there is also a real increase in prevalence due to an unidentified cause remains an open question. Interestingly, in contrast to scientific opinion that the increase in ASD prevalence is mainly due to changes in awareness and diagnostic practices, many laypeople continue to believe that the increase is due to increased exposure to new environmental, medical, and technological hazards (e.g., vaccinations, cell phone towers) (Russell, Kelly, & Golding, 2009).

ASD is found in all social classes and has been identified worldwide. It is about four to five times more common in boys than in girls, a ratio that has remained fairly constant over the years, even with increasing prevalence estimates (CDC, 2014). The sex difference is most apparent among children with IQs in the average to above-average range, perhaps being as high as 10:1 in higher-functioning individuals. However, among children with ASD and profound ID, the numbers of boys and girls are similar. Thus, although girls are less often affected by ASD than are boys, when they are affected, they tend to have more severe intellectual impairments (Dworzynski et al., 2012). Girls with ASD who do not have an intellectual impairment are more likely to be formally diagnosed at a later age than boys (Giarelli et al., 2010). Girls with comparable high levels of ASD symptom severity as boys are also less likely to be diagnosed, suggesting that there is a bias in diagnosis or that in the absence of co-occurring intellectual or behavioral deficits girls may be better able to cope with the same level of ASD symptoms (Constantino & Charman, 2012). In general, the clinical manifestations of ASD are quite similar for boys and girls, although there may be some differences in their cognitive profiles (Carter et al., 2007). For example, it has been found that girls with ASD engage in more pretend play than do boys, suggesting that impairment in pretense may be less of a problem for girls (Knickmeyer, Wheelwright, & Baron-Cohen, 2008). Findings also suggest brain differences underlying ASD in males and females (Lai et al., 2013).

In considering the high ratio of males to females with ASD, Simon Baron-Cohen (2002, 2009) proposed the *extreme male brain theory of ASD*. Those with ASD are presumed to fall at the extreme high end of a continuum of cognitive abilities associated with systemizing (understanding the inanimate world), and at the extreme low end of abilities associated with empathizing (understanding our social world). Both abilities are present in all males and females, but males are presumed to show more systemizing and females more empathizing. Frequent interests and behaviors that occur among individuals with ASD (e.g., attention to detail, collecting, an interest in mathematics, mechanical knowledge, and scientific and technical information) are presumed to reflect an extreme on the systemizing dimension of the male brain, and a relative absence of empathizing (e.g., mindreading, empathy, eye contact, and communication) (Baron-Cohen et al., 2003). Interestingly, one brain study found that females with ASD displayed masculinization at the neural level (Lai et al., 2013). The extreme male brain theory is intriguing but somewhat controversial. Further research into the neurocognitive aspects of these dimensions in individuals with ASD will be needed before we can infer that they are "from Mars and not Venus."

Rates of ASD are comparable across different racial and ethnic groups. Where differences are found, prevalence is higher among non-Hispanic white children than among members of other groups, most likely because of underidentification in non-Hispanic black and Hispanic children (CDC, 2012a). Different racial and ethnic groups do not differ in core symptoms of or risk factors for ASD (CDC, 2012a; Cuccaro et al., 2007). However, African American children are nearly three times more likely than white children to receive another diagnosis such as ADHD or adjustment disorder before being diagnosed with ASD, and they are nearly three times more likely to experience delays in receiving intervention (Mandell et al., 2007). Societies differ in how they integrate ASD into their cultural frameworks. For example, in contrast to viewing ASD as a disorder, some cultures view children with ASD as having special skills or as being more in touch with the spirit world. Cultural views range from those of the Navajo, who embrace their children with ASD as being blessed (Kapp, 2011), to the South Koreans, who may hide their children with ASD to protect siblings from being considered tainted and unmarriageable (Grinker, 2007).

## Age at Onset

The *diagnosis* of ASD is usually made in the preschool period or later. However, most parents of children with ASD become seriously concerned a year or more before a diagnosis is made, typically during the months preceding their child's second birthday (McConkey, Truesdale-Kennedy, & Cassidy, 2009) At this time, their child's lack of progress in language, imaginative play, and social relations stands in sharp contrast to rapid developments in these areas by other children of the same age. Although deficits of ASD become increasingly noticeable around age 2, elements are probably present and noticed earlier, as reflected in Anne-Marie's solemn reaction to her first birthday party (Yirmiya & Charman, 2010).

**ANNE-MARIE**

### First Birthday

We were celebrating Anne-Marie's first birthday and had just paraded in, bearing the cake with much fanfare. Daniel, her big brother, almost two and a half years old, and greatly excited, joined us in singing. Anne-Marie, in her high-chair, gazed solemnly at the cake, her baby body still, her mouth unsmiling. … I couldn't help once again making a silent comparison to her brother, who at his first birthday party had squealed with delight. … Who knows, really, what the first sign was, at what point Anne-Marie began to slip away from us? Was it around that first celebration, or after or before? (Based on Maurice, 1993b)

At present, the period from 12 to 18 months seems to be the earliest point in development at which ASD can be reliably detected. For example, an interesting study found that children with ASD generally did not show signs of the disorder at 6 months of age, but between 6 and 12 months they failed to gain new social skills or showed a loss of previously acquired ones (Ozonoff et al., 2010). Most children with ASD showed a subtle and gradual loss of specific social skills between 6 and 18 months that went unnoticed by parents. These findings suggest that traditional views that symptoms of ASD are present at birth or that the child shows dramatic regression at a later age may not accurately depict how ASD develops. Instead, the onset of symptoms may be more accurately represented as being on a continuum based on the amount and timing of loss of previously acquired skills (Ozonoff et al., 2009). Consistent with this view are recent findings that an early decline in eye contact over the first 6 months may precede the gradual loss of specific social skills that occurs from 6 to 12 months of age. Children who later developed ASD and typically developing children did not differ in eye contact in the first month of life, but for those who later developed ASD, eye contact gradually decreased over the first 6 months of life (Jones & Klin, 2013).

Currently, diagnoses of ASD that are made around age 2 to 3 years are stable for most children (Bryson, Rogers, & Fombonne, 2003; Kleinman et al., 2008). However, with increasing research into key early indicators, systematic screening and direct observation of infants at risk for ASD (e.g., those with older siblings with the disorder), and universal screening of young infants, it is likely that ASD can and will be reliably detected at earlier ages, particularly for those with low IQ (Bryson et al., 2008; Oosterling et al., 2010; Pierce et al., 2011). Features of atypical development, that are very similar to those found in ASD but are less severe, have recently been detected in infant siblings of children with ASD by the infants' first birthday (Ozonoff et al., 2014). Possible early indicators of ASD may include: "uses few gestures to express social interest," "doesn't respond when name is called," "rarely makes eye contact when interacting," "limited babbling, particularly in a social context," and "displays odd or repetitive ways of moving hands and/or fingers" (Zwaigenbaum et al., 2009). Children with ASD have been found to differ from typically developing children on most of these indicators between the ages of 12 and 24 months. However, in one study, only early communicative gestures were found to distinguish children with ASD from those with developmental delay or language impairment (Vaness et al., 2012). As part of its campaign to raise awareness about the importance of early identification for intervention, the American Academy of Pediatrics (AAP) recommended that all children be screened for ASD at 18 months and 24 months (Hampton, 2007). The organization Autism Speaks has a video glossary on its website (www.autismspeaks.org) where you can view fascinating video clips that show some of the early red flags for ASD as well as examples of commonly used treatments. To date, efforts to implement screening approaches in community settings have had positive results. However, the extent to which these results have brought about reduced time to diagnosis and enrollment in services has not yet been tested (Daniels et al., 2013).

## Course and Outcome

Children with ASD develop along different pathways. Some show abnormal behavior soon after birth; some, 25% or more, show seemingly normal development for the first year or longer followed by *regression* (the loss of previously acquired language and social skills, with an onset of ASD) (Parr et al., 2011); while others appear to improve significantly over time (Fein et al., 2013). The symptoms of children with ASD change over time. Most symptoms gradually improve with age, even though children continue to experience many problems. During adolescence, some symptoms, such as hyperactivity, self-injury, and compulsivity, may worsen (Spector & Volkmar, 2006). During later adolescence and adulthood, abnormalities such as stereotyped motor movements, anxiety, and socially inappropriate behaviors are common, even in high-functioning individuals; these individuals also often experience loneliness, social disadvantage and exclusion,

and work difficulties (Howlin, 2013). Complex obsessive–compulsive rituals may develop, and talking may be characterized by idiosyncratic and perseverative speech, monotonous tone, and self-talk (Newsom & Hovanitz, 2006).

Findings from early studies of children with ASD who received limited help indicated that an overwhelming majority (70% or more) showed poor outcomes with limited progress and continuing handicaps that did not permit them to lead an independent existence (Lotter, 1978). More recent follow-up studies report slightly better, but quite similar, outcomes (Eaves & Ho, 2008; Howlin et al., 2004; Howlin et al., 2013). Very few adults with ASD achieve high levels of independence. Most remain quite dependent on their family and other support services, with few friends and no permanent job (Roux et al., 2013). These adults continue to display problems in communication, stereotyped behaviors and interests, and poor reading and spelling abilities. Overall, children with better language skills, higher intellectual ability, and higher scores on measures of reciprocal social interaction at the time of diagnosis show better long-term outcomes, but outcomes can be variable even for high-functioning individuals (Bennett et al., 2008). It is possible that better long-term outcomes will be achieved by more recent generations of children with ASD who were diagnosed at a younger age, are higher-functioning, and received intensive early intervention. However, future longitudinal research will be needed before we know. Whatever the outcome, the reality is that children with ASD grow up, and most will continue to require age-appropriate supports and services. To date, far greater attention has been given to research, programs, and services for children with ASD than to adolescents and adults with ASD. Further efforts to address the needs of older individuals with ASD are sorely needed (Bailey, 2012; Wilczynski, 2013).

## Section Summary

### Prevalence and Course of ASD

- ASD is a disorder that affects as many as 1 in 68 children, or between one and two percent. It is four to five times more common in boys than in girls. ASD is found across all social classes and has been identified in every country in which it has been studied.
- ASD is most often identified around age 2 years or older, although elements are present at a much earlier age.
- Children with ASD may develop along different pathways. Some show abnormal behavior soon after birth; others show seemingly normal development for the first year or longer followed by regression; while others appear to improve significantly over time.
- Most children with ASD show gradual improvement of their symptoms with age, although they continue to display social impairments that make them different from other people throughout their lives.
- The two strongest predictors of adult outcomes in children with ASD are intellectual ability and language development.

# CAUSES OF ASD

No single abnormality can account for all the impairments associated with ASD, or for the many forms of the disorder, ranging from mild to severe. Although the precise causes of ASD are still not known, our understanding of possible mechanisms has increased dramatically (Klinger et al., 2014). These advances are evident when we consider that, not long ago, autism was being attributed to cold and unloving parents. It is now generally accepted that ASD is a biologically based neurodevelopmental disorder with multiple causes involving genetic and environmental risk factors (Faja & Dawson, 2013). To understand ASD, we must consider problems in early development, genetic influences, and neuropsychological and neurobiological findings.

## Problems in Early Development

Children with ASD experience more health problems prenatally, at birth, or immediately following birth than do other children. Although not proven as independent risk factors, prenatal and neonatal complications such as preterm birth, bleeding during pregnancy, toxemia (blood poisoning), viral infection or exposure, a lack of vigor after birth, and others have been identified in a small percentage of children with ASD (Gardener, Spiegelman, & Buka, 2009, 2011). One study found that very preterm birth (gestational age of <26 weeks) was associated with a much higher rate of ASD, with a prevalence of 8% diagnosed by age 11 (Johnson et al., 2010). Other risk factors that affect the prenatal environment may place the fetus at increased risk for ASD. These include increased maternal age, in vitro fertilization, maternal use of prescription and nonprescription drugs, toxic chemicals in the environment during pregnancy, maternal fever or maternal illnesses such as diabetes or infections during pregnancy, chronic hypertension, and prepregnancy obesity (Szatmari, 2011). For example, with regard to parental age, a study of over 7 million

children in California found that older mothers and fathers were more likely to have a child with ASD than were younger parents (Grether et al., 2009). It was found that an increase of 10 years in maternal age was associated with a 38% greater risk of ASD and that the same increase in paternal age was associated with a 22% greater risk. The relationship between increasing parental age and ASD suggests that age could be a contributing factor in the increase in ASD, and also raises questions about possible mechanisms including age-related gene variants or epigenetic dysfunction (Sandin et al., 2012). Exposure to antidepressant medication (SSRIs) during the first trimester of pregnancy has also been found to increase the risk of ASD (Croen et al., 2011). Although problems during pregnancy and birth may not be the primary cause of ASD, they do suggest that fetal or neonatal development has been compromised (Szatmari, 2011).

A controversial and widely publicized proposal was that some cases of ASD in children who speak only a few words and have other social–communicative behaviors that disappear in the second year of life might be linked to vaccinations. Two hypotheses attracted the most attention. The first incriminated the measles components of combination vaccines for measles–mumps–rubella (MMR) (Wakefield et al., 1998; *retracted* February, 2010). The second lay blame on exposure to ethyl mercury (thimerosal), a preservative used in other vaccines (Ball, Ball, & Pratt, 2001). Both hypotheses claimed that the apparent ASD "epidemic" coincided with the introduction of MMR vaccines and/or increased exposure to thimerosal as a result of the increased number of recommended childhood vaccinations in the first 3 years of life. However, current scientific evidence does not support an association between MMR vaccines or thimerosal and ASD (Fombonne, 2008; Institute of Medicine, 2004; see also The Editors of The Lancet, 2010). Nevertheless, a large number of parents of children with ASD still believe that their child's disorder was caused by vaccinations (Harrington et al., 2006).

## Genetic Influences

Studies of specific chromosomal anomalies and gene disorders, findings from family and twin studies, and specific gene studies indicate a substantial role for genetic factors in ASD (Rutter, 2005). However, despite strong evidence for a genetic contribution and some noteworthy findings emerging from tests of hundreds of genes, the rate of progress in gene discovery has been slow, and the genetic architecture of ASD remains largely unknown (El-Fishawy & State, 2010).

### Chromosomal and Gene Disorders

The discovery of the fragile-X anomaly (see Chapter 5) in about 2% to 3% of children with ASD led to increased attention to this and other chromosomal defects that might be related to ASD (Turk & Graham, 1997). In general, individuals with ASD have an elevated risk, about 5%, for chromosomal anomalies (Barton & Volkmar, 1998; Dykens & Volkmar, 1997). However, these anomalies alone do not indicate the specific gene sites underlying the disorder, because ASD has been associated with anomalies involving several chromosomes (Freitag et al., 2010).

ASD is also associated with *tuberous sclerosis,* a rare single-gene disorder. The manifestations of this disorder can vary widely from mild to severe; they may include neural deficits, seizures, and learning disabilities. Most cases are derived from de novo mutations, cases in which no family history of the disorder existed (Bailey, Phillips, & Rutter, 1996). About 25% or more of children with tuberous sclerosis also have ASD. This makes the association between ASD and tuberous sclerosis greater than that for any other genetically based condition.

### Family and Twin Studies

Some studies have found that as many as 15% to 20% of siblings of individuals with ASD also have the disorder, a number nearly twice that seen in earlier reports (Ozonoff et al., 2011). Also, at 3 years of age, high-risk siblings who do not receive a diagnosis of ASD show greater severity of symptoms of ASD and lower levels of developmental functioning than do low-risk children (Messinger et al., 2013). In addition, family members of children with ASD display higher-than-normal rates of social and language deficits and unusual personality features that are very similar to those found in ASD but are less severe (Gerdts et al., 2013; Ozonoff et al., 2014). Referred to as the *broader autism phenotype,* these deficits include social oddities such as aloofness, lack of tact, and rigidity; pragmatic language problems such as overcommunicativeness or undercommunicativeness; and poor verbal comprehension. Family members with the broader phenotype do not, however, display the atypical language (e.g., pronoun reversal), extreme stereotyped repetitive behavior, or the ID and epilepsy that are often associated with a formal diagnosis of ASD (Rutter, 2000). These findings are consistent with a general family risk for ASD that is genetically mediated. In addition, a growing number of studies have reported similar neurophysiological correlates (e.g., atypical brain activation, reduced white matter) for children with ASD *and* their "unaffected siblings," suggesting a family susceptibility to ASD involving a

wide array of brain regions and networks (Barnea-Goraly, Lotspeich, & Reiss, 2010; Belmonte, Gomot, Baron-Cohen, 2010).

Twin studies have reported concordance rates for ASD in identical twins ranging from 70% to 90%, in contrast to near-zero rates for fraternal twins (Rutter, 2005). These findings indicate that the heritability of an underlying liability for ASD may be as high as 90% and suggest that almost all the variance in the expression and stability of ASD over time can be attributed to inherited genetic influences (Freitag et al., 2010; Holmboe et al., 2013; Lichtenstein et al., 2010). One exception is a finding that a large proportion of the variance in susceptibility to ASD could be explained by *shared environmental experiences* (58%), with heritability accounting for a smaller amount (38%) (Hallmayer et al., 2011). If replicated, this finding suggests that susceptibility to ASD may have a moderate genetic heritability component and a substantial shared twin environmental component. To date, the major focus of research on ASD has been on genetic influences, with minimal attention paid to environmental factors. The finding that shared environmental experiences have a significant influence on ASD susceptibility suggests that environmental risk factors occurring prior to or by the end of the first year of life could play an important role. Thus, further research into problems in early development of the type discussed previously, such as low birth weight, multiple births, maternal infections during pregnancy, and parental age, may help to advance our understanding of ASD (Hertz-Picciotto, 2011).

### *Molecular Genetics*

New research using molecular genetics has pointed to particular areas on many different chromosomes as possible locations for *susceptibility genes* for ASD (Klinger et al., 2014). Susceptibility genes are causally implicated in the susceptibility to ASD but do not cause it directly on their own. Although several searches for major ASD genes have been undertaken, they have not yielded consistent results (Freitag et al., 2010). No single gene has been found to be relevant for most cases of ASD. Inconsistent findings in gene studies may be due to the considerable etiologic heterogeneity within ASD and the diverse ways in which it appears. Rather than a single gene, ASD is associated with rare mutations that have a strong effect for a very small proportion of individuals with ASD who have such genes, and a few common variants of small effect in several genes that seem to be a factor for many cases of ASD (Anney et al., 2012). Thus, ASD is likely to be a complex genetic disorder resulting from both rare mutations and simultaneous genetic variations (e.g., submicroscopic deletions or insertions of segments of DNA) in multiple genes (El-Fishawy & State, 2010). Moreover, the expression of ASD gene(s) may be influenced by environmental factors (e.g., exposure to drugs, maternal illness)—a "a second hit" that occurs primarily during fetal brain development. The possible role of such gene–environment interactions (GxE) and also gene–environment correlations (rGEs) in ASD requires further study (Corrales & Herbert, 2011; Meek et al., 2013).

Finally, there are a number of situations in which epigenetic dysregulation (changes in gene expression caused by mechanisms other than changes in the underlying DNA sequence) may be associated with the development of ASD, For example, co-morbid genetic conditions such as fragile-X syndrome or genes or genomic regions exhibiting abnormal epigenetic regulation (Grafodatskaya et al., 2010) may be associated with ASD. Thus, in searching for genetic alterations responsible for ASD, it may also be necessary to look beyond mutations and variations in specific genes into epigenetic regulation of gene function (Rangasamy, D'Mello, & Narayanan, 2013).

## Brain Abnormalities

Although there is no known biological marker for ASD, impressive advances have been made in documenting the neurobiological basis of the disorder (Neuhaus, Beauchaine, & Bernier, 2010; Pelphrey et al,, 2011). Current research suggests that the behavioral features of ASD may result from abnormalities in brain structure and functioning that are consistent with early disturbances in neural development, possibly tracing back to prenatal development (Minshew, Johnson, & Luna, 2000; Stoner et al., 2014; Xiao et al., 2014). Importantly, although many brain regions are implicated, the disorder does not seems to lie in an abnormality localized in one part of the brain. Rather, it results from a lack of normal connectivity across brain networks that underlie the core features of ASD (Rudie et al., 2012; Stigler et al., 2011; Waterhouse & Gillberg, 2014).

Neuropsychological impairments in ASD occur in many domains, including verbal intelligence, orienting and selective attention, memory, pragmatic language, and executive functions (Dawson et al., 2002). The widespread nature of these deficits suggests that multiple regions of the brain are involved at both the cortical and subcortical levels (Happé & Frith, 1996). The types of neuropsychological deficits also vary as a function of the severity of the child's disorder. For example, low-functioning children with ASD may show impairments in basic memory functions, such as visual recognition memory, which are mediated by the brain's

medial temporal lobe (Barth, Fein, & Waterhouse, 1995). In contrast, high-functioning children may have more subtle deficits in working memory or in encoding complex verbal material, suggesting the involvement of higher cortical functions (Dawson, 1996).

### Biological Findings

Brain imaging studies have looked for structural and functional abnormalities in brain development or consistently localized brain lesions associated with the symptoms of ASD (Williams & Minshew, 2007). In terms of abnormal brain development, one longitudinal study examined brain growth at multiple points in time from ages 1.5 to 5 years in normal toddlers and toddlers who received a confirmed diagnosis of ASD at around 4 years of age (Schumann et al., 2010). The toddlers with ASD showed evidence of overgrowth of cerebral gray and white matter in all regions by age 2.5 years, around the time that their clinical symptoms began to appear. Almost all brain regions were found to develop at an abnormal rate; this finding was more pronounced in girls.

In terms of localized brain abnormalities, studies have consistently identified structural abnormalities in the cerebellum and in the medial temporal lobe and related limbic system structures (Bauman & Kemper, 2005; Courchesne et al., 2007). The *cerebellum,* a relatively large part of the brain located near the brain stem, is most frequently associated with motor movement. However, it is also partially involved in modulating emotion, language, executive function, learning, thought, and attention (Hodge et al., 2010). Specific areas of the cerebellum are found to be significantly smaller than normal in youngsters with ASD, particularly in those with a higher level of functioning (Scott et al., 2009). It has been proposed that cerebellar abnormalities may underlie the problem that children with ASD have in rapidly shifting their attention from one stimulus to another (Courchesne et al., 2007).

A second localized brain abnormality is in the medial temporal lobe and connected limbic system structures such as the amygdala and hippocampus (Groen et al., 2010; Johnson et al., 2013; Schumann & Amaral, 2009). These areas of the brain are associated with functions that are often disturbed in children with ASD—for example, learning, memory, and emotion regulation (Mazefsky et al., 2013). The amygdala plays an especially important role in recognizing the emotional significance of stimuli, in orienting toward social stimuli, in the perception of eye gaze direction, and, along with the hippocampus, in long-term memory (Schulkin, 2007). Findings from brain scan studies suggest that there are both structural and functional abnormalities in the amygdala of those with ASD (although not in all cases) (Monk, 2008). For example, enlargement of the amygdala in toddlers with ASD is correlated with the severity of their social and communication impairments (Schumann et al., 2009).

Studies of brain metabolism in individuals with ASD suggest decreased blood flow in the frontal and temporal lobes. Studies have also found a decrease in the functional connections between cortical and subcortical regions and a delay in the maturation of the frontal cortex, as indicated by reduced cerebral blood flow in the frontal brain regions of preschool-age children with autism (Zilbovicius et al., 1995).

In relation to connections among specific brain regions and tracts (parts of the brain that carry signals from one brain region to another and allow communication between the two hemispheres), abnormalities in the corpus callosum, frontal lobe cortex, and other brain tracts have been found in youngsters with ASD (Kumar et al., 2010; Shukla, Keehn, & Muller, 2011) and in their unaffected siblings (e.g., Barnea-Goraly et al., 2010). Reductions in the area of the corpus callosum (the main fiber tract connecting the hemispheres) have been found in children with ASD, supporting the role of abnormalities in connectivity in the disorder (Frazier & Hardan, 2009). Another study found significantly reduced interhemispheric connectivity specific to brain regions with functional relevance to ASD (Anderson et al., 2011). Postmortem studies of single axons in prefrontal regions of the brain have revealed a disconnection of long-distance brain pathways, excessive connections between adjacent areas, and inefficiency in pathways for emotion, which may account for why individuals with ASD have difficulty in shifting attention, engage in repetitive behavior, and avoid social interactions (Zikopoulos & Barbas, 2010).

Atypical patterns of connectivity in the *default mode network* (DMN) have also been found in children with ASD (von dem Hagen et al., 2013). The DMN is a network of brain regions that are active when the individual is not focused on the external world and the brain is at wakeful rest—focusing on internal tasks such as daydreaming, thinking about the future, retrieving autobiographical memories, and assessing others' perspectives. These findings are important, since the DMN includes brain regions (e.g., medial prefrontal cortex, medial temporal lobe) hypothesized to be involved in the higher-order social–cognitive processes that are impaired in children with ASD—for example, memory, theory of mind, and integration of information (Stigler et al., 2011).

## ASD as a Disorder of Risk and Adaptation

Based on the causal factors we have discussed, a model of risk and adaptation is needed to understand how

ASD develops. Genetic and environmental factors lead to abnormalities in brain development, which in turn lead to generalized disturbances in how the child processes information and interacts with his or her environment (Faja & Dawson, 2013). These disturbances are likely to disrupt critical input affecting brain development during early periods of sensitivity (Dawson et al., 2002). Therefore, the relationship between the child's early risk for ASD and later outcomes will be mediated by alterations in how the child interacts with and adapts to his or her environment. For example, Wan et al. (2013) found that the quality of interaction between infants at risk for ASD and their caregivers at 12 to 15 months correlated with an autism diagnosis at age 3 years. Depending on the interaction between early risk factors and the environment in which the child develops, different children will follow different developmental pathways. Although pathways may change at any point in development, the longer the child is on a maladaptive pathway, the more difficult it is for change to occur. Thus, as we discuss in the next section on treatment, the earlier the risk for ASD can be identified and the sooner intervention begins, the greater the likelihood that the child will have a better outcome (Sigman, Spence, & Wang, 2006).

## Section Summary

### Causes of ASD

- ASD is a biologically based neurodevelopmental disorder that may result from multiple causes.
- Some children with ASD experience prenatal and neonatal complications such low birth weight, bleeding during pregnancy, toxemia (blood poisoning), viral infection or exposure, and a lack of vigor after birth.
- ASD is a genetic disorder, although specific genes with large effects have not been identified. More likely, ASD is a complex genetic disorder resulting from rare mutations and simultaneous genetic variations in multiple genes. Shared environmental experiences and epigenetic factors may also be involved.
- Nonautistic relatives of individuals with ASD display higher-than-normal rates of social, language, and cognitive deficits that are similar in quality to those found in ASD, but are less severe and are not associated with intellectual deficits or epilepsy.
- Neuropsychological impairments occur in many areas of functioning, including intelligence, attention, memory, language, and executive functions.
- Structural abnormalities in the cerebellum and the medial temporal lobe, prefrontal cortex, and related limbic system structures have been found.
- ASD is not represented by a localized abnormality in one part of the brain but rather by a lack of normal connectivity and communication among brain networks that underlie the core features of ASD.
- The relationship between the child's early risk for ASD and later outcomes will be mediated by how the child interacts with and adapts to his or her environment.

## TREATMENT OF ASD

*I have not counted the trials of medication, the diets, … the behavioral programs. If they total five hundred, there are five hundred fewer to try. … I'm a believer. … I believe my son can get well.*

—From Swackhamer (1993)

*Autistic people suffer from a biological defect. Although they cannot be cured, much can be done to improve their lives.*

—U. Frith (1997)

These two sentiments—the first by the mother of a child with ASD, the second by an ASD expert—underscore the promise, pain, and uncertainty that surround efforts to help children with ASD and their families. Parents of children with ASD report having tried, on average, seven to nine different therapies for their child, and are currently using four to six (Goin-Kochel, Myers, & Mackintosh, 2007). It has been estimated that about *400 different treatments* are being used by individuals with ASD (Interactive Autism Network, 2011). The fact that no one treatment has been successful in eliminating the symptoms of ASD makes many parents vulnerable to new claims of dramatic improvements. This is especially true for a dizzying array of widely publicized treatments such as vitamins, nutritional supplements, special diets (e.g., gluten- and casein-free diets), medications (e.g., antipsychotics, stimulants, antidepressants), hyperbaric oxygen therapy (sealing the child in a pressurized oxygen chamber), chelation therapy (removal of heavy metals from the body), weighted vests (to provide "calming" stimulation), secretin (a hormone that controls digestion), immunotherapy (use of substances that target a variety of hypothesized but as yet unproven immune system abnormalities), auditory training, music therapy, dance/movement therapy, repetitive transcranial magnetic stimulation (stimulating key motor cortical sites to improve motor activity), sensory integration, facilitated communication, horseback riding, use of trained service dogs, and even swimming with dolphins (Schreck, Russell, & Vargas, 2013). Unfortunately, most of these treatments have not lived up to

their claims under close scientific scrutiny, and some may have harmful effects (Research Autism, 2013; Umbarger, 2007).

Although behavioral, educational, and medical treatments may improve learning and behavior, and may permit a few children to achieve near-normal functioning, there is no known cure for ASD. The goals for most treatments are to minimize the core problems of ASD, maximize the child's independence and quality of life, and help the child and family cope more effectively with the disorder (Myers, Johnson, and the Council on Children with Disabilities, 2007). These goals can be facilitated by treatments designed to enhance development and learning, to reduce associated maladaptive behaviors, and to educate and support parents in meeting these goals. Understanding parents' beliefs about the causes of their child's ASD may also be important for treatment (Dardennes et al., 2011).

Promising new programs of early intervention, community-based education, and community living options are all reasons for optimism about improving outcomes for children with ASD (Rogers & Wallace, 2011; Volkmar et al., 2014). The most benefit is likely to come from developmentally oriented, early behavioral interventions that involve parents and that are used along with special educational methods (Rutter, 2006b). Most children treated using these newer evidence-based methods show significant gains in language, communication, and measured IQ and a modest reduction in the severity of the core symptoms of autism (Virués-Ortega, 2010; Young et al., 2010). However, questions remain concerning how intensive the interventions need to be (e.g., 20 vs. 40 hours per week), how much change can be achieved, and the extent to which changes can be directly attributed to the intervention. Additional controlled studies are needed before long-term outcomes can be fully assessed (Charman, 2011; Vismara & Rogers, 2010).

## Overview

### EMILIE

### A Full-Time Job

When Emilie was 2 she was diagnosed with autism, Emilie's mother recalled, her eyes brimming with tears. "We've been relying on ourselves ever since." Emilie's mother and father have read about children with autism who became accomplished scientists and musicians—but progress for Emilie, now age 4, has been slow. Two months ago they hired a specialist to teach them a new one-on-one approach for getting through to Emilie with a reward system. Pictures of food are taped to hallway walls. On the fridge is a cutout of a glass of milk. After years of shrieking and kicking for what she wants, Emilie is learning to express her needs. When she points to what she wants, she gets a reward—a potato chip or an activity she likes. Every afternoon mother and daughter spend 2 hours on the floor, face to face, their legs interlocked. "Listen to maman, Emilie. Look at me. Look at me. Say 'yes.' Say 'yes.' Do you like chips, Emilie? You can have one if you just say the word, 'yes.'"

Emilie's mother coaxes patiently, firmly, holding out a bowl of chips. But Emilie runs to the radiator and climbs it, teetering there. When her mother pulls her down Emilie shrieks, kicks, and falls to the floor crying. In a minute the episode is over, and the lesson begins again. This time Emilie looks at her mother, says "yes," and holds out her hand for a chip. "Bravo, sweetheart. You did it. I knew you could," her mother beams. Emilie's mother has used the reward system to build Emilie's vocabulary to 22 words. That, to her parents, has been a monumental breakthrough.

"We have to motivate her," says her mother, whose only respite is an evening out once or twice a month with

AP Images/Ed Betz

**The demands of parenting a child with ASD are considerable.**

her husband. "If we let her be, she'd just climb or hide under the cover all day long. That's my nightmare, that she'll end up in a psychiatric hospital, withdrawn from the world. I can see that we are slowly beginning to get through to her," Emilie's mother says with a deep sigh. "She didn't pay any attention to us at all before. She never showed any affection or made eye contact. But now she looks at me and says 'maman.' Sometimes she hugs me. It doesn't happen every day. But it grabs my heart when it does."

From Susan Semenak, *The Gazette*, November 21, 1996, pp. A1 and A15. Reprinted by permission.

Emilie's case captures the demands, frustrations, aspirations, and hopes of a family trying to do the best possible for their child with ASD. A number of treatments are available for helping children with ASD, such as Emilie, and their families. These treatments focus on the specific social, communication, behavioral, and cognitive deficits of ASD that we have discussed throughout this chapter. They include strategies for engaging children in treatment; decreasing disruptive behaviors; teaching appropriate social behavior, including joint attention, imitation, and reciprocal interaction; increasing functional, spontaneous communication; promoting cognitive skills such as symbolic play and perspective taking; and teaching adaptive skills that prepare the child for increased responsibility and independence. Family interventions enable parents to participate fully in their child's treatment and to cope with the substantial demands and parenting-related stress associated with raising a child with ASD (Estes et al., 2014; Rivard et al., 2014). In addition, educational interventions and speech and language therapy are commonly used. Also, for some children, antipsychotic medications (e.g., risperidone, aripiprazole) may help to decrease interfering and challenging behaviors and symptoms such as irritability, severe tantrum behavior, physical aggression, and repetitive behaviors (Volkmar et al., 2014), particularly when they are combined with intensive behavioral intervention (Arnold et al., 2012; Frazier et al., 2010). However, the effectiveness of these medications must be balanced against their known adverse effects, such as weight gain or liability to cause metabolic disorders (McPheeters et al., 2011).

Because children with ASD have great difficulty making changes and generalizing previously learned skills to new environments, these areas must be directly addressed in treatment. It is also critical that treatment be tailored to meet the needs of the individual child and the family, thus making it possible for each child to meet his or her full potential. In the following paragraphs we highlight how several of the treatment components mentioned previously are implemented.

### Initial Stages

Initially, treatment focuses on building rapport and teaching the child learning-readiness skills. Various procedures help the child feel comfortable being physically close to the therapist and to identify rewards to strengthen the child's social behavior, affection, and play. Imitating the child's use of toys may increase eye contact, touching, and vocalizations directed toward the therapist. Prompting the child to engage in play with a preferred toy may decrease social avoidance.

Children with ASD must learn to sit in a chair, come when called, and attend to their teacher if they are to progress. These readiness skills are taught using two approaches. The first is a step-by-step approach to presenting a stimulus and requiring a specific response, referred to as **discrete trial training**. The second attempts to strengthen behavior by capitalizing on naturally occurring opportunities, referred to as **incidental training.** Most interventions use a combination of these approaches (Ghezzi, 2007).

### Reducing Disruptive Behavior

Young children with ASD display many disruptive and interfering behaviors, such as tantrums or throwing objects, as well as self-stimulation, aggression, and self-injury. These behaviors are common reactions to demands on the child that are made early in treatment, and they must be eliminated if the child is to learn more adaptive forms of social interaction and communication. Many procedures are effective in eliminating disruptive behavior, including rewarding competing behaviors, ignoring the behavior, and mild forms of punishment.

### Teaching Appropriate Social Behavior

Teaching appropriate social behavior is a high treatment priority (White, Keonig, & Scahill, 2007). The salience of social cues may be increased by pairing people with whom the child has contact with actions, activities, and events that the child finds pleasant or useful. Younger children are also taught ways to express affection through smiling, hugging, tickling, or kissing—behaviors that enable them to return the affection they receive from others. Other ways to enhance social interaction include teaching social toy play, social pretend play, and specific social skills such as initiating and maintaining interactions, taking turns and sharing, and including others in activities. Developmental and relationship approaches foster parental use of child-centered responsive interactions that embed numerous opportunities for teaching social and emotional behaviors into play. Parent-assisted Children's Friendship Training programs for school-age children

with ASD target conversational skills, peer group entry skills, developing friendship networks, good sportsmanship, good host behavior during play dates, and handling teasing (Laugeson et al., 2012). Group social skills interventions have also been shown to improve social behaviors in high functioning children with ASD (Derosier et al., 2010).

One strategy for teaching appropriate social behavior to children with ASD involves teaching normal or mildly handicapped peers to interact with them. Peers are taught to initiate age-appropriate social behaviors such as playing with toys, commenting about activities, or acknowledging their partner's responses. Teachers may signal and reward the peers' social initiations with the child with ASD. Other strategies use prompts and rewards for teaching the child with ASD to initiate interactions, and in some cases to involve siblings as trainers (Kohler, Strain, & Goldstein, 2005).

### *Teaching Appropriate Communication Skills*

Several strategies are used to help children with ASD communicate more appropriately. **Operant speech training** is a step-by-step approach that first increases the child's vocalizations and then teaches imitation of sounds and words, the meanings of words, labeling objects, making verbal requests, and expressing desires. The emphasis is on teaching the child to use language more spontaneously and more functionally in everyday life situations to influence others and to communicate better (Newsom & Hovanitz, 2006).

### *Executive Function Intervention*

Recent interventions have also focused on the executive functioning deficits displayed by children with ASD. One such school-based program, Unstuck and On Target [UOT], uses cognitive–behavioral strategies to reduce insistence on sameness and to teach flexibility, goal-setting, and planning (Kenworthy et al., 2013). A controlled study compared third- and fifth-graders with ASD and average or above-average intellectual ability who received the UOT intervention with a comparable group of children with ASD who received social skills training. After intervention, children in both groups improved, but those receiving UOT showed significantly greater improvement in their problem-solving, flexibility, and planning/organizing skills. When observed in their classroom, children who had received UOT were better able to follow rules, make transitions, and be more flexible (Kenworthy et al., 2013). Both groups made equivalent gains in social skills. These findings are promising in showing that children with ASD with average or above-average intellectual ability can learn higher-level cognitive skills and apply them in a mainstream classroom.

Nir Alon/Alamy

**The mother of Max, a 4-year-old boy with ASD, spends hours each day teaching communication skills to her son.**

## Early Intervention

As methods to identify ASD at a very young age are developed, possibilities for effective early intervention with infants and toddlers increase dramatically (Wallace & Rogers, 2010). The promise of early intervention derives, in part, from the plasticity of neural systems early in development (Mundy & Neal, 2001) and the fascinating hypothesis that providing very young children with ASD with intensive and highly structured experiences may alter their developing brains in ways that permit outcomes that are not otherwise possible (Dawson, 2008). In an important investigation, Dawson et al. (2012) found that 18-to-30–month-old children with ASD who received early intervention showed significantly greater improvement than comparison children after intervention in their ASD symptoms, IQ, language, and adaptive and social behaviors. In support of the hypothesis of altering the developing brain through early intervention, at 48 to 77 months of age, children who had received the early intervention also displayed more normal patterns of brain activation when viewing faces versus objects than did comparison children, and these brain changes were associated with improved social behavior. Nowadays, whenever possible, intensive interventions for children with ASD begin before the age of 3—the earlier the intervention, the better the outcome is likely to be (Rogers et al., 2012).

Comprehensive early-intervention programs include many of the specific treatments for ASD that we have described (Harris, Handleman, & Jennett, 2005). A variety of early-intervention programs are available, some based on a learning/behavioral model (e.g., Applied Behavior Analysis; Lovaas & Smith, 2003; Smith, 2011), others

based on a structured teaching model (e.g., TEACCH; Mesibov, Shea, & Schopler, 2005), and others based on developmental (e.g., Early Start Denver Model; Dawson et al., 2010) and/or relationship-focused (DIR or Floor Time; Greenspan & Wieder, 2006) approaches. Although these and other programs may differ in philosophy and emphasis, they share many common goals and features, which is why different models may result in similar outcomes when applied in standard-practice preschool or school settings (Kasari & Smith, 2013). There is a growing consensus that the most effective interventions for children with ASD include the following features (Myers et al., 2007):

- *Early:* Begin intervention as soon as an ASD diagnosis is seriously considered.
- *Intensive:* Active engagement of the child at least 25 hours a week, 12 months a year, in systematically planned, developmentally appropriate educational activities with specific objectives.
- *Low Student–Teacher Ratio:* Allow sufficient one-on-one time and small-group instruction to meet specific individualized goals.
- *High Structure:* Use predictable routines, visual activity schedules, and clear physical boundaries to minimize distractions.
- *Family Inclusion:* Include a family component, with parent training as indicated.
- *Peer Interactions:* Promote opportunities for interactions with typically developing peers.
- *Generalization:* Teach child to apply learned skills in new settings and situations and to maintain the use of these skills.
- *Ongoing Assessment:* Monitor child's progress and make adjustments in treatment as needed.

The average age of children with ASD entering early-intervention programs has been 3 to 4 years or younger. These children have an average IQ in the mid-50s, although many are not testable at the time of their intake for treatment. Early intervention provides direct one-to-one work with the child for 15 to 40 hours per week and active involvement of the family. In effect, these programs become a way of life for the family—24 hours a day, 7 days a week. Programs are carried out at home and in the preschool, and efforts are made to include the child in interactions with normal peers, especially later in treatment.

Comprehensive reviews of outcomes for children with ASD completing early-intervention programs find that many of them are able to function in regular educational placements, although the type of setting and amount of support services needed varied considerably. Most children also show developmental gains, as reflected in improvements in their social behavior and communication, IQ scores, and scores on developmental tests and as found on classroom observations (Dawson et al., 2010; Eikeseth et al., 2007; Howard et al., 2005).

### The UCLA Young Autism Project

The UCLA Young Autism Project began about 50 years ago under the direction of Dr. Ivar Lovaas (1927–2010). Now referred to as the Applied Behavior Analysis (ABA) or Early Intensive Behavioral Intervention (EIBI) approach, it is the most detailed and labor-intensive of the early intervention programs and begins the earliest. It is one of the few programs that currently qualifies as an evidence-based treatment for ASD based on outcomes evaluated against control groups of similar-age children receiving less-intensive intervention (Lord & Jones, 2013; Rogers & Vismara, 2008). For these reasons, we focus on this approach, although we recognize that other potentially useful approaches have yet to be evaluated and that a few others that have been evaluated are also available.

The program includes many of the key elements of early intervention and is based on principles of applied behavior analysis, including the use of rewards and punishment and shaping by successive approximation (Lovaas, 2003; Smith, 2011). Parents are taught to act as the primary therapists for their children, with direction and help from therapists who work with them in the home. The average age of children entering the program is 32 months.

In a landmark research investigation, children with ASD were assigned to one of three groups. Although this assignment was not carried out randomly, the groups were found to be comparable with respect to age, language, intellectual functioning, and other measures prior to intervention. The experimental group of 19 children received 40 hours per week of intensive intervention. Control group 1 consisted of 19 children

Janine Wiedel Photolibrary/Alamy

Early intervention is essential for children with ASD.

who attended special education classes and received 10 hours per week of one-to-one instruction. Control group 2 consisted of 21 children from a larger study who also attended special education classes but did not receive one-to-one instruction.

For children in the experimental group, the first year of the program emphasized reducing disruptive behaviors and teaching appropriate behaviors such as compliance, imitation, and appropriate toy play. The second year emphasized expressive and abstract language and interactive play. Children were also taught how to function in a preschool group and, if possible, were enrolled in regular education preschool. The third year of the program emphasized the appropriate expression of emotions, pre-academic tasks, assertiveness, and observational learning.

The first outcome data were obtained when the children were 7 years old. Remarkably, 47% of the children in the experimental group were found to be functioning educationally and intellectually at a normal level. They successfully completed a regular first-grade class without support, were recommended for promotion by their teachers to a regular second-grade class, and scored at or above average on standardized IQ tests. On average, the IQ scores of these children increased by 37 points, from 70 to 107; overall, the experimental-group children showed an increase in mean IQ from 53 to 83. Children in the two control conditions did not fare nearly as well, resulting in only 1 of 40 children placed in a regular first-grade class; overall, these children showed only minimal increases in IQ scores. The experimental-group children were assessed again when their average age was 13 years, with similar results. The nine children who were placed in regular classrooms were virtually indistinguishable from same-age normal peers.

Nearly all children with ASD benefit from early intervention, but uncertainty remains as to: (1) how much the rate of progress depends on the severity of the child's ASD, the child's IQ and language ability, and the area of functioning being targeted (i.e., cognitive performance, language skills, adaptive behavior), and (2) what the long-term outcomes will be (Institute of Education Sciences, 2010; Reichow & Wolery, 2009; Warren et al., 2011). Additional studies have supported the effectiveness of the ABA approach in comparisons with other treatments and when used in community settings (Rogers & Vismara, 2008; Magiati, Charman, & Howlin, 2007). Claims have been made that some children with ASD can achieve normal functioning if given intensive intervention before age 3. However, earlier reports of "recovery" have not been replicated, and the question of whether they can achieve full recovery is a matter of some debate (Gresham & MacMillan, 1997; Smith & Lovaas, 1997).

## Medications

Many children with ASD receive psychotropic medication, most commonly antidepressants, stimulants, and tranquilizers/antipsychotics (Mandell et al., 2008). As with many of the other childhood disorders we have discussed, medication use for children with ASD has also increased, and many of these children receive multiple medications (Oswald & Sonenklar, 2007; Spencer et al., 2013). Although certain medications may help in the alleviation of specific behavioral symptoms, their benefits are limited, variable from child to child, and do not alter the core deficits of children with ASD (McCracken, 2011). Given the limited evidence of the effectiveness of medications, particularly for very young children, it is crucial that their risks, benefits, and costs be carefully evaluated (McPheeters et al., 2011).

## Section Summary

### Treatment of ASD

- Treatments for ASD are directed at maximizing the child's potential and helping the child and family cope more effectively with the disorder.
- Treatments for ASD focus on the specific social, communication, cognitive, and behavioral deficits displayed by children with this disorder.
- The most effective treatments use highly structured skill-oriented strategies that are tailored to the individual child and provide education and supportive counseling for the family.
- Nearly all children with ASD benefit from early intervention; however, controlled studies are needed to evaluate long-term outcomes.
- Medications may help in alleviating some symptoms. However, their benefits are limited, variable from child to child, and do not change the core deficits of children with ASD.

Paul Doyle/Alamy

**A teenage boy with ASD and his parents: Active family involvement is a key ingredient for successful treatment outcomes.**

## CHILDHOOD-ONSET SCHIZOPHRENIA (COS)

*I have a special power in my nose and I can control what's on TV and what people say or do.*

—From "Schizophrenia: Hidden Torment," by M. Nichols, *Maclean's,* January 30, 1995

This statement by a young girl with schizophrenia highlights the seriousness of this disorder. **Schizophrenia** is a neurodevelopmental disorder of the brain that is expressed in abnormal mental functions and disturbed behavior (White & Hilgetag, 2011). It is characterized by severe psychotic symptoms, including: bizarre delusions (false beliefs), hallucinations (false perceptions), thought disturbances, grossly disorganized behavior or catatonic behavior (motor dysfunctions ranging from wild agitation to immobility), extremely inappropriate or flat affect, and significant deterioration or impairment in functioning (APA, 2013).

Early-onset or **childhood-onset schizophrenia** (COS) is a progressive neurodevelopmental disorder that causes significant distress and disability (Rapoport & Gogtay, 2011). Rather than being a distinct form of schizophrenia, COS is a rare and possibly more severe form of schizophrenia that has an onset prior to age 18 and worse long-term outcomes (Kyriakopoulos & Frangou, 2007; Remschmidt et al., 2007). Although there is some overlap in symptoms, susceptibility genes, and social–cognitive patterns in children with ASD and those with COS, the two are distinct disorders. Clinically, those with COS have a later age at onset of their problem, less intellectual impairment, less severe social and language deficits, less ritualistic and repetitive behavior, hallucinations and delusions as the child gets older, and periods of remission and relapse (J. R. Asarnow & Asarnow, 2003).

Initially, a category for schizophrenia in childhood, distinct from the category of schizophrenia in adults, was used to diagnose the disorder. However, current thinking is that the criteria used to diagnose schizophrenia in adults can also be used to diagnose this disorder in young people (Asarnow, Tompson, & McGrath, 2004). Nevertheless, there are differences in presentation. For example, COS most often presents with hallucinations, thought disorder, and flattened affect; systematic delusions are observed less frequently (McClellan, Stock, & American Academy of Child and Adolescent Psychiatry [AACAP] Committee on Quality Issues [CQI], 2013).

In the initial stages of COS, the afflicted youngster may have difficulty concentrating, sleeping, or doing schoolwork, and may start to avoid friends. As the illness progresses, she or he may begin to speak incoherently and see or hear things that no one else does. Periods of improvement may be followed by terrifying relapses that are characterized by disordered thinking in which the youngster leaps illogically from one idea to another. The youngster may experience hallucinations, paranoia, and delusions. During their psychotic phases, youngsters with schizophrenia may be convinced that they have godlike powers or that people are spying on them. When in the grip of a psychosis, they may behave unpredictably and may become violent and suicidal.

Several of the clinical features of COS are illustrated in the case of Mary, a girl who first began to display symptoms of the disorder when she was about 10 years old.

### MARY

### Depressed, Disorderly, Doomed

Mary had always been a very shy child. At times she would become mute, she had severe difficulty making friends, was frequently oppositional, and occasionally wet the bed. By age 10, Mary had problems in school in addition to her continuing social isolation. She became depressed, felt that the devil was trying to make her do bad things, believed that her teacher was trying to hurt her, and became preoccupied with germs. Her behavior became increasingly disorganized; she talked of killing herself, appeared disheveled, and ran in front of a moving car in an apparent suicide attempt.

This episode precipitated an inpatient psychiatric evaluation, during which Mary continued to show bizarre behavior. She lapsed into periods of intense anxiety and had one episode of uncontrolled screaming. At times she would stare blankly into space and was frequently mute. Although Mary's functioning improved during hospitalization and she returned to her family, throughout her childhood and adolescence she was tormented by fears, hallucinations, the belief that others were out to get her, and occasional bouts of depression, often accompanied by suicide attempts. She continued to be socially isolated and withdrawn and to perform poorly at school. At age 17, after several brief inpatient hospitalizations, Mary was admitted to a state hospital, where she remained until the age of 19. During this period her affect was increasingly flat, and her psychotic symptoms persisted. One week after discharge from the hospital, Mary went into her room, locked the door, and overdosed on her medications. She was found dead the next morning.

Based on J. R. Asarnow & Asarnow, 2003.

Mary's tragic story illustrates several key features of COS.

- Although most cases have their onset during late adolescence or early adulthood, schizophrenia does occur during childhood (Nicolson & Rapoport, 1999).
- COS has a gradual rather than a sudden onset in childhood, with the child displaying a wide range of impairments that precede his or her psychotic symptoms (Nicolson et al., 2000).
- When the disorder is present in childhood, the symptoms likely will persist into adolescence and adulthood.
- COS has a profound negative impact on the child's developing social and academic competence.

Mary's futile 10-year struggle with schizophrenia underscores the tremendous pain and personal suffering experienced by youngsters with this illness.

## DSM-5: DEFINING FEATURES OF SCHIZOPHRENIA

DSM-5 criteria for schizophrenia are presented in Table 6.2. In addition to the presence of hallmark symptoms such as delusions, hallucinations, grossly disorganized speech and behavior, and negative symptoms such as flat affect, continuous signs of disturbance must persist for at least 6 months. In addition, the individual must show a significant decrement in one or more areas of functioning or, in the case of children and adolescents, a failure to achieve expected levels of interpersonal, academic, or occupational achievement. Although minor modifications in the symptom criteria for schizophrenia were made in DSM-5, research has found minimal effects of these changes on diagnosis, with over 98% of patients previously diagnosed with the disorder using DSM-IV

**TABLE 6.2** | **Diagnostic Criteria for Schizophrenia**

**DSM-5**

**(A)** *Severe disturbance in sensory functioning and/or behavior*: Two (or more) of the following, each present for a significant portion of the time during a 1-month period (or less if successfully treated). At least one of these must be (1), (2), or (3):

(1) Delusions.

(2) Hallucinations.

(3) Disorganized speech (e.g., frequent derailment or incoherence).

(4) Grossly disorganized or catatonic behavior.

(5) Negative symptoms (i.e., diminished emotional expression or avolition)

**(B)** *Social/occupational dysfunction*: For a significant portion of the time since the onset of the disturbance, level of functioning in one or more major areas such as work, interpersonal relations, or self-care is markedly below the level achieved prior to the onset (or when the onset is in childhood or adolescence, there is failure to achieve expected level of interpersonal, academic, or occupational functioning).

**(C)** *Duration*: Continuous signs of the disturbance persist for at least 6 months. This 6-month period must include at least 1 month of symptoms (or less if successfully treated) that meet Criterion A (i.e., active-phase symptoms) and may include periods of prodromal or residual symptoms. During these prodromal or residual periods, the signs of the disturbance may be manifested by only negative symptoms or by two or more symptoms listed in Criterion A present in an attenuated form (e.g., odd beliefs, unusual perceptual experiences).

**(D)** *Schizoaffective and Mood Disorder exclusion*: Schizoaffective disorder and depressive or bipolar disorder with psychotic features have been ruled out because either 1) no major depressive or manic episodes have occurred concurrently with the active-phase symptoms, or 2) if mood episodes have occurred during active-phase symptoms, they have been present for a minority of the total duration of the active and residual periods of the illness.

**(E)** *Substance/medical condition exclusion*: The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.

**(F)** *Relationship to autism spectrum or communication disorder*: If there is a history of autism spectrum disorder or a communication disorder of childhood-onset, the additional diagnosis of schizophrenia is made only if prominent delusions or hallucinations, in addition to the other requirements of schizophrenia, are also present for at least 1 month (or less if successfully treated).

*Specify* if: **With catatonia**

*Specify* current severity:

Severity is rated by a quantitative assessment of the primary symptoms of psychosis including delusions, hallucinations, disorganized speech, abnormal psychomotor behavior and negative symptoms. Each of these symptoms may be rated for its current severity (more severe in the last 7 days) on a 5-point scale ranging from 0 (not present) to 4 (present and severe).

**Note**: Diagnosis of schizophrenia can be made without using this severity specifier.

*Source:* Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition. American Psychiatric Association.

criteria also receiving a DSM-5 diagnosis (Tandon, Bruijnzeel, & Rankupalli, 2013). The traditional subtypes of schizophrenia (e.g., paranoid, disorganized) were eliminated in DSM-5, and catatonia is included as a specifier, not a subtype.

The use of the same diagnostic criteria for children and adults facilitates comparisons between cases of childhood-onset and adult-onset schizophrenia and the identification of important continuities in the disorder during the course of development. However, schizophrenia may be expressed differently at different ages. For example, hallucinations, delusions, and formal thought disturbances are extremely rare and difficult to diagnose before the age of 7; when they do occur, they may be less complex and reflect childhood themes (Caplan, 1994). A failure to adjust diagnostic criteria for developmental changes, such as social withdrawal or peer problems, may overlook children who show early signs of schizophrenia but may not develop the full-blown adult type until a later age (McClellan et al., 2003).

Other developmental considerations may also come into play in making a diagnosis. For example, it is sometimes difficult to distinguish between pathological symptoms, such as delusions, and the rich imaginative fantasies typical of many young children. Also, because cognitive and language processes are developing during childhood, it is extremely difficult to diagnose the symptom of disorganized speech (e.g., switching topics midsentence, incoherent or tangential speech). One difference between children with schizophrenia and adults with schizophrenia is that young children may not see their psychotic symptoms as distressing or disorganizing. Thus, when psychotic symptoms appear early in development, children may have difficulty distinguishing them from normal experience (Russell, 1994).

Symptoms of schizophrenia fall into two categories. The first, positive symptoms (also called psychotic or active symptoms), involve excesses or disturbances in normal functioning, such as delusions or hallucinations. The second, negative symptoms, involve a loss in normal functioning, for example, disturbances in sleep patterns.

### *Positive Symptoms*

Youngsters with COS may display psychotic symptoms such as delusions and hallucinations (Polanczyk et al., 2010). **Delusions** are disturbances in thinking involving disordered thought content and strong false beliefs that are misrepresentations of reality in the context of one's life experience and culture. **Hallucinations** are disturbances in perception in which things are seen, heard, or otherwise sensed even though they are not real or present. They can present in any sensory modality, including smell or touch. The most common presenting symptom for children with schizophrenia is auditory hallucinations (e.g., hearing voices that other people cannot hear and are experienced as separate from the individual's thoughts); they occur in about 80% of patients who have an onset prior to age 11. About 40% to 60% of children with schizophrenia also experience visual hallucinations, delusions (David et al., 2011), and thought disorders characterized by loose associations, illogical thinking, and impaired discourse skills (Caplan et al., 2000). Examples of psychotic symptoms reported by children with schizophrenia are presented in A Closer Look 6.4.

### *Negative Symptoms*

Negative symptoms of schizophrenia may include slowed thinking, speech, and movement; emotional apathy; lack of drive; indifference to social contact; and

A CLOSER **LOOK 6.4**

## Psychotic Symptoms in Children with Schizophrenia

**Hallucinations**

An 8-year-old boy stated: "I once heard a noise coming from the south and the east; one told me to jump off the roof and one told me to smash my mom."

A 12-year-old boy saw a ghost (man) with a red, burned, scarred, and cut face on multiple occasions and in different locations. He had been seeing this since age 5.

**Delusions**

A 9-year-old boy was convinced he was a dog (his parents were German Shepherds) and was growing fur; on one occasion, he refused to leave a veterinarian's office unless he received a shot.

An 11-year-old boy described "waste" produced when the good and bad voices fought with each other; the "waste" came out of his feet when he swam in chlorinated pools.

**Thought Disorder**

"I used to have a Mexican dream. I was watching TV in the family room. I disappeared outside of this world and then I was in a closet. Sounds like a vacuum dream. It's a Mexican dream. When I was close to that dream earth, I was turning upside down. I don't like to turn upside down. Sometimes I have Mexican dreams and vacuum dreams. It's real hard to scream in dreams."

Source: From A. T. Russell, The Clinical Presentation of Childhood-Onset-Schizophrenia, Schizophrenia Bulletin, 1994, volume 20, 631–646.

self-neglect. These symptoms generally reflect a loss of motivation and can range from minor to severe. Although these symptoms are much less dramatic than the positive symptoms, they can be very persistent and difficult to treat. They also may be difficult to recognize in young people, because some of these changes are behaviors that might be expected to occur to some degree during adolescence.

## Section Summary

### DSM-5: Defining Features of Schizophrenia

- Schizophrenia is a neurodevelopmental disorder of the brain that is expressed in abnormal mental functions and disturbed behavior.
- Unlike children with ASD, children with COS have a later age at onset, show less intellectual impairment, display less severe social and language deficits, develop hallucinations and delusions as they get older, and experience periods of remission and relapse.
- COS is a more severe form of adult-onset schizophrenia rather than a different disorder.
- Youngsters with COS may display psychotic symptoms, such as delusions and hallucinations, and negative symptoms, such as a slowing of thinking, speech, and movement, and indifference to social contact.

# PRECURSORS AND COMORBIDITIES

For a majority of children with COS, the onset of their disorder is gradual rather than sudden, with nearly 95% showing a clear history of behavioral, social, and psychiatric disturbances before the onset of psychosis (Paya et al., 2013). For example, Mary was oppositional and had difficulty making friends well before she began to display her psychotic symptoms.

Developmental precursors for the onset of schizophrenia include speech and language problems, learning problems, static cognitive impairments and maturational delays, problems in motor development, movement abnormalities (e.g., facial tics, grimaces), higher levels of social impairment (e.g., social withdrawal, social oddities), unusual thought content, suspicion/paranoia, substance abuse, and a genetic risk for schizophrenia with a recent deterioration in functioning (Cannon et al., 2008; Dickson et al., 2012; Paya et al., 2013).

About 10% to 20% of children and adolescents with COS have IQs in the borderline range of intellectual functioning or lower. They also display impairments in attention, memory, and executive functions, as well as global intellectual deficits (Frangou, 2013). Children with COS often present with other symptoms and disorders, such as anxiety and depression, ADHD, conduct problems, movement abnormalities, and suicidal tendencies (Frazier et al., 2007). About 70% of children meet criteria for another diagnosis, most commonly mood disorder or oppositional/conduct disorder (Russell, Bott, & Sammons, 1989). Returning to the question of whether ASD and COS are related, one study found that COS is preceded by ASD in 30% to 50% of cases (Rapoport et al., 2009). In addition, epidemiological and family studies have found associations between the two disorders, and both display deficits in theory of mind and mirror neuron impairments (mirror neurons fire both when an individual acts and when they see the same action performed by another) impairments. Both disorders also show signs of accelerated brain development at ages near the onset of the disorder, and brain imaging studies also find similar abnormalities in neural connectivity (de Lacy & King, 2013). A small but significant genetic correlation and similar risk genes and rare small chromosomal variants have also been found (Cross-Disorder Group of the Psychiatric Genetics Consortium, 2013; King & Lord, 2011). Taken together, these findings suggest several potentially important links between COS and ASD.

## Prevalence

Schizophrenia is extremely rare in children under 12 years of age. It begins to increase dramatically in frequency in adolescence and early adulthood, with a modal onset at around 22 years of age (Abel, Drake, & Goldstein, 2010; McClellan et al., 2013)

- Estimates of COS indicate a prevalence of less than 1 child per 10,000, with an increased rate in more recent years (J. R. Asarnow & Asarnow, 2003; Okkels et al., 2013).
- These estimates suggest that schizophrenia occurs at least 100 times more often in adults than in children (Bromet & Fennig, 1999).
- COS has an earlier (by 2 to 4 years) age at onset in boys as in girls (Häfner et al., 1998), and an onset prior to 12 years of age is about twice as common in boys as in girls. However, this sex difference disappears in adolescence (Frazier et al., 2007).

The reasons that more males have COS are not known; a greater general biological vulnerability of males for neurodevelopmental disorders and different causes have both been suggested as possibilities. For adults, the rates of schizophrenia are higher in lower socioeconomic groups. However, there is little

information regarding the relationship between social class and COS. In a related vein, the incidence rates and pattern of symptoms in adults with schizophrenia are similar across cultures, countries, and racial groups (Myers, 2011), but little information regarding cross-cultural patterns is available for COS (J. R. Asarnow & Asarnow, 1994).

## Section Summary

### Precursors and Comorbidities

- For a majority of children with COS, the onset of the disorder is gradual rather than sudden, with nearly 95% of children showing a clear history of behavioral, social, and psychiatric disturbances before the onset of psychosis.
- Developmental precursors for the onset of schizophrenia include speech and language problems, cognitive impairments and delays, problems in motor development, social impairment, unusual thought content, substance abuse, and a genetic risk for schizophrenia with a recent deterioration in functioning.
- Children with COS often display other symptoms and disorders, such as anxiety and depression, ADHD, conduct problems, movement abnormalities, and suicidal tendencies.
- Recent findings suggest several links between COS and ASD.

### Prevalence

- Schizophrenia is extremely rare in children under 12 years of age, occurring much less often than in adolescents and adults.
- COS has an earlier age at onset in boys, and an onset prior to 12 years of age is about twice as common in boys as in girls. This sex difference disappears in adolescence.

# CAUSES AND TREATMENT OF COS

We are just beginning to understand the possible causes of COS and ways of helping these children and their families. In the following sections, we discuss research into biological and family factors that have been implicated thus far, as well as recent treatments for this disorder.

## Causes

A key issue in understanding schizophrenia is why a genetically based, neurobiological disorder is not expressed clinically until 15 to 20 years after birth, at which time it progressively disables its victims. In understanding this issue, investigators have proposed a **neurodevelopmental model of schizophrenia** in which a genetic vulnerability and early neurodevelopmental insults result in impaired connections between many brain regions, including the cerebral cortex, white matter, hippocampus, cerebellum, and parts of the limbic system (Rapoport, Addington, & Frangou, 2005; Rapoport, Giedd, & Gogtay, 2012). This model is supported by findings suggesting that gene mutations leading to schizophrenia may produce disruptions in the development of fetal prefrontal cortical circuitry (Gulsuner et al., 2013). This defective neural circuitry creates a vulnerability to dysfunction that is revealed by developmental processes and events during puberty (e.g., synaptic and hormonal changes), and by exposure to stress (Lewis & Lieberman, 2000). The neurodevelopmental model provides a useful framework for understanding how a condition like schizophrenia, which first presents as a disorder in adolescence or early adulthood, can be partly understood as a function of events occurring much earlier in development (Owen et al., 2011). A model of early-occurring neural pathology in schizophrenia is consistent with the findings that infants and children who later develop COS often display developmental impairments well before the onset of their psychotic symptoms, including deficits in motor, language, and cognitive and social functioning (Marenco & Weinberger, 2000).

### *Biological Factors*

Evidence suggests a strong genetic contribution to schizophrenia in childhood, with heritability estimates around 80% (R. F. Asarnow et al., 2001; Gejman, Sanders, & Duan, 2010). For example, the rate of schizophrenia among relatives of children with COS is about double the rate for family members of adults with schizophrenia. One landmark twin study found concordance rates of 88% and 23%, respectively, for identical versus fraternal twins with schizophrenia with an onset prior to 15 years of age (Kallman & Roth, 1956). Molecular genetic studies have identified a number of potential susceptibility genes for COS, many of which have been previously linked with schizophrenia in adults (Kyriakopoulos & Frangou, 2007). Several common gene variants with small effects have been implicated, along with some extremely rare gene variants with potentially large effects (Pogue-Geile & Yokley, 2010). However, specific gene findings account for a very small amount of the variance, and most of the genetic influences on susceptibility to schizophrenia have yet to be identified. Given the large number of weak genetic (and environmental) risk factors, it is likely that COS is best represented by a continuum of risk involving many GxE interactions (Rapoport & Gogtay, 2011). For example, one study found a ninefold increased risk of schizophrenia in cases in which the presence of a

parent with psychosis was combined with maternal depression during pregnancy (Maki et al., 2010). In light of the enormous number of genes, gene regulatory mechanisms associated with brain development, and mutational mechanisms that can disrupt these processes, it is possible that most affected individuals with COS have a unique genetic cause (Kuniyoshi & McClellan, 2014).

The occurrence of central nervous system dysfunction among individuals with schizophrenia, and the dramatic improvements associated with the administration of medications, suggest that schizophrenia is a disorder of the brain (Lewis & Lieberman, 2000). Brain scan studies of youngsters with COS have found enlarged ventricles and a shrinkage in brain gray matter that spreads across the brain during adolescence, beginning in the rear brain structures involved in attention and perception and spreading to the frontal parts of the brain involved in executive functions such as planning and organization (Vidal et al., 2006). The progressive loss of gray matter was accompanied by delayed/disrupted white matter growth, hippocampal volume loss, and a progressive decline in cerebellar volume. Interestingly, most of these changes were also found in the nonpsychotic siblings of the children with COS. However, siblings showed later normalization of the earlier gray matter abnormalities, suggesting a role for restorative/protective factors. In contrast, the hippocampal volume loss across age in children with COS was not shared by their siblings; thus it appears to be specific to schizophrenia (Rapoport & Gogtay, 2011). Another study found that atypical neural activity in a network of language-associated brain regions during discourse processing was associated with subsequent thought disorder severity and social outcome in youth at risk for psychosis (Sabb et al., 2010). Findings like this are important in identifying potential biomarkers for COS that may suggest strategies for intervention and prevention.

No single brain lesion has been identified in all cases of COS, and the lesions that have been found in some cases are not specific to schizophrenia (Kyriakopoulos & Frangou, 2007). In addition, atypical developmental patterns of brain development over time are often more prominent than are anatomic brain differences at any one time point (Rapoport & Gogtay, 2011). In general, brain research on COS points to a widespread developmental disruption of neural connectivity. These disrupted neural connections likely involve susceptibility genes that impact developmental processes involved in establishing connectivity within and between brain regions (Karlsgodt et al., 2008; Rapoport et al., 2012).

### *Environmental Factors*

COS is a familial disorder, but the less than 100% concordance rates for identical twins suggest that nongenetic influences contribute to the likelihood of a child developing schizophrenia. Nongenetic factors, including exposure to infectious, toxic, or traumatic insults and stress during prenatal or postnatal development, may interact with a genetic susceptibility for schizophrenia (Arseneault et al., 2011; Lahti et al., 2009; Rapoport et al., 2005). Several nongenetic factors occurring during pregnancy and birth are associated with an increased risk for later schizophrenia, including: maternal diabetes, low birth weight, older paternal age, winter birth, and prenatal maternal stress (King, St-Hilaire, & Heidkamp, 2010). Each of these factors alone is associated with a slight increase in risk, which multiplies when they are combined with each other and/or with other risk factors. In considering other nongenetic influences, the elevated likelihood of psychiatric illness in parents of children with schizophrenia will likely have a negative effect on the ability of the affected parent to function in a parental role.

By themselves, psychosocial factors do not cause COS. However, they may interact with biological risk factors to affect the onset, course, and severity of the disorder. Parents of children with schizophrenia score higher than parents of children with depression on **communication deviance,** which is a measure of interpersonal signs of attentional and thought disturbance. Children from families with high communication deviance display the most severe impairment and the poorest attentional functioning. These findings suggest that communication deviance may be associated with a severe form of schizophrenia or that family interaction may worsen the severity of dysfunction (J. R. Asarnow, Goldstein, & Ben-Meir, 1988). Parents of children with schizophrenia are more likely to use harsh criticism of their children than are parents of depressed children or normal controls, which could be a reaction to their child's severe difficulties.

Support for the role of the family environment comes from studies showing that exposure to a poor family environment and certain patterns of communication may interact with a genetic risk for schizophrenia to further increase a child's risk for developing a schizophrenia-spectrum disorder. For example, in a longitudinal study of children of biological mothers with schizophrenia-spectrum disorders who were adopted at an early age, poor child-rearing environments and communication deviance of the adoptive parents predicted which adoptees developed a schizophrenia-spectrum disorder (Wahlberg et al., 2004; Wynne et al., 2006). Children

with a dysfunctional child-rearing environment and adoptive parents who displayed high rates of communication deviance were significantly more likely to develop schizophrenia-spectrum disorder than those raised in more positive family environments. For children with a low genetic risk for schizophrenia, a poor family environment and deviant parent communication patterns did not increase the risk for the later development of a schizophrenia-spectrum disorder.

Family findings highlight the stress, distress, and personal tragedy often experienced by families of children with schizophrenia (J. R. Asarnow & Asarnow, 2003). In the words of June Beeby, the mother of 17-year-old Matthew, who was diagnosed with schizophrenia and believed that God wanted his mother and his sister to die:

> "It's quite horrendous. First of all, you've got somebody that you love, a child that you've raised. And then suddenly, the child becomes a crazy person" (M. Nichols, 1995, p. 70). On a dark and cold winter day, June Beeby arrived home to find her son dead in a pool of blood. "He had taken two ordinary dinner knives … and plunged them into his eyes until they pierced his brain" (p. 70). In a diary entry that he had made 2 years before he took his life, Matthew had described an encounter with God: "He used his power and he controlled my brain for nine months. … God wanted me to feel that I would die, in order for individuals to live forever in heaven" (p. 74).

## Treatment

*As a parent you feel you have a tremendous responsibility to keep a son or daughter safe. … But when your child is schizophrenic you can't do that, because the person doesn't want help.*

—From "Schizophrenia: Hidden Torment," by M. Nichols, *Maclean's,* January 30, 1995

COS is a chronic disorder with a poor long-term outcome for most sufferers, although some youngsters may display more positive outcomes (McClellan et al., 2013; Röpcke & Eggers, 2005). In either case, outcomes for most afflicted individuals are vastly improved over what they once were. Current treatments emphasize the use of antipsychotic medications (e.g., clozapine, risperidone) combined with psychotherapeutic and social and educational support programs (McClellan et al., 2013; National Institute of Health and Clinical Excellence [NICE], 2013b). Although we know far less about the use of antipsychotic medications with children than with adults, they are widely used to treat young people with schizophrenia, and a majority of youngsters with schizophrenia will spend much of their life on some medication (Findling et al., 2014). Medications help control psychotic symptoms in children with schizophrenia by blocking dopamine transmission at the D2 dopamine receptor. However, adverse effects with antipsychotic treatment are prevalent and are associated with reduced adherence to treatment. Depending on the type of medication, these side effects can be serious and may include increased levels of prolactin, motor dysfunction (e.g., tremor), weight gain, sedation, or dysregulation of glucose. Thus, it is extremely important that these side effects be carefully monitored and managed with changes in dose or type of medication as needed (Tiffin, 2007). There is also a need for psychosocial treatments, such as family intervention, social skills training, and cognitive–behavioral therapy (Addington, Piskulic, & Marshall, 2010). The need for educational support that provides factual information about the illness and its treatment within a recovery-focused discussion with the patient and family is also widely recognized in clinical practice (McDonnell & Dyck, 2004; Tiffin, 2007). Although findings from psychosocial treatments with older individuals with schizophrenia are promising, more controlled studies with children and adolescents are needed (Tiffin & Welsh, 2013).

Using a prevention framework, recent efforts have focused on a variety of pharmacological, biological, psychosocial, and family interventions for high-risk younger individuals well before the onset of psychotic symptoms. Findings to date have suggested that these interventions can be effective in reducing the risk of transition to full-blown psychosis over the short term but over the longer term may only delay transition to psychosis (Preti & Cella, 2010). Further research into the prediction and prevention of psychosis in youths at high clinical risk for psychosis is needed, particularly in relation to possible long-term benefits (Addington & Heinssen, 2012).

## Section Summary

### Causes and Treatment of COS

- Current views regarding the causes of COS are based on a neurodevelopmental model in which a genetic vulnerability and early neurodevelopmental insults result in impaired connections between many brain regions. This impaired neural circuitry may increase the child's vulnerability to stress.

(*continues*)

## Section Summary (*continued*)

- COS is a disorder that involves multiple genes and is associated with environmental and developmental vulnerability factors.
- Brain studies in COS suggest a shrinkage in brain gray matter that spreads across the brain during adolescence; it begins in the rear brain structures involved in attention and perception and spreads to the frontal parts of the brain involved in executive functions such as planning and organization.
- Although medications may help control psychotic symptoms in children with schizophrenia, psychosocial treatments such as social skills training, family intervention, cognitive–behavioral therapy, and educational support are also needed.

# Study Resources

### SECTION SUMMARIES



### KEY TERMS

# 7

# Communication and Learning Disorders

*If you can read this, thank a teacher.*

—Anonymous

## CHAPTER PREVIEW

EVERYONE HAS IMPORTANT NEEDS and ideas. Imagine not being able to get them across. Sights and sounds surround you, but you cannot focus your attention long enough to make sense of them. When you are shown how to read or add, you find that the letters and numbers look and sound too much alike. Children and adolescents with communication and learning disorders experience these difficulties daily. Everyday tasks can be confusing and frustrating, and sometimes result in a cycle of academic failure and lowered self-esteem.

### JAMES

#### Smart but Can't Read

James, age 9, was a growing concern for his teacher: "James is obviously a very bright boy, and he wants to do well. I've noticed that he likes art, and is always wanting to draw. But he gets really upset when I ask him to do some work in class.

He looks like he dreads coming to school. And he complains that some words he tries to read don't make sense to him. I'm worried that his increasing frustration is going to cause other problems in school or with friends. Sometimes he gets mad at something and he has trouble calming down. If he is trying to create something that doesn't turn out the way he envisioned it, he explodes and slams his fist against the wall."

What James's mother heard was all too familiar. She knew that her son would get involved in something only if he could do it his own way. Her mind wandered briefly to when he was a toddler and sometimes got so anxious and worried about something that he had trouble sleeping or felt sick. She shared with his teacher her frustration at trying to find out what the problem was: "Getting him to read at home is like pulling teeth. He won't read at all on his own because he knows he can't read many of the words." (Based on authors' case material.)

### FRANCINE

#### Shunned and Falling Behind

Francine, age 7, was entering a new school for the second time in 2 years. The first school was too challenging, and the other kids teased her because she "doesn't know what 2 plus 2 is." She is content to play for hours by herself and is not interested in the things that other kids her age are doing. "Most of the time," her mother explained, "Francine seems sad and in a bit of a fog." Although school performance was a major concern, her mother was also quite worried about Francine's lack of friends and the way other children treated her.

Her mother and father proudly shared their daughter's early childhood history and developmental milestones with me during our first interview. "Francine walked before she was a year old, and was a very talkative baby and toddler, who picked up new words quite quickly. She was a healthy and normal baby—we can't figure out why she seems so uninterested in school and other kids." They went on to explain: "When she entered preschool and kindergarten, she seemed uninterested in making friends. The other kids basically ignored her, even though she didn't do anything to bother them. My husband and I didn't think much of it at first. In fact, we bragged about how she took an early interest in reading and would spend a lot of her time alone with a book or magazine, even when she was 4 or 5, although she didn't usually understand what she read. But we grew more concerned around age 5 because she paid little attention to popular movies, toys, and things other kids her age played with. When she was a preschooler, we also noticed that she had trouble with numbers and understanding concepts like "more," "less," or "bigger." She knows what these words mean now, but she is still confused when we ask her to count something.

Yesterday I gave her her allowance and just for the heck of it, I used pennies, nickels, and dimes to see if she could add them up. No matter how hard we tried, she became confused, switching from one coin to the other, and she thought she had a bigger allowance if I stacked the pennies up! And if you ask her to arrange something, like setting the table for dinner, you never know what you'll end up with!" (Based on authors' case material.)

Children with communication or learning disorders can learn, and they are as intelligent as anyone else. Their disorders usually affect only certain limited aspects of learning, and rarely are they severe enough to impair the pursuit of a normal life—but they can be very stressful. Consider the experiences of James and Francine: James and Francine have different learning problems. James's are with language and reading. His ability to distinguish between different language sounds (phonemes) is underdeveloped, which is the primary reason for his poor word recognition and writing ability. Francine's problems are mostly with nonverbal learning, such as math. She can read quite well, but she has difficulty understanding some of the subtleties of others' facial expressions and gestures. She also confuses terms and instructions that describe numerical or spatial relationships, such as "larger than" or "sit beside the couch."

The field of learning and communication disorders, broadly referred to as "learning disabilities,"

has changed dramatically during the past 50 years. For many years, learning problems were attributed to poor motivation or poor instruction. Fortunately, breakthroughs in neuroimaging techniques has led to increased recognition of the differences in the neurological makeup and development of children with problems in language and related cognitive tasks. With recent advances in detection and intervention aimed at early language development, signs of communication problems are detected at an early age and children are taught using alternative methods that build on their developmental strengths.

In this chapter, we emphasize the relationship between language development and the subsequent appearance of a learning problem once the child enters school. We put these problems in a developmental context by showing how communication disorders (diagnosed primarily in early childhood) and learning disorders (identified most often during early school years) have interconnected features and underlying causes. As a case in point, preschoolers with communication disorders are more likely to develop a learning disability by middle childhood or early adolescence (Beitchman & Brownlie, 2014).

Opus/amanaimages/Jupiter Images

Slowly but surely, most children learn the letters of the alphabet and how to use them to read and write words. For children with certain learning disabilities, however, the shapes and sounds of different letters continue to be confusing.

## DEFINITIONS AND HISTORY

*Learning disability* (LD) is still commonly used as a general term for learning problems that occur in the absence of other obvious conditions, such as intellectual disability or brain damage. In the *Diagnostic and Statistical Manual of Mental Disorder*, 5th edition (DSM-5) two more specific terms are used, *communication disorders* and *learning disorders*, but the common use of the term *learning disability* requires that it be clarified and defined.

A learning disability affects how individuals with normal or above-average intelligence take in, retain, or express information. Incoming or outgoing information can be scrambled as it passes between the senses and the brain. Unlike most physical disabilities, a learning disability is hidden and is often undetected in young children (Lovett & Lewandowski, in 2014). Thus, children with learning disabilities often must cope not only with their limitations in reading, writing, or math but also with the frustration of convincing others that their problems are as legitimate as visible disabilities.

Learning difficulties often show up in schoolwork and can impede a child's ability to learn to read, write, or do math, but they also can affect many other parts of life, including work, daily routines, family life, and friendships. Some learning problems are specific and affect a narrow range of ability, whereas others may affect many different tasks and social situations. Each type of learning disability, whether it is related to reading, writing, math, or language, is characterized by distinct definitions and diagnoses. Knowledge of communication and learning disorders is growing rapidly as a result of increased scientific interest and research support. We now recognize that a learning disability, though challenging, does not have to be a handicap. Many well-known people with learning difficulties used their talents in exceptional ways, including Albert Einstein, Winston Churchill, and Thomas Edison.

The main characteristic all children with learning difficulties share is failing to perform at their expected level in school. Otherwise, symptoms vary tremendously (Beitchman & Brownlie, 2014). Many children and adults who are unable to acquire academic skills at a normal rate have been helped by recognizing and attending to specific learning problems.

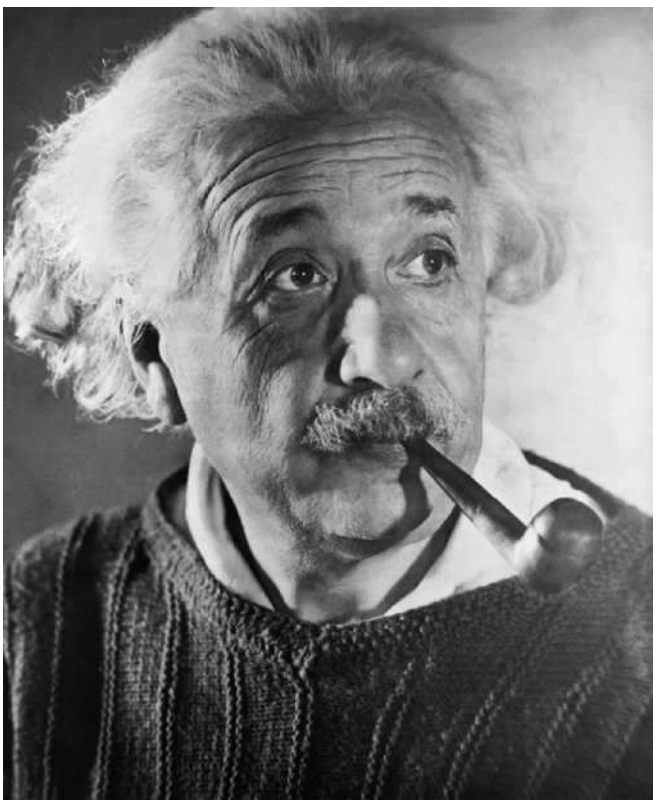
Children with learning disabilities constitute a third of all children in the United States and Canada who receive special education services (National Center for Educational Statistics, 2012). Yet, experts still struggle to adequately define learning disabilities because of their many forms and overlapping symptoms, which you will note in the following lengthy definition:

**Learning disability** is a lay term (not a diagnostic term) that refers to significant problems in mastering one or more of the following skills: listening, speaking, reading, writing, reasoning, and mathematics. Learning disabilities do not include visual, hearing, or physical

impairments; intellectual disability; emotional disturbance; or environmental disadvantage. Emotional and social disturbances and other adaptive deficiencies may occur with learning problems, but they do not by themselves constitute a learning disability (Individuals with Disabilities Education Improvement Act [IDEA], 2004).

In Chapter 5 we described intellectual disability as involving deficits in basic cognitive abilities that include problem solving, verbal skills, and mental reasoning. But the broader concept of intelligence also includes logical, mathematical, and language abilities that reflect a pattern of relative strengths and weaknesses possessed by everyone. For example, we are all stronger in some areas of learning and performance than in others (e.g., we enjoy writing and reading, but don't ask us to fix your car). Similarly, children with specific learning disorders who have normal intelligence show a pattern of relative strengths and weaknesses that can make some learning tasks much more difficult. This pattern is noteworthy mostly because it is so extreme and unexpected for a child who otherwise shows normal cognitive and physical development.

**Communication disorder** is a diagnostic term that refers to deficits in language, speech, and communication (APA, 2013). Communication disorders include the following diagnostic categories:



Bettmann/Corbis

Few realize that Albert Einstein had early speech and language difficulties, given his monumental contributions to society.

- language disorder (problems using language to communicate, such as spoken words or sign language, or understanding what other people say)
- speech sound disorder (deficits in productive speech sounds)
- childhood-onset fluency disorder (problems in speech fluency, such as stuttering)
- social (pragmatic) communication disorder

These communication disorders are developmentally connected to the later onset of learning disorders.

**Specific learning disorder** is a diagnostic term that refers to specific problems in learning and using academic skills. The DSM-5 integrates the frequently co-occurring problems in reading, mathematics, and written expression into this one category, and uses specifiers to designate impairments in one or more of these areas. Specific learning disorder is determined by achievement test results that are substantially below what is expected for the child's age, schooling, and intellectual ability.

An unexpected pattern of strengths and weaknesses in learning was first noted and studied during the late nineteenth century by physicians who were treating patients with medical injuries (Hammill, 1993). Franz Joseph Gall, a pioneer of language disorders, was struck by what he observed among some of his brain-injured patients: They had lost the capacity to express their feelings and ideas clearly through speech, yet they did not seem to suffer any intellectual impairment. One of his patients could not speak, but had no problem writing his thoughts on paper. Because he knew that this patient had normal speech before the head injury, Gall reasoned that the problem must have resulted from brain damage that had disrupted the neurological processes related to speech. For the first time, scientists began to pinpoint areas in the brain that control the ability to express and receive language processes.

These early observations, based on known medical injuries, raised the possibility that people with learning disabilities differ from people with intellectual disability in terms of relative strengths and deficits. People with learning disabilities have normal intellectual processes in most areas but are relatively weaker in others, which is known as having an **unexpected discrepancy** between measured ability and actual performance. This premise remained the foundation of most definitions of learning disability for many years. However, researchers and practitioners eventually agreed that subaverage performance on achievement tests, rather than a discrepancy between potential and actual performance, was a better way to capture these learning difficulties (Maehler & Schuchardt, 2011).

The links between intellectual disability, organic brain damage, and learning problems fascinated scientists, who had a firmer understanding of brain–behavior relationships by the 1940s. During that time, the question still remained as to why some children who did not fit the definition of intellectual disability based on IQ had significant problems in learning. Could intellectual disability be restricted to only certain intellectual abilities? Were academic problems the same as those assessed by measures of general intelligence?

Strauss and Werner (1943) shed light on this issue by pointing out that children learn in individual ways, challenging the concept that learning is a relatively uniform, predictable process in children without intellectual disabilities. Three important concepts from this period continue to influence the field to this day (Hallahan, Pullen, & Ward, 2013):

1. Children approach learning in different ways, so each child's individual learning style and uniqueness should be recognized and used to full advantage.
2. Educational methods should be tailored to an individual child's pattern of strengths and weaknesses; one method should not be imposed on everyone.
3. Children with learning problems might be helped by teaching methods that strengthen existing abilities rather than emphasize weak areas.

By the early 1960s, the modern learning disabilities movement had begun. Parents and educators were dissatisfied that children often had to be diagnosed with an intellectual disability in order to receive special education services. A category was needed to describe learning problems that could not be explained on the basis of intellectual disability, lack of learning opportunities, psychopathology, or sensory deficits (Lyon, Fletcher, & Barnes, 2003).

Thus, the emerging concept of learning disabilities made intuitive sense to many who were familiar with the varied needs of children, and it was welcomed as states and provinces began to support special education programs and services. The domination of physicians and psychologists in the field gave way to greater input from educators, parents, and clinicians. Teacher training expanded to include new ways to teach youngsters who could not respond to typical classroom methods. Professionals trained in speech and language pathology became an important part of school-based services.

As the focus of the learning disabilities movement shifted from the clinic to the classroom, parents and educators assumed a major role in programming and placement. They were encouraged by the fact that the term *learning disabled* did not stigmatize children, but rather brought them needed services (Hammill, 1993). The fact that these children had normal intelligence gave parents and teachers hope that difficulties in reading, writing, and math could be overcome if only the right set of instructional conditions and settings could be identified (Lyon et al., 2003). These developments led to the recent Response-to-Intervention (RTI) movement, which views LD in terms of what academic help children need rather than what disability they might have (Lovett & Lewandowski, in press). Thus, with the collaborative leadership of parents, educators, and specially trained professionals, the field of learning disabilities grew from its beginning in the 1960s to the major component of educational services it is today.

## Section Summary

### Definitions and History

- *Learning disability* is a general lay term for communication and learning problems that occur in the absence of other obvious conditions such as intellectual disability or brain damage.
- Children and adults with learning disabilities show specific deficits in using spoken or written language, often referred to as relative strengths and weaknesses.
- Parents and educators assumed a major role in bringing recognition and services to children with learning disabilities.

# LANGUAGE DEVELOPMENT

From birth, infants selectively attend to parental speech sounds and soon learn to communicate with basic gestures and sounds of their own. Usually, by their first birthday they can recognize several words and use a few of their own to express their needs and emotions. Over the next 2 years, their language development proceeds at an exponential pace, and their ability to formulate complex ideas and express new concepts is a constant source of amazement and amusement for parents. Adults play an important role in encouraging language development by providing clear examples of language and enjoying the child's expressions.

Language consists of **phonemes**, which are the basic sounds (such as sharp *ba*'s and *da*'s and drawn-out *ee*'s and *ss*'s) that make up language. When a child hears a phoneme over and over, receptors in the ear stimulate the formation of dedicated connections to the brain's auditory cortex. A perceptual map forms that represents similarities among sounds and helps the infant learn to discriminate among different phonemes. These

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From an early age, children love to express themselves.

maps form quickly; 6-month-old children of English-speaking parents already have auditory maps different from infants in non-English-speaking homes, as measured by neuron activity in response to different sounds (Kuhl et al., 2006). By their first birthday, the maps are complete, and infants are less able to discriminate sounds that are not important in their own language.

Rapid development of a perceptual map is why learning a second language after—rather than with—the first language is difficult; brain connections are already wired for English, and the remaining neurons are less able to form basic new connections for, say, Swedish. Once the basic circuitry is established, infants can turn sounds into words, and the more words they hear, the faster they learn language. The sounds of words serve to strengthen and expand neural connections that can then process more words. Similar cortical maps are formed for other highly refined skills, such as musical ability (Huss et al., 2011). A young child who learns to play a musical instrument may strengthen the neural circuits that underlie not only music, but verbal memory as well (Ho, Cheung, & Chan, 2003).

## Phonological Awareness

Not all children progress normally through the milestones of language development. Some are noticeably delayed, continuing to use gestures or sounds rather than speech. Others progress normally in some areas, such as following spoken directions and attending to commands, but have trouble finding the words to express themselves clearly.

Although the development of language is one of the best predictors of school performance and overall intelligence (Sattler, 2008), delays or differences in development are not a definitive sign of intellectual disability or cognitive disorder. Rather, such deviations from normal may be just that—deviations—and may be accompanied by superior abilities in other areas of cognitive functioning. Albert Einstein, who is considered an intellectual genius, began speaking late and infrequently, causing his parents to worry that he was "subnormal." According to family members, when his father asked his son's headmaster what profession his son should adopt, the answer was simply, "It doesn't matter, he'll never make a success of anything" (R. W. Clark, 1971, p. 10).

Since language development is an indicator of general mental development, children in whom language fails to develop or who show severe delays in acquiring language are considered at risk of having a language-based learning disability. Albert Einstein notwithstanding, early language problems are considered highly predictive of subsequent communication and learning disorders (Heim & Benasich, 2006; Williams, 2010).

**Phonology** is the ability to learn and store phonemes as well as the rules for combining the sounds into meaningful units or words. Deficits in phonology are a chief reason that most children and adults with communication and learning disorders have problems in language-based activities such as learning to read and spell (Larkin & Snowling, 2008; Nation, Snowling, & Clarke, 2007).

A young child is required to recognize that speech is segmented into phonemes (the English language contains about 42, such as *ba*, *ga*, *at*, and *tr*). This task is difficult for many children because speech does not consist of separate phonemes produced one after another. Instead, sounds are *co-articulated* (overlapped with one another) to permit rapid communication, rather than pronounced sound-by-sound (Liberman & Shankweiler, 1991). About 80% of children can segment words and syllables into their proper phonemes by the time they are 7 years old. The other 20% cannot, and it is these children who struggle hardest to read (S. E. Shaywitz & Shaywitz, 2013; Vellutino et al., 2004).

Generally, early language problems surface as learning problems when children enter school, because in school children are taught to connect spoken and written language. Those who do not easily learn to read and write often have difficulty learning the alphabetic system—the relationship of sounds to letters. They also cannot manipulate sounds within syllables in words, which is called a lack of phonological awareness and is a precursor to reading problems (Rvachew, 2007).

**Phonological awareness** is a broad construct that includes recognizing the relationship between sounds and letters, detecting rhyme and alliteration, and being

aware that sounds can be manipulated within syllables in words. Primary-grade teachers detect phonological awareness as they ask children to rhyme words and manipulate sounds. For example, the teacher can say "hat" and ask the child to say the word without the *h* sound, or say "trip" and have the child say the word without the *p*. To assess the child's ability to blend sounds, teachers can say the three sounds *t*, *i*, and *n*, for example, and see whether the child can pull the sounds together to say "tin."

In addition to serving as a prerequisite for basic reading skills, phonological awareness and processing also appear highly related to expressive language development (Boada & Pennington, 2006). Readers with core deficits in phonological processing have difficulty segmenting and categorizing phonemes, retrieving the names of common objects and letters, storing phonological codes in short-term memory, and producing some speech sounds. Reading and comprehension depend on the rapid and automatic ability to decode single words. Children who are slow and inaccurate at decoding have the most difficulties in reading comprehension (Melby-Lervåg, Lyster, & Hulme, 2012).

## Section Summary

### Language Development

- Language development is based on innate ability and environmental opportunities to learn, store, and express important sounds in the language. It proceeds very rapidly during infancy.
- Deficits in phonological awareness—the ability to distinguish the sounds of language—have been identified as a major cause of communication and learning disorders.

# COMMUNICATION DISORDERS

Children with communication disorders have difficulty producing speech sounds, using spoken language to communicate, or understanding what other people say. In DSM-5, communication disorders include the diagnostic subcategories of *language disorder, speech sound disorder, childhood-onset fluency disorder (stuttering),* and *social (pragmatic) communication disorder*. These subcategories are distinguished by the exact nature of the child's impairment.

Recall that during development phonological problems appear before problems in language reception or expression, yet they have strong similarities. The following discussion focuses on language disorder in an effort to highlight early childhood problems that represent the fundamental features of communication disorders. (Stuttering has a unique clinical feature and developmental course, so it is discussed separately.)

Consider Jackie's communication problems at age 3 years:

**JACKIE**

### Screaming, Not Talking

Jackie's mother explained with no hesitation why she asked for help: "My 3-year-old daughter is a growing concern. Since she was a baby, she has been plagued by ear infections and sleep problems. Some nights she screams for hours on end, usually because of the ear infections. She has violent temper outbursts and refuses to do simple things that I ask her to do, like get dressed or put on her coat."

The child, waiting in the playroom, could be heard screaming over her mother's voice. Jackie was asking my assistant for something, but she could not make out what Jackie was saying. It was pretty obvious how frustrated both the child and her mother must feel on occasion. Her mother explained how she and Jackie's father had divorced when Jackie was less than 2 years old, and that after weekend exchanges it sometimes took a few days for Jackie's routine to return to some degree of normalcy.

I opened the letter she had brought from Jackie's preschool teacher, someone who I knew had a great deal of experience with children of this age. "Jackie is a bright and energetic child," the letter began, "but she is having a great deal of difficulty expressing herself with words. When she gets frustrated, she starts to give up or becomes angry—she won't eat her meals or she fights with staff at nap time, even if she is hungry or tired. If a new teacher at day care is introduced, it takes Jackie a long time to get used to the new person. Jackie seems to understand what she is being asked, but can't find the words to express herself, which understandably leads to an emotional reaction on her part." (Based on authors' case material.)

## Language Disorder

Jackie's problems met the criteria for a **language disorder,** which is a communication disorder characterized by difficulties in the comprehension or production of spoken or written language. As a result of these deficits, Jackie showed her frustration loudly and inappropriately.

Children's language development follows specific steps, although each child may proceed through the steps at a different pace. Normal variations can make it difficult to predict that a given child's early

communication problems will later become major problems in learning. A common example is the child who points to different objects and makes grunting or squealing noises that the parent quickly recognizes as "more milk" or "no peas." Prior to age 3 or so, many children communicate this way unless parents actively encourage using words and discourage nonverbal communications. Nevertheless, despite plenty of verbal examples and proper language stimulation, some children do not develop in some areas of speech and language, and they later have problems in school. This developmental connection makes the study of communication disorders highly pertinent to understanding and treating subsequent learning problems.

Table 7.1 shows the major features of the DSM-5 diagnostic criteria for language disorder. Children with a language disorder, such as Jackie, do not suffer from intellectual disability or from autism spectrum disorder, which affect speech and language. Rather, they show persistent difficulties in acquiring and using language to communicate (Criterion A). A child's ability to use language depends on both receptive skills (i.e., receiving and comprehending language) and expressive skills (i.e., production of vocal, gestural, or verbal signals). Thus, children with this disorder often show reduced vocabulary, limited sentence structure, or impairments in their ability to carry on a conversation. A child's expressive and receptive abilities may differ such that his or her language comprehension, for example, is stronger than his or her language expression. For example, when asked by her parents to go upstairs, find her socks, and put them on, Jackie was quite capable of complying. When asked by her mother to describe what she has just done, however, she might respond simply, "find socks."

The linguistic abilities of children with language disorders vary significantly, based on the severity of the disorder and the age of the child. Most often, these children begin speaking late and progress slowly in their speech development. Their vocabulary often is limited and is marked by short sentences and simple grammatical structure, as in Jackie's response. To fit the diagnostic criteria, these problems must be substantially below the abilities of other children of the same age, resulting in functional limitations in communication, social participation, or academic achievement (Criterion B). In addition, the symptoms must begin in the early developmental period (Criterion C) and not be attributable to other sensory impairments or medical conditions (Criterion D).

Although their hearing is normal, children with language disorder may have difficulty understanding particular types of words or statements, such as complex if–then sentences. In severe cases, the child's ability to understand basic vocabulary or simple sentences may be impaired, and there may be deficits in auditory processing of sounds and symbols and in their storage, recall, and sequencing. Understandably, these problems make the child seem inattentive or noncompliant, and the disorder can be easily misdiagnosed. Imagine how it would feel to be in Greece visiting an English-speaking host and her Greek husband. Unless your host is present, trying to engage in friendly conversation can be frustrating and uncomfortable. Even if both you and the husband can each understand a few words the other is saying, you probably cannot actually converse. If you have ever faced a similar communication barrier, you probably have an appreciation of the frustration and discomfort that accompany a language disorder.

When the developmental language problem involves articulation or sound production rather than word knowledge, a **speech sound disorder** may be an appropriate diagnosis. Children with this disorder have trouble controlling their rate of speech, or lag behind playmates in learning to articulate certain sounds. Typically, children learn phonemes and use intelligible speech by the age of 3 years or so, with the exception of some of the more difficult sounds such as *l, r, s, z, th,* and *ch,* which may take a few years longer to articulate (APA, 2013).

**TABLE 7.1** | **Diagnostic Criteria for Language Disorder**

**DSM-5**

**(A)** Persistent difficulties in the acquisition and use of language across modalities (i.e., spoken, written, sign language, or other) due to deficits in comprehension or production that include the following:

- **(1)** Reduced vocabulary (word knowledge and use).
- **(2)** Limited sentence structure (ability to put words and word endings together to form sentences based on the rules of grammar and morphology).
- **(3)** Impairments in discourse (ability to use vocabulary and connect sentences to explain or describe a topic or series of events or have a conversation).

**(B)** Language abilities are substantially and quantifiably below those expected for age, resulting in functional limitations in effective communication, social participation, academic achievement, or occupational performance, individually or in any combination.

**(C)** Onset of symptoms is in the early developmental period.

**(D)** The difficulties are not attributable to hearing or other sensory impairment, motor dysfunction, or another medical or neurological condition and are not better explained by intellectual disability (intellectual developmental disorder) or global developmental delay.

*Source:* Diagnostic and Statistical Manual of Mental Disorders, 5th edition. American Psychiatric Association.

Depending on the severity of the disorder, the speech quality of these children may be unusual, and even unintelligible. For example, at age 6, James still said "wabbit" instead of "rabbit" and "we-wind" for "rewind." Preschoolers, of course, often mispronounce words or confuse the sounds they hear, which is a normal part of learning to speak. When these problems persist beyond the normal developmental range (age 4) or interfere with academic and social activities by age 7, they deserve separate attention.

### Prevalence and Course

Children usually reveal problems in speech articulation and expression as they attempt to tackle new sounds and express their own concepts. Even though prevalence estimates account for normal variations in language development and are based on individuals who meet specific diagnostic criteria, the degree of severity can vary considerably. For example, in early childhood, milder forms of speech sound disorder are relatively common, affecting close to 10% of preschoolers. Many of these children outgrow their earlier difficulties, so only 2% to 3% of preschoolers meet the criteria for speech sound disorder. However, language disorder is a bit more common, affecting about 7% of younger school-age children across studies (Beitchman & Brownlie, 2014; Heim & Benasich, 2006).

Communication disorders are identified almost twice as often in boys as in girls (Pinborough-Zimmerman et al., 2007); boys' language difficulties are more often accompanied by behavior problems, and consequently, they are referred and diagnosed with communication learning disorders more often than girls (Vellutino et al., 2004). By 4 years of age a child's individual differences in language stabilize, so problems that remain past this age are highly predictive of later outcomes. About 50% fully outgrow their problems, whereas the other 50% may show improvement but still have some degree of impairment into adulthood. Children with receptive language impairments, in particular, have a poor prognosis as compared with those with primarily expressive impairments. Receptive language problems are more resistant to treatment and are often associated with reading difficulties throughout their education (APA, 2013).

Even though language problems usually diminish with time, children with communication disorders often have higher-than-normal rates of negative behaviors that began at an early age (van Daal, Verhoeven, & van Balkom, 2007). Associated behavior problems, such as attention-deficit/hyperactivity disorder (ADHD) and social skill limitations, can add to communication problems and further alter the course of development in terms of how these children relate to peers or keep up with educational demands (Durkin & Conti-Ramsden, 2010). To give children with special needs the opportunity to interact with typically developing children, school systems have begun to include these children in regular, rather than segregated, classrooms. **Inclusion** education strategies are based on the premise that the abilities of children with special needs will improve from associating with normally developing peers and that by doing so these children will be spared the effects of labeling and special placements.

### Causes

Notable findings that support the role of genetics, brain function, and environmental risk factors associated with a higher incidence of learning disorders are discussed in the following sections.

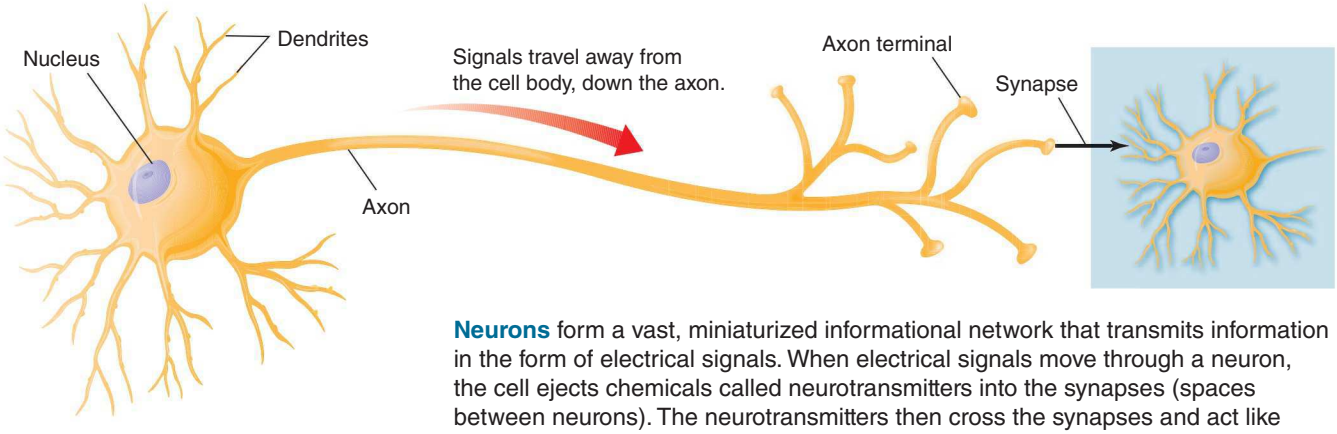
#### Genetics

Language processes appear to be heritable to a significant degree, although the specific genetic underpinnings are difficult to pinpoint. About 50% to 75% of all children with a language disorder show a positive family history of some type of learning disability (American Speech–Language–Hearing Association [ASHA], 2008; Heim & Benasich, 2006). Twin studies and adoption studies also suggest a genetic connection (McGrath et al., 2007; Plomin, Haworth, & Davis, 2010; Whitehouse et al., 2011).

Scientists are zeroing in on specific deficits in brain functioning that lead to communication disorders and may be heritable. Studies comparing language-impaired children with and without an affected parent suggest that *temporal processing deficits* occur significantly more often in children with a positive family history for a language-based learning disability (Caylak, 2011; Flax et al., 2003). That is, affected children have more difficulty deciphering certain speech sounds because of subtle but important differences in the way neurons fire in response to various sounds. In a twin study, Bishop et al. (1999) found that the variation in temporal processing was due to environmental factors and not to genetics because twin–twin correlations were similar for monozygotic (MZ) and dizygotic (DZ) twins. However, what does appear to be genetic is a deficit in phonological short-term memory.

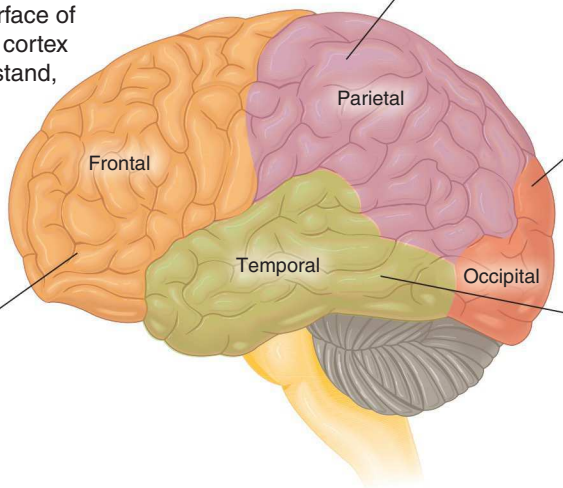
#### The Brain

Language functions develop rapidly and are housed primarily in the left temporal lobe of the brain




**Neurons** form a vast, miniaturized informational network that transmits information in the form of electrical signals. When electrical signals move through a neuron, the cell ejects chemicals called neurotransmitters into the synapses (spaces between neurons). The neurotransmitters then cross the synapses and act like switches to turn adjacent cells on or off.

The **cortex** is a thin layer of cells that essentially covers the entire surface of the forebrain. A well-developed cortex allows a person to read, understand, talk about, and remember the concepts in this text. The vast majority of our neurons are located in the cortex.




The parietal lobe is involved with perception and sensory experiences.

The occipital lobe is involved with processing visual information.

The frontal lobe is involved with personality, emotions, and motor behaviors.

The temporal lobe is involved with hearing and speaking.

● **FIGURE 7.1** | Areas of the brain involved in language functions.

From Plotnik/Kouyoumdjian. Introduction to Psychology, 9th ed. 2011 Wadsworth, a part of Cengage Learning, Inc

(see ● Figure 7.1). A circular feedback loop helps strengthen the developmental process of language reception and expression. The better children comprehend spoken language, the better they will be able to express themselves. Feedback from their own vocalizations, in turn, helps shape their subsequent expressions. Lack of comprehension and absence of feedback reduces verbal output, and thus interferes with the development of articulation skills (Vellutino et al., 2007).

Anatomical and neuroimaging studies show that deficits in phonological awareness and segmentation are related to problems in the functional connections between brain areas, not to a specific dysfunction of any single area of the brain (Lyon et al., 2006; Richlan, Kronbichler, & Wimmer, 2011). Some brain imaging studies have indicated that poor performance on tasks demanding phonological awareness is associated with less brain activity in the left temporal region, suggesting that phonological problems may stem from neurological deficits or deviations in posterior left-hemisphere systems that control the ability to process phonemes (Richlan et al., 2011; S. E. Shaywitz et al., 2009). We return to these findings on brain function later, in our discussion of reading disorders.

Recurrent otitis media (middle ear infection) in early childhood was long thought to contribute to language difficulties, because hearing loss accompanies frequent or long bouts of infections. Although otitis media that occurs often during early childhood may lead to speech and language delays, these delays improve relatively quickly and largely disappear by age 7 (Zumach et al., 2010).

In summary, although biological findings point to abnormal brain functioning, how this abnormality originates is still unclear. The best guess is that communication

disorders result from an interaction of genetic influences, slowness or abnormalities of brain maturation, and possibly, minor brain lesions that escape clinical detection (S. E. Shaywitz, Morris, & Shaywitz, 2008).

### *Home Environment*

How much does the home environment contribute to communication disorders? Do some parents fail to provide adequate examples to stimulate their children's language? Because of the important role parents play in children's development, psychologists have studied this issue carefully.

We noticed when we first visited Jackie at home that her stepfather was a very quiet man who often communicated nonverbally—a gesture, a frown, a short phrase. Her mother used very simple speech when talking to Jackie but not when talking to Jackie's 6-year-old sister. These observations match those of researchers (St. James-Roberts & Alston, 2006; Whitehurst & Lonigan, 1998) who compared verbal interactions of families with and without a child who had a language disorder. They found that parents changed the way they spoke to their children, depending on their children's abilities. When the child spoke in simple, two- or three-word sentences, the parents adjusted their speech accordingly. Note that, except in extreme cases of child neglect or abuse, it is unlikely that communication disorders are caused by parents. Parental speech and language stimulation may affect the pace and range of language development, but not the specific impairments that characterize the disorders (Glascoe & Leew, 2010; McGrath et al., 2007).

### *Treatment*

Although communication disorders in some children may self-correct by age 6 or so, those with more severe communication and language difficulties will continue to lag behind their peers and are at risk of havingbehavioral or social problems if the difficulties are left untreated. Thus, parents should seek help in understanding their child's speech delays and to ensure that they are doing everything possible to stimulate language development. In general, treatment for children with communication disorders is based on three principles (Beitchman & Brownlie, 2014): (1) treatment to promote the child's language competencies; (2) treatment to adjust the environment in ways that accommodate the child's needs; and (3) therapy with the child (or youth) to equip him or her with knowledge and skills to reduce behavioral and emotional symptoms.

Specialized preschools, for example, have had good results using a combination of computer- and teacher-assisted instruction to teach early language skills to young children, which helps to pace the child's practice of new skills (Hatcher et al., 2006; Loo et al., 2010; Smith-Lock et al., 2013).

For Jackie, we designed ways that her parents and day-care teachers could build on her existing strengths. Her day-care teacher had an excellent idea: Because Jackie loved to draw and to talk about her artwork, why not use her interest in drawing to increase her enthusiasm for speaking? When I visited her class, Jackie ran up to show me her drawing, exclaiming, "I draw picture of mom, dad, kitty, and lake." We agreed that her behavior problems could be managed by simple forms of ignoring and distracting and the occasional time-out. Jackie became attached to computer graphics and images, and she soon was able to identify letters and small words and to move shapes around the screen. All the while, her expressive language improved, and by age 5 she could pronounce all the letters of the alphabet and was eager to start kindergarten.

## Childhood-Onset Fluency Disorder (Stuttering)

Childhood-Onset Fluency Disorder (Stuttering) is the repeated and prolonged pronunciation of certain syllables that interferes with communication. It is quite normal for children who are still learning to speak to go through a period of nonfluency, or unclear speech, as part of their development. It takes practice and patience for a child to develop the coordination for the tongue, lips, and brain to work in unison to produce unfamiliar or difficult combinations of sounds. For most children, this period of speech development passes without notice, and for most parents it is full of wonder and amusement as their children wrestle with new words. Some children, however, progress slowly through this stage, repeating (*wa-wa-wa*) or prolonging (*n-ah-ah-ah-o*) sounds; they struggle to continue or develop ways to avoid or compensate for certain sounds or words. Four-year-old Sayad has speech problems that typify the pattern of stuttering:

**SAYAD**

### Family Legacy

Sayad's parents had received a lot of informal advice from friends and relatives about their son's speech problems, but most of what they said was worrisome. "He'll struggle with this for most of his life," his grandmother had warned. "If something isn't done right away, he'll become a stutterer, and be so self-conscious that he won't be able to keep up in school or with his friends."

(*continues*)

(*continued*)

Sayad started repeating and prolonging some of his words when he was about 2, but now his problem had grown more noticeable. As he spoke, he pursed his lips, closed his eyes, and shortened his breathing, seeming to tense up his face. Yet his interactions with me were friendly and at ease. "M-m-m-m-y words get stuck in m-m-m-m-y m-m-mouth," he explained, "and I-I-I-I talk t-t-t-t-too fast. Wh-wh-wh-why can't I talk right?" I soon discovered why his grandmother was so concerned: The child's great-grandfather and great-uncle both stuttered, and Sayad's father had been a stutterer until he was a teenager.

Sayad's mother had been trying to ignore the problem and not draw attention to it, but she was growing more aware that Sayad's peers teased and imitated him. She explained why she came for an assessment: "We were on the way to the store when Sayad kept saying 'where' over and over. After I stopped the car and unfastened his seatbelt, he finished his question—'is daddy?' After that, I gave up on my 'leave it alone' notion and began trying ways to slow Sayad down a bit." (Based on authors' case material.)

**TABLE 7.2** | **Diagnostic Criteria for Childhood-Onset Fluency Disorder (Stuttering)**

**DSM-5**

**(A)** Disturbances in the normal fluency and time of patterning of speech that are inappropriate for the individual's age and language skills, persist over time, and are characterized by frequent and marked occurrences of one (or more) or the following:

- **(1)** Sound and syllable repetitions.
- **(2)** Sound prolongations of consonants as well as words.
- **(3)** Broken words (e.g., pauses within a word).
- **(4)** Audible or silent blocking (filled or unfilled pauses in speech).
- **(5)** Circumlocutions (word substitutions to avoid problematic words).
- **(6)** Words produced with an excess of physical tension.
- **(7)** Monosyllabic whole-word repetitions (e.g., "I-I-I-I" see him).

**(B)** The disturbance causes anxiety about speaking or limitations in effective communication, social participation, or academic or occupational performance, individually or in any combination.

**(C)** The onset of symptoms is in the early developmental period (*Note:* Later-onset cases are diagnosed as adult-onset fluency disorder).

**(D)** The disturbance is not attributable to a speech-motor or sensory deficit, dysfluency associated with neurological insult (e.g., stroke, tumor, trauma), or another medical concern and is not better explained by another mental disorder.

*Source:* Diagnostic and Statistical Manual of Mental Disorders, 5th ed. American Psychiatric Association.

DSM-5 diagnostic criteria for Childhood-Onset Fluency Disorder (Stuttering) are shown in Table 7.2. This disorder involves disturbance in the normal fluency and time patterning of speech that is atypical for the child's age and that occurs often and persists over time. Stuttering is characterized by sound and syllable repetitions, sound prolongations, pauses within a word, word substitutions to avoid problematic words, and similar pronunciation and speech difficulties. These difficulties lead to anxiety about speaking or participating in activities that require effective communication or social participation. Over time the child may develop a fearful anticipation of speaking in front of others and attempt to avoid speech situations such as talking by telephone or in class. The disorder may be accompanied by motor movements such as eye blinks, tics, tremors of the lips or face, etc. (APA, 2013).

### Prevalence and Course

Stuttering is relatively common as young children learn to articulate sounds clearly and appropriately. Population-based surveys indicate that 11% of children stutter by age 4, with girls affected as much as boys (Reilly et al., 2013; Yairi & Ambrose, 2013). However, few children receive a diagnosis of childhood-onset fluency disorder because about 80% recover from stuttering as they attend school for a year or so (Packman, Code, & Onslow, 2007). The prevalence of stuttering across the lifespan (i.e., the number of individuals of all ages who meet the diagnostic criteria at any point in time) is below 1% (Yairi & Ambrose, 2013). Higher rates of parent-reported stuttering have been noted among African American and Hispanic children in the United States, although how racial and cultural factors may affect stuttering remains unclear (Boyle et al., 2011).

### Causes and Treatment

Many myths and falsehoods surround stuttering. The widely held view that stuttering is caused by an unresolved emotional problem or by anxiety is not supported by any evidence (Packman et al., 2007). Because the problem runs in families, researchers have focused on family characteristics as the major causes. However, it is not likely this behavior is acquired primarily as a function of the child's linguistic environment. Sayad's grandmother and mother would be relieved to know that the communicative behavior of mothers does not significantly contribute to the development of stuttering (Howell & Davis, 2011).

Genetic factors play a strong role in the etiology of stuttering, accounting for approximately 70% of the variance in the causes of stuttering (Dworzynski et al., 2007). Environmental factors, such as premature birth or parental mental illness, account for the remaining causal influences (Ajdacic-Gross et al., 2010). Genetic factors most likely influence speech by causing an abnormal development in the location of the most prominent speech centers in the brain, which are usually in the left hemisphere. This biological source for stuttering explains many of its clinical features, including the loss of spontaneity and occasional problems in self-esteem (Howell, 2011; Kell et al., 2009).

Since most children outgrow stuttering, one of the most frustrating problems for parents and therapists is to decide whether therapy would be intervention or interference. Therapy is usually recommended if sound and syllable repetitions are frequent, if the parent or child is concerned about the problem, or if the child shows, like Sayad, facial or vocal tension. A common psychological treatment for children who stutter is to teach parents how to speak to their children slowly and use short and simple sentences, consequently removing the pressure the child may feel about speaking (Howell, 2011; Rousseau, Packman, Onslow, Harrison, & Jones, 2007). Other beneficial treatments for stuttering include contingency management, which uses positive consequences for fluency and negative consequences for stuttering (Bothe et al., 2006; Murphy, Yaruss, & Quesal, 2007), and habit reversal procedures, such as learning to regulate breathing (Bate et al., 2011).

## Social (Pragmatic) Communication Disorder

Social (Pragmatic) Communication Disorder (SCD) is a new disorder in DSM-5. It involves persistent difficulties with pragmatics—the social use of language and communication (APA, 2013). Pragmatics are culturally specific practices and skills related to social uses of language, conversational norms, and the use of nonverbal communication, such as eye contact and gestures (Beitchman & Brownlie, 2014). Pragmatic difficulties involve both expressive and receptive skills—being able to adapt one's communication to the social context and being able to understand the nuances and social meanings expressed by others.

The first requirement for a diagnosis of social (pragmatic) communication disorder involves persistent difficulties across four areas (Table 7.3):

- Deficits in using communication for social purposes. A child may show difficulty greeting others or sharing information appropriately.
- Difficulties changing their communication to match the situation or the listener, such as the classroom versus the playground.
- Problems following the rules of language, such as taking turns in a conversation,
- Difficulties understanding what someone is not explicitly saying, such as being able to make inferences based on the context of the situation.

A diagnosis of SCD is not typically made until the child is 4 or 5 years old, to determine whether he or she has shown adequate developmental progress in speech and language. Signs of language impairment, such as a history of delay in reaching language milestones, are common, but it is the specific deficits in social communication that determine this disorder. As with other

**TABLE 7.3** | **Diagnostic Criteria for Social (Pragmatic) Communication Disorder**

**DSM-5**

**(A)** Persistent difficulties in the social use of verbal and nonverbal communication as manifested by all of the following:

- **(1)** Deficits in using communication for social purposes, such as greeting and sharing information, in a manner that is appropriate for the social context.
- **(2)** Impairment of the ability to change communication to match context or the needs of the listener, such as speaking differently in a classroom than on a playground, talking differently to a child than to an adult, and avoiding use of overly formal language.
- **(3)** Difficulties following rules for language and storytelling, such as taking turns in conversation, rephrasing when misunderstood, and knowing how to use verbal and nonverbal signals to regulate interaction.
- **(4)** Difficulties understanding what is not explicitly stated (e.g., making inferences) and nonliteral or ambiguous meanings of language (e.g., idioms, humor, metaphors, multiple meanings that depend on the context for interpretation).

**(B)** The deficits result in functional limitations in effective communication, social participation, social relationships, academic achievement, or occupational performance, individually or in combination.

**(C)** The onset of the symptoms is early in the developmental period (but deficits may not become fully manifest until social communication demands exceed limited capacities).

**(D)** The symptoms are not attributable to another medical or neurological condition or to low abilities in the domains of word structure and grammar, and are not better explained by autism spectrum disorder, intellectual disability (intellectual developmental disorder), global developmental delay, or another mental disorder.

*Source:* Diagnostic and Statistical Manual of Mental Disorders, 5th ed. American Psychiatric Association.

communication disorders, onset must be early in development and result in functional limitations in communication, social participation, social relationships or academic functioning.

Social (pragmatic) communication disorder was added to the DSM because of the number of children who did not meet conventional criteria for an autism spectrum disorder (ASD) yet who had persistent difficulties with social aspects of communication and peer relations. SCD is differentiated from ASD largely on the basis of fewer restricted/repetitive patterns of behaviors and interests (Gibson et al., 2013). The symptoms of SCD also overlap with ADHD, social anxiety disorder, and intellectual disability in that they share problems in social, pragmatic communication. Although this diagnostic category is new, studies based on similar samples of children suggest that some children show improvement over time while others continue to show social communication deficits into adulthood. Regardless of improvements in social communication, children with SCD may suffer lasting impairments in peer relations due to their early difficulties. Thus, peer-assisted interventions are recognized as effective ways to build pragmatic communication and social skills for these children (Murphy, Faulkner, & Farley, 2014).

## Section Summary

### Communication Disorders

- Speech and language problems that emerge during early childhood include difficulty producing speech sounds, demonstrating speech fluency, using spoken language to communicate, or understanding what other people say.
- Even though most children with communication disorders acquire normal language by mid-to-late adolescence, early communication disorders are developmentally connected to the later onset of learning disorders.
- Language disorder is a communication disorder involving difficulties in comprehension or production of spoken or written language.
- Childhood-Onset Fluency Disorder (Stuttering) is relatively common among younger children, and declines significantly once the child enters school.
- Social (Pragmatic) Communication Disorder is new to DSM-5. Its primary characteristics involve difficulties in the social use of verbal and nonverbal communication.
- Causes of communication disorders include genetic influences and slow or abnormal brain maturation.
- Many communication disorders resolve themselves after children begin attending school. Treatment is recommended for children who show significant language delays or difficulties; it involves accommodating the child's needs to strengthen speech and language skills.

# SPECIFIC LEARNING DISORDER

*People do not understand what it costs in time and suffering to learn how to read. I have been working at it for eighty years, and I still can't say that I've succeeded.*

—Goethe (1749–1832)

Whether we are studying Roman history or calculus, applying ourselves to the task of learning requires exertion and concentration. Like physical activities, some learning activities are more difficult than others, especially for younger children who have not developed a foundation of good study habits and successful learning experiences. Parents and teachers may notice that a child is struggling unusually hard to master a particular skill, such as reading, and wonder why. The problem may be formally assessed by an IQ test and various standardized tests that assess abilities in specific academic areas.

When achievement in reading, math, or writing is well below average for the child's age and intellectual ability, he or she may be diagnosed with a specific learning disorder (SLD). In other words, a child with a specific learning disorder is intellectually capable of learning key academic concepts of reading, writing, and math, but seems unable to do so. The phrase "unexpected academic underachievement" captures this notion that the child's learning problems are indeed *specific* and not due to intellectual disability or global developmental delay (APA, 2013).

### JAMES

### Strong Points Shine

The look on the 9-year-old's face said it all—he did not want to be here. "I'm tired of talking to people" was his terse greeting. I wondered for a moment whether he would talk to me at all, but as soon as he saw my computer, he brightened a bit. To allow time for him to feel more comfortable, I invited James to play a quick game or two. His skill at the action games told me a lot about his basic energy and problem-solving ability—he was a whiz at figuring out the rules of each game and getting a high score. We spoke casually during the warm-up, but it was clear to me that he preferred to concentrate on the game.

A half hour passed, with little more than a few sentences exchanged. A quick trip to the snack bar gave us the common ground we needed to open up and talk a bit. "Why does my teacher want me to come here?" he reasonably asked. As he listened and replied to my explanation, his language problems stood out. His sentences were short, simple, and rapid. Here is an example:

"James, tell me something about your favorite story or a recent movie you've seen."
"I like the movie. Lots of dogs."
"What movie is that, James?"
"Dog movie."

During testing, James often tried to start before I had finished telling him what to do. He was eager to do what I asked, but he stopped abruptly as soon as he had trouble. James could focus on only one sound at a time, so if he missed early cues or initial instructions, he would become disoriented, frustrated, and uncooperative. James wanted to do well, but I could see he was struggling. He completed the WISC-IV (Wechsler Intelligence Scale for Children, 4th ed.) in less than an hour, hurrying almost as if to escape his own mistakes. His measured general intelligence was within the normal range, but his performance abilities (performance IQ, 109) were much stronger than his verbal abilities (verbal IQ, 78). It was obvious as well that the test underestimated his true ability, as a result of his eagerness to finish and his difficulty with understanding some of the instructions.

To my surprise, James was ready to continue on to the next test after only a short computer game break. He explained why this was so: "I put things together, like puzzles. I make cars and planes at my house." As long as I gave him small breaks on the computer, he was willing to tackle the material on the tests. Some of his spelling errors stood out immediately, such as *skr* for *square*, and *srke* for *circle*. When asked to write the sentence "he shouted a warning," he wrote "he shtd a woin." He read "see the black dog" as "see the black pond," and "she wants a ride to the store" as "she was rid of the store." He seemed to use a "best guess" strategy in tackling reading, based on the sounds that he knew: When asked to write the word bigger, he wrote just her. But I noticed that James's enthusiasm picked up a bit as he began telling stories from pictures he was shown, and he marveled at his own ability to rotate shapes on the computer to complete a picture. He left my office more animated and talkative than when he arrived, which showed how nice it must have felt for him to experience success. (Based on authors' case material.)

James, at age 9, had problems primarily in reading and spelling. Contrast his reading difficulties with those of Tim, who struggles with spatial orientation and mathematical reasoning.

**TIM**

## Warming with Interest

When I first saw Tim, he seemed aloof and disinterested. His eyes stayed focused on the floor, and his body remained expressionless, as if to say, "Leave me alone, and let me outta here." As I searched for something to say, I asked Tim to tell me a little about his family: "Do you have any brothers or sisters? Does your family like to do anything special together?" His tired response, "I have two brothers, my father works all day, mom plays piano. We want a boat," sent me a clear message as to his mood and interest in this activity. My usual ploy of turning on the computer games fell flat—"I hate computers" was Tim's preemptive response. I wondered, "Is he depressed, angry, hurt, frustrated? Just what is going on here?"

Having looked at his school record, I knew he was struggling, especially in math and physical sciences, but his speech and affect expressed more than only academic problems. His school records flashed the news that Tim had a specific learning disorder, as evidenced by his WISC-IV performance score of 79 that fell in the borderline-to-low-average range, and his verbal score of 108 that fell in the average range. The test administrator had politely described Tim's test-taking approach as "reluctant." Notes by teachers indicated that he commonly had problems on tasks involving drawing, particularly if they required memory, and his math and social skills were far below those of others in his class.

I pulled out my *Where's Waldo?* book and we began looking at it together. In addition to being fun, looking for Waldo and his friends (small figures amidst millions of figures and colors) required Tim to be patient. At first he balked, but I noticed that he improved if he used his own verbally mediated strategy to solve the problem. Tim talked to himself as he thought aloud: "Look around the edges first, then start to look closer and closer to the middle of the page. Look for Waldo's red and white shirt—look closely at each section!" The more interested he was, the more he would talk. Once he warmed up, his smile appeared, along with his admission that "this sure beats math lesson." (Based on authors' case material.)

James's pattern of strengths and weaknesses shows that although he has reading problems, other strengths compensate for this disability. He has strong talents for figuring out how things work and for drawing ideas on paper. Tim has several strengths, too, especially in linguistic skills such as word recognition, sentence structure, and reading. In contrast to James, Tim has problems primarily in the visual, spatial, and organizational spheres, which show up as difficulties with tactile (touch) perception, psychomotor activity (e.g., throwing and catching), and nonverbal problem solving (e.g., figuring out math problems and assembling things).

Both boys fit the diagnostic criteria for SLD. Note how Tim's academic problems, in particular, were almost masked by his frustration and low self-esteem.

Zigy Kaluzny/The Image Bank/Getty Images

For children with specific learning disorders, following simple instructions can be confusing and frustrating.

Emotional problems are often seen in children who are bright enough to recognize that their performance is below that of others and are frustrated with their poor performance at school. The limitations of both James and Tim can affect every aspect of their formal education as well as their interpersonal abilities; therefore, these disorders require comprehensive and ongoing treatment plans.

To understand the nature of specific learning disorders, picture yourself asking for directions to a famous monument at an information booth in an unfamiliar town. The attendant hands you a map with written directions: "Go out the driveway and turn right. Go till you reach the second light, turn left, and look for the sign to Amityville. It's about 3 miles down the road. You'll pass a cemetery and a red schoolhouse, and go under a railroad trestle before you get to Highway 18. When you see the sign, turn right." Most of us would have trouble recalling these verbal directions, so having them written in the map makes them easier to follow. However, children or adults with an SLD in reading experience confusion in these common situations that involve understanding the meaning of what is read. Specific learning problems can be difficult to recognize because, for most of us, the material in question is straightforward and simple. The child may be blamed for not listening, not paying attention, or for being "slow," which further disguises the true nature of the learning problems.

The main diagnostic feature of SLD is that the child has difficulties learning keystone academic skills of reading, writing, spelling, or math (see Table 7.4). These difficulties may appear in one or more of these

**TABLE 7.4 | Diagnostic Criteria for Specific Learning Disorder**

**DSM-5**

**(A)** Difficulties learning and using academic skills, as indicated by the presence of at least one of the following symptoms that have persisted for at least six months, despite the provision of interventions that target those difficulties:

- **(1)** Inaccurate or slow and effortful word reading (e.g., reads single words aloud incorrectly or slowly and hesitantly, frequently guesses words, has difficulty sounding out words).
- **(2)** Difficulty understanding the meaning of what is read (e.g., may read text accurately but not understand sequence, relationships, inferences, or deeper meanings of what is read).
- **(3)** Difficulties with spelling (e.g., may add, omit, or substitute vowels or consonants).
- **(4)** Difficulties with written expression (e.g., makes multiple grammatical or punctuation errors within sentences; employs poor paragraph organization; written expression of ideas lacks clarity).
- **(5)** Difficulties mastering number sense, number facts, or calculation (e.g., has poor understanding of numbers, their magnitude, and relationships; counts on fingers to add single-digit numbers instead of recalling the math fact as peers do; gets lost in the midst of arithmetic computation and may switch procedures).
- **(6)** Difficulties with mathematical reasoning (e.g., has severe difficulty applying mathematical concepts, facts, or procedures to solve quantitative problems).

**(B)** The affected academic skills are substantially and quantifiably below those expected for the individual's chronological age, and cause significant interference with academic or occupational performance, or with activities of daily living, as confirmed by individually administered standardized achievement measures and comprehensive clinical assessment. For individuals aged 17 years and older, a documented history of impairing learning difficulties may be substituted for the standardized assessment.

**(C)** The learning difficulties begin during school-age years but may not become fully manifest until the demands of those affected academic skills exceed the individuals limited capacities (e.g., as in timed tests, reading or writing lengthy complex reports for a tight deadline, excessively heavy academic loads).

**(D)** The learning difficulties are not better accounted for by intellectual disabilities, uncorrected visual or auditory acuity, other mental or neurological disorders, psychosocial adversity, lack of proficiency in the language of academic instruction, or inadequate educational instruction.

*Note:* The four diagnostic criteria are to be met based on a clinical synthesis of the individual's history (developmental, medical, family, educational), school reports, and psychoeducational assessment.

(*continues*)

**TABLE 7.4 | Diagnostic Criteria for Specific Learning Disorder** (*continued*)

*Specify if*:

**With impairment in reading:**

Word reading accuracy

Reading rate or fluency

Reading comprehension

**With impairment in written expression:**

Spelling accuracy

Grammar and punctuation accuracy

Clarity or organization of written expression

**With impairment in mathematics:**

Number sense

Memorization of arithmetic facts

Accurate or fluent calculation

Accurate math reasoning

*Specify* current severity:

**Mild:** Some difficulties learning skills in one or two academic domains, but of mild enough severity that the individual may be able to compensate or function well when provided with appropriate accommodations or support services, especially during the school years.

**Moderate:** Marked difficulties learning skills in one or more academic domains, so that the individual is unlikely to become proficient without some intervals of intensive and specialized teaching during the school years. Some accommodations or supportive services at least part of the day at school, in the workplace, or at home may be needed to complete activities accurately and efficiently.

**Severe:** Severe difficulties learning skills, affecting several academic domains, so that the individual is unlikely to learn these skills without ongoing intensive individualized and specialized teaching for most of the school years. Even with an array of appropriate accommodations or services at home, at school, or in the workplace, the individual may not be able to complete all activities efficiently.

*Source:* Diagnostic and Statistical Manual of Mental Disorders, 5th ed. American Psychiatric Association.

skills, including inaccurate or slow reading, difficulty understanding the meaning of what is read, difficulties with spelling and/or written expression, or difficulties mastering number sense, calculation, or mathematical reasoning. The affected academic skills would need to be substantially below what it should be for the child's age and intellectual ability. In practice, this often means that the child's achievement test scores in academic subjects are at least 1.5 standard deviations below average for their age and sex (which translates to a standard score of 78, or below the seventh percentile).

To be classified as a disorder, the performance problems must significantly interfere with academic achievement or daily living, and to persist for more than 6 months despite efforts to improve them. (Some children and adults have found ways to compensate for their learning problems and therefore do not display a disability, despite their test findings or poor achievement.) Finally, the learning difficulties appear during the school-age years, and cannot be better accounted for by a sensory problem (such as impaired hearing or sight), intellectual disability, psychosocial adversity, or inadequate educational instruction.

Because many aspects of speaking, listening, reading, writing, and arithmetic overlap and build on the same functions of the brain, it is not surprising that a child or adult can have more than one form of SLD (Scanlon, 2013). Recall that phonological awareness facilitates the ability to speak and, later on, to read and write. A single gap in the brain's functioning can disrupt many types of cognitive activity. These disruptions, in turn, can interfere with the development of important fundamental skills and compound the learning difficulties in a short time. Moreover, as we saw with both James and Tim, numerous secondary problems can emerge,

such as temper outbursts and withdrawal from social situations, as a result of frustration and lack of success.

As noted earlier in this chapter, DSM-5 integrates the frequently co-occurring problems in reading, mathematics, and written expression into one category, and uses specifiers to designate all academic domains and subskills that are impaired. Table 7.4 also describes the degrees of severity (mild, moderate, or severe) associated with each impairment. Degrees of severity reflect both the extent of the child or adolescent's learning difficulties as well as the appropriate accommodations or supports he or she requires to learn the academic skill(s) and complete activities at school, work or home as efficiently as possible. Below we take a closer look at the three core academic skill impairments specified in SLD.

## SLD with Impairment in Reading

*He has only half learned the art of reading who has not added to it the more refined art of skipping and skimming.*

—Arthur James Balfour

Children are naturally attracted to reading, and its importance in our society is unequaled by any other academic accomplishment. We are surrounded by written signs and messages and, by about age 5 or so, most children want to know what they mean. (Capitalizing on this natural curiosity, advertisers have become expert in pairing recognizable symbols with the names of their product or establishment so that children can "read" more quickly.) By the first grade, natural interest and developmental readiness are channeled into formally learning how to read. For many children, this process is difficult and tedious; for a sizable minority, however, it can be confusing and upsetting. The role of parents in this process is critical, because children need positive feedback and need to feel satisfied with their performance, regardless of their speed and accuracy.

When you consider everything involved in learning the basics of reading, such as associating shapes of letters (graphemes) with sounds (phonemes), it is not surprising that some children have difficulty and can quickly fall behind. Read the following sentence: "I believe that abnormal child psychology is one of the most fascinating and valuable courses I have taken." As you read the sentence, did you notice that you had to simultaneously:

- Focus attention on the printed marks and control your eye movements across the page?
- Recognize the sounds associated with letters?
- Understand words and grammar?
- Build ideas and images?
- Compare new ideas with what you already know?
- Store ideas in memory?

Most of us have forgotten all the effort that goes into reading, especially in the beginning. Not surprisingly, children's initial attempts are laborious and monotonous as they wrestle with the sounds and complexities of combined letters. Such mental processing requires a complex intact network of nerve cells that connect our vision, language, and memory centers (Grigorenko, 2007). A small problem in any area can cause reading difficulties. The most common underlying feature of a reading disorder, however, is an inability to distinguish or to separate the sounds in spoken words. Phonological skills are fundamental to learning to read, and therefore this deficit is critical.

To assess a child's need for additional practice in mastering phonemes and words, it is important to understand that there are two systems that operate when one reads words, which are essential in the development of reading. The first system operates on individual units (phonemes) and is relatively slow; the second system operates on whole words more quickly. In normal readers, whole words are learned through the development of phonologically based word analysis. However, persistently poor readers seem to rely on rote memory for recognizing words (S. E. Shaywitz & Shaywitz, 2013).

Many clinical signs of reading disorders are first evident only to a trained eye. Some testing methods developed by teachers and school psychologists show how children with reading disorders function in the classroom. They often have trouble learning basic sight words, especially those that are phonetically irregular and must be memorized, such as *the, who, what, where, was, laugh, said,* and so forth. These children have developed their own unique and peculiar reading patterns, which signal the need for different teaching methods. Typical errors include *reversals (b/d; p/q), transpositions* (sequential errors such as *was/saw, scared/sacred*), *inversions (m/w; u/n)*, and *omissions* (reading *place* for *palace* or *section* for *selection*). However, these errors are common in many younger children who are just learning to read and write and do not necessarily imply a reading disorder.

To assess a child's need for additional practice in certain areas, teachers may log the types of errors the child makes while reading out loud. In addition to decoding words, reading comprehension is assessed by having the student retell a story or suggest the next episode. Average readers rely heavily on auditory and visual modalities for gathering new information, but children with reading disorders may prefer a mode

of touch or manipulation to assist them in learning. These various patterns of strengths and weaknesses, if adequately assessed, can then be used to the child's advantage in planning additional teaching methods such as computer-based learning (S. E. Shaywitz et al., 2008).

A child with a SLD with impairment in reading lacks the critical language skills required for basic reading: word reading accuracy, reading comprehension, and reading rate or fluency. **Dyslexia** is an alternative term sometimes used to describe this pattern of reading difficulties. These core deficits stem from problems in **decoding**—breaking a word into parts rapidly enough to read the whole word—coupled with difficulty reading single small words (Vellutino et al., 2007). When a child cannot detect the phonological structure of language and automatically recognize simple words, reading development will very likely be impaired (Peterson & Pennington, 2010). The slow and labored decoding of single words requires substantial effort and detracts from the child's ability to retain the meaning of a sentence, much less a paragraph or page.

## SLD with Impairment in Written Expression

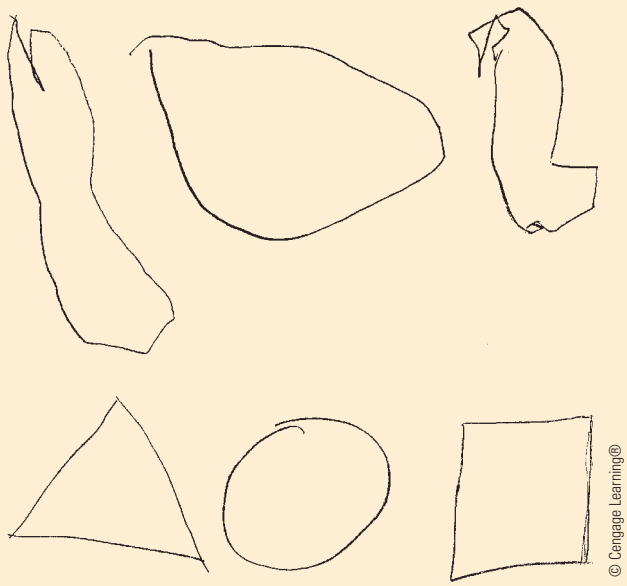
### CARLOS

### Slowly Taking Shape

Carlos, age 7, was about to finish second grade when his teacher and parents met to discuss his handwriting problems. The year had gone well in general, but his parents were bracing for bad news. Smiling and pulling out some workbooks, Carlos's teacher lined up examples of how he had gradually become able to print some letters over the course of the year. But what his parents saw was self-explanatory: His shapes were very poor and looked more like those of his 3-year-old sister. Sensing both parents' apprehension, his teacher clarified: "Carlos is having a few problems in his fine motor coordination, in activities such as artwork, putting puzzles together, and similar tasks. He goes too fast when trying to do these tasks, and he forgets to be careful or to follow the pattern. He makes a half-hearted attempt on his writing assignments and then starts talking to his classmates. I'd like him to be seen by a psychologist for testing, and hopefully next fall his new teacher can strengthen his writing and fine motor skills with some additional exercises."

During the initial interview, Carlos took an immediate interest in my computer games, exclaiming how easy it was to use the mouse to draw figures. When asked to use a pencil and paper, however, Carlos balked. I asked him to copy by hand some of the figures he drew on the computer, after first printing them for him on paper. In doing so, he switched to his preferred hand in the middle of the task. He also showed several letter reversals (b/d; p/q), and pushed down very hard on the pencil in an attempt to trace or draw the figures. Throughout these tasks he talked freely and asked a lot of questions, making me wonder at times who was assessing whom.

Carlos showed evidence on neuropsychological testing of finger *agnosia* (he could not tell which finger I touched when his hand was behind his back), especially with his left hand. He also had considerable difficulty copying a triangle, a circle, and a square based on examples shown to him (see ● Figure 7.2). On the WISC-IV he obtained a performance score of 91, in the low-average range, and a verbal IQ score of 117, in the high-average range. On performance subtests he had particular problems with block design and puzzles, such as object assembly. He had more difficulty with verbal IQ subtests that involved concentration and attention, such as math and digit-span tasks. Throughout the testing, I found Carlos to be impulsive and sometimes quite defiant: If he didn't want to do something, he simply would not do it. These observations were consistent with his parents' frustration at his immature behavior and defiance at home.




● **FIGURE 7.2** | *Top*: Drawings produced by Carlos when asked to copy a triangle, a circle, and a square. *Bottom*: Examples of a triangle, circle, and square from a typically developing 7-year-old boy.

(Based on authors' case material.)

Carlos has a specific learning disorder related to written expression. He has strong language and reasoning abilities, as well as normal problem-solving skills for his

age, yet he is considerably weaker in his visual–motor abilities, as shown by his writing, figure copying, and figure rotation. Like reading and math, writing derives from several interconnected brain areas that produce vocabulary, grammar, hand movement, and memory.

SLD with impairment in written expression may manifest as problems in spelling accuracy, grammar and punctuation accuracy, and/or clarity or organization of written expression. This particular SLD is often found in combination with SLD in reading or mathematics, which also have underlying core deficits in language and neuropsychological development.

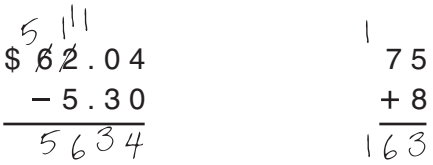
Children with impairment in written expression often have problems with tasks that require eye–hand coordination, despite their normal gross motor development. Teachers notice that, as compared with children who have normal writing skills, children with impairments in writing produce shorter, less interesting, and poorly organized essays and are less likely to review spelling, punctuation, and grammar to increase clarity (Hooper et al., 2011, 2013). However, spelling errors or poor handwriting that do not significantly interfere with daily activities or academic pursuits do not qualify a child for this diagnosis. In addition, problems in written expression signal the possibility of other learning problems because of shared metacognitive processes: planning, self-monitoring, self-evaluation, and self-modification (Heim & Benasich, 2006).

## SLD with Impairment in Mathematics

During their preschool years, children are not as naturally drawn to mathematical concepts as they are to reading. This changes rapidly as they discover that they need to count and add to know how much money it takes to buy something or how many days remain until vacation. As in reading, the need to know propels children to learn new and difficult concepts, and little by little their new skills help them understand the world better.

For some children, like Francine and Tim, this curiosity about numbers is compromised by their inability to grasp the abstract concepts inherent in many forms of numerical and cognitive problem solving. Francine's difficulty with numbers and concepts began to show up well before she attended school, which is typically the case. When she encountered math concepts in second grade that required some abstract reasoning, she fell further and further behind.

The DSM-5 criteria for SLD with impairment in mathematics include difficulties in number sense, memorization of arithmetic facts, accurate or fluent calculation, and/or accurate math reasoning. *Dyscalculia* is an alternative term sometimes used to describe this pattern of math difficulties. Many skills are involved in arithmetic: recognizing numbers and symbols, memorizing facts (the multiplication table), aligning numbers, and understanding abstract concepts such as place value and fractions. Any or all may be difficult for children with a mathematics disorder (Vukovic & Siegel, 2010). Children and adults with this disorder may have difficulty not only in math, but also in comprehending abstract concepts or in visual–spatial ability. Examples of calculation errors typical of children with a mathematics disorder are shown in ● Figure 7.3, an example that points out errors that suggest spatial difficulties and directional confusion.




● **FIGURE 7.3** | Errors in math computation by a 10-year-old girl with a mathematics disorder.

From "Learning Disabilities" by H.G. Taylor, 1988, p. 422. In E.J. Mash and L.G. Terdal (Eds.) "Behavioral Assessment of Childhood Disorders", 2nd ed.

Children with an SLD with impairment in mathematics typically have core deficits in arithmetic calculation and/or mathematics reasoning abilities, which include naming amounts or numbers; enumerating, comparing, and manipulating objects; reading and writing mathematical symbols; understanding concepts and performing calculations mentally; and performing computational operations (Andersson, 2010; Lyon et al., 2006). These deficits imply that the neuropsychological processes underlying mathematical reasoning and calculation are underdeveloped or impaired.

### *Prevalence and Course*

Estimates of the prevalence of SLD across all three domains (reading, writing, and math) range from 5% to 15% among school-aged children (APA, 2013). Reasons for this large range focus on the notion that SLD in reading—the most common form—may be part of a continuum of reading abilities rather than a discrete, all-or-none phenomenon. Children with reading disorders are essentially those who fall at the lower end of the reading continuum (Snowling, 2008). This consideration of the range of ability is useful and important because, clearly, there are strong readers and weak readers, and no definitive cutoff point easily distinguishes the two. Estimates of the prevalence of SLD with impairment in mathematics or written expression

are unclear due to the overlap among all three subtypes, although consensus is that they occur at a much lower rate than do reading difficulties (Landerl & Moll, 2010).

SLD impairments are considered lifelong, although the course varies based on severity and available supports. Recognition of SLD typically emerges during elementary school years, when a student falls significantly behind classmates in one or more of these subjects, though parents often note problems in language delays or counting in early childhood. Parents and teachers may notice specific delays in early skill development, or notice behavioral signs of the child's struggles, such as his or her unwillingness to learn to read, write, or work with numbers. In elementary school, the child shows marked difficulty in learning letter–sound correspondence and may commit reading errors by connecting sounds and letters (e.g., "big" for "got") and have difficulty sequencing numbers and letters (APA, 2013).

By the middle grades children with SLD may show poor reading comprehension and poor spelling and written work. They may be able to read and pronounce the first part of a word correctly but then guess the rest of the word. As they struggle with these difficulties throughout elementary school, being fearful of or refusing to read aloud is common. By adolescence through to adulthood, these patterns often shift from basic coding difficulties to marked problems in reading comprehension and written expression, including poor spelling and poor mathematical problem solving. Over time, teens and adults learn to manage these difficulties to the best of their ability, but may avoid situations that require reading, writing, or numerical ability. Thus, over the life span SLD is associated with many functional consequences, such as lower academic achievement, higher school dropout rates, poor overall mental health and well-being, and lower employment and income (APA, 2013).

### Cultural, Class, and Gender Variations

Social and cultural factors are less relevant to SLD than other types of cognitive and behavioral problems; in fact, the diagnostic criteria state that they cannot be attributed to these factors. Nevertheless, some cultural and ethnic factors may affect how children with SLD are identified and treated (Johansson, 2006).

Many childhood disorders reflect an interaction between the child's inherent abilities and resources and the opportunities that exist in the child's local environment, as emphasized throughout this text. In the case of learning to read, some teaching approaches do not explicitly emphasize specific sound–symbol relationships that are inherent in the dialect of children from diverse ethnic backgrounds. For example, an interesting study by Wood et al. (1991) illustrated the point that deficits in phonological awareness occur more frequently among populations that use nonstandard English. They followed a random sample of 485 Caucasian (55%) and African American (45%) children from first grade through third grade, and they found that although African American youngsters read at the same grade level as Caucasian children at the beginning of the first grade, they show marked declines in reading by the third grade and severe declines by the fifth grade. These findings suggest that greater attention to differences in dialect can lead to better learning opportunities.

Whereas attention to cultural and ethnic issues pertaining to SLD is a recent addition to research, sex differences have a long and contentious history. Boys are more often referred for learning difficulties than girls, perhaps because boys are more likely to show associated behavior problems such as aggression or inattention. Girls with learning problems often are quiet and withdrawn rather than loud and attention seeking, and they may be overlooked unless educators and parents are well informed. Nonetheless, when male–female ratios of SLD are derived from epidemiological estimates rather than from referrals, the ratio of boys to girls falls between 2:1 and 3:1 (APA, 2013).

### Psychological and Social Adjustment

For many years, a diagnosis of learning disability or disorder required a discrepancy between IQ and performance, which hampered early identification because the assessment often was not done until the child had attempted and failed at reading, usually by the third grade. By that time, the child's achievement would be low enough to warrant a diagnosis, but the child had failed in reading for 2 to 3 years and may have developed other behavioral and emotional problems as a result.

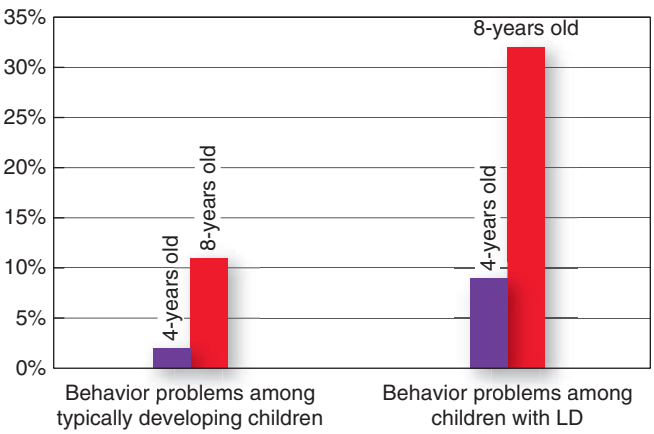
Today's recognition of SLD as an early neurodevelopmental disorder is improving detection of children with difficulties, but they still face significant obstacles in their peer adjustment and academic progress. Children with SLD often do not know how or why they are different, but they do know how it feels to be unable to keep up with others in the classroom. Hearing themselves described as "slow," "different," or "behind," they may identify more with their disabilities rather than with their strengths. These daily experiences may cause some children to act out by either withdrawing or becoming angry and noncompliant. Like James, they may stop trying to learn. Like Francine, they may become isolated and limit their participation in activities that their peers enjoy.

Students with SLD with reading impairment feel less supported by their parents, teachers, and peers than

do normal readers, and they are more likely to express poor academic or scholastic self-concepts (Heim & Benasich, 2006). Perhaps as a result of the interaction of their disorder and their environment, children and adolescents with SLD are more likely than their peers to show internalizing problems such as anxiety (Nelson & Harwood, 2011) and mood disorders (Maughan et al., 2003), as well as externalizing behaviors such as ADHD (Goldston et al., 2007). The range and types of problems are generally similar for both younger and older age groups. Accordingly, issues pertaining to both younger and older children and adolescents with SLD are considered jointly unless particular developmental differences warrant attention. Many of these issues are common to all domains of SLD unless otherwise noted.

The connection between SLD and behavioral or emotional disorders has generated considerable interest but only cautious conclusions. Common sense suggests that children with SLD encounter considerable challenges that are likely to take a toll on self-esteem and, in time, their social relationships. However, children's self-concepts in sports and appearance are usually less affected (Lyon et al., 2006).

Parents and teachers describe children with SLD as being more difficult to manage than typical children, beginning at an early age. Although overall reports of behavior problems increase considerably for all children between early and middle childhood, behavior problems among children with SLD are about three times higher than typically developing children by 8 years of age (i.e., 32% vs. 9%; Benasich, Curtiss, & Tallal, 1993) (see ● Figure 7.4). Most of these problems are not specific to SLD but cover a broad range of problems that overlap with features of conduct disorder (CD), oppositional defiant disorder (ODD), and attention-deficit/hyperactivity disorder (ADHD) across all ages (APA, 2013).




● **FIGURE 7.4** | Percentage of clinically significant behavior problems among children with and without learning disorders, at 4 years and 8 years of age.

Data from Benasich et al., 1993

These co-occurring problems are often interpreted as individual reactions and coping styles in response to failure, frustration, and, in some instances, punishment and negative attention. However, in terms of development, it is hard to say which comes first: Behavior problems may precede, follow, or co-occur with learning problems (Hinshaw, 1992). Whereas many of these behavioral and emotional problems gradually decrease from childhood to adolescence, adolescents with SLD continue to face challenges in their social relationships (St Clair et al., 2011).

Based on a review of over 150 studies, Kavale and Forness (1996) found that about three of every four students with SLD have significant deficits in social skills. As a group, they are more isolated and less popular among peers than other children, and they tend to make negative impressions on others (Durkin & Conti-Ramsden, 2010). Like Francine, who was described by her mother as "humorless and in a bit of a fog," most children with SLD have difficulty grasping the nuances of social interaction and may not know how to greet others, make friends, or join in playground games. Subtle cues of social interaction may be missed or ignored. These children may not always interpret correctly or respond appropriately to the frequent nonverbal—but very expressive—communication of other children, such as rolling the eyes to show dislike or disinterest. When children with SLD misunderstand the situation and act inappropriately, other children turn away.

A child with SLD also can be an emotional burden for family members. Parents may experience a wide range of emotions, including denial, guilt, blame, frustration, anger, and despair. Brothers and sisters often feel annoyed, embarrassed, or jealous of the attention their sibling receives. Because behavioral problems are usually so disruptive, a child's distress and emotional needs may easily be overlooked.

### *Adult Outcomes*

Unfortunately, the social and emotional difficulties connected to communication and learning disorders may continue into adulthood, largely because of inadequate recognition and services (Johnson et al., 2010). Adults may find ways to disguise their problems, such as watching television news rather than reading newspapers. On the other hand, many excel in nonacademic subjects such as art, music, dance, or athletics. Still others may become outstanding architects and engineers,

or they may have extraordinary interpersonal skills (Lyon et al., 2006). Each child and adolescent has many strengths that can be developed to compensate for his or her known deficits. Thus, despite their earlier risk for academic failure and psychosocial problems, many adults with SLD lead successful and productive lives (Lovett & Lewandowski, in press).

Men with SLD with impairments in reading do not differ from their peers regarding feelings of global self-worth; symptoms of depression; feelings of competency and satisfaction with jobs, marriages, and other relationships; or frequency of antisocial behavior (Boetsch, Green, & Pennington, 1996). However, men still perceive lower levels of social support from parents and relatives—the only people still in their lives who knew of their problems as children—which confirms the indelible impressions left by early experiences.

One adult describes his own way of compensating for learning problems:

> I faked my way through school because I was very bright. I resent most that no one picked up my weaknesses. Essentially I judge myself on my failures. . . . [I] have always had low self-esteem. . . . A blow to my self-esteem when I was in school was that I could not write a poem or a story. . . . I could not write with a pen or pencil. The computer has changed my life. I do everything on my computer. It acts as my memory. I use it to structure my life and for all of my writing since my handwriting and written expression has always been so poor. (Polloway, Schewel, & Patton, 1992, p. 521)

Whereas the long-term outlook for men with SLD is generally positive, the troublesome issue of sexism arises when considering how adult women with SLD fare over time. As a group, women with SLD have more adjustment problems than men as they leave school and face the demands of adult life. Similar to other adults with disabilities, they also face greater risk of sexual assault and related forms of abuse (Brownlie et al., 2007). Problems and breakdowns in relationships are common, which may reflect the lack of opportunity available to these women to achieve in areas that capitalize on their strengths.

Reading problems often cause poorly qualified graduates to take relatively undemanding and unrewarding jobs. Women who lack competitive skills and strong career options because of school failure tend to get involved at an early age in intimate relationships that are generally unsupportive (Fairchild, 2002). Young men, in contrast, have more wide-ranging options once they leave school, which facilitates more positive social functioning in adulthood. Thus, if they are able to select their own environments in adulthood (and women have more obstacles in this regard than men), both men and women with SLD can build on their existing strengths, skills, and talents (Hatch, Harvey, & Maughan, 2010).

It is safe to say that even though aspects of SLD may remain, people who are given proper educational experiences have a remarkable ability to learn throughout their life spans (Gregg, 2011). A Closer Look 7.1 describes some of these important opportunities that increase resilience. Adults can learn to read, although it is difficult because brain development slows down after puberty. Current gains in knowledge of the causes and early signs of SLD are likely to have a positive impact on early recognition and proper instruction. Nonintrusive electrophysiological measurements of brain reactivity may permit an early diagnosis based on underlying deficits in phonological processing rather than on performance alone. Thus, early identification and intervention may be the key to preventing the long-term consequences of these disorders (S. E. Shaywitz & Shaywitz, 2013).

A CLOSER **LOOK 7.1**

### Factors That Increase Resilience and Adaptation

Several personal characteristics and circumstances aid those with learning disorders in their successful adaptation from childhood, through adolescence, to young adulthood. As part of a longitudinal study of all children born in 1955 on the island of Kauai, Hawaii, E. E. Werner (1993) followed 22 children with learning disabilities and 22 matched controls. She found that most children with learning disabilities adapted successfully to adult life. Those who showed the greatest resilience and flexibility over time had: (1) a basic temperament that elicited positive responses from others; (2) a well-developed sense of efficacy, preparedness, and self-esteem that guided their lives; (3) competent caregivers and supportive adults; and (4) opportunities for a second chance if they made mistakes or got into trouble with the law. Although some of these characteristics are present from birth (e.g., temperament), many of the other supportive factors can be increased through the efforts of family members, schools, and communities. (Based on authors' case material.)

## *Causes*

Most learning disorders do not stem from problems in a single area of the brain, but from difficulties in bringing information from various brain regions together so that information can be integrated and understood

(Damasio et al., 2004; Meyer & Damasio, 2009). Minute disturbances may underlie phonological processing deficits. Emerging evidence suggests that in many cases these subtle disturbances begin very early during development, perhaps prenatally (McGrath et al., 2007; Raschle, Chang, & Gaab, 2011).

Recent findings suggest two distinguishable types of children with reading disorders—children who are persistently poor readers and those who are accuracy-improved (i.e., they learn ways to compensate for their reading difficulties and improve over time) (S. E. Shaywitz, Mody, & Shaywitz, 2006). Persistently poor readers and accuracy-improved readers have comparable reading skills and socioeconomic status when they begin school, but by the time they are young adults, the accuracy-improved readers show better cognitive ability. The presence of compensatory factors, such as stronger cognitive ability, may allow the accuracy-improved individuals to minimize the consequences of their phonological defect over time (Ferrer et al., 2010). These compensatory factors may be genetically based, and thus the child's ability improves with maturity, whereas the persistently poor readers may face greater environmental challenges, often associated with poverty and inequality, that reduce reading opportunities.

### Genetic and Constitutional Factors

Children who lack some of the skills needed for reading, such as hearing the separate sounds of words, are more likely to have a parent with a related problem. Around the turn of the twentieth century this problem was studied largely by physicians, who considered reading disorders to be an inherited condition called *congenital word blindness* (W. P. Morgan, 1896). Today, estimates based on behavioral genetic studies indicate that heritability accounts for over 60% of the variance in reading disorders (V. M. Bishop, 2006; Plomin et al., 2010), although the exact mode of transmission remains undetermined.

Most attention paid to heritability is aimed at genetic transmission of critical brain processes underlying phonetic processing (Scerri & Schulte-Körne, 2010; Vellutino et al., 2004). Because a parent's learning disorder may take a slightly different form in the child—the father may have a writing disorder and his child an expressive language disorder—it seems unlikely that subtypes of specific learning disorders are inherited directly. More likely, what is inherited is a subtle brain dysfunction that, in turn, can lead to a learning disorder (Newbury et al., 2011; Richlan et al., 2011; Scerri & Schulte-Körne, 2010). For example, on chromosome 6 an area has been identified that predisposes children to reading disorders (Grigorenko, 2007). Genetic transmission provides a plausible explanation for the relative risk of SLD in reading or mathematics being 4 to 8 times and 5 to 10 times higher, respectively, in first-degree relatives of persons with SLD as compared to those without it (APA, 2013; Shalev, 2007). Keep in mind that environmental factors also play a role in moderating genetic influences on SLD outcomes (i.e., gene–environment interaction; Petrill, 2013).

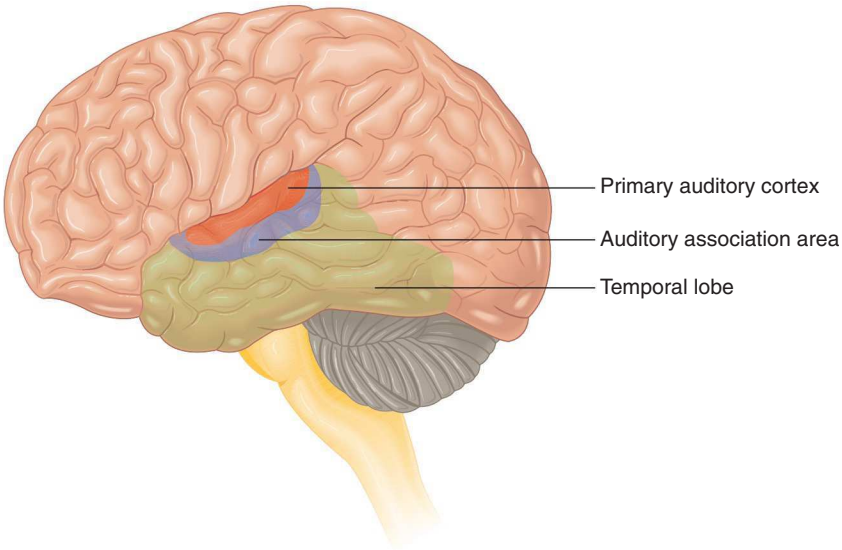
### Neurobiological Factors

Our understanding of learning disorders, particularly reading-based and language-based problems, took an important new direction in the mid-1980s with the discovery that the brains of people with these problems were characterized by cellular abnormalities in the left hemisphere, which contains important language centers (Galaburda et al., 1985). The fact that these cellular abnormalities could occur only during the fifth to seventh months of fetal development strengthened the view that learning disorders evolve from subtle brain deficits present at birth (Lyon et al., 2003). Initial autopsy findings were confirmed by sophisticated brain imaging technology that reveals the brain directly at work and makes it possible to detect subtle malfunctions that never could be seen before.

The suspected deficits, which likely are genetically based, involve specific discrimination tasks, such as detecting visual and auditory stimuli, as well as more pervasive visual–organizational deficits associated with reasoning and mathematical ability (Benassi et al., 2010; Pennington, 2006). A probable location of these deficits is a structure called the "planum temporale," a language-related area in both sides of the brain. In a normal brain, the left side of the planum temporale is usually larger than the right side; however, in the brain of an individual with a reading disorder, the two sides are equal (Tallal, 2003).

B. A. Shaywitz et al. (2002) found lower activation in numerous sites—primarily the left hemisphere of the brains of dyslexic children as compared with nonimpaired children—including the inferior frontal, parietotemporal, and occipitotemporal gyri. These three areas of the brain are responsible for understanding phonemes, analyzing words, and automatically detecting words, respectively. Once a word is learned, this three-part center recognizes it automatically, without first having to decipher it phonetically (S. E. Shaywitz & Shaywitz, 2013).

From a cognitive standpoint, these neurological findings suggest that children with learning disorders are distinctively disadvantaged as compared with average readers in terms of the processing underlying their short-term and working memory. Impairments in short-term memory affect the recall of phonemes




**How the brain processes speech**

1. The primary auditory cortex (shown in red), which is located on the top edge of each temporal lobe, receives electrical signals from receptors in the ears and transforms these signals into meaningless sound sensations, such as vowels and the consonants in ***ba*** and ***ga***.

2. The meaningless sound sensations are sent from the primary auditory cortex to another area in the temporal lobe, called the auditory association area.

3. The auditory association area (shown in blue), which is located directly below the primary auditory cortex, transforms basic sensory information, such as noises or sounds, into recognizable auditory information, such as words or music. Here, sounds are matched with existing patterns that have been previously formed and stored.

● **FIGURE 7.5** | How the brain processes speech.



and numbers; similarly, impairments in working memory affect how such information is processed and stored so that it can be rapidly accessed. Considerable support now exists concerning how memory deficits explain the performance difficulties of children with a learning disorder (Carretti et al., 2009; Maehler & Schuchardt, 2011; Swanson, Zheng, & Jerman, 2009).

We have stressed that most children with reading and writing disorders have difficulty distinguishing phonemes that occur rapidly in speech. But why is this so? Consider what is involved, as shown in ● Figure 7.5. The sound must be processed by various brain areas as it is carried by nerve impulses from the ear to the thalamus to the nerve cells within the auditory cortex, where it is matched to existing patterns, or phonic bins, that have been previously formed and stored.

Compare this process to listening to music. When you first hear a new song, do you recognize aspects that resemble other recordings by that group or another group? Can you distinguish the music of one group from another? As we listen, we tend to cluster sounds into various categories, acquiring our taste for music as we store more collections and melodies into memory. Each time we hear new music, we match it to what we already know and appreciate. Young people are particularly adept at assimilating new sounds, thereby broadening their tastes. In contrast, people who have already formed specific musical tastes tend to stick to what they know, rejecting unrecognizable sounds. This gap in music appreciation is analogous to the gap researchers describe in the phonic abilities of children with learning disorders—they lack certain auditory sites that allow certain sounds to be recognized, so their appreciation of certain words is compromised.

Each neuron in the language-processing areas of the brain has immense specificity. Some neurons fire when you silently name an object but not when you read the object's name out loud, and vice versa. Certain

neurons are activated when bilingual people speak one language, but not when they speak the other (Ojemann, 1991). Someone can have an expressive language problem despite full comprehension, because the same neurons that are active when a person hears a word are not active when that person speaks it.

In the visual system, different aspects of what you see, such as form, color, and motion, are routed to different regions of the visual cortex. When something moves in your visual field, the region of the cortex that responds to visual motion is activated. Eden et al. (1996) first discovered that adults with reading disorders show no activation in visual motion when asked to view randomly moving dots. A specific defect in the perception of visual motion may interfere with many different brain functions, and it has been noted among children with autism as well as those with learning disorders (Benassi et al., 2010; Skottun & Skoyles, 2008). To detect differences between consonant sounds—such as *b* and *t*—we must be able to distinguish between very rapid changes in sound frequency. A subtle neurological deficit in sensitivity could prohibit this distinction, which would then show up clinically as problems in reading and phonological processing (Raschle et al., 2011).

Thus, two major findings implicate specific biological underpinnings of reading disorders: (1) language difficulties for people with reading disorders are specifically associated with the neurological processing of phonology and storage of such information into memory; and (2) behavioral and physiological abnormalities are found in the processing of visual information. It is not surprising, therefore, that phonological and visual processing problems often coexist among people with reading disorders (Skottun & Skoyles, 2008).

Studies of the causes of SLD mostly involve children with reading disorders, but the findings apply to disorders in written expression and mathematics as well. Many—but not all—disabled writers show deficits in reading (Lyon et al., 2003), and some mathematical concepts require reading and writing as well as mathematics skills. Similar to how deficits in phonological awareness underlie SLD with reading impairment, certain cognitive deficits involving concepts of numbers appear to underlie SLD with impairment in mathematics (Geary, 2013). For instance, if you show small sets of dots, usually up to four dots per set, to typically developing children they can tell you instantly (i.e., without counting the items) how many items are in each set, a process known as *subitizing*. But children with SLD with impairments in mathematics appear to have trouble subitizing even three items, suggesting a deficit in their rapid visual processing of enumeration concepts (Ashkenazi, Mark-Zigdon, & Henik, 2013). Similarly, most children can quickly say which of two numbers is larger (e.g., "13" vs. "31"), but students with mathematics disorder are slower and less accurate than their peers at doing so (Rousselle & Noël, 2007).

Recall that Francine had well-developed word recognition and spelling abilities, but significantly worse mechanical–arithmetic skills. SLD with impairment in mathematics, and perhaps SLD with impairment in written expression as well, are associated with brain deficits that differ from those described for language-based learning disorders. These deficits are largely found in areas not related to verbal ability, which has led to the term nonverbal learning disability. **Nonverbal learning disability** (**NLD**) is associated with deficits related to right-hemisphere brain functioning, which are characteristic of children who perform considerably worse at math than reading. These deficits involve social/emotional skills, spatial orientation, problem solving, and the recognition of nonverbal cues such as body language (Hulme & Snowling, 2009). In addition to math deficiencies, NLD may be accompanied by neuropsychological problems such as poor coordination, poor judgment, and difficulties adapting to novel and complex situations (Lyon et al., 2003; Semrud-Clikeman et al., 2010). To date, the unique aspects of NLD remain unconfirmed. Critics argue that it may simply be a form of SLD with impairment in mathematics (Fine et al., 2013; Spreen, 2011).

### *Social and Psychological Factors*

Emotional and behavioral disturbances and other signs of poor adaptive ability often accompany SLD. This is no surprise, because children with one neurodevelopmental disorder (i.e., communication disorder, SLD, ADHD, or intellectual disability) are about 40% more likely to have another neurodevelopmental disorder, most likely because of shared etiological factors (Gooch et al., 2013). The overlap between SLD with impairment in reading and ADHD, for example, ranges from 30% to 70% depending on how ADHD is defined (Del'Homme et al., 2007; Fletcher, Shaywitz, & Shaywitz, 1999). Although this degree of overlap suggests that behavioral and learning problems have certain common aspects, they are still distinct and separate disorders (Lyon et al., 2006). SLD is commonly associated with deficits in phonological awareness, whereas ADHD has more variable effects on cognitive functioning, especially in areas of rote verbal learning and memory (Jakobson & Kikas, 2007). ADHD, moreover, is relatively unrelated to phonological awareness tasks. However, some children with SLD show symptoms similar to those of ADHD, including inattention, restlessness, and hyperactivity (Del'Homme et al., 2007).

### *Prevention and Treatment*

What can be expected of Francine, James, Tim, and Carlos during their school years and beyond? Proper planning and goal setting are the cornerstones of the helping strategies for home and for school. Specific learning disorders are not usually outgrown, but there is reason for optimism if educational planning and accommodations are ongoing (Beitchman & Brownlie, 2014).

Although SLD has strong biological underpinnings, intervention methods rely primarily on educational and psychosocial methods. Psychosocial treatments for James, Francine, Carlos, and Tim must be comprehensive and ongoing, with each new task broken down into manageable steps, including examples, practice, and ample feedback. Combined with proper teaching strategies, children and their families may benefit from counseling aimed at helping the children develop greater self-control and a more positive attitude toward their own abilities. Support groups for parents also can fill an important gap between the school and the home by providing information, practical suggestions, and mutual understanding.

Someday, breakthroughs in brain research may lead to new medical interventions, but at present no biological treatments exist for speech, language, and academic disabilities. In cases in which significant problems coexist in concentration and attention, some children respond favorably to stimulant medications that may temporarily improve attention, concentration, and the ability to control their impulsivity, albeit with little or no improvement in learning. Typically, the medication schedule ensures that the drug is active during peak school hours, when reading and math are taught.

Francine's problems in making friends were a major concern to everyone, and we believed that they were directly linked to her learning disability. A cognitive–behavioral intervention plan was developed in conjunction with her educational program. Because of Francine's strong verbal skills, we taught her to problem solve through role playing, and encouraged her mother to invite one child at a time for her to play with so that she could practice her skills. Francine had drifted into being a loner and seemed disinterested in looking after herself, so we also discussed ways to develop better self-care at home by giving her an allowance for completing household chores. We spent considerable time explaining the nature of her problems to her parents, and this guidance led to relief and understanding.

We saw the family once again one year later; although some of Francine's problems still existed, her social abilities had improved. She still had difficulties in developing friendships and tended to prefer being alone, but the problem had clearly lessened from the previous year. (Based on authors' case material.)

Consider the coordinated planning and effort that went into the treatment programs for Francine: Francine was able to get help because her problems were detected; recall, however, that by the time she was referred, she had already begun to fail at formal schooling. The first step in solving any problem is to realize that it exists. The nature of learning disorders makes this difficult for many children and parents. Although numerous signs of language-based learning disorders are present from early childhood, sophisticated means of assessing problems are not yet available before children are old enough to be formally tested.

**FRANCINE**

## Slowly but Surely Improving

To reduce Francine's difficulties with math and, especially, with peer relationships, we considered several factors. First, we decided that teaching should be primarily verbal, with an understanding that she would have the most difficulty in math and science. Her teachers favored allowing Francine to use a calculator and a computer to assist her in learning new concepts. An emphasis on physical education was also planned, to help her with her visual–motor coordination. Her math teacher agreed that using graph paper might help her visualize numerical relationships, which led to noticeable improvements in her schoolwork.

Phanie/Science Source

Treatment of specific learning disabilities usually begins with a careful assessment of a child's abilities.

Issues of identification are important because a brief window of opportunity may exist for successful treatment. If a problem is detected in early childhood—say, by kindergarten—then language-based deficits can often be remediated successfully. If the problem is not detected until age 8 or so, the rates of response to treatment are much lower (Hatcher et al., 2006). This is why prevention of reading difficulties is a hot topic: Training children in phonological awareness activities at an early age may prevent subsequent reading problems among children at risk (Duff & Clarke, 2011; Snowling & Hulme, 2012). These activities involve games of listening, rhyming, identifying sentences and words, and analyzing syllables and phonemes. For example, the child might analyze *sand* as *s-and* and then synthesize it into *sand,* or colored alphabet blocks might be used to break the word into separate phonetic sounds (*s-a-n-d*).

Knowledge of communication and learning disorders has played leapfrog with the philosophy and practice of classroom instruction during the past decade. Discoveries in neurosciences, as noted above, challenged some prevailing educational practices, leading to more systematic ways of assisting children with learning disorders, as explored in the following sections.

### The Inclusion Movement

Integrating children with special needs into the regular classroom began as the *inclusion movement* during the 1950s, based on studies showing that segregated classes for students with disabilities were ineffective and possibly harmful (Baldwin, 1958). Resource rooms and specially trained teachers replaced the special classes that had been in vogue, a change that had the further advantage of removing the need to label and categorize children. The Education for All Handicapped Children Act of 1975 in the United States (currently known as Individuals with Disabilities Education Improvement Act [IDEA], 2004) and the provincial Education Acts in Canada mandate that children with special needs must be afforded access to all educational services, regardless of their handicaps. Today, children with special educational needs in the United States, Canada, and many other countries are placed in regular classrooms whenever possible.

In 2002, the No Child Left Behind Act was signed into law in the United States. This act allowed for more intensified efforts by each state to improve the academic achievement of public school students considered at risk for school failure. Today, almost 14% (about 6.6 million) of school-age children in the United States from all walks of life receive some level of support through special education, and students with specific learning disabilities account for close to half of these students (National Center for Educational Statistics (2012).

### Response to Intervention Models

IDEA provides for the use of response to intervention (RTI) models to identify and assess children. RTI consists of tiered instruction, in which children who have difficulty learning to read using typical methods of instruction are provided with small-group, intensive instruction. Those who need additional intervention may receive one-on-one special education. This approach seeks to provide each child with the appropriate level of instruction required for his or her individual needs (National Institutes of Health, 2007).

Initiatives to allow children with special needs to receive services without being diagnosed or labeled as intellectually disabled, learning disabled, and so forth have become widely available and hold considerable promise. However, implementation and teacher training, as well as the question of whether such initiatives succeed in meeting the special needs of students, continue to be unresolved (Lovett & Lewandowski, in 2014).

### Instructional Methods

Although controversy remains over the practical aspects of including all children in regular classrooms, most educators today favor direct instruction for children with learning disorders. **Direct instruction** is a straightforward approach to teaching based on the premise that to improve a skill, the instructional activities must approximate those of the skill being taught (see example in A Closer Look 7.2) (Hammill et al., 2002). Direct instruction in word structure is necessary because of the child's phonological deficits. Direct instruction in reading emphasizes the specific learning of word structure and word reading until the skill is learned, without concern for the full context of the sentence or story. This method is based on the premise that a child's ability to decode and recognize

A CLOSER **LOOK 7.2**

**Steps in Direct Behavioral Instruction**

1. Review the child's existing abilities.
2. Develop a short statement of goals at the beginning of each lesson.
3. Present new concepts and material in small steps, each followed by student practice.
4. Provide clear and detailed instructions and explanations.
5. Provide considerable practice for all students.

6. Check student understanding of concepts continually, in response to teacher questions.
7. Provide explicit guidance for each student during initial practice.
8. Provide systematic feedback and corrections.
9. Provide explicit instruction and practice for exercises completed by students at their desks.

*Source:* From Treatment of Learning Disabilities by G. R. Lyons and L. Cutting, 1998. In E. J. Mash and L. C. Terdal (Eds.), Treatment of Childhood Disorders.

The following example illustrates how the steps in direct behavioral instruction are applied.

**Example: Direct Instruction Lesson**

A typical DI lesson includes explicit and carefully sequenced instruction provided by the teacher (model) along with frequent opportunities for students to practice their skills (independent practice) over time (review). For example, if the sound /m/ appeared for the first time, the teacher might say, "You're going to learn a new sound. My turn to say it. When I move under the letter, I'll say the sound. I'll keep on saying it as long as I touch under it. Get ready. mmm" (model). "My turn again. Get ready. mmm" (model). "Your turn. When I move under the letter, you say the sound. Keep on saying it as long as I touch under it. Get ready." (independent practice). "Again. Get ready." (independent practice). If an error occurs during instruction, the teacher would model the sound ("My turn. mmm"), use guided practice ("Say it with me. Get ready. mmm"), and have students practice independently ("Your turn. Get ready"). A "starting over" would be conducted based on this error; this might include starting over at the top of a column or row of sounds so that students get increased practice on the /m/ sound. The /m/ would appear throughout the lesson and in subsequent lessons to ensure skill mastery (firm responding) over time.

*Source*: Marchand-Martella, Martella, & Ausdemore (2005). An Overview of Direct Instruction.

words accurately and rapidly must be acquired before reading comprehension can occur (Hammill et al., 2002; Haager, Klinger, & Vaughn, 2007).

To prevent dyslexia, it is important to provide early interventions that teach both phonological and verbal abilities. Children must be able to learn the sounds of words to decode them, but they must also understand the meaning of a word to understand the message of the text (S. E. Shaywitz & Shaywitz, 2013). The techniques that have been demonstrated to work are practicing manipulating phonemes, building vocabulary, increasing comprehension, and improving fluency, which helps strengthen the brain's ability to link letters to sounds (Nation et al., 2007).

In brief, the components of effective reading instruction are the same whether the focus is prevention or intervention—phonemic awareness and phonemic decoding skills, fluency in word recognition, construction of meaning, vocabulary, spelling, and writing (Snowling & Hulme, 2012). Evidence-based evaluations show dramatic reductions in the incidence of reading failure when direct and explicit instruction in these components is provided by the classroom teacher (Duff & Clarke, 2011; Haager et al., 2007; Schuele & Boudreau, 2008). Empirical support for teaching phonics from an early age also is emerging from brain imaging studies. For example, instruction in phonemic awareness, phonics, and other reading skills produces more activation in the automatic recognition process, noted previously (see section on "Causes," above). After undergoing such training, brain scans of people who were once poor readers begin to resemble those of good readers (National Institutes of Health, 2007).

We now turn to some practical examples of how reading, writing, and math can be taught by applying well-established principles of learning. Behavioral and cognitive–behavioral strategies have been highly beneficial in remediating the problems of children and adolescents with communication and learning disorders (Lyon et al., 2006). In addition, new methods based on the use of technology offer some children additional ways to acquire basic and advanced academic skills.

### *Behavioral Strategies*

Many problems that children with communication and learning disorders have stem from the fact that the material is simply presented too fast for them (Tallal & Benasich, 2002). Thus, a strategy to provide children with a set of verbal rules that can be written out and reapplied may be more beneficial than one that relies on memory or on grasping the concept all at once. Tried-and-true behavioral principles of learning are well suited to this task of teaching systematically.

In addition to academic concepts, some of the associated problems with peers can be addressed in the same fashion, as we saw with Francine. A simple, gradual approach is more beneficial than an approach that tries to solve the problem all at once. Children also need help learning to generalize new information to different situations. An individualized, skills-based approach does not have to be boring or routine; in fact, speech and language therapists are skilled at providing a stimulating but structured environment for hearing and practicing language patterns. During an engaging activity with a younger child, the therapist may talk about toys and then encourage the child to use the same sounds or words. The child may watch the therapist make the sound, feel the vibration in the therapist's throat, and then practice making the sounds himself or herself in front of a mirror.

Behavioral methods often are used in conjunction with a complete program of direct instruction, which typically proceeds in a cumulative, highly structured manner (Wright & Jacobs, 2003), as shown in A Closer Look 7.2. Because this method places a strong emphasis on the behavior of the teacher in terms of explicit correction, reinforcement, and practice opportunities, it is sometimes referred to as "faultless instruction": Each concept should be so clearly presented that only one interpretation is possible. Each lesson is structured according to field-tested scripts. Teachers work with one small group of students at a time, and shoot questions at them at a rate as high as 10 to 12 per minute.

This highly structured, repetitive method is clearly effective. Students who receive direct behavioral instruction typically outperform students who receive standard classroom instruction by almost 1 standard deviation on various learning measures (Lyon et al., 2003).

### Cognitive–Behavioral Interventions

Cognitive–behavioral interventions are also highly suited for children with communication and learning disorders. Like behavioral methods, these procedures actively involve students in learning, particularly in monitoring their own thought processes. Considerable emphasis is placed on self-control by using strategies such as self-monitoring, self-assessment, self-recording, self-management of reinforcement, and so on (Alwell & Cobb, 2009; Cobb et al., 2006). Essentially, children are taught to ask themselves several questions as they progress, to make themselves more aware of the material. Try it yourself: "Why am I reading this? What's the main idea the authors are trying to get across? Where can I find the answer to this question? How does this follow from what I learned a minute ago?"

Carlos's treatment program shows how some of these procedures were applied to his particular writing problems:

**CARLOS**

**Plans**

In third grade, Carlos's treatment plan was to integrate a cognitive–behavioral approach into regular teaching methods. Rather than using one-to-one instruction, I discussed with his teacher ways of blending some behavioral methods into the classroom. For example, his strengths are in the areas of thinking and speaking, so I discussed using computers and tape recorders to help him learn the materials. He seemed to like these methods, and they helped him bypass some aspects of his writing disability. I discussed practice strategies for visual–motor integration, such as drawing and tracing and gradually made the task more complex. Because cursive writing is often easier for children than printing, I suggested that Carlos bypass learning to print. A continuous pattern of output is easier for Carlos to plan and produce than a discrete form of output, such as printing.

To help Carlos write a paper, I adopted a basic planning strategy from Graham, MacArthur, Schwatz, and Voth (1992), which helped him structure the tasks into related subproblems. The acronym PLANS helps him to remember to:

**P**ick goals (related to length, structure, and purpose of the paper)
**L**ist ways to meet goals
**A**nd
make **N**otes
**S**equence notes

This mnemonic was used in a three-step writing strategy to assist Carlos to: (1) do PLANS, (2) write and say more, and (3) evaluate whether he is successful in achieving his goals. (Based on authors' case material.)

### Computer-Assisted Learning

Studies have shown that a similar level of efficacy in phonetic ability can be achieved by teachers as by clinicians (Duff & Clarke, 2011), which has led to a growing number of computer and Internet training programs. One problem in reading instruction is maintaining a balance between the basic, but dull, word decoding and the complex, but engaging, text comprehension. Not all the issues have been resolved, but computer-assisted methods for spelling, reading, and math provide more academic engagement and achievement than traditional pencil-and-paper methods.

Computers have been used as simple instructional tools to deliver questions and answers since the 1970s. Since discovering phonological awareness and timing problems in the brain, researchers are now testing whether computers can remedy some basic auditory problems. Some children with communication and learning disorders are unable to process information that flashes by too quickly, such as the consonant sounds *ba* and *da*, and this deficit interferes with vital speech processes. Computer programs are able to slow down these grammatical sounds, allowing young children to process them more slowly and carefully (Loo et al., 2010; Gaab et al., 2007; Palmer, Enderby, & Hawley, 2007).

BSIP/UIG via Getty Images

New research raises cautious hope that computer games and exercises can help children with learning disabilities develop key mental skills.

Whether taught by computers, teachers, or both, studies of interventions for learning disorders indicate that successful approaches typically include explicit instruction in phonemic awareness and phonemic decoding. These interventions also provide students with practice reading text and comprehending what they read, with ample assistance and almost daily sessions (Torgesen et al., 2010).

In summary, treatment methods for communication and learning disorders are varied and beneficial. A Closer Look 7.3 reviews some of the basic elements of a successful beginning reading program, elements that apply to other disabilities as well. For children with reading disorders to learn how to read, they must receive a balanced intervention program composed of direct and explicit instruction in phonemic awareness, a systematic way to generalize this learning to the learning of sound–symbol relationships (phonics), and many opportunities to practice these coding skills by reading meaningful, interesting, and controlled texts. The sooner this intervention occurs in schools, the better (Haager et al., 2007).

## A CLOSER **LOOK 7.3**

### Critical Elements for a Successful Beginning Reading Program

1. *Provide direct instruction in language analysis*. Identify at-risk children early in their school careers—preferably in kindergarten—and teach phonological awareness skills directly.
2. *Provide direct teaching of the alphabetic code*. Code instruction should be structured and systematic, in a sequence that goes from simple to more complex. Teach the regularities of the English language before introducing the irregularities. Nothing should be left to guesswork—be as explicit as possible. Teach a child who is overly reliant on letter-by-letter decoding to process larger and larger chunks of words.
3. *Teach reading and spelling in coordination*. Children should learn to spell the words they are reading correctly.
4. *Provide intensive reading instruction*. Children may need 3 or more years of direct instruction in basic reading skills to ensure competency. As they progress, they should practice more and more reading that is contextualized. Reading materials should have controlled vocabularies that contain mostly words the children can decode. As children develop a core sight vocabulary, introduce only those irregular words that can be read with high accuracy. Guessing is counterproductive.
5. *Teach for automaticity*. Once basic decoding is mastered, children must be exposed to words often enough that they become automatically accessible. This usually requires a great deal of practice, which should be as pleasant and rewarding as possible.

*Source:* From Rebecca H. Felton, Effects of Instruction on the Decoding Skills of Children with Phonological-Processing Problems, Journal of Learning Disabilities, 26, 583–589

## Section Summary

### Specific Learning Disorder

- Specific learning disorder (SLD) includes problems in reading, mathematics, or writing ability, with reading disorders being the most common. Mathematics and writing disorders overlap considerably with reading disorders.
- Although SLD overlaps with behavioral disorders, they are distinct problems. Opportunities to develop and use particular strengths lead to more successful adult outcomes.
- SLD in reading may be caused by phonological problems that arise from physiological abnormalities in the processing of visual information in the brain. These deficits are believed to be largely inherited.
- Treatments for children with communication and learning disorders involve educational strategies that capitalize on existing strengths, and behavioral strategies involving direct instruction.
- Cognitive–behavioral techniques and computer-assisted instruction are also used successfully.

# Study Resources

## SECTION SUMMARIES



## KEY TERMS

# 8

# Attention-Deficit/Hyperactivity Disorder (ADHD)

*ADHD is not a problem with knowing what to do; it is a problem with doing what you know.*

—Russell A. Barkley (2006a)

## CHAPTER PREVIEW

## DESCRIPTION AND HISTORY

WE BEGIN OUR DISCUSSION of attention-deficit/hyperactivity disorder (ADHD) with a description of the primary symptoms and behaviors of children with this disorder. We then consider the different views of ADHD that have been presented since it was first described as a disorder more than 200 years ago.

### JOHN

### Inattentive, Hyperactive, Impulsive

John is a 7-year-old whose mother is desperate for help. "He walked at 10 months and has kept me running ever since. As a child he was always bouncing around the house and crashing into things. He's in constant motion, impulsive, and never listens. When I ask him to put his shirt in the hamper, I find him playing, his shirt still on the floor. John has no routines and seldom sleeps. Discipline doesn't work, nor do the techniques that work for my other boys. He's oblivious to his behavior. He never finishes anything, and except for sitting down to play a video game, rarely watches TV except on the run."

John's teacher says his main problems in school are staying on task and keeping track of what's happening. "He blurts things out in class and is constantly fidgeting or out of his chair," she says. Although John can complete his assignments, he forgets to bring home the book he needs to do his homework. When he does complete his homework, he forgets to put it in his backpack or to hand it in. John has great difficulty waiting his turn or following rules with other children. Other kids think he's weird and don't want to play with him. John's parents are demoralized and don't know what to do.

From *The Hyperactive Child Book*, by Patricia Kennedy, Leif Terdal, and Lydia Fusetti, pp. 8–9. New York: St. Martin's Press.

Fidgety Phil, 1845, Blackie & Son, Ltd., Glasgow

Jose Azel/Aurora/Getty Images

Fidgety Phil, 1845, and Dusty N., 1994: Mealtimes are an especially trying time for children with ADHD and their parents

## Description

**Attention-deficit/hyperactivity disorder (ADHD)** describes children who, like John, display persistent age-inappropriate symptoms of inattention, hyperactivity, and impulsivity that are sufficient to cause impairment in major life activities (APA, 2013).

The term *ADHD* may be new, but children who display overactive and unrestrained behaviors have been around for some time. In 1845, Heinrich Hoffmann, a German neurologist, wrote in a child's storybook one of the first known accounts of hyperactivity. His humorous poem described the mealtime antics of a child aptly named "Fidgety Phil," who "won't sit still; / He wriggles, / And jiggles," and "swings backwards and forwards, / And tilts up his chair." When his chair falls, Philip screams and grabs the tablecloth, and "Down upon the ground they fall, / Glasses, plates, knives, forks, and all" (Hoffmann, 1845).

More recently, a compelling article about ADHD titled "Life in Overdrive" described the behavior of 7-year old Dusty N.:

> Dusty awoke at 5:00 one recent morning in his Chicago home. Every muscle in his 50-pound body flew in furious motion as he headed downstairs for breakfast. After pulling a box of cereal from the cupboard, Dusty started grabbing cereal with his hands and kicking the box, scattering the cereal across the room. Next he began peeling the decorative paper covering off the TV table. Then he started stomping the spilled cereal to bits. After dismantling the plastic dustpan he had gotten to clean up the cereal, he moved on to his next project: grabbing three rolls

of toilet paper from the bathroom and unraveling them around the house. (Adapted from *Time*, July 18, 1994, p. 43)

Although the accounts of Phil and Dusty N. are separated by almost 150 years, the mealtime behaviors of both boys typify the primary symptoms of ADHD. The boys are **inattentive**, not focusing on mealtime demands and behaving carelessly; **hyperactive**, constantly in motion; and **impulsive**, acting without thinking.

ADHD has no distinct physical symptoms that can be seen in an x-ray or a lab test. It can only be identified by characteristic behaviors that vary considerably from child to child. As we shall discuss, ADHD has become a blanket term used to describe several different patterns of behavior that likely have different causes.

The behavior of children with ADHD is puzzling and full of contradictions. Rash and disorganized behaviors are a constant source of stress for the child and for parents, siblings, teachers, and classmates. Why can't he sit still? Why can't she ever get anything done? Why does he make so many careless mistakes? Nothing seems physically wrong with the child, and at certain times or in some situations the child with ADHD seems fine. Such inconsistencies may cause others to think the child could do better if only she tried harder or if her parents or teachers would set firmer limits. However, increased effort and stricter rules usually don't help, because most children with ADHD are already trying hard. They want to do well but are constantly thwarted by their limited self-control. As a result, they experience the hurt, confusion, and sadness of being blamed for not paying attention or being called names like "space cadet." They may be scolded, put down, or even spanked for failing to complete homework or chores. Unfortunately, they may not know why things went wrong or how they might have done things differently.

Feelings of frustration, being different, not fitting in, and hopelessness may overwhelm a child with ADHD (Young et al., 2008). For example, David says: "I got no friends cause I don't play good and when they call me Dope Freak and David Dopey I cry, I just can't help it" (Ross & Ross, 1982). Such comments leave little doubt that ADHD can severely disrupt an individual's life, consume vast amounts of energy, produce emotional pain, damage self-esteem, and seriously disrupt relationships. In addition to the individual's personal suffering and exposure to stigmatizing attitudes by others (Lebowitz, 2013), the societal costs of ADHD in youth are also high, with an estimated cost of $38 billion to $72 billion a year in the United States, the highest costs related to health care and education. Estimated costs for adults with ADHD, which take productivity and income losses into account, are nearly two to three times higher than for young people. These estimates, along with the additional billions in spillover costs borne by family members of individuals with ADHD, indicate that the economic impact of ADHD across the lifespan for individuals with ADHD in the United States is considerable (Doshi et al., 2012).

## History

The symptoms of ADHD were first described in a 1775 medical textbook by the German physician, Melchior Adam Weikard (Barkley & Peters, 2012). Since then there have been numerous explanations for the troublesome behaviors of ADHD (Barkley, 2014e). In 1798, a Scottish-born physician, Sir Alexander Crichton described a syndrome similar to ADHD that included early onset, restlessness, inattention, and poor school performance. These individuals described themselves as having "the fidgets," and displayed a severe problem attending no matter how hard they tried (Palmer & Finger, 2001). Symptoms of overactivity and inattention were described as a disorder in 1902 by the English physician George Still (what a coincidence!), who believed that the symptoms arose out of poor "inhibitory volition" and "defective moral control" (see ● Figure 8.1). In the early 1900s, the onset of widespread compulsory education demanded self-controlled behavior in a group setting, which further focused attention on children with the symptoms of ADHD.

Another view of ADHD arose from the worldwide influenza epidemic from 1917 to 1926. A number of children who had developed encephalitis (brain inflammation) and survived experienced multiple behavior problems, including irritability, impaired attention, and hyperactivity. These children and others who had suffered birth trauma, head injury, or exposure to toxins displayed behavior problems that were labeled *brain-injured child syndrome*, which was associated with intellectual disability. In the 1940s and 1950s, this label was then erroneously applied to children displaying similar behaviors, but with no evidence of brain damage or intellectual disability, and led to the terms *minimal brain damage* and *minimal brain dysfunction (MBD)* (Strauss & Lehtinen, 1947). These terms provided a convenient way to attribute behavior problems to a physical cause. Although certain head injuries can explain some cases of ADHD, the brain damage theory was eventually rejected because it did not explain the majority of cases (Rie, 1980).

In the late 1950s, ADHD was referred to as *hyperkinesis*, which was attributed to poor filtering of stimuli

**THE LANCET, APRIL 19, 1902.**

## The Goulstonian Lectures

ON

## SOME ABNORMAL PSYCHICAL CONDITIONS IN CHILDREN.

*Delivered before the Royal College of Physicians of London on March 4th, 6th, and 11th, 1902,*

BY GEORGE F. STILL, M.A., M.D. CANTAB., F.R.C.P. LOND.,

ASSISTANT PHYSICIAN FOR DISEASES OF CHILDREN, KING'S COLLEGE HOSPITAL; ASSISTANT PHYSICIAN TO THE HOSPITAL FOR SICK CHILDREN, GREAT ORMOND-STREET.

### LECTURE II.

*Delivered on March 6th.*

MR. PRESIDENT AND GENTLEMEN.—In my first lecture I drew your attention to some points in the psychology and development of moral control in the normal child and then considered the occurrence of defective moral control in association with general impairment of intellect; before going further it may be well to review briefly the points which have been raised. Moral control, we saw, is dependent upon three psychical factors, a cognitive relation to environment, moral consciousness, and volition, which in this connexion might be regarded as inhibitory volition. Moral control, therefore, is not present at birth, but under normal psychical conditions is gradually developed as the child grows older. The variation in the degree of moral control which is shown by different children at the same age and under apparently similar conditions of training and environment suggested that the innate capacity for the development of such control might also vary in different individuals.

Courtesy of *The Lancet*, April 19, 1902.

● **FIGURE 8.1** | English physician George Still was one of the first to describe the symptoms of ADHD.

entering the brain (Laufer, Denhoff, & Solomons, 1957). This view led to the definition of the *hyperactive child syndrome*, in which motor overactivity was considered the main feature of ADHD (Chess, 1960). However, it was soon realized that hyperactivity was not the only problem; there was also the child's failure to regulate motor activity in relation to situational demands.

In the 1970s, it was argued that in addition to hyperactivity, deficits in attention and impulse control were also primary symptoms of ADHD (Douglas, 1972). This view was widely accepted and has had a lasting impact on the criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) for defining ADHD. In the 1980s, interest in children with ADHD increased dramatically, and the sharp rise in the use of stimulants generated controversy that continues to this day (Mayes & Rafalovich, 2007).

More recently, in addition to inattention and hyperactivity–impulsivity, the problems of poor self-regulation, difficulty in inhibiting behavior, and reward and motivational deficits have been emphasized as central impairments of the disorder (Nigg, Hinshaw, & Huang-Pollack, 2006). Increasingly, "multipathway models" have emerged that include both attention-related and motivation-related theories. These models propose different pathways to ADHD with different neural substrates, meaning that different children with ADHD may have different reasons for their behavior (Nigg & Barkley, 2014). Although there is growing agreement about the nature of ADHD, views continue to evolve as a result of new findings and discoveries. As you will learn, despite the label for this disorder, the main difficulties in ADHD are far more complex than simply a deficit in attention (Nigg & Barkley, 2014).

## Section Summary

### Description and History

- Attention-deficit/hyperactivity disorder (ADHD) is manifested in children who display persistent age-inappropriate symptoms of inattention, hyperactivity, and impulsivity that cause impairment in major life activities.
- ADHD can only be identified by characteristic patterns of behavior, which vary quite a bit from child to child.
- The behavior of children with ADHD is a constant source of stress and frustration for the child and for parents, siblings, teachers, and classmates; it also has high costs to society.
- The disorder that we now call ADHD has had many different names, primary symptoms, and presumed causes, and views of the disorder are still evolving.

## CORE CHARACTERISTICS

ADHD is included in DSM-5 as a *neurodevelopmental disorder* because it has an early onset and persistent course, is associated with lasting alterations in neural development, and is often accompanied by subtle delays and problems in language, motor, and social development that overlap with other neurodevelopmental disorders such as autism spectrum disorder (ASD) and specific learning disorder (APA, 2013). Experts developed the DSM-5 criteria for ADHD after reviewing research, re-analyzing data, conducting field trials with children throughout North America, and receiving several rounds of public feedback (APA, 2013). Table 8.1

**TABLE 8.1** | Diagnostic Criteria for Attention-Deficit/Hyperactivity Disorder

**DSM-5**

**(A)** A persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development, as characterized by (1) and/or (2):

(1) **Inattention:** Six (or more) of the following symptoms have persisted for at least 6 months to a degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities:

**Note:** The symptoms are not solely the manifestation of oppositional behavior, defiance, hostility, or failure to understand tasks or instructions. For older adolescents and adults (age 17 and older), at least five symptoms are required.

**(a)** Often fails to give close attention to details or makes careless mistakes in schoolwork, at work, or during other activities (e.g., overlooks of misses details, work is inaccurate).

**(b)** Often has difficulty sustaining attention in tasks or play activities (e.g., has difficulty remaining focused during lectures, conversations, or lengthy reading).

**(c)** Often does not seem to listen when spoken to directly (e.g., mind seems elsewhere, even in the absence of any obvious distraction).

**(d)** Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (e.g., starts tasks but quickly loses focus and is easily sidetracked).

**(e)** Often has difficulty organizing tasks and activities (e.g., difficulty managing sequential tasks: difficulty keeping materials and belongings in order; messy, disorganized work; has poor time management; fails to meet deadlines).

**(f)** Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (e.g., schoolwork or homework; for older adolescents and adults, preparing reports, completing forms, reviewing lengthy papers).

**(g)** Often loses things necessary for tasks or activities (e.g., school materials, pencils, books, tools, wallets, keys, paperwork, eyeglasses, mobile telephones).

**(h)** Is often easily distracted by extraneous stimuli (for older adolescents and adults, may include unrelated thoughts).

**(i)** Is often forgetful in daily activities (e.g., doing chores, running errands; for older adolescents and adults, returning calls, paying bills, keeping appointments).

(2) **Hyperactivity and Impulsivity:** Six (or more) of the following symptoms have persisted for at least 6 months to a degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities:

**Note:** The symptoms are not solely a manifestation of oppositional behavior, defiance, hostility, or a failure to understand tasks or instructions. For older adolescents and adults (age 17 or older), at least five symptoms are required.

**(a)** Often fidgets with or taps hands or feet or squirms in seat.

**(b)** Often leaves seat in situations when remaining seated is expected (e.g., leaves his or her place in the classroom, in the office or other workplace, or in other situations that require remaining in place).

**(c)** Often runs about or climbs in situations where it is inappropriate

**Note:** In adolescents or adults, may be limited to feeling restless.

**(d)** Often unable to play or engage in leisure activities quietly.

**(e)** Is often "on the go," acting as if "driven by a motor" (e.g., is unable to be or is uncomfortable being still for extended time, as in restaurants, meetings; may be seen by others as being restless or difficult to keep up with).

**(f)** Often talks excessively.

**(g)** Often blurts out answers before a question has been completed (e.g., completes people's sentences; cannot wait for a turn in conversation).

**(h)** Often has difficulty waiting his or her turn (e.g., while waiting in line).

**(i)** Often interrupts or intrudes on others (e.g., butts into conversations, games or activities; may start using other people's things without asking or receiving permission; for adolescents and adults, may intrude into or take over what others are doing).

**(B)** Several inattentive or hyperactive–impulsive symptoms were present before age 12 years.

**(C)** Several inattentive or hyperactive-impulse symptoms are present in two or more settings (e.g., at home, school, or work; with friends or relatives; in other activities).

**(D)** There must be clear evidence that the symptoms interfere with, or reduce the quality of, social academic, or occupational functioning.

**(E)** The symptoms do not occur exclusively during the course of schizophrenia or another psychotic disorder and are not better explained by another mental disorder (e.g., mood disorder, anxiety disorder, dissociative disorder, personality disorder, substance intoxication or withdrawal).

(*continues*)

**TABLE 8.1** | **Diagnostic Criteria for Attention-Deficit/Hyperactivity Disorder** (*continued*)

*Specify* whether:

**Combined presentation:** If both Criterion A1 (inattention) and Criterion A2 (hyperactivity-impulsivity) are met for the past 6 months.

**Predominantly inattentive presentation:** If Criterion A1 (inattention) is met but Criterion A2 (hyperactivity–impulsivity) is not met for the past 6 months.

**Predominantly hyperactive–impulsive presentation:** if Criterion A2 (hyperactivity–impulsivity) is met but Criterion A1 (inattention) is not met for the past 6 months.

*Specify* if:

**In partial remission:** When full criteria were previously met, fewer than the full criteria have been met for the past 6 months, and the symptoms still result in impairment in social, academic, or occupational functioning.

*Specify* current severity:

**Mild:** Few, if any, symptoms in excess of those required to make the diagnosis are present, and symptoms result in no more than minor impairments in social or occupational functioning.

**Moderate:** Symptoms or functional impairment between "mild" and "severe" are present.

**Severe:** Many symptoms in excess of those required to make the diagnosis, or several symptoms that are particularly severe, are present, or the symptoms result in marked impairment in social or occupational functioning.

*Source:* Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition. American Psychiatric Association.

shows the two lists of key symptoms that were identified for defining ADHD and distinguishing it from related problems. The first list includes symptoms of *inattention*; the second list includes symptoms of *hyperactivity–impulsivity*.

Quantitative studies support a model of ADHD consisting of a unitary ADHD component with two separable specific dimensions of inattention and hyperactivity–impulsivity (Burns et al., 2013). These two dimensions are well documented in research with thousands of individuals across various age, ethnic, and cultural groups throughout the world (Toplak et al., 2012). The two dimensions are highly correlated but they do predict different behavioral and cognitive impairments and likely have different neural correlates (Kuntsi et al., 2013; Willcutt et al., 2012). For example, symptoms of inattention tend to predict academic problems and peer neglect, whereas those of hyperactivity–impulsivity tend to predict aggressive behavior and peer rejection, among other problems.

To define the two core dimensions of ADHD as inattention and hyperactivity–impulsivity oversimplifies the disorder. First, each dimension includes many distinct processes that have been defined and measured in various ways. Second, although we discuss attention and impulse control separately, the two are closely connected developmentally—attention helps the child regulate behavior, emotions, and impulses (Nigg et al., 2006).

## Inattention

### LISA

### Just Can't Focus

At age 17, Lisa struggles to pay attention and act appropriately. But this has always been hard for her. She still gets embarrassed thinking about the time that her parents took her to a restaurant to celebrate her 10th birthday. She was so distracted by the waitress's bright red hair that her father had to call her name three times before she remembered to order. Then, before she could stop herself, she blurted, "Your hair dye looks awful!"

In school, Lisa was quiet and cooperative but often seemed to be daydreaming. She was smart, yet couldn't improve her grades no matter how hard she tried.

Several times she failed exams. She knew the answers, but couldn't keep her mind on the test. Her parents responded to her low grades by taking away privileges and scolding her, "You're just lazy, Lisa. You could get better grades if you only tried."

Lisa found it agonizing to do homework. Often, she forgot to plan ahead by writing down the assignment or bringing home the right books. And when trying to work, every few minutes she found her mind drifting to something else. As a result, she rarely finished and her work was full of errors. One day, after Lisa had failed yet another exam, her teacher found her sobbing, "What's wrong with me?"

Adapted from National Institute of Mental Health [NIMH], 1994a.

**Inattention** refers to an inability to sustain attention or stick to tasks or play activities, to remember and follow through on instructions or rules, and to resist distractions. It also involves difficulties in planning and organization and in timeliness and problems in staying alert (Nigg & Barkley, 2014). Children who are inattentive find it difficult, during work or play, to focus on one task. While playing soccer, as the rest of the team heads downfield with the ball, the child with ADHD may get sidetracked by playing in a mud puddle. The child may attend automatically to enjoyable things, but have great difficulty focusing on less enjoyable tasks. Common complaints about inattention are that the child doesn't or won't listen, follow instructions, or finish chores or assignments. Since inattention can result from a failure in one or more cognitive processes that control attention (Petersen & Posner, 2012), it is not sufficient to say that a child has an attention deficit. The child could have a deficit in only one type or in more than one type.

**Attentional capacity** is the amount of information we can remember and attend to for a short time. When someone gives you directions or a phone number, how much information can you attend to and remember briefly? Children with ADHD do not have a deficit in their attentional capacity. They can remember the same amount of information for a short time as do other children (Taylor, 1995).

**Selective attention** is the ability to concentrate on relevant stimuli and ignore task-irrelevant stimuli in the environment. When you're studying for a test (relevant stimuli), how easily are you distracted by voices in another room?

**Distractibility** is a term commonly used to indicate a deficit in selective attention. Distractions can be disruptive to all children, including those with ADHD. However, children with ADHD are much more likely than others to be distracted by stimuli that are *highly salient and appealing* (Milich & Lorch, 1994).

**Sustained attention**, or *vigilance*, is the ability to maintain a persistent focus over time on unchallenging, uninteresting tasks or activities or when fatigued (Langner & Eickhoff, 2013). When you're tired and have to study for a test, can you still pay attention until you've reviewed all the required material? A primary attention deficit in ADHD seems to be sustained attention. When children with ADHD are assigned an uninteresting or repetitive task, their performance is poor as compared with that of other children. Although no one likes to work on uninteresting tasks, most of us will when we have to. Children with ADHD may not be able to persist at such tasks even when they want to. They work best on self-paced tasks that they themselves have chosen—playing a computer game or building a model airplane—and on tasks they find especially interesting that do not require sustained attention. Most tasks, though, require sustained attention for successful performance, and many tasks are not particularly interesting.

Deficits in sustained attention are one of the core features of ADHD. However, children with ADHD may show performance deficits from the very beginning of a task or response, not just a decline over time. This suggests that their attentional problems may also be in alerting and preparing for the task from the outset, and not only in sustaining attention during the task. **Alerting** refers to an initial reaction to a stimulus; it involves the ability to prepare for what is about to happen. It helps the child achieve and maintain an optimally alert attentional state. A child with an alerting deficit (such as the lack of alertness you may experience when you are very tired) may respond too quickly in situations requiring a slow and careful approach and too slowly in situations requiring a quick response. This pattern of responding is often seen in children with ADHD. Thus, one view is that the deficit in sustaining attention may be partly related to the difficulty in alerting (Mullane et al., 2011).

William Haefeli / The New Yorker Collection / www.cartoonbank.com

*"I need you to line up by attention span."*

## Hyperactivity–Impulsivity

**MARK**

### Junior Wild Man

Mark, age 14, has more energy than most boys his age. But then, he's always been overactive. At age 3 he was a human tornado, dashing around and disrupting everything in his path. At home, he darted from one activity to the next, leaving a trail of toys behind him. At meals, he upset dishes and talked non-stop. He was reckless and impulsive, running into the street despite oncoming cars, no matter how often his mother explained the danger or scolded him. At the playground, his tendency to overreact—like socking playmates simply for bumping into him—had already gotten him into trouble several times. His parents didn't know what to do. Mark's doting grandparents reassured them, "Boys will be boys. Don't worry, he'll grow out of it." But he didn't.

Adapted from NIMH, 1994a.

**Hyperactivity–impulsivity** involves the undercontrol of motor behavior, poor sustained inhibition of behavior, the inability to delay a response or defer gratification, or an inability to inhibit dominant responses in relation to ongoing situational demands (Nigg & Barkley, 2014). Although the symptoms of hyperactivity and impulsivity are conceptually distinct, when children display one symptom they usually display the other as well. These symptoms are best viewed as a single dimension of behavior called *hyperactivity–impulsivity* (Willcutt et al., 2012). The strong link between hyperactivity and impulsivity suggests a deficit in regulating behavior. There are different reasons for hyperactivity–impulsivity. For example, a child may be constantly out of his seat in the classroom because he wants to look outdoors, because he is anxious about completing an assigned task, or because he can't control his motor behavior.

Britt Erlanson/The Image Bank/Getty Images

Sitting through a class lesson is hard for a child with ADHD

### *Hyperactivity*

The image of a motor-driven ball of speed is the stereotype of a child with ADHD. Sitting still through a class lesson can be impossible for children with ADHD. They may fidget, squirm, climb, run about the room aimlessly, touch everything in sight, or noisily tap a pencil. Parents and teachers describe them as "always on the go" and "talking incessantly." Their activity is excessively energetic, intense, inappropriate, and not goal-directed. The children are extremely active, but unlike other children with a high energy level, they accomplish very little. Recordings of body movements indicate that even when they sleep, children with ADHD display more motor activity than other children (Teicher et al., 1996). However, the largest differences are found in situations requiring the child to inhibit motor activity—to slow down or sit still in response to the structured task demands of the classroom.

### *Impulsivity*

Children who are impulsive seem unable to bridle their immediate reactions or think before they act. They may take apart an expensive clock with little thought about how to put it back together. It's very hard for them to stop an ongoing behavior or to regulate their behavior in accordance with the demands of the situation or the wishes of others. As a result, they may blurt out inappropriate comments or give quick, incorrect answers to questions that have not yet been completed. Because it is difficult to wait or take turns, they interrupt conversations, intrude on others' activities, and lash out in frustration when upset. They also experience difficulty resisting immediate temptations and delaying gratification (Sonuga-Barke et al., 2008). Minor mishaps are common, such as spilling drinks or knocking things over, but more serious accidents and injuries can result from reckless behavior, such as running into the street without looking (Brehaut et al., 2003).

Impulsivity may take different but related forms and is expressed across many life domains, including school and interpersonal relationships (Sharma, Markon, & Clark, 2013; Tsukayama, Duckworth, & Kim, 2013). *Cognitive impulsivity* is reflected in disorganization, hurried thinking, and the need for supervision. (Remember John not handing in his homework even though it was done?) It may also involve impulsive decision making, for example, in the child's valuing of immediate rewards. *Behavioral impulsivity* includes impulsively calling out in class or acting without considering the consequences. Children who are behaviorally impulsive have difficulty inhibiting their response when the situation requires

it. A child may touch a stove to see if it is hot even when she is old enough to know better. Cognitive and behavioral impulsivity (and inattention) predict problems with academic achievement, particularly in reading (Rabiner, Coie, & the Conduct Problems Prevention Research Group, 2000). Only behavioral impulsivity, however, predicts rule-breaking behavior and thus may be a specific sign of increased risk for conduct problems (Willoughby et al., 2000). *Emotional impulsivity* is demonstrated by impatience, low frustration tolerance, hot temper, quickness to anger, and irritability. The term generally refers to how quickly and how likely an individual will react with negative emotions in response to negative events as compared with others of the same age or developmental level (Barkley, 2014c). Although less studied than other types of impulsivity, some findings suggest that emotional impulsivity may be an important component of ADHD that contributes to poor educational, occupational, and other adult outcomes beyond those associated with inattention and hyperactivity–impulsivity (Barkley, 2014c).

In summary, the core features of ADHD—inattention and hyperactivity–impulsivity are made up of many processes. Children with ADHD display a unique constellation and severity of symptoms but may not differ from comparison children on all types and measures of inattention and hyperactivity–impulsivity. The primary attention deficit in ADHD is an inability to engage and sustain attention and to follow through on directions or rules while resisting salient distractions. The primary impairment in hyperactivity–impulsivity involves undercontrol of motor behavior, poor sustained inhibition of behavior, the inability to delay a response or defer gratification, and an inability to voluntarily inhibit dominant responses in relation to situational demands.

## Presentation Type

Investigators have become increasingly interested in identifying the different ADHD presentations (Milich, Balentine, & Lynam, 2001). **Presentation type** refers to a group of individuals with something in common—symptoms, etiology, problem severity, or likely outcome—that makes them distinct from other groupings. DSM specifies three presentation types of ADHD based on the individual's primary symptoms:

- **Predominantly inattentive presentation (ADHD-PI)** describes children who meet symptom criteria for inattention but not hyperactivity–impulsivity.
- **Predominantly hyperactive–impulsive presentation (ADHD-HI)** describes children who meet symptom criteria for hyperactivity-impulsivity but not inattention.
- **Combined presentation (ADHD-C)** describes children who meet symptom criteria for both inattention and hyperactivity–impulsivity.

Children with ADHD-PI are described as inattentive to details, easily distracted, careless, not listening, unfocused, disorganized, unable to sustain effort, and forgetful (Interestingly, the same 1845 storybook that described the hyperactive–impulsive behavior of "Fidgety Phil" also described a very inattentive, distractible young boy named "Hans Look-in-the-Air"). They may have a learning disability, find it hard to remember things, and display low academic achievement (Massetti et al., 2008). They are often described as anxious and apprehensive and socially withdrawn and may display mood disorders (Maedgen & Carlson, 2000). Children with ADHD-PI represent the most common presentation in the general population, but they are less often referred to clinics than are those with ADHD-C (Willcutt, 2012).

One concern about the ADHD-PI presentation is that it may contain at least three diagnostic subgroups (Diamond, 2005; Milich, Balentine, & Lynam, 2001). One group includes children who display both clinically significant symptoms of inattention and subclinical (below the DSM cutoff), but still substantial, levels of hyperactivity–impulsivity. Thus, it is not clear whether this group is qualitatively different from those with ADHD-C or just different in their degree of impairment. Another group with the ADHD-PI presentation consists of children whose inattentive symptoms are linked to problems with arousal and **sluggish cognitive tempo (SCT)**, a cluster that includes symptoms such as daydreaming, trouble staying awake/alert, mentally foggy/easily confused, slow processing of information, stares a lot, spacey, loses train of thought, forgets what was going to say, and appearing lethargic, hypoactive, or even sleepy (Barkley, 2013b; McBurnett et al., 2013). Symptoms of SCT have been viewed both as a core component of some forms of ADHD or as an entirely separate disorder from ADHD that coexists with it in up to 50% of all cases (Barkley, 2014f). Qualitative differences exist between individuals with the SCT subset of ADHD-PI and those with "subthreshold" ADHD-C, suggesting that these groups may have different disorders (Barkley, 2013b). A third subgroup of individuals who meet criteria for ADHD-PI consists of those who originally met criteria for the ADHD-C presentation but experience an age-related reduction in symptoms of hyperactivity–impulsivity and, as a result, no longer meet criteria for the ADHD-C presentation. These individuals with ADHD-PI likely have a different

disorder from those whose symptoms have always been consistent with the ADHD-PI presentation.

Children with the ADHD-HI and ADHD-C presentations are more likely to display problems in inhibiting behavior and in behavioral persistence (Solanto et al., 2007). They are also more likely to be aggressive, defiant, rejected by peers, and suspended from school or placed in special education classes (Short et al., 2007). Children with ADHD-C are the ones most often referred for treatment (Willcutt, 2012). ADHD-HI is the rarest presentation and includes primarily preschoolers. Many of those diagnosed with ADHD-HI as preschoolers do not meet diagnostic criteria for ADHD at a later age, suggesting that the preschool diagnosis reflects a typical developmental phase or possibly a time-limited behavior disorder (Roberts, Milich, & Barkley, 2014).

Although children with different ADHD presentation types are diverse, research findings do not fully support the three subgroup distinctions (Nigg, Tannock, & Rohde, 2010). Presentations may also be unstable over time—a child described as ADHD-PI at one time may be categorized as ADHD-HI or ADHD-C at another point in time, and vice versa (Lahey & Willcutt, 2010). Valo and Tannock (2010) found that 50% of ADHD cases were reclassified from one presentation to another depending on the number of people reporting on the child's symptoms, the methods used to assess symptoms, and how information across reporters and methods was combined.

The heterogeneity of ADHD symptoms is widely acknowledged, but many issues remain regarding the reliability and validity of the presentations specified in DSM-5 and their symptom criteria (Nikolas & Nigg, 2013). Our knowledge of ADHD presentations is clearly still "under construction." It is important to keep this in mind since inconsistencies in the literature may reflect findings that mix together samples of children with different presentations.

## Additional DSM Criteria

Not every child who displays inattention and/or hyperactive–impulsive behavior has ADHD. Most children blurt out things they didn't mean to say, jump from one activity to another, make careless mistakes, or become forgetful and disorganized at times. This doesn't mean they will have a lifelong disorder. ADHD also differs in *severity*—the number of symptoms in excess of those required to make the diagnosis and the degree of impairment in functioning. Symptoms and degree of impairment are used to rate the child's "current severity" of ADHD, ranging from mild to moderate to severe. To diagnose ADHD using DSM-5, the symptoms must also:

- appear before age 12 [*Note:* This represents a change from DSM-IV, which required that symptoms appear before age 7. The age criteria was raised because there was little difference between children with an onset before or after age 7; about half of children with ADHD-PI are not identified until well after age 7; and extending the requirement from age 7 to age 12 does not significantly change the overall prevalence of ADHD (Kieling et al., 2008; Polanczyk et al., 2010)]
- persist for more than 6 months;
- occur more often and with greater severity than in other children of the same age and sex;
- occur across two or more settings (e.g., home, school, other activities)
- interfere with, or reduce the quality of, social, academic, or occupational functioning; and
- not be better explained by another mental disorder (e.g., mood disorder, anxiety disorder).

Illnesses, accidents, middle ear infections, mild seizures, chronic abuse, or stressful life events such as a major move can result in behaviors that mimic the symptoms of ADHD. A normally agreeable 9-year-old boy who becomes inattentive or argumentative immediately after his parents separate is likely having an adjustment reaction, not experiencing ADHD. The disruptive behaviors of youngsters with mild intellectual disabilities, learning disorders, or conduct problems may be mistaken for ADHD, as can the inattentive or restless behaviors of those with anxiety disorders. In fact, most people have trouble concentrating if they are overloaded with too many things to do or a lot of pressure to hurry—sound familiar? Therefore, it is essential to investigate other possible reasons for the individual's symptoms (Smith, Barkley, & Shapiro, 2007).

Depending too heavily on individual symptoms or rating scales to diagnose ADHD can be unreliable and misleading (Solanto & Alvir, 2009). Before a diagnosis of ADHD is made, it is essential to carry out a thorough assessment that includes a developmental history, parent and teacher reports, normed assessment instruments, and behavioral observations (Johnston & Mah, 2008). Importantly, since there is only a modest relationship between ADHD symptoms and impairment in childhood, a child can display ADHD symptoms without necessarily displaying significant impairment. Conversely, a child may display remitted or subclinical levels of ADHD symptoms but still suffer significant maladjustment (Mick et al., 2011). Thus, in evaluating ADHD, it is important to assess both the child's symptoms and impairment in functioning (Gathje, Lewandowski, & Gordon, 2008).

## What DSM Criteria Don't Tell Us

The DSM-5 criteria for ADHD have a number of limitations (Roberts et al., 2014):

- *Developmentally insensitive*. Although DSM states that clinical judgment may be used to assess whether symptoms are "inconsistent with developmental level," it applies the same symptoms to individuals of all ages, even though some symptoms, particularly for hyperactive–impulsive behaviors (running and climbing), apply more to young children (Pine et al., 2011). Similarly, the number of symptoms needed to make a diagnosis is not adjusted for age or level of maturity in younger children, even though hyperactive–impulsive symptoms show a general decline with age. One age adjustment that is included in DSM-5 is a reduced number of ADHD symptoms (from six to five) needed to make the diagnosis in older adolescents and adults (age 17 and older) (APA, 2013). The DSM-5 also includes clarifying examples of how these symptoms might be expressed in teens and adults. The validity of these changes in symptom number and expression in diagnosing and predicting impairment in teens and adults with ADHD has yet to be tested.
- *Categorical view of ADHD*. According to DSM, ADHD is a disorder that a child either has or doesn't have. However, because the number and severity of symptoms are also calculated on a scale, children who fall just below the cutoff for ADHD are not necessarily different from children just above the cutoff. In fact, over time, some children may move in and out of the DSM category as a result of fluctuations in their behavior. Both statistical and neurobiological research support the idea that ADHD is a dimensional rather than a categorical disorder, representing an extreme or a delay in normal traits that all children possess to a degree (Shaw et al., 2011; Sonuga-Barke, 2013). However, it may still be useful to talk about categories even when a disorder is of a continuous or changing nature. For example, there is no magic cutoff for defining high blood pressure, but most of us would agree that people with high blood pressure are at greater risk for certain negative outcomes.

These limitations highlight the fact that DSM criteria are designed to classify and diagnose. They help shape our understanding of ADHD but are also shaped by—and in some instances lag behind—new research findings.

## Section Summary

### Core Characteristics

- DSM-5 uses two lists of symptoms to define ADHD. The first list includes symptoms of inattention, poor concentration, and disorganization. The second list includes symptoms of hyperactivity–impulsivity.
- Children who are inattentive find it difficult to sustain mental effort during work or play and find it difficult to resist salient distractions while doing so.
- Children with ADHD are extremely active, but unlike other children with a high energy level, they accomplish very little.
- Children with ADHD are impulsive, which means they seem unable to bridle their immediate reactions or they may fail to think before they act.
- DSM specifies three presentation types of ADHD based on primary symptoms: predominantly inattentive, predominantly hyperactive–impulsive, or both.
- A diagnosis of ADHD requires the appearance of symptoms before age 12, a greater frequency and severity of symptoms than in other children of the same age and gender, persistence of symptoms, occurrence of symptoms in several settings, and impairments in functioning.
- Although useful, the DSM criteria have several limitations; an important one is developmental insensitivity.

# ASSOCIATED CHARACTERISTICS

In addition to their primary difficulties, children with ADHD often display other problems. For example, Lisa was failing in school and Mark was getting into fights. In the sections that follow, we consider the characteristics and problems commonly associated with ADHD, including cognitive deficits, speech and language impairments, medical and physical concerns, and social problems.

## Cognitive Deficits

Children and adolescents with ADHD display a variety of cognitive deficits, including deficits in executive functions, intellectual deficits, impairments in academic functioning, learning disorders, and distorted self-perceptions. These are discussed in the sections that follow.

### *Executive Functions*

**Executive functions** (EFs) are cognitive processes in the brain that activate, integrate, and manage other brain functions (Pennington & Ozonoff, 1996). They underlie the child's capacity for self-regulation functions such as self-awareness, planning, self-monitoring, and self-evaluation. Having EFs in the brain is like having

an air traffic control system at a busy airport to manage the arrivals and departures of dozens of planes on multiple runways. They help us focus on multiple streams of information at the same time, filter distractions, and revise plans as necessary (Center on the Developing Child at Harvard, 2011). EFs are varied and include (Barkley, 2012):

- *Cognitive processes*, such as working memory (holding facts in mind while manipulating information), mental computation, planning and anticipation, flexibility of thinking, and the use of organizational strategies.
- *Language processes*, such as verbal fluency and the use of self-directed speech.
- *Motor processes*, such as allocation of effort, following prohibitive instructions, response inhibition, and motor coordination and sequencing.
- *Emotional processes*, such as self-regulation of arousal level and tolerating frustration.

For most children, these different processes work in concert, enabling them to exercise deliberate control of their attention and impulses and to maintain problem-solving behaviors in order to attain a future goal. Many EFs reflect abilities that emerge and develop rapidly in preschool children, continue to mature in older children and adolescents, and peak in young adults (Best & Miller, 2010). However, most (not all) children with ADHD show deficits in one or more EFs, especially response inhibition, vigilance, working memory, and planning (Holmes et al., 2010). In fact, as shown in Table 8.2, many symptoms of inattention and hyperactivity–impulsivity reflect impairments in EFs. The number of children with ADHD who display EF deficits may vary depending on how these deficits are measured, with EF rating scales generally finding more deficits than psychometric tests (Weyandt & Gudmundsdottir, 2014). It is also important to keep in mind that children with ADHD may differ in the type of EF deficits they display; for example, some may show poor inhibitory control, others may show poor set shifting, and others may not display any EF deficits (Roberts, Martel, & Nigg, 2013). Children with ADHD with different EF deficit profiles may also differ in other characteristics; for example, their intellectual ability, academic achievement, and co-occurring symptoms or disorders (Roberts et al., 2013).

In light of their close connection with symptoms of ADHD, impairments in EF are viewed as a key deficit in ADHD. However, EF deficits are not *uniquely* associated with ADHD. They also occur in children with other disorders, such as ASD and conduct disorder (Banaschewski et al., 2005; Salcedo-Marin et al., 2013). In addition, EF deficits occur in only about half of children with ADHD (Lambek et al., 2011), suggesting that they are one important component of ADHD but that other deficits also need to be examined. One study of children with ADHD with and without EF deficits found that the two groups were comparable in ADHD symptoms and school functioning. However, those with EF deficits had lower IQ scores and displayed greater response variability. In contrast, those with ADHD without EF deficits showed more aversion to delay of reward. If confirmed, these and other findings suggest multiple pathways for ADHD (Lambek et al., 2010), a point we return to in a later section on causes.

**TABLE 8.2** | **Impaired Executive Functions in ADHD and Examples of Resulting Impairments**

| Impaired Executive Function | Resulting Impairment |
|---|---|
| 1. Organize, prioritize, and activate | Trouble getting started<br>Difficulty organizing work<br>Misunderstand directions |
| 2. Focus, shift, and sustain attention | Lose focus when trying to listen<br>Forget what has been read and need to reread<br>Easily distracted |
| 3. Regulate alertness, effort, and processing speed | Excessive daytime drowsiness<br>Difficulty completing a task on time<br>Slow processing speed |
| 4. Manage frustration and modulate emotion | Very easily irritated<br>Feelings hurt easily<br>Overly sensitive to criticism |
| 5. Working memory and accessing recall | Forget to do a planned task<br>Difficulty following sequential directions<br>Quickly lose thoughts that were put on hold |
| 6. Monitor and regulate action | Find it hard to sit still or be quiet<br>Rush things, slapdash<br>Often interrupt, blurt things out |

*Source:* Based on Brown, 2000.

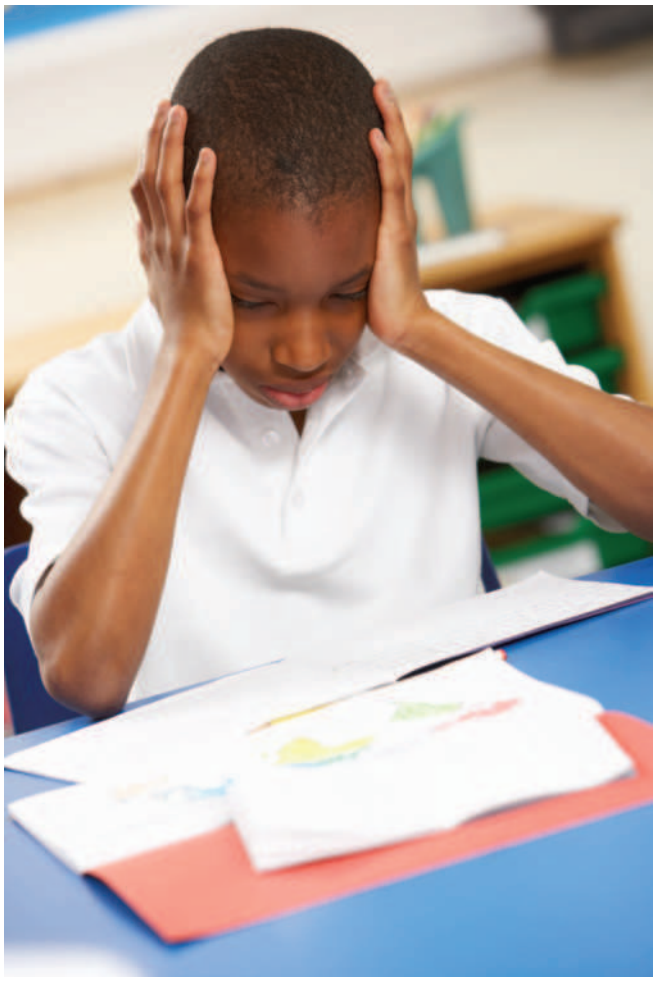
### *Intellectual Deficits*

Most children with ADHD are of at least average overall intelligence, and some are quite bright (Antshel et al., 2008). Their difficulty lies not in a lack of intelligence, but rather in applying their intelligence to everyday life situations (Barkley, 2006a). As a result, the children never quite live up to their potential. They do score about

5 to 9 points lower on IQ tests than both control children and their own siblings (McConaughey et al., 2009). Since IQ tests such as the Wechsler Intelligence Scale for Children, Fourth Edition (WISC-IV) include subtests related to specific deficits of children with ADHD (e.g., working memory), their lower test scores are not surprising (Frazier, Demaree, & Youngstrom, 2004). In addition, lower IQ scores can be the direct result of the effects of ADHD symptoms on test-taking behavior. For example, a child who scores lower on an IQ test because he or she is not paying attention to instructions or is engaging in off-task behaviors is not necessarily less intelligent. On average, inattention has been estimated to account for about a 2- to 5-point lowering of overall IQ-test scores in children with ADHD (Jepsen, Fagerlund, & Mortensen, 2009). Other factors that may contribute to a lower overall IQ score include possible family influences, co-occurring learning disabilities, and deficits in executive functioning.

### *Impaired Academic Functioning*

Most children with ADHD experience severe difficulties in school; this is especially true for those with co-occurring disorders (DuPaul & Langberg, 2014). They frequently have lower productivity, grades, and scores on achievement tests. They may also fail to advance in grade or may be placed more frequently in special education classes. Finally, they may be expelled and fail to finish high school or obtain post-secondary education (Loe & Feldman, 2007). Particularly disturbing are findings that the academic skills of children with ADHD are impaired before they enter the first grade (Barkley, Shelton et al., 2002).



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The structured demands of a classroom can be painful for a child with ADHD

### *Specific Learning Disorder*

As many as 45% of children with ADHD have a specific learning disorder (DuPaul, Gormley, & Laracy, 2013); that is, they have trouble with certain academic skills, such as reading, spelling, and math (Weyandt & Gudmundsdottir, 2014). When learning disorder is broadly defined as performance below expected grade level, nearly 80% of children with ADHD qualify for a learning disorder by late childhood. However, when defined more narrowly as a significant delay in reading, arithmetic, or spelling that is relative to the child's general intellectual functioning, or defined as extremely low achievement in a specific academic subject(s), the number drops to around 25% (Barkley, 2006a). Children with ADHD and those with learning problems show distinct patterns of cognitive deficits, which may be present in combination in those with both disorders (Gooch, Snowling, & Hulme, 2011).

Different pathways may underlie the association between ADHD and learning disorders (Taylor, 2011). For example, the child's cognitive and intellectual deficits may directly lead to learning problems. The impact of childhood ADHD symptoms on long-term academic achievement may also be indirect—they influence later school grades as a result of their effects on homework management and classroom performance (Langberg et al., 2011). ADHD may also predispose the child to conduct problems (to be discussed in Chapter 9) in school that may in turn result in poor academic performance. The association could also be due to common neuropsychological deficits (McGrath et al., 2011; Rajendran et al., 2013) or a common genetic link, although findings suggest that the two disorders are transmitted independently within families (Del'Homme et al., 2007).

### *Distorted Self-Perceptions*

The many failures experienced by children with ADHD have led to the common belief that these children must suffer from low self-esteem—and there is some support for this (Treuting & Hinshaw, 2001). However, many children with ADHD report a higher self-esteem than warranted by their behavior (Owens et al., 2007). For example, they

may perceive their relationships with their parents, teachers, or peers no differently than do control children, even though their parents, teachers, or peers see things in a more negative light (Gerdes, Hoza, & Pelham, 2003; Normand et al., 2013). This exaggeration of one's competence is referred to as a **positive bias.** Positive bias can occur in relation to social competence, where it may be particularly problematic, or in other areas such as academic performance or behavioral conduct (Hoza et al., 2012). Some findings suggest that self-esteem in children with ADHD may vary with the type of ADHD presentation, the accompanying disorders, and the area of performance being assessed (e.g., conduct, scholastic achievement). Children with ADHD who display inattentive and depressive/anxious symptoms tend to report lower self-esteem, whereas those with symptoms of hyperactivity–impulsivity and conduct problems appear to exaggerate their self-worth (Owens et al., 2007). The bias in the latter group is most dramatic in the areas of performance in which the child is most severely impaired. Increases in positively biased self-perceptions of behavior in children with ADHD have also been found to predict greater aggression over time (Hoza et al., 2010). In general, research has found that children with ADHD and a positive social or behavioral bias are more likely to display persistent social impairments and more negative behaviors than those with ADHD without this bias (McQuade & Hoza, 2014).

Several explanations for the positive bias in children with hyperactivity–impulsivity have been proposed—it serves a self-protective function that allows the child to cope every day despite frequent failures; it reflects a diminished self-awareness as a result of impairments in executive functions (McQuade et al., 2011); or it is a result of not knowing what constitutes successful or unsuccessful performance (Ohan & Johnston, 2002). To date, there is some support for the self-protective function of positive bias (Hoza et al., 2010), although other explanations may also apply.

Children with ADHD also display distortions in their perceptions of **quality of life**, which refers to a person's subjective perception of their position in life as evidenced by their physical, psychological, and social functioning. According to parents, the impact of their child's ADHD on the child's quality of life is substantial, particularly when the child has co-existing emotional and conduct problems (Schei et al., 2013). However, despite experiencing many life difficulties, children with ADHD rate their own quality of life more positively than others rate it (Danckaerts et al., 2010).

## Speech and Language Impairments

About 30% to 60% of children with ADHD also have impairments in their speech and language (Helland et al., 2012). Interestingly, the type of speech and language impairment may be related to the child's specific ADHD symptoms. For example, one study with preschoolers found that symptoms of hyperactivity–impulsivity were related to poor language skills, whereas those of inattention were more highly correlated with weaker receptive and expressive vocabulary skills (Gremillion & Martel, 2013). In addition to showing a higher prevalence of formal speech and language disorders (see Chapter 7), children with ADHD may have difficulty in understanding others' speech and in using appropriate language in everyday situations (McInnes et al., 2003; Wassenberg et al., 2010). The pragmatic aspects of speech, along with impaired verbal working memory and discourse analysis, are primary difficulties (Bellani et al., 2011). Impairment in pragmatic language skills has been related to these children's social difficulties, and may, in part account for these difficulties (Staikova et al., 2013). Excessive and loud talking, frequent shifts and interruptions in conversation, inability to listen, and inappropriate conversation are a few common examples of impairments.

Children with ADHD not only ramble on, but also their conversation is characterized by speech production errors, fewer pronouns and conjunctions, tangential and unrelated comments, abandoned utterances, and unclear links (Mathers, 2006; McGrath et al., 2008). Can you understand the following statement by a boy with ADHD?

> And all of a sudden the soldiers—and all of a sudden he gets faint and you know when he says "Good doctors, I want to talk with you" and all of a sudden he goes in the door and inside they come off it from the thing. So he puts—I think this something on the doorknob. (Tannock et al., 1995)

When speech is unclear, as in this example, it is difficult for the listener to understand who and what the child is talking about. Unfortunately, miscommunication is all too common in children with ADHD.

## Medical and Physical Concerns

In addition to the difficulties we have discussed, children and adolescents with ADHD also experience a number of medical and physical concerns, including having health-related problems, being accident-prone, and demonstrating risk-taking behaviors (Barkley, 2014d; Nigg, 2013).

### *Health-Related Problems*

In terms of specific problems, some studies have reported higher rates of enuresis and encopresis (Shreeram et al., 2009) and asthma (Fasmer et al., 2011). One prospective study found that asthma in early life increased

the risk of developing ADHD during the school years (Chen et al., 2013). Other health concerns for which individuals with ADHD are at elevated risk include dental health problems, poor fitness, obesity, and eating problems and disorders (in females) (Barkley, 2014d). Sleep disturbances are also common in children with ADHD (Cortese et al., 2009, 2013; Owens et al., 2013). Resistance to going to bed, difficulty in falling asleep, fewer total hours asleep, and involuntary sleep movements such as teeth grinding or restless sleep may be the most significant disturbances (Spruyt & Gozal, 2011). Some of the sleep problems in children with ADHD may be related to their use of stimulant medications, and/or co-occurring conduct or anxiety disorders, rather than to only their ADHD (Mick, Biederman et al., 2000).

### *Accident-Proneness and Risk Taking*

Given their problems with impulsivity, motor inhibition, and lack of planning and forethought, it is not surprising that over 50% of parents of children with ADHD describe their child as accident-prone. These children are about three times more likely to experience serious accidental injuries, such as broken bones, lacerations, severe bruises, burns, poisonings, or head injuries (Barkley, 2014d). Young adult drivers with ADHD are at higher risk than others for traffic accidents, and deviant peer associations may play an important role (Cardoos, Loya, & Hinshaw, 2013; Cox, Madaan, & Cox, 2011).

ADHD is a significant risk factor for the early initiation of cigarette smoking, substance-use disorders, Internet- and videogame-use problems and addictions, and risky sexual behaviors such as multiple partners and unprotected sex (Barkley, 2014d; Lee et al., 2011). Substance-use disorders among young people with ADHD are also more frequent, severe, and persistent than substance-use disorders in those without ADHD (Charach et al., 2011). There are a number of possible reasons that ADHD may lead to substance-use problems, a notable one being co-occurring or later conduct problems (Wilens, 2011). Overall, these findings suggest a progression of hyperactive–impulsive behaviors during childhood to a pattern of irresponsible and risky adolescent and adult behavior.

In support of this, a prospective 33-year follow-up study of 8-year-old boys with ADHD but without conduct disorder found that as adults they had relatively more risky driving behaviors, sexually transmitted diseases, head injuries, and emergency department admissions than a comparison group and that lifetime risk-taking was related to negative health outcomes and more deaths not related to specific medical conditions. Importantly, the relationship between ADHD and risk taking was accounted for by the later development of Conduct Disorder (CD)/Antisocial Personality Disorder (APD). Over their lifetime, individuals with ADHD who did not develop CD/APD did not differ from the comparison group in risk-taking behaviors (Ramos Olazagasti et al., 2013).

Impulsive behavior is the most significant childhood characteristic that predicts reduced life expectancy (an average of 8 years less), according to a longitudinal study spanning over a half century (Friedman et al., 1995). A reduced life expectancy for individuals with ADHD may also be predicted by a pattern of accident-proneness, auto accidents, and risk taking, combined with a reduced concern for health-promoting behaviors, such as exercise, proper diet, safe sex, and moderate use of tobacco, alcohol, and caffeine, especially for those with co-occurring CD/APD symptoms (Barkley, 2014d).

The need for further research into health-related problems in children and adolescents with ADHD is accentuated by findings that show they have significantly higher rates of inpatient and outpatient hospitalizations and emergency department visits. Average medical costs for children with ADHD are more than double the costs for those without ADHD (Le et al., 2013; Leibson et al., 2001) and at least comparable to the costs for children with asthma (Chan, Zhan, & Homer, 2002). The multitude of health and related problems and costs indicate that "ADHD is more than just a serious mental health problem—it is a serious *public health* problem" (Barkley, 2014d).

## Social Problems

**DENNIS**

### Nothing Sticks

With my other children, I could tell them one time, "Don't do that," and they would stop. But Dennis, my child with ADHD, I could tell him a hundred times, "Dennis, don't carve soap with my potato peeler," "Don't paint the house with used motor oil," or "Don't walk on Grandma's white sofa in your muddy shoes," but he still does it. It's like every day is a brand new day and yesterday's rules are long forgotten. … I just cannot stay one step ahead of him. He does things my other kids never thought of.

From *The Hyperactive Child Book*, by Patricia Kennedy, Leif Terdal, and Lydia Fusetti, pp. 8–9. New York: St. Martin's Press.

Social problems in family life and at school are common in children with ADHD (Johnston & Chronis-Tuscano, 2014; McQuade & Hoza, 2014). Those who experience the most severe social disability are at greatest risk for poor adolescent outcomes and other disorders, such as depression and conduct disorder (Greene et al., 1996). Children with ADHD don't listen

and are often hostile, argumentative, unpredictable, and explosive. As a result, they are frequently in conflict with adults and other children. It is common for children with ADHD to be removed from swimming lessons because of disruptive behavior or to be kicked out of gymnastics. For one child with ADHD taking dance lessons, the teachers offered to buy back her shoes if only her parents would take her out!

To get along with others, you must follow social rules and respect conventions. Children with ADHD do not play by the same rules as others and don't seem to learn from past mistakes, despite their awareness of expected social behaviors and a desire to conform to them. Many social blunders by children with ADHD appear more thoughtless than intentional. Even with good intentions, their behaviors have an annoying quality that is a source of great distress for their parents, siblings, teachers, and classmates. In the words of one mother: "Our grade one parent interview was highly traumatic; her teacher cried!"

### Family Problems

Families of children with ADHD experience many difficulties, including interactions characterized by negativity, noncompliance by the child, excessive parental control, and sibling conflict (Johnston & Chronis-Tuscano, 2014). Parents may experience high levels of distress and related problems—most commonly, depression in mothers and antisocial behavior, such as substance abuse, in fathers. Further stress on family life stems from the fact that parents of children with ADHD may themselves have ADHD and other associated conditions (Johnston et al., 2012). This can also be a barrier to effective treatment for the child (Sonuga-Barke, Daley, & Thompson, 2002).

Families of children with ADHD also report less parenting competence, fewer contacts with extended family members, greater caregiver strain, less instrumental support, and slightly higher rates of marital conflict, separation, and divorce (Bussing et al., 2003; Johnston & Mash, 2001). They report generally higher levels of parenting stress, which may fluctuate over the course of the day in relation to the child's ADHD-related behaviors (Theule et al., 2013; Whalen et al., 2011). Parents of these children also show increased alcohol consumption that, in some instances, could be a direct result of stressful interactions with their children (Pelham & Lang, 1999). Many also report stigmatizing experiences, including concerns about how society would label their child, social isolation and rejection, and perceptions that health care and school personnel are dismissive of their concerns (dosReis et al., 2010; Moldavsky & Sayal, 2013). Finally, when interviewed, siblings of children with ADHD report that they feel victimized by their ADHD sibling and that this experience is often minimized or overlooked (Kendall, 1999).

It is important to note that the links between ADHD and high levels of family conflict, parental psychopathology, and marital discord are, in many cases, due to the child's co-occurring conduct problems rather than to ADHD symptoms alone (Johnston & Mash, 2001).

Things I've learned from my ADHD child (honest and no kidding):

> If you hook a dog leash over a ceiling fan, the motor is not strong enough to rotate a 42-pound boy wearing batman underwear and a superman cape. It is strong enough, however, if tied to a paint can, to spread paint on all four walls of a 20 × 20 foot room. (An anonymous mother in Austin, Texas)

### Peer Problems

Peer problems in both boys and girls with ADHD are apparent at an early age and are quickly evident when the child enters a new social situation. These children display little of the give-and-take that characterizes other children (McQuade & Hoza, 2014). Children with ADHD can be bothersome, stubborn, socially awkward, and socially insensitive. Others describe them as socially conspicuous, loud, intense, and quick to react. They are socially active but usually "off the mark" with respect to the style, content, or timing of their behavior, which often has an annoying quality that brings out the worst in other children (Whalen & Henker, 1992). Children with ADHD seem to get into trouble even when trying to be helpful, and although their behavior seems thoughtless, it is often unintentional. They often see their own behavior more favorably than it is perceived by others, and they may be puzzled by others' negative reactions (Nijmeijer et al., 2008).

In light of their social difficulties, it is not surprising that children with ADHD are disliked and uniformly rejected by peers, have few friends, and report receiving low social support from peers (Gardner & Gerdes, 2013; McQuade & Hoza, 2014). Their lack of skill in correctly recognizing emotions in others and in regulating their own emotions and behavior (Kats-Gold, Besser, & Priel, 2007) and the aggressiveness that frequently accompanies ADHD often lead to social conflict and a negative reputation (Erhardt & Hinshaw, 1994). For girls with ADHD-PI, internalizing symptoms may play a particularly salient role in their being disliked or rejected by peers (Becker et al., 2013). The social problems of these children may increase the later risk of having disorders other than ADHD and may spill over into other areas of development (Murray-Close et al., 2010). Once they are labeled "ADHD" by their peers, a negative process begins whereby the child

suffers more negative treatment and rejection by peers, leading to a cascading of negative effects over time.

Children with ADHD are not deficient in social reasoning or understanding (Whalen & Henker, 1992). They simply do not use what they know during social exchanges, and they may continue to be dominant or assertive even when the situation changes and requires accommodation, negotiation, or submission (Landau & Milich, 1988). Their social agenda may also differ from the agenda of their peers, especially when ADHD is accompanied by aggression. They may actually value and prefer troublemaking, sensation seeking, and having fun at the expense of following rules and getting along with others (Melnick & Hinshaw, 1996).

Despite their many social problems with peers, some adolescents with ADHD may meet their social needs by maintaining one or two positive close friendships (Glass, Flory, & Hankin, 2012). The social premise for such relationships may differ from those of other teens, possibly with a mutual focus on "having fun" rather than on seeking emotional support. Positive friendships may buffer the negative outcomes of peer rejection commonly seen in children with ADHD. Since most research on ADHD has examined peer relationships in general, further research into close friendships is needed to determine their nature and function during adolescence.

## Section Summary

### Associated Characteristics

- Besides their primary difficulties, children with ADHD display other problems, such as cognitive and learning deficits, speech and language impairments, motor incoordination, medical and physical concerns, and social problems.
- Children with ADHD display deficits in executive functions (EFs), the higher-order mental processes that underlie the child's capacity for planning and self-regulation.
- Children with ADHD score slightly lower on IQ tests, but most are of normal intelligence. Their difficulty is in applying their intelligence to everyday life situations.
- Children with ADHD experience school performance difficulties, including lower grades, a failure to advance in grade, and more frequent placements in special education classes.
- Many children with ADHD have a specific learning disorder, typically in reading, spelling, or math.
- Some children with ADHD report a higher self-esteem than is warranted by their behavior, referred to as a " positive bias."
- They often have speech and language impairments and have difficulty using language in everyday situations.
- They may experience many health-related problems, including enuresis and encopresis, asthma, obesity, eating problems, and sleep disturbances and tend to be accident-prone. The costs of and medical service use in those with ADHD are high.
- They experience numerous social problems with family members, teachers, and peers.

## ACCOMPANYING PSYCHOLOGICAL DISORDERS AND SYMPTOMS

Children with ADHD typically present a mixed bag of symptoms and impairments, with ADHD being only one, albeit a significant one. Nearly two-thirds of parents report behaviors other than the core symptoms of ADHD as being the most worrisome, typically aggression and defiance (Findling et al., 2009). Co-occurring psychological disorders are also common, usually with an onset after that of the ADHD (Yoshimasu et al., 2012). In community samples, up to 44% of children with ADHD have at least one other disorder, and 43% have at least two or more other disorders (Willcutt et al., 2012). One reason that ADHD is so challenging is that as many as 80% of clinic-referred children with ADHD have a co-occurring psychological disorder, and up to 50% have two or more disorders (Pliszka, 2014). Common co-occurring disorders include oppositional and conduct disorders, anxiety disorders, mood disorders, and motor coordination and tic disorders. As we noted previously, learning disorders are also quite common, as are substance-use disorders in adolescence (see Chapter 13). Some children with ADHD display symptoms of ASD (see Chapter 6), possibly originating from similar familial/genetic factors (Rommelse et al., 2011). Children with ADHD and ASD traits display higher rates of psychopathology, neuropsychological deficits, social difficulties with peers, and restricted and repetitive behaviors than do those with ADHD without ASD traits (Kotte et al., 2013; Martin et al., 2014).

### Oppositional Defiant Disorder and Conduct Disorder

**SHAWN**

**Bad Boy**

Shawn, now an energetic and talkative young adult, recalls his childhood with ADHD as a total disaster: "I did really bad in school. My parents and teachers were always on my back. They bugged me about being too loud, too defiant, too explosive, and too aggressive. Then I began to use drugs: marijuana, and later, cocaine. I barely managed to squeak through high school. I

(*continues*)

(*continued*)

couldn't concentrate at all. I'd study for hours and then forget everything I'd read. I had to cheat my way through high school." (Based on authors' case material.)

About half or more of all children and adolescents with ADHD—mostly boys, like Shawn—meet criteria for oppositional defiant disorder (ODD) by age 7 or later (Pliszka, 2014). Children with ODD overreact by lashing out at adults and other kids. They are stubborn, short-tempered, argumentative, and defiant. The symptoms of ODD generally fall into two types—irritability (e.g., tantrums, crankiness) and defiance (e.g., talking back, argumentativeness) (Kuny et al., 2013). About 30% to 50% of children with ADHD eventually develop conduct disorder (CD) (Beauchaine, Hinshaw, & Pang, 2010), which is more severe than ODD. Children with CD violate societal rules and are at high risk for getting into serious trouble at school or with the police. They may fight, cheat, steal, set fires, or destroy property. CD is also associated with the use of illegal drugs, which may explain why children with ADHD may have a higher risk for developing substance-use problems in adolescence (Szobot & Bukstein, 2008). ADHD that occurs early, particularly when it is accompanied by severe symptoms of hyperactivity–impulsivity, increases the odds of ODD/CD by about 10-fold, making it one of the most reliable predictors of these disorders (Angold, Costello, & Erkanli, 1999). Longitudinal studies have found that ADHD leads to ODD and CD rather than vice versa (Thapar et al., 2006). Interestingly, persistent and severe ODD and CD outcomes among children with ADHD are related to variations in a specific gene (*COMT*) known to be associated with the regulation of neurotransmitters in the areas of the brain implicated in ADHD. These findings suggest the existence of a subgroup of children with ADHD who are at biological risk for later developing conduct problems (Caspi et al., 2008). Finally, ADHD is also a risk factor for the later development of antisocial personality disorder (APD) (Storebø & Simonsen, 2013), a pervasive pattern of disregard for, and violation of, the rights of others, as well as involvement in multiple illegal behaviors.

ADHD, ODD, and CD run together in families, which suggests a common predisposing cause. In support of this, there is a substantial common genetic contribution for the three disorders, especially between ADHD and ODD (Coolidge, Thede, & Young, 2000; Hamshere et al., 2013). There is also evidence for a contribution from a shared environment, perhaps related to family adversity and deficits in parenting (Burt et al., 2001). We discuss ODD, CD, and APD in greater detail in Chapter 9.

## Anxiety Disorders

**T.J.**

### Overactive and Anxious

T. J. was first referred for help at age 6. He had been very active and impulsive since he was a toddler. His parents reported that he had trouble sleeping and would wake up several times each night. They also said that he showed great anxiety during even brief separations from them and seemed to be worrying about something the whole time. T. J. confirmed that he had "terrible bad dreams" and felt that no one liked him.

Based on Tannock, 2000.

"Sam, neither your father nor I consider your response appropriate."

Edward Koren / The New Yorker Collection / www.cartoonbank.com

About 25% or more of children with ADHD experience excessive anxiety (Manassis, 2007). These children worry about being separated from their parents, trying something new, taking tests, making social contacts, or visiting the doctor. They may feel tense or uneasy and constantly seek reassurance that they are safe and protected. Because these anxieties are unrealistic, more frequent, and more intense than normal, they have a negative impact on the child's thinking and behavior. Findings regarding whether or not co-occurring anxiety worsens the symptoms or severity of ADHD are inconsistent (Hammerness et al., 2010). However, children with co-occurring ADHD and anxiety display

social and academic difficulties and experience greater long-term impairment and mental health problems than those with either condition alone (Manassis et al., 2007).

## Mood Disorders

As many as 20% to 30% of young people with ADHD experience depression (Daviss, 2008; Spencer et al., 2000), and even more will develop depression or another mood disorder by early adulthood (Fischer et al., 2002). Being diagnosed with ADHD between 4 and 6 years of age is a risk factor for future depression and suicidal behavior in adolescence, particularly for girls (Chronis-Tuscano, Molina, et al., 2010). These youths feel so sad, hopeless, and overwhelmed that they are unable to cope with everyday life. Depression lowers self-esteem; reduces interest or pleasure in favorite activities; increases irritability; and disrupts sleep, appetite, and the ability to think (Mick, Santangelo et al., 2000). Youths with ADHD have higher rates of suicidal ideation and deliberate self-harm than controls, although the highest risk for suicide is among those with ADHD with co-occurring depression and conduct problems (Pliszka, 2014).

The association between ADHD and depression may be a function of the fact that family risk for one disorder increases the risk for the other. This suggests that depression in a child with ADHD is not due solely to the child's demoralization as a result of their ADHD symptoms (Biederman, Mick, & Faraone, 1998).

Controversy abounds regarding the association between ADHD and pediatric bipolar mood disorder (BP) (to be discussed in Chapter 10). In part, this is due to the difficulty in distinguishing between symptoms of ADHD and the unregulated high energy level, poor judgment, and over-talkativeness of children with BP, as well as to possible shared underlying mechanisms (Youngstrom & Algorta, 2014; Youngstrom, Arnold, & Fraser, 2010). The relationship between the two disorders seems to go mainly in one direction—a diagnosis of childhood BP sharply increases the child's risk for previous or co-occurring ADHD, but a diagnosis of ADHD does not appear to increase the child's risk for BP (Pataki & Carlson, 2013; Skirrow et al., 2012).

## Developmental Coordination and Tic Disorders

As many as 30% to 50% of children with ADHD display motor coordination difficulties—such as clumsiness, poor performance in sports, or poor handwriting—especially when they attempt to execute complex motor sequences (Fliers et al., 2010). Impairments often appear in the motor skills domains of strength, visual motor coordination, adjusting speed, and dexterity. As many as 50% of children with ADHD may have a **developmental coordination disorder (DCD)**, a condition characterized by marked motor incoordination and delays in achieving motor milestones (Brossard-Racine et al., 2012; Fliers et al., 2012). About 20% of children with ADHD also have **tic disorders**—sudden, repetitive, nonrhythmic motor movements or sounds such as eye blinking, facial grimacing, throat clearing, and grunting (Murphy et al., 2013; Simpson, Jung, & Murphy, 2011). These children experience more behavioral, social, and academic difficulties than do those with ADHD alone. When present, tic disorders decline to low rates by adolescence and do not appear to significantly affect later psychosocial functioning (Peterson et al., 2001).

## Section Summary

### Accompanying Psychological Disorders and Symptoms

- A factor that makes ADHD so challenging is that children with the disorder have much higher than expected rates of other psychiatric disorders, particularly conduct problems, anxiety, and mood disorders.
- As many as 50% of children with ADHD also meet criteria for oppositional defiant disorder or conduct disorder.
- About 25% or more of children with ADHD experience excessive anxiety. The presence of co-occurring anxiety is associated with more social and academic difficulties, and greater long-term impairment and mental health problems.
- As many as 20% to 30% children with ADHD experience depression or another mood disorder. Although depression may be partly related to demoralization as a result of their symptoms, it also can result from an elevated risk for depression in families of children with ADHD.
- The relation between ADHD and bipolar disorder is controversial. A diagnosis of childhood bipolar disorder appears to sharply increase the child's risk for previous or co-occurring ADHD, but a diagnosis of ADHD does not appear to elevate the child's risk for bipolar disorder.
- Children with ADHD may display motor coordination difficulties and tic disorders.

# PREVALENCE AND COURSE

*When we began our studies in the 1960's no one believed such children existed; while now people find them under every rock.*

—Dr. Leon Eisenberg, child psychiatrist and ADHD pioneer (1922–2009)

The striking increase in ADHD alluded to in the above quote likely reflects an increase in diagnostic practices rather than an epidemic of ADHD. This increase is based

partly on growing knowledge about ADHD, pressures from parents seeking treatment for children, and adults identifying their own ADHD (Taylor, 2009). The growing recognition of ADHD has established that it affects millions of children throughout the world and across all socioeconomic levels (Erskine, 2013). Although rates can vary widely with sampling methods (Rowland et al., 2013), the best estimate is that about 5% to 9% of all children and adolescents 4 to 17 years old in North America are currently diagnosed with ADHD; worldwide, just over 5% have ADHD (APA, 2013; Polanczyk et al., 2007; Visser et al., 2013). These estimates suggest that about 3 million to 5 million school-age children and adolescents in the United States are affected. As many as half of all children referred to clinics display ADHD symptoms either alone or in combination with other disorders, making ADHD one of the most common referral problems (Barkley, 2006a).

Reports from parents, teachers, and doctors are all used to identify children with ADHD. However, these people do not always agree, because the child's behavior may differ from setting to setting. Also, different adults may emphasize different symptoms when making a judgment. Teachers, for example, are most likely to rate a child as inattentive when the child also displays oppositional symptoms (Abikoff et al., 1993). Prevalence rates and patterns of comorbidity also differ when teacher's reports are compared with parent's reports (Gadow & Nolan, 2002). Since adults may disagree, prevalence estimates of ADHD are much higher when based on one person's opinion rather than on a consensus.

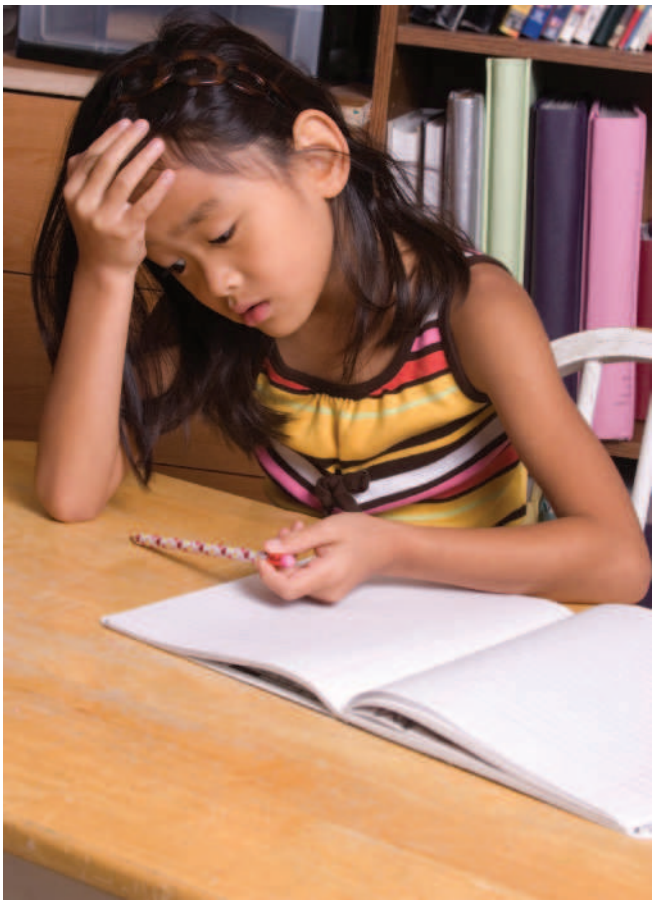
## Gender

ADHD occurs more frequently in boys than in girls, with estimates ranging from 2% to 4% for girls and 6% to 9% for boys 6 to 12 years of age (Owens, Cardoos, & Hinshaw, 2014; Polanczyk & Jensen, 2008). In adolescence, overall rates of ADHD decrease slightly for both sexes, but boys still outnumber girls by the same ratio of about 2.5:1, a ratio that declines by adulthood to about 1.6:1 or lower (Kessler et al., 2005; Merikangas et al., 2010), possibly because of an underidentification of girls in childhood. This ratio is even higher in clinic samples, in which boys outnumber girls by 6:1 or more—most likely because boys are referred more frequently because of their overt defiance and aggression or because parents and teachers may think that learning assistance is less effective for girls than for boys with ADHD (Ohan & Visser, 2009).

ADHD in girls may go unrecognized and unreported because teachers fail to recognize and report inattentive behavior unless it is accompanied by the disruptive symptoms normally demonstrated by boys. In addition, the symptoms used to diagnose ADHD may also contribute to the sex difference in prevalence. DSM criteria were developed and tested mostly with boys with ADHD, and many of the symptoms—excessive running around, climbing, and blurting out answers in class— are generally more common in boys than girls. Thus, the specified cutoffs and symptoms may be more appropriate to boys than to girls, because girls with ADHD may have to display not only extreme behavior but also behavior that is uncharacteristic as compared with their same-sex peers before they will be referred (Hinshaw & Blachman, 2005). Interestingly, when girls with ADHD also display defiance and aggression, they are referred at a younger age than boys, a finding that implies lower tolerance by adults or a greater concern for these behaviors when they occur in girls (Silverthorn et al., 1996). Girls with ADHD may be more likely than boys to display inattentive/disorganized symptoms characteristic of a sluggish cognitive tempo, including forgetfulness, lethargic behavior, mental confusion, drowsiness, tendency to daydream (McBurnett, Pfiffner, & Frick, 2001), anxiety, depression (Rucklidge & Tannock, 2001), and hyperverbal rather than hyperactive motor behavior (Nadeau, Littman, & Quinn, 1999). Thus, current DSM criteria may be insensitive to the problems that are especially relevant to girls (Ohan & Johnston, 2005). Although it may be premature to expand the current symptom lists for ADHD to include additional items appropriate to girls, it is clear that sampling, referral, and definition biases may all contribute to reports of ADHD being more prevalent in boys than in girls.

In the past, girls with ADHD were a highly understudied group (Hinshaw & Blachman, 2005). Although girls with ADHD tend to display inattentive/disorganized symptoms, some research shows more similarity between girls and boys with ADHD than was previously thought to exist. Some studies have found that among clinic-referred school-age children with ADHD, boys and girls are quite similar with respect to their expression and severity of symptoms, brain abnormalities, deficits in response inhibition and executive functions, level of impairment, family correlates, response to treatment, and outcomes (Biederman et al., 2006; Owens et al., 2014). Follow-up studies of girls with ADHD indicate clear evidence of major problems through adolescence and young adulthood, including anxiety; depression; romantic relationship difficulties; conflict with mothers; significant peer rejection and conduct problems; large deficits in academic achievement; continuing deficits in attention, executive functions, and language; impaired decision making; and high rates of service utilization (Babinski et al., 2010; Biederman et al., 2010; Mick et al., 2011; Miller et al., 2013; Owens et al., 2014).

It is noteworthy that girls with ADHD who display hyperactive–impulsive behaviors are more likely to develop symptoms of eating disorders (bingeing–purging



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Girls with ADHD may be described by their teachers as "spacey" or "in a fog." Without hyperactivity and disruptive behavior, ADHD in girls may go unrecognized or be ignored.

behaviors, body dissatisfaction) and to engage in self-injury and suicide attempts than are girls with ADHD who display only symptoms of inattention or girls who do not have ADHD (Hinshaw et al., 2012; Mikami et al., 2008; Swanson, Owens, & Hinshaw, 2013). These findings support the need to consider behaviors and outcomes for girls with ADHD that reflect female-relevant domains of impairment.

## Socioeconomic Status and Culture

ADHD affects children from all social classes, although there are slightly more children with ADHD in lower socioeconomic status (SES) groups (Akinbami et al., 2011). Low family income in childhood is also associated with an increased likelihood of ADHD (Larsson et al., 2013). The relationship between ADHD and SES may be related to co-occurring conduct problems in children with ADHD, since conduct problems are related to conditions that accompany low SES, such as family adversity and stress (Szatmari, Offord, & Boyle, 1989). Consistent with this view, family conflict and attachment links ADHD with SES (Russell et al., 2014).

Research on the relationships among ADHD, race, and ethnicity have been inconsistent, and it remains unclear whether current tools for assessing ADHD adequately capture the expression of ADHD in minority groups. By kindergarten entry, children in the United States who are black are 70% less likely to be diagnosed with ADHD than otherwise similar white children—even though they are equally likely to display ADHD-related behaviors in the classroom (Morgan et al., 2014). However, for older children, teacher-rated ADHD and observed rates of ADHD behavior are higher for black than for white children, which are not explained by rater bias or SES (Miller, Nigg, & Miller, 2009). Slightly lower rates of ADHD have been reported for Hispanic, Asian, American Indian, and Pacific Islander children (Cuffe, Moore, & McKeown, 2005). Knowledge about ADHD and access to treatment seem to be greater among Caucasian, non-Hispanic, and more highly educated families (McLeod et al., 2007; Miller et al., 2009). However, some research suggests that when families from different ethnic groups do receive treatment, they do not differ in the benefits derived (Jones et al., 2010).

ADHD has been identified in every country where it has been studied. Estimates vary across countries and cultures, with the highest rates found in South America and Africa (8% to 12%) and the lowest rates in Japan and China (2% to 5%). European and North American rates are in between (4% to 9%). These variations are mainly the result of differences in source of information (e.g., parents, teachers) and the way ADHD is diagnosed across studies done in different parts of the world. When a uniform diagnostic method is used, the rates of ADHD are highly similar worldwide (Buitelaar et al., 2006).

Varying cultural norms and tolerance for the symptoms of ADHD may explain the different diagnosis rates across cultures (Weisz, Weiss et al., 2006). In cultures that value reserved and inhibited patterns of child behavior, such as Thailand, symptoms of ADHD are less common than in the United States. Moreover, when ADHD symptoms do occur, teachers in Thailand view them as more problematic—likely because of their culture-linked values and expectations (Weisz, Chayaisit et al., 1995). Thus, a child being identified as having ADHD is partly a function of the discrepancy between a child's behavior and cultural expectations about how children ought to behave (Moffitt & Melchior, 2007).

## Course and Outcome

In the sections that follow, we describe how symptoms of ADHD change with development. These changes over the life span are illustrated with the experiences of Alan, now age 35, who was diagnosed with ADHD in grade 1.

### *Infancy*

**ALAN**

#### Off and Running

Our baby-sitter swears I was sitting up watching TV by 4 months. Then from a crawling position I ran, and we were off. … I was all over the house, into everything, and Mom soon realized I could not be left alone. Darting here, there, and anywhere, I didn't like playing with my toys, preferring to explore on my own.

From R. A. Barkley and L. J. Pfiffner, "Off to School on the Right Foot: Managing Your Child's Education. In: Barkley, R. A. Taking Charge of ADHD: The Complete, Authorized Guide for Parents, 1995, p. 208.

It is likely that signs of ADHD are present at birth (one mother reported her child was so overactive in the womb that the kicking nearly knocked her over!). Home-based activity-level assessments in the home—using motion detectors—are related to mothers' ratings of ADHD symptoms in children as young as 2 years of age (Ilott et al., 2010). Similarly, when parents of an older child with ADHD describe what their child was like as a baby, they often say their baby had a difficult temperament—extremely active, unpredictable, oversensitive or undersensitive to stimulation, and irritable with erratic sleep patterns or feeding difficulties. Early markers of ADHD symptoms may be present in infancy and toddlerhood, but reliable identification of ADHD is difficult prior to age 3 (Arnett, MacDonald, & Pennington, 2013). In addition, there are issues with these reports that suggest the early presence of ADHD. First, parents' recollections may be colored by their child's later difficulties. Second, most infants with a difficult temperament do not develop ADHD. Although a difficult temperament in infancy may indicate something amiss in development—and in some cases may be a risk factor for later ADHD—it cannot by itself be taken as an early sign of ADHD. For example, one study reported an association between persistent crying during infancy and a 10-fold higher risk for hyperactivity at 8 to 10 years of age (Wolke, Rizzo, & Woods, 2002). However, most infants who cry persistently do not go on to develop ADHD.

### *Preschool*

**ALAN**

#### Preschool Outcast

I often wondered why I wasn't in group-time in preschool. The teacher sent me in the corner to play with a toy by myself. Because of being singled out I didn't have many friends. I was different, but I didn't know why or what it was.

From R. A. Barkley and L. J. Pfiffner, "Off to School on the Right Foot: Managing Your Child's Education. In: Barkley, R. A. Taking Charge of ADHD: The Complete, Authorized Guide for Parents, 1995, p. 208.

With the growing number of hyperactive–impulsive symptoms at 3 to 4 years of age, ADHD becomes an increasingly visible and significant problem (Greenhill et al., 2008). Preschoolers with ADHD act suddenly and without thinking, dashing from activity to activity, grabbing at immediate rewards; they are easily bored and react strongly and negatively to routine activities (Campbell, 2006). Parents find it very difficult to manage the hyperactivity and noncompliance of their child, who may also be defiant and aggressive. Preschoolers with ADHD often roam about the classroom or daycare, talking excessively and disrupting other children's activities. Those who display a persistent pattern of hyperactive–impulsive and oppositional behavior for at least 1 year are likely to continue on to difficulties into middle childhood and adolescence (Olson et al., 2000). Difficulties in resisting temptation, delaying gratification, and inhibiting behavior during the preschool years have also been found to predict ADHD symptoms in third grade (Campbell & von Stauffenberg, 2009). At this age, the combination of severe ADHD-related symptoms and disruptions in the parent–child relationship is especially predictive of continuing ADHD behavior patterns (Campbell, Shaw, & Gilliom, 2000).

### *Elementary School*

**ALAN**

#### I Couldn't Do Anything Right

Toward the middle half of first grade, the teacher called my Mom in for a conference. She was telling my Mom, "I'm always having to call on Alan. 'Alan, be still. Please. Yes, you can sharpen your pencil for the third time. You have to go to the bathroom again?'" By the time I got to third grade things were getting off track. I felt like nothing I did was right. I would try to do good work. My teacher would write on my papers, "Needs to concentrate more on answers," "Needs to turn in all work," "Needs to follow directions." I really didn't think my teacher liked me. She was very stern, never seemed to smile, and was always watching me.

From R. A. Barkley and L. J. Pfiffner, "Off to School on the Right Foot: Managing Your Child's Education. In: Barkley, R. A. Taking Charge of ADHD: The Complete, Authorized Guide for Parents, 1995, p. 208.

Symptoms of inattention become especially evident when the child starts school. Classroom demands for sustained attention and goal-directed persistence are formidable challenges for these children (Kofler, Rapport, & Alderson, 2008). Not surprisingly, this is when children are usually identified as having ADHD and referred for special assistance. Symptoms of inattention continue through grade school, resulting in low academic productivity, distractibility, poor organization, trouble meeting deadlines, and an inability to follow through on social promises or commitments to peers. The hyperactive–impulsive behaviors that were present in preschool continue, with some decline, from 6 to 12 years of age (Barkley, 2006a).

During elementary school, oppositional defiant behaviors may increase or develop. By 8 to 12 years of age, defiance and hostility may take the form of serious problems, such as lying or aggression. During the school years, ADHD increasingly takes its toll, as children experience problems with self-care, personal responsibility, chores, trustworthiness, independence, social relationships, and academic performance (Stein et al., 1995).

### *Adolescence*

**ALAN**

#### A Parent's Viewpoint

It wasn't until Alan was 13 that I understood that ADHD was a lifelong condition. His inability to block out the high level of activity in junior high caused him to become a frequent visitor to the principal's office. And he began to do poorly in math, the subject he had always done well at, because he couldn't concentrate on all the steps involved. I had him thoroughly evaluated for ADHD again and discovered he wasn't outgrowing it. In fact, it was causing him more trouble, not less. It was then that I realized how ADHD shapes personality, torments the victims, and fragments relationships.

From R. A. Barkley and L. J. Pfiffner, "Off to School on the Right Foot: Managing Your Child's Education. In: Barkley, R. A. Taking Charge of ADHD: The Complete, Authorized Guide for Parents, 1995, p. 208.

Many children with ADHD do not outgrow their problems when they reach adolescence, and sometimes their problems can get much worse. Although hyperactive–impulsive behaviors decline significantly by adolescence, they still occur at a higher level than in 95% of same-age peers who do not have ADHD. The disorder continues into adolescence for at least 50% or more of clinic-referred elementary school children (Spencer, Biederman, & Mick, 2007). In addition, most teens continue to display significant impairments in their emotional, behavioral, and social functioning (Barkley, 2006b; Lee et al., 2008). Childhood symptoms of hyperactivity–impulsivity (more so thansymptoms of inattention) are generally related to poor adolescent outcomes (Barkley, 2006b).

### *Adulthood*

**ALAN**

#### Adult Challenges

Alan is now 35 years old. He frequently feels restless, cannot sit at a desk for more than a few minutes, cannot get organized, does not follow through on plans because he forgets them, loses his keys and wallet, and fails to achieve up to his potential at work. During conversations, his mind wanders and he interrupts others, blurting out whatever comes to mind without considering the consequences. He often gets into arguments. His mood swings and periodic outbursts make life difficult for those around him. Now his marriage is in trouble. He feels helpless and frustrated. (Based on authors' case material)

Although difficult to confirm, many well-known and highly successful adults, including inventor Thomas Edison, recording star and actor Justin Timberlake, celebrity Paris Hilton, and 18-time Olympic gold medal winner in swimming Michael Phelps may have had ADHD as children. Some children with ADHD either outgrow their disorder or learn to cope with it, particularly those with mild ADHD and without conduct or oppositional problems. Better outcomes are more likely for children whose symptoms are less severe and who receive good care, supervision, and support from their parents and teachers and who have access to economic and community resources, including educational, health, and mental health services (Kessler et al., 2005).

Unfortunately, like Alan, most children with ADHD will continue to experience problems, leading to a lifelong pattern of suffering and disappointment (Barkley, 2014a, b). Once thought of primarily as a disorder of childhood, ADHD is now well established as an adult disorder. Adults with ADHD are restless, easily bored, and constantly seeking novelty and excitement; they may experience work difficulties, impaired social relations, and suffer from depression, low self-concept, substance abuse, and personality disorder (Barkley, 2014a, b). Although the situation is changing, many adults with ADHD have never been diagnosed, particularly those without accompanying behavior problems. As a result, they may feel that something is wrong with them, but they don't know what it is. Since many adults with ADHD are bright and creative individuals, they often feel frustrated about not living up to their potential. Alan has

had to contend with a number of significant problems as he continues to adapt to adult responsibilities of marriage, finances, and work. The help he has received for his ADHD helps him cope, but it is an ongoing challenge.

A few words of caution are in order regarding the developmental course and outcomes for ADHD. First, age at onset, course, and outcomes may differ depending on the samples, measures used, accompanying disorders, and the presentation type of ADHD (Faraone, Biederman, & Mick, 2006; Owens et al., 2014). For example, negative outcomes are much greater in clinic samples as compared with community samples and for children with accompanying conduct problems. Little information is currently available regarding long-term outcomes for youngsters with ADHD-PI. Interestingly, ADHD symptoms and impairments in adulthood are more severe when reported by other adults than by the person with ADHD (Barkley et al., 2002). Thus, the developmental course and outcomes we have presented describe an overall pattern, but additional follow-up studies are needed to fill in important details.

## Section Summary

### Prevalence and Course

- The best estimate is that ADHD affects about 5% to 9% of all school-age children.
- The diagnosis of ADHD is about two to three times more common in boys than in girls.
- Girls with ADHD have a significant disorder; clinic-referred girls with ADHD display many of the same features and outcomes as boys with ADHD.
- ADHD occurs across all socioeconomic levels and has been identified in every country where it has been studied.
- Symptoms of ADHD change with development. A difficult temperament as an infant may be followed by hyperactive–impulsive symptoms at 3 to 4 years of age, which are followed, in turn, by the increasing visibility of symptoms of inattention around the time that the child begins school.
- Although some symptoms of ADHD may decline in prevalence and intensity as children grow older, for many individuals ADHD is a lifelong and painful disorder.

# THEORIES AND CAUSES

*The word cause is an altar to an unknown god.*

—William James (1842–1910)

Many explanations for ADHD have been advanced, and some are highly controversial. For example, it has been argued (without much support) that ADHD is a trait left over from our evolutionary past as hunters (Hartmann, 1993). Others contend that ADHD is a myth, a disorder that has been fabricated because as a society we need it (Baughman, 2006; Breggin, 2001).

Clear answers about the nature and causes of ADHD have been elusive because diagnostic practices are not standardized and research is challenging. Nevertheless, research into the basic nature of ADHD leads to fascinating theories about possible mechanisms and causes (Martel, 2009; Nigg & Barkley, 2014). As summarized in A Closer Look 8.1, theories

## A CLOSER LOOK 8.1

### Interrelated Theories of ADHD

**Cognitive Functioning Deficits**

Children with ADHD display specific cognitive deficits in sustained attention, response inhibition (i.e., inability to delay initial reactions to events or to stop behavior once it gets going), working memory, and executive functions. These in turn may lead to other cognitive, language, and motor difficulties. Cognitive deficits are important for understanding ADHD. However, since more than 50% of children with ADHD do not show major impairment on any specific cognitive task, the evidence does not support a *single* cognitive deficit as the cause of ADHD (Nigg, 2005).

**Reward/Motivation Deficits**

Children with ADHD display an abnormal sensitivity to rewards (i.e., higher reward threshold) and usually a heightened sensitivity or an aversion to delay (Sonuga-Barke et al., 2008). As a result, they have difficulty motivating themselves and performing well when rewards are unavailable or delayed (Aase & Sagvolden, 2006). In support of this theory, some research has connected ADHD with disruptions in the dopamine reward pathways of the brain (Volkow et al., 2009).

**Arousal Level Deficits**

Children with ADHD have an abnormal level of arousal—either too high or, more commonly, too low. Hyperactivity–impulsivity reflects an underaroused child's effort to maintain an optimal level of arousal by excessive self-stimulation (Zentall, 1985). Although this theory has received some support (Antrop et al., 2000), it has not yet been presented as a comprehensive model to account for the full range of problems found in children with ADHD.

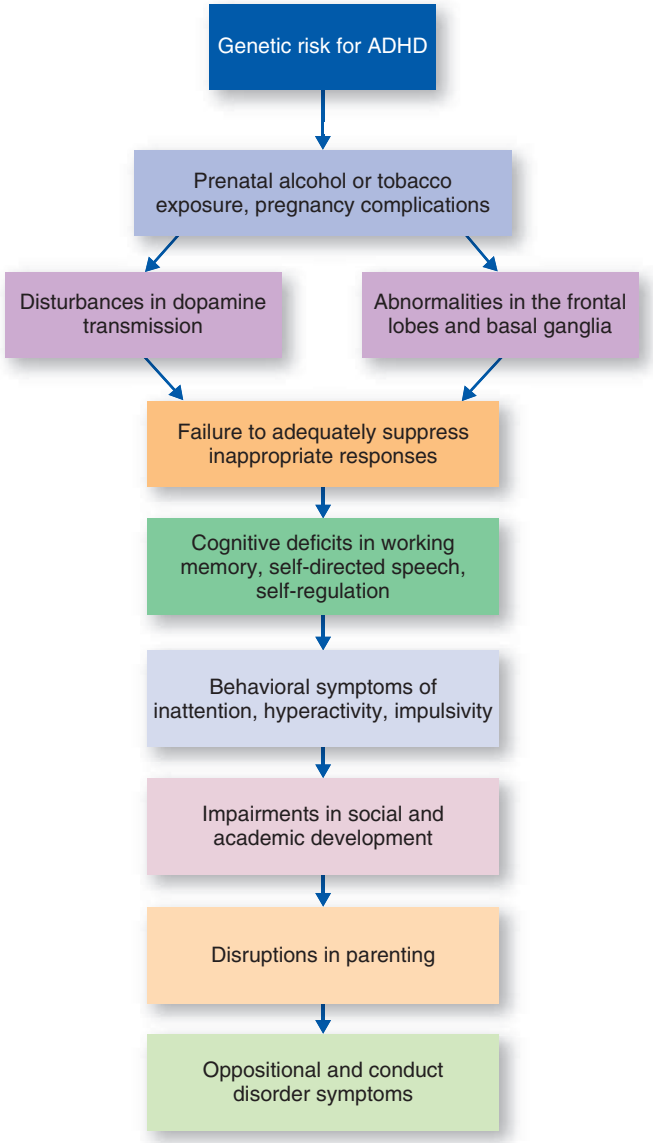
**Self-Regulation Deficits**

Children with ADHD have a higher-order deficit in their ability to self-regulate—to use thought and language to direct their behavior. Deficiencies in self-regulation and effortful control lead to impulsivity, poor maintenance of effort, poor modulation of arousal level, emotion dysregulation, and attraction to immediate rewards. Self-regulatory theories examine the interplay among cognitive, arousal, and reward/motivational processes to understand how individuals with ADHD regulate their behavior in specific contexts (Douglas, 1999; Martel, 2009).

emphasize deficits in cognitive functioning, reward and motivation, arousal level, and self-regulation. No single theory can explain the many difficulties associated with ADHD. For example, despite having similar levels of ADHD symptoms, children with ADHD may show differences in the kinds of problems they experience related to response inhibition, arousal, and response variability (Fair et al., 2012). However, identifying the influences of the processes emphasized by each theory helps increase our understanding of ADHD and develop more integrative models (Shiels & Hawk, 2010).

Numerous causes for ADHD have been proposed. However, many have not been adequately tested or have fallen by the wayside in the face of weak, inconsistent, or nonexistent support. Among these are that ADHD is caused by too much sugar intake, yeast, fluorescent lighting, motion sickness, bad parenting, poor school environment, urban living, or too much TV.

While many factors may lead to ADHD, current research strongly suggests that ADHD is a neurodevelopmental disorder for which genetic and neurobiological factors play a central role (Faraone, 2014; Kieling, Gonclaves, Tannock, & Castellanos, 2008; Scasselatti et al., 2012). However, biological and environmental risk factors together shape the development of behavioral and emotional regulation and the expression of ADHD symptoms over time, following several different pathways (Froehlich et al., 2011; Nigg et al., 2006; Sonuga-Barke et al., 2005). Since ADHD is a complex and chronic disorder involving genetic, neural, cognitive, and behavioral mechanisms, any explanation that focuses on just one cause is likely to be inadequate (Banerjee, Middleton, & Faraone, 2007; Coghill et al., 2005). Although data do not yet permit a comprehensive causal model, ● Figure 8.2 shows a possible developmental pathway for ADHD that highlights several known causal influences and outcomes. We discuss each of these causal influences in the sections that follow.






● **FIGURE 8.2** | A possible developmental pathway for ADHD.

## Genetic Influences

Several lines of evidence point to genetic influences as key causal factors in ADHD (Faraone & Mick, 2010).

- *ADHD runs in families*. About one-third of the biological relatives of children with ADHD also have the disorder (Smalley et al., 2000). Remarkably, if a parent has ADHD, the risk to their child is nearly 60% (Biederman et al., 1995).
- *Adoption studies*. Rates of ADHD are nearly three times higher in biological parents of children with ADHD, as compared with adoptive parents of children with ADHD (Sprich et al., 2000).
- *Twin studies*. Twin studies report extraordinarily high heritability estimates for ADHD, averaging about 75% for hyperactive–impulsive and inattentive behaviors, making ADHD among the most heritable of the childhood disorders (Nikolas & Burt, 2010). Further, ADHD concordance rates for identical twins average 65%, about twice that for fraternal twins (Levy & Hay, 2001).
- *Specific gene studies*. Molecular genetic analysis suggests that specific genes may contribute to the expression of ADHD (Banaschewski et al., 2010).

The focus has been on genes involved in dopamine regulation, for three primary reasons. First, dopamine is a neurotransmitter used by the brain and has a central role in psychomotor activity and reward seeking. Second, brain structures implicated in ADHD (see below) are rich with dopamine innervation, and neuroimaging studies have found evidence for dopamine dysregulation in these brain structures (Spencer et al., 2007). Third, primary medications that reduce ADHD symptoms act primarily by blocking the dopamine transporter (DAT1), a receptor on the presynaptic neuron involved in the re-uptake of dopamine, thereby increasing the availability of dopamine in the synapse. Research findings on the association between variants in DAT1 and ADHD have been mixed (Li et al., 2006). However, this may be related to a gene–environment interaction. For example, children with this genetic risk who are also exposed to environmental risks such as psychosocial adversity or maternal smoking during pregnancy may develop a greater number of ADHD symptoms than those who are not exposed to the environmental risk (Laucht et al., 2007).

Studies consistently show an association between ADHD and a variant of one of the dopamine receptor genes, DRD4 (seven-repeat form). Interestingly, this gene has previously been linked to the personality trait of sensation seeking (high levels of thrill seeking, impulsive, exploratory, and excitable behavior) (Ebstein et al., 1996); it affects responsiveness to medication; and it impacts parts of the brain associated with executive functions and attention. In addition to the association between ADHD and the DRD4 dopamine receptor gene, several other genes related to the regulation of dopamine, as well as noradrenaline, have recently been identified (Faraone & Mick, 2010; Nikolas et al., 2010).

Findings that implicate specific genes within the dopamine system in ADHD are intriguing, and they are consistent with a model suggesting that reduced dopaminergic activity may be related to the behavioral symptoms of ADHD. For example, preliminary findings indicate that brain activity related to inhibitory control of behavior in children with ADHD may differ as a function of variations in specific genes within the dopamine system (Bédard et al., 2010). Keep in mind, however, that the effects of individual gene variants are very small and—in the vast majority of cases—the heritable components of ADHD are likely to be the result of multiple genes interacting on several different chromosomes (Neale et al., 2010). For example, associations have also been found between ADHD and genes within the serotonin system (Guimarães et al., 2009), and gene mutations that impair maternal serotonin production during pregnancy may increase the risk of ADHD-related symptoms in offspring (Halmøy et al., 2010). Some work has also implicated genes associated with serotonin function in aversion to reward delay, suggesting that different genes may regulate different ADHD behaviors (Sonuga-Barke et al., 2011b). Thus, it is extremely unlikely that ADHD is caused by only one gene. As Kates (2007) noted, "Finding a gene mutation that transmits risk for ADHD is like looking for a needle in a haystack of the 3 billion base pairs of DNA that constitute the human genome" (p. 547). Nevertheless, taken together, the findings from family, adoption, twin, and specific gene studies strongly indicate that the risk for ADHD is inherited, although the precise mechanisms are not yet known (Mick et al., 2010). Findings from genomewide association studies are just beginning to reveal the specific neurodevelopmental networks that may be involved in ADHD (Poelmans et al., 2011). To date, these findings indicate that many common DNA variants combine to create a genetic predisposition to ADHD that accounts for about 20% to 30% of the disorder's heritability (Smoller et al., 2003; Yang et al., 2013). These findings from genomewide studies do not specify the particular DNA or variants that make up the predisposition to ADHD but do suggest that such variants will likely be found as more data are collected (Faraone, 2014). Importantly, the more we discover about the genetics of ADHD, the more evident it becomes that environmental factors play a powerful role in shaping neurodevelopmental processes and pathways. Therefore, the role of environment will need to be incorporated into any explanation of ADHD based on genetic influences (Sonuga-Barke, 2010).

## Pregnancy, Birth, and Early Development

Many factors that can compromise the development of the nervous system before and after birth may be related to ADHD symptoms, including pregnancy and birth complications, maternal exposure to environmental toxins or severe stress during pregnancy, low birth weight, malnutrition, early neurological insult or trauma, and diseases of infancy (Lindström, Lindblad, & Hjern, 2011; Linnet et al., 2003; Martel et al., 2007). Although these early factors predict later symptoms of ADHD, they may not be specific to ADHD; that is, they may elevate the risk of developing many later problems in addition to ADHD.

A mother's use of cigarettes, alcohol, or other drugs during pregnancy can have damaging effects on her unborn child. One study found that mothers of children with ADHD reported higher rates of heavy smoking

(10 cigarettes per day) and more psychosocial stress during pregnancy as compared with mothers of unaffected control children (Motlagh et al., 2010). Consistent support has been found for an association between maternal cigarette smoking during pregnancy and ADHD, particularly for female offspring, and for children who carry a specific genetic risk for ADHD (Braun et al., 2006; Neuman et al., 2007). Whether this association is causal or due to other unmeasured environmental or genetic factors is unclear (Obel et al., 2011; Thapar et al., 2009). Evidence suggests that most (but not necessarily all) of the association between maternal cigarette smoking during pregnancy and ADHD is due to genetic mediation, not to the direct effects of nicotine exposure in the womb (Rutter & Solantaus, 2014). Exposure to alcohol before birth may also lead to symptoms of inattention, hyperactivity, impulsivity, and associated impairments in learning and behavior (Mick et al., 2002).

Evidence suggests that mothers of children with ADHD use more alcohol, tobacco, and drugs than do control parents, even when they are not pregnant (Mick et al., 2002). Since parental substance use is often associated with a chaotic home environment both before and after birth, it is difficult to disentangle the influence of substance abuse and other factors that occur prior to birth from the cumulative impact of a negative family environment that occurs during later development. Other substances used during pregnancy, such as cocaine, can adversely affect the normal development of the brain and lead to higher-than-normal rates of ADHD and other psychiatric disorders (Weissman et al., 1999). To summarize, although pregnancy and birth complications and substance use during pregnancy are not the cause of most cases of ADHD, they may be important contributing factors for some children.

## Neurobiological Factors

ADHD is far from well understood, but there is substantial support for neurobiological causal factors (Krain & Castellanos, 2006). Evidence comes from research showing differences between children with and without ADHD on measures of brain function (Barkley, 2006a; Kieling et al., 2008), including:

- Differences on psychophysiological measures (e.g., electroencephalography, galvanic skin response, and heart-rate deceleration), suggesting diminished arousal or arousability (Beauchaine et al., 2001; Loo & Makieg, 2012).
- Differences on measures of brain activity during vigilance tests, suggesting underresponsiveness to stimuli and deficits in response inhibition (Pliszka, Liotti, & Woldorff, 2000).
- Differences in blood flow to the prefrontal regions of the brain and the pathways connecting these regions to the limbic system and cerebellum, suggesting decreased blood flow to these regions (Hendren, De Backer, & Pandina, 2000).

### *Brain Abnormalities*

Brain-imaging studies make it possible to test hypotheses about the location of brain dysfunction in ADHD and to provide assessments of brain structure and function (Cortese et al., 2012). Findings from these studies suggest abnormalities primarily in the **frontostriatal circuitry of the brain** (Bush, 2008). This region consists of the prefrontal cortex and interconnected areas of gray matter located deep below the cerebral cortex, collectively known as the *basal ganglia* (basement structures) (Hart et al., 2013). These areas of the brain are associated with attention, executive functions, delayed responding, and response organization. Lesions in this region result in symptoms similar to those of ADHD. Children with ADHD have a smaller right prefrontal cortex than children without ADHD (Filipek et al., 1997) and have structural abnormalities in several regions of the basal ganglia (Sobel et al., 2010). Interestingly, in identical twins discordant for ADHD, it is only the twin with ADHD who displays abnormalities in these brain structures (Castellanos et al., 2003).

Brain differences may not be restricted exclusively to the prefrontal cortex and parts of the basal ganglia. Some findings indicate that children with ADHD have smaller total and right cerebral volumes (by 3% to 4%) and a smaller cerebellum. These data provide support for the growing notion that some deficits associated with ADHD, such as learning temporal associations and events and their consequences, may involve a cerebellar–prefrontal–striatal network (Mackie et al., 2007; Valera et al., 2007). Some work also suggests that specific regions of the thalamus may also be involved in the brain circuitry of ADHD, with different thalamic subcircuits associated with differing ADHD symptoms linked with the regulation of motor and emotional responses (Li et al., 2012). The default mode network (discussed in Chapter 6) also operates abnormally in ADHD. This network tends to be active at rest but tends to shut off during task engagement—but not as effectively in those with ADHD (Cortese et al., 2012).

In addition to identifying brain abnormalities, brain-scan studies now focus on how brain connections and networks are wired during development. Newer studies suggest that circuits may develop

differently or later in ADHD (Fair et al., 2010; Shaw et al., 2007). For example, one study (see A Closer Look 8.2) identified a delay in brain maturation in children with ADHD, particularly in the prefrontal regions (Shaw et al., 2007). Another study found differences in brain development between individuals with ADHD whose symptoms persisted into adulthood and those whose symptoms did not persist. Those whose symptoms persisted showed an increase in cortical thinning in areas of the prefrontal cortex. In contrast, only individuals whose ADHD remitted showed a trajectory of cortical thickening or minimal thinning (Shaw et al., 2013). These findings help to advance our understanding of developmental pathways to adult ADHD.

In summary, brain-scan studies indicate the importance of the frontostriatal region of the brain in ADHD and the pathways connecting this region with the limbic system (via the striatum) and the cerebellum and thalamus. However, further research is required to reliably identify more localized abnormalities. Furthermore, although neuroimaging studies can tell us that children with ADHD have a structural difference or less activity in certain regions of the brain, they don't tell us why.

A few additional words of caution are needed when evaluating the neurobiological evidence for ADHD. First, samples have been defined differently across studies and sample sizes are generally small. This gives rise to inconsistent findings and presents problems

## A CLOSER **LOOK 8.2**

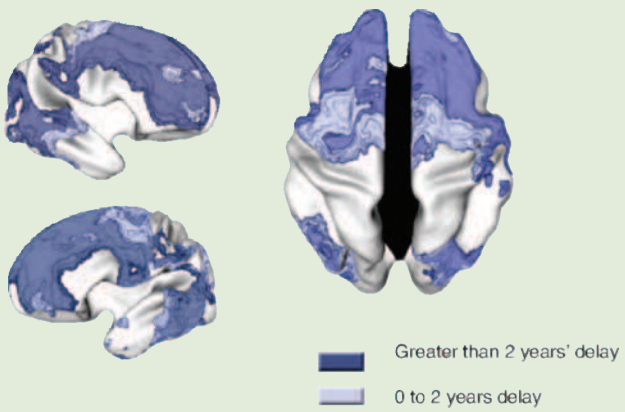
### Does the Brain Develop Abnormally in Children with ADHD, or Is It Just Delayed?

Findings from brain-imaging studies suggest the latter. Dr. Philip Shaw and his colleagues at the National Institute of Mental Health studied a large group of children with ADHD and a comparison group of typically developing children at different ages (Shaw et al., 2007). Using magnetic resonance imaging (MRI), the researchers tracked changes in the cortical thickness of the children's brains. Typically, the cortex reaches a peak thickness at around 7 or 8 years of age. However, as shown in the accompanying photograph, in children with ADHD, there was about a 2- to 3-year delay in reaching this peak thickness as compared with typically developing controls. The greatest delay (about 5 years) was in the prefrontal cortex, which regulates self-control.

The overall pattern of brain development was the same for both groups, suggesting a developmental delay and not abnormal development in children with ADHD. The brains of children with ADHD did reach one developmental milestone earlier than did the brains of typically developing children—maturation of the motor cortex, which plans and controls movements. The researchers proposed that the delay in the development of areas of the cortex related to self-control, combined with the earlier development of the motor cortex, may account for the fidgety, restless, and uncontrolled hyperactive behavior of children with ADHD.

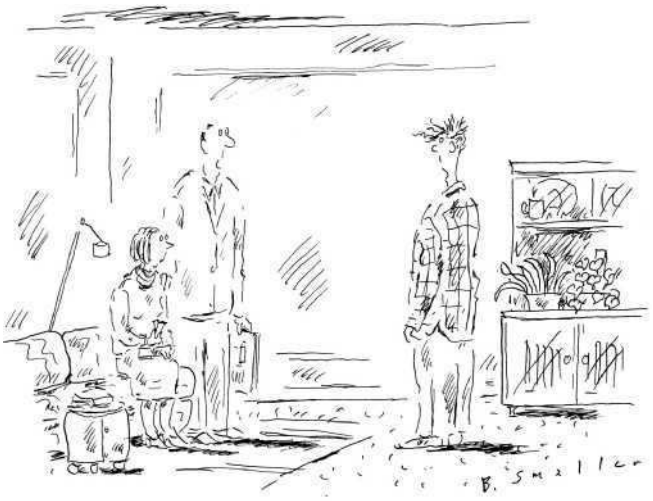
Interestingly, other brain-imaging studies have found that delayed cortical maturation in the prefrontal cortex is associated with a greater number of symptoms of hyperactivity–impulsivity, even in typically developing children who have not been diagnosed with ADHD (Shaw et al., 2011).

Although these brain-scan findings are captivating, they raise other important issues. First, the cause of the maturational delay in children with ADHD is not known at this time, although some work suggests that delayed or decreased myelination may be a factor (Nagel et al., 2011). Second, if ADHD is just a delay in development, why do so many children continue to display ADHD as adults? Delay alone may not fully characterize all children with ADHD. In addition, even when brain maturation "catches up," the expression of behavior may still be different, since the timing of brain maturation may be as important as its end point (Fair et al., 2010). Answers to these questions await further research. The hope is that brain-imaging studies will increase our understanding of how ADHD develops and in doing so may one day help to tailor treatments for children with ADHD. (Based on authors' case material.)




Regions where the ADHD group had delayed cortical maturation, as indicated by an older age of attaining peak cortical thickness

From Proceedings of the National Academy of Sciences, "Attention-deficit/hyperactivity disorder is characterized by a delay in cortical maturation," edited by Leslie G. Ungerleider, National Institutes of Health, Bethesda, MD, and approved October 5, 2007, © 2007 by The National Academy of Sciences of the USA.



"Young man, go to your room and stay there until your cerebral cortex matures."

Barbara Smaller/The New Yorker Collection/cartoonbank.com

with interpretation and generalization. Second, ADHD includes a complex set of symptoms, making the search for a single neurobiological cause too simplistic. Third, the differences in brain structure and activity seen in ADHD (and other disorders) are usually meaningful only when comparing group statistics. The amount of variation in brain structure or function from one brain to the next is simply too great to be diagnostic or predictive for an individual, given what we currently know (Insel, 2010). Thus, although research using brain imaging has greatly increased our understanding of ADHD, much research remains to be done before neuroimaging procedures can be integrated into clinical practice (Bush, 2008).

### *Neurophysiological and Neurochemical Findings*

Precise neurophysiological and neurochemical abnormalities underlying ADHD have been extremely difficult to document (Scassellati et al., 2012). No consistent differences have been found between the biochemistry of urine, plasma, blood, and cerebrospinal fluid of children with and without ADHD. At a neurochemical level, the known action of effective medications for ADHD suggests that the neurotransmitters dopamine, norepinephrine, epinephrine, and serotonin may be involved, with most evidence suggesting a selective deficiency in the availability of both dopamine and norepinephrine (Barkley, 2006a). However, we must be cautious in drawing conclusions from the effects of medications alone. Using medication for effective treatment of ADHD symptoms does not prove that deficits in the chemical or its action are the cause of the symptoms, any more than the elimination of a headache by aspirin implies that the headache was caused by aspirin deficiency. Medications may operate at levels of neuroanatomy and neurochemistry that are far removed from the primary causal influences.

## Diet, Allergy, and Lead

The connection between sugar and hyperactivity achieved epic significance when "What is sugar?" was the correct question for the answer "The major cause of hyperactivity in North America" on the popular TV show *Jeopardy* (Barkley, 1995). However, study after study has conclusively shown that sugar is not the cause of hyperactivity (Milich, Wolraich, & Lindgren, 1986). So why do nearly half of the parents and teachers who are asked think that children are sugar-sensitive? It may be the power of suggestion.

In one study, mothers who believed their children were sugar-sensitive were told that their children would be given a drink of Kool-Aid™ containing either sugar or, as a placebo, the sugar substitute aspartame. After the children drank their Kool-Aid, they and their mothers spent time playing and working together. In fact, none of the children was given sugar—they all received Kool-Aid with aspartame. But the mothers who thought their children had received sugar rated them as more hyperactive than the mothers who believed their children had received aspartame. Perhaps even more telling was that during play and task interactions, mothers who thought their children had received sugar were more critical of them, hovered more, and talked to them more frequently (Hoover & Milich, 1994). These findings suggest that what parents believe about the causes of their children's ADHD can affect their views of their children and how they treat them (Johnston & Leung, 2001). Such beliefs may be fueled by media portrayals—for example, when Bobby Hill, the son on the TV show *King of the Hill,* is diagnosed with ADHD after eating at least four bowls of "Cookie Crunch" cereal (the last with extra sugar!). His mother, Peggy, "a hotshot substitute teacher," wonders if she should stay at home more often so she can give Bobby more attention, a notion reinforced by Bobby's father, Hank, who comments, "Well, it is called attention *deficit* disorder" (Kennedy, 2008).

There has been a long-time controversy about the possibility that allergic reactions and diet cause ADHD. A popular view in the 1970s and 1980s was that food additives caused children to be hyperactive and inattentive, and parents were encouraged to withhold foods containing artificial flavorings, colorings, preservatives, and sugars. By the 1990s, food additives appeared to have been discarded as an explanation for ADHD (McGee, Stanton, & Sears, 1993). However, some studies have revived this idea, focusing on the moderating role of genetic factors to explain why food additives may affect the behavior of

some children more than that of others (McCann et al., 2007; Stevenson, 2010; Stevenson et al., 2010). In a close vote (8 to 6), a Food and Drug Administration Advisory Panel (March 31, 2011) concluded that foods that contain artificial dyes do not need warning labels, but this issue continues to be debated. Dietary research on ADHD is now focusing on micronutrients, with much interest currently in essential fatty acids—which are commonly lacking in the diet of North American children—as well as in other nutrients such as zinc and iron, which may be metabolized abnormally in some children (Arnold & DiSilvestro, 2005; Howard et al., 2011; Stevens et al., 2003).

Exposure to low levels of lead found in dust, water, soil, and flaking paint in areas where leaded gasoline and paint were once used may be associated with ADHD symptoms in the classroom (Fergusson, Horwood, & Lynskey, 1993). Although most children with ADHD do not have significantly elevated lead levels in their teeth or blood (Kahn, Kelly, & Walker, 1995), some work persistently links ADHD to slight, subclinical elevations in lead exposure (Goodlad, Marcus, & Fulton, 2013). All children have a little lead in their blood, and those with ADHD have a little more. In addition, lead exposure in combination with other risk factors, such as exposure to nicotine during pregnancy, may further increase a child's risk for ADHD (Froehlich et al., 2009). These findings suggest a possible role of lead exposure that requires further follow-up using causally informative prospective studies (Nigg et al., 2010).

## Family Influences

Twin studies find that psychosocial factors in the family account for only a small amount of the variance in ADHD symptoms (Nikolas & Burt, 2010), and explanations for ADHD based exclusively on negative family influences have received little support (Barkley, 2006a). Nevertheless, family influences are important in understanding ADHD for several reasons (Johnston & Chronis-Tuscano, 2014; Mash & Johnston, 2005).

- *Family influences may lead to ADHD symptoms or to a greater severity of symptoms*. In some cases, ADHD symptoms may be the result of interfering and insensitive early caregiving practices (Carlson, Jacobvitz, & Sroufe, 1995), especially in children with a specific genetic risk for ADHD (Martel et al., 2011). Thus, parenting practices may interact with the child's genetic makeup to moderate risk for ADHD. In addition, for children at risk for ADHD, family conflict may raise the severity of their hyperactive–impulsive symptoms to a clinical level (Barkley, 2003). Especially important is the **goodness of fit**, or the match between the child's early temperament and the parent's style of interaction (Chess & Thomas, 1984). An overactive child with an overstimulating parent is a seemingly poor fit. As we have seen, many parents of children with ADHD also have the disorder, which means that the parents' ADHD symptoms may disrupt early parent–child interactions (Johnston et al., 2012). For example, mothers with higher levels of ADHD symptoms have been found to show less involvement, less positive parenting, and more inconsistent discipline with their children than mothers with lower levels of ADHD symptoms (Chronis-Tuscano et al., 2008). Finally, one study found that mothers with variants in DAT1 were more like to display negative and controlling behaviors when interacting with their children, particularly when their children were highly disruptive (Lee et al., 2010). Although preliminary, these findings are fascinating, in that they suggest the possible importance of a parent-gene × child-gene interaction in families of children with ADHD, with the effects mediated by the child-rearing environment.
- *Family problems may result from interacting with a child who is impulsive and difficult to manage* (Mash & Johnston, 1990). The clearest support for this child-to-parent direction of effect comes from double-blind placebo-controlled drug studies in which children with ADHD who received stimulant medications showed a decrease in their symptoms. The decreases in children's ADHD symptoms produced a corresponding reduction in the negative and controlling behaviors that parents had previously displayed when their children were not medicated (Barkley, 1988).
- *Family conflict is likely related to the presence, persistence, or later emergence of associated oppositional and conduct disorder symptoms*. In children with an inherited biological risk for ADHD, family conflict may heighten the emergence of early ODD and later comorbid ADHD and CD (Beauchaine et al., 2010). For example, children with ADHD report observing more interparental conflict than do children without ADHD, which may worsen ADHD and related ODD and CD symptoms in those who have a genotype that makes them particularly vulnerable to the effects of the emotional stress and self-blame associated with interparental conflict (Nikolas et al., 2010). Many ADHD interventions aim to change patterns of family interaction to prevent an escalating cycle of oppositional behavior and conflict. Family influences may play a major role in determining the outcome of ADHD and its associated problems, even if the influences are not the primary

cause of ADHD (Johnston, Hommersen, & Seipp, 2009; Kaiser, McBurnett, & Pfiffner, 2011).

In summary, ADHD has a strong biological basis and is an inherited condition for many children. It is likely that ADHD is a heterogeneous disorder, particularly at the level of neurobiology. Although evidence is converging on specific brain areas and circuits, findings are correlational, and we do not yet know the specific causes of the disorder. We are just beginning to understand the complex ways in which biological risk factors, brain development, early environmental experiences and events, family relationships, and broader system influences interact to shape the development and outcome of ADHD (Nigg & Barkley, 2014).

## Section Summary

### Theories and Causes

- Theories about possible mechanisms and causes for ADHD have emphasized deficits in cognitive functioning, reward/motivation, arousal level, and self-regulation.
- There is strong evidence that ADHD is a neurodevelopmental disorder; however, biological and environmental risk factors together shape its expression.
- Findings from family, adoption, twin, and specific gene studies suggest that ADHD is inherited, although the precise mechanisms are not yet known.
- Many factors that compromise the development of the nervous system before and after birth may be related to ADHD symptoms, such as pregnancy and birth complications, maternal smoking during pregnancy, low birth weight, malnutrition, maternal alcohol or drug use, early neurological insult or trauma, and diseases of infancy.
- ADHD appears to be related to abnormalities and developmental delays in the frontostriatal circuitry of the brain and the pathways connecting this region with the limbic system, the cerebellum, and the thalamus.
- Neuroimaging studies tell us that in children with ADHD there is a structural difference or less activity in certain regions of the brain, but they don't tell us why.
- The known action of effective medications for ADHD suggests that several neurotransmitters are involved, with most evidence suggesting a selective deficiency in the availability of both dopamine and norepinephrine.
- Psychosocial factors in the family do not typically cause ADHD, although they are important in understanding the disorder. Family problems may lead to a greater severity of symptoms and relate to the emergence of co-occurring conduct problems.
- ADHD is likely the result of a complex pattern of interacting influences, perhaps giving rise to the disorder through several nervous system pathways.

## TREATMENT

### MARK

### Medication and Behavior Therapy

In third grade, Mark's teacher threw up her hands and said, "Enough!" In one morning, Mark had jumped out of his seat six times to sharpen his pencil, each time accidentally charging into other children's desks and toppling books and papers. He was finally sent to the principal's office when he began kicking a desk he had overturned. In sheer frustration, his teacher called a meeting with his parents and the school psychologist.

But even after they developed a plan for managing his behavior in class, Mark showed little improvement. Finally, after an extensive assessment, they found that he had ADHD with symptoms of both inattention and hyperactivity–impulsivity. He was put on Ritalin, a stimulant medication, to control the hyperactivity during school hours. With a psychologist's help, his parents learned to reward desirable behaviors and to have Mark take time out when he became too disruptive. Soon Mark was able to sit still and focus on learning.

Adapted from NIMH, 1994a.

### LISA

### Behavior Therapy and Counseling

Because Lisa wasn't disruptive in class, it took a long time for teachers to notice her problem. Lisa was first referred to the school evaluation team when her teacher realized that she was a bright girl with failing grades. The team ruled out a learning disability but determined that she had the inattentive subtype of ADHD. The school psychologist recognized that Lisa was also dealing with depression.

Lisa's teachers and the school psychologist developed a treatment plan that included a program to increase her attention and develop her social skills. They also recommended that Lisa receive counseling and cognitive behavior therapy to help her recognize her strengths and overcome her depression.

Adapted from NIMH, 1994.

In recent years, the number of children with ADHD receiving help has more than doubled. However, it is still the case that less than half, particularly those in greatest clinical need, actually receive specialty services for ADHD (Visser et al., 2013; Zima et al., 2010). Of those who do receive services, many do not continue their

treatment for any length of time (Hechtman, 2006). Although there is no known cure for ADHD, a variety of treatments can be used to help children like Mark and Lisa cope with their symptoms and any secondary problems that may arise (Antshel & Barkley, 2008; Pelham & Fabiano, 2008). An overview of these treatments is presented in Table 8.3.

The primary treatment approach, as recommended by *Consumer Reports* and the U.S. Surgeon General, combines stimulant medication, parent management training, and educational intervention (AACAP, 2007c; Kaiser & Pfiffner, 2011; NICE, 2013a). Interventions that use elements of all these approaches have also been provided in intensive summer treatment programs and controlled clinical trials (Smith & Shapiro, 2014). Additional treatments, for which there is far less evidence, include family counseling and support groups and child-focused treatments, such as individual counseling and traditional forms of social skills training (Mikami, 2014; Smith, Barkley, & Shapiro, 2006). Other treatments, such as brain-wave neurofeedback/biofeedback (Lofthouse et al., 2011) and complementary and alternative biomedical treatments, such as special diets (e.g., restricted elimination diets, artificial food color elimination) and vitamin/mineral supplements (e.g., supplementation with free fatty acids) (Hurt & Arnold, 2014; Millichap & Lee, 2012) are used despite limited or inconsistent supporting evidence to date (Sonuga-Barke et al., 2013; Vollebregt et al., 2013). However, given that some studies have reported modest beneficial effects for both neurofeedback and dietary treatments for a small proportion of children with ADHD, further evaluation of these approaches seems warranted (Gevensleben et al., 2014; Nigg et al., 2012; Stevenson et al., 2014).

**TABLE 8.3** | **Treatments for Children with ADHD**

| **Primary Treatments** | **Focus of Treatment** |
|---|---|
| Stimulant medication | Managing ADHD symptoms at school and home |
| Parent management training | Managing disruptive child behavior at home, reducing parent–child conflict, and promoting prosocial and self-regulating behaviors |
| Educational intervention | Managing disruptive classroom behavior, improving academic performance, teaching prosocial and self-regulating behaviors |
| **Intensive Treatment** | **Focus of Treatment** |
| Summer treatment programs | Enhancing present adjustment at home and future success at school by combining many of the primary and additional treatments in an intensive summer treatment program |
| **Additional Treatments** | **Focus of Treatment** |
| Family counseling | Coping with individual and family stresses associated with ADHD, including mood disturbance and marital strain |
| Support groups | Connecting adults with other parents of children with ADHD, sharing information and experiences about common concerns, and providing emotional support |
| Individual counseling | Providing a supportive relationship in which the youth can discuss personal concerns and feelings |



A rationale and procedures for early detection and early intervention for ADHD are now beginning to emerge (Sonuga-Barke et al., 2011a). However, one 6-year follow-up study of 207 children 3 to 5 years old who had ADHD who were treated with stimulant medication found little improvement, suggesting that further study of moderating variables (e.g., co-occurring behavior problems, family adversity) and other interventions for this age group may be needed (Riddle et al., 2013). Focused interventions for specific ADHD core deficits—for example, working memory (Beck et al., 2010; Rapport et al., 2014), inattention (Rabiner et al., 2010), self-control (Diamond, 2012), and organization, time-management, and planning (Abikoff et al., 2013) are also receiving attention (Evans, Owens, & Bunford, 2013). Further work is needed to determine whether these approaches produce changes that are long-lasting and generalize to other tasks and situations beyond the training context (Dunning, Holmes, & Gathercole, 2013).

Although similar treatments are used for children and adolescents with ADHD, research with teens has been extremely limited (Barkley, 2006b). Similarly, only a few studies have looked at treatment efficacy for specific ADHD presentation types, such as children with predominantly inattentive symptoms (e.g., Pfiffner et al., 2007). Therefore, although recognizing that children with different ADHD symptoms may have different treatment needs (Solanto et al., 2009), most of our discussion centers on treatments for school-age children with ADHD who show symptoms of both inattention and hyperactivity–impulsivity.

## Medication

*When I'm not medicated … my muscles just don't want to relax. They don't want to settle down. I just feel very "go-go-go." I can't be still. My body tries to keep up with my mind but it can't just do it fast enough.*

—Angelique (Waite & Ramsey, 2010, p. 429)

The use of stimulants and other medications to treat the symptoms of ADHD in children has been the subject of considerable and heated debate. Most of you likely have opinions about stimulants for ADHD—perhaps that they are overprescribed, used as a quick fix, do not let kids be kids, lead to overdiagnosis, or lead to later substance use. Let's look at the role of stimulant medications and the controversy that surrounds them.

### *Stimulants*

**Stimulant medications** have been used to treat the symptoms of ADHD since the chance discovery of their effectiveness in the 1930s (see A Closer Look 8.3).

A CLOSER **LOOK 8.3**

#### The "Accidental" Discovery of Math Pills

The use of stimulant medication for children with learning and behavior problems was first reported in 1937 by Charles Bradley, the medical director of a small hospital for children with major difficulties in learning or behavior. Dr. Bradley described dramatic improvements in some of the children he treated with Benzedrine. Why did Dr. Bradley decide to use stimulants to treat these problems in the first place?

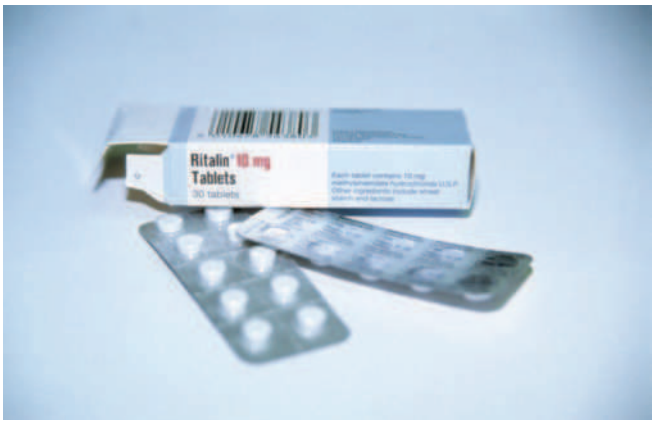
Dr. Bradley was a very conscientious physician, and all patients were given careful workups. These workups included a spinal tap, which naturally led to headaches afterward that frequently were lasting, severe, or both, and were presumed to be due to the loss of spinal fluid. Dr. Bradley speculated that if he could stimulate the choroid plexus to secrete spinal fluid at a faster rate, the headaches would be relieved more quickly. He decided to proceed along these lines and chose the most potent stimulant available at the time, Benzedrine. (*Note:* This type of powerful stimulant is no longer used.) The effect on the headaches was negligible, but to his astonishment, the teachers reported major improvements in learning and behavior in many children that lasted until the Benzedrine regimen was withdrawn. The children themselves noted the greater ease of learning and called the medication "math pills," presumably because mathematics was the hardest subject for them, and their improved ability to learn was most noticeable in that subject.

*Source:* Based on Gross, 1995.

Several other types of medications have also been used, including noradrenergic drugs, antidepressants, and antihypertensives (Daughton & Kratochvil, 2009). We focus our discussion on stimulants because they are the most studied, most effective, and most commonly used treatment for the management of symptoms of ADHD and its associated impairments. Nevertheless, it is important to keep in mind that many other potentially effective medication options are available for children with ADHD, including nonstimulant drugs such as atomoxetine (Strattera) and a patch that releases stimulant medication directly through the skin into the bloodstream, eliminating the need to take medication orally (Pelham et al., 2011). Other medications may be used for children who do not respond well to stimulants, cannot tolerate the side effects of stimulants, or have other conditions along with ADHD (Connor, 2014).

Stimulants come in several types, including both short- and long-acting forms. Among the most effective stimulants in treating individuals with ADHD are dextroamphetamine (Dexedrine or Dextrostat), amphetamine-dextroamphetamine (Adderall), and **methylphenidate** (Ritalin), the most commonly used drug by far (Faraone & Buitelaar, 2010). These medications alter activity in the frontostriatal region of the brain by affecting neurotransmitters (dopamine) important to this region. Interestingly, preliminary findings from brain-scan studies of individuals with ADHD suggest that stimulants may also help to normalize structural abnormalities and functional connections within this region (Sheridan, Hinshaw, & D'Esposito, 2010; Sobel et al., 2010).

For about 80% of children with ADHD, stimulants produce dramatic increases in sustained attention, impulse control, and persistence of work effort and decreases in task-irrelevant activity and noisy and disruptive behaviors. Stimulants may also improve the child's academic productivity; cooperation and social interactions with parents, teachers, and peers; and, occasionally, physical coordination such as handwriting or sports ability (Barkley, 2006a; Prasad et al., 2013). Stimulant medications used appropriately and with proper supervision are usually quite safe (Connor, 2014). Some children may experience side effects such as reduced appetite, weight loss, slowing of expected gains in height and weight, increase in heart rate and blood pressure, or problems falling asleep. However, most potential side effects are typically benign and can be carefully monitored and often eliminated by reducing the dose of the medication (Graham et al., 2011). Although stimulants can be addictive if misused or abused (one recreational name for Ritalin is Vitamin R!), they are not addictive for most children who take them, nor do they lead to an increased risk for later




Tracy Dominey/Science Source

Stimulant medications are commonly used to treat children with ADHD

substance abuse (Chang et al., 2013; Humphreys, Eng, & Lee, 2013; Molina et al., 2013). Stimulants seldom make children "high," nervous, or jumpy, or turn them into nonfeeling zombies. Qualitative findings suggest that young people taking medication are generally positive about taking it and report that it reduces their disruptive behavior, improves their relationships with peers, and increases their ability to meet normative expectations (Singh, 2013; Singh et al., 2010).

The short-term benefits of medication are well documented (Spencer, Biederman, & Wilens, 2000). Unfortunately, follow-up studies raise questions about their long-term benefits (Jensen et al., 2007). The effects of stimulants are temporary and occur only while the individual is taking the medication. In this sense, the use of stimulants is similar to other important treatments for chronic conditions, such as insulin used for diabetes; however, they are not a cure. Many young people receiving stimulants for severe behavior problems remain impaired, despite many years of medication treatment. The limited long-term benefits of stimulants raise important issues about their clinical use that have yet to be resolved, particularly their use in preschool children (Riddle et al., 2013).

### *Controversy: The Ritalin Wars*

*There is no pharmacologic free lunch. The key question is always this: Is the gain to public health from proper use of an agent … greater than the danger of wide-scale misuse or the social cost of the regulatory machinery itself?*

—Leon Eisenberg (2007)

Public awareness of and controversy about the potential misuse of medication is evidenced by efforts(ultimately unsuccessful) to introduce legislation in the U.S. Congress with the express purpose "to protect children and their parents from being coerced into administering a controlled substance in order to attend school, and for other purposes" (Child Medication Safety Act of 2007). Why the concern? Community and physician surveys, increased production of Ritalin, and pharmacy audits all indicate that stimulant consumption has more than tripled since 1990 in North America (Scheffler et al., 2007). The co-prescription of stimulants with other medications, most commonly antidepressants, has also increased during this period (Comer, Olfson, & Mojtabai, 2010). Over 3.5 million children and adolescents in the United States (6% of 4- to 17-year-olds) are currently taking stimulants for ADHD, a 28% increase from 2007–2008 to 2011–2012 (Visser et al., 2013). Moreover, the use of Ritalin is at least five times higher in North America than in the rest of the world, although its use worldwide is also increasing. This increase may stem from a widening of the diagnostic criteria for ADHD, greater use of stimulants among girls and older individuals, widespread third-party medication coverage, and direct-marketing efforts by drug companies. Also, as changes in public policy and laws increase eligibility for special education and other services for individuals with ADHD, more individuals may receive this diagnosis and subsequently be prescribed medication (DuPaul & Stoner, 2003). Some findings indicate that although stimulant use in children ages 15 and younger in the United States has leveled off over the past decade, it continues to increase in older adolescents and young adults (McCabe & West, 2013). This increase is related to the growing recognition of ADHD in adults, and has also raised concerns about a possible diversion of stimulants from medical to nonmedical use in older individuals (Rabiner, 2013). In fact, among high school seniors in the United States, the self-reported lifetime prevalence of medical and nonmedical use of stimulants is the same (9.5%) (McCabe & West, 2013).

Given the astronomic increase in the use of stimulants to treat ADHD in North America, despite the similarities of prevalence across different parts of the world, we need to ask whether ADHD is overdiagnosed and whether stimulants are overprescribed (Eisenberg, 2007). Because of the wide variability in diagnostic practices, treatment decisions, and rates of stimulant use in various schools, communities, geographic regions, and populations, it is not surprising that research findings concerning diagnosis and medication use are inconsistent (Angold et al., 2000; Jensen et al., 1999). Perhaps the best overall conclusion to be drawn from the research is that in many cases stimulants are currently being used inappropriately: underprescribed in some cases and

overprescribed in others. We need a better understanding of the factors leading to the diagnosis of ADHD and stimulant use so that further steps can be taken to increase appropriate use—for example, improved screening and diagnosis and better education of service providers (Jensen, 2000; Kovshoff et al., 2013). Consider the following comments by the parents of a child with ADHD:

> When all is said and done, we stand in the middle on the issue of medication—not dramatically opposed, but not wildly enthusiastic either. Ideally, stimulants should be prescribed, monitored carefully, and there should be ongoing communication with parents and school personnel. However, the world being what it is, a lot of people seem to be falling short of the ideal a lot of the time. (McCluskey & McCluskey, 2000, p. 11)

Despite their limitations, stimulants—when properly used—remain among the most effective treatments for managing symptoms of ADHD. Nevertheless, since stimulants do not address many of the associated individual, family, academic, and peer problems of children with ADHD, additional primary interventions such as parent management training and educational interventions are needed.

## Parent Management Training (PMT)

Being the parent of a child who is overactive, disorganized, irritable, and does not listen or follow directions is difficult and exhausting. Usual discipline tactics such as reasoning, warning, or scolding often don't work. Thus, parents may feel powerless and at a loss as to what to do. Out of frustration, they may spank, ridicule, or yell at their child, even though they know it is not effective. These reactions leave everyone in the family feeling more upset than ever. **Parent management training (PMT)** focuses on teaching both effective parenting practices and strategies for coping with the challenges of parenting a child with ADHD (Chacko et al., 2014). It provides parents with a variety of skills to help them:

- manage their child's oppositional and noncompliant behaviors;
- cope with the emotional demands of raising a child with ADHD;
- contain the problem so that it does not worsen; and
- keep the problem from adversely affecting other family members.

Parents are first taught about ADHD so that they understand the biological basis of the disorder. This helps to remove the burden of guilt from parents who may think they have caused the problem. Parents are also given a set of guiding principles for raising a child with ADHD, such as using more immediate, frequent, and powerful consequences; striving for consistency; planning ahead; not personalizing the child's problems; and practicing forgiveness (Barkley, 2013c).

Parents are next taught behavior management principles and techniques, such as identifying behaviors they wish to encourage or discourage, using rewards and sanctions to achieve specified goals, establishing a home token program, noticing what their child does well, and praising their child's strengths and accomplishments. For disruptive behavior, parents also learn to use penalties such as loss of privileges or time-out as well as how to manage noncompliance in public places. Parents may also learn to use a school–home-based reward program, in which teachers evaluate the child on a daily report card (Owens et al., 2012). This card serves as a means for rewards or punishments (usually tokens) that will be administered at home for classroom conduct. Parents also learn how to manage future misconduct and are given follow-up sessions (Smith et al., 2006).

Parents are encouraged to spend time each day sharing an enjoyable activity with their child. They learn to structure situations in ways that will maximize the child's success and minimize failures. For example, if the child has difficulty completing tasks, it may be necessary to break the task into smaller steps and then praise the completion of each step. In PMT, parents also learn to reduce their own levels of arousal through relaxation, meditation, or exercise. Reduced arousal or anger allows parents to respond more calmly to their child's behavior.

Numerous studies support the effectiveness of PMT and other psychosocial interventions in treating children with ADHD (Chacko et al., 2014; Evans et al., 2013; Fabiano et al., 2009; Mulqueen, Bartley, & Bloch, 2013); however, the relative advantages and long-term benefits of PMT when used on its own to treat ADHD continue to be debated (Chronis-Tuscano, Chacko, & Barkley, 2013; Sonuga-Barke et al., 2013). As we will discuss, the effects of stimulants appear to be as strong as or stronger than the effects of PMT in treating the primary symptoms of ADHD. PMT may produce additional therapeutic benefits by treating the associated problems, improving family functioning, and increasing consumer satisfaction (Chronis et al., 2004a). To date, PMT has focused mainly on teaching parents to manage the overt disruptive behaviors that accompany their child's ADHD rather than on changing the deficits underlying the child's ADHD. New approaches that combine PMT with therapy that is directed at the parent–child processes that mediate the development of attention and self-regulatory skills may provide incremental benefits for children with ADHD (Sonuga-Barke et al., 2006).

## Educational Intervention

**ALAN**

### Boxed in at School

My teacher wanted to make me concentrate better, so one day she put my desk in the far corner, separated from the rest of the class. A few days had passed. I still wasn't finishing my work on time, but I was trying. My teacher didn't care; it wasn't finished. She then put a refrigerator box around my desk so I couldn't see anyone. I could hear as other kids in class would make fun of me. It really hurt; I was ashamed of myself and mad at my teacher. I couldn't tell my Mom because I might get into trouble. I hated school, didn't like my teacher, and started not liking myself. … It was hard to face the next day. A week had passed, and I poked holes in the cardboard so I could see who was making fun of me. I started peeping through the holes, making the other kids laugh. The teacher would get so annoyed. So I became the class clown. I was expelled for two days. When my Mom found out what was going on, boy, did she get angry; she was mad that the teacher would do this and mad that the principal allowed it and no one could see what this was doing to me.

From R. A. Barkley and L. J. Pfiffner, "Off to School on the Right Foot: Managing Your Child's Education. In: Barkley, R. A. Taking Charge of ADHD: The Complete, Authorized Guide for Parents, 1995, p. 208.

Classroom requirements to sit still, pay attention, listen to instructions, wait your turn, complete assignments, and get along with classmates are not easily met by children with ADHD. Their inattention and hyperactivity–impulsivity make learning very difficult, at times even painful. Although some children with ADHD are placed in a special education class for all or part of the day, most remain in the regular classroom. Whenever possible, it is preferable to keep children with ADHD in classes with their peers.

Educational interventions focus on managing inattentive and hyperactive–impulsive behaviors that interfere with learning and on providing a classroom environment that capitalizes on the child's strengths (DuPaul & Stoner, 2003). Techniques for managing classroom behavior are similar to those recommended to parents. The teacher and child set realistic goals and objectives, set up a mutually agreed-upon reward system, carefully monitor performance, and reward the child for meeting goals. Disruptive or off-task classroom behaviors may be punished with **response-cost procedures** that involve the loss of privileges, activities, points, or tokens following inappropriate behavior or with brief periods of time-out. These procedures have proved to be effective in reducing disruptive classroom behavior and enhancing academic productivity (Pfiffner & DuPaul, 2014).

Many strategies for instructing children with ADHD are simply good teaching methods. Letting children know what is expected of them, using visual aids, providing cues for expected behavior, and giving written as well as oral instructions all help children focus their attention and remember important points. In addition, children with ADHD may require other accommodations to help them learn. For example, the teacher may seat the child near the teacher's desk, provide a designated area in which the child can move about, establish a clearly posted system of rules, and give the child frequent cues for expected behaviors. A card or a picture on the child's desk can provide a visual reminder for acceptable behavior such as raising a hand instead of shouting out. Repeating instructions, providing extra time, writing assignments on the board, and listing all of the books and materials needed for a task may increase the likelihood that children with ADHD will complete their work (Pfiffner & DuPaul, 2014).

School-based interventions for ADHD have received considerable support. A meta-analysis of 60 studies spanning 15 years found that school-based programs, including academic, contingency management, and self-regulation interventions, were related to moderate to large improvements in academic and behavioral functioning of students with ADHD (DuPaul, Eckert, & Vilardo, 2012). Some efforts have focused on a variety of school-based interventions for ADHD, including those for individual students in regular and special education classes, combined home and school interventions, and schoolwide interventions that incorporate both universal and targeted treatments (Pfiffner & Dupaul, 2014; Waschbusch, Pelham, & Massetti, 2005).

## Intensive Interventions

There are no quick cures for ADHD. More intensive (and ongoing) treatments than previously used may be required to produce meaningful changes in long-term outcomes. As described below, the Summer Treatment Program and the Multimodal Treatment Study for Children with ADHD are two examples of programs that have provided intensive treatment to children with ADHD and their families.

### *Summer Treatment Program*

Over the past 30 years, Dr. William Pelham and his colleagues have developed and disseminated an exemplary intensive summer treatment program (Pelham et al., 2010). In this program, treatment is provided to

children with ADHD ages 5 to 15 in a camplike setting where they engage in classroom and recreational activities with other children. Summer treatment has two major advantages over other interventions: It maximizes opportunities to build effective peer relations in normal settings, and it provides continuity to academic work to ensure that gains made during the school year are not lost. These programs are coordinated with stimulant medication trials, parent management training, social skills training, and educational interventions in an all-out treatment effort.

The Summer Treatment Program packs 360 hours of day-treatment into a period of 8 weeks, the equivalent of 7 years of weekly therapy. Ratings by parents and counselors suggest that children who participate show overall improvements in behavior, decreases in problem severity, and improvements in social skills and academic performance. Children also rate themselves as doing better, and parents report higher levels of self-efficacy. Dropout rates are low and consumer satisfaction is high. The program is also cost-effective as compared with more traditional treatments. Preliminary findings from controlled studies of outcomes are promising (Chronis et al., 2004b; O'Connor et al., 2012; Pelham et al., 2000; Sibley et al., 2013); however, although growing in use, these programs are not yet widely available and it is still too early to tell whether this kind of intensive program will make a long-term difference for these children.

### The MTA Study

The Multimodal Treatment Study of Children with ADHD (MTA Study) is a landmark multisite study sponsored by the U.S. National Institute of Mental Health (NIMH) and the U.S. Department of Education. It represents the first large, randomized clinical trial for children with ADHD. The study sought to answer three questions: How do long-term medication and behavioral treatments compare with one another? Are there additional benefits when they are used together? What is the effectiveness of systematic carefully delivered treatments versus routine community care? (MTA Cooperative Group, 1999a).

Courtesy of William E. Pelham

Courtesy of William E. Pelham

Children with ADHD participating in a Summer Treatment Program

Carefully diagnosed children 7 to 9 years of age with ADHD were randomly assigned to one of four treatment groups, followed by major assessments at periodic intervals during and after treatment.

- *Medication Management:* This group received stimulant medication 7 days a week;
- *Behavioral treatment:* This group received 35 sessions of parent management training, up to 10 teacher and school visits per year, and participation in an intensive 8-week summer treatment program, which taught academic and social skills and had a classroom aide who continued to reinforce strategies learned in the summer treatment program in the child's actual classroom for half a day, 5 days per week, for 12 weeks;
- *Combined behavioral treatment and medication:* This group received both medication and behavioral treatment; or
- *Routine community treatment:* This group received treatment as it was routinely delivered in community care. In fact, 66% of children in this group received stimulant medication.

The major finding from the MTA Study after 14 months of active treatment was that all groups showed reductions in ADHD symptoms over time, but there were significant variations in the amount of change. First, stimulant medication was superior to behavioral treatment and to routine community care in treating the symptoms of ADHD. Second, combining behavioral treatments with medication resulted in no additional benefits for the core symptoms of ADHD over medication alone, but it did provide modest benefits for non-ADHD symptoms and other outcomes related to positive functioning (MTA

Cooperative Group, 1999a). Composite outcome measures showed that combined treatment was best, followed by medication, then behavior therapy, and finally, community treatment (Conners et al., 2001; Swanson et al., 2001).

The benefits of combined treatment were also found at 24 months of follow-up (MTA Cooperative Group, 2004a, b). However, by 36 months there were no significant treatment group differences, with all groups showing equal benefits for ADHD symptoms (Jensen et al., 2007). Consistent with this result, findings from the MTA Study at 6 and 8 years after enrollment in the program indicate that the effects of both medication and behavioral treatments either decline or cease entirely when the treatment stops (Molina et al., 2009). Thus, the efficacy of treatment for children with ADHD will require that these treatments continue to be provided in a comprehensive, carefully monitored, and ongoing fashion. The interventions in the MTA Study were more intensive and monitored more closely than is typically the case in real-world clinical practice. Further research will be needed to determine the intensity of intervention and frequency of monitoring needed to maintain treatment gains and optimal functioning over time and across contexts in light of available treatment resources (Abikoff, 2009).

Other questions from the MTA Study that continue to be addressed concern which treatments work best for which children, for which outcomes, and why? (Hinshaw, 2007b). For example, children with ADHD and comorbid anxiety and children from families on social assistance may benefit more from behavioral treatments than those without these difficulties (MTA Cooperative Group, 1999b), and behavioral treatments may be associated with less substance use at a later age (Molina et al., 2007). In general, the long-term findings from the MTA study indicate that the initial clinical presentation in childhood (e.g., severity, co-occurring conduct problems, social disadvantage) and the strength of ADHD symptom response to *any* treatment are better predictors of adolescent outcomes than the type of treatment received in childhood (Molina et al., 2009).

The MTA Study findings can be interpreted in different ways and are likely to be debated for some time to come. It seems that for children who have uncomplicated ADHD with no co-occurring disorders, adequate social functioning, and good academic performance, medication management may be the best treatment option. However, for those who have ADHD complicated by oppositional symptoms, poor social functioning, and ineffective parenting, combining medication and behavioral treatment may be the best option. In both cases, ongoing interventions will likely be needed. Whatever the final verdict, findings from the MTA study continue to raise numerous questions of clinical importance for children with ADHD and their families.

## Additional Interventions

Other interventions have been used to provide support to children with ADHD and their families. Among these interventions are family counseling and support groups and individual counseling for the child. (A brief overview of these interventions can be found in Table 8.3.)

### *Family Counseling and Support Groups*

Many families of children with ADHD experience frustration, blame, and anger for some time. As we have discussed, siblings may feel neglected or resent the time their parents spend with the child with ADHD. Family members may require special assistance not only in managing behavior but also in dealing with their own thoughts and feelings. Counseling the family helps everyone develop new skills, attitudes, and an ability to relate more effectively.

Support groups for people who are coping with ADHD in various ways can be very helpful to members. There are many local and national support groups for parents of children with ADHD. Members share information, emotional support, personal frustrations and successes, referrals to qualified professionals, discoveries about what works, and their aspirations for their children and themselves. There are also online bulletin boards and discussion groups. Sharing experiences with others that have similar concerns helps parents feel that they are not alone.

### *Individual Counseling*

Life can be very hard for children with ADHD. They have few successes on which to build their sense of self-competence. Perhaps as a result, even when they succeed, they may attribute their success to uncontrollable factors such as task ease or luck (Hoza et al., 2000). Being punished or told they are stupid or bad is often their main form of attention. They have few friends and are constantly in trouble. The cumulative impact can leave them feeling isolated and believing that they are abnormal, stupid, or doomed to failure. Individual counseling attempts to address these concerns, although evidence for its effectiveness in treating children with ADHD is limited. Children usually come into counseling with many questions about ADHD and treatment that are addressed at the outset and in later sessions (see A Closer Look 8.4. How would you answer these questions?).

## A CLOSER LOOK 8.4

### Questions Asked by Children and Adolescents with ADHD

**Children (Ages 4 to 10)**

I just found out I have ADHD. How can I keep this secret from my brother?
I heard ADHD means you're weird. Is that right?
Is it true that if you have ADHD you can think faster than other people?
Will the medicine make me smarter?

**Adolescents (Ages 11 to 17)**

How do you know the medicine isn't dangerous?
Any advice on how to deal with the fact that I feel like a reject because I have ADHD?
How long am I going to have ADHD?
How can I convince [my teacher] that ADHD exists and that it affects my performance?

*Source*: Based on Hallowell and Ratey, 1994.

## A Comment on Controversial Treatments

Understandably, parents want to explore all possible ways to help their children with ADHD. Over the years, many treatments that sound plausible have been proposed. Some are enthusiastically endorsed by professionals, and individual patient reports claim dramatic success; others are pure charlatanism. Treatments proposed for children with ADHD that have not been scientifically substantiated include allergy treatments, homeopathic treatments, medication to correct inner ear problems, vestibular stimulation, running, walks in the park, treatment for yeast infection, megavitamins, sensory integration training, chiropractic adjustment, eye training, special colored glasses, metronome therapy, and applied kinesiology (realigning bones in the skull). Untested or fad treatments may prove to be expensive, provide false hope for a quick cure, delay the use of evidence-based treatments that are known to be of some benefit, and in some cases may even be harmful (AACAP, 2013; Waschbusch & Hill, 2003).

## Keeping Things in Perspective

**MARK**

### Good Support System

Through my years so far, I've been through a lot. My Mom says I have a good heart; I care about those in need. I'm not dumb. You can't always measure smartness by tests. I feel I'm doing better now. It helps to talk to people who understand. What I'm trying to say is: no matter what comes my way, I can survive. I have those who really care, and from that I draw my strength.

From R. A. Barkley and L. J. Pfiffner, "Off to School on the Right Foot: Managing Your Child's Education. In: Barkley, R. A. Taking Charge of ADHD: The Complete, Authorized Guide for Parents, 1995, p. 208.

Young people with ADHD have problems that should not be minimized, especially if doing so prevents children and adolescents with ADHD and their families from receiving help. However, as Mark's comments illustrate, in helping those with ADHD and their families, it is important not to lose sight of the fact that each child is unique and has assets and resources that need to be recognized and supported. These assets can serve as a buffer in reducing the child's behavior problems and referral concerns (Short et al., 2007).

In closing, many of our current treatments for ADHD developed prior to advances in our theory and knowledge about the possible causes of ADHD. Efforts are now underway to design more effective treatments that are sensitive to the different types of ADHD and to specific cognitive and behavioral deficits of individual children (Casey, Nigg, & Durston, 2007).

## Section Summary

### Treatment

- There is no cure for ADHD, but a variety of treatments can be used to help children cope with their symptoms and any secondary problems that may arise over the years.
- The primary approach to treatment combines stimulant medication, parent management training, and educational intervention.
- Stimulants are the most effective treatment for managing symptoms of ADHD; however, their limited long-term benefit raises important issues about their clinical use that are yet to be resolved.
- Parent management training (PMT) provides parents with a variety of skills to help them manage their child's oppositional and defiant behaviors and cope with the difficulties of raising a child with ADHD.
- Educational interventions focus on managing inattentive and hyperactive–impulsive behaviors that interfere with learning and on providing a classroom environment that capitalizes on the child's strengths.

- Findings from the MTA Study, a landmark controlled comparison of intensive treatments for ADHD, suggest that for children with uncomplicated ADHD, medication may be the best treatment option; however, for those with ADHD and oppositional symptoms, poor social functioning and ineffective parenting, combining medication and behavioral treatment may be the best option.
- Additional interventions for ADHD include family counseling and support groups, and individual counseling for the child.

# Study Resources

## SECTION SUMMARIES



## KEY TERMS

# 9

# Conduct Problems

*Our youth now love luxury. They have bad manners, contempt for authority and disrespect for their elders. Children nowadays are tyrants.*

—Socrates, 470–399 B.C.E.

## CHAPTER PREVIEW

CHILDREN'S CONDUCT PROBLEMS HAVE long been a societal concern and been considered to be forerunners of juvenile delinquency and adult criminality. However, despite enormous public, scientific, and professional attention, substantial numbers of youths continue to display antisocial, destructive, and violent behaviors, many of which are hidden from public view. Many types of adolescent conduct problems have increased substantially over the past 25 years, a change that has affected males and females, all social classes, and all family types (Collishaw et al., 2004). Since 1994, the most lethal forms of youth violence in the United States steadily decreased and then leveled off in the 2000s, and the number of youths arrested for violent crimes in 2010 was at its lowest in at least 30 years (Puzzanchera, 2013). However, the prevalence of other forms of antisocial behavior (e.g., aggravated assault) remains alarmingly high, and the proportion of females involved in violent crimes has increased (Zahn et al., 2008). A nationally representative survey of U.S. high school students in 2011 found that about 33% had been in a physical fight at least once in the past year, 25% reported being bullied on school property, 17% reported carrying a weapon in the past month, and 8% reported being threatened or injured with a weapon on school property (CDC, 2012b; Robers, Kemp, & Truman, 2013). Tragically, there are many victims of youth violence. Guns killed more children in the United States in 2008–2009 than the number of U.S. military deaths in both the Iraq and Afghanistan wars to date (Children's Defense Fund, 2012).

Masterfile

Early conduct problems may be forerunners of delinquency and adult criminal behavior

The high prevalence of youths with conduct problems and the harm inflicted on their victims create an urgent need for understanding and assistance. Tragic school shootings by youths, such as the mass murder–suicide at Sandy Hook Elementary School in Newtown, Connecticut, in 2012 that resulted in the deaths of 20 children and 6 adult staff members, provide stark reminders of the societal impact of youth violence. These youths often have a background that suggests a history of social isolation and rejection, unusual social behavior, and a fascination with violent themes. As such, these incidents raise important questions about factors that contribute to violence and other antisocial behavior by young people in our society (McNamara & Findling, 2008).

Unfortunately, school shootings and media portrayals of extreme antisocial acts may also fuel popular beliefs that aggression is inherent in humans, that some children are born bad, or that youth violence is symptomatic of a decaying society. In fact, although it is an ongoing and extremely serious problem, much progress has been made in understanding, reducing, and preventing youth violence as well as less harmful but still serious forms of antisocial conduct (Moffitt et al., 2008).

## DESCRIPTION OF CONDUCT PROBLEMS

*"[T]hose who violate social and criminal codes do so for very many reasons. . . .[B]efore studying our cases we must do our best to group them into different sorts. . . ."*

—John Bowlby (1907–1990)

### ANDY

### Young Rage

"Andy threw his booster seat in my face and hit my jaw. He thought it was funny. He was acting up, and I think he had already had one time-out for yelling and screaming and interrupting us at the table. And I said, "Fine, you are not having dessert." He flew into a rage. He picked up a metal fork and threw it at me with all his force, and hit me—barely missed my eye. There was blood on my forehead. I was hysterical. I was terrified to see that type of rage in a 4-year-old."

Based on Troubled Families—Problem Children: Working with Parents: A Collaborative Process by C. Webster-Stratton and M. Herbert, 1994, pp. 44–45

### MARVELLE

## Defiant

"She just drives me up the wall. She's irritable all the time and never does anything I ask her to do. When she doesn't get her way she throws a full-blown tantrum. Her behavior is also a problem at school. Her teacher can't get her to do schoolwork—she simply refuses. She's also defiant, won't stay in her seat, and talks constantly. She's disrupting the entire class. I'm worried that she's headed for serious problems if she doesn't shape up soon." (Based on authors' case material)

### NICK

## Not Like Other Kids

Outwardly, Nick is a normal 10-year-old. He loves sports, especially football. He has a talent for drawing and an aptitude for math … but Nick isn't like other kids. At age 2, he put a can of cat food on the stove, and lit the burner—it exploded. In one 5-day period last March, he threw a rock at a girl at the YMCA, hitting her in the head and drawing blood; set fire to his room; pushed his sister down the stairs; whipped the family dog with a chain; and stole $20 from his mother's wallet.

Adapted from Colapinto, 1993, p. 122.

**Conduct problem(s)** and **antisocial behavior(s)** are terms used to describe a wide range of age-inappropriate actions and attitudes of a child that violate family expectations, societal norms, and the personal or property rights of others (Kimonis, Frick, & McMahon, 2014). These children experience problems in controlling their emotions and behavior. Like the children in our examples, youths with conduct problems display a variety of disruptive and rule-violating behaviors, ranging from annoying but relatively minor behaviors such as whining, swearing, and temper tantrums to more serious forms of antisocial behavior such as vandalism, theft, and assault. Given such diversity, we need to consider many types, pathways, causes, and outcomes of conduct problems.

Although we may be shocked by their actions, children with severe conduct problems frequently (not always) grow up in extremely unfortunate family and neighborhood circumstances, where they experience physical abuse, neglect, poverty, or exposure to criminal activity (Lahey, Miller et al., 1999). Thus, in many cases, aggressive behaviors are an adaptation to home and neighborhood violence and neglect. These circumstances do not excuse the behaviors, but they do provide an important backdrop for understanding and preventing these problems. Consider the case of Steve.

### STEVE

## Not without Cause

Twelve-year-old Steve was referred because he stabbed his father in the leg and stole a car. He had a history of lying, fighting at school, and theft and was constantly in trouble with school personnel and police. He readily admitted stabbing his father in the leg, but his story included some interesting details that hadn't come up previously. He and his two brothers were in their parents' bedroom while the father was raping the mother. She was screaming for help and panicked. Steve went and got a knife from the kitchen; his brothers tried to restrain him but could do so only partially. He stabbed his father in the calf, deeply and with a long cut. After the stabbing, Steve felt he was going to get beaten, because his father had a long history of physically abusing the boys. He fled to his grandfather's house, took the car keys without permission, drove off, and crashed the car in a field. The police brought Steve to us. By all accounts, he had stabbed his father. And indeed he stole a car.

Based on Kazdin, A. E. (1995). Conduct disorders in childhood and adolescence (2nd ed.). Thousand Oaks, CA: Sage.

Steve's tragic family situation may evoke sympathy and concern. Youths with severe conduct problems are often seriously disturbed and need help. At the same time, the callousness of their deeds often evokes outrage, concern for innocent victims, and a desire to severely punish or confine them. This creates an inconsistency between society's concern for children who experience early adversity or abuse and the tendency to criminalize and demonize youths who display violent behaviors. As they grow older, these youths walk a fine line between pleas from the mental health and juvenile justice systems for understanding and rehabilitation and demands from the general public and the criminal justice system to punish the offenders and protect the victims (Steinberg, 2009). Most people have opinions about the nature of youth violence and what can be done about it. To examine some of your own views, consider the statements in A Closer Look 9.1.

## A CLOSER **LOOK 9.1**

### Beliefs about Youth Violence: True or False?

| | | |
|---|---|---|
| Most future offenders can be identified during early childhood. | T | F |
| Child abuse and neglect inevitably lead to violent behavior later in life. | T | F |
| African American and Hispanic youths are more likely to become involved in violence than other racial or ethnic groups. | T | F |
| Getting tough with juvenile offenders by trying them in adult criminal courts reduces the likelihood that they will commit more crimes. | T | F |
| Most violent youths will end up being arrested for a violent crime. | T | F |
| Nothing works with respect to treating or preventing violent behavior. | T | F |

*Note:* All of the above statements are false. Such false ideas can be harmful when they fail to recognize the true nature of a problem or when they lead to inappropriate policies or practices.

Based on Department of Health and Human Services (2001). Youth Violence: A Report of the Surgeon General. Washington, DC: Author.

## Section Summary

### Description of Conduct Problems

- Conduct problems or antisocial behavior(s) are age-inappropriate actions and attitudes of a child that violate family expectations, societal norms, and the personal or property rights of others. These children display problems in the self-control of emotions and behaviors.
- The nature, causes, and outcomes of conduct problems in children are wide-ranging, requiring that we consider several different types and pathways.
- Many children with severe conduct problems grow up in extremely unfortunate family and neighborhood circumstances.

# CONTEXT, COSTS, AND PERSPECTIVES

*"[T]he extent to which any of us conform with the social and criminal codes is a matter of degree. . . ."*

—John Bowlby (1950)

To understand antisocial behavior in young people and its impact on society, we next consider its expression in the context of normal development, its societal costs, and the different ways in which such behavior has been viewed by the juvenile justice, mental health, and public health systems.

## Context

Most young people break the rules from time to time. Did you ever defy authority, lie, spread rumors, fight, skip school, run away, break curfew, destroy property, steal, text while driving, or drive under the influence of alcohol? If so, welcome to the club—many young people admit to these antisocial acts. In 2011, about 71% of high school students in the United States had consumed alcohol, 45% smoked cigarettes, 40% used marijuana, 33% texted or e-mailed while driving, and 8% had driven after drinking alcohol (CDC, 2012b). In fact, very few adolescents (about 6%) refrain from antisocial behavior entirely, and those who do describe themselves as excessively conventional, anxious, and socially inhibited—not well adjusted at all during adolescence (Moffitt et al., 2002).

Antisocial behaviors appear and then decline during normal development (Tremblay, 2003). Most toddlers hit, kick, intentionally break things, tell lies, and resist adult authority, but most also learn to control these behaviors by the time they enter school. About 50% of parents report that their preschoolers steal, lie, disobey, or destroy property, in contrast to 10% of parents who report the same about young adolescents (Achenbach, 1991a). This decline partially reflects the parents' lack of awareness of the trouble their teens may be getting into. However, teens also report that their antisocial behaviors decrease with age (Achenbach, 1991b). Frequencies of three common antisocial behaviors for clinic-referred and non-referred boys and girls of different ages, as reported by their parents, are shown in ● Figure 9.1.

The graphs in Figure 9.1 illustrate several important features of antisocial behaviors in the context of normal development:

- Antisocial behaviors vary in severity, from minor disobedience to fighting.

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Policies and practices that place youths with conduct problems together can increase their antisocial and delinquent behavior.

- Some antisocial behaviors decrease with age (e.g., disobeying at home), whereas others increase with age and opportunity (e.g., hanging around with kids who get into trouble).
- Antisocial behaviors are more common in boys than in girls during childhood, but this difference narrows in adolescence.

Even though many antisocial behaviors decrease with age, children who are the most physically aggressive in early childhood maintain their relative standing over time (Broidy et al., 2003). Longitudinal studies find aggressive acts such as persistent physical fighting to be highly stable, with an average correlation of about 0.70 for measures of these behaviors taken at different times (Loeber, Green et al., 2000). This makes aggressive behavior about as stable as IQ scores!

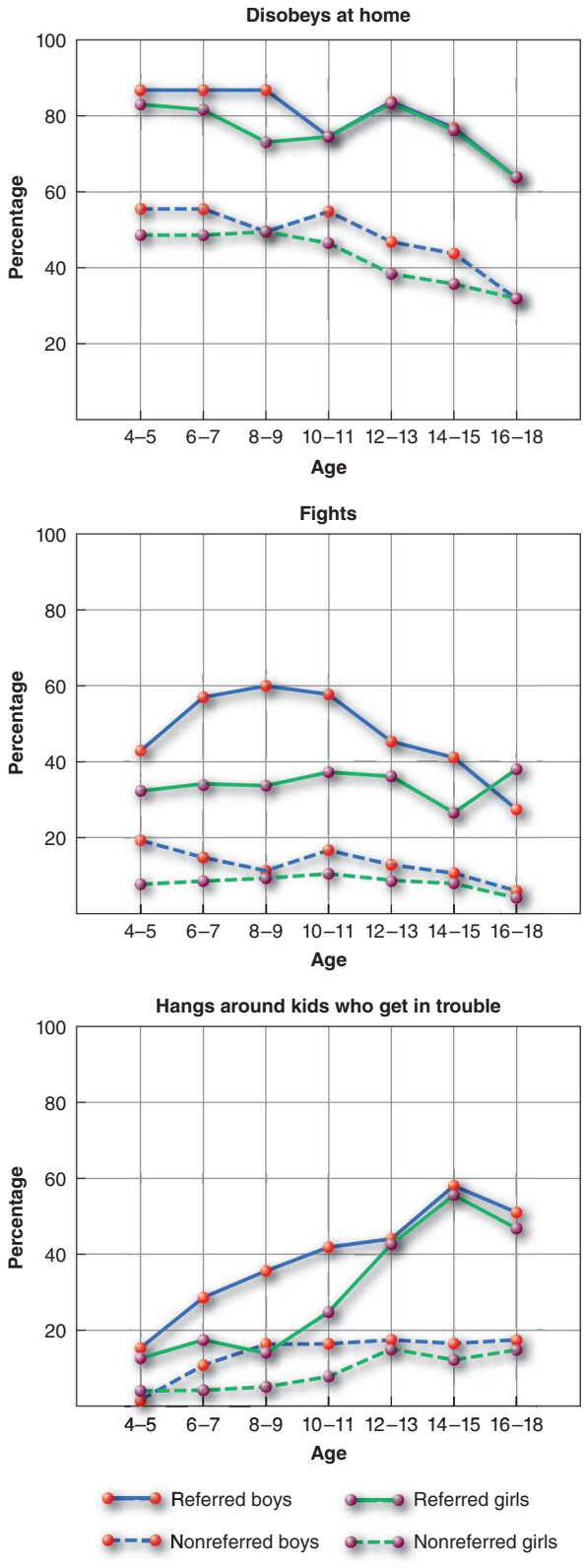
## Social and Economic Costs

The staggering costs borne by the educational, health, criminal justice, social service, and mental health systems that deal with youths with conduct problems make it the most costly mental health problem in North America (Welsh et al., 2008). Although antisocial acts are universal in young people, an early, persistent, and extreme pattern of antisocial behavior occurs in only about 5% of children (Hinshaw & Lee, 2003). These children cause considerable and disproportionate amounts of harm, accounting for over 50% of all crime in the United States, and about 30% to 50% of clinic referrals (Loeber, Burke et al., 2000).

More teenagers in the United States die from firearm injuries than from all diseases combined, and they are more than twice as likely as adults to be victims of violence, most often committed by other teens (Snyder & Sickmund, 2006). The costs of antisocial behavior can be understood in terms not only of lives but also of dollars. As much as 20% of all mental health expenditures in the United States are attributable to crime (National Institute of Justice, 1996). The additional public costs per child with conduct problems across the health care, juvenile justice, and educational systems are enormous—at least $10,000 or more a year (Foster, Jones, & The Conduct Problems Prevention Research Group, 2005). The lifetime costs to society for one youth to leave high school for a life of crime and substance abuse have been estimated to be about $3.2 million to $5.5 million (Cohen & Piquero, 2009).

## Perspectives

Conduct problems have been viewed from several perspectives, each using different terms and definitions to




● **FIGURE 9.1** | Parent-reported frequencies for common antisocial behaviors in clinic and nonreferred boys and girls ages 4 to 18.

From *Manual for the Child Behavior Checklist/4-18 and 1991 Profile* by T. M. Achenbach, 1991, pp. 131, 134, 138, 145. University of Vermont. Copyright by T. M. Achenbach. Reproduced by permission.

describe similar patterns of behavior. These include the legal, psychological, psychiatric, and public health perspectives (Loeber, Burke, & Pardini, 2009).

### Legal

Legally, conduct problems are defined as delinquent or criminal acts. The broad term **juvenile delinquency** describes children who have broken a law, ranging from sneaking into a movie without paying to homicide. Delinquent acts include property crimes (e.g., vandalism, theft, breaking and entering) and violent crimes (e.g., robbery, aggravated assault, homicide). Legal definitions depend on laws that change over time or differ across locations. Delinquency, the legal definition, involves apprehension and court contact and excludes the antisocial behaviors of very young children that usually occur at home or school. It is also important to distinguish official records of delinquency from self-reported delinquency. Youths who display antisocial behavior and are apprehended by police may differ from youths who display the same patterns but are not apprehended because of their intelligence or resourcefulness. Debate is ongoing about the age at which children should be held responsible for their delinquent behavior. The minimum age of criminal responsibility ranges from 7 to 14 years in most states and provinces, but this has fluctuated over the years in relation to society's tolerance or intolerance of antisocial behavior in youth.

Given the large numbers of youths involved in criminal activities, we must ask whether these behaviors are understandable (albeit objectionable) adaptations to a hostile environment—the most common reason that youths give for carrying a weapon is self-defense (Simon, Dent, & Sussman, 1997). Unfortunately, no clear boundaries exist between delinquent acts that are a reaction to environmental conditions, such as a high-crime neighborhood, and those that result from factors within the child, such as impulsivity. Some criminal behaviors, such as arson and truancy, are arbitrarily included in current mental health definitions, whereas selling drugs, receiving stolen property, and prostitution are not. Similarly, some symptoms of mental health problems do not necessarily violate laws (e.g., bullying, staying out late without permission). A legal definition of delinquency may result from one or two isolated acts, whereas a mental health definition usually requires the child to display a variety and persistent pattern of antisocial behaviors. Thus, only a subgroup of youths who meet a legal definition of delinquency will also meet the definition for a mental disorder (Waldman & Lahey, 2013).

### Psychological

From a psychological perspective, conduct problems fall along a continuous dimension of **externalizing behavior** (Burns et al., 1997). Children at the upper extreme of this dimension, usually one or more standard deviations above the mean, are considered to have conduct problems. The externalizing dimension itself consists of two related but independent subdimensions, labeled "rule-breaking behavior" and "aggressive behavior" (Achenbach & Rescorla, 2001). Rule-breaking behaviors include running away, setting fires, stealing, skipping school, using alcohol and drugs, and committing acts of vandalism. Aggressive behaviors include fighting, destructiveness, disobedience, showing off, being defiant, threatening others, and being disruptive at school.

Two additional independent dimensions of antisocial behavior have been identified: overt–covert and destructive–nondestructive (Frick et al., 1993). The **overt–covert dimension** ranges from overt visible acts such as fighting to covert hidden acts such as lying or stealing. Children who display overt antisocial behavior tend to be negative, irritable, and

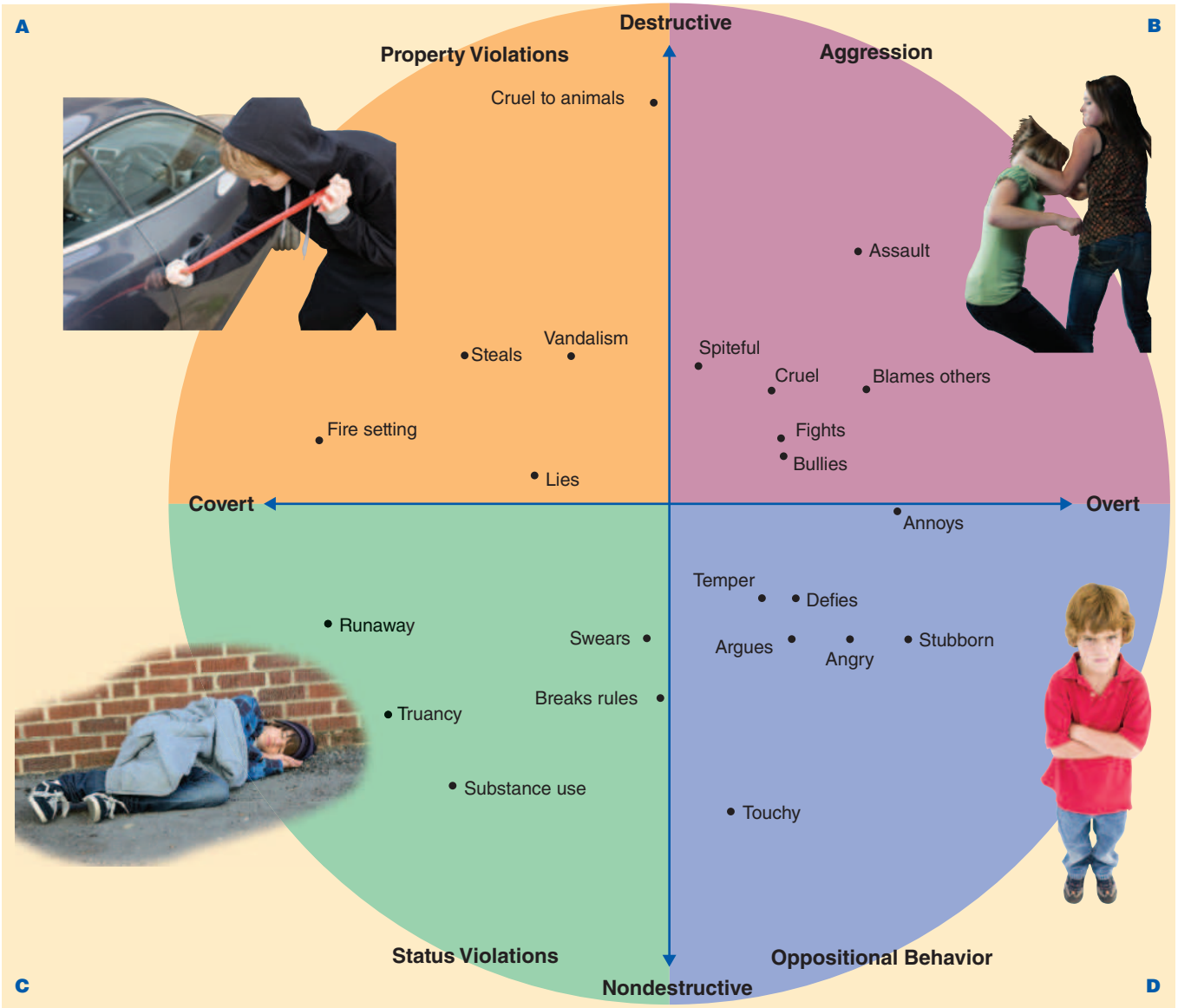
Chaos/Christopher T Stein/Jupiter Images

KidStock/Blend Images/Getty Images

Two sides of the externalizing dimension: overt (left) and covert (right)

resentful in their reactions to hostile situations and to experience higher levels of family conflict (Kazdin, 1992). In contrast, those displaying covert antisocial behavior are less social, more anxious, and more suspicious of others and come from homes that provide little family support. Most children with conduct problems display both overt and covert behaviors. These children are in frequent conflict with authority, show the most severe family dysfunction, and have the poorest long-term outcomes (Loeber, Lahey, & Thomas, 1991). The **destructive–nondestructive dimension** ranges from acts such as cruelty to animals or physical assault to nondestructive behaviors such as arguing or irritability.

As shown in ● Figure 9.2, crossing the overt–covert with the destructive–nondestructive dimension results in four categories of conduct problems: (A) covert–destructive, or property violations; (B) overt–destructive, or aggression; (C) covert–nondestructive, or status violations; and (D) overt–nondestructive, or oppositional behavior. Children who display overt–destructive behaviors, particularly persistent physical fighting, are at especially high risk for later psychiatric problems and impairment in functioning (Broidy et al., 2003).




● **FIGURE 9.2** | Four categories of conduct problems.

Based on "Oppositional Defiant Disorder and Conduct Disorder: A Meata-Analytic Review of Factor Analyses and Cross-Validation in a Clinic Sample," by P. J. Frick, Y. Van Horn, B. B. Lahey, M.3A. G. Christ, R. Loeber, E. A. Hart, L. Tannenbaum & K. Hanson, Clinical Psychology Review, 13, 319–340.

*Photo Credits:* (a) Paul Bradbury/OJO Images/Getty Images; (b) Weston Colton/Getty Images; (c) ©iStockphoto.com/plherrera; (d) © Monkey Business Images/Dreamstime.com

### *Psychiatric*

From a psychiatric perspective, conduct problems are defined as distinct mental disorders based on DSM-5 symptoms (APA, 2013). DSM-5 contains the general category of disruptive, impulse-control, and conduct disorders. All disorders in this category involve problems in the self-control of emotions and behaviors, including two that refer to persistent patterns of antisocial behavior in youth—oppositional defiant disorder (ODD) and conduct disorder (CD). This general category also includes *intermittent explosive disorder* (i.e., impulsive aggressive outbursts in response to minor provocations), *pyromania* (i.e., multiple episodes of deliberate and purposeful fire setting), and *kleptomania* (i.e., recurrent failure to resist impulses to steal items not needed for personal use or monetary value). In this chapter, we focus on ODD and CD, often collectively referred to as conduct problems or **disruptive behavior disorders.**

**Note:** Both categorical (psychiatric) and dimensional (psychological) perspectives have proven validity for classifying conduct problems in youth. Categories such as CD or ODD are associated with different patterns of behaviors and outcomes. On the other hand, dimensional measures of externalizing behavior in adolescence may be better predictors of adult outcomes than categorical measures (Fergusson, Boden, & Horwood, 2010). In other words, each perspective provides useful information.

### *Public Health*

This perspective blends the legal, psychological, and psychiatric perspectives with public health concepts of prevention and intervention (U.S. Department of Health and Human Services, 2001). The goal is to reduce the number of injuries and deaths, personal suffering, and economic costs associated with youth violence, in the same way that other health concerns such as automobile accidents or tobacco use are addressed. The public health approach cuts across disciplines and brings together policy makers, scientists, professionals, communities, families, and individuals to understand conduct problems in youths and determine how they can be treated and prevented (Dodge, 2011).

## Section Summary

### Context, Costs, and Perspectives

- For most children, antisocial behaviors appear and then decline during normal development, although children who are most aggressive maintain their relative standing over time.
- Costs to the educational, health, social service, criminal justice, and mental health systems that deal with youth make conduct problems one of the most costly mental health problems in North America.
- From a legal perspective, conduct problems are defined as criminal acts that result in apprehension and court contact and are referred to as "delinquency."
- From a psychological perspective, conduct problems fall along a continuous dimension of externalizing behavior, which includes a mix of impulsive, overactive, aggressive, and rule-breaking acts.
- From a psychiatric perspective, conduct problems are viewed as distinct categories of mental disorder based on DSM symptoms. These are called disruptive, impulse control, and conduct disorders, and include oppositional defiant disorder (ODD) and conduct disorder (CD).
- A public health perspective cuts across disciplines and blends the legal, psychological, and psychiatric perspectives with public health concepts of prevention and intervention.

## DSM-5: DEFINING FEATURES

In this section, we discuss the defining features and characteristics of ODD and CD. Both disorders have been found to predict future psychopathology and enduring impairment in life functioning (Burke, Waldman, & Lahey, 2010). Because of its relevance to understanding youth conduct problems and their adult outcomes, we also consider the relationship between these disorders, antisocial personality disorder (APD), and psychopathic symptoms.

### Oppositional Defiant Disorder (ODD)

**GORDON**

#### Enjoying His Power

He just digs his heels in, "That's it, I am not wearing these socks! Forget it, I'm not going!" And he is right. He's gone to school in his pajamas, without lunch, in the pouring rain without a coat. … He will explain to me, "Mom, we are done with this discussion." … He doesn't have an easy-going bone in his body. He is not ever going to say, "Okay, I'll put that turtleneck on." It's going to be, "I will do something but only on my terms. … I will do nothing that you want me to do and furthermore I'll throw such a tantrum and throw this cereal bowl all over the wall, so you will be late, and mad at me when you clean it up." … He enjoys that power.

Adapted from *Troubled Families—Problem Children: Working with Parents: A Collaborative Process* by C. Webster-Stratton and M. Herbert, 1994, p. 47.

Gordon's frequent arguing and active defiance of his mother are consistent with a diagnosis of **oppositional defiant disorder (ODD)**. These children display an age-inappropriate recurrent pattern of stubborn, hostile, disobedient, and defiant behaviors (see Table 9.1 for DSM-5 diagnostic criteria for ODD). ODD usually appears by age 8, and it was included in the DSM to capture early displays of antisocial and aggressive behavior by preschool and school-age children (Keenan, 2011). Many of these behaviors, such as temper tantrums or arguing, are extremely common in young children. However, severe and age-inappropriate ODD behaviors can have extremely negative effects on parent–child interactions (Greene & Doyle, 1999). Children with ODD also are at considerable risk for developing later impulse-control, substance-use, and mood and anxiety disorders, even after controlling for common co-occurring childhood disorders such as ADHD and CD (Frick & Nigg, 2012; Nock et al., 2007). Child and adolescent ODD symptoms also predict a variety of social and interpersonal difficulties in early adulthood, including poor functioning with peers and poor romantic relationships (Burke, Rowe, & Boylan, 2014).

Some findings indicate that symptoms of ODD can be grouped into three dimensions that reflect *negative affect* (angry/irritable mood), *defiance* (defiant/headstrong behavior), and *hurtful behavior* (vindictiveness), which differentially predict later emotional and behavioral disorders in early adulthood (Burke, 2012; Burke, Hipwell, & Loeber, 2010; Stringaris, Maughan, & Goodman, 2010). For example, in one study, all three dimensions of ODD were related to CD, whereas only negative affect predicted later depression, defiance predicted later behavior disorders, and vindictiveness was related to callous and unemotional behavior (Stringaris & Goodman, 2009). All three dimensions of ODD are highly correlated. However, symptoms of the hurtful behavior dimension do not seem to occur as consistently with symptoms of the other two dimensions, suggesting that they might be more related to the severe conduct problems of CD than to ODD (Burke et al., 2010; Rowe et al., 2010).

DSM-5 organizes ODD symptoms into three symptom clusters, which is consistent with research: *angry/irritable mood* (i.e., loses temper, touchy or easily annoyed, angry and resentful); *argumentative/defiant behavior* (i.e., argues with or defies authority figures, annoys or blames others); and *vindictiveness* (i.e., spiteful and vindictive) (see Table 9.1).

DSM-5 uses severity ratings for ODD of "mild," "moderate," or "severe," depending on whether symptoms are present in one, two, or three or more settings. Settings are home, school, and with peers.

**TABLE 9.1** | Diagnostic Criteria for Oppositional Defiant Disorder

**DSM-5**

**(A)** A pattern of angry/irritable mood, argumentative/defiant behavior, or vindictiveness lasting at least 6 months as evidenced by at least four symptoms from any of the following categories, and exhibited during interaction with a least one individual who is not a sibling.

**Angry/Irritable Mood**

**(1)** Often loses temper.

**(2)** Is often touchy or easily annoyed.

**(3)** Is often angry or resentful.

**Argumentative/Defiant Behavior**

**(4)** Often argues with authority figures or, for children and adolescents, with adults.

**(5)** Often actively defies or refuses to comply with requests from authority figures or with rules.

**(6)** Often deliberately annoys others.

**(7)** Often blames others for his or her mistakes or misbehavior.

**Vindictiveness**

**(8)** Has been spiteful or vindictive at least twice within the past 6 months.

*Note:* The persistence and frequency of these behaviors should be used to distinguish a behavior that is within normal limits from behavior that is symptomatic. For children younger than 5 years, the behavior should occur on most days for a period of at least 6 months unless otherwise noted (Criterion A8). For individuals 5 years or older the behavior should occur at least once per week for 6 months, unless otherwise noted (Criterion A8). While these frequency criteria provide guidance on a minimal level of frequency to define symptoms, other factors should also be considered, such as whether the frequency and intensity of the behaviors are outside a range that is normative for the individual's developmental level, gender, and culture.

**(B)** The disturbance in behavior is associated with distress in the individual or others in his or her immediate social context (e.g., family peer group, work colleagues), or it impacts negatively on social, educational, occupational, or other important areas of functioning.

**(C)** The behaviors do not occur exclusively during the course of a psychotic, substance-use, depressive, or bipolar disorder. Also, the criteria are not met for disruptive mood disorder.

Specify current severity:

**Mild:** Symptoms are confined to only one setting (e.g., at home, at school, at work, with peers).

**Moderate:** Some symptoms are present in at least two settings.

**Severe:** Some symptoms are present in three or more settings.

*Source:* Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition. American Psychiatric Association.

Most clinic-referred children with ODD (about 90%) display symptoms in two or more settings, but even those who show impairments only at home still display significant adjustment problems, albeit not as severe as those of children who display impairments across multiple settings (Kimonis et al., 2014).

## Conduct Disorder (CD)

### GREG

### Dangerous Distress

Greg, age 10, was referred because of his excessive fighting, hyperactivity, temper tantrums, and disruptive behavior at home and at school. At home, Greg argued with his mother, started fights with his siblings, stole from his parents, and constantly threatened to set fires when disciplined. On three separate occasions, he actually had set fires to rugs, bedspreads, and trash in his home. One fire led to several thousand dollars in damages. Greg also lied frequently; at school, his lying got others into trouble, precipitating frequent fights with peers and denials of any wrongdoing.

Greg was brought to the clinic because his parents felt that he was becoming totally unmanageable. A few incidents were mentioned as unusually dangerous—for example, Greg's attempt to suffocate his 2-year-old brother by holding a pillow over his face. Also, Greg had recently wandered the streets at night and had broken windows of parked cars.

Greg's parents occasionally resorted to severe punishment, using paddles and belts, or locking him in his room for 2 to 3 days. His father has been employed only sporadically for the past 2 years, and spent much of his time at home sleeping or watching TV. The loss of income led to increased stress. Greg said that he could not stand to be with his dad because his dad got mad all the time over little things. Greg's mother worked full time and was not at home very much. She had a history of depression, with two suicide attempts in the past 3 years. She was hospitalized on each occasion for approximately 2 months. Greg's behavior became even worse during these periods.

Although Greg's intelligence was within the normal range, his academic performance was behind grade level, and he was in a special class because of his overactive and disruptive behavior. His parents were told that unless they got help, Greg could not return to the school the next year. His parents did not know where to turn. They talked about giving Greg up or putting him in a special boarding school, where more discipline might make him "shape up."

Based on Conduct Disorders in Childhood and Adolescence by A. E. Kazdin, pp. 2–3.

Greg's chronic and unmanageable behavior qualifies for a diagnosis of **conduct disorder (CD)**. Children with conduct disorder display a repetitive and persistent pattern of severely aggressive and antisocial acts that involve inflicting pain on others or interfering with the rights of others through physical and verbal aggression, stealing, or vandalism. The DSM-5 groups symptoms of CD into four dimensions: aggression to people and animals (e.g., bullying, physical cruelty), destruction of property (e.g., fire setting, vandalism), deceitfulness or theft (e.g., conning, shoplifting), and serious violations of rules (e.g., truancy, running away from home). (see Table 9.2 for DSM-5 diagnostic criteria for CD).

Greg's case illustrates several key features of CD (Kazdin, 1995):

- Children with CD engage in severe antisocial behaviors. Greg set fires and tried to suffocate his 2-year-old brother. He also displayed less severe problems, such as noncompliance and temper tantrums, but these weren't the main reasons for referral. DSM-5 includes severity ratings for CD of "mild," "moderate," and "severe" based on the number of symptoms in excess of the three required to make the diagnosis or the amount of harm caused to others.
- They often have co-occurring problems such as ADHD, academic deficiencies, and poor relations with peers.
- Their families often use child-rearing practices, such as harsh punishment, that contribute to the problem and often have their own problems and stresses, such as marital discord, psychiatric problems, and unemployment. Greg's mother had a history of depression and his father was frequently unemployed.
- Their parents feel these children are out of control, and they feel helpless to do anything about it. Greg's parents want to give him up or put him in a boarding school.

### *CD and Age at Onset*

Is the age at which symptoms of CD first occur important? DSM makes the distinction between youths with an early or late onset of CD. Those with **childhood-onset conduct disorder** display at least one symptom of the disorder before age 10, whereas those with **adolescent-onset conduct disorder** do not. Increasing evidence points to the importance of age at onset in diagnosing and treating children with CD (Odgers et al., 2008). Children diagnosed with childhood-onset CD are more likely to be boys, show more aggressive symptoms, account for a disproportionate amount of

**TABLE 9.2** | Diagnostic Criteria for **Conduct Disorder**

**DSM-5**

**(A)** A repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated, as manifested by the presence of at least three of the following 15 criteria in the past 12 months from any of the categories below, with at least one criterion present in the past 6 months:

**Aggression to People and Animals**

**(1)** Often bullies, threatens, or intimidates others.

**(2)** Often initiates physical fights.

**(3)** Has used a weapon that can cause serious physical harm to others (e.g., a bat, brick, broken bottle, knife, gun).

**(4)** Has been physically cruel to people.

**(5)** Has been physically cruel to animals.

**(6)** Has stolen while confronting a victim (e.g., mugging, purse snatching, extortion, armed robbery).

**(7)** Has forced someone into sexual activity.

**Destruction of Property**

**(8)** Has deliberately engaged in fire setting, with the intention of causing serious damage.

**(9)** Has deliberately destroyed others' property (other than by fire setting).

**Deceitfulness or Theft**

**(10)** Has broken into someone else's house, building, or car.

**(11)** Often lies to obtain goods or favors or to avoid obligations (i.e., "cons" others)

**(12)** Has stolen items of nontrivial value without confronting a victim (e.g., shoplifting, but without breaking and entering; forgery).

**Serious Violations of Rules**

**(13)** Often stays out at night despite parental prohibitions, beginning before age 13 years.

**(14)** Has run away from home overnight at least twice while living in parental or parental surrogate home, or once without returning for a lengthy period.

**(15)** Is often truant from school, beginning before age 13 years

**(B)** The disturbance in behavior causes clinically significant impairment in social, academic, or occupational functioning.

**(C)** If the individual is 18 years or older, criteria are not met for Antisocial Personality Disorder.

*Specify* whether:

**Childhood-onset type:** Individuals show at least one symptom characteristic of conduct disorder prior to age 10 years.

**Adolescent-onset type:** Individuals show no symptom characteristic of conduct disorder prior to age 10 years.

**Unspecified onset:** Criteria for a diagnosis of conduct disorder are met, but there is not enough information available to determine whether the onset of the first symptom was before or after age 10 years.

*Specify* if:

**With limited prosocial emotions:** To qualify for this specifier, an individual must have displayed at least two of the following characteristics persistently over at least 12 months and in multiple relationships and settings. These characteristics reflect the individual's typical pattern of interpersonal and emotional functioning over this period and not just occasional occurrences in some situations. Thus, to assess the criteria for the specifier, multiple information sources are necessary. In addition to the individual's self-report, it is necessary to consider reports by others who have known the individual for extended periods of time (e.g., parents, teachers, co-workers, extended family members, peers).

**Lack of remorse or guilt:** Does not feel bad or guilty when he or she does something wrong (excludes remorse when expressed only when caught and/or facing punishment). The individual shows a general lack of concern about the negative consequences of his or her actions. For example, the individual is not remorseful after hurting someone or does not care about the consequences of breaking rules.

**Callous–lack of empathy:** Disregards and is unconcerned about the feelings of others. The individual is described as cold and uncaring. The person appears more concerned about the effects of his or her actions on himself or herself, rather than their effects on others, even when they result in substantial harm to others.

**Unconcerned about performance:** Does not show concern about poor/problematic performance at school, at work, or in other important activities. The individual does not put forth the effort necessary to perform well, even when expectations are clear, and typically blames others for his or her poor performance.

**Shallow or deficient affect:** Does not express feelings or show emotions to others, except in ways that seem shallow, insincere, or superficial (e.g., actions contradict the emotion displayed; can turn emotions "on" or "off" quickly) or when emotional expressions are used for gain (e.g., emotions displayed to manipulate or intimidate others).

(*continues*)

**TABLE 9.2** | **Diagnostic Criteria for Conduct Disorder** (*continued*)

*Specify* severity:

**Mild:** Few if any conduct problems in excess of those required to make the diagnosis are present, and conduct problems cause relatively minor harm to others (e.g., lying, truancy, staying out after dark without permission, other rule breaking).

**Moderate:** The number of conduct problems and the effect on others are intermediate between those specified in "mild" and those in "severe" (e.g., stealing without confronting a victim, vandalism).

**Severe:** Many conduct problems in excess of those required to make the diagnosis are present, or conduct problems cause considerable harm to others (e.g., forced sex, physical cruelty, use of a weapon, stealing while confronting a victim, breaking and entering).

*Source:* Diagnostic and Statistical Manual of Mental Disorders, 5th ed. American Psychiatric Association.

illegal activity, and persist in their antisocial behavior over time (Lahey, Goodman, et al., 1999). In contrast, youths diagnosed with adolescent-onset CD are as likely to be girls as boys and do not display the severity or psychopathology that characterizes the childhood-onset group. They are also less likely to commit violent offenses or to persist in their antisocial behavior as they get older. Age at onset does make a difference.

### *The ODD and CD Connection*

There is much overlap between the symptoms of ODD and CD. This raises the question of whether ODD is a separate disorder from CD; a milder, earlier version; or a reflection of the same underlying temperament and deficits (Waldman & Lahey, 2013). Symptoms of ODD typically emerge 2 to 3 years before CD symptoms, at about 6 years of age for ODD versus 9 years for CD (Nock et al., 2007). Since ODD symptoms emerge first, it is possible that they are precursors of CD for some children. However, nearly half of all children with CD have no prior ODD diagnosis (Rowe et al., 2010), and most children who display ODD do not progress to more severe CD—at least 50% maintain their ODD diagnosis without progressing, and another 25% cease to display ODD problems entirely (Burke et al., 2010). Thus, for most children, ODD is an extreme developmental variation and a strong risk factor for later ODD and other problems, but not one that necessarily signals an escalation to more serious conduct problems (Keenan et al., 2011). Thus, ODD and CD appear to be distinguishable yet highly correlated aspects of child psychopathology.

## Antisocial Personality Disorder (APD) and Psychopathic Features

Persistent aggressive behavior and CD in childhood may be a precursor of adult **antisocial personality disorder (APD)**, a pervasive pattern of disregard for, and violation of, the rights of others, including repeated illegal behaviors, deceitfulness, failure to plan ahead, repeated physical fights or assaults, reckless disregard for the safety of self or others, repeated failure to sustain work behavior or honor financial obligations, and a lack of remorse (APA, 2013). Research has found that as many as 40% of children with CD develop APD as young adults (Lahey et al., 2005). In addition to their early CD, adolescents with APD may also display **psychopathic features**, which are defined as a pattern of callous, manipulative, deceitful, and remorseless behavior—the more menacing side of human nature (Blair et al., 2006). Consider these chilling comments by Jason.

**JASON**

### No Conscience

Jason, age 13, had been involved in serious crime—including breaking and entering, thefts, and assaults on younger children—by age 6. Listening to Jason talk was frightening. Asked why he committed crimes, this product of a stable, professional family replied, "I like it. My f___ parents really freak out when I get in trouble, but I don't give a sh ___ as long as I'm having a good time. Yeah, I've always been wild." About other people, including his victims, Jason had this to say: "You want the truth? They'd screw me if they could, only I get my shots in first." He liked to rob homeless people, especially "f_gots," "bag ladies," and street kids, because, "They're used to it. They don't whine to the police. … One guy I got into a fight with pulled a knife and I took it and rammed it in his eye. He ran around screaming like a baby. What a jerk!"

Adapted from Hare, 1993, p. 162.

Like Jason, youths who display psychopathic features appear to be aware that their aggressive behavior will cause others to suffer—but they don't care. Rather, their goals in conflict situations involve revenge,

dominance, and forced respect (Pardini, 2011). Although less is known about psychopathic features in children than in adults this situation is changing. Signs of a lack of conscience occur in some children as young as 3 to 5 years (Kochanska et al., 1994). Other children, like Jason, began committing brutal acts of violence at age 6 with little remorse. A subgroup of preschoolers with behavior problems show a worrisome increase in their lack of concern for others as they begin to enter middle childhood (Hastings et al., 2000). Finally, adolescents with CD are less likely than peers to show affective empathy or embarrassment, which suggests a failure to inhibit emotions and actions in accordance with social conventions (Lovett & Sheffield, 2007).

These and many other findings point to a subgroup of children with CD whose lack of concern for others may place them at especially high risk for extreme antisocial and aggressive acts and for poor long-term outcomes. They display a **callous and unemotional (CU) interpersonal style** characterized by an absence of guilt, lack of empathy, uncaring attitudes, shallow or deficient emotional responses, and related traits of narcissism and impulsivity (Frick et al., 2014; Kahn et al., 2012). Children with CU traits display a greater number and variety of conduct problems, and they have more frequent contact with police and a stronger parental history of APD than other children with conduct problems (Frick & White, 2008). Research with children and adolescents has found that CU interpersonal and affective traits predict persistent delinquency, future recidivism, and symptoms of APD in early adulthood (Byrd, Loeber, and Pardini, 2012; McMahon et al., 2010; Pardini & Loeber, 2008). CU symptoms in childhood are about as stable as ODD and CD symptoms over time, but developmental changes have also been noted, suggesting that these are not unchanging characteristics of the child. For example, some children display stable high levels of CU traits, others show increasing or decreasing levels, and others show stable low levels (Fontaine et al., 2011). CU traits in childhood and early adolescence are likely precursors of adult forms of psychopathy, although further research is needed to confirm this (Lynam et al., 2007).

Given the evidence in support of CU traits in identifying an important characteristic of children with conduct problems, DSM-5 uses the specifier "**with limited prosocial emotions**" to describe youth with CD who display a persistent and typical pattern of interpersonal and emotional functioning involving at least two of the following three characteristics: lack of remorse or guilt; callous–lack of empathy; and unconcerned about performance. The term "limited prosocial emotions" was used, in part, in DSM-5 to avoid the possible negative connotations associated with the term "callous-unemotional" (Frick & Nigg, 2012).

At this point you might want to consider A Closer Look 9.2 to sharpen your knowledge of DSM-5criteria for ODD and CD by considering whether or not TV cartoon personality Bart Simpson qualifies for a diagnosis of one, both, or neither of these disorders.

## Section Summary

### DSM-5: Defining Features

- Children with oppositional defiant disorder (ODD) display an age-inappropriate pattern of stubborn, hostile, and defiant behaviors that reflect symptoms of emotionality

### A CLOSER **LOOK 9.2**

### Bart Simpson: What's the Diagnosis?

Sharpen your knowledge of DSM-5 criteria for ODD and CD by considering whether TV cartoon personality Bart Simpson qualifies for a diagnosis of one, both, or neither of these disorders. Here is a list of antisocial acts displayed by Bart:

- Flushes a cherry bomb down the toilet
- Rearranges party snacks to say "Boy our party sucks"
- Loosens the top on Milhouse's salt shaker
- Lights Homer's tie on fire
- Tricks Flanders kids into giving cookies away
- Pretends to be Timmy (trapped in a well)
- Blames Lisa for making long distance calls
- Pulls carpet up, writes "Bart" on carpet
- Plays with and later breaks grandpa Abe's false teeth
- Flushes Homer's wallet and keys down toilet
- Cuts all of baby Maggie's hair off
- Paints extra lines on parking lot
- Leaves box factory tour
- Pops heads off Mr. Burns's statues/floods his car
- Smashes Mr. Burns's windows
- Recounts throwing mail in sewer with Milhouse
- Phones 911 to get babysitter into trouble

*Comment:* Based on Bart's symptoms of aggression, destruction of property, deceitfulness, and serious violation of rules, he easily qualifies for a DSM diagnosis of CD. Like most children with CD, Bart also displays symptoms of ODD (e.g., not complying with rules, deliberately annoying others, blaming others for his misbehavior, engaging in spiteful behavior) and qualifies for this diagnosis as well. (Based on authors' case material.)

and temperamental activity. ODD symptoms can be grouped into three dimensions: negative affect, defiance, and vindictiveness.

- Conduct disorder (CD) describes children who display severe aggressive and antisocial acts involving inflicting pain upon others or interfering with the rights of others through physical and verbal aggression, stealing, or committing acts of vandalism.
- Children who display childhood-onset CD (before age 10) are more likely to be boys, show more aggressive symptoms, account for a disproportionate amount of illegal activity, and persist in their antisocial behavior over time.
- Children with adolescent-onset CD are as likely to be girls as boys and do not display the severity or psychopathology that characterizes the childhood-onset group.
- There is much overlap between CD and ODD. However, most children who display ODD do not progress to more severe CD.
- Persistent aggressive behavior and conduct problems in childhood may be a precursor of adult antisocial personality disorder (APD), a pervasive pattern of disregard for, and violation of, the rights of others.
- A subgroup of children with conduct problems display psychopathic features, including callous–unemotional (CU) traits such as lacking in guilt, not showing empathy, and not displaying feelings or emotions. These children also display a preference for novel and perilous activities and a diminished sensitivity to cues for danger and punishment when seeking rewards.
- DSM-5 uses the specifier "with limited prosocial emotions" to describe youth with CD who display a pattern of interpersonal and emotional functioning involving a lack of remorse or guilt, empathy, or concern about performance.

## ASSOCIATED CHARACTERISTICS

Many child, family, peer, school, and community factors are associated with conduct problems in youths. Some factors co-occur with conduct problems, others increase their likelihood, and still others are the result of these problems. To fully understand conduct problems, we must examine these various factors and how they interact over time.

### Cognitive and Verbal Deficits

Although most children with conduct problems have normal intelligence, they score nearly 8 points lower than their peers on IQ tests (Pajer et al., 2008). This IQ deficit may be greater (more than 15 points) for children with childhood-onset CD, and cannot be accounted for solely by socioeconomic disadvantage, race, or detection by the police (Lynam, Moffitt, & Stouthamer-Loeber, 1993). Lower IQ scores in children with CD may be related to the co-occurrence of ADHD (Waschbusch, 2002). When ADHD is also present, the association between a lower IQ and an increased risk for CD is clear. It is less clear how a lower IQ mediates this risk (Rutter, 2003b).

Verbal IQ is consistently lower than performance IQ in children with CD, suggesting a specific and pervasive deficit in language (Zadeh, Im-Bolter, & Cohen, 2007). This deficit may affect the child's receptive listening, reading, problem solving, pragmatic language, expressive speech and writing, and memory for verbal material (Brennan et al., 2003; Gremillion & Martel, 2013; Jaffee & D'Zurilla, 2003). Some suggest that verbal and language deficits may contribute to conduct problems by interfering with the development of self-control, emotion regulation, or the labeling of emotions in others, which may lead to a lack of empathy (Hastings et al., 2000). One study found that poor language ability predicted the development of later conduct problems, suggesting that targeting language deficits may be useful in preventing or treating conduct problems (Peterson et al., 2013).

Verbal deficits are present early in a child's development, long before the emergence of conduct problems. However, their presence alone does not predict future aggression—family factors are also important. Children with both verbal deficits and family adversity display four times as much aggressive behavior as children with only one factor (Moffitt, 1990). Thus, verbal deficits may increase the child's vulnerability to the effects of a hostile family environment. How this occurs is not known, but one possibility is that a child's verbal deficits may make it more difficult for parents to understand their child's needs, which leads to parents' frustration, fewer positive interactions, more punishment, and greater difficulties in teaching social skills (Patterson, 1996). Verbal deficits, such as poor receptive language skills, may also lead to rejection by mainstream peers, adding to the development of conduct problems (Menting, van Lier, & Koot, 2011).

It is important to keep in mind that the relationship between different cognitive/verbal deficits and antisocial behavior may vary for specific types of antisocial behaviors. For example, one study found that verbal abilities were negatively related to physical aggression but positively associated with theft, and that inductive reasoning was negatively associated with increases in theft across adolescence (Barker et al., 2011). These findings highlight the importance of studying specific types of conduct problem behaviors in order to better understand their possible underlying mechanisms.

Children with conduct problems rarely consider the future consequences of their behavior or its impact


"How am I supposed to think about consequences before they happen?"

David Sipress/Cartoon Bank.com

on others. They fail to inhibit their impulsive behavior, keep social values or future rewards in mind, or adapt their actions to changing circumstances. This pattern suggests deficits in executive functions similar to those of children with ADHD (Raine et al., 2005). Because ODD/CD and ADHD frequently co-occur, the observed deficits in executive functions in these children could be due to the presence of co-occurring ADHD (Pennington & Ozonoff, 1996).

It is also possible that the types of executive functioning deficits experienced by children with ODD and CD may differ from those experienced by children with ADHD (Nigg et al., 2006). For example, Rubia (2010) has made the distinction between *cool* (as in temperature, not as in Lady Gaga) cognitive executive functions, such as attention, working memory, planning, and inhibition, and *hot* executive functions that involve incentives and motivation. Both cool and hot executive functions are associated with distinct but interconnected brain networks. Cool executive function deficits are thought to be more characteristic of children with ADHD, whereas hot executive function deficits are more characteristic of children with conduct problems. Children with both ADHD and conduct problems, which is common, likely display a combination of the two types of executive function deficits.

## School and Learning Problems

*Every time you stop a school, you will have to build a jail.*

—Mark Twain (November 23, 1900)

Children with conduct problems display many school difficulties, including academic underachievement, grade retention, special education placement, dropout, suspension, and expulsion (Roeser & Eccles, 2000). Although the frustration and demoralization associated with school failure can lead to antisocial behavior in some children (Maughan, Gray, & Rutter, 1985), there is little evidence that academic failure is the primary cause of conduct problems, particularly in early childhood. Since many young children display patterns of disruptive behavior long before they enter school, it is more likely that a common factor, such as a neuropsychological or language deficit, lack of self-control, or socioeconomic disadvantage, underlies both conduct problems and school difficulties (Lahey & Waldman, 2003).

Over time, underachievement and conduct problems influence each other. Subtle early language deficits may lead to reading and communication difficulties, which in turn may heighten conduct problems in elementary school. Children with poor academic skills are increasingly likely to lose interest in school and to associate with delinquent peers. By adolescence, the relationship between conduct problems and underachievement is firmly established, which, in one pathway, may lead to anxiety or depression in young adulthood (Masten et al., 2005).

## Family Problems

*He is so violent with his sister. He split her lip a couple of times. And he almost knocked her out once when he hit her over the head with a 5-pound brass pitcher. He's put plastic bags over her head.*

—Webster-Stratton and Herbert (1994)

Family problems are among the strongest and most consistent correlates of conduct problems (Dishion & Patterson, 2006). Two types of family disturbances are related to these problems in children. *General family disturbances* include parental mental health problems, a family history of antisocial behavior, marital discord, family instability, limited resources, and antisocial family values. *Specific disturbances in parenting practices and family functioning* include excessive use of harsh discipline, lack of supervision, lack of emotional support and involvement, and parental disagreement about discipline.

The two types are interrelated, since general family disturbances such as maternal depression often lead to poor parenting practices that can lead to antisocial behavior and feelings of parental incompetence that may lead to increased maternal depression, which completes the circle.

High levels of conflict are common in families of children with conduct problems. So, too, are poor parenting practices such as ineffective discipline, negative control, inappropriate use of punishment and rewards, failure to follow through on commands, and a lack

of involvement in child rearing (Larsson et al., 2008; Trentacosta & Shaw, 2008). Parents may also exhibit social–cognitive deficits similar to those of their children, which suggests that the tendency of antisocial children to infer hostile intent of others may mirror the social perceptions of their parents (Healy et al., 2013). Finally, there is often a lack of family cohesion, which is reflected in emotional detachment, poor communication and problem solving, low support, and family disorganization (Henggeler, Melton, & Smith, 1992). Household chaos—characterized by high noise levels, crowding, people coming and going all the time—and a lack of predictability and family routines is also associated with child conduct problems (Deater-Deckard et al., 2009).

From Cain and Abel to TV's Bart and Lisa Simpson, conflict between siblings has generated much attention (Johnston & Freeman, 1998). Conflict is especially high between children with conduct problems and their siblings. Nonreferred siblings sometimes display as much negative behavior as their referred siblings, even when the sibling with conduct problems is not present (Dishion & Patterson, 2006). This suggests that their difficulties are not simply immediate reactions to the annoying behaviors of their antisocial brother or sister. There are many possible reasons for the similarities in the problem behaviors of siblings, including poor parenting practices, the effects of modeling, direct influence of the other sibling, marital discord, parent mental health problems, and shared hereditary influences. Whatever the reasons, the collaboration of siblings in one another's deviant behavior can be as powerful as deviant peer relationships in heightening the risk for later conduct problems, and may also contribute to later aggression toward peers (Ensor et al., 2010).



Conflict in families of children with conduct problems is common.

In this section, we have described many common problems in families of children with conduct problems. Later in this chapter, we consider how these family problems might combine with other factors to cause additional difficulties.

## Peer Problems

*He is so aggressive around other children. We can't really trust him not to walk up and wallop the smaller ones. He pokes them in the eyes or pushes them down. … It's almost like he seeks out other children to hurt them.*

—Webster-Stratton and Herbert (1994)

Young children with conduct problems display verbal and physical aggression toward other children as well as poor social skills (Miller & Olson, 2000). Preschoolers who show poor self-regulation have difficulty understanding the perspectives of others, experience corporal punishment from their parents, and display higher levels of peer aggressiveness during the transition to grade school (Olson et al., 2011). As they grow older, most children with conduct problems are rejected by their peers, although some may remain quite popular (Rodkin et al., 2000). Peer rejection in elementary school is a strong risk factor for adolescent conduct problems. For example, children rejected for 2 or 3 years by grade 2 are about 5 times more likely than others to display conduct problems later in adolescence (Laird et al., 2001). As they enter school, some of these children become bullies, a particularly offensive pattern associated with continuing conduct problems into adolescence and adulthood (see A Closer Look 9.3).

Children with conduct problems are able to make friends. Unfortunately, their friendships are often with like-minded antisocial individuals (Button et al., 2007). Notably, the combination of early antisocial behavior and associating with deviant peers is a powerful predictor of conduct problems during adolescence (Laird et al., 2005). Involvement with antisocial peers becomes increasingly stable during childhood and

## Bullies and Their Victims

For 2 years, Johnny, a quiet 13-year-old, was a human plaything for some of his classmates. The teenagers badgered Johnny for money, forced him to swallow weeds and drink milk mixed with detergent, beat him up in the restroom, and tied a string around his neck, leading him around as a "pet" (Olweus, 1995, p. 196).

Bullying among school children is a very old, familiar, and particularly offensive form of antisocial behavior. **Bullying** occurs when one or more children intentionally and repeatedly expose another child, who cannot readily defend himself or herself, to negative actions (Olweus, 2013). Such actions may take the form of physical contact, offensive words, making faces or dirty gestures, and intentional exclusion from a group. Bullying usually involves an imbalance of power, so that the victim has difficulty defending herself or himself (Guerra, Williams, & Sadek, 2011). The scope of this problem is large. In 2007, about 32% of students 12 to 18 years of age reported having been bullied at school, most commonly by being made fun of; being the subject of rumors; or being pushed shoved, tripped, or spit on (Robers et al., 2013). Boys are much more likely than girls to bully other children and are also somewhat more likely to be the victims of bullying. Victims are typically perceived as vulnerable, weak, or different. The following comments by high school students capture the diverse factors related to victimization and being seen as different (Guerra et al., 2011):

> You can get bullied because you are weak or annoying or because you are different. Kids with big ears get bullied. Dorks get bullied. You can also get bullied because you think too much of yourself and try to show off. Teacher's pet gets bullied. If you say the right answer too many times in class you can get bullied. … If you do not want to get bullied you have to stay under the radar, but then you might feel sad because no one pays attention to you. (p. 306)

No youth who is seen as different is exempt. For example, at a 2011 White House Conference on the Prevention of Bullying, President Barack Obama admitted that as a youth he was a victim of bullying: "I have to say, with big ears and the name that I have, I wasn't immune. I didn't emerge unscathed" (Brown, 2011). Although victimization by a bully is strongly associated with emotional problems, some children are resilient. Interestingly, in one study, youths with a particular genotype (involving the serotonin transporter 5-HTT gene) were found to be less likely to suffer adverse effects following bullying victimization—another example of gene-environment interaction (Sugden et al., 2010).

Unfortunately, widespread access to the Internet and smart phones has led to new forms of bullying by youths who use electronics to taunt, insult, threaten, harass, or intimidate a peer. Electronic/Internet or cyberbullies may use instant text messaging, e-mails, tweets, defaming websites, and online "slam books" to aggress against peers by circulating rumors,

Radius Images/Masterfile

secrets, insults, and threats to harass, manipulate, and harm their victims (Kowalski et al., 2014). Most cyberbullying occurs outside of school hours, although it is likely that many, if not most, episodes originate in school settings. Media accounts and some studies have reported that cyberbullying is a rapidly growing problem, maybe even be more common than face-to-face bullying. However, some findings suggest that rates of cyberbullying have remained relatively constant in recent years, and are approximately 65% to 75% lower than traditional forms of bullying (Olweus, 2013). There is also a huge overlap between traditional and cyberbullying. About 90% of traditional bullies are also cyberbullies, and about 90% of victims of traditional bullies are also victims of cyberbullies (Olweus, 2013; Raskauskas & Stoltz, 2007). This suggests that the new electronic media have actually created relatively few new victims and bullies. These findings should not downplay or trivialize the significance of cyberbullying but rather serve to provide perspective on its prevalence and nature relative to traditional forms of bullying.

A child's status as a victim or a bully is likely to be stable over time, and victims and bullies display certain typical characteristics. Typical victims are characterized by anxious and submissive patterns of behavior, low self-esteem, and, in the case of boys, by physical weakness. These children send a signal to others that if they are attacked or insulted, they won't retaliate. Typical bullies are distinguished by their aggressiveness toward both peers and adults. They are often impulsive, need to dominate other people, are stronger than other boys, show little empathy for their victims, and derive satisfaction and, often, material gain from inflicting injury and suffering on their victims. One study found that nearly 40% of boys who were bullies in school were later convicted of three or more criminal offenses by the

(*continues*)

(*continued*)

time they were 24 years old (Olweus, 1995). Thus, bullying in school appears to be part of a more general pattern of antisocial behavior.

The high prevalence of bullying and its impact on victims (sadly, some may commit suicide) make it a significant social problem. Studies of long-term outcomes for former bullies and victims provide strong evidence that bullying is not simply a harmless and passing school problem, but one that has serious adjustment and public health consequences with large costs to society (Copeland et al., 2013; Olweus, 2013). To combat this problem, websites (e.g., http://stopbullyingnow.hrsa.gov) and schoolwide interventions and policies that increase awareness of the problem, develop clear rules against bullying, and provide support and protection for victims have been developed and successfully used in countries throughout the world (Karna et al., 2011).

supports the transition to adolescent criminal acts such as stealing, truancy, and substance abuse (Patterson, 1996). In fact, about two-thirds of all recorded youth offenses are committed in the company of two to three peers (Snyder & Sickmund, 2006). Involvement with deviant peers is also one of the strongest predictors of accelerated autonomy and early sexual activity in adolescence (French & Dishion, 2003).

## TOM AND MATTHEW

### Murderous Meeting of Minds

On February 16, 1995, in the small Minnesota town of Delano, 14-year-old Tom and his best friend Matthew ambushed and killed Tom's mother. … These boys spent much time together. They admitted to planning the ambush (one saying they had planned it for weeks, the other, for a few hours). They were armed and waiting when Tom's mother came home from work. One conclusion seems relatively certain: This murder was an unlikely event until these antisocial friends reached consensus about doing it.

Adapted from Hartup, 1996, p. 1.

Friendships between antisocial boys are abrasive, unstable, of short duration, and not very productive (Dishion & Patterson, 2006). Positive exchanges, when they do occur, are compromised by the bossy and coercive behaviors that accompany them. Antisocial friends may engage in "deviant talk," selectively rewarding one another for discussions of rule breaking, but having little to say about prosocial behavior. As a result of this differential reinforcement, they may become more alike in their antisocial tendencies over time, leading to a further escalation in the frequency and variety of their antisocial activities (Piehler & Dishion, 2007).

The fact that deviant peer involvement is an especially strong predictor of substance use, delinquent behavior, and violence makes intervention in this area a high priority. Unfortunately, many well-intentioned programs such as group therapy, unstructured after-school programs, summer programs, or boot camps tend to create groups for youths with conduct problems—the very situation that may produce the most damage (Dishion, Bullock, & Granic, 2002; Gottfredson, 2010).

Aggressive children also show distortions in how they think about social situations. They underestimate their own aggressiveness and its negative impact, and they overestimate the amount of aggression directed at them. Subgroups of aggressive children may think about social situations in different ways. For example, *reactive–aggressive* children (those showing an angry, defensive response to frustration or provocation) display a **hostile attributional bias**, which means they are more likely to attribute hostile and mean-spirited intent to other children, especially when the intentions of others are unclear (e.g., when another child accidentally bumps into him, a reactive–aggressive child is likely to think the other child did it on purpose). In contrast, *proactive–aggressive* children (those who use aggressive behavior deliberately to obtain a desired goal) are more likely to view their aggressive actions as positive and to value social goals of dominance and revenge rather than affiliation (Crick & Dodge, 1996). Proactive-aggressive children display a lack of concern for others, and their solutions to social problems are few in number, mostly aggressive, and inappropriate (Hastings et al., 2000; Dodge & Pettit, 2003). Proactive–aggressive youths are also likely to display reactive–aggression, whereas reactive–aggressive youths display only reactive aggression and are less aggressive overall (Crapanzano, Frick, & Terranova, 2010).

The hostile attributions of children with conduct problems are directly related to similar attributions by their parents. For example, one longitudinal study found that maternal hostile attributions were related to psychosocial adversity, poor parenting behavior, hostile attributions of the child, and child aggression at 5 years of age (Healy et al., 2013). It is important to keep in mind that many children with conduct problems live in highly aggressive and threatening circumstances. In some cases, their bias toward seeing threat and aggression in others may be an accurate reflection of the realities of living in a hostile social world, and their aggressive style of responding may be an adaptive reaction to that world.

## Self-Esteem Deficits

Although many children with conduct problems have low self-esteem, there is little support for the view that low self-esteem is the primary cause of conduct problems. Rather, these problems seem to be related to an inflated, unstable, and/or tentative view of self (Baumeister, Bushman, & Campbell, 2000). For example, aggressive children may overestimate their social competence and acceptance by other children (David & Kistner, 2000). Any perceived threat to their biased view of self (e.g., rejection) may lead to aggressive behavior, which provides a way to avoid a lowering of self-concept (Orobio de Castro et al., 2007). Consistent with this view, self-esteem among youth gang members seems to conform to a pattern in which any increment in self-esteem—from increased status, respect, or prestige—for one group member takes away from what is available for others (Anderson, 1994). Thus, youths with conduct problems may experience high self-esteem that over time permits them to rationalize their antisocial conduct (Menon et al., 2007).

## Health-Related Problems

Young people with persistent conduct problems engage in many behaviors that place them at high risk for personal injuries, illnesses, drug overdoses, sexually transmitted diseases, substance abuse, and physical problems as adults (Odgers et al., 2007a). Rates of premature death (before age 30) due to various causes (e.g., homicide, suicide, accidental poisoning, traffic accident, or drug overdose) are three to four times higher in boys with conduct problems than in boys without these problems (Kratzer & Hodgins, 1997). Antisocial behavior in childhood predicts an early onset and persistence of sexual activity and sexual risk-taking by age 21 (Ramrakha et al., 2007; Wymbs et al., 2013). This exposes young people to more years at risk for contracting sexually transmitted diseases and unwanted pregnancies through contact with multiple partners and a failure to use contraceptives.

Substance-use disorders and adolescent antisocial behavior are strongly associated with each other (Conner & Lochman, 2010). For example, youths who have used or sold drugs are more likely than nonusers to carry a handgun, belong to a gang, use alcohol, or engage in a host of other antisocial behaviors (Snyder & Sickmund, 2006). Adolescent substance abuse is related to the imminent dangers of accidents, violence, school dropout, family difficulties, and risky sexual behavior (Gilvarry, 2000). Early conduct problems are a known risk factor for adolescent substance use (Fergusson, Horwood, & Ridder, 2007). The prevalence of delinquent behavior varies with the severity of substance abuse, with about 10% of adolescents who use multiple drugs committing more than 50% of all felony assaults, felony thefts, and various other reported crimes (Johnston et al., 2012). Thus, the evidence indicates that conduct problems during childhood are a risk factor for adolescent and adult substance abuse, and this relationship is mediated by drug use and delinquency during early and late adolescence (Fergusson et al., 2007; Hopfer et al., 2013). We talk more about substance use problems in Chapter 13, when we discuss health-related disorders.

### Section Summary

#### Associated Characteristics

- Many children with conduct problems show cognitive, verbal, and language deficits, despite their normal intelligence.
- These children experience a variety of school difficulties, including academic underachievement in language and reading, which may result from co-occurring ADHD.
- General family disturbances, and disturbances in parenting practices and family functioning, are among the strongest and most consistent correlates of conduct problems.
- Children with conduct problems have interpersonal difficulties with peers, including rejection and bullying. Their friendships are often with other antisocial children.
- Antisocial behavior may be related to an inflated, unstable, and/or tentative view of self.
- Youths with conduct problems engage in many behaviors that place them at high risk for health-related problems, including personal injuries, illnesses, sexually transmitted diseases, and substance abuse.

# ACCOMPANYING DISORDERS AND SYMPTOMS

Most children with conduct problems suffer from one or more additional disorders, most commonly ADHD, depression, and anxiety (Waldman & Lahey, 2013).

## Attention-Deficit/Hyperactivity Disorder (ADHD)

More than 50% of children with CD also have ADHD. There are several possible reasons for this overlap (Beauchaine, Hinshaw, & Pang, 2010; Rhee et al., 2008), among them:

- A shared predisposing vulnerability such as impulsivity, poor self-regulation, or temperament may lead to both ADHD and CD.

- ADHD may be a catalyst for CD by contributing to its persistence and escalation to more severe forms, particularly when shaped by ineffective parenting emotional reactions and behaviors.
- ADHD may lead to childhood onset of CD, which is a strong predictor of continuing problems.

Despite the large overlap, two lines of research suggest that CD and ADHD are distinct disorders. First, a model that includes both CD and ADHD consistently provides a better fit to the data than a model based on only a single disorder (Waschbusch, 2002). Second, CD is less likely than ADHD to be associated with cognitive impairments, neurodevelopmental abnormalities, inattentiveness in the classroom, and higher rates of accidental injuries (Hinshaw & Lee, 2003).

## Depression and Anxiety

About 50% of youths with conduct problems also receive a diagnosis of depression or anxiety (Wolff & Ollendick, 2006). Some evidence suggests that it is ODD and not CD that best accounts for the connection between conduct problems and depression and that this relationship is driven by the negative mood symptoms of ODD (e.g., anger/irritability) rather than by its behavioral symptoms of defiance (Burke & Loeber, 2010). Boys with combined conduct and internalizing problems have poor outcomes in early adulthood, including having the highest risk of later psychiatric disorders and criminal offenses (Sourander et al., 2007). Most girls with CD develop a depressive or anxiety disorder by early adulthood, and for both sexes, increasing severity of antisocial behavior is associated with increasing severity of depression and anxiety (Zoccolillo et al., 1992). Adolescent CD is also a risk factor for completed suicide in young people with a family history of depression (Renaud et al., 1999).

Findings regarding the relation between anxiety disorders and antisocial outcomes for children with conduct problems are puzzling but quite interesting (Drabick, Ollendick, & Bubier, 2010). In some studies, co-occurring anxiety has been identified as a protective factor that inhibits aggressive behavior (Pine et al., 2000). However, other studies have found that anxiety increases the risk for later antisocial behavior (Rutter, Giller, & Hagell, 1998). Boys with CD and anxiety disorder show a higher level of salivary cortisol associated with a greater degree of behavioral inhibition, which supports the theory of anxiety as a protective factor (McBurnett et al., 1991). In boys with CD only, lower levels of salivary cortisol are directly associated with more aggressive and disruptive behaviors (McBurnett et al., 2000). It has been hypothesized that the relation between anxiety and antisocial outcomes may depend on the type of anxiety. In this formulation, anxiety related to shyness, inhibition, and fear may protect against conduct problems, whereas anxiety associated with negative emotionality and social avoidance/withdrawal based on a lack of caring about others may increase the child's risk for conduct problems (Lahey & Waldman, 2003). Consistent with this view, children with callous–unemotional traits show less anxiety than other children with conduct problems (Frick et al., 1999). It has been proposed that different pathways underlie the relationship between conduct problems and anxiety, such that anxiety may serve as a buffer or facilitator of conduct problems, depending on the underlying conditions (Drabick et al., 2010).

### Section Summary

#### Accompanying Disorders and Symptoms

- About 50% of children with CD also have ADHD. Despite the overlap, CD and ADHD appear to be distinct disorders.
- About 50% of children with conduct problems are diagnosed with depression or a co-occurring anxiety disorder. Symptoms of negative mood associated with ODD best account for the relationship between conduct problems and depression.
- Anxiety related to shyness, inhibition, and fear may protect against conduct problems, whereas anxiety associated with negative emotionality and social avoidance/withdrawal based on a lack of caring about others may increase the child's risk for conduct problems.

# PREVALENCE, GENDER, AND COURSE

In the sections that follow we consider the prevalence of conduct problems, the important role that gender plays in the expression of antisocial behavior, and the different ways that conduct problems emerge over the course of development.

## Prevalence

ODD is more prevalent than CD during childhood, but by adolescence they occur equally often. *Lifetime prevalence estimates* are 12% for ODD (13% for males, and 11% for females), and 8% for CD (9% for males and 6% for females) (Merikangas et al., 2010). The reason overall lifetime prevalence rates are comparable is that ODD either declines or stays constant from early childhood to adolescence, whereas CD increases over the same time period. Prevalence estimates for CD and ODD are similar across cultures, although most comparisons to date have been made between Western

countries rather than between Western and non-Western countries (Canino et al., 2010; Erskine et al., 2013).

## Gender

*In all of the recorded history of the more than ten million animal species, including four thousand mammals which populate the planet, only two species have been documented to engage in warfare… male chimpanzees and male humans.*

—From Eme (2007)

**ANN**

### Runaway

Until recently, Ann, age 13, lived with her mother, stepfather, and younger brother. For the past 6 months, she has been living in a youth shelter under the custody of the courts, because she repeatedly ran away from home. Ann was described by her parents as defiant and argumentative, and she frequently lied and stole. She often stole clothes and jewelry from the homes of relatives and friends, as well as from her parents. … Over the past 3 years, Ann had run away from home on four occasions. Each time, the police had to be called. Running away was precipitated by being grounded for stealing or smoking cigarettes. … One time, Ann was gone for 3 nights. The police found her wandering the streets late at night on the other side of town (about 10 miles from her home). Ann would not tell them who she was or where she lived.

Based on Conduct Disorders in Childhood and Adolescence by A. E. Kazdin, p. 17.

Clear gender differences in the frequency and severity of antisocial behavior are evident by 2 to 3 years of age (Dodge, Coie, & Lynam, 2006). During childhood, rates of conduct problems are about 2 to 4 times higher for boys than for girls, with boys showing an earlier age at onset and greater persistence (Eme, 2007; Lahey et al., 2006). Boys also display more conduct problems and report using more physical aggression than girls across countries throughout the world (Erskine et al., 2013; Lansford et al., 2012). This gender difference does not imply that girls do not display severe conduct problems, including physically aggressive behavior, they just do so much less often than boys.

The gender disparity in conduct problems increases through middle childhood, narrows greatly in early adolescence—due mainly to a rise in covert nonaggressive antisocial behavior in girls (McDermott, 1996)—and then increases again in late adolescence when boys are at the peak of their delinquent behavior (Lahey et al., 2006). Ann steals, lies, and runs away from home, but she is not physically aggressive. In contrast to boys, whose early symptoms of CD are aggression and theft, early symptoms for girls are usually sexual misbehaviors (Offord, Alder, & Boyle, 1986). Antisocial girls are more likely than others to develop relationships with antisocial boys, then become pregnant at an earlier age and display a wide spectrum of later problems, including anxiety, depression, and poor parenting (Foster, 2005).

Although gender differences in the overall amount of antisocial behavior decrease in early adolescence, boys remain more violence-prone than girls throughout their life span, and are more likely to engage in repeated acts of physical violence (Odgers & Moretti, 2002). For conduct problems that are chronic from early childhood to adulthood, the male-to-female ratio is marked, about 10:1. In contrast, more transient forms of antisocial behavior in adolescence show a male-to-female ratio of about 2:1 (Moffitt et al., 2001).

In addition, physical aggression by girls during childhood, when it does occur, does not seem to forecast continued physical violence and other forms of delinquency in adolescence, as it does for boys (Broidy et al., 2003). This does not mean that girls are nonviolent—about 25% of teenage girls commit at least one violent act such as getting into a serious fight at school or work, taking part in a group-against-group fight, or attacking others with the intent to seriously harm them (compared with about 50% of teenage boys) (Substance Abuse and Mental Health Services Administration [SAMHSA], 2009). Interestingly, the sex difference in antisocial behavior has decreased by more than 50% over the past 60 years, suggesting that females may be more susceptible to or more affected by contemporary risk factors, such as family discord or media influences and/or that there is a growing recognition of these problems in girls (Rutter et al., 1998). Unfortunately, antisocial behavior is increasingly becoming an equal

Photofusion Picture Library / Alamy

Girls will be girls.

opportunity affliction—conduct problems are one of the most common mental disorders in adolescent girls.

### *Explaining Gender Differences*

The precise reasons for gender differences in antisocial behavior are not known, although genetic, neurobiological, and environmental risk factors have all been implicated (Eme, 2007; Messer et al., 2006). Genetic and environmental risk factors for antisocial behavior in childhood may also be qualitatively different for males and females (Meier et al., 2011). Gender differences may be partly related to definitions of conduct problems that place a strong emphasis on physical aggression and minimal emphasis on the less physically aggressive forms of antisocial behaviors shown by girls (Crick, Bigbee, & Howes, 1996). When girls are angry they are more likely to use indirect forms of **relational aggression** (see A Closer Look 9.4), such as verbal insults, gossip, tattling, ostracism, threatening to withdraw one's friendship, getting even, or third-party retaliation rather than physical forms of aggression (Cote et al., 2007; Crapanzano et al., 2010). In addition, girls are more likely than boys to become emotionally upset by aggressive social exchanges (Crick, 1995). As girls move into adolescence, the function of their aggressive behavior increasingly revolves around group acceptance and affiliation, whereas for boys, aggression remains confrontational (Crick & Rose, 2000).

Fewer differences in antisocial behaviors exist between boys and girls referred for treatment than for children in community samples. Although boys and girls with conduct problems who are referred to clinics display comparable amounts of externalizing behavior (Dishion & Andrews, 1995), referred girls are more deviant than boys in relation to their same-age, same-sex peers (Webster-Stratton, 1996; Zoccolillo, 1993). Girls' behavior is considered more covert because boys typically engage in more rough and tumble play, bullying, fighting, and noncompliance than girls. With overt antisocial behavior more common in boys, their symptoms are more noticeable at a younger age, which could account for the reported earlier age at onset of conduct problems in boys. Longitudinal research has found that age at onset is prior to age 10 in nearly 90% of girls with CD (Keenan et al., 2010). Although research does not support the development of gender-specific criteria for CD at this time (Frick & Nigg, 2012), it does suggest that further study of relational aggression and callous–unemotional features in girls may prove useful in detecting girls with CD at a younger age (Keenan et al., 2010; Kroneman et al., 2011).

Some girls have an early menarche, which may indirectly heighten their conduct problems by increasing their

## A CLOSER **LOOK 9.4**

### Social Aggression in Girls: "I Hurt Her through the Grapevine"[1]

Over the course of a school day, Rachel Simmons (2002) met with eight groups of 9th-grade girls and began each meeting with the same question:

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Girls are more likely than boys to use indirect forms of social aggression, such as gossiping and spreading rumors.

"What are some of the differences between the ways guys and gals are mean?"

From periods one through eight she heard the same responses: "Girls can turn on you for anything." "Girls whisper." "They glare at you." "They destroy you from the inside." "Girls are manipulative." "There's an aspect of evil in girls that there isn't in boys." "Girls target you when they know you're weakest." "Girls do a lot behind each other's backs." "Girls plan and premeditate."

"In bold, matter-of-fact voices, girls described themselves … as disloyal, untrustworthy, and sneaky. They claimed girls use intimacy to manipulate and overpower others. They said girls are fake, using each other to move up the social hierarchy. They described girls as unforgiving and crafty, lying in wait for a moment of revenge that will catch the unwitting victim off guard, and with an almost savage eye-for-an-eye mentality."

[1]Crick et al. (2001, p. 15)

Adapted from Simmons, 2002, pp. 15–16.

involvement with deviant peers (Burt et al., 2006). Interestingly, an early onset of menarche predicts increased delinquency primarily for girls who attend mixed-gender schools rather than all-girl schools. In mixed-gender schools, exposure to boys who model antisocial behavior and pressure girls for early sexual relations may interact with the early physical maturation of girls. Such exposure may lead to antisocial behavior in these girls, who are more likely to find rewards and opportunities for antisocial activities in the company of boys than girls (Moffitt et al., 1992). In contrast to earlier findings, one study found that girls reaching menarche at an early age had a greater risk of early pregnancy and sexually transmitted infection by age 18, but it found little evidence to indicate that age at menarche was related to later antisocial or criminal behavior (Boden, Fergusson, & Horwood, 2011). Although the reason for these discrepant findings is not known, they do suggest that early age at menarche plays a role in determining sexual behavior outcomes but not necessarily in determining longer-term outcomes in other areas of adjustment, such as antisocial behavior.

Another study found that *both* early maturing girls and early maturing boys were at risk for being exposed to peers who may draw them into delinquent behavior (Negriff, Ji, & Trickett, 2011). However, for early-maturing children who had been maltreated, exposure to abuse had a greater effect on the development of delinquency than did exposure to delinquent peers. These findings suggest that different mechanisms may underlie the relationship between early maturation and delinquency for different groups of children. Most likely, both early-maturing girls and early-maturing boys are vulnerable to an interaction between genetic and environmental risks for delinquent behavior (Harden & Mendle, 2012).

## Developmental Course and Pathways

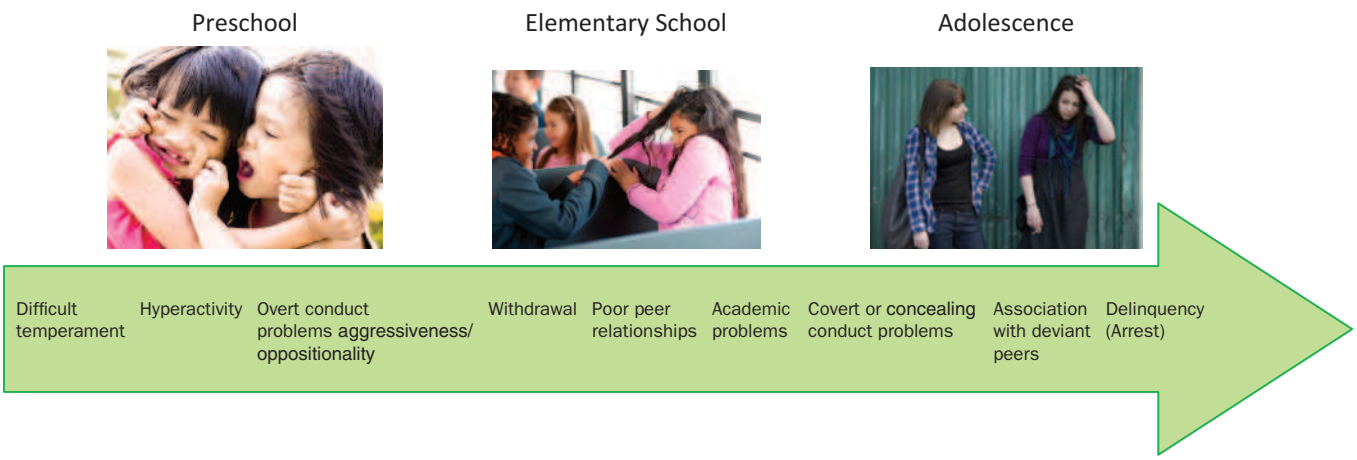
Longitudinal studies have greatly advanced our understanding of antisocial patterns by revealing both a general developmental progression and important variations on this theme (Frick & Viding, 2009).

### *General Progression*

An approximate ordering of the different forms of disruptive behavior and antisocial behavior from early childhood through adolescence is shown in ● Figure 9.3.

Although there are isolated reports, such as that of a 9-month-old infant being expelled from day care for punching other children, early signs of conduct problems are usually not so obvious (Kazdin, 1995, p. 27). The earliest indications of conduct problems may be a *difficult temperament* in the first few years of life, expressed as fussiness, irritability, irregular sleeping and eating patterns, or fearfulness in response to novel events. Interestingly, fussiness in the first year of life was a stronger predictor of later conduct problems in boys, whereas fearfulness was a stronger predictor of later conduct problems in girls (Lahey et al., 2008). As is the case for ADHD, although difficult temperament often precedes later conduct problems, it may not be specific to these problems. The evidence regarding early temperament and later conduct problems suggests a general link, but has not yet identified specific aspects of temperament that predict distinct types of maladjustment (Loeber et al., 2009).

During the preschool and early school years, a child with a difficult temperament displays an increase in hyperactivity and impulsivity with growing mobility, weak emotion-regulation skills, and a heightened risk for




● **FIGURE 9.3** | Approximate ordering of the different forms of disruptive and antisocial behavior from childhood through adolescence.

Based on Development and Risk Factors of Juvenile Antisocial Behavior and Delinquencyby R. Loeber, 1990, 'Clinical Psychology Review', 10, 1–41. *Photo Credits (left to right):* © Andrewblue/Dreamstime.com; © iStockphoto.com/ Christopher Futcher; Elena Rostunova/Shutterstock.com

simple forms of oppositional and aggressive behaviors that peak during the preschool years (Tremblay, 2000). Preschoolers with ODD display stubbornness, temper tantrums, irritability, and spitefulness—problems that remain stable from 2 to 5 years of age. Discipline problems and poor self-control and emotion regulation during early childhood, especially when accompanied by harsh parenting and high levels of stress, are strong indicators that the child will continue to experience behavior problems and negative outcomes across nearly every area of life functioning in adolescence and adulthood (Campbell et al., 2010; Fergusson, Boden, & Horwood, 2013; Moffitt et al., 2011).

Most children with conduct problems show *diversification*—they add new forms of antisocial behavior over time rather than simply replacing old behaviors. Poor social skills and social–cognitive deficits often accompany early oppositional and aggressive behaviors, predisposing the child to poor peer relationships, rejection by peers, and social isolation and withdrawal. When the child enters school, impulsivity and attention problems may result in reading difficulties and academic failure. Covert conduct problems, such as truancy or substance use, also begin to appear during the elementary school years and increase into early adolescence. From ages 8 to 12, behaviors such as fighting, bullying, fire setting, vandalism, cruelty to animals and people, and stealing begin to emerge.

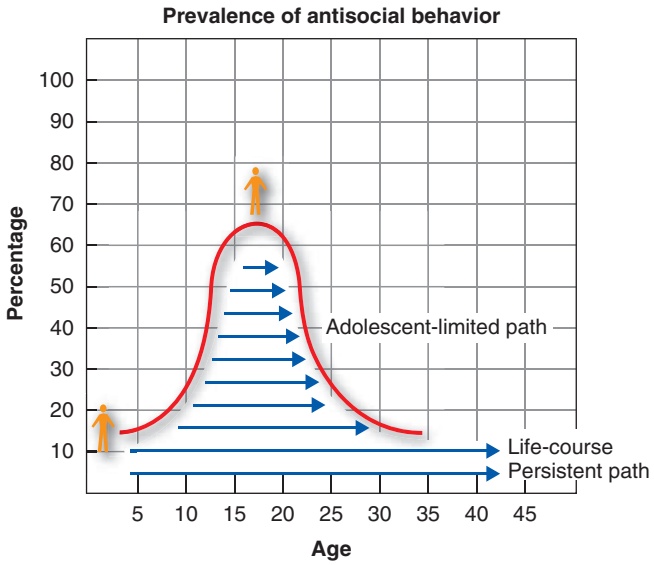
In this progression, we see a snowballing negative cycle over time, where one deficit or problem behavior produces direct and indirect changes in others. For example, peer rejection leads to social–cognitive deficits and aggression; social–cognitive deficits lead to peer rejection and aggression; aggression leads to peer rejection (Lansford et al., 2010). Conversely, better social–cognitive skills may increase peer acceptance and lower aggressiveness. These cascading effects highlight the importance of looking at the progression of antisocial behavior over time as a dynamic developmental process involving relationships among neurobiological dispositions, social environments, cognitions, and behavior (Lansford et al., 2010).

Across cultures, major conduct problems become more frequent during adolescence. Delinquent behavior shows a dramatic rise in middle adolescence and peaks around the age of 17, followed by an equally dramatic drop in late adolescence and young adulthood (Hirschi & Gottfredson, 1983). Adolescence is characterized by a growing association with deviant peers and by increasing rates of arrest, re-arrest, and conviction as the minimum age of criminal responsibility is met. From ages 12 to 14, property destruction, running away from home, truancy, mugging, breaking and entering, use of a weapon, and forced sex occur with increasing frequency (Lahey & Waldman, 2003). By age 18, many young people with conduct problems display antisocial personality development and behaviors that forecast an antisocial future, including substance dependence, unsafe sex, dangerous driving habits, delinquent friends, and unemployment (Moffitt et al., 1996).

Does this developmental progression mean that every young child with conduct problems goes on to become a delinquent adolescent? Definitely not. The sequence in Figure 9.3 shows a maximum progression that begins early in life and persists through adolescence. Although some children display this maximum progression, others will desist their antisocial behavior at a young age. About 50% of children with early conduct problems do improve. Those who do so are those who tend to display less extreme levels of early conduct problems, have higher intelligence and socioeconomic status (SES), fewer delinquent friends, mothers who were not teenagers when they gave birth, and parents with more social skills and fewer mental health problems (Lahey et al., 2002; Nagin & Tremblay, 2001). It is important to note, however, that even among children who display antisocial behavior and then desist at a young age, other problems may emerge in young adulthood, suggesting that their recovery is far from complete (Moffitt et al., 2002). Some children may not display problems until adolescence, and not all children display the full range of difficulties described. Still others may display a chronic low level of persistent antisocial behavior from childhood or adolescence through adulthood (Fergusson & Horwood, 2002). These differences lead us to consider important variations in the general progression.

### Pathways

There are likely as many unique pathways to the development of antisocial behavior as there are children who display these problems. Although a number of different pathways have been identified (Hoeve et al., 2008), evidence across cultures and countries supports two common pathways—the life-course–persistent (LCP) path and the adolescent-limited (AL) path (Moffitt, 2006). As shown in ● Figure 9.4, a small number of individuals with conduct problems (less than 10%) show a persistent pattern of antisocial behavior throughout their lives, whereas the majority display antisocial behavior that occurs mainly during adolescence. Keep in mind that, although the designation of two pathways is a useful way to think about how conduct problems develop in different ways, it is likely that children display a range of levels of severity and continuity over time and that many do not fit neatly on one pathway or the other (Waldman & Lahey, 2013). For example, other pathways that have been identified include the low-level chronic offense path and the adult-onset antisocial behavior path (Loeber et al., 2009).




● **FIGURE 9.4** | The changing prevalence of participation in antisocial behavior across the life span.

Based on Adolescence-Limited and Life-Course-Persistent Antisocial Behavior: A Developmental Taxonomy by T. E. Moffitt, 1993, 'Psychological Review', 100, 674–701.

The **life-course–persistent (LCP) path** describes children who engage in aggression and antisocial behavior at an early age and continue to do so into adulthood (Moffitt et al., 1996). They may display "biting and hitting at age 4, shoplifting and truancy at age 10, selling drugs and stealing cars at age 16, robbery and rape at age 22, and fraud and child abuse at age 30" (Moffitt, 1993, p. 679). Their underlying disposition remains, but the way it is expressed changes with new "opportunities" at different points in development. For these children, antisocial behavior begins early because of subtle neuropsychological deficits that may interfere with their development of language, memory, and self-control, resulting in cognitive deficits and a difficult temperament by age 3 or younger. These deficits heighten the child's vulnerability to antisocial elements in the social environment, such as abuse or poor parenting, which in turn lead to oppositional and conduct problems (Lansford et al., 2011; Moffitt, 1993). These children experience greater social adversity (e.g., maternal insensitivity, single parenting, low income) than their peers from infancy through mid-adolescence (Roisman et al., 2010).

About half of the children who display high levels of childhood-onset antisocial behavior continue on the LCP path by engaging in less serious nonaggressive antisocial behaviors (e.g., stealing and truancy) during middle childhood, followed by affiliation with delinquent peers and more serious delinquent activities during adolescence (Brame, Nagin, & Tremblay, 2001; Dandreaux & Frick, 2009). This subgroup of teens is most likely to commit violent crimes and to drop out of school. LCP youths display consistency in their behavior across situations—for example, by lying at home, stealing from stores, and cheating at school. As young adults, they have difficulty forming lasting relationships and may display a hostile mistrust of others, aggressive dominance, impulsivity, and psychopathic features. Complete spontaneous recovery is rare after adolescence. The LCP path is associated with a family history of externalizing disorders and is often perpetuated by the progressive accumulation of its own consequences (Odgers et al., 2007b). For example, poor self-control and diminished verbal intellect may lead to irreversible decisions, such as dropping out of school or abusing drugs, which further limit opportunities for recovery.

The **adolescent-limited (AL) path** describes youths whose antisocial behavior begins around puberty and continues into adolescence, but who later cease these behaviors during young adulthood. This path includes most juvenile offenders whose antisocial behavior is limited primarily to their teen years (Hamalainen & Pulkkinen, 1996). Teens on the AL path display less extreme antisocial behavior than those on the LCP path, are less likely to drop out of school, and have stronger family ties. Their delinquent activity is often related to temporary situational factors, especially peer influences. The behavior of AL youths is not consistent across situations; they may use drugs or shoplift with their friends while continuing to follow rules and to do well in school. Although these children do not display antisocial behavior in childhood, they do experience, like youngsters on the LCP path, greater social adversity and personal risk during childhood relative to other youths, suggesting that the AL pathway is not simply part of normal adolescent development (Roisman et al., 2010).

## MARCUS

### Call of the Wild

"I grew up in a real poor family. My mom was on welfare all my life—we never had much. As soon as I got to the age of 11, I was interested in other kids who were breaking the rules. I used to see what they used to do—and what they had."

Marcus joined a gang when he turned 13. Two years later, after a number of arrests and four detentions in a juvenile facility, he became disillusioned with gang life and managed to turn his life around. He is now 17 and works as a youth minister for a church dedicated to helping other young people like himself.

Adapted from Goldentyer, 1994.

The attraction of still-forbidden adult privileges, such as drinking alcohol, driving a car, and having sex, may motivate some youths with few previous risk signs to engage in antisocial behavior as they enter adolescence. These youths may observe their LCP peers obtaining desired adult privileges via illicit means and may mimic their delinquent activities. Eventually, when access to adult privileges becomes available, AL youths cease breaking laws and rely instead on the more adaptive and prosocial behaviors and values they learned prior to adolescence (Moffitt, Lynam, & Silva, 1994).

Contrary to expectations, some youths on the adolescent-limited path continue to display antisocial behavior well into their 20s before they eventually stop. Others do not desist in their 20s at all but continue to display higher-than-normal levels of impulsivity, substance abuse and dependence, property crimes, and mental health problems (Moffitt et al., 2002). Persistence in early adulthood is often the result of *snares,* or outcomes of antisocial behavior that close the door to getting a good job, pursuing higher education, or attracting a supportive partner. Common snares include unplanned parenthood, dropping out of school, addiction to drugs or alcohol, disabling injuries, unemployment or erratic work history, severed family connections, imprisonment, bad reputation, and a delinquent self-image (Moffitt et al., 1994). Thus, despite their potential, some individuals with no history of childhood antisocial behavior who initiate delinquent activity in adolescence continue to experience problems well into adulthood (Moffitt et al., 2002). Therefore, referring to these individuals as "adolescent-limited" is somewhat misleading.

The identification of the LCP and AL pathways (and their variations) helps us understand why adult antisocial behavior is almost always preceded by antisocial behavior during childhood and adolescence (Brame et al., 2001). Nevertheless, most antisocial adolescents do not go on to become antisocial adults. At the crossroads of early adulthood, LCP and most AL teens go different ways. Antisocial behavior is stable for youths on the LCP path, who continue on the same road, but unstable for those on the AL path.

## Adult Outcomes

The number of active offenders decreases by about 50% in the early 20s, and almost 85% of former delinquents desist from offending by their late 20s. This general relationship between age and crime applies to males and females, for most types of crimes, and in numerous Western nations (Caspi & Moffitt, 1995). Most children with conduct problems do not grow up to be antisocial adults (Maughan & Rutter, 2001). However, adult outcomes depend not only on the type and variety of conduct problems developed during childhood and adolescence, but also on the number and combination of risk and promotive factors in the child, family and community (Kokko & Pulkkinen, 2000). Also, even when antisocial behavior decreases in adulthood, coercive interpersonal styles may sometimes persist, along with family, health, and work difficulties.

A significant number of children with conduct problems, particularly those on the LCP path, do go on as adults to display criminal behavior, psychiatric problems, social maladjustment, health problems, lost productivity, and poor parenting of their own children (Fergusson, Horwood, & Ridder, 2005). As adults, they are more likely to be downwardly socially mobile and to display an erratic work history, perhaps because of lower skill attainment and difficulties in getting along with co-workers and supervisors. They also have more violent marriages and cohabitations and higher rates of divorce and are more likely than others to select partners with similar antisocial characteristics, providing the next generation with a double dose of both genetic and environmental risk (Moffitt et al., 2002). One follow-up study of adult women who were arrested for severe conduct problems in adolescence found that most continued to display these problems. A majority had depressive and anxiety disorders, 6% died a violent death, many had dropped out of school, one-third were pregnant before the age of 17 years, half were re-arrested, and many had suffered traumatic physical injuries (Zoccolillo & Rogers, 1991, 1992). Thus, males and females experience different but poor adult outcomes. Males are at higher risk for criminal behavior, work problems, and substance abuse, whereas females are more likely to experience depression, suicidal behavior, and health problems (Moffitt et al., 2001).

## Section Summary

### Prevalence, Gender, and Course

- ODD is more prevalent than CD during childhood, but by adolescence the two occur about equally. The lifetime prevalence rates for ODD and CD are about 12% and 8%, respectively.
- During childhood, conduct problems are about 2 to 4 times more common in boys than in girls. This difference narrows greatly in early adolescence, due mainly to a rise in covert nonaggressive antisocial behavior in girls, and then increases again in late adolescence and beyond.
- Girls are more likely than boys to use indirect forms of relational aggression—for example, verbal insults, gossip, or third-party retaliation.

- There is a general progression of antisocial behavior from difficult early temperament and hyperactivity, to oppositional and aggressive behavior, to social difficulties, to school problems, to delinquent behavior in adolescence, to antisocial personality development, to criminal behavior in adulthood.
- The life-course–persistent (LCP) path describes children who display antisocial behavior at an early age and who continue to do so into adulthood.
- The adolescent-limited (AL) path describes teens whose antisocial behavior begins around puberty and continues into adolescence and who later cease these behaviors in young adulthood.
- A significant number of children with conduct problems continue to experience difficulties as adults, including criminal behavior, psychiatric problems, social maladjustment, health and employment problems, and poor parenting of their own children.

## CAUSES

When it comes to conduct problems, there are no simple or single causes. Consider two brothers—one, John Edgar Wideman, is an award-winning author (*Brothers and Keepers*), while his younger brother Robby is in prison for murder. How do we account for such striking differences between brothers raised in the same family? Are they due to differences in genetic makeup, neurobiological functioning, birth complications, temperament, intelligence, family experiences, peer influences, difficulties in school, or some combination of these factors?

Historically, conduct problems were viewed as either the result of an inborn characteristic or learned through poor socialization practices. Early theories focused mainly on the child's aggression and invoked one primary cause, such as an aggressive drive, frustration, poor role models, or reinforcement. However, most of these "smoking gun" explanations can be challenged on one point or another. For example, not all children behave aggressively, as would be predicted by the aggressive-drive theory, and frustration sometimes leads to cooperation rather than aggression. Although each single-cause theory highlights a potentially important determinant, no single theory can explain all forms of antisocial behavior.

We next consider a wide number and diversity of risk factors for youth conduct problems. Although we examine them separately, conduct problems are best accounted for by the interplay among predisposing child, family, community, and cultural factors that operate in a transactional fashion over time (Granic & Patterson, 2006). It is also important to recognize that subgroups of youths with conduct problems (e.g., child versus adolescent onset, those with CU traits) may have distinct risk factors underlying their antisocial and aggressive behaviors (Kimonis et al., 2014). These risk factors are summarized in Table 9.3.


"We won't know till they're older which one is the evil twin."

Sidney Harris/Cartoon Bank.com

### Genetic Influences

*Heredity is something a man believes in until his own son begins behaving like a delinquent.*

—Author unknown

The universality of aggressive behavior and antisocial behavior in humans and the fact that such behaviors run in families within and across generations highlight the importance of genetic influences. Adoption and twin studies indicate that 50% or more of the variance in antisocial behavior is attributable to heredity for both males and females. This influence is somewhat higher for aggressive versus nonaggressive conduct problems and in childhood versus adolescence. Research indicates that parents pass on a general liability for externalizing disorders to their children that may be expressed in different ways, including oppositional and conduct problems, inattention, and hyperactivity–impulsivity (Bornovalova et al., 2010). The heritability of conduct problems also varies by age at onset and other factors (Burt & Neiderhiser, 2009). For example, the strength of the genetic contribution is higher for children who display the LCP versus the AL pattern

and for those with callous–unemotional traits (Viding et al., 2008). However, all externalizing disorders appear to share substantial genetic influences, suggesting at least some common causal factors among them(Lahey et al., 2011). Overall, adoption and twin studies suggest that both genetic and environmental factors contribute to antisocial behavior across development. The studies do not, however, specify the mechanisms by which the factors operate.

**TABLE 9.3 | Summary of Risk Factors for Antisocial Behaviors**

| | |
|---|---|
| **Child** | Genetic risk, prenatal and birth complications, exposure to lead and other toxins, low arousal and reactivity, anterior and posterior cingulate cortex development, functional and structural deficits in prefrontal cortex, reduced amygdala activity, blunted emotional and cortisol reactivity (CU -type), insensitivity to stress (CU-type), fearlessness/low anxiety (CU-type), difficult temperament, emotion dysregulation, attention-deficit/hyperactivity disorder (ADHD), insecure/disorganized attachments, childhood onset of aggression, social avoidance and withdrawal, social–cognitive deficits (hostile attributional bias), lowered verbal intelligence and verbal deficits, executive functioning deficits |
| **Family** | Antisocial family values, parental antisocial or criminal behavior, paternal antisocial personality disorder, maternal depression, parental substance abuse, marital discord, teen motherhood, single parenthood, family stress/conflict/instability, chaotic household, large family, low socioeconomic status, low education of mother, family carelessness in permitting access to weapons |
| **Ineffective Parenting** | Poor supervision and monitoring, inconsistent discipline, avoidance of discipline due to concerns about the child's reaction, harsh discipline and maltreatment, discordant parent–child interactions, poor communication and problem solving, low parental involvement, parental neglect, low parental warmth, parental hostile attributional bias |
| **Peers** | Early peer aggression, rejection by peers, association with deviant siblings, association with deviant peers, bullying |
| **School** | Poor academic performance, weak bonding to school, low educational aspirations, low school motivation, poorly organized and functioning schools |
| **Neighborhood and Community** | Neighborhood disadvantage and poverty, disorganized neighborhoods, gang membership, availability of weapons |
| **Sociocultural** | Media portrayal of violence, cultural attitudes encouraging use of aggression, socialization of children for aggression |

Adapted from Loeber and Farrington, 2000, p. 749.

It is likely that genetic risks for antisocial behavior operate via several pathways (Rutter, 2003b). First, genetic factors may be related to a difficult temperament, lack of response to distress in others, impulsivity, a tendency to seek rewards, or an insensitivity to punishment that combine to create an antisocial "propensity" or "personality" (Waldman et al., 2011). Second, genetic factors may increase the likelihood that a child will be exposed to environmental risk factors, such as prenatal stress, parental maltreatment, divorce, or other negative life events that are associated with an increased risk of antisocial behavior. Third, children's genotype and neurobiology may moderate their susceptibility to these environmental insults in determining whether they later develop antisocial behavior (Ellis & Boyce, 2011; Zohsel et al., 2014). These and other pathways will need to be addressed if the causes of antisocial behavior are to be understood (Rutter, 2006a).

Exciting new studies into gene variants have identified possible gene–environment (G×E) interactions in the development of conduct problems (Dodge, 2009). A variant of the gene that encodes the neurotransmitter-metabolizing enzyme monoamine oxidase A (MAOA) has been of particular interest because this gene is related to neural systems involved in aggression. When threatened or provoked, humans naturally feel rage and an impulse to react aggressively. Activation of the MAOA enzyme helps us inhibit that response; thus, it plays a key role in regulating behavior following threatening events. Research has found that maltreated children with a low-active MAOA genotype are much more likely to develop antisocial behavior than maltreated children who do not have this genotype (Kim-Cohen et al., 2006). An interaction between the low-active MAOA genotype and physical discipline before age 5 has also been found to be related to adolescent delinquent behavior (Edwards et al., 2010). Brain imaging studies have also found that individuals with the low-active MAOA genotype show patterns of arousal in areas of the brain that are associated with aggression in response to emotion-provoking stimuli (Buckholtz & Meyer-Lindenberg, 2008).

In addition to MAOA, other genes and G×E interactions have been implicated in the association between negative parenting and childhood conduct problems (Albaugh et al., 2010; Lahey et al., 2011). Findings to date suggest that some of these genes may not be specific to any one type of externalizing disorder but will predispose individuals to a broad spectrum of conduct problems (Dick, 2007). G×E interaction effects in conduct disorders are fascinating. However, research into these effects is just beginning, and replication

studies are needed (Hebebrand et al., 2010). Similarly, research into how G×E interaction effects develop over time is still in its infancy (Dodge, 2009).

## Prenatal Factors and Birth Complications

A number of pregnancy and birth factors (e.g., low birth weight) are related to the development of serious conduct problems (Brennan, Grekin, & Mednick, 2003). Malnutrition during pregnancy is associated with later antisocial behavior, which may be mediated by protein deficiency (Raine, 2002). Exposure to lead before and after birth and the mother's use of nicotine, marijuana, and other substances during pregnancy may also be associated with later conduct problems (Carpenter & Nevin, 2010; Gaysina et al., 2013; Murray et al., 2010; Nigg & Breslau, 2007). There is also support for maternal alcohol use during pregnancy playing a role in conduct problems—the greater the amount of alcohol consumed, the greater the risk of child conduct problems (D'Onofrio et al., 2007; Larkby et al., 2011). Although pregnancy and birth factors are correlated with conduct problems, strong evidence of direct biological causation is lacking (Hodgins, Kratzer, & McNeil, 2001). For example, it is likely that the relation between mothers' smoking during pregnancy and adolescent conduct problems and criminality is best accounted for by the transmission of an underlying antisocial tendency from mother to child and other family background variables, rather than by exposure to cigarette byproducts during pregnancy (D'Onofrio et al., 2008, 2010b; Gatzke-Kopp & Beauchaine, 2007).

## Neurobiological Factors

Gray (1987) proposed that people's behavioral patterns are related to two subsystems of the brain, each having distinct neuroanatomical regions and neurotransmitter pathways. The **behavioral activation system (BAS)** stimulates behavior in response to signals of reward or nonpunishment. In contrast, the **behavioral inhibition system (BIS)** produces anxiety and inhibits ongoing behavior in the presence of novel events, innate fear stimuli, and signals of nonreward or punishment. Other behavioral patterns may result from the relative balance or imbalance of activity in these two neural systems. Think of the BAS as similar to the gas pedal and the BIS as similar to the brake pedal—some individuals ride one more heavily than the other.

It has been proposed that antisocial patterns of behavior result from an overactive BAS *and* an underactive BIS—a pattern determined primarily by genetic predisposition. Consistent with an overactive BAS, children with conduct problems show a heightened sensitivity to rewards (Frick et al., 2003). In addition, they fail to respond to punishment and continue to respond under conditions of no reward—patterns that are consistent with an underactive BIS (Fowles, 2001). Strikingly, a lack of fear conditioning at age 3 has been found to predict criminal offending 20 years later (Gao et al., 2010).

Individual differences in antisocial behavior have been related to variations in stress-regulating mechanisms, including the hypothalamic–pituitary–adrenal (HPA) axis and the autonomic nervous system (ANS), serotonergic functioning, and structural and functional deficits in the prefrontal cortex of the brain (van Goozen et al., 2007). Children with CD who show an early onset of aggressive symptoms display low psychophysiological/cortical arousal and low reactivity of the autonomic nervous system (e.g., a lower resting heart rate) (Lorber, 2004; Raine, 2002). Low arousal and autonomic reactivity may lead to diminished avoidance learning in response to warnings or reprimands, a poor response to punishment, and a fearless, stimulus-seeking temperament. In turn, this may lead to antisocial behavior, a failure to develop the anticipatory fear needed to avoid such behavior, and a lack of conscience. Most children respond to discipline and punishment by reducing their antisocial behavior. Often, the opposite occurs with children with conduct problems—when disciplined or punished they may increase their antisocial behavior and become even more defiant (Briggs-Gowan et al., 2014).

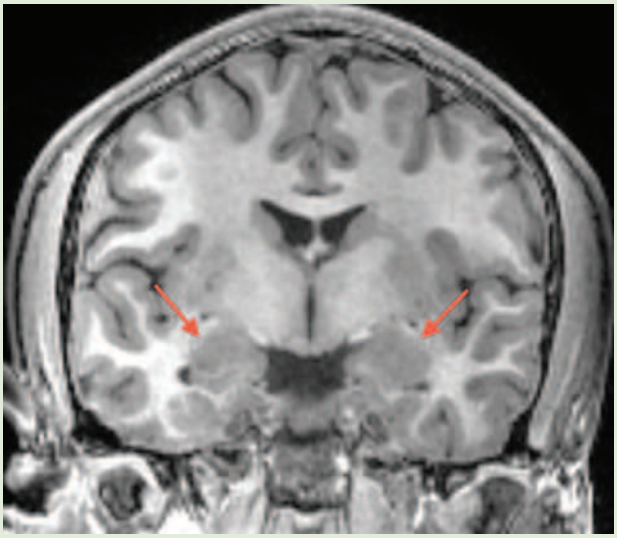
Neuroimaging studies have identified structural and functional brain abnormalities in several brain regions in youths with conduct disorders, including in those with high levels of psychopathic features (Pardini et al., 2013). These brain regions include the amygdala, prefrontal cortex, posterior and anterior cingulate, and insula, as well as interconnected regions. Imaging studies also show reduced activation in some of these areas (e.g., the amygdala) when viewing emotional stimuli such as angry or sad faces, or during tasks that require learning not to respond to punishing stimuli (Finger et al., 2011; Huebner et al., 2008; Passamonti et al., 2010). These brain regions are involved in processing social and emotional information. Therefore, abnormalities in these regions likely underlie the social–cognitive and emotional deficits that characterize youths with conduct problems. Interestingly, as described in A Closer Look 9.5, similar brain abnormalities may be present both in youths with early-onset and those with adolescent-onset conduct disorders (Fairchild et al., 2011).

Although much additional work is needed, early findings from neuroimaging and other studies suggest three neural systems underlying cognitive, social, and emotional differences across different types of conduct problems. The first includes subcortical neural systems that lead to

A CLOSER **LOOK 9.5**

## Do the Brains of Children with Early-Onset Conduct Disorders Differ from Those of Children with Adolescent-Onset Conduct Disorders?

Despite the many differences between children with early-onset and those with adolescent-onset CD, brain-imaging findings suggest that the two groups may display similar brain abnormalities as compared with children without CD. Using magnetic resonance imaging (MRI), neuroscientists in England measured the size of specific brain regions of 65 teenage boys with CD and 27 teenage boys without CD (Fairchild et al., 2011). They found that the amygdala, a region of the brain involved in reading others' emotions, empathy, and recognizing when others are distressed, was markedly smaller in teens with CD than in the healthy comparison group. However, no differences were found between teens with early-onset and those with adolescent-onset conduct disorders. The image to the right, shows the amygdala (for each side of the brain), the region of the brain for which the reduction in volume was largest for teens with CD versus healthy comparison children.



Courtesy of Dr. Giuseppe Iaria

These preliminary findings are important, since it has been argued that early-onset CD is a neurodevelopmental condition, whereas adolescent-onset CD is mostly the result of teens associating with and mimicking other teens who are getting into trouble. However, this study shows that abnormalities in brain structures underlying social-information processing may contribute to the emergence of both adolescent-onset as well as early-onset CD (Fairchild et al., 2011).

aggressive behavior. In this context, dysfunction in the integrated functioning of brain circuits involving the amygdala has been implicated (Blair, 2011). The second neural circuit includes prefrontal cortex decision-making circuits and socioemotional information-processing circuits that assess social cues and evaluate the consequences of aggressing or not aggressing; the third neural circuit includes frontoparietal regions involved in regulating emotions and impulsive motivational urges (Coccaro et al., 2011). In the future, further research into these neural circuits may help to reveal mechanisms through which inborn dispositions may place a child at risk for later conduct problems (Viding & Jones, 2008).

## Social–Cognitive Factors

**Social–cognitive abilities** refer to the skills involved in attending to, interpreting, and responding to social cues. There is a strong relationship between social–cognitive deficits and antisocial behavior across all types of conduct-problem trajectories (e.g., childhood limited, adolescent-onset, early-onset persistent), especially for children showing early-onset persistent conduct problems. As many as 40% of boys and 25% of girls with persistent conduct problems display significant social–cognitive impairments (Oliver et al., 2011). The connection between children's thinking in social situations and their aggressive behavior has been looked at in several ways. Some approaches focus on immature forms of thinking, such as egocentrism, a lack of social perspective taking, theory of mind deficits, or deficits in moral reasoning (Blair, 2010; Olson et al., 2011). Others emphasize cognitive deficiencies, such as a child's failure to use verbal mediators to regulate his or her behavior (Meichenbaum, 1977), or cognitive distortions, such as interpreting a neutral event as an intentionally hostile act (Crick & Dodge, 1994). Other approaches focus more broadly on the social–cognitive processes involved in antisocial decision making (Fontaine et al., 2010). Some research has also found deficits in facial expression recognition and eye contact in children with conduct problems, which may further contribute to their antisocial behavior and social difficulties (Dadds et al., 2011; Fairchild et al., 2009).

Dodge and Pettit (2003) have presented a comprehensive social–cognitive framework to account for aggressive behavior and antisocial behavior in children. In their model, cognitive and emotional processes play a central mediating role. Children are presumed to develop social knowledge about their world based on a unique set of predispositions, life experiences, and sociocultural contexts. In specific social situations, children

**TABLE 9.4** | **Steps in the Thinking and Behavior of Aggressive Children in Social Situations**

| |
|---|
| **Step 1: Encoding.** Socially aggressive children use fewer cues before making a decision. When defining and resolving an interpersonal situation, they seek less information about the event before acting. |
| **Step 2: Interpretation.** Socially aggressive children attribute hostile intentions to ambiguous events. |
| **Step 3: Response Search.** Socially aggressive children generate fewer and more aggressive responses and have less knowledge about social problem solving. |
| **Step 4: Response Decision.** Socially aggressive children are more likely to choose aggressive solutions. |
| **Step 5: Enactment.** Socially aggressive children use poor verbal communication and strike out physically. |

*Source:* From A Review and Reformulation of Social information Processing Mechanism in Children's Social Adjustment by N. R. Crick and K. A. Dodge, 1994, Psychological Bulletin', 115, 74–101.

then use this social knowledge to guide their processing of social information in ways that lead directly to certain behaviors. For example, when teased in the schoolyard by peers, does the child laugh with the crowd, walk away, or strike back aggressively? A set of emotional and thought processes are presumed to occur between the social stimulus of being teased and the child's reaction. The thinking and behavior of antisocial/aggressive children in social situations are often characterized by deficits in one or more of these steps, as outlined in Table 9.4.

## Family Factors

*I am convinced that increasing rates of delinquency are due to parents who are either too careless or too busy with their own pleasure to give sufficient time, companionship, and interest to their children.*

—Former FBI director J. Edgar Hoover, *The New York Times*, December 6, 1947

Many family factors have been implicated as possible causes of children's antisocial behavior—early maternal age at childbearing, poor disciplinary practices, harsh discipline, a lack of parental supervision, a lack of affection, marital conflict, family isolation, and violence in the home (D'Onofrio et al., 2009; Hoeve et al., 2008; Lansford et al., 2011). For children who are at genetic risk for antisocial behavior, positive parenting practices may reduce the influence of the child's genotype on later antisocial behavior, whereas negative parenting practices can have the opposite effect (Feinberg et al., 2007). Although the association between family factors and conduct problems is well established, the nature of this association and the possible causal role of family factors continue to be debated.

Family difficulties are related to the development of both ODD and CD, with a stronger association for children on the LCP as compared with those on the AL path (Lahey et al., 1992). A combination of individual child risk factors (e.g., difficult temperament) and extreme deficits in family management skills most likely accounts for the more persistent and severe forms of antisocial behavior (Caspi & Moffitt, 1995).

Family factors are related to children's antisocial behavior in complex ways. For example, physical abuse is a strong risk factor for later aggressive behavior. One reason for this link between factors appears to be deficits in the child's social information processing that result from the physical abuse (Dodge & Pettit, 2003). As we have seen, the child's genotype can also moderate the link between maltreatment and later antisocial behavior, a possible reason that not all children who have been abused grow up to victimize others (Caspi et al., 2002).

Several factors may affect the consequences of marital conflict on children's aggressive behavior; these include the parents' unavailability, the use of

Bob Kalman / The Image Works

Physical abuse is a strong risk factor for later aggressive behavior.

inconsistent or harsh discipline, lax monitoring, and how the child interprets conflict between parents (Cummings & Davies, 2002). Other conditions associated with marital conflict or divorce such as stress, depression, loss of contact with one parent, financial hardship, and greater responsibility at home may also contribute to antisocial behavior (Emery, 1999). Interestingly, contact with an absent father after the breakup of the parents' marriage can be either a risk or a protective factor for antisocial behavior, depending on whether or not the father is antisocial (Jaffee et al., 2003).

**Nick's mother says:**

> Nick hit a neighborhood kid on the head with a two-by-four; the injured child required 16 stitches. Then he killed another kitten by jumping on it from his bunk bed. I lost control. I told him I hated him, I grabbed him by the cheek, I pinched it a little too hard. I didn't know what to do. (Colapinto, 1993, p. 150)

Cruel and aggressive child behaviors can evoke strong reactions, like the anger and overly harsh response by Nick's mother. An important concept for understanding family influences on antisocial behavior is **reciprocal influence**, which means that the child's behavior is both influenced by and influences the behavior of others. Negative parenting practices and parent–child conflict may lead to antisocial behavior, but they may also be a reaction to the oppositional and aggressive behaviors of their children (Klahr et al., 2011).

For example, in an interesting study of reciprocal influence, mothers of boys with and without CD were asked to interact with three boys—their own son, a boy with CD, and a boy without CD (Anderson, Lytton, & Romney, 1986). All mothers were more demanding and negative when interacting with a child with CD, which supports a child-to-parent effect. However, mothers of boys with CD responded most negatively to their own sons, suggesting that previous negative interactions with their child also had an effect. Reciprocal influence is a useful way to think of the interplay between family influences and antisocial behavior over the course of development. However, it is also possible that some aspects of the family environment are related to antisocial behavior as a result of a shared genetic predisposition that leads parent and child to display similar behavior patterns.

Some studies generally support the view that child behaviors exert greater influence on parenting behaviors than the reverse, perhaps more so for mothers than fathers (Narusyte et al., 2011). This suggests that the inborn level of emotional dysregulation that children bring to their interactions with parents may have a greater influence on outcomes than ineffective parenting behaviors (Loeber et al., 2009). Nevertheless, as we will discuss, interventions directed at changing ineffective parenting behaviors are among the most effective methods for reducing children's conduct problems.

© Masterfile

A child's oppositional behavior may also lead to negative parenting behavior.

### *Coercion Theory*

Gerald Patterson's **coercion theory** contends that parent–child interactions provide a training ground for the development of antisocial behavior (Patterson, Reid, & Dishion, 1992). This occurs through a four-step, escape-conditioning sequence in which the child learns to use increasingly intense forms of noxious behavior to escape and avoid unwanted parental demands. The *coercive parent–child interaction* described in A Closer Look 9.6 begins when a mother finds her son Paul, who is failing in school, watching TV rather than doing his homework. Coercive parent–child interactions are made up of well-practiced actions and reactions, which may occur with little awareness. This process is called a "reinforcement trap" because, over time, all family members become trapped by the consequences of their own behaviors. For example, mothers of antisocial children are eight times *less* likely to enforce demands than are mothers of nonproblem children (Patterson et al., 1992).

The relationship between parenting and conduct problems also appears to be affected by a child's callous–unemotional traits. In one report, ineffective parenting was related to conduct problems, but only in children who were rated low on CU traits (Wootton et al., 1997). Children with CU traits displayed significant conduct problems regardless of the quality of parenting they received. The relationship between parental discipline and conduct problems may also be affected by the amount of discipline—too much or too little can both have adverse effects. The relationship between parental discipline and antisocial behavior may

## A CLOSER LOOK 9.6

### Coercive Parent–Child Interaction: Four-Step Escape Conditioning Sequence

**Step 1**

Raising her voice, Paul's mother scolds, "Why are you sitting in front of the TV when you should be doing your homework?"

**Step 2**

Paul snaps back, "School is boring, my teachers are stupid, and I don't have any homework to do." Paul's arguing has the immediate effect of punishing his mother for her scolding and, over time, may reduce her efforts to do something about his homework and school problems.

**Step 3**

Paul's mother withdraws her demand for him to complete his homework, allowing herself to be satisfied that he does not have any homework to do. She lowers her voice and says, "Does Mrs. Smith still put everyone to sleep in her English class?" The mother's withdrawal of her demand for homework reinforces Paul's arguing and increases the chances that the next time she makes an issue of homework, he will argue with her. Over time, Paul may also turn up the volume of his negative reactions by shouting or throwing things.

**Step 4**

As soon as Paul's mother withdraws her demand, Paul stops arguing and engages in neutral or even positive behavior. He says "You're sure right about Mrs. Smith, Mom. It's tough to keep your eyes open in her class." Paul, by ceasing his noxious behavior, reinforces his mother for giving in and increases the likelihood that she will do so again in response to his arguing and protests.

Based on Antisocial Boys, by G. R. Patterson, J. B. Reid, and T. J. Dishion, 1992, p. 41.

also vary with the family's cultural background, the emotional climate in which discipline is used, and the gender of the parent–child pair. For example, discipline may be most effective in same-gender parent–child pairs: discipline of daughters by mothers and sons by fathers (Deater-Deckard & Dodge, 1997).

### *Attachment Theories*

Attachment theories emphasize that the quality of children's attachment to parents will determine their eventual identification with parental values, beliefs, and standards. Secure bonds with parents promote a sense of closeness, shared values, and identification with the social world. Attachment theories contend that children refrain from antisocial behavior because they have a stake in conformity.

Children with conduct problems often show little internalization of parent and societal standards. Even when they comply with parental requests, they may do so because of perceived threats to their freedom or physical safety (Shaw & Bell, 1993). When these threats are not present, such as when the child is unsupervised, antisocial behavior is likely to occur. Weak bonds with parents may lead the child to associate with deviant peers, which in turn may lead to delinquency and substance abuse (Elliott, Huizinga, & Menard, 1989).

Research findings support a relationship between insecure attachments, particularly for children with disorganized attachments, and the development of antisocial behavior during childhood and adolescence (Pasco Fearon et al., 2010). However, it is unclear whether attachment quality by itself can predict current or future variation in the severity of conduct problems. It is likely that the relationship between attachment and antisocial behavior is affected by many factors, including the child's gender, clinical status, temperament, and family management practices (Pasco Fearon et al., 2010).

## Other Family Problems

In addition to the negative parenting practices and attachment problems that we have discussed, other family factors such as family instability and stress, and parental criminality and psychopathology may also contribute to children's conduct problems.

### JAKE AND REGGIE

#### All Odds Against Them

Linda M., single mother of 2-year-old Jake and 4-year-old Reggie, sought treatment because Reggie was engaging in severe and uncontrollable aggressive behaviors, including hitting, kicking, and biting Jake. She was depressed and at risk for suicide. Her boyfriend Hank is the father of the two children. He lives nearby and demands that she come over so he can see the children. During these visits, he engages her in what she refers to as "forced sex" (i.e., rape), and he demands that Jake and Reggie remain with them and watch. In principle, Linda could have refused the visits. However, Hank threatened that if she did not comply, he would stop paying child support, take Jake and Reggie away in a custody battle, kill himself, or come over to the house and kill her and the two boys. These threats of violence were to be taken seriously because Hank had a prior arrest record for assault and brandished a gun.

Based on Conduct Disorders in Childhood and Adolescence by A. E. Kazdin, p. 17.

### Family Instability and Stress

Families of children with conduct problems are often characterized by an unstable family structure, with frequent transitions, including changes in parents and changes in residence (Dishion & Patterson, 2006). Family instability is related to a child's heightened risk for antisocial behavior, academic problems, anxiety and depression, association with deviant peers, and criminal conviction (Kasen et al., 1996). In most cases, the impact of divorce on a child's antisocial behavior is related to the family disruption and conflict that accompany it (Emery, 1999). In some cases, a child's antisocial behavior may contribute to family instability by increasing the chances of divorce (Block, Block, & Gjerde, 1986).

High family stress is associated with negative child behavior in the home, and may be both a cause and an outcome of antisocial behavior. Unemployment, low SES, and multiple family transitions are all related to childhood conduct problems. Among family stressors, poverty is one of the strongest predictors of children having CD and high rates of criminal activities (Pagani et al., 1999). But what constitutes the "active ingredient" in the link between poverty and antisocial behavior? In this regard, instability, residential mobility, and disruptions in parenting practices have all been found to be important (Dodge, Pettit, & Bates, 1994b). The **amplifier hypothesis** states that stress amplifies the maladaptive predispositions of parents (e.g., poor mental health), thereby disrupting family management practices and compromising parents' ability to be supportive of their children (Conger et al., 1994).

### Parental Criminality and Psychopathology

Aggressive and antisocial tendencies run in families, within and across generations (Blazei, Iacono, & Krueger, 2006; D'Onofrio et al., 2007). In fact, children's aggression is correlated with their parents' childhood aggression at the same age (Huesmann et al., 1984). Parents of antisocial children have higher rates of arrests, motor vehicle violations, license suspensions, and substance abuse (Dishion & Patterson, 2006). Antisocial individuals are likely to be ineffective parents, especially during disciplinary confrontations during which they display an irritable, explosive style of interaction. Certain types of parental psychopathology, such as APD, are strongly and specifically related to CD in their children (Herndon & Iacono, 2005). This relationship is particularly clear for fathers, as is the link between paternal criminal behavior and substance abuse and child antisocial patterns (Waldman & Lahey, 2013). The strong association between paternal APD and a child's antisocial behavior is independent of whether the father lives in the home and of the degree of contact between father and child (Tapscott, Frick, Wootton, & Kruh, 1996). For mothers, antisociality, histrionic personality (excessive emotionality and attention seeking), and depression are related to children's antisocial behavior (Dishion & Patterson, 2006).

## Societal Factors

Causes of antisocial behavior at the level of the individual and family tell only part of the story, since they interact with the larger societal and cultural context in determining conduct problems (Sampson, 1992). There is little doubt that poverty, neighborhood crime, family disruption, and residential mobility are related to crime and delinquency in young people (Cooley-Strickland et al., 2009; Schonberg & Shaw, 2007). However, the specific mechanisms by which these conditions lead to crime and delinquency are not known.

Theories of social disorganization propose that community structures impact family processes that then affect the child's adjustment (Sampson & Laub, 1994). Adverse contextual factors (e.g., low SES) are associated with poor parenting, particularly coercive and inconsistent discipline and poor parental monitoring (Lahey, Van Hulle et al., 2008). In turn, these factors are associated with an early onset of antisocial behavior, arrests at an early age, and chronic offending during adolescence (Capaldi & Patterson, 1994). A vicious cycle of adaptational failure and added stress places downward pressure on both the parent and the child. An antisocial individual is more vulnerable and at greater risk of entering a class of divorced, unemployed, and disadvantaged people (Dishion & Patterson, 2006). For example, social disadvantage, increased mobility, divorce, early sexual activity, and working-mother status may lead to an increase in mothers who are at greater risk for antisocial parenting practices. Also, less skilled antisocial mothers may drift into areas of large cities that isolate them from family and neighbors and lead them to function in an atmosphere of mistrust and minimal communication. When these women become pregnant again, they may have reduced access to public health services. Poor diet and drugs may result in a higher incidence of low birth weight, prematurity, and birth defects in their offspring, which in turn make their infants and toddlers more difficult to parent. The combination of a difficult infant and an unskilled parent increases the likelihood of antisocial behavior and the subsequent likelihood of behaviors that result in arrest. In this way, the generation of conduct problems cycles again and again.

### Neighborhood and School

Antisocial behavior in youths is disproportionately concentrated in poor neighborhoods characterized by

a criminal subculture that supports drug dealing and prostitution, peer group violence, delinquent gang membership, frequent transitions and mobility, and low social support from neighbors or religious groups (Leventhal & Brooks-Gunn, 2000). In addition, antisocial people tend to select neighborhoods populated by other people who are like them (Harden et al., 2009). The **social selection hypothesis** states that people who move into different neighborhoods differ from one another before they arrive, and those who remain differ from those who leave. For individuals with antisocial traits, this creates a community organization that minimizes productive social relations and effective social norms, leading to the antisocial behavior becoming the rule (Sampson, Raudenbush, & Earls, 1997). The effects of community characteristics on crime and delinquency are likely to be reinforced by neighborhood social disorganization characterized by few local friendship and acquaintance networks, low participation in local community organizations, and an inability to supervise and control teenage peer groups (Sampson & Groves, 1989). In fact, the main influence of effective parents in high-risk neighborhoods seems to be in countering gang membership (Tolan, Gorman-Smith, & Henry, 2003).

In high-risk neighborhoods, enrollment in a poor-quality school is associated with antisocial and delinquent behavior, whereas a positive school experience can be a protective factor for the development of these behaviors (Rutter, 1989). A good school environment characterized by clear requirements for homework completion, high academic expectations, clear and consistent discipline policies, and incentives for appropriate school behavior and achievement may partially compensate for poor family circumstances. Systematic interventions to promote these school characteristics have resulted in schoolwide reductions in children's conduct problems (Gottfredson, Gottfredson, & Hybel, 1993).


Bob Daemmrich/Alamy

Weapons signs, such as this one in the area of Austin, Texas, are routinely posted outside schools.

### Media

*I believe that this kind of vicarious adventure, escape, excitement, even blood and thunder is necessary and important to most children as outlets for their own emotions, particularly their feelings of aggression.*

—Josette Frank, Media Consultant

*Programs interestingly depicting antisocial conduct, crime, and murder, influence children to antisocial attitudes and lead to aggression.*

—Judge Jacob Panken, New York City Children's Court

These contrasting expert opinions were presented about 70 years ago (*The New York Times*, April 14, 1946), in reference to the influence of radio on children. The controversy regarding media influences on aggression in young people continues today. Parents, teachers, policymakers, and the press have expressed concerns about the possible adverse impact of violence in movies, TV, video games, text messages, and the Internet on children's social development and aggressive behavior. In considering media influences on children's antisocial behavior, we discuss the relation between TV violence and children's aggressive behavior because most of the research has been conducted in this area. Research into the new media is increasing at a rapid pace (Varnhagen, 2006). The findings from studies of TV violence and children's aggressive behavior should help us in answering questions about young people's use of the Internet, their exposure to unwanted or undesirable information, and how they are affected by this exposure.

At one extreme, some researchers claim that TV violence verges on child maltreatment and that we can reduce murders by unplugging the TV; others argue that there is little evidence for a causal relation between TV violence and aggressive behavior. By the time a child in the United States reaches grade 6, he or she has witnessed 8,000 or more murders on TV and well over 100,000 other acts of violence (Leland, 1995). The concern is that this steady diet of violence leads children to think violence is normal, to become desensitized to the suffering of real people, or to become aroused by images they see and want to mimic those violent acts. For example, one 5-year-old boy, after watching his favorite cartoon characters pull one of their famous arson stunts, set his house ablaze; his younger sister was killed in the fire. Thus, exposure to media violence can be both: (1) a short-term *precipitating* factor for aggressive and violent behavior that results from priming, excitation, or imitation of specific behaviors, and (2) a long-term *predisposing* factor for aggressive behavior acquired via desensitization to violence and observational learning of an aggression-supporting belief

system (i.e., "the world is a hostile place," "aggression is acceptable," "aggression can be used to solve social problems") (Huesmann et al., 2003).

Exposure to media violence may reinforce preexisting antisocial tendencies in some children. For example, in a series of studies spanning more than a decade, children with conduct problems were found to view relatively large amounts of violent material, prefer aggressive characters, and believe fictional content to be true (Gadow & Sprafkin, 1993). However, it is not only children with preexisting violent tendencies who are likely to be affected. Long-term studies have found that childhood exposure to media violence between ages 6 and 9, identification with aggressive TV characters, and perceived realism of TV violence predict serious aggressive and criminal behavior 15 years later (Huesmann et al., 2003).

The correlation between TV violence and aggression is indisputable—but does TV violence cause aggression, and if so, how? Although research suggests a causal relation (Anderson & Bushman, 2002; Johnson et al., 2002), answers to these questions remain elusive despite decades of research and a pressing urge to act on research findings through social policies to filter violent content and inform users. It is unlikely that media influences (TV or other forms) alone can account for the substantial amount of antisocial behavior in young people (Rutter & Smith, 1995). Like other risk factors, media influences interact with child, family, community, and cultural factors in contributing to conduct problems. But clearly they are an important and unique contributing factor. Exposure to media violence will not turn an otherwise well-adjusted child into a violent criminal. However, "just as every cigarette one smokes increases a little bit the likelihood of a lung tumor some day … every violent TV show increases a little bit the likelihood of a child growing up to behave more aggressively in some situation" (Huesmann et al., 2003, p. 218).

## Cultural Factors

Cultural differences in the expression of aggressive behavior are dramatic. Across cultures, socialization of children for aggression has been found to be one of the strongest predictors of aggressive acts such as homicide and assault. As the following examples of contrasting socialization practices illustrate, aggression may be an inadvertent consequence of a culture's emphasis on training "warriors":

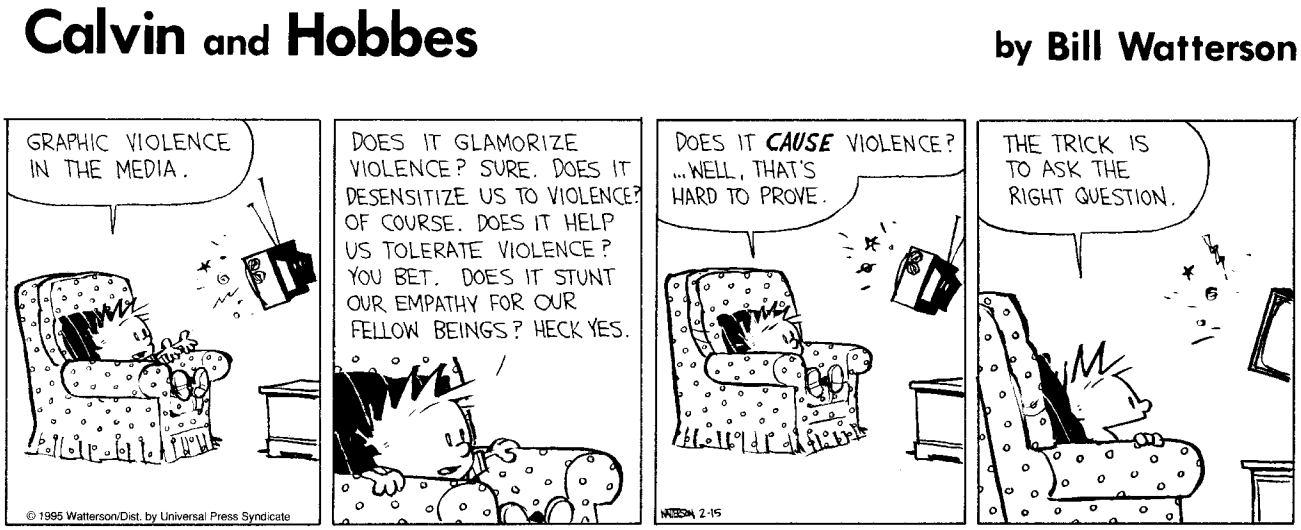
*The Kapauku of Western New Guinea:*

> At about 7 years of age, a Kapauku boy begins to be under the father's control, gradually sleeping and eating only with the men and away from his mother. … His training [to be a brave warrior] begins when the father engages his son in mock stick fights. Gradually the fights become more serious and possibly lethal when the father and son shoot real war arrows at each other. Groups of boys play at target shooting; they also play at hitting each other over the head with sticks. (Ember & Ember, 1994, p. 639–640)

The homicide rate among the Kapauku from 1953 to 1954 was estimated at 200 per 100,000, approximately 40 times the current murder rate in the United States.

*The Lepcha of the Indian Himalayas:*

> The Lepcha are very clear about what they expect from their children. "Good children help out with the work, tell the truth, listen to teaching from elders, help old people, and are peaceable. Bad children quarrel with and insult people, tell lies, draw their



knives in anger when reprimanded, and do not do their share of the work." (Ember & Ember, 1994, p. 641)

Interviews with the Lepcha people revealed that the only authenticated murder in their culture had occurred about 200 years ago (Ember & Ember, 1994).

Rates of antisocial behavior vary widely across and within cultures, and not necessarily in relation to technological gains, material wealth, or population density. For example, some third world countries that value interdependence are characterized by high rates of prosocial behavior, and some places with high population density have very low rates of violence. The United States is by far the most violent of all industrialized nations, with homicide rates about 5 times higher than those in Europe and 10 times higher than those in Japan (OECD, 2013).

Minority status is related to antisocial behavior in the United States, with elevated rates of antisocial behavior in African American, Hispanic American, and Native American youths (Elliott, Huizinga, & Ageton, 1985). However, studies of national samples have reported either no or very small differences in antisocial behavior related to race or ethnicity when SES, gender, age, and referral status are controlled for (Lahey et al., 1995). Thus, although externalizing problems are reported to be more frequent among minority-status youths, this finding is likely related to disparities that include economic hardship, limited employment opportunities, residence in high-risk urban neighborhoods, and membership in antisocial gangs (Children's Defense Fund, 2007; Egley & Howell, 2013). Importantly, both Mexican American and African American children who live in dangerous or disadvantaged neighborhoods and who have high levels of family support show fewer antisocial behaviors than children who have low or no family support (Schofield et al., 2012). For immigrant groups in the United States, the risk for CD also seems to vary according to migration status and amount of exposure to American culture. For example, one study found that the risk for CD was highest among Mexican American children of U.S.-born parents as compared with children of Mexican-born immigrants raised in the United States or the general population of Mexico (Breslau et al., 2012).

## Section Summary

### Causes

- Conduct problems in children are best accounted for by multiple causes or risk and protective factors that operate in a transactional fashion over time.
- Adoption and twin studies indicate that genetic influences account for about 50% of the variance in antisocial behavior.
- Genetic contributions to overt forms of antisocial behavior, such as aggression, are stronger than for covert acts, such as stealing or lying.
- Antisocial behavior may result from an overactive behavioral activation system (BAS) and an underactive behavioral inhibition system (BIS). Low levels of cortical arousal and autonomic reactivity and deficits in the amygdala, prefrontal cortex, and other brain regions play an important role, particularly for childhood-onset/persistent CD.
- Many family factors have been implicated as possible causes of children's antisocial behavior, including marital conflict, family isolation, violence in the home, poor disciplinary practices, a lack of parental supervision, and insecure attachments.
- Family instability and stress, parental criminality and antisocial personality, and antisocial family values are risk factors for conduct problems.
- The structural characteristics of the community provide a backdrop for the emergence of conduct problems by giving rise to community conditions that interfere with the adoption of social norms and the development of productive social relations.
- School, neighborhood, and media influences are all potential risk factors for antisocial behavior, as are cultural factors, such as minority group status and ethnicity.

## TREATMENT AND PREVENTION

### SCOTT

### Salvageable?

Scott, age 10, was referred after setting a fire in the schoolyard. While his therapist saw Scott as "potentially salvageable," his parents were not willing to pursue therapy. As a result, Scott was placed in a boarding school for "troubled boys." … After 3 weeks at this school, he was expelled for burning down the dorm. … Charges were pressed and he was sent to a group home for delinquent boys. He remained there for 3 months before he and two older boys ran away. They were caught a few days later when they attacked a homeless man, stealing his money ($4.85) and beating him. As a result of this crime, Scott was sent to a detention facility until he turned 18. His therapist heard nothing further.

Based on Morgan, 1999.

Many forms of treatment will be tried throughout the life of a child with severe conduct problems. Treatment may begin during the preschool years or, more typically, as was the case with Scott, when severe antisocial behavior at school leads to referral. Ongoing contacts with

the educational, mental health, and judicial systems may result in referral for one or more of a wide range of treatments. The most promising treatments use a combination of approaches that are applied across individual, family, school, and community settings (Kazdin, 2007; Lochman et al., 2011; Sanders, 2012). In addition, treatment frequently requires that related family problems, such as maternal depression, marital discord, abuse, and other stressors be addressed if gains are to be generalized and maintained (McMahon, Wells, & Kotler, 2006).

Most people understand that family dysfunction, abuse, school expulsion, association with drug-using peers, residence in a high-crime area, and minimal parental supervision contribute to serious conduct problems (Henggeler, 1996). However, despite this recognition, typical and often court-mandated treatments such as psychotherapy, group therapy, tutoring, punishment, wilderness programs, and boot camps fail to meaningfully address these determinants, and thus are among the least effective approaches (Lipsey, 1995). Despite their lack of effectiveness in treating serious antisocial behavior, office-based individual counseling and family therapy are often provided because they can be relatively inexpensive (Tate, Reppucci, & Mulvey, 1995). Group treatments that bring together antisocial youth may only make the problem worse, since associating with like-minded individuals often encourages antisocial behavior (Dishion & Dodge, 2005).

As we saw for Scott, restrictive approaches such as residential treatment, inpatient psychiatric hospitalization, and incarceration also show little effectiveness and have the additional disadvantage of being extremely expensive (Henggeler & Santos, 1997). Unfortunately, a significant proportion of mental health dollars for youths continues to be spent on restrictive out-of-home placements that may cause more harm than good (Sondheimer, Schoenwald, & Rowland, 1994). Incarceration may not even serve a community protection function, since youths who are incarcerated and then released often commit more crimes than youths kept at home and given treatment (Henggeler, 1996).

Since youth conduct problems are known to show a developmental progression, diversification, and escalation over time, treatments must be sensitive to where a youth is in this trajectory. Treatment methods and goals will differ for preschoolers, school-age children, and adolescents and will differ according to the type and severity of the child's conduct problems. In general, the more progressed the antisocial behavior, the greater is the need for intensive interventions and, unfortunately, for children like Scott, the poorer is the prognosis. In fact, if early-onset antisocial behavior is not changed by the end of grade 3, it might best be treated as a chronic condition, much like diabetes, which cannot be cured but can be managed or contained through ongoing interventions and supports (Kazdin, 1995). This troubling situation of high treatment effort and cost with less return for older children has led to a reevaluation of priorities and a growing emphasis on early intervention and prevention (Powell, Lochman, & Boxmeyer, 2007). A comprehensive, two-pronged approach to the treatment of conduct problems is needed that includes (Frick, 2000):

- *Early intervention/prevention* programs for young children at risk for or just starting to display problem behaviors
- *Ongoing interventions* to help older youths and their families cope with the many associated social, emotional, and academic problems

To illustrate the many treatments for children and adolescents with conduct problems, we next highlight three representative treatment approaches that have had some proven success (Eyberg, Nelson, & Boggs, 2008)—parent management training (PMT), problem-solving skills training (PSST), and multisystemic therapy (MST) (see Table 9.5). We also discuss promising new

**TABLE 9.5 | Effective Treatments for Children with Conduct Problems**

| Treatment | Overview |
|---|---|
| **Parent Management Training (PMT)** | Teaches parents to change their child's behavior in the home and in other settings using contingency management techniques. The focus is on improving parent–child interactions and enhancing other parenting skills (e.g., parent–child communication, monitoring, and supervision). |
| **Problem-Solving Skills Training (PSST)** | Identifies the child's cognitive deficiencies and distortions in social situations and provides instruction, practice, and feedback to teach new ways of handling social situations. The child learns to appraise the situation, change his or her attributions about other children's motivations, be more sensitive to how other children feel, and generate alternative and more appropriate solutions. |
| **Multisystemic Therapy (MST)** | An intensive approach that draws on other techniques such as PMT, PSST, and marital therapy, as well as specialized interventions such as special education, and referral to substance abuse treatment programs or legal services. |

preventive interventions for young children. Almost all forms of treatment provide corrective interpersonal experiences with parents, siblings, and peers because most antisocial acts, including violence, occur between the child and family members or peers. In addition, given the pervasiveness of conduct problems across settings, nearly all treatments include components designed to change the child's behavior at home, in the community, and at school (Liber et al., 2013).

## Parent Management Training (PMT)

**Parent management training** (**PMT**) teaches parents to change their child's behavior at home and in other settings (Brinkmeyer & Eyberg, 2003; McMahon & Forehand, 2003). Its underlying assumption is that maladaptive parent–child interactions are at least partly responsible for producing and sustaining the child's antisocial behavior. Changing the way parents interact with their child will lead to improvements in the child's behavior. Although both child and parent behavior jointly contribute to negative parent–child interactions, the easiest and most desirable point of entry in modifying these interactions is changing parent behavior. The goal of PMT is for the parent to learn specific new skills (Forgatch & Patterson, 2010). To achieve this goal, many of the same procedures that we have discussed for working with children with ADHD and their families are used (see Chapter 8). These include teaching parents to monitor their children's behavior, to present clear commands and rules, and to systematically provide rewards and minor forms of punishment such as time out from positive reinforcement. Many variations of PMT can be individual versus group training, training in the clinic versus in the home, or the use of live versus videotaped training materials.

PMT has a number of strengths and some limitations (McMahon et al., 2006). Many excellent treatment manuals and training materials have been developed that facilitate its widespread use (e.g., Barkley, 2013a; McMahon & Forehand, 2003). In addition, PMT has been evaluated more than any other treatment for conduct problems (Eyberg et al., 2008). These evaluations have repeatedly demonstrated short-term effectiveness in producing changes in parent and child behavior. The average child whose parents participate in PMT shows better adjustment after treatment than 80% of referred children whose parents do not participate (Serketich & Dumas, 1996). In addition to changes in the referred child, PMT has also been associated with reductions in the problem behaviors of siblings and reduced stress and depression in the parents.

PMT has been most effective with parents of children younger than 12 years of age and less so with adolescents (Dishion & Patterson, 1992). In light of this, promising adaptations of these interventions have also been developed for working with older adolescents and their families (Dishion & Kavanagh, 2003). Although PMT can produce short-term gains, its long-term effectiveness is less clear (McMahon et al., 2006). In addition, PMT makes numerous demands on parents to master and implement procedures in the home, attend meetings, and maintain phone contact with the therapist. For families under stress and with few resources, these demands may be too great to allow the family to continue in treatment (Lundahl, Risser, & Lovejoy, 2006). In order to increase engagement of low-income families, smart phone–enhanced versions of PMT have been developed to include series of skills videos, brief daily surveys, text message reminders, video recording of home practice, and midweek video calls (Jones et al., 2014). There is also growing recognition of the importance of identifying barriers to, and facilitators of, access/engagement of parents in PMT programs (Koerting et al., 2013).

The application of PMT is rarely straightforward. The need to change their own parenting practices may not be recognized by parents who believe that difficulties occur because their child is stubborn, their marriage is bad, work is interfering with the time they spend together, or school personnel are unfair. In fact, parents of children with conduct problems frequently believe they use good parenting practices but their child fails to respond. It is important to address these parental beliefs and concerns if treatment is to be successful (Morrisey-Kane & Prinz, 1999). In addition, PMT practitioners have increasingly come to recognize the importance of marital and social support, therapy style and engagement, and ethnic and cultural factors in treatment (Scott et al., 2010; Yasui & Dishion, 2007).

## Problem-Solving Skills Training (PSST)

**Problem-solving skills training** (**PSST**) is a form of cognitive behavioral therapy that focuses on the cognitive deficiencies and distortions displayed by children and adolescents with conduct problems in interpersonal situations (Kazdin, 2010). PSST is used both alone and in combination with PMT, as required by the family's circumstances. The underlying assumption of PSST is that the child's perceptions and appraisals of environmental events will trigger aggressive and antisocial responses, and that correcting faulty thinking will lead to changes in behavior. As described in A Closer Look 9.7, the child is taught to use five problem-solving steps to identify thoughts, feelings, and behaviors in problem social situations.

During PSST, the therapist uses instruction, practice, and feedback to help the child discover different

A CLOSER **LOOK 9.7**

### Cognitive Problem-Solving Steps

**Problem Situation**
Jason, one of the kids in your class, has taken your Nintendo game. You want to get it back. What do you do?

**Step 1: What Am I Supposed to Do?**
I want to get my Nintendo game back from Jason.

**Step 2: I Have to Look at All My Possibilities**
I can beat him up and take it back, ask him to give it back, or tell my teacher.

**Step 3: I Had Better Concentrate and Focus**
If I beat him up, I would get into trouble. If I asked him, he might give it back.

**Step 4: I Need to Make a Choice**
I'll try asking him, and if that doesn't work, I will tell my teacher.

**Step 5: I Did a Good Job or I Made a Mistake**
I made a good choice. I won't get into trouble. Jason and I can still be friends if he returns my Nintendo game. If not, I did my best to get it back before asking my teacher for help. I did a good job!

Based on Kazdin, A. E. (1996). "Problem solving and parent management training in treating aggressive and antisocial behavior." In E. D. Hibbs & P. S. Jensen (Eds.), Psychosocial Treatments for Child and Adolescent Disorders (pp. 377–408). Washington, DC: American Psychological Association.

ways to handle social situations. To accomplish this, children learn to appraise the situation, identify self-statements and reactions, and alter their attributions about other children's motivations. They also learn to be more sensitive to how other children feel, to anticipate others' reactions, and to generate appropriate solutions to social problems.

PSST is effective with children and youths who are clinically referred for conduct problems, with benefits extending to parent and family functioning (Kazdin, 2010). Research supports the emphasis on the relationship between maladaptive cognitions and aggressive behavior on which PSST is based, and PSST procedures are carefully specified in treatment manuals. Until relatively recently it was not clear whether changes in maladaptive cognitions were responsible for behavioral improvements. Indeed, the alteration of social–cognitive processes may not necessarily lead to changes in behavior. However, in one major study using a multifaceted intervention (Fast Track) it was found that 27% of the intervention's impact on antisocial behavior was mediated by its effect on three social–cognitive processes: reducing hostile-attribution biases, increasing competent response generation to social problems, and devaluing aggression (Dodge, Godwin, & The Conduct Problems Prevention Research Group, 2013). Finally, although most children improve as a result of PSST, some may continue to display more problems than their nondeviant peers. Thus, more enduring PSST and other interventions are being developed to meet the needs of families of children with conduct problems whose problems are particularly severe.

## Multisystemic Therapy (MST)

**Multisystemic therapy** (**MST**) is an intensive empirically supported family and community-based treatment for adolescents with severe conduct problems that make out-of-home placement highly likely (Henggeler & Schaeffer, 2010). MST views the adolescent with conduct problems as functioning within interconnected social systems, including the family, school, neighborhood, and court and juvenile services (Henggeler et al., 2009). Antisocial behavior results from, or can be maintained by, transactions within or between any of these systems. MST seeks to empower caregivers to improve youth and family functioning (Cunningham et al., 1999). Thus, treatment is carried out with all family members, school personnel, peers, juvenile justice staff, and other individuals in the child's life. MST is an intensive approach that also draws on PMT, PSST, and marital therapy, as well as specialized interventions such as special education and referral to substance-abuse treatment programs or legal services. In effect, MST attempts to address the many determinants of severe antisocial behavior (Wells et al., 2010). The guiding principles of MST are outlined in Table 9.6.

Outcome studies of MST with extremely antisocial and violent youths have found this approach to be superior to usual services, individual counseling, community services, and psychiatric hospitalization. In addition, studies have found decreases in delinquency and aggression with peers, improved family relations, and reductions in out-of-home placements (Weiss et al., 2013). Importantly, MST has been found to reduce long-term rates of criminal behavior for as long as 5 years after treatment. MST is also cost-effective; its costs are 10 times less than conventional interventions and its estimated savings over the years are about $10 to $20 for each dollar spent on MST (Klietz, Borduin, & Schaeffer, 2010).

Since studies of MST have not yet differentiated between adolescents who show life-course–persistent and those with adolescence-limited patterns of antisocial behavior, it is difficult to know whether successful outcomes reported for this approach apply

**TABLE 9.6** | **The Nine Principles of Multisystemic Therapy (MST)**

1. **Finding the fit** The primary purpose of assessment is to understand the "fit" between the identified problems and their broader systemic context.
2. **Positive and strength-focused** Therapeutic contacts emphasize the positive and use systemic strengths as levers for change.
3. **Increasing responsibility** Interventions are designed to promote responsible behavior and decrease irresponsible behavior among family members.
4. **Present-focused, action-oriented, and well defined** Interventions are present-focused and action-oriented, targeting specific and well-defined problems.
5. **Targeting sequences** Interventions target sequences of behavior within and between multiple systems that maintain identified problems.
6. **Developmentally appropriate** Interventions are developmentally appropriate and fit the developmental needs of the youth.
7. **Continuous effort** Interventions are designed to require daily or weekly effort by family members.
8. **Evaluation and accountability** Intervention efficacy is evaluated continuously from multiple perspectives, with providers assuming accountability for overcoming barriers to successful outcomes.
9. **Generalization** Interventions are designed to promote treatment generalization and long-term maintenance of therapeutic change by empowering caregivers to address family members' needs across multiple systemic contexts.

Based on Table 2.1 in Multisystemic Therapy for Antisocial Behavior in Children and Adolescents (2nd ed.), by S. W. Henggeler, S. K. Schoenwald, C. M. Borduin, M. D. Rowland, & P. B. Cunningham. New York: Guilford Press.

equally to both groups. It is possible that part of the success of MST may be in helping youths on the adolescent-limited path decrease their association with deviant peers and, by doing so, lowering the age at which they cease delinquent behavior.

## Preventive Interventions

Until recently, treatments for older children with conduct problems were given far greater attention than programs for early intervention and prevention. Fortunately, this situation is changing, with a growing recognition of the need for intensive home and school-based interventions that can compete with the child's negative developmental history, poor family and community environment, and deviant peer associations (Prinz & Sanders, 2007; Wilson & Lipsey, 2007). Annual family checkups that provide tailored PMT to preschool-age children within the context of early childhood social, health, and educational services have also been developed to prevent early-onset pathways of antisocial behavior (Dishion et al., 2013). The main assumptions of preventive interventions are (Webster-Stratton, 1996):

- Conduct problems can be treated more easily and more effectively in younger than in older children.
- By counteracting risk factors and strengthening promotive factors at a young age, it is possible to limit or prevent the escalating developmental trajectory of increased aggression, peer rejection, self-esteem deficits, conduct disorder, and academic failure that is commonly observed in children with childhood-onset conduct problems.
- In the long run, preventive interventions will reduce the substantial costs to the educational, criminal justice, health, and mental health systems that are associated with conduct problems.

Carolyn Webster-Stratton has developed an intensive and multifaceted early-intervention program for parents and teachers of 2- to 10-year-old children with or at risk for conduct problems (Incredible Years; Webster-Stratton & Reid, 2010). This program uses interactive videotapes as a foundation for training, which permits widespread use at a relatively low cost. In addition to teaching child management skills, the program also addresses the associated individual, family, and school difficulties that accompany conduct problems. Parents are taught personal self-control strategies for managing anger, depression, and blame. As a result, they learn effective communication skills, strategies for coping with conflict at home and at work, and ways to strengthen social supports. Teachers are taught ways to strengthen positive relationships with students, effective classroom discipline, strategies for teaching social skills, anger management, problem-solving skills, and how to increase collaboration with parents (Webster-Stratton & Herman, 2010). In addition to the parent and teacher training programs, there is also an Incredible Years Child Training program for 3- to 8-year-olds, who meet with a therapist in groups of six for 2 hours a week. Children view videotapes of conflict situations at school and home that illustrate problem-solving and social skills. Following this, children discuss feelings, generate ideas for more effective responses, and role-play alternative behaviors.

A number of studies have provided support for the effectiveness of these early interventions in reducing later conduct problems and maintaining positive outcomes in adolescence for two-thirds or more of children whose parents are involved (Webster-Stratton, Reid, & Hammond, 2004; Webster-Stratton, Rinaldi, & Reid, 2010). This early intervention/prevention program is

now more frequently based in schools, with a growing emphasis on matching the type, timing, and amount of intervention to the level of risk and specific needs of the child and family (Webster-Stratton & Reid, 2010).

An innovative program designed to prevent the development of serious chronic antisocial behavior in high-risk children is Fast Track (Conduct Problems Prevention Research Group, 2007, 2010)—a multisite, collaborative research project that exemplifies the comprehensive effort needed to treat children at risk for serious conduct problems. The program was directed at high-risk kindergarten children who were identified in terms of their disruptive behavior and poor peer relations. The intervention began in grade 1 and continued through grade 10. It was divided into an elementary school phase and a transition to middle and high school phase, with each phase including goals and interventions relevant to successful adjustment during each of these developmental periods.

The goals were to reduce disruptive and aggressive behaviors at home and school and to improve the quality of the child's relationships with parents, teachers, and peers. Children were taught the social–cognitive skills needed for effective interpersonal problem solving and emotion regulation. Other important goals were to strengthen academic skills, especially reading, and to improve the quality of the relationship between family members and school personnel. During the transition to adolescence, issues related to peer affiliation and peer influence, academic achievement and orientation, social cognition and identity development, and parent and family relations were addressed.

Five integrated treatment components were used to achieve these goals: (1) parent management training; (2) home visiting/case management; (3) social–cognitive skills training; (4) academic tutoring; and (5) teacher-based classroom intervention. Fast Track interventions were implemented with close collaboration among parents, teachers, and project staff. The strengths of the program were that they targeted the deficits and determinants that research has shown to be important in youths with conduct problems and that they used treatment procedures for which there is already some empirical support (Eyberg et al., 2008).

The findings from the Fast Track intervention are complex, since multiple behaviors and attitudes were assessed over a wide age range and outcomes for various behaviors and areas of functioning differed over time. In general, the overall results indicate that the intervention had a significant impact, particularly with respect to reducing conduct problems and enhancing the child's social competence and family relations. However, interventions were less successful in reducing disruptive behavior in the classroom or improving academic performance. Importantly, by grade 9, for children who initially had the highest risk for conduct problems (top 3%), the intervention prevented 75% of CD cases. In contrast, the intervention had little impact on children who were initially at only moderate levels of risk (Slough, McMahon, & The Conduct Problems Prevention Research Group, 2008). Taken together, the findings support the efficacy of the Fast Track intervention for children at highest initial risk for conduct problems. For these children, Fast Track was effective in preventing diagnoses of CD, ODD, and ADHD, highlighting the importance of interventions that target children with the highest risk at a young age (Conduct Problems Prevention Research Group, 2011). Only time will tell whether this all-out effort will result in changes in public policy and will achieve its intended long-term goal of preventing serious chronic antisocial behavior and to enhance psychosocial outcomes into late adolescence and young adulthood (Dodge & McCourt, 2010).

Although tremendous advances have been made in the treatment and prevention of conduct problems, much work remains to be done. The main conclusion to be drawn from intervention and prevention efforts over the past 100 years is that *the degree of success or failure in treating antisocial behavior depends on the type and severity of the child's conduct problem and related risk and protective factors* (Kazdin & Wassell, 1999). Children who come from mostly middle-class healthy families and who have mild conduct problems are likely to benefit from individual, parent, family, and school-based interventions; those who come from highly dysfunctional homes and poor neighborhoods and who display severe and persistent problems are likely to benefit very little, if at all, unless early, much more intensive, and long-term interventions are used. If interventions are to succeed, it will also be necessary to find cost-effective interventions and ways to help families persevere with interventions that could prove to have real benefits (Rutter, 2003b). Although significant short-term gains for children with severe conduct problems have been achieved using intensive interventions, the degree of normalization and long-term impact of these approaches is yet to be determined.

## Section Summary

### Treatment and Prevention

- Considerable efforts to help children and adolescents with conduct problems have led to several approaches with some proven success.

- The focus of parent management training (PMT) is on teaching parents to change their child's behavior in the home.
- The underlying assumption of problem-solving skills training (PSST) is that faulty perceptions and appraisals of interpersonal events trigger antisocial responses. The focus is on changing behavior by changing the way the child thinks in social situations.
- Multisystemic therapy (MST) is an intensive approach that is carried out with all family members, school personnel, peers, juvenile justice staff, and other individuals in the adolescent's life.
- Recent efforts have focused on trying to prevent conduct problems through intensive programs of early intervention/prevention.
- The degree of success or failure in treating antisocial behavior depends on the type and severity of the child's conduct problem and related risk and protective factors.

# Study Resources

## SECTION SUMMARIES



## KEY TERMS

10

# Depressive and Bipolar Disorders

*This is my depressed stance. When you're depressed, it makes a lot of difference how you stand. The worst thing you can do is straighten up and hold your head high because then you'll start to feel better. If you're going to get any joy out of being depressed, you've got to stand like this.*

—Charlie Brown (Charles M. Schulz, 1922–2000)

## CHAPTER PREVIEW

**DONNA**

### Desperate Despair

Donna, age 12, says, "Sometimes I feel like jumping off the roof or finding some other way to hurt myself." Over the past 3 months, Donna has become more and more withdrawn, and her feelings of sadness, worthlessness, and self-hatred scare her. Her teacher describes Donna as "a loner who seems very troubled and unhappy." She's always been a good student, but she is now having difficulty concentrating, is failing tests, and feels totally unmotivated. At home, Donna is having trouble sleeping, has no appetite, and frequently complains of headaches. Most days she stays in her room and does nothing. When her mother asks her to do something, Donna becomes extremely upset. Her mother says Donna is "moody and irritable most of the time." (Based on authors' case material)

**MICK**

### Up and Down

Mick, age 16, is moody all of the time. Sometimes he is sad, sullen, and apathetic. At other times he is full of life and energy, or intensely angry. When full of energy, he can go with little or no sleep for days without feeling tired. He moves constantly, talks incessantly, and cannot be interrupted. These extreme changes in mood make Mick feel out of control, and sometimes he thinks about hurting himself. He is frightened by his thoughts and drinks or uses drugs when they are available to reduce the pain. (Based on authors' case material)

PERHAPS YOU KNOW A child or teen who seems constantly unhappy, shows little enthusiasm for anything, is moody, or—at worst—thinks life just isn't worth living. This child may have a **mood disorder** (also called an *affective disorder*), in which a disturbance in mood is the central feature. Mood is broadly defined as a feeling or emotion—for example, sadness, happiness, anger, elation, or crankiness. Children with mood disorders suffer from extreme, persistent, or poorly regulated emotional states, such as excessive unhappiness, ongoing irritability or anger, or swings in mood from deep sadness to high elation. Mood disorders are one of the most common, chronic, and disabling illnesses in young people (Kessler et al., 2012a, b).

## OVERVIEW OF MOOD DISORDERS

Mood disorders come in several types. At one end of the spectrum is severe depression. Like Donna, children who have severe depression suffer from **dysphoria**, a state of prolonged bouts of sadness (Kovacs & Yaroslavsky, 2014). They feel little joy in anything they do and lose interest in nearly all activities, a state known as **anhedonia**. In the words of one depressed teen:

> Depression makes you lose interest in all the stuff you used to think was fun. You might quit playing guitar or drop out of yearbook, and claim that you just don't have the energy or desire to pursue extracurricular activities—or curricular activities, for that matter. From Solin, 1995.

Many young people with depression express these combined feelings of sadness and loss of interest or pleasure. However, some may never report feeling sad. Rather, they express their depression through their irritable mood. **Irritability** refers to easy annoyance and touchiness, characterized by an angry mood and temper outbursts (Stringaris, 2011). Others may describe these children as cranky, grouchy, moody, short-fused, or easily upset. Being around them is difficult because any little thing can set them off. Irritability is one of the most common co-occurring symptoms of depression, present in as many as 80% of clinic-referred and 36% of community samples of youngsters with depression (Stringaris et al., 2013).

At the other end of the mood spectrum are a smaller number of youths, those like Mick, who also experience episodes of **mania**, an abnormally elevated or expansive mood, increased goal-directed activity and energy, and feelings of **euphoria**, which is an exaggerated sense of well-being. They suffer from an ongoing combination of extreme highs and extreme lows, a condition known as **bipolar disorder** (**BP**) or *manic–depressive illness*. Their highs may alternate with lows, or they may feel both extremes at about the same time.

The two major types of mood disorders in the *Diagnostic and Statistical Manual of Mental Disorders*, 5th edition (DSM-5) are depressive disorders and bipolar disorders. We discuss each of these in the sections that follow.

## Section Summary

### Overview of Mood Disorders

- Children with mood disorders suffer from extreme, persistent, or poorly regulated emotional states—for example, excessive unhappiness, irritability, or swings in mood from deep sadness to high elation.

(*continues*)

## Section Summary (*continued*)

- Mood disorders are common and are among the most persistent and disabling illnesses in young people.
- There are two major types of mood disorders: depressive disorders and bipolar disorders.

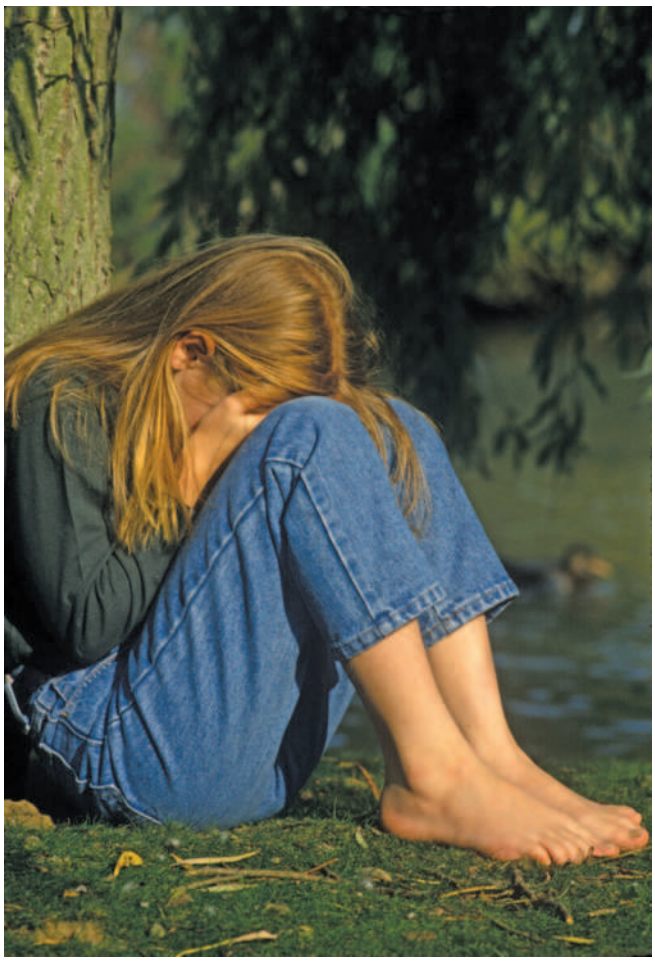
# DEPRESSIVE DISORDERS

*"And how are you?" said Winnie-the-Pooh.*
*Eeyore shook his head from side to side.*
*"Not very how," he said. "I don't seem to have felt at all how for a long time."*

—A. A. Milne, *Winnie-the-Pooh* (1926)

Depression refers to a pervasive unhappy mood, the kind of gloomy feeling displayed by Eeyore, the sad and indecisive old gray donkey in *Winnie-the-Pooh*. The symptoms of depression are so universal that depression is sometimes called "the common cold of psychopathology." Everyone feels sad, blue, out of sorts, or "down in the dumps" at times. (Even reading or writing about depression can be a real downer—can anyone think of a way to put a positive spin on feelings of dejection, hopelessness, irritability, loneliness, or self-blame?) Sometimes our sadness is a normal reaction to an unfortunate event in our lives like losing a friend or a job. At other times, we may feel depressed without really knowing why. These feelings soon pass, however, and we resume our normal activities. Clinical depression, in contrast, is more severe than the occasional blues or mood swings that everyone gets from time to time.

Childhood is usually thought of as a happy and carefree time, a period unfettered by the worries, burdens, and responsibilities of adulthood. We tend to think of young people as positive and upbeat, not depressed. In fact, a common reaction to hearing that a child is depressed is "What does she have to be depressed about?" Even when children experience disappointment, disapproval, or other inevitable negative events in their lives, their sadness, frustration, and anger are expected to be short-lived. When children become sad, irritable, or upset, parents often attribute the negative moods to temporary factors—such as a lack of sleep or not feeling well—and expect the moods to pass. Thus, for a long time it was thought that children did not get depressed, and when they did, the depression would be short-lived. We now know this is not true. Current estimates indicate that more than 3 million children and adolescents in the United States suffer from significant depression each year (Kessler et al., 2012a).



David Robinson /Bubbles Photolibrary / Alamy

Depression in children goes well beyond normal mood swings.

Unlike most children, who bounce back quickly when they are sad, children who are depressed cannot seem to shake their sadness, and it begins to interfere with their daily routines, social relationships, school performance, and overall functioning. Depressed youths often have accompanying problems such as anxiety or oppositional/conduct disorders. Although clinical depression may resemble the normal emotional dips of childhood, for many young people it is pervasive, disabling, long-lasting, and life-threatening (Abela & Hankin, 2008b; Hammen, Rudolph, & Abaied, 2014). Unfortunately, depression often goes unrecognized and untreated because parents and, in some cases, teachers may not recognize the child's underlying subjective negative mood.

## History

Not long ago, people doubted the existence of depression in children. This mistaken belief was rooted in traditional psychoanalytic theories, which viewed

depression as hostility or anger turned inward. Because children lacked sufficient superego development to permit aggression to be directed against the self, it was believed that they were incapable of experiencing depression (Rochlin, 1959). In another mistaken view, symptoms of depression were considered normal and passing expressions of certain stages of development, a belief that also has proved false. Depression in young people is a recurrent problem, as it is for adults.

As depression in children was acknowledged, a popular view emerged that children express depression in a much different way than adults, ways that are often indirect and hidden. This idea came to be known as *masked depression*. It was thought that any known clinical symptom in children, including hyperactivity, learning problems, aggression, bed-wetting, separation anxiety, sleep problems, and running away, could be a sign of an underlying but masked depression (Cytryn & McKnew, 1974). Because this concept is too encompassing to be useful, the once popular notion of masked depression has been rejected. Depression in children is not masked, but it may simply be overlooked because it frequently co-occurs with more visible disorders, such as conduct problems.

## Depression in Young People

Almost all young people experience some symptoms of depression, and many experience significant depression at some time (Avenevoli et al., 2008). These youngsters display lasting depressed mood while facing real or perceived distress and experience disturbances in their thinking, physical functioning, and social behavior. Suicidal behavior among teens, which is frequently associated with depression, is also a very serious concern (Cha & Nock, 2014).

As many as 90% of youngsters with depression show significant impairment in their daily functions, and, even when they recover from their depression, they are likely to experience recurrent bouts of depression and continued impairments (Simonoff et al., 1997). The long-lasting emotional suffering, problems in everyday living, and heightened risk of these youths for suicide, substance use, other mental health problems, poorer health outcomes, and higher health care costs make depression in young people a significant concern (Fombonne et al., 2001a; Keenan-Miller, Hammen, & Brennan, 2007).

## Depression and Development

Children express and experience depression differently at different ages (Weiss & Garber, 2003). An infant may show sadness by being passive and unresponsive; a preschooler may appear withdrawn and inhibited; a school-age child may be argumentative and combative or complain of feeling sick; a teenager may express feelings of guilt and hopelessness, sulk, or feel misunderstood. These examples are not various types of depressions, but likely represent different stages in the developmental course of the same process.

No one pattern fits all children within a particular age group or developmental period, and depression is not clearly recognizable as a clinical disorder using DSM criteria until children are older. Depression in children under the age of 7 is diffuse and less easily identified. However, some studies have found that age-adjusted diagnostic criteria can be used to identify and treat depression in children as young as 3 to 5 years (Luby, 2013). It is important to recognize depressive symptoms in preschool children, since their symptoms can persist or reoccur and develop into depressive disorders during late childhood or early adolescence (Luby, Si et al., 2009).

We know the least about depression in infants (Guedeney, 2007). In the 1940s, American psychoanalyst René Spitz described a condition he called *anaclitic depression,* in which infants raised in a clean but emotionally cold institutional environment displayed reactions that resembled depression (Spitz & Wolf, 1946). These infants displayed weeping, withdrawal, apathy, weight loss, and sleep disturbance. They also showed an overall decline in development, and in some cases, death. Although Spitz attributed this depression to an absence of mothering and the lack of opportunity to form an attachment, other factors, such as physical illness and sensory deprivation, may also have played a role. Nevertheless, even young children exposed to institutional neglect and later adopted may display emotional and behavioral disturbances that place them at heightened risk for depression and other internalizing disorders (Stellern et al., 2014).

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Depression in institutionalized infants: When observed over time, these infants display a physical appearance that in an adult might be described as depression.

It also became clear that similar symptoms could occur even in noninstitutionalized infants raised in severely disturbed families in which the mother was depressed, psychologically unavailable, or physically abusive. These infants may experience sleep disturbances, loss of appetite, increased clinging, apprehension, social withdrawal, crying, and sadness (Goodman & Brand, 2009).

Preschool children who are depressed may appear extremely somber and tearful. They generally lack the exuberance, bounce, and enthusiasm in their play that characterize most preschoolers. They may display excessive clinging and whiny behavior around their mothers, as well as fears of separation or abandonment. In addition to getting upset when things do not go their way, many are irritable for no apparent reason. Negative and self-destructive verbalizations may occur, and physical complaints such as stomachaches are common (Luby et al., 2003).

School-age children with depression display many of the symptoms of preschoolers in addition to increased irritability, disruptive behavior, temper tantrums, and combativeness. A parent may say, "Nothing ever pleases my child—she hates herself and everything around her." School-age children may look sad, but are often unwilling to talk about their sad feelings. Physical symptoms may include weight loss, headaches, and sleep disturbances. Academic difficulties and peer problems are also common, and may include frequent fighting and complaints of not having friends or being picked on. Suicide threats may also begin to occur at this age.

Preteens and teens with depression display many of the symptoms of younger children, in addition to increasing self-blame and expressions of low self-esteem, persistent sadness, and social inhibition. A child may say, "I'm stupid" or "Nobody likes me." Feelings of isolation from family are also common. The preteen may also experience an inability to sleep or may sleep excessively. Disturbances in eating are also common. Teens show increased irritability, loss of feelings of pleasure or interest, and worsening school performance. Angry discussions with parents regarding normal parent–teen issues, such as choice of friends or curfew, are also more common. Other symptoms at this age include a negative body image and self-consciousness, physical symptoms such as excessive fatigue and energy loss, feelings of loneliness, guilt, and worthlessness, and suicidal thoughts, plans, and attempts.

Many of these symptoms and behaviors may also occur in children and teens who are developing normally or in those with other disorders or conditions. Therefore, the presence of sad mood, diminished interest or pleasure, or irritability is essential for diagnosing depression. In addition, regardless of the child's age, the symptoms must reflect a change in behavior, persist over time, and cause significant impairment in functioning (Rudolph & Lambert, 2007).

## Anatomy of Depression

The term *depression* has been used in various ways. It is important to distinguish between depression as a symptom, depression as a syndrome, and depression as a disorder.

As a *symptom*, depression refers to feeling sad or miserable. Depressive symptoms often occur without the existence of a serious problem, and they are relatively common at all ages. For most children, symptoms of depression are temporary, related to events in the environment, and not part of any disorder.

As a *syndrome*, depression is more than a sad mood. A syndrome refers to a group of symptoms that occur together more often than by chance. Along with sadness, the child may display a reduced interest or pleasure in activities, cognitive and motivational changes, and somatic and psychomotor changes. As a syndrome, depression represents an extreme on a dimension reflecting the number or severity of co-occurring symptoms that the child displays. The occurrence of depression as a syndrome is far less common than isolated depressive symptoms, and it often includes mixed symptoms of anxiety and depression, which tend to cluster on a single dimension of *negative affect* (Ollendick et al., 2003).

As a *disorder*, depression comes in several forms. We will consider three types. The first, **major depressive disorder (MDD)**, has a minimum duration of 2 weeks and is associated with depressed or irritable mood, loss of interest or pleasure, other symptoms (e.g., sleep disturbances, difficulty concentrating, feelings of worthlessness), and significant distress or impairment in functioning. The second, **persistent depressive disorder [P-DD]**, or **dysthymia**, is associated with depressed or irritable mood, generally fewer, less severe, but longer-lasting symptoms (a year or more in children) than MDD, and significant impairment in functioning. The third, **disruptive mood dysregulation disorder (DMDD)**, is a recently introduced depressive disorder characterized by: (1) frequent and *severe temper outbursts* that are extreme overreactions to the situation or provocation; and (2) chronic, persistently *irritable or angry mood* that is present between the severe temper outbursts.

The common characteristic of all depressive disorders is the presence of sad, empty, or irritable mood, along with somatic and cognitive symptoms that interfere with the individual's functioning. The differences among depressive disorders are related to their duration, timing, associated features, or presumed causes. As we discuss next, these disorders are defined using DSM-5 criteria.

## Section Summary

### Depressive Disorders

- Depression in young people involves numerous and persistent symptoms, including impairments in mood, behavior, attitudes, thinking, and physical functioning.
- For a long time it was mistakenly believed that depression did not exist in children in a form comparable to depression in adults.
- It is now known that depression in young people is prevalent, disabling, and often under-referred.
- The way in which children express and experience depression changes with age.
- It is important to distinguish between depression as a symptom, a syndrome, and a disorder.
- Three types of DSM-5 depressive disorders are major depressive disorder (MDD), persistent depressive disorder [P-DD], or dysthymia, and disruptive mood dysregulation disorder (DMDD).

# MAJOR DEPRESSIVE DISORDER (MDD)

### JOEY

#### Feeling Worthless and Hopeless

Ten-year-old Joey's mother and teacher are concerned about his irritability and temper tantrums at home and at school. With little provocation, he bursts into tears, yells, and throws objects. In class he seems to have difficulty concentrating and seems easily distracted. Increasingly shunned by his peers, he plays by himself at recess, and at home spends most of his time in his room watching TV. His mother notes that he has been sleeping poorly and has gained 10 pounds over the past couple of months from constant snacking. The school psychologist has ruled out learning disabilities or ADHD; instead, she says Joey is a deeply unhappy child who expresses feelings of worthlessness and hopelessness, and even a wish that he would die. These feelings began about 6 months ago when Joey's father, divorced from his mother for several years, remarried and moved to another town, and now spends far less time with Joey.

Adapted from Hammen & Rudolph, 2003.

### ALISON

#### "I Couldn't Take It Any More"

Alison, age 17, gets high grades, is a talented musician, and is attractive. However, for the past 3 years, she has been fighting to stay alive. "There are times when I was in school and I would start to cry—I had no idea why. My friends would say, 'What have you got to be depressed about, Alison? You're smart, talented, and can have any boy you want.' When my closest friend moved away 3 years ago, I was really lonely," says Alison. "I'd write notes about suicide and talk about killing myself. I couldn't eat and was tired most of the time. Even the smallest decision was overwhelming. Some days I'd never get out of bed I was so depressed. I couldn't stand school and hated everyone." Alison's feelings of hopelessness lasted for days, then weeks, then months. Finally, "I couldn't take it anymore," says Alison. "I wanted to die—so I tried to kill myself." (Based on authors' case material)

Although Joey and Alison differ in age and symptoms, both display the key features of MDD: sadness, loss of interest or pleasure in nearly all activities, irritability, plus a number of additional specific symptoms that are present during the same 2-week period. These symptoms must also represent a change from previous functioning. DSM-5 criteria for MDD are presented in Table 10.1.

A diagnosis of MDD depends on the presence of a major depressive episode plus the exclusion of other conditions, such as the prior occurrence of a manic episode (in this case, a diagnosis of bipolar disorder would be made). It also requires ruling out physical factors such as the physiological effects of a substance, another medical condition that may have caused or prolonged the depression, depression that is part of normal bereavement, and underlying thought disorders. Finally, the symptoms must cause clinically significant distress or impairment in important areas of life functioning (e.g., social, academic).

If full criteria are currently met for MDD, DSM-5 also provides for severity ratings of "mild," "moderate," or "severe" based on the number of symptoms in excess of those required to make the diagnosis, the amount of symptom distress and its manageability, and the extent of impairment in life functioning caused by the symptoms. In addition to severity, other specifiers are used to designate whether this is a single or recurrent depressive episode; whether a previous episode is in partial or full remission; whether the episode includes psychotic features (presence of delusions and/or hallucinations); or, whether the episode is accompanied by other features, for example, anxious distress (e.g., feeling tense, restless, or that something awful may happen).

The cases of Joey and Alison highlight three important points about the diagnosis of MDD in children and adolescents (Hammen et al., 2014):

**TABLE 10.1** | **Diagnostic Criteria for Major Depressive Disorder**

**DSM-5**

**(A)** Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

**Note:** Do not include symptoms that are clearly attributable to another medical condition.

**(1)** Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad, empty, hopeless) or observation made by others (e.g., appears tearful). (**Note:** In children and adolescents, can be irritable mood.)

**(2)** Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by subjective account or observation).

**(3)** Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. (**Note:** In children, consider failure to make expected weight gains).

**(4)** Insomnia or hypersomnia nearly every day.

**(5)** Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).

**(6)** Fatigue or loss of energy nearly every day.

**(7)** Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).

**(8)** Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others)

**(9)** Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

**(B)** The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

**(C)** The episode is not attributable to the physiological effects of a substance or to another medical condition.

**Note:** Criteria A-C represent a major depressive episode.

**Note:** Responses to a significant loss (e.g., bereavement, financial ruin, losses from a natural disaster, a serious medical illness or disability) may include the feelings of intense sadness, rumination about the loss, insomnia, poor appetite, and weight loss noted in Criterion A, which may resemble a depressive episode. Although such symptoms may be understandable or considered appropriate to the loss, the presence of a major depressive episode in addition to the normal response to a significant loss should also be carefully considered. This decision inevitably requires the exercise of clinical judgment based on the individual's history and the cultural norms for the expression of distress in the context of loss.

**(D)** The occurrence of the major depressive episode is not better explained by schizoaffective disorder, schizophrenia, schizophreniform disorder, delusional disorder, or other specified and unspecified schizophrenia spectrum and other psychotic disorders.

**(E)** There has never been a manic episode or hypomanic episode.

**Note:** This exclusion does not apply if all of the manic-like or hypomanic-like episodes are substance-induced or are attributable to the physiological effects of another medical condition.

*Source:* Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, American Psychiatric Association.

- The same DSM-5 criteria for diagnosing adults can be used to diagnose school-age children and adolescents.
- Because children's disruptive behaviors attract more attention, or are more easily observed as compared with internal, subjective suffering, depression in children can be easily overlooked.
- Some features of depression are likely more common in children and adolescents than in adults—notably, irritable mood. In light of this, DSM-5 specifies that irritable mood can substitute for depressed mood in diagnosing depression in children. However, most children with depression display either depressed mood alone (58%) or depressed and irritable mood (36%)—irritable mood alone is rare (6%) (Stringaris et al., 2013).

Young people with MDD frequently display similar symptoms and have comparable rates of comorbidity and recurrence as adults (Birmaher et al., 2004). However, as compared with adults, clinic-referred youths with MDD have almost exclusively first-episode depressions, will recover somewhat faster from their depressive episodes, and are at greater risk for developing bipolar disorder. Children who develop MDD suffer from their disorder for many years longer than adults, making early-onset of this disorder a particularly severe form of affective illness (Kovacs, 1996).

## Prevalence

Between 2% and 8% of all youths aged 4 to 18 experience MDD each year (Costello, Erkanli, & Angold, 2006; Kessler et al., 2012a). Depression is relatively rare among preschool and school-age children (about 1% to 3%; Bufferd et al., 2012; Egger & Angold, 2006), but increases twofold to threefold by adolescence (NIMH, 2003). Since depression comes and goes, prevalence estimates vary with the time frame during which symptoms are assessed. Lifetime prevalence estimates—whether a young person has ever been depressed—range from about 10% to 20% or higher (Merikangas et al., 2010; Rohde, Lewinsohn et al., 2013).

Even though these rates are so high, they may underestimate the problem. First, the estimates using a DSM-5 diagnosis of MDD might be lower than the self-reported symptoms of depression. Second, many children who just barely fail to meet diagnostic criteria for MDD still show significant impairments in their social competence, cognitive attributions, coping skills, family relations, and experience of stress. They are also at greater risk than other youths for developing future depression and other disorders, such as substance abuse (Gotlib, Lewinsohn, & Seeley, 1995).

The modest increase in depression from preschool to elementary school is likely not biologically based, but rather is a reflection of the school-age child's growing self-awareness and cognitive capacity, verbal ability to report symptoms, and increased performance and social pressures. In contrast, the sharp increase in depression in adolescence appears to be the result of biological maturation at puberty interacting with important developmental changes that occur during this tumultuous period. This hypothesis is supported by the emergence of large sex differences in depression after puberty, the emergence of bipolar disorder, and the relative stability in rates of depression through adolescence (Birmaher et al., 1996).

## Comorbidity

**RAYMOND**

### Depressed and Enraged

Raymond, age 16, lives alone with his single mother. For the past few months he has been persistently sad and unhappy, overcome with feelings of worthlessness. He is socially withdrawn, and he spends most of his time alone at home or avoiding contact with peers on the days when he manages to attend school. He is constantly tired but still finds it difficult to sleep, lying awake at night for hours and then struggling to drag himself from bed in the morning. Both he and his mother are concerned about his weight, which has increased substantially due to his inability to control his appetite for chips, candy, and soda. Even if he makes it to school, he finds he is unable to concentrate on his work.

Raymond's listlessness and withdrawal are countered, however, by his defiance and repeated outbursts of anger and aggression. He frequently lashes out in rage at his mother, and he recently punched his fist through a wall and a door at home. He also has been in several fights with other students at school as a result of being teased by his peers. He rarely complies with rules and limits at home or at school, leading to frequent conflicts with his mother and with school authorities. The event that precipitated Raymond's current referral was his arrest for shoplifting at a local store.

(Adapted from Compas & Hammen, 1994)

Like Raymond, who has MDD and a co-occurring conduct disorder, as many as 90% of young people with depression have one or more other disorders, and 50% have two or more (Simonoff et al., 1997). The most frequent co-occurring disorders in youngsters with MDD are anxiety disorders, particularly generalized anxiety disorders, specific phobias, and separation anxiety disorders. Depression and anxiety become more visible as separate but co-occurring disorders as the severity of the child's problem increases and as the child gets older (Gurley et al., 1996). Persistent depressive disorder, conduct problems, attention-deficit/hyperactivity disorder (ADHD), and substance-use disorder are also common in youngsters with MDD (Birmaher et al., 1996; McKowen et al., 2014). In the case of conduct problems, the extent to which young people with MDD experience oppositional defiant disorder (ODD) or conduct disorder (CD) seems to be directly related to the presence of irritable mood (Stringaris et al., 2013). Further, about 60% of adolescents with MDD have a comorbid personality disorder, which is most commonly borderline personality disorder—characterized by instability of interpersonal relationships, self-image, and affects and marked impulsivity (Muehlenkamp et al., 2011).

Many co-occurring disorders are present before MDD manifests, and they are likely to persist after the child is no longer depressed. For example, an early-onset anxiety disorder is a strong predictor of later depression and precedes depression in 85% of young people with both disorders (Kessler et al., 2012c; Rohde, 2009). A multiple-pathways model for understanding the strong relationship between anxiety and depression has been proposed by Cummings, Caporino, and Kendall (2013). In one common pathway, the child may have a general propensity (temperamental, biological, environmental) to anxiety.

If left untreated, this may lead to anxiety-related impairments (e.g., cognitive biases, negative affectivity) that become risk factors for the development of depression.

The presence of a co-occurring disorder is significant because it can increase the risk for recurrent depression, increase the duration and severity of depressive episodes, and increase the risk for suicide attempts. The presence of another disorder also decreases a depressed youth's response to treatment and is related to less effective treatment outcomes (Birmaher et al., 1996).

## Onset, Course, and Outcome

The onset of depression in adolescence may be gradual or sudden. Either way, a youth typically has a history of milder episodes of depression that do not meet DSM-5 diagnostic criteria. Most adults with depression recall having their first depressive episode between the ages of 15 and 19 (Kessler, Merikangas, & Wang, 2007). However, prospective studies of children and adolescents usually find earlier ages at onset, most commonly between the ages of 13 and 15 (Merikangas et al., 2010).

The average episode of MDD in clinically referred children and adolescents lasts about 8 months, with longer episodes if a parent has a history of depression (Kaminski & Garber, 2002). Initial episodes are of shorter duration in community samples, with the average ranging from 4 months in childhood to 2 months in adolescence (Rohde, Lewinsohn et al., 2013). Although almost all young people eventually recover from their initial depressive episode, their disorder itself, unfortunately, does not go away (Birmaher, Arbelaez, & Brent, 2002). MDD has a chance of recurrence of about 25% within 1 year, 40% within 2 years, and 70% within 5 years. Thus, a significant number of youngsters develop a chronic, relapsing disorder that persists into young adulthood (Fombonne et al., 2001a).

Those with an onset of depression prior to age 15 and a recurrent episode prior to age 20 display more severe, chronic, suicidal depressions; greater co-occurring anxiety and worse social functioning at age 15; and poorer psychosocial outcomes at age 20 (Hammen et al., 2008). For youths who are hospitalized for depression, nearly half will be rehospitalized within 2 years after remission. In addition, about one-third of adolescents with MDD will develop a bipolar disorder within 5 years after the onset of their depression, known as a *bipolar switch* (DelBello et al., 2003). Thus, depression is a condition that endures over the course of development, creating a long-term social, emotional, and economic burden for the youth and the family.

Why do depressive episodes re-occur, and why does the length of time between episodes get progressively shorter? One possible explanation is that the first episode may sensitize the child to future episodes (Rudolph & Flynn, 2007). According to this idea, the first episode may be linked to a specific stressor and is accompanied by lasting changes in biological processes that heighten future reactivity to stress (Post et al., 1996). The initial externally produced changes in the brain can be conditioned so that following the first depressive episode, individuals are increasingly vulnerable to stress, and even nonsevere stress or minor events that resemble loss or stress experiences may result in depression (Stroud et al., 2011). This process is known as *stress sensitization* (Post & Weiss, 1998).

In addition to their recurring bouts of depression, the immediate and long-term prospects for children with MDD may include many other negative outcomes (Fergusson, Boden, & Horwood, 2007). For example, a history of depression during the school years also increases the risk for later delinquency, tobacco use, substance-use disorder, suicidal behavior, impairment, school dropout, poor work record, marital problems, and health-service use (Gotlib et al., 1998; Rice et al., 2007).

The overall outcome for young people with depression is not optimistic. Although almost all youths will recover from their depression, they continue to be at high risk for later episodes of mood and other disorders and for impaired social and academic functioning. One mother of a depressed teen paints a realistic picture of the long-term outcome for a child who suffers from depression:

> Depression in kids, when it hits them in their teens, leaves a hole in their lives. When they're young and just starting out in life, they're supposed to become independent. But that doesn't happen with depressed kids. They're out of synch and get left behind. And they never really catch up. That leaves a permanent scar. (Adapted from Owen, 1993, p. C1)

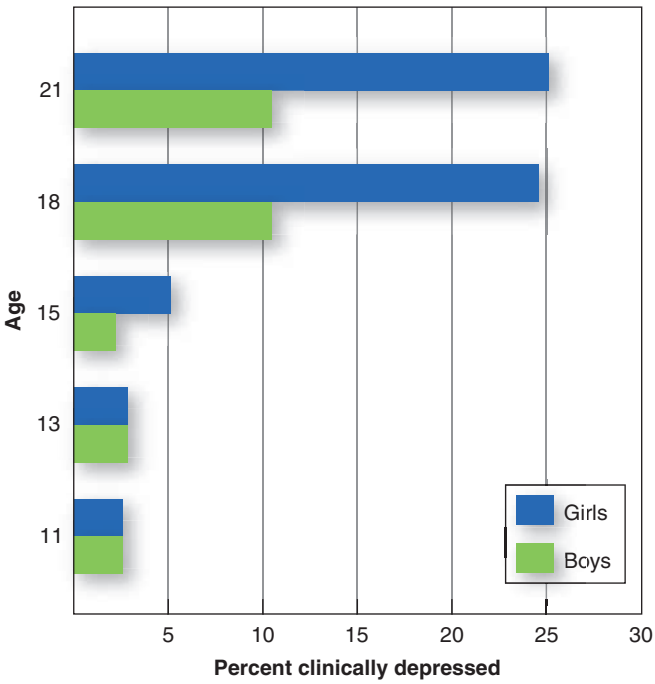
As they become adults, youngsters with a history of MDD continue to experience many negative long-term outcomes that include a high rate of suicidal behavior, adult depression and other psychiatric disturbances, high rates of psychiatric and medical hospitalizations, alcohol abuse/dependence, psychosocial impairments, lower educational achievement, and employment problems (Fombonne et al., 2001b; Jaycox et al., 2009). In general, these outcomes underscore the need for effective prevention and early intervention programs for young people with depression, which we discuss in a later section.

## Gender

In what has been called depression's double standard, females are twice as likely as males to suffer from

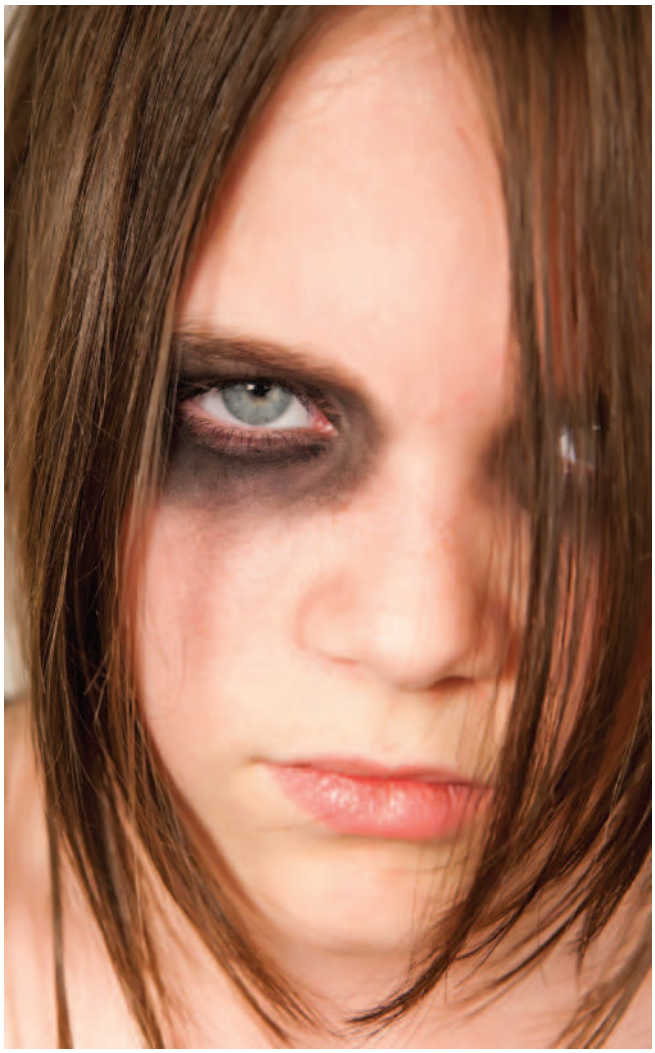
depression, are more susceptible to milder mood disorders, and are more likely to experience recurrent episodes (Zahn-Waxler, Race, & Duggal, 2005). This sex difference is not present among children ages 6 to 11, at which ages depression is reported to be equally common in boys and girls (Kessler et al., 2012a; Merikangas et al., 2010). However, sex differences in emotional reactivity are present in children who are depressed or at risk for depression as early as the preschool period, with boys displaying more anger and girls more sadness (Luby, Essex et al., 2009). In addition, sex differences in specific symptoms that forecast later depression (e.g., fearfulness, feelings of inadequacy, negative self-evaluation, and negative affect) may in fact be present prior to 10 years of age, with girls reporting significantly more of these symptoms than boys (Rudolph, Hammen, & Daley, 2006).

Sex differences in diagnosable depression begin between ages 13 and 15, when the rate rises for girls (Wade, Cairney, & Pevalin, 2002). As shown in ● Figure 10.1, rates of depression as well as sex differences in rates increase dramatically between ages 15 and 18 (Hankin, Wetter, & Cheely, 2008). The ratio of girls to boys is about 2:1 to 3:1 after puberty, a pattern that continues throughout adolescence and adulthood.




● **FIGURE 10.1** | The overall rate of depression and the proportion of females with depression increase dramatically during adolescence.

Based on "Development of Depression from Preadolescence to Young Adulthood: Emerging Gender Differences in a 10-year-Longitudinal Study" by B. L. Hankin L. Y. Abramson, T. E. Moffitt, P. A. Silva, R. McGee & K. E. Andell, 1998, 'Journal of Abnormal Psychology', 107, 128–140.



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Teenage girls are particularly vulnerable to depression and related problems.

Although depression occurs more frequently in girls than in boys, symptom presentation is generally quite similar for the two sexes. (Slightly more girls than boys report symptoms related to weight and appetite disturbances and feelings of worthlessness and guilt [Lewinsohn, Pettit et al., 2003].) However, the correlates of depression may differ for the sexes. For example, depression is more highly related to school-related stress in boys than in girls (Sund, Larsson, & Wichstrom, 2003).

The increase in depression during adolescence and the emergence of sex differences in depression at this time have led to a special interest in this developmental period (Rudolph et al., 2006). Many physical, psychological, and social changes during adolescence may heighten the risk for depression in girls. Hormonal changes in estrogen and testosterone may affect brain function, increasing sexual maturity may affect social

roles, interpersonal changes and expectations may result in heightened exposure to stressful life events, and nonnormative changes such as early maturation may lead to isolation from one's peer group (Hankin et al., 2008).

These changes may diminish self-worth, lead to depressed mood, and evoke self-focused attention. It is also thought that girls may be at higher risk than boys because they have a greater orientation toward cooperation and sociality. They also use ruminative coping styles to deal with stress (focusing on the symptoms of distress and its causes rather than on solutions)—especially stress involving interpersonal loss and disruptions. These two characteristics may put girls at a disadvantage during adolescence, when they face somewhat greater biological and stressful role-related challenges than boys (Zahn-Waxler et al., 2005). Interpersonal stress and a lack of social support are particularly salient aspects of depression for adolescent girls (Rudolph & Flynn, 2007).

Low birth weight has been found to predict depression in adolescent girls but not in adolescent boys, and girls born at a low birth weight are especially vulnerable to adversity after puberty (Costello et al., 2007; Van Lieshout & Boylan, 2010). This suggests that low birth weight may be a marker for poor intrauterine conditions that lead to adjustments in fetal development, which in turn have long-term consequences for girls' response to stress in adolescence. Research also suggests that increased levels of testosterone and estrogen at puberty, particularly when they occur in combination with social stress, increase the risk for depression in girls (Angold, Worthman, & Costello, 2003). Hormones and sleep cycles, which can alter mood, differ dramatically between boys and girls. One study of blood flow in regions of the brain during periods of sadness in men and in women found that although men and women considered themselves to be equally sad, their brain activity differed. When asked to feel sad on cue, both sexes activated regions of the prefrontal cortex, but women showed a much wider activation of the limbic system (George et al., 1996). These and other findings suggest that sex differences in depression may be partly rooted in biological differences in the brain processes that regulate emotions (Martel, 2013).

## Ethnicity and Culture

The incidence of depression has been found to vary across regions worldwide (Culbertson, 1997); however, few studies have examined ethnic, racial, or cultural differences in clinically depressed youths, and findings have been inconsistent (Anderson & Mayes, 2009). One study compared the prevalence of MDD across nine ethnic groups in a large community sample of children in grades 6 to 8 (Roberts, Roberts, & Chen, 1997). Of these groups, African American and Hispanic youths both had significantly higher rates of depression. However, only Hispanic youths with depression showed an elevated risk for impaired functioning. In another study, pubertal status was found to be a better predictor of depressive symptoms than chronological age in Caucasian girls, but not in African American or Hispanic girls (Hayward et al., 1999). Similarly, obesity in the sixth grade was found to be associated with a greater likelihood of depressed mood in the eighth grade for Caucasian girls but not for African American or Hispanic girls (Anderson et al., 2011).

A large community study of high school students found that nonwhite (African American, Hispanic, and Asian) adolescents reported more symptoms of depression than white adolescents (Rushton, Forcier, & Schectman, 2002). However, these differences likely reflect differences in socioeconomic status (SES), since depression and lower SES are related. Race and ethnicity are known sources of varying levels of exposure to stress and availability of resources. As a result, low SES may increase vulnerability to stress, and by doing so it may increase the likelihood of depression. In a longitudinal study of four race–ethnic groups (whites, African Americans, Hispanics, and Asians) during the transition from adolescence to young adulthood, it was found that race and ethnicity were important in understanding depressive symptoms during this period (Brown, Meadows, & Elder, 2007). In females, initial rates of depressive symptoms were highest for Hispanic and Asian teens and lowest for whites, with African American youths falling in between. As expected, males displayed lower levels of symptoms, but the findings for race–ethnic group differences were similar to those for females. Within gender, all groups showed decreases in symptoms over time; however, whites continued to display fewer depressive symptoms than the other three groups, particularly as compared with African Americans. This lasting race–ethnic inequality in depressive symptoms creates a risk for emotional and physical health in later life, as stress may accumulate in the context of a lack of resources.

## Section Summary

### Major Depressive Disorder (MDD)

- The key features of MDD are sadness, loss of interest or pleasure in nearly all activities, and irritability, plus many specific symptoms that are present for at least 2 weeks.
- The overall prevalence of MDD annually for youths 4 to 18 years of age is between 2% and 8%, with rates that are low during childhood but increase dramatically during adolescence. The likelihood that a youth has ever had MDD is higher, from 10% to 20% or more.

- The most frequent accompanying disorders in young people with MDD are anxiety disorders, persistent depressive disorder, conduct problems, ADHD, and substance-use disorder.
- Almost all young people recover from their initial depressive episode, but about 70% have another episode within 5 years and many develop bipolar disorder.
- Depression in preadolescent children is equally common in boys and girls, but the ratio of girls to boys is about 2:1 to 3:1 after puberty.
- The relationship between depression and race/ethnicity during childhood and adolescence is an understudied area.

In the next section, we discuss persistent depressive disorder, a milder but more chronic form of depression, about which we know relatively little as compared with MDD. Many children with persistent depressive disorder eventually develop MDD; therefore, the two disorders are related.

## PERSISTENT DEPRESSIVE DISORDER [P-DD] (DYSTHYMIA)

### DEBORAH

### A Childhood without Laughter

A few months ago, my mother unearthed some pictures of me as a baby that I had never seen before. One showed me at about 9 months old, crawling on the grass of Golden Gate Park. I was looking directly at the camera, my tongue sticking out of the corner of my mouth, and I was laughing happily. My face was lit from within and looked more than a little mischievous. I was absolutely transfixed by that photo for days. I would continually take it out of my wallet and stare at it, torn between laughter and tears. For a while I couldn't figure out what it was about the picture that drew me. Finally it hit me; this was the only picture of myself as a child that I had seen that showed me laughing. All the photos I had ever seen depicted a child staring solemnly or smiling diffidently, but never laughing. I looked at the Golden Gate Park picture and wished that I had remained that happy and that depression had not taken away my childhood. When I first was diagnosed with depression at age 24, I discussed my childhood with my doctor. Although it is hard to diagnose a child from 20 years in the past, it seemed clear to both of us that I had suffered from dysthymia (mild, long-term depression) probably from the time I was a small child.

Based on A Childhood Without Laughter by D. M. Deren.

Like Deborah, young people who suffer from **persistent depressive disorder (P-DD)** experience symptoms of depressed mood that occur for most of the day, on most days, and persist for at least 1 year. They are unhappy or irritable most of the time. (The sad and gloomy life of Eeyore the donkey in the 100 Acre Wood likely qualifies for a diagnosis of P-DD.) Combined with their chronic depressed (or irritable) mood, these children also display at least two somatic (e.g., eating problems, sleep disturbances, low energy) or cognitive symptoms (e.g., lack of concentration, low self-esteem, feelings of hopelessness) that are present while they are depressed. Although the symptoms of P-DD are chronic, they are less severe than those for children with MDD.

P-DD is a "new" category in DSM-5; it combines the previous DSM-IV categories of Dysthymic Disorder and MDD—Chronic. This was done because of the lack of differences between youths with a dysthymic disorder and those with a chronic type of major depression. In comparison to nonchronic MDD, chronic forms of depression, whether referred to as dysthymic disorder, chronic major depression, or P-DD are associated with a poorer response to treatment, greater long-term morbidity at follow-up, and greater familial loading for affective disorders (McCullough et al., 2003).

Children with P-DD are characterized by poor emotion regulation, which includes constant feelings of sadness, feelings of being unloved and forlorn, self-deprecation, low self-esteem, anxiety, anger, and temper tantrums (Masi et al., 2003). Some may experience **double depression**, in which MDD is superimposed on the child's previous P-DD, causing the child to present with both disorders (Klein, Shankman, & Rose, 2008).

The chronic nature of P-DD raises the issue of whether it is a mood disorder or a general personality style (Daley et al., 1999). For example, we all know people we would describe as "sad sacks"—nothing ever seems to make them happy. However, P-DD seems to follow a chronic course that is typical of mood disorders, and the similarities between P-DD and MDD in young people suggest that it is a mood disorder, not a personality style (Renouf & Kovacs, 1995). One study found that children with either MDD or P-DD alone did not differ in their clinical features, demographics, or associated characteristics, leading to unanswered questions about the validity of this distinction. However, those with both disorders were more severely impaired than children with just one of them (Goodman et al., 2000).

### Prevalence and Comorbidity

Rates of P-DD are lower than those of MDD, with approximately 1% of children and 5% of adolescents

displaying the disorder (Birmaher et al., 1996). The most prevalent co-occurring diagnosis with P-DD is MDD. During the course of their P-DD, as many as 70% of children may have an episode of major depression (Renouf & Kovacs, 1995). About half of the children with P-DD also have one or more co-occurring non-affective disorders that preceded the P-DD, including anxiety disorders, CD, and ADHD (Kovacs et al., 1994).

## Onset, Course, and Outcome

P-DD develops about 3 years earlier than MDD, most commonly around 11 to 12 years of age (Kovacs et al., 1997). Since P-DD frequently precedes MDD, it could be a precursor to its development (Lewinsohn, Hops et al., 1993). Childhood-onset P-DD has a prolonged duration, with an average episode length of 2 to 5 years.

Almost all children eventually recover from P-DD. On the other hand, they also have an extremely high risk of developing other disorders, especially MDD, anxiety disorders (separation anxiety disorder and generalized anxiety disorder are the most common), and conduct disorder (Klein et al., 2008; Masi et al., 2003). They are also at increased risk for the subsequent development of bipolar and substance-use disorders (Kovacs et al., 1994).

Adolescents with a history of P-DD report receiving less social support from friends. This finding appears to be unique to children with P-DD as compared with children with MDD (Klein, Lewinsohn, & Seeley, 1997). Those who recover from their P-DD have the same family relationships, cognitive styles, and school functioning as other children. The only area that continues to be affected is psychosocial functioning (Klein et al., 1997). However, it is not known whether deficits in psychosocial functioning precede or follow P-DD. They may be a predisposing factor for the development of P-DD, or a lasting scar of the illness (Renouf & Kovacs, 1995).

The early onset and extended duration of P-DD make it a serious problem. Children who develop the disorder at age 9 then recover 4 years later will have spent more than 30% of their entire lives and over 50% of their school-age years being depressed. Since depression is associated with many other academic, cognitive, family, and social problems, these long-lasting episodes of P-DD can have extremely harmful effects on development (Renouf & Kovacs, 1995). Since early-onset P-DD is almost always followed by MDD and sometimes by bipolar disorder, its early diagnosis may help to identify children at risk for later mood disorders and has important implications for prevention.

## Section Summary

### Persistent Depressive Disorder [P-DD] (Dysthymia)

- Children with P-DD display a depressive or irritable mood for most of the day, on most days for at least 1 year. While depressed, they also experience a number of somatic and cognitive symptoms.
- About 5% of children and adolescents have an episode of P-DD by the end of adolescence.
- The most common disorders accompanying P-DD are superimposed MDD, anxiety disorders, CD, and ADHD.
- The most common age at onset for P-DD is between 11 and 12 years, with an average episode length of between 2 and 5 years.
- Almost all young people eventually recover from their P-DD, but many will develop MDD.
- Children who recover from their P-DD differ from other children mainly on measures of psychosocial functioning.
- P-DD is a revised category in DSM-5 that combines the previous DSM-IV categories of Dysthymic Disorder and MDD—Chronic. This was done because of the lack of differences between youths with a dysthymic disorder and those with a chronic type of major depression.

# DISRUPTIVE MOOD DYSREGULATION DISORDER (DMDD)

The central feature of **disruptive mood dysregulation disorder (DMDD)** is chronic, severe persistent irritability. This severe irritability has two main clinical features. The first is frequent verbal or physical *temper outbursts* that usually occur in response to frustration and are totally out of proportion to the provocation or situation. These outbursts must occur frequently (three or four times a week) over 1 year in at least two of three settings (i.e., at home, at school, with peers), and must be age-inappropriate. The second feature of severe irritability is a chronic, persistently *irritable or angry mood* that is present most of the day, nearly every day, between the severe temper outbursts. This mood must have an onset prior to age 10 years; be characteristic of the child; be present most of the day, nearly every day; and be noticeable to others. A diagnosis of DMDD cannot coexist with ODD (in this case. a diagnosis of DMDD only would be made) or bipolar disorder (in this case. a diagnosis of BP would be made), but can co-occur with MDD, ADHD, CD, or substance-use disorder (APA, 2013).

DMDD is a new depressive disorder in DSM-5, and it is the one that we know the least about. In addition, its inclusion in DSM-5 as a depressive disorder has generated some controversy. In light of this, we briefly

consider the context in which DMDD was established as a diagnostic category and some of the issues that surrounded its development. DMDD was formulated in two contexts. The first included findings on severe irritability as a salient characteristic of mood, not just as a manifestation of MDD (Stringaris, 2011). For example, studies have found that irritability at age 3 predicts depression, ODD, and functional impairment in early childhood (Dougherty et al., 2013) and that irritability in adolescence predicts self-reports of depressive and anxiety disorders up to 20 years later (Stringaris et al., 2009). The second context was research on bipolar disorder (to be discussed later in this chapter) that identified children with severe mood dysregulation problems whose symptoms did not fit neatly into traditional definitions of bipolar disorder (Towbin et al., 2013). Importantly, the development of the diagnosis of DMDD was a direct response to concerns about increasing rates of bipolar disorder diagnoses in young children and the growing use of medications to treat these children. Thus, much of the initiative in creating the category of DMDD was to provide an alternative to diagnosing BP in young children too frequently (Youngstrom & Algorta, 2014).

As described by Youngstrom and Algorta (2014), the definition of DMDD changed several times during its development in DSM-5, first described as "severe mood dysregulation disorder," then as "temper dysregulation disorder," and lastly as a "disruptive mood regulation disorder." Significantly, it was also moved from the Disruptive, Impulse-Control, and Conduct Disorders section of DSM-5 to the Depressive Disorders section—not to the Bipolar Disorders section. Significant concerns have been expressed about using DMDD clinically as a new diagnosis, given the absence of data about its prevalence, course, and response to treatment. A central concern is the extent to which the criteria developed for DMDD can be used to reliably differentiate it from other mood and behavior disorders, particularly ODD, which as you may recall, also includes irritability, anger, and defiance as key features (Axelson et al., 2011). This concern appears to be justified, as field trials report overall reliabilities of the DSM-5 criteria for DMDD in clinical practice to be quite low (kappa = 0.25), and to vary widely across settings (Regier et al., 2013). Thus, it may prove difficult for clinicians to reliably distinguish DMDD from other mood and behavioral problems (Youngstrom & Algorta, 2014).

According to DSM-5, DMDD is common in clinic samples, occurs predominantly in males and in school-age children, has high comorbidity with anxiety, mood, and disruptive behavior disorders, and markedly disrupts the youth's family and peer relationships and school performance. Currently, we know there are a number of youngsters with severe irritability and mood dysregulation problems who do not meet criteria for bipolar disorder. What we do not know is whether DMDD as currently defined is distinct from other mood and conduct problems, its prevalence, characteristic course and outcomes, or how it can be best treated (Axelson et al., 2011; Towbin et al., 2013). Further research and clinical data are sorely needed to determine if the DMDD diagnosis will prove to be reliable, valid, and useful in clinical practice.

## Section Summary

### Disruptive Mood Dysregulation Disorder (DMDD)

- Disruptive mood dysregulation disorder (DMDD) is characterized by frequent and severe temper outbursts and chronic, persistently irritable or angry mood.
- DMDD is a new disorder in DSM-5, and it is the one we know the least about.
- The development of the DMDD category was a response to increasing rates of bipolar disorder (BP) diagnoses in young children; it was intended to provide an alternative to diagnosing BP in young children too frequently.
- Further research and clinical data are needed to determine whether the DMDD diagnosis will prove to be reliable, valid, and useful in clinical practice.

Now that you have some familiarity with MDD, P-DD, and DMDD, we next consider their associated characteristics and possible causes.

# ASSOCIATED CHARACTERISTICS OF DEPRESSIVE DISORDERS

Young people with depressive disorders experience deficits in intellectual performance and academic achievement and disturbances in self-perceptions, self-esteem, social problem solving, interpersonal behavior, and life stressors (Garber & Kaminsky, 2000). Since depression often occurs with anxiety and other disorders, we do not always know whether these associated deficits and disturbances are specific to depression or related to the presence of psychopathology in general. In addition, it is often difficult to know whether cognitive and psychosocial deficits are an outcome or a cause of depression.

## Intellectual and Academic Functioning

Certain depressive symptoms—difficulty concentrating, loss of interest, and slowness of thought and movement—are likely to have a harmful effect on a child's intellectual and academic functioning. However,

the overall intellectual potential of youths who are depressed is comparable to the potential of those who are not depressed. The association between severity of depression and children's overall intelligence is weak, suggesting that the effects of depression on cognitive functions may be selective. For example, depression may be associated with impairments while performing nonverbal tasks that require attention, coordination, speed, or recall of emotionally coded information, such as facial expressions (Guyer et al., 2011), but not necessarily on tasks that require verbal skills or overall intelligence (Wilkinson & Goodyer, 2006). Depression may also be associated with broad impairments in executive functions, for example, maintaining task goals in working memory (Snyder, 2013).

Youngsters with depression perform more poorly than others in school. They score lower on standard achievement tests, are rated by their teachers as achieving less academically, and have lower levels of grade attainment (Cole, 1990). Poor concentration and thinking ability, slowed movement or agitation, fatigue, insomnia, and somatic symptoms may lead to repeating a grade, being late or skipping school, failure to complete homework, and dissatisfaction with or refusal of school. Jenn, a 15-year-old girl with MDD, and her mother had this to say about school:

> "School is a big waste of time," says Jenn. "I don't want to be there. I don't have the energy or motivation for school. I just say I'm sick so I can stay at home in bed and sleep all day." Jenn's mother says, "We used to fight about school so much that eventually I'd let her stay home—just to avoid having another fight."

It is difficult to determine whether depression is a cause or an outcome of learning difficulties. Most likely it can be both. For example, learning difficulties in adolescents, particularly girls, has been found to lead to feelings of inadequacy as a student, which predict depressive symptoms (Kiuru et al., 2011). It is unclear whether depression has an enduring effect on school performance. Some studies find that school difficulties do not follow the child's recovery from depression, while others report that academic problems continue even after recovery (Kovacs & Goldston, 1991). In general, the association between depression and school difficulties is not as strong as the association between depression and social dysfunction (Lewinsohn, Gotlib, & Seeley, 1997).

## Cognitive Biases and Distortions

*"Good morning, Pooh Bear," said Eeyore gloomily. "If it is a good morning," he said. "Which I doubt."*

—A. A. Milne, *Winnie-the-Pooh* (1926)

**ELLIE**

### Life's Hardly Worth It

"… like everything's worthless, like it's just not worth it to even be. … It's—it seems like it's a silly thing to even go through life and exist. And from one day to the next you're always wondering if you're going to make it to the next day if it's—if you can stand it, if it's worth trying to get to tomorrow. … It's just—just, I feel like—I feel mostly like I'm worthless, like there's something wrong with me. It's really not a pleasant feeling to know that you're a total failure, a complete nothing, and I get the feeling that I never do nothing right or worthwhile or anything."

Adapted from McKnew, Cytryn, & Yahraes, 1983.

Many children with depression experience biases, deficits, and distortions in their thinking (Lakdawalla, Hankin, & Mermelstein, 2007). These children commonly notice depression-relevant cues such as sad facial expressions more often than positive cues such as happy facial expressions (Ehrmantrout et al., 2011; Hankin et al., 2010). Given the importance of accurately reading emotional cues for successful social relationships, these selective attentional biases can contribute to adverse relationships with family members and peers.

Some cognitive disturbances, such as Ellie's painful feelings of worthlessness, are part of the diagnosis of depression. Negative beliefs ("I never do nothing right.") and attributions of failure ("I'm a total failure.") are not part of the diagnosis but typically accompany the disorder. Negative thoughts that are self-critical and automatic, such as "I'm a real loser," "I'm ugly," or "I'm gonna fail," are common. Unfortunately, these thoughts can't simply be swept aside by suggesting to a depressed youth that she or he "look at the bright side."

Depressed children often devalue their own performance by not acknowledging their accomplishments. They dismiss praise when it is given and frequently make inaccurate interpretations of their experiences (Fichman, Koestner, & Zuroff, 1996). To focus narrowly and passively on negative events for long periods is referred to as a **depressive ruminative style** (Nolen-Hoeksema, Girgus, & Seligman, 1992). These youngsters view themselves as ineffective in most areas of their lives, and they make self-directed disparaging comments when faced with further failure or rejection (e.g., "It must be my fault."). They misread situations, feel slighted by harmless remarks, and are easily frustrated—small setbacks are seen as major catastrophes. Negative thinking and faulty conclusions are generalized across situations, so the depressed youth sees no hope of gaining any pleasure or satisfaction.

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Depression in young people is associated with negative thoughts and feelings of worthlessness.

## Negative Self-Esteem

*Eeyore, the old grey Donkey, stood by the side of the stream and looked at himself in the water. "Pathetic," he said. "That's what it is. Pathetic."*

—A. A. Milne, *Winnie-the-Pooh* (1926)

**FARAH**

### Never Good Enough

Fifteen-year-old Farah's mother says that Farah is a "model daughter," who is near the top of her class, active in school activities, and extremely popular. Her mother is concerned about "how hard Farah is on herself, thinking that she has to be perfect." If Farah doesn't get the highest grade on a test, she won't allow herself to see her friends for a week, and spends most of the time in her room studying. Farah acknowledges that she sets very high standards for herself and if she fails to meet these standards becomes extremely self-critical and self-punitive. She has even slapped herself in the face after what she saw as academic "failure" (getting an A minus rather than an A). Farah's accomplishments bring her little satisfaction, and any perceived failure leads to immediate self-condemnation. Farah's overall self-worth is low and her sense of self, which is based on competency in academic achievement, is highly vulnerable. (Based on authors' case material)

It is not unusual for young people with depression to think that no one can help them out of their misery. Many report hopelessness or negative expectations about the future that are related to diminished self-esteem and to suicidal ideations and attempts (Marciano & Kazdin, 1994). Since feelings of hopelessness dominate their lives, they experience a vicious downward cycle in which self-defeating negative thoughts become pervasive and impair performance at school and home. As performance deteriorates they perceive more failure, and receive—and even seek—further negative feedback. These outcomes maintain their low opinions of themselves and their view of an inability to change, and these lead to further impairments in functioning.

The pessimistic outlook of young people with depression also places them at greater risk for depressive symptoms, especially in response to stressful life events. Since their pessimism may continue after remission of depressive symptoms, they remain at risk for future depressive episodes. We will return to the role of cognitive disturbances in depression later, in the section on cognitive theories.

Almost all young people with depression experience negative self-esteem. In fact, low self-esteem is the symptom that seems most specifically related to depression in adolescents (Lewinsohn et al., 1997). Self-esteem in children with depression is also highly reactive to daily life events, and such daily fluctuations in self-esteem appear to be related to depression following exposure to major life stresses (Roberts & Gotlib, 1997). Thus, both low self-esteem and unstable self-esteem seem to play an important role in depression.

Since physical appearance and approval from peers are especially important as sources of self-esteem for most adolescents, perceived incompetence in these areas may heighten the risk for depression. The fact that self-esteem problems in adolescent girls are often related to a negative body image may partly contribute to their higher risk for depression (Hankin & Abramson, 2001).

An interesting developmental model of self-esteem and depression hypothesizes that young people seek and receive feedback from others about their competence or incompetence in several domains: academics, social relations, sports, conduct, and physical appearance

(Jordan & Cole, 1996). Self-views are constructed from this feedback, and the outcome may be a varied and positive self-view leading to optimism, energy, and enthusiasm. Or it may be a narrow and negative self-view leading to pessimism, a sense of helplessness, and possibly, depression (Seroczynski, Cole, & Maxwell, 1997). Children whose self-views are negative and narrowly focused in one domain—for example, in academics—may show instability in their self-esteem because they lack alternative compensatory areas of functioning, such as sports or social relations. This may make them vulnerable to developing depression when faced with stress in their primary domain.

## Social and Peer Problems

Young people who are depressed experience significant disruptions in their relationships. They have few friends or close relationships, feel lonely and isolated, feel that others do not like them (which, unfortunately, often becomes a reality), and display extensive impairments in their social skills (Rudolph, Flynn, & Abaied, 2008). The low social status of youngsters with depression has been found to emerge via two pathways (Agoston & Rudolph, 2013). In the first pathway, depressive symptoms promote socially helpless behavior and subsequent neglect by peers. In the second pathway, depressive symptoms promote aggressive behavior and subsequent rejection by peers. Chronic peer-related loneliness during childhood has also been found to predict depressive symptoms in early adolescence (Qualter et al., 2010). In addition, children with depression who report poor friendships at the time of referral have a reduced likelihood of recovery from depression (Goodyer et al., 1997). Even when children recover from their depression, they continue to experience some social impairment.

Social withdrawal is common in youngsters with depression. They often spend significant amounts of time alone, show little interest in seeing friends, and engage in few activities. Their social withdrawal may reflect an inability to maintain social interactions—possibly related to negative, irritable, and aggressive behavior toward others—and deficits in initiating conversations or making friends (Rockhill et al., 2007). These factors can seriously interfere with social development, depriving youngsters with depression of the social exchanges that lead to healthy interpersonal relationships.

Youngsters who are depressed use ineffective styles of coping in social situations. For example, they use less active and problem-focused coping and more passive, avoidant, ruminative, or emotion-focused coping (Hammen et al., 2014). A strong risk factor for the onset of depression in adolescent females is **co-rumination**, a negative form of self-disclosure and discussion between peers focused narrowly on problems or emotions to the exclusion of other activities or dialogue (Stone et al., 2011). Co-rumination seems to be one mechanism underlying adolescent females' heightened risk for depression. Ironically, co-rumination between peers is associated with higher ratings of friendship quality and closeness, which in turn, have been found to predict increases in co-rumination. Thus, what appear to be socially rewarding and supportive relationships with peers not only fail to protect female teens from distress, but also may increase their risk for depression when based on maladaptive styles of interaction (Stone et al., 2011).

Depressed teens may also make poor choices in dealing with social problems, such as turning to alcohol or drugs in response to a break-up with a boyfriend or girlfriend. In the words of Page, age 17:

> I was so unhappy that I didn't care about myself—even about being safe. I was out drinking a lot, doing a lot of pot. Sometimes I would just black out and not know what was happening. One night I think a bunch of guys had sex with me when I passed out, I don't know. I never remembered anything, it was all hearsay the next day. I made some really bad boyfriend choices. I would date guys who reinforced my view of myself as ugly, stupid, and uncool. I dropped my preppy boyfriend and started dating a 20-year-old guy who was living in his own apartment and playing in a band. He had tattoos on his arms and stomach. I would date guys just so I could get a ride, even though I didn't like them. I would pick boyfriends who were depressed or ones that my parents really didn't like. (From "I Did Not Want to Live," by Sabrina Solin Weill, 1995. *Seventeen*, April 1, 1995, pp. 154–156, 176.)

Interestingly, the basic understanding required for appropriate social relations appears to be relatively intact in youngsters with depression. They are generally capable of providing cognitive solutions to interpersonal problems (Kovacs & Goldston, 1991). However, as with Page, their deficits in social problem solving and behavior in real-life situations, particularly when they are under stress, are in sharp contrast to their social understanding (Calhoun et al., 2012). Adolescents with depression and poor social problem-solving skills are likely to show increases in the severity of their depression over time (Becker-Weidman et al., 2010).

## Family Problems

Youngsters with depression experience less supportive and more conflictual relationships with their mothers, fathers, and siblings than do children who do not have depression. They report feeling socially isolated from


"It's my youth, and I don't have to enjoy it if I don't want to."

Barbara Smaller/Cartoon Bank.com

their families and prefer to be alone rather than with them. In family situations, the child's social isolation may not be a social skill deficit, but rather a reflection of the child's desire to avoid conflict. Family relationship difficulties have been found to persist even when children are no longer clinically depressed (Sheeber et al., 2007).

During interactions, these youngsters may be quite negative toward their parents, and their parents in turn may respond in a negative, dismissing, or harsh manner. When repeated over time, these interactions may adversely affect family relationships. Children with depression who are irritable, unresponsive, and unaffectionate provide little positive reinforcement for their parents, and they frustrate their parents' desire for satisfaction in the parenting role (Kovacs, 1997).

## Depression and Suicide

### CARLA

### "It Became Too Much"

Carla, age 12, was admitted to the intensive care unit unconscious and unstable after ingesting eight of her mother's 50-mg Elavil tablets, an unknown quantity of antidepressants, and approximately 20 tablets of Tylenol 3. This suicide attempt, her first, came after arguing with her father over chores and restrictions imposed because her grades were so bad. Carla said she went to the medicine cabinet and ingested everything she could find because "it became too much" and she "did not want to live." For the previous month, she had displayed a noticeable change of mood, behaving with more instability and depression, feeling worthless and hopeless. During this period she had lost her appetite and had dropped two dress sizes. She had increasingly isolated herself, staying alone in her room. Her school performance, for which her father had restricted her, had declined from B's the previous term to D's.

From Adolescent Suicide: Assessment and Intervention by A. L. Berman and D. A. Jobes, 1991, p. 144.

Carla's case illustrates the profound feelings of hopelessness, helplessness, and despair that often lead a youngster with depression to attempt suicide. Most youngsters with depression report suicidal thinking, and as many as one-third who think about killing themselves actually attempt it (Goldston, Daniel, & Arnold, 2006). Drug overdose and wrist cutting are among the most common methods for adolescents who attempt suicide. In one long-term follow-up study, adolescents with MDD had a fivefold increased risk of a first suicide attempt as compared with controls without MDD, and nearly 8% of them committed suicide within 15 years of their first episode of MDD (Weissman et al., 1999b). For adolescents who complete suicide, the most common methods are firearms (45%), suffocation (40%), and poisoning (8%) (CDC, 2013).

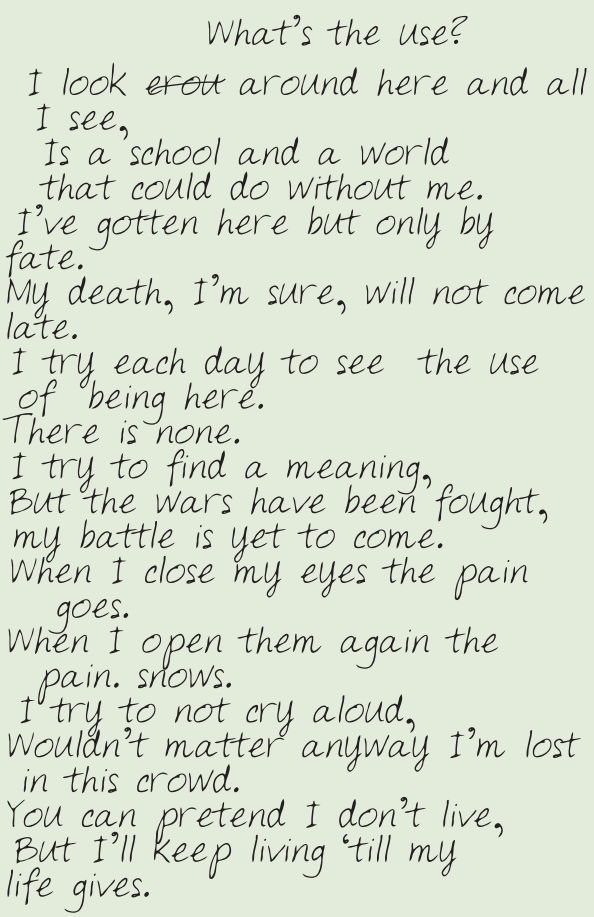
The link between depression, suicidal behavior, and completed suicide is undeniable, strong, and sobering (Dervic, Brent, & Oquendo, 2008). Suicidal ideation (e.g., thinking about killing oneself) is common across many different types of psychological disorders, but actual suicide attempts are much more common during depression (Nock et al., 2013) (see A Closer Look 10.1). In one 7-to 9-year follow-up of youngsters with psychiatric disorders, 84% of all suicide attempts were found to occur because of depressive disorders (Shaffer et al., 1996).

About 60% of youngsters who are clinically depressed report having thoughts about suicide, and 30% attempt suicide by 17 years of age, with most attempts coming within the first year after the onset of suicidal thoughts. Unfortunately, about half of them eventually make further attempts (AACAP, 2001). The suicide attempts of youngsters with depression almost never occur during times when they are symptom-free—90% or more have depressive features at the time of their suicidal episode. Finally, among youngsters who kill themselves, the odds of having major depression are 27 times higher than among controls (Brent et al., 1993; Shaffer et al., 1996).

Although rates of suicidal behavior vary across countries, the two strongest risk factors for suicidal behavior are consistent worldwide—having a mood disorder and

## A CLOSER **LOOK 10.1**

### Depressive Disorder Is Associated with Suicide Thoughts and Suicide Attempts



What's the use?

I look ~~erou~~ around here and all
I see,
Is a school and a world
that could do without me.
I've gotten here but only by
fate.
My death, I'm sure, will not come
late.
I try each day to see the use
of being here.
There is none.
I try to find a meaning,
But the wars have been fought,
my battle is yet to come.
When I close my eyes the pain
goes.
When I open them again the
pain. shows.
I try to not cry aloud,
Wouldn't matter anyway I'm lost
in this crowd.
You can pretend I don't live,
But I'll keep living 'till my
life gives.

Teri's note

**Teri: What's the Use?**

Teri, age 15, had been depressed since her father died when she was 11. According to her mother, over the past 14 months her behavior had gone from moody to sullen. She had disobeyed restrictions imposed as punishments and had run away from home on several occasions. She labeled herself as "stupid," spoke and wrote often of death and suicide (see accompanying note). On three occasions she had cut her wrists, albeit only superficially. Her school performance had declined and she spoke now of hating school. Her peer associations were almost exclusively with other alienated teens, described by her as "punks and other anarchists."

Source: Adolescent Suicide: Assessment and Intervention by A. L. Berman and D. A. Jobes, 1991, p. 144.

being a young female (Cha & Nock, 2014; Nock et al., 2008). In general, young females with depression show more suicidal ideation and attempt suicide more often than young males (Nock et al., 2013). The risk factors for nonfatal suicide attempts are similar for males and females (Thompson & Light, 2010). However, since girls typically do not use guns, they are usually less successful in completing suicide than boys (Goldston et al., 2006). Ages 13 and 14 are peak periods for a first suicide attempt by youngsters with depression. Suicide prior to puberty is rare, most likely because depression and substance abuse before puberty are also rare. In adolescents with depression, suicide attempts double during the teen years but show an abrupt decline after age 17 or 18. It is possible that as young people mature, they are better able to tolerate their negative mood states and acquire more resources for coping, thus making it less likely that they will attempt suicide during periods of sadness (Borowsky, Ireland, & Resnick, 2001).

In light of the strong connection between symptoms of depression and suicidal ideation and behavior, a primary strategy for reducing suicide in young people is to increase the availability of effective treatments for depression (Brown et al., 2007). We discuss these treatments later in this chapter. Prevention and treatment programs for suicidal behavior generally focus on family involvement and support, and they emphasize intervening early after the suicidal crisis (Brent et al., 2013; Pineda & Dadds, 2013). Since racial and ethnic groups are known to differ in rates of suicidal behaviors and the circumstances under which they occur (e.g., precipitants, risk and protective factors, and patterns of seeking help), it is also important that suicide prevention and treatment programs are sensitive to these cultural differences (Goldston et al., 2008).

Our discussion of suicidal behavior in this chapter has focused primarily on its relationship with depressive disorders. However, it is important to recognize that self-injurious thoughts and behaviors in young people is a significant topic in its own right, and one that is just beginning to receive the attention it deserves (Cha & Nock, 2014). Self-injurious thoughts and behaviors range from nonsuicidal self-injury such as self-cutting, to suicidal ideation, suicide attempts, and completed suicide. Suicide is especially worrisome, as it is the second leading cause of death among adolescents and young adults in the United States, resulting in about 4,600 deaths per year (CDC, 2013). DSM-5 includes two newly proposed disorders in its section on conditions for further study: Suicidal Behavior Disorder describes individuals who have made a suicide attempt within the past 24 months, and Nonsuicidal Self Injury describes individuals who engage in intentional self-inflicted damage to the surface

of the body (e.g., cutting, burning, stabbing, excessive rubbing) without suicidal intent. These proposed diagnoses and other efforts will hopefully bring further attention and understanding to self-injurious behaviors in young people so we can learn how to best predict and prevent these behaviors (Cha & Nock, 2014).

## Section Summary

### Associated Characteristics of Depressive Disorders

- Youngsters with depression have normal intelligence, although certain symptoms such as difficulty concentrating, loss of interest, and slowness of thought may negatively affect intellectual functioning.
- They perform more poorly than others in school, score lower on standard achievement tests, and have lower levels of grade attainment.
- They often experience deficits and distortions in their thinking, including negative beliefs, attributions of failure, and self-critical automatic negative thoughts.
- Almost all youngsters with depression experience low or unstable self-esteem.
- Youngsters with depression have few friends and close relationships, feel lonely and isolated, and feel that others do not like them.
- They experience poor relationships and conflict with their parents and siblings, who in turn may respond in a negative, dismissing, or harsh manner.
- Most youngsters with depression report suicidal thinking, and about 30% who think about killing themselves actually attempt it.

# THEORIES OF DEPRESSION

Many theories have been proposed to explain the onset and course of depression. Until recently, however, most were developed to explain depression in adults, then directly applied to children with minimal regard for developmental differences (Garber & Horowitz, 2002). In the sections that follow, we consider several of these theories. Keep in mind, however, that depression is likely a final, common pathway for interacting influences that predispose a child to develop the disorder (Hammen et al., 2014). No one theory can explain all forms of depressive disorder and differences in symptoms and severity within the same disorder. An overview of the primary theories of depression is presented in Table 10.2.

**TABLE 10.2 | Overview of Theories of Depression**

| | |
|---|---|
| **Psychodynamic** | Actual or symbolic loss of love object (e.g., caregiver) that is loved ambivalently; anger toward love object turned inward; excessive severity of the superego; loss of self-esteem |
| **Attachment** | Insecure early attachments; distorted internal working models of self and others |
| **Behavioral** | Lack or loss of reinforcement or quality of reinforcement; deficits in skills needed to obtain reinforcement |
| **Cognitive** | Depressive mindset; distorted or maladaptive cognitive structures, processes, and products; negative view of self, world, and future; poor problem-solving ability; hopelessness |
| **Self-Control** | Problems in organizing behavior toward long-term goals; deficits in self-monitoring, self-evaluation, and self-reinforcement |
| **Interpersonal** | Impaired interpersonal functioning related to grief over loss; role dispute and conflict; role transition; interpersonal deficit; single parenting; social withdrawal; interaction between mood and interpersonal events |
| **Socio-environmental** | Stressful life circumstances and daily hassles as vulnerability factors; social support, coping, and appraisal as protective factors |
| **Neurobiological** | Neurochemical and receptor abnormalities; neurophysiological abnormalities; neuroendocrine abnormalities; genetic variants; abnormalities in brain structure and function; effects of early experience on the developing brain |

Based on A Developmental Cognitive Model of Unipolar Major Depression, by D. J. A. Dozois, unpublished manuscript.

## Psychodynamic

Early psychodynamic theories viewed depression as the conversion of aggressive instinct into depressive affect. Depression is presumed to result from the loss of a love object (e.g., mother). This loss can be actual, as in the case of the death of a parent, or symbolic, as a result of emotional deprivation, rejection, or inadequate parenting. The individual's subsequent rage toward the love object is then turned against the self. Since children

and adolescents were believed to have inadequate development of the superego or conscience, the hostility directed against internalized love objects that have disappointed or abandoned them does not produce guilt, so they do not become depressed (Bemporad, 1994; Poznanski, 1979). However, more recent studies have found that high levels of maladaptive guilt and shame are related to the onset of depression in children as young as 3 to 5 years of age (Luby, Belden et al., 2009). Furthermore, the fact that depression does occur in many youngsters who do not experience loss or rejection—and doesn't occur in many children who do—casts doubt on the psychodynamic model. Contrary to this theory, many children do experience clinical depression.

## Attachment

Attachment theory focuses on parental separation and disruption of an attachment bond as predisposing factors for depression. John Bowlby hypothesized that a child confronted with unresponsive and emotionally unavailable caregiving goes through a typical sequence involving protest, despair, and detachment (Bowlby, 1961). A parent's consistent failure to meet the child's needs is associated with the development of an insecure attachment, a view of the self as unworthy and unloved, and a view of others as threatening or undependable. These factors may place the child at risk for later depression, particularly in the context of stressful interpersonal relationships (Rudolph, Hammen, & Burge, 1997). Attachment relationships also serve to regulate biological and behavioral systems related to emotion. For example, a secure attachment may help to reduce distress, whereas an insecure attachment may lead to difficulties in regulating emotion, which in turn may become a risk factor for later depression. In support of this theory, children with insecure attachments are more likely than children with secure attachments to display symptoms of depression (e.g., Toth & Cicchetti, 1996). In addition, children and adolescents with depression are more likely to experience disturbances in attachment than are children without depression (Stein et al., 2000). In one study, only 8% of adolescents with depression were securely attached (vs. 52% of controls), and 40% of them had an insecure attachment that was unresolved with regard to loss or abuse (Ivarsson et al., 2010).

## Behavioral

Behavioral views emphasize the importance of learning, environmental consequences, and skills and deficits during the onset and maintenance of depression. Depression is related to *a lack of response-contingent positive reinforcement* (Lewinsohn, 1974). This lack of positive reinforcement may occur for three reasons. First, a youngster may be unable to experience available reinforcement, often because of interfering anxiety. Second, changes in the environment, such as the loss of a significant person in the child's life, may result in a lack of availability of rewards. Finally, a youngster may lack the skills needed to have rewarding and satisfying social relationships.

Children may also receive sympathy for their sadness, which produces the desired attention and concern. However, this sympathy is usually short-lived because even people who care about the youngster begin to avoid him or her. This reduction in attention may then lead to withdrawal, impairment in functioning, and heightened feelings of depression. Few studies have tested specific behavioral hypotheses with children, and this model seems incomplete in the light of what is known about other factors that may lead to a vulnerability to depression. Nevertheless, the behavioral model highlights the importance of learning processes in the emergence, expression, and outcome of depression in young people.

## Cognitive

Cognitive theories focus on the relation between negative thinking and mood (Abela & Hankin, 2008a). The underlying assumptions are that how young people view themselves and their world will influence their mood and behavior and that cognitive vulnerabilities interact with negative events to increase depressive symptoms. A variety of negative cognitions, attributions, misperceptions, and deficiencies in cognitive problem-solving skills are related to depression in young people (Lakdawalla et al., 2007). Cognitive theories emphasize **depressogenic cognitions**, which are the negative perceptual and attributional styles and beliefs associated with depressive symptoms.

For example, **hopelessness theory** proposes that depression-prone individuals tend to make internal, stable, and global attributions to explain the causes of negative events. In other words, when something bad happens, they think that they are responsible (internal attribution), that the reason they are to blame will not change over time (stable attribution), and that the reason that something bad happened applies to most things they do and in most situations (global attribution) (Abramson, Seligman, & Teasdale, 1978). In contrast, they attribute positive events to something outside themselves (external), which is not likely to happen again (unstable), and is seen as unique to this event (specific). A *negative*

*attributional style* results in the individual's taking personal blame for negative events in his or her life and leads to helplessness and avoidance of these events in the future. Helplessness may in turn lead to hopelessness about the future, which promotes further depression (Abramson, Metalsky, & Alloy, 1989).

The cognitive model developed by Aaron Beck (1967) proposes that depressed individuals make negative interpretations about life events because they use biased and negative beliefs as interpretive filters for understanding these events. Depressed individuals show cognitive problems in three areas.

First, they display *information-processing biases*, or errors in their thinking in specific situations, called *negative automatic thoughts*. These often include thoughts of physical and social threat, personal failure, and hostility (Schniering & Rapee, 2004). They may selectively attend to negative information, assume blame for negative events, maximize and exaggerate negative events, and minimize positive events. They also assign negative labels to events and then react emotionally to the label rather than to the event. For example:

- EVENT: Child didn't receive an invitation to Ashley's party.
- LABEL: "I didn't receive an invitation because Ashley doesn't like me. *Nobody likes me*."
- EMOTIONAL REACTION: Unhappiness and depression.

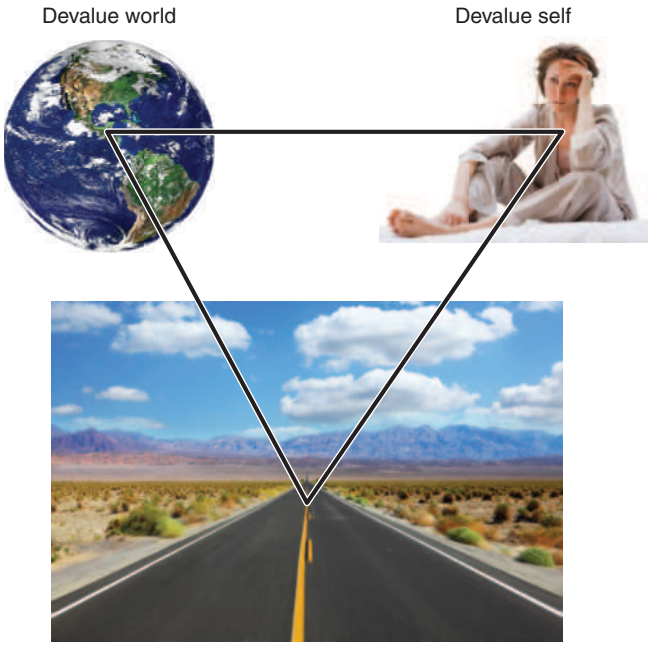
Second, depression is believed to be associated with a negative outlook in the following three areas, referred to as the **negative cognitive triad** (see ● Figure 10.2):

- Negative views about *oneself* (e.g., "I'm no good," "I'm boring")
- Negative views about the *world* (day-to-day experiences) (e.g., "They're no good," "It's too hard")
- Negative views about the *future* (e.g., "It's always going to be this bad," "I'll never graduate")

These negative views become increasingly more stable with age, maintain feelings of helplessness, undermine the youngster's mood and energy level, and are related to the child's severity of depression (LaGrange et al., 2008).

Third, depressed youngsters have **negative cognitive schemata**, which are stable structures in memory that guide information processing, including self-critical beliefs and attitudes. These schemata are rigid and resistant to change even in the face of contradictory evidence and may heighten the youngster's sensitivity to depression, especially when activated by stress.

Applying cognitive theories to depression in young people raises questions about the cognitive capacities




● **FIGURE 10.2** | The Negative Cognitive Triad: Depression is related to a devaluation of self, the world, and the future.

*Photo* credits: Tom Merton/OJO Images/Getty Images; ©worker/Shutterstock.com; ©kavram/Shutterstock.com

of children at various stages of development and the development and stability of cognitive structures that may be involved in their depressive thinking (Abela & Hankin, 2008a). A well-developed sense of self and a time perspective for the future are needed to experience depression; these cognitive processes are still developing in children. In addition, many of the cognitive errors and distortions discussed so far, such as illogical thinking or faulty attributions, are normal ways of thinking in young children!

Although higher rates of negative thinking are found in youngsters with depression, there are still many unanswered questions about the relation between cognition and depression (Lakdawalla et al., 2007). More information is needed about how negative cognitions develop. Are the negative cognitions that accompany depression the result of parental rejection and negative parenting practices? Is there a relation between maternal and child cognitions, as suggested by the relationship between mothers' and children's negative thinking (Stark, Schmidt, & Joiner, 1996)? One study found an association between a maternal depressogenic cognitive style during pregnancy and offspring cognitive style 18 years later (Pearson et al., 2013). How and when does a cognitive vulnerability for depression interact with stress to result in depression (Cole et al.,

2008)? At this time, there is support for a cognitive vulnerability–stress interaction in adolescents, and possibly for children as young as 7 years of age (Hayden et al., 2013; Lakdawalla et al., 2007). Longitudinal studies are needed to answer these and other questions concerning the role of cognition in the development of depression in young people.

## Other Theories

*Self-control theories* view youngsters with depression as having difficulty organizing their behavior in relation to long-term goals and as displaying deficits in self-monitoring, self-evaluation, and self-reinforcement. As a result, they selectively attend to negative events and to the immediate consequences of their behavior. These youngsters set excessively high standards for performance, make negative causal attributions, administer insufficient self-rewards, and use excessive self-punishment. Research suggests that children with depression display a number of these deficits (Rehm & Sharp, 1996).

*Interpersonal models* view disruptions in interpersonal relationships, especially with family and peers, as the basis for the onset and maintenance of depression (Hammen, 1999). Depressive symptoms in adolescence are associated with increases in the negative quality and decreases in the positive quality of relationships over time (Oppenheimer & Hankin, 2011). The behaviors of a depressed youngster are unpleasant to others, leading family members and others to become annoyed and frustrated. As the youngster becomes more aware of how others are reacting, he or she feels even more needy, and then unthinkingly and annoyingly seeks excessive reassurance, which in turn leads to further interpersonal rejection (Joiner, 1999). Interpersonal models also propose that the child's depression may serve a function in the family—for example, to reduce conflict between parents.

*Socioenvironmental models* emphasize the relationship between stressful life events and depression. Adolescents with depression experience significantly more psychosocial adversity than controls or adolescents with other psychiatric disorders (Ivarsson et al., 2010). Some life situations related to the onset of depression are being social disadvantaged, having an unemployed parent, having a single parent, being part of a large family, experiencing a personal loss, being abused, and having poor social support. Stressful life events may be linked to depression in several ways. First, depression can be a direct reaction to the occurrence of stressful life events, such as the loss of a parent. Second, the impact of stress may be moderated by individual risk factors, such as genetic risk. This is referred to as the **diathesis–stress model of depression** because the occurrence of depression depends on the interaction between the youngster's personal vulnerability (diathesis) and life stress. Third, negative environmental events may be internalized as negative cognitive styles (e.g., rumination), which then predispose the child to depression (Abela & Hankin, 2011). Finally, depression may result in behaviors and impairments in functioning that generate stressful life circumstances that in turn lead to depressive reactions (Hammen, Brennan, & Le Brocque, 2011). The generation of stress following depression may also be heightened by early developmental risk factors such as child abuse and neglect (Harkness, Lumley, & Truss, 2008).

*Neurobiological models* of depression in young people focus on genetic vulnerabilities and neurobiological processes, including the effects of early experiences such as stress, child maltreatment, or maternal depression on the developing brain. Several neurobiological abnormalities have been identified, although findings are less consistent for children than for adults (NIMH, 2003). We consider possible biological and other factors in the next section, on causes.

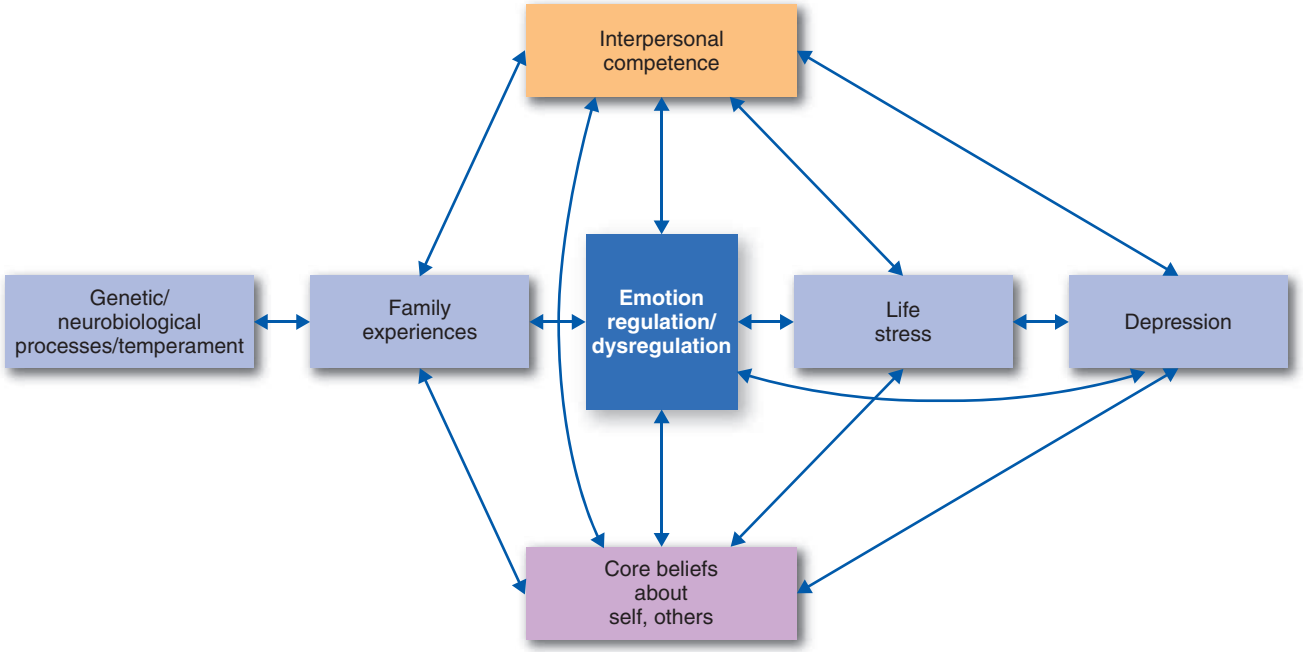
## Section Summary

### Theories of Depression

- Psychodynamic theories presume that depression results from the actual or symbolic loss of a love object and view depression as the conversion of aggressive instinct into depressive affect.
- Attachment theories focus on insecure attachment, a view of the self as unworthy and unloved, and a view of others as threatening or undependable as risk factors for later depression, particularly in the context of stressful interpersonal relationships.
- Behavioral views emphasize the importance of learning, environmental consequences (particularly a lack of response-contingent reinforcement), and skills deficits during the onset and maintenance of depression.
- Cognitive theories of depression focus on the relation between negative thinking and mood, with the underlying assumption that how young people view themselves and their world will influence their mood and behavior.
- Other theories of depression have emphasized the role of deficits in self-control, interpersonal disturbances, stressful life events, and genetic and neurobiological processes.

# CAUSES OF DEPRESSION

In light of the many vulnerability, risk, and protective factors that have been implicated, an integrative framework is necessary to account for depression in young






● **FIGURE 10.3** | A developmental framework for depression in young people.

people and for its nonoccurrence in the presence of risk (Hammen et al., 2014; Miller, 2007). The framework presented in ● Figure 10.3 highlights possible causes of depression in young people and the interplay among genetic, neurobiological, family, cognitive, emotional, interpersonal, and environmental factors. Given these many interacting influences, multiple pathways to depression are likely (Eaves, Silberg, & Erkanli, 2003).

Within this framework, genetic risk influences neurobiological processes and is reflected in an early temperament characterized by oversensitivity to negative stimuli, high negative emotionality, and a disposition to feeling negative affect. These early dispositions increase exposure to and are shaped by negative experiences within the family and continue to exert influence throughout development. Core beliefs about self and others develop as a result of experiences within the family. Parenting that is insensitive, disengaged, or rejecting may lead to an insecure attachment and a view of the self as incompetent, other people as threatening or unresponsive, and relationships as negative and unpredictable. Negative family experiences may also create an inconsistent emotional and social environment, which makes it difficult for the child to effectively regulate emotions and interpersonal behavior and to cope with stress (Compas et al., 2001).

Cognitive, emotional, and interpersonal problems may lead directly to depression, or they may elicit conflict, rejection by others, and social isolation, which will eventually lead to depression. In other instances, negative beliefs, poor social relationships, and difficulty in regulating emotions may create a *vulnerability* to develop depression when confronted with life stresses. In any of these scenarios, the child's depression may then interfere with future development by further disrupting interpersonal relationships, damaging existing competencies, producing further difficulties in regulating emotions, creating additional stress, and confirming the child's already negative views about self and others (Hammen et al., 2014).

In the sections that follow, we examine several of these possible interacting causal influences for depression.

## Genetic and Family Risk

Twin and other genetic studies suggest a moderate genetic influence on depression in children and adolescents, with heritability estimates ranging from 30% to 45% across studies for males and females (Franic et al., 2010; Lemery & Doelger, 2005). There is consistent evidence that MDD in young people runs in families across generations (Oquendo et al., 2013). In fact, the single best predictor of a child's risk for MDD is a high family incidence for this disorder (Weissman et al., 2005). Children with a parent who suffered from depression as a child are 14 times more likely than controls to become depressed themselves *before the age of 13* (Weissman et al., 1988).

Children of parents with depression have about 3 times the risk of having depression as compared

with children of parents with no psychiatric disorders (Weissman et al., 2006). The child's risk for depression is even higher when both parents have a mood disorder. Children of depressed parents also have an earlier age at onset for their depression (by about 3 years of age) and are more likely to show an onset before puberty than children of nondepressed parents (Weissman et al., 1997). This is a significant factor because a family history of depression is most likely associated with recurrence of depression and a continuation of depression into adulthood for children with an onset of depression before puberty (Wickramaratne, Greenwald, & Weissman, 2000). In addition to depression and other mood disorders, children of depressed parents also display a variety of other emotional and behavioral disturbances, including anxiety, conduct problems, and substance-use disorders (Batten et al., 2012; Oquendo et al., 2013).

The lifetime prevalence of depression in mothers of children with depression is also high, about 50% to 75% (Kovacs, 1997). A family history of depression is also greater in first-degree relatives of children with depression than in children without depression (Wickramaratne et al., 2000). Although depression in young people is a family disorder, the extent to which transmission in families is genetic, psychosocial, or, most likely, both is not yet known. Causal influences may also differ with development, with support for a greater role for environmental influences for depression during childhood in contrast to a greater genetic influence during adolescence (Scourfield et al., 2003).

Studies into possible genetic markers for early-onset depressive disorders have implicated regions on several chromosomes. However, findings generally suggest that no specific region makes a large contribution to the risk of MDD and that multiple regions are involved (Holmans et al., 2007). Studies of specific genes have focused primarily on those involved in the synthesis, release, and reuptake of the neurotransmitter serotonin and to a lesser extent on other genes, such as brain-derived neurotrophic factor (BDNF), that have been implicated in brain plasticity and response to stress (Chen, Li, & McGue, 2013; Levinson, 2006).

In general, family and twin studies and specific gene studies suggest that a vulnerability to negative affect may be inherited and that certain environmental stressors may be required for these vulnerabilities to result in depression (Eley & Stevenson, 2000; Rice, Harold, & Thapar, 2003). Support for gene–environment (G×E) interactions comes from several studies. One 3-year longitudinal study found that the effects of family conflict on depressive symptoms were greater for children and adolescents at genetic risk for depression (Rice et al., 2006). A second study found that individuals with variants in the serotonin transporter gene displayed more depressive symptoms, diagnosable depression, and suicidality in relation to stressful life events than those who did not (Caspi et al., 2003). Research has also found that youths with variants in the serotonin transporter gene who experienced more stressors as compared with the typical level displayed increases in depressive but not anxious symptoms over time, suggesting a G×E interaction specific to depression (Hankin et al., 2011). Another study found a higher risk for depression in children who were maltreated, but only in children with variants in both the BDNF and serotonin transporter genes. Importantly, social support was also found to ameliorate the child's genetic risk for depression (Kaufman et al., 2006). Finally, other genes (such as COMT) have been found to reduce the risk for depressive symptoms in children exposed to severe psychosocial deprivation as a result of being raised in an institution from a young age (Drury et al., 2010).

The findings that specific genes may increase or decrease sensitivity to stress through their impact on the brain's emotional and arousal systems and, by doing


*"Son, it's important to remember that it's O.K. to be depressed."*

Robert Weber The New Yorker Collection/The Cartoon Bank.com

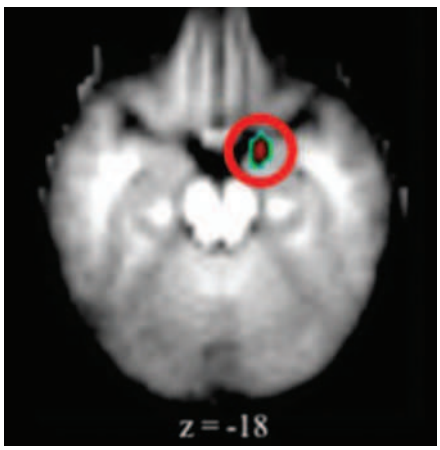
so, heighten or reduce the child's risk for depression are fascinating (Bradley et al., 2008; Gatt et al., 2010). However, it is important to note that there have been few studies with children and that results have been inconsistent (e.g., Karg et al., 2011; Risch et al., 2009). Thus, findings in this area, particularly those for the serotonin transporter gene (Culverhouse et al., 2013), must be viewed cautiously until they can be confirmed in studies that consider how multiple genes interact with multiple sources of environmental adversity in youngsters with and without depression (Fergusson et al., 2011). Findings from some studies in animals have also suggested that epigenetic processes known to cause stable and lasting changes in gene function may be involved in depression, although further studies will be needed to extend these findings to human depression (Mahgoub & Monteggia, 2013).

## Neurobiological Influences

Although we cannot point to one part of the brain that causes a young person to become depressed, abnormalities in the structure and function of several brain regions have been implicated. Most studies of young people with depression have focused on neural systems that regulate emotional functions such as neuroendocrine stress responses, autonomic activity, and reward sensitivity. Brain scan studies have identified multiple abnormalities in the structure and function of the amygdala, cingulate and prefrontal cortex, and related limbic and striatal brain areas (Miller, 2007). For example, the amygdala, hippocampus, and thalamus have been found to have smaller volumes in adolescents and adults with depressive disorders. Interestingly, maternal depression during pregnancy is related to the microstructure of the right amygdala of newborn infants (Rifkin-Graboi et al., 2013). Smaller volumes of several of the aforementioned brain structures in infants as young 6 weeks of age have been associated with higher levels of internalizing behaviors at 18 and 36 months of age (Herba et al., 2010). These findings suggest a possible biological vulnerability for the development of internalizing problems that may be present early in life. Studies have also identified cortical thinning in the right hemispheres of children and adults with or at risk for depression based on family history (Fallucca et al., 2011; Peterson et al., 2009). Cortical thinning in the right hemisphere might produce disturbances in arousal, attention, and memory for social stimuli that predispose the individual to developing a depressive disorder.

In general, brain activity has been found to be less active than normal in regions of the brain associated with attention, executive functions, and sensory processes, but more active than normal in regions involved in recognizing and regulating emotions, mediating stress responses, and learning and recalling emotion-arousing memories (Ho et al., 2013; Sylvester et al., 2013; Yurgelun-Todd, Sava, & Dahlgren, 2007). For example, the amygdala may overstimulate brain structures involved in forming certain types of memories, perhaps accounting for the tendency of depressed youngsters to ruminate on past negative life events. Overactivity of the amygdala may also affect the recognition and consolidation of social stimuli (e.g., faces, tone of voice) from a very early age so that ordinary interpersonal events are seen or recalled as aversive or emotionally arousing (Gaffrey et al., 2011; Lau et al., 2009; Monk et al., 2008). Consistent with this hypothesis, one study found elevated amygdala activity during face processing in 4- to 6-year-old children with depression, which was also related to parent-reported child emotion dysregulation and negative affect (Gaffrey et al., 2013) (see ● Figure 10.4). Neuroimaging studies of youngsters with MDD have also identified disruptions in neural activity in areas of the brain associated with decision making about future rewards and responses to rewarding outcomes (Forbes et al., 2006).

The hippocampus, one of the brain's memory centers, has also been implicated in depression. Parts of the hippocampus are involved in recognizing the environmental contexts for reward or danger, including sensitivity to stress. Brain-scan studies have found that individual variations in hippocampal volume interact with family stress to prospectively predict differences in depressive symptoms in adolescent girls over a period of 2.5 years (Whittle et al., 2011). Because of




● **FIGURE 10.4** | Brain scans of preschoolers with depression revealed elevated activity in the amygdala (the small area in the red circle) during face processing when compared with scans of young children exhibiting no signs of depression.

Washington University School of Medicine

variants in the hippocampus, individuals with depression may experience a constant state of anxiety and have difficulty recognizing situations that are safe (Davidson, Pizzagalli, & Nitschke, 2002). Studies of other brain regions have found that healthy adolescents who respond to peer rejection with greater activation of the anterior cingulate cortex are more likely to show an increase in depressive symptoms over the following year (Masten et al., 2011). These findings suggest that activity in brain regions involved in affective processing of socioemotional stimuli may provide a possible neurobiological marker for predicting healthy youngsters' future risk for depression.

Other studies into the neurobiological correlates of depression in young people have focused on hypothalamic–pituitary–adrenal (HPA)-axis dysregulation; sleep abnormalities suggestive of reduced neuroplasticity; variants in BDNF, which is involved in nerve growth and development; and the brain neurotransmitters serotonin, dopamine, and norepinephrine, which are widely spread throughout brain circuits thought to underlie mood disorders (Miller, 2007). Although findings related to these neurobiological correlates are suggestive, keep in mind that studies of children are few in number, and the findings are far less consistent than those for adults (Kaufman et al., 2001).

HPA-axis dysregulation is evidenced by abnormal cortisol responses in children and adolescents with depression, including higher baseline levels and atypical or overactive responses to stressors (Lopez-Duran, Kovacs, & George, 2009). HPA-axis and other neurobiological findings have led to a strong interest in the impact of early exposure to stress on later negative moods. Mounting evidence suggests that early adversity (e.g., prenatal stress, harsh or neglectful parenting) may produce HPA-axis abnormalities (e.g., alterations in corticotropin-releasing hormone [CRH] circuits), which sensitize the child to later stress, thus increasing the risk for developing depression (Heim & Nemeroff, 2001; Huizink, Mulder, & Buitelaar, 2004).

Infants of depressed mothers show higher levels of salivary cortisol (the stress hormone) and less relative left frontal lobe electrical activity than infants of mothers without depression (Dawson et al., 1997). Like higher levels of cortisol, decreased relative left frontal lobe activity may be a vulnerability factor for negative emotional states and later onset of depression (Nusslock et al., 2011; Forbes et al., 2008), although not all studies support this finding (e.g., Shankman et al., 2011). Nevertheless, research suggests that interactions between depressed mothers and their infants may produce biochemical and neurological changes that form and perpetuate a lasting basis for depressive disorder (Cytryn & McKnew, 1996; Post et al., 1996).

In summary, findings from studies of neurobiological correlates suggest that youngsters with depression may have a heightened sensitivity to stress. Repeated neuroendocrine activation related to stress might increase youngsters' susceptibility to chronic depressive symptoms, which in turn may lead to further extreme biological activation and psychosocial stress. Neurobiological findings over a wide age range of children are suggestive of widespread abnormalities in executive, affective, and motor networks of the brain, and in brain areas supporting emotional regulation (Ho et al., 2013). However, further research will be needed to clarify the specific neural circuits underlying depression in young people. The characteristics, severity, course, and outcome of a depressive episode may depend on the extent to which different neural circuits and processes are involved in response to environment demands and on when during development these networks are formed (Gabbay et al., 2013; Goodyer, 2008).

## Family Influences

*"I was always able to explain away my daughter's symptoms," says the mother of a 12-year-old. "When she was 10 and fought with me about everything, I just wrote it off as preadolescent hissy fits. When she dropped out of gymnastics—which had been her raison d'être—and started losing weight, I told myself she was just searching for a new identity. But when her best friend came to me and told me that my daughter was talking about suicide, I was forced to face the truth. I keep blaming myself. What did I do to cause this depression? What could I have done to prevent it?"*

—From "Childhood Depression," by K. Levine, pp. 42–45, *Parents*, October 1995.

Family influences play an important role in the development, onset, maintenance, and course of depression in young people (Schwartz et al., 2013; Restifo & Bögels, 2009). One approach to examining these influences looks at families of children and adolescents with depression; the second approach considers families in which parents, particularly mothers, are depressed.

### *When Children Are Depressed*

Families of children with depression display more critical and punitive behavior toward their depressed child than toward other children in the family. As compared with families of youngsters without depression, these families display more anger and conflict, greater use of control, poorer communication, more overinvolvement, and less warmth and support (Sheeber et al., 2007; Stein et al., 2000). They often experience high

levels of stress, disorganization, marital discord, and a lack of social support (Messer & Gross, 1995; Slavin & Rainer, 1990). Youngsters with depression describe their families as less cohesive and more disengaged than do youngsters without depression (Kashani et al., 1995).

Research points strongly to the link between childhood depression and family dysfunction. One longitudinal study found that less support and more conflict in the family were associated with more depressive symptoms in adolescents both concurrently and prospectively over a 1-year period. In contrast, more depressive symptoms did not predict a worsening of family relationships over the same time period. Thus, family problems precede and may be directly related to the development of depressive symptoms (Sheeber et al., 1997).

### When Parents Are Depressed

*To mother appropriately requires the action of systems that regulate sensation, perception, affect, reward, executive function, motor output and learning. When a mother is at risk to engage in less than optimal mothering, such as when she is depressed … the function of many or all of maternal and related systems may be affected.*

—Barrett & Fleming, 2011, p. 368

**MRS. D.**

#### Not Up to Mothering

Mrs. D. is depressed and has been helpless and needy for most of her 5-year-old daughter Maria's life. She moves ever so slowly to prepare breakfast for Maria and herself. Wringing her hands, she pays little attention to events around her. Maria has been tugging at her mother for some time, apparently wanting food. Mrs. D. mumbles something, sobs continuously, and wipes tears from her cheek as she moves between the cupboard and kitchen table. Maria persists in trying to gain her mother's attention, and finally Mrs. D. hugs her and strokes her hair. At first Maria pulls back; then she snuggles against her mother's legs. Finally, Mrs. D. fills a bowl with cereal, and she and Maria sit down to eat in total silence, during which Mrs. D. looks sadly at her daughter. Deep bouts of depression periodically incapacitate Mrs. D., and any problem that Maria has sends her to bed. Mostly, Maria is left on her own to handle problems.

Adapted from Radke-Yarrow & Zahn-Waxler, 1990.

Depression interferes with a parent's ability to meet the basic physical and emotional needs of a child, including feeding, bedtime routines, medical care, and safety practices. Mothers who suffer from depression, like Mrs. D., also create a child-rearing environment teeming with negative mood, irritability, helplessness, less emotional flexibility, and unpredictable displays of affection. When their children display negative emotions and distress, mothers with a history of depression are less likely to respond supportively with comfort, empathy, or assistance and are more likely to disapprove, dismiss, punish, or ignore their child's negative emotions (Silk et al., 2011). Depressed mothers also display less energy in stimulating play, less consistent discipline, less involvement, poor communication, lack of affection, and more criticism and resentment of their children than mothers without depression (Goodman, 2007). High levels of marital conflict, family discord, and stress may also be present in the home when a parent is depressed (Hammen, 2002). Critically, this type of negative family environment in combination with a child's genetic predispositions can adversely affect the development of stress regulatory systems and predispose the child to a lifetime of depressive illness and other negative health outcomes (Taylor, Way, & Seeman, 2011).

Maternal depressions during and shortly after pregnancy have been found to be independent risk factors for major depression in the offspring at age 18 years (O'Connor, Monk, & Fitelson, 2014; Pearson et al., 2013). The first year of a child's life seems to be a particularly sensitive period for the effects of maternal depression on the child's later behavior and other adverse outcomes (Bagner et al., 2011). Depressed mothers may differ from one another in their styles of interaction; some are more intrusive and others are more withdrawn. These differences are important because they may be associated with different child outcomes. For example, children of depressed mothers with an intrusive maternal style display avoidance and "tuning out," whereas children of depressed mothers with a withdrawn maternal style display heightened

Chris Rout / Alamy

Maternal depression interferes with the mother's ability to meet the needs of her children

sociability toward strangers (Hart, Jones, & Field, 2003). Thus, the offspring of mothers with depression attempt to cope with the unpredictability of their environment in different ways, which are often maladaptive and show reactions ranging from aggressive behavior to withdrawal, failure to thrive, school refusal, depression, and even suicidal behavior (Goodman & Tully, 2008). Like Maria, they must take care of themselves and learn how to handle their own problems.

It is not surprising that children of depressed mothers show cognitive deficits, emotional delays, separation difficulties, insecure attachments, and less positive affect during their development (Olino et al., 2011). These children also display early signs of a cognitive vulnerability to depression. They tend to be self-critical, display a negative attributional style, and have a lower self-concept. They also have difficulties regulating their emotions and experience decreased social acceptance as young as age 5 (Dagne & Snyder, 2011; Maughan et al., 2007). As a result of these disturbances in emotion regulation, the children are ill equipped to cope effectively with stressful events, which subsequently places them at risk for higher levels of depression, lower functioning across multiple domains, and lower perceived competence (Garber & Cole, 2010; Goodman et al., 2011). Given the emotion-regulation disturbances seen in both depressed mothers and their children, it is not surprising that by adolescence or earlier, maternal depression is associated with greater *mutual* engagement in negative affect during parent–child interactions, rather than solely reflecting the mothers' own negativity (Connell et al., 2011).

Follow-up studies of these children confirm the risks associated with growing up in a family with a depressed parent. Over a 10-year period, children of depressed parents had not only increased rates of depression, particularly before puberty, but also higher rates of phobias, panic disorder, and alcohol dependence (Weissman et al., 1997). As compared with controls, offspring of depressed parents received more outpatient treatment over 10 years and had poorer overall functioning in work, family, and marital relationships. In terms of health-care use, children of depressed parents have a higher rate of medically attended physical injuries in the home, emergency and sick visits, and inpatient and specialty-service use and a lower rate of well-child care visits (Phelan et al., 2008; Sills et al., 2007). The findings from follow-up studies are sobering in documenting the serious long-term negative outcomes and impairments in children whose parents suffer from depression (Weissman et al., 2006).

Several issues regarding the relationship between maternal depression, child depression, and family factors need to be considered. First, the kinds of family difficulties we have described are related to many other child disorders, and they may not be specific to depression. Second, it is difficult to know whether family problems are the result of one or more co-occurring conditions, such as child conduct problems, or maternal anxiety disorder or antisocial behavior, rather than depression. It is generally found that, although statistically significant, severity of maternal depression by itself accounts for only a small amount of the variance in child outcomes, indicating that other risk factors will also need to be considered (Goodman, 2014). Third, most studies are correlational, making it impossible to determine the direction of influence. An adverse family environment can lead to child depression, but child depression may also evoke negative and critical reactions from family members and produce distress in others. Fourth, another factor, such as genetic risk, may account for both depression and family disturbances. Fifth, protective factors in the mother or child may reduce the risk of negative child outcomes. For example, one study reported that the presence of a specific maternal genotype markedly reduced the impact of maternal depression on child psychopathology (Apter-Levy et al., 2013). Some support exists for all these mechanisms of family influence on child depression.

Finally, a shortcoming of existing research has been the relative lack of attention to fathers with depression. A steadily growing number of studies indicate that paternal depression has significant but small effects on parenting, with depressed fathers showing less positive and more negative parenting behaviors than those who are not depressed (Wilson & Durbin, 2010). However, few studies have examined paternal behavior in relation to child outcomes. Although internalizing and externalizing problems in children are more strongly associated with depression in mothers as compared with depression in fathers, depression in fathers still may play an important moderating role—for example, through its impact on the marital relationship (Connell & Goodman, 2002; Kane & Garber, 2004).

The high frequency of maternal depression combined with the numerous associated developmental, health, and behavioral problems in children of depressed mothers creates a pressing need for effective treatments for depressed mothers and their children (Wachs, Black, & Engle, 2009). Importantly, some studies have found that reductions in parents' depressive symptoms with treatment can lead to both immediate and longer-term decreases in their children's problem behaviors and symptoms and to favorable changes in their child's global functioning (Wickramaratne et al., 2011). Regulated early child-care services may also help to buffer the negative effects of maternal depression on children's internalizing problems (Herba et al., 2013).

## Stressful Life Events

**CARLINE**

### How Depression Acts

I don't feel depressed all the time. It comes and goes. Usually it takes something to set it off. It could be something big, like when we moved, but anything, no matter how small, can really get to me, and then I start feeling bad and can't do anything. So today things are OK and I don't feel so bad. But tomorrow, or the next day, something might happen, no matter how minor, and I just might not want to get out of bed, or do anything. (Based on authors' case material)

Depression is associated with both severe and nonsevere stressful life events (Rudolph et al., 2006). Severely stressful events may include a move to a new neighborhood, a change of schools, a serious accident or family illness, an extreme lack of family resources, a violent family environment, or parental conflict or divorce (Gilman et al., 2003; Goodyer et al., 1997). At times, nonsevere stressful events, or "daily hassles," such as a poor grade on a test, an argument with a parent, criticism from a teacher, a fight with a boyfriend, or a broken date, may also result in depression. Relative to nondepressed youngsters, those who become depressed experience significantly more severe and nonsevere stressful life events in the year preceding their depression—especially events related to romantic relationships, education, relationships with friends or parents, work, and health (Birmaher et al., 1996).

Triggers for depression often involve interpersonal stress or actual or perceived personal losses, such as the death of a loved one, abandonment, rejection, or a threat to one's self-esteem (Eley & Stevenson, 2000; Goodyer, 1999). For example, a recent relationship break-up seems to be an especially significant predictor for first versus later episodes of major depression during adolescence (Monroe, Rohde, Seeley, & Lewinsohn, 1999). Sadness and depression following loss are common. In children 6 to 17 years of age who had recently suffered the most horrible loss possible—the loss of a parent—all experienced sadness, grief, and other symptoms (Cerel et al., 2006). Thirty percent of adolescents who had lost a friend or peer through suicide developed a depressive disorder within 6 months after the loss (Brent et al., 1992). Yet depressive disorder is not an inevitable outcome to personal losses—most children who experience the loss of a parent or friend show far fewer depressive symptoms than children who are clinically depressed, and they do not develop major depression (Cerel et al., 2006).

## Emotion Regulation

**Emotion regulation** refers to the processes by which emotional arousal is redirected, controlled, or modified to facilitate adaptive functioning and the balance maintained among positive, negative, and neutral mood states (Cole & Hall, 2008). Youngsters demonstrate wide differences in regulating their emotions and managing their negative mood states (Keenan, 2000). For example, if a favorite playmate cannot be found, one child may cry and cannot be comforted; another may cry for a short time and then find someone else to play with; and another child may look to an adult for comfort. Children's strategies for self-regulation play a crucial role in overcoming, maintaining, or preventing negative emotional states. As we have discussed, young children who experience prolonged periods of emotional distress and sadness, or who are exposed to maternal negative moods, may have problems regulating negative emotional states and may be prone to the development of depression (Dagne & Snyder, 2011; Durbin & Shafir, 2008).

A variety of skills are necessary to manage one's own emotions. These include recognizing changes in emotion, accurately interpreting the conditions that led to mood change, setting goals to change one's mood, and implementing effective coping responses. Youngsters with depression may show deficits in one or more of these regulatory skills and, as a result, have difficulty overcoming their negative moods (Sheeber et al., 2000; Yap et al., 2011). They may use avoidance or negative behavior to regulate their distress, rather than more problem-focused and adaptive coping strategies. Since emotion regulation encompasses neurobiological regulatory processes, acquired behavioral and cognitive strategies, and external resources for coping, depression may result from difficulties in any one or more of these areas (Cole & Hall, 2008).

## Section Summary

### Causes of Depression

- Depression is likely a final common pathway for interacting influences that predispose a child to develop the disorder.
- Family and twin studies and specific gene studies suggest that what may be inherited is a vulnerability to depression and anxiety and that certain environmental stressors may be required to express these disorders.
- Youngsters with depression may experience heightened reactions to stress that increase their vulnerability to depression. Studies of neurobiological correlates have focused on limbic and prefrontal neural circuits; the HPA axis; sleep abnormalities; growth hormone; variants in

(*continues*)

## Section Summary (*continued*)

BDNF, which is involved in nerve growth and development; and the brain neurotransmitters serotonin, dopamine, and norepinephrine.

- Families of children with depression display anger and conflict, greater use of control, less effective communication, more overinvolvement, and less warmth and support than families of children who are not depressed.
- Children of depressed parents experience increased rates of depression before puberty; higher rates of phobias, panic disorder, and alcohol dependence as adolescents and adults; and other negative health outcomes.
- Depression is associated with both severe stressful life events, such as a move to a new neighborhood, and less severe stressful events or daily hassles, such as criticism from a teacher or an argument with a boyfriend.
- Young children who experience prolonged periods of emotional distress and sadness may have problems in regulating their negative emotional states and may be prone to the development of depression.

# TREATMENT OF DEPRESSION

### LEETA

### Feeling Better

Leeta, age 16, sat slumped in her chair. Disheveled and distracted, she answered questions in a vague and unfocused manner. She was admitted to the hospital after she slit her wrists with a knife; she had become despondent, irritable, and out of control at home. Leeta's thoughts and reasoning were distorted. She expressed a pervasive sense of hopelessness and was certain that she would remain in hospitals for the rest of her life.

Fortunately, this was not the case. She became involved in cognitive–behavioral therapy that focused on accurate reasoning, a more positive self-image, and ways to lessen family turmoil and was also treated with antidepressant medication. One year later, Leeta entered our office for a follow-up interview with energy and excitement. "I never thought that I would feel like hanging out with friends and doing things again. It's not that I don't get sad once in a while, but it doesn't take over my whole life."

Adapted from Oster & Montgomery, 1995.

Many potentially effective psychosocial and psychopharmacological treatments are available to treat youngsters with depression (Brent, Poling, & Goldstein, 2011; David-Ferdon & Kaslow, 2008; Maalouf & Brent, 2012). Despite this availability, less than half of children with depression receive help for their problem (Olfson et al., 2003). Rates of treatment vary by racial/ethnic background, being highest for non-Hispanic white youths (40%) and lowest for Asian youths (19%). About one-third of African American and Hispanic youths receive treatment for their depression (Cummings & Druss, 2011). In one study of adolescents who killed themselves, most of whom had a depressive disorder, only 7% had been in treatment prior to their suicide (Brent et al., 1988). The high comorbidity, associated deficits, and recurrence of depression in young people require a combination of treatments, with an emphasis on eliminating depressive symptoms, maintaining positive outcomes, and preventing relapse (AACAP, 2007d; Cheung et al., 2007).

Cognitive–behavioral therapy (CBT), the treatment used with Leeta, has shown the most success in treating children and adolescents with depression (Watanabe et al., 2007). In addition, Interpersonal Psychotherapy for Adolescent Depression (IPT-A), which focuses on improving interpersonal functioning by enhancing communication skills in significant relationships, has also proven to be an effective treatment (Mufson et al., 2004). CBT and IPT-A are also more efficacious for treating depression than other forms of therapy (e.g., family therapy or nondirective supportive therapy) (David-Ferdon & Kaslow, 2008).

With regard to psychopharmacological treatment, tricyclic antidepressants have not proven to be nearly as effective for depressed young people as for depressed adults (Papanikolaou et al., 2006). In contrast, new-generation antidepressants, especially the selective serotonin reuptake inhibitors (SSRIs), have demonstrated moderate efficacy in controlled studies with young people (Fombonne & Zinck, 2008). However, concerns have been raised about their effectiveness, overuse, and possible side effects, which we discuss later in this section. In addition to CBT, IPT-A, and medications, many variations in these treatments (e.g., individual versus group format, increasing amounts of parental involvement, combined treatments, computer-based treatments, and online treatments) are available. Other treatments that emphasize self-control, social support, family relationships, and increasing social and academic functioning, have also been used with varying degrees of success (David-Ferdon & Kaslow, 2008). Screening for and treating maternal depression may also benefit the child's response to his or her own treatment and help the mother to be a more effective participant in her child's treatment (Kovacs & Yaroslavsky, 2014). To date, studies have reported some benefits for older depressed children whose mothers were treated for depression, but short-term or no benefits for infants and toddlers whose mothers received treatment. These

findings suggest that direct treatment of very young children with depression may be needed to sustain developmental changes (Luby, 2013).

An early onset of depression places youngsters at greater risk for experiencing multiple episodes of major depression throughout their lives. Therefore, it is critical that treatment begin as soon as possible; very early and aggressive intervention is warranted to reduce the length of a depressive episode, reduce the likelihood of future episodes, minimize associated impairments in functioning, and reduce the risk of suicide. An overview of the main treatments for youngsters with depression is presented in Table 10.3.

## Psychosocial Interventions

Most psychosocial interventions for depression in young people use an integrated approach derived from two traditions—behavior therapy and cognitive therapy. These two approaches for depression were originally developed with adults, but have since been adapted and extensively applied with children and adolescents.

*Behavior therapy* maintains that depression results from and is sustained by a lack of reinforcement due to a restricted range of potential reinforcers, few available reinforcers, or inadequate skills for obtaining rewards (Lewinsohn, 1974). Consequently, the treatment focuses on increasing pleasurable activities and events and providing the youngster with the skills needed to obtain more reinforcement. Interventions such as social skills training teach children assertiveness, communication, how to accept and give feedback, social problem solving, and conflict resolution skills in order to increase positive social interactions. Strategies such as daily monitoring, structuring activities, and scheduling activities are used to help youngsters with depression become more active, engage in rewarding experiences, and solve problems (McCauley et al., 2011).

*Cognitive therapy* teaches youngsters with depression to identify, challenge, and modify negative thought processes such as misattributions, negative self-monitoring, short-term focus, excessively high performance standards, and a failure to self-reinforce. They are taught to identify and eliminate negative thoughts, such as "It's my fault," or "What's the point?" and taught to replace them with positive thoughts, such as "She really likes me," or "I'm an interesting person." A child who has been rejected by a friend might be encouraged to think, "She was in a bad mood," rather than "She hates me." When youngsters are presented with specific situations and examples of irrational negative thinking, they are taught to substitute alternative logical explanations that are more positive. For example:

- SITUATION: Two girls, Diana and Colleen, both ask friends to get together with them after school. Both girls' friends say they can't because they have too much homework to do.
- IRRATIONAL THINKING: Diana feels rejected and thinks, "Because my friend won't get together with me, she doesn't like me, and she'll never want to do anything with me again."
- RATIONAL THINKING: In contrast, Colleen thinks, "Well, my friend is busy today, but we can get together some other time. She's still my best friend."

**TABLE 10.3 | Treatments for Youngsters with Depression**

| | |
|---|---|
| **Behavior Therapy** | Aims to increase behaviors that elicit positive reinforcement and to reduce punishment from the environment. May involve teaching social and other coping skills, and using anxiety management and relaxation training. |
| **Cognitive Therapy** | Focuses on helping the youngster with depression become more aware of pessimistic and negative thoughts, depressogenic beliefs and biases, and causal attributions of self-blame for failure. Once these self-defeating thought patterns are recognized, the child is taught to change from a negative, pessimistic view to a more positive, optimistic one. |
| **Cognitive–Behavioral Therapy (CBT)** | The most common form of psychosocial intervention. Combines elements of behavioral and cognitive therapies in an integrated approach. Attribution retraining may also be used to challenge the youngster's pessimistic beliefs. |
| **Interpersonal Psychotherapy for Adolescent Depression (ITP-A)** | Explores family and interpersonal interactions that maintain depression. Family sessions are supplemented with individual sessions in which youngsters with depression are encouraged to understand their own negative cognitive style and the effects of their depression on others and to increase pleasant activities with family members and peers (Mufson, et al., 2004). |
| **Medication** | Treats mood disturbances and other symptoms of depression using antidepressants, especially selective serotonin reuptake inhibitors (SSRIs). |

In practice, behavior therapy and cognitive therapy are integrated into a unified cognitive behavior therapy (CBT) approach, in which more adaptive cognitions are hypothesized to lead to more adaptive behavior and vice versa. Examples of these integrated CBT approaches for children and adolescents follow.

### Primary and Secondary Control Enhancement Training (PASCET)

John Weisz and his colleagues (2003) have developed a 15-session, individualized CBT-based program for youngsters 8 to 15 years of age who have depression. In treatment sessions and in take-home assignments, youngsters learn and practice two types of coping skills:

- *Primary control skills* (ACT skills) for changing objective events in their lives (e.g., changing the activities they engage in, learning to relax) to conform with their wishes.
- *Secondary control skills* (THINK skills) for altering the subjective impact of stressful life events (e.g., altering their negative thoughts and feelings).

The focus of the PASCET program is to help the child change conditions that are changeable and to change the subjective impact of those that are not. Parents are also involved in the program and are encouraged to support their children in using these coping skills. PASCET is an excellent example of a program that has evolved since its initial use in the schools to reduce depressive symptoms. It is now being implemented and evaluated in community mental health clinics with youngsters who have been referred for depression (Bearman & Weisz, 2009). A current and ongoing priority for PASCET and similar CBT programs is on identifying the treatment components needed to effectively deploy them in intervention settings where youngsters with depression are typically referred for help and treated by clinical practitioners (Bearman et al., 2010; Weisz et al., 2013).

### The ACTION Program

Kevin Stark and his colleagues (2012) have developed a comprehensive CBT approach for children with depression. The primary components of this treatment are appropriate for children and adolescents and for boys as well as girls. However, the current format is designed to be gender-sensitive, with treatment activities, skills emphasized, and a focus on interpersonal relationships specific to girls in the 9- to 13-year age range (Stark et al., 2008). Like the PASCET program, ACTION uses a holistic approach that involves both child and parents. The ACTION acronym is used to nourish the idea that youngsters can have an impact on their moods, and it is presented to them as follows (Stark & Kendall, 1996, p. 14):

**A** = Always find something to do to feel better.
**C** = Catch the positive.
**T** = Think about it as a problem to be solved.
**I** = Inspect the situation.
**O** = Open yourself to the positive.
**N** = Never get stuck in the negative muck.

Multiple treatment procedures are used to reduce the child's mood disturbances, behavioral deficits, and cognitive symptoms:

- *Dysphoria, anger, anhedonia,* and *excessive anxiety* are treated by educating the child about the relation between mood, thinking, and behavior, and by using anger management procedures, scheduling pleasant activities, and relaxation training.
- *Interpersonal deficits* are treated using social skills training.
- *Cognitive distortions* and *negative and self-critical thinking* are addressed by using cognitive-restructuring procedures and training in effective problem-solving and self-control procedures.

Interventions can be carried out in both individual and group formats, and they make use of a workbook that includes a variety of exercises such as this one:

- SITUATION: You accidentally drop your books … a group of classmates are talking and laughing at the other side of the room.
- NEGATIVE THINKING: Now look at what I've done. They must think I'm a complete idiot.
- COPING RESPONSE: No, they're probably laughing at something else. Besides, I know them. They're not like that. It's not like I'm the first person ever to drop her books. It's really no big deal.

Interventions with parents are used to facilitate the child's use of effective coping strategies outside of treatment and to change events that may contribute to and prolong the child's problems. Since negative parent–child interactions may result in negative thinking, changing maladaptive patterns within the family is an important feature of the ACTION program. Several methods are used to change parental and family cognitions and behavior, including teaching parents effective forms of discipline, ways to manage anger, and ways to change negative thinking. Interventions with the entire family teach negotiation and conflict-resolution skills, recreational planning, and effective problem solving and family communication (Stark et al., 2012). ACTION is a promising intervention built on a sound theoretical and research base. The program continues

to be evaluated as a comprehensive treatment package for depression (Stark et al., 2010).

### Adolescent Coping with Depression Program (CWD-A)

One of the most well-established and comprehensive CBT programs for the treatment of depression in adolescents is the Adolescent Coping with Depression Program (CWD-A) (Clarke, Lewinsohn, & Hops, 2001). CWD-A is a nonstigmatizing psychoeducational approach that emphasizes skills training to promote adolescents' control over their moods and enhancement of their ability to cope with problematic situations. Treatment is provided in 16 two-hour sessions over an 8-week period for groups of up to 10 adolescents ages 13 to 18. Adolescents use a workbook that includes brief readings, short quizzes, structured learning tasks, and forms for homework assignments for each session. The core treatment sessions with adolescents involve group activities and role playing. In addition, complementary therapy with the youngsters' parents is carried out to accelerate and support the learning of new skills, and to assist in applying the skills learned in the group to everyday life situations. Periodic "booster sessions" help to maintain the skills taught during treatment (Clarke & DeBar, 2010).

Initially, adolescents learn that depression can have many causes, including inherited tendencies, stress, and excessive negative thinking. Relaxation training is then used to quickly provide a successful experience and some immediate relief. Subsequent sessions include the following components (Clarke & DeBar, 2010):

- Self-change skills, such as self-monitoring of mood and behavior, and ways to establish realistic goals, are taught.
- Pleasurable activities and opportunities for reinforcement are increased.
- Positive thinking is increased by identifying, challenging, and changing negative cognitions.
- Training in social, communication, and problem-solving skills is integrated throughout the program.
- Specific skills are taught, such as conversational skills, ways to plan social activities, and ways to make friends.
- Goal setting is used to identify short- and long-term life goals and potential barriers to these goals.
- Final sessions emphasize integrating the skills learned and making plans for the future.

The CWD-A program and modified versions (e.g., group format, brief treatment protocol) have demonstrated beneficial, albeit moderate and in some cases not long-lasting, treatment and prevention effects in many controlled studies by its developers and others (Cuijpers et al., 2010; Rohde, Stice et al., 2010, 2013). However, as with the other treatments we have discussed, there is a need for further evaluation by independent investigators, extending its use to a wider variety of depressed youngsters, longer-term follow-up studies, and comparisons with other treatments for depression, particularly medication (Clarke & DeBar, 2010).

### Interpersonal Psychotherapy for Adolescent Depression (IPT-A)

IPT-A is based on the idea that adolescent depression affects relationships, which in turn affect mood. Thus, treatment focuses on the adolescent's depressive symptoms and the social context in which these symptoms occur (Young & Mufson, 2008). The emphasis in IPT-A is on increasing adolescents' independence and negotiating their interdependence on others by addressing relevant developmental issues such as romantic relationships, separation from parents, and peer relationships. The adolescent takes an active role in identifying a specific problem area (e.g., loss and grief, interpersonal disputes, role transitions, interpersonal deficits), discussing communication and problem-solving techniques for that area, practicing these skills in session, and applying them outside sessions in the context of significant relationships. The treatment is structured around addressing the identified problem areas, and both the therapist and adolescent are expected to play an active role (Mufson, et al., 2004).

IPT-A is designed as a once-weekly, 12-session outpatient program. Treatment is divided into three phases. The *initial phase* (4 sessions) addresses the diagnosis of depression, educates the adolescent and family about depression, introduces the principles of IPT-A and the structure of treatment, identifies an interpersonal problem area, and makes a treatment contract. The *middle phase* (5 sessions) further clarifies the problem, identifies strategies for effectively targeting the problem, and implements interventions to resolve the problem. A number of techniques are used. For example, if an adolescent reports a fight with her boyfriend, she would be asked how this made her feel and whether or not this affected her depressive symptoms. This helps to educate the adolescent about the link between interpersonal events and mood, and makes her feel more comfortable and skilled at identifying and communicating feelings. Other techniques in this phase focus on helping the adolescent to recognize the impact of her communication on others, the feelings generated, and how modifying the communication may impact the outcome of the interaction and the adolescent's associated feelings. Additional techniques involve problem solving in interpersonal situations, role playing both communication and problem-solving skills, and

the use of homework to practice these skills between sessions (Young & Mufson, 2008).

The *termination phase* (3 sessions) reviews progress in the identified problem area, links changes in interpersonal functioning and relationships to improved mood and decreased depressive symptoms, and identifies strategies that have been most helpful. It also addresses the importance of continuing to use the learned strategies following treatment, highlights areas that still need improvement, and considers what to do if symptoms of depression return.

IPT-A has been shown to be an effective treatment for adolescent depression in a number of controlled studies in clinic, school, and community settings, using both individual and group formats (Mufson, 2010; Mufson et al., 2012). In an effort to reach more youngsters, IPT-A is also being developed as a preventive intervention ("Teen Talk") for adolescents in grades 7 to 10 who display elevated levels of depressive symptoms. Preliminary findings suggest that IPT-A and Teen Talk may useful approaches to preventing more severe forms of depression and reducing symptoms of anxiety (Young et al., 2012; Young, Mufson, & Davies, 2006).

In concluding our discussion of psychosocial treatments, the good news is that a wide variety of treatments for young people with depression have been shown to be effective for most youths who receive them (March & Vitiello, 2009; TADS Team, 2009). However, nearly half of those who recover, especially girls, have a relapse of their depression (Curry et al., 2011), and effect sizes have been moderate and smaller than those reported for adults (Weisz, McCarty, & Valeri, 2006). Hence, there is a need to explore more effective treatments that build on our growing understanding of childhood depression; reduce the rate of depression relapse or deterioration; personalize treatment to meet the child's cognitive, emotional, and developmental profile; and are likely to be used in clinical practice settings (Brent & Maalouf, 2009; Weisz et al., 2013). There is also a need to study treatments of longer duration and the use of booster sessions following treatment and to evaluate outcomes over longer follow-up periods.

## Medications

*"I kept hearing about Prozac in the news," says one father, "and when we finally brought my 9-year-old to a psychiatrist, I thought he could just give her this pill and change our lives. After a year, I can say that things are a bit better. But it took lots of trials with lots of different pills."*

—From "Childhood Depression," by K. Levine, pp. 42–45, *Parents,* October 1995. Reprinted by permission of the author/

Antidepressant medications are commonly used to treat youngsters with depression. An estimated 1.4 million youngsters in the United States received antidepressant medication in 2002 (Vitiello, Zuvekas, & Norquist, 2006), with over 60% of those treated in outpatient settings filling prescriptions for these drugs (Olfson et al., 2014). For many youngsters, antidepressant medications can shorten a depressive episode and return them to the important developmental tasks of childhood and adolescence. As we have noted, although tricyclic antidepressants are effective with adults, they have consistently failed to demonstrate any advantage over placebo in treating depression in young people, and may have some potentially serious cardiovascular side effects (Fombonne & Zinck, 2008). As a result they are no longer regarded as primary drugs for the management of depressive symptoms in young people.

SSRIs have clearly become the first line of antidepressant medication treatment for youngsters with depression, with one national survey reporting that over 90% of prescriptions written for these youngsters were for SSRIs (Olfson et al., 2003). Among the most commonly used SSRIs are fluoxetine (Prozac), sertraline (Zoloft), and citalopram (Celexa). SSRIs achieve their antidepressant effects by blocking the reuptake of serotonin, thereby increasing its availability in the synapse and stimulating the postsynaptic neuron. At present, the only SSRI that is approved by the Food and Drug Administration (FDA) for use with children and adolescents with MDD is fluoxetine.

A number of controlled investigations have demonstrated that some SSRIs are moderately effective in reducing symptoms of depression in children and adolescents. One meta-analysis found that 40% to 60% of children responded to Prozac versus 20% to 35% of those on placebo (Hetrick, McKenzie, & Merry, 2010). Support for the effectiveness of other SSRIs was limited. There was also little evidence that children and adolescents who took SSRIs showed improvement in their school performance, interpersonal relations, or social functioning on a day-to-day basis (Hetrick et al., 2010). Others have argued that research supports the use of SSRIs, that combined treatments using medication and CBT in combination are likely to be the most effective, and that greater treatment effects are obtained for children with more severe depressions (March, 2010).

After they were first marketed in the late 1980s, the use of Prozac and other SSRIs increased dramatically. For example, nearly three-quarters of a million prescriptions for SSRIs for children ages 6 to 18 were written in 1996—an 80% increase in only 2 years (*APA Monitor,* December 1997). However, despite some support for their efficacy, both professional and public concerns have been voiced about their use with children and adolescents. The main concerns are

possible serious side effects such as suicidal thoughts and self-harm and a lack of information about the long-term effects of these medications on the developing brain. Related to these concerns and warnings by the FDA, the use of SSRIs with young people has decreased by about 20% in more recent years (Gibbons et al., 2007; Libby et al., 2007). In 2004, the FDA asked all manufacturers of antidepressant medications to include in their labeling a boxed warning (black box) and Patient Education Guide to alert consumers about the increased risk of suicidal thinking and behavior in youngsters treated with these medications. A summary of the main points included in these black box warnings is presented in A Closer Look 10.2.

The FDA warnings were based on a pooling of findings from 24 short-term, placebo-controlled studies of antidepressant trials with more than 4,400 youngsters with MDD and other disorders. The overall findings indicated an increased risk of suicidal thinking or behavior in youngsters with depression (4% on active medication vs. 2% on placebo) (Hammad, Laughren, & Racoosin, 2006). The risk for untreated youths with depression, the long-term effects of medication, and the combination of medication and psychosocial interventions were not evaluated, and there were no completed suicides in any of the studies. In addition, findings regarding increases in suicidality from other studies have been inconsistent (Gibbons et al., 2012). Also inconsistent are findings regarding the use of medications either alone or in combination with psychosocial interventions. Some studies have found a benefit of combined treatment versus medication alone, with an enhancement of the safety of medications when used in combination with CBT (e.g., Treatment of Adolescents with Depression Study [TADS] Team, 2004, 2007), whereas others have not (e.g., Goodyer et al., 2007).

Thus, despite much research, the risks, long-term safety, and benefits associated with the use of antidepressant medications with young people remain uncertain (Moreno et al., 2007a). In light of the potential effectiveness of the psychosocial treatments for depression in youths that we have discussed, careful consideration must be given to determining which youngsters are most or least likely to benefit from antidepressant medication (AACAP, 2007d).

Notwithstanding these concerns, untreated depression has profound long-term consequences, including a high risk for suicide, and there is some evidence that a higher use of antidepressant medications across counties in the United States is associated with lower rates of suicide in young people (Gibbons et al., 2006). Thus, there may be possible risks that go along with not using medications relative to the risks from suicidal ideation and suicide attempts, especially when numerous research and clinical studies indicate that many young people benefit from drug treatment (Bridge et al., 2007).

In the absence of better data regarding drug effects, side effects, and long-term safety of medication use with depressed children, there are currently no easy answers to this dilemma. Some say it is unethical to treat depressed children using medications in light of the potential dangers. Others say that the risks associated with drug treatment are no greater than risks for other treatments and that it is unethical to withhold treatment in light of the known benefits. Given the many social, political, and economic implications surrounding the use of medications to treat depression in young people, we may hear a lot more about this issue for some time to come (e.g., Riddle, 2004).

In concluding our discussion of the treatment of depression, we note that controlled studies of psychological treatments and medication have found that up to 60% of youngsters with depression respond to placebo (Bridge et al., 2007) and about 15% to 30% respond to brief treatment (Goodyer et al., 2007). Thus, in youngsters with mild or brief depression, an absence of suicidality, and minor impairment in functioning, the use of education, support, and case management related to school and family stressors may be effective. However, for those who are more severely depressed, display suicidal ideation and behavior, and show significant impairment in functioning, the specific types of

A CLOSER **LOOK 10.2**

### Summary of Food and Drug Administration (FDA) Black Box Warnings for the Use of Antidepressants with Children and Adolescents

- Youths with MDD are at an increased risk for suicidal thinking or behavior.
- When considering an antidepressant for a child or adolescent, it is important to weigh the increased risk of suicidality with the possible benefits of the medication.
- When starting young people on antidepressants, they must be very closely monitored for worsening of symptoms, suicidality, or unusual changes in behavior.
- Family members must closely observe the youngster for increases in symptoms or worsening of functioning, and immediately communicate any such observations to their provider.
- A statement needs to be included regarding whether the medication is approved for use with children and adolescents.


psychological and pharmacological treatments that we have discussed will likely be needed (AACAP, 2007d; Cheung, Kozloff, & Sacks, 2013).

## Prevention

In view of the recurring nature of depression, successful efforts during childhood and adolescence to prevent the onset of depression may reduce a lifelong risk of illness and reduce the use and costs of health care resources (Horowitz & Garber, 2006; Stice et al., 2009).

Early studies of prevention with grade school and high school students with subclinical symptoms of depression found CBT/problem-solving approaches to be effective in reducing depressive symptoms and lowering the risk for developing depression up to 2 years after treatment (Gillham & Reivich, 1999; Shochet et al., 2001). However, not all programs have reported benefits. For example, in a later controlled study, a comprehensive school-based program for adolescents attempted to develop individual resiliency skills and enhance protective factors in the environment. The program failed to produce significant changes in levels of depressive symptoms or risk or protective factors in participating adolescents over the 3 years of the study (Sawyer et al., 2010). These negative results, despite extensive efforts to use evidence-based interventions, highlight some of the challenges in implementing school-based, universal prevention programs. Among these challenges are the need for effective teacher training over a large and diverse geographical area so that programs are implemented as prescribed, the difficulty in engaging young adolescents in prevention programs, and the amount of time required to implement policy and practice changes at "whole school" levels (Sawyer et al., 2010).

Other large-scale prevention efforts (e.g., *Columbia Teen Screen*) have been directed at the early detection of high school students at risk for depression and suicide to ensure that these students receive help (Shaffer et al., 2004). The importance of school-based screening is highlighted by the finding that although 90% of parents report that they are confident in their ability to tell if their child is thinking about suicide, the parents of only about one-third of teens with mental health problems know that their child has these problems. School-based screening for suicide has had moderate success in identifying students who are at high risk for suicide and other mental health problems, but concerns have been raised regarding the number of youngsters who are falsely identified as being at risk (Scott, Wilcox, et al., 2010).

In recent years, prevention efforts have also focused on providing cognitive–behavioral prevention to adolescents at risk for depression by virtue of having a parent with a history of a depressive disorder. In a randomized controlled study, a cognitive–behavioral prevention program demonstrated sustained effects as compared with a usual-care group in preventing the onset of depressive episodes in at-risk youths over a 3-year period (Beardslee et al., 2013). However, group differences were not found when parents were actively depressed at baseline. Other similar prevention programs have used family cognitive–behavioral interventions with 9- to 15-year-old children of parents with a history of MDD (Compas et al., 2011). Depressed parents and their children are taught a wide array of problem-solving and coping skills, including teaching children ways of coping with their parent's depression. The goals of the program are to educate families about depression, to increase awareness of the impact of stress and depression on functioning, to help families recognize and monitor stress, to facilitate the use of effective ways of coping with stress, and to improve parenting skills. In a randomized controlled study, children who participated in the preventive intervention were compared with those in a comparison group who received written information. Those who received the intervention showed significantly lower rates of MDD over a 2-year period, significantly lower rates of internalizing and externalizing symptoms at 18 months, and significantly lower rates of externalizing symptoms at 24 months. Marginal effects were found for reductions in parents' symptoms of depression at 18 and 24 months but not for episodes of MDD (Compas et al., 2011). Preventive programs

like these have the potential to protect unaffected children of depressed parents from developing the disorder and to improve outcomes for children with depression who are currently receiving treatment.

A high priority must be given to the development and continued refinement of identification, early intervention, and prevention efforts for youngsters at risk for depressive disorders (Barrera, Torres, & Munoz, 2007; Farrell & Barrett, 2007). The development of programs for preschool children with depression (Lenze, Pautsch, & Luby, 2011) and online and computer-based interactive programs for use in primary care, school, and other settings are examples of promising new prevention approaches (Gladstone et al., 2014; Spence et al., 2011).

## Section Summary

### Treatment of Depression

- Cognitive–behavioral therapy and interpersonal psychotherapy have had the most success in treating depression in young people.
- SSRIs have been recommended as the first line of drug treatment for children with depression, but concerns have been raised about their use.
- A high priority needs to be given to programs aimed at preventing depression in young people.

# BIPOLAR DISORDER (BP)

*In a sense, depression is a view of the world through a dark glass, and mania is that seen through a kaleidoscope—often brilliant but fractured.*

—K. R. Jamison (1997)

### BEN

### Extreme Mood Swings

Ben, age 14, was living in a residential treatment center. He had a history of moodiness, hyped-up activity, sleeplessness, and a sexual preoccupation with the girls in his class—he had even approached his teacher with offers of sexual intimacy. Ben's thoughts raced, his speech was rapid and fragmented, and he had wide mood swings. At the high extreme, Ben rarely slept, and he yelled, sang, and disturbed everyone—charging about the residence day and night with a seemingly endless supply of energy. He felt "absolutely terrific" at these times and thought he could fly. The low extreme found Ben curled up in a ball beneath a stack of blankets, a withdrawn and hopeless young man who expressed feelings of worthlessness and thoughts of suicide. (Based on authors' case material)

Ben displays the essential features of **bipolar disorder (BP):** a striking period of unusually and persistently elevated, expansive, or irritable mood, accompanied by increased goal-directed activity or energy, and alternating with or accompanied by one or more major depressive episodes. The two mood states associated with the manic phase of BP are elation and a profound sense of well-being (euphoria). However, these feelings can quickly change to anger and hostility if something interferes with the youngster's behavior. Since many youngsters with BP have simultaneous feelings of depression, they are easily reduced to tears. It is at once remarkable and almost inconceivable how a young person with this disorder such as Ben can be so manic, elated, energetic, and wild at one moment; so depressed and immobile the next; and at other times seemingly so normal (Geller & Luby, 1997).

Symptoms of BP in young people were recognized as early as 150 C.E., when the physician Aretaeus of Cappadocia described manic behavior in young men in puberty (Kotsopoulos, 1986). In modern times, BP was generally considered an adult illness, receiving very little attention in children and adolescents (Geller & DelBello, 2003). More recently, there has been upsurge of professional and public interest in children and adolescents with BP as reflected in increasing rates of research, diagnosis, treatment, and media coverage (Fristad & Algorta, 2013; Youngstrom & Algorta, 2014). For example, since 1994, the estimated number of youth outpatient office visits for BP has increased at least 40-fold, with about 90% of the youngsters receiving prescriptions for psychotropic medication during these visits (Moreno et al., 2007b). As we will discuss, along with the increased interest has come considerable controversy—BP in young people is difficult to identify because it occurs infrequently, shows extreme variability of clinical presentation within and across episodes, and overlaps in symptoms with more common childhood disorders such as ADHD and conduct problems (Youngstrom & Algorta, 2014).

Debate continues as to the appropriate criteria for diagnosing BP in children and adolescents (Carlson, 2011). At the center of the controversy is whether BP can be diagnosed in prepubertal children. Some clinicians avoid the use of this label entirely, and instead label young children who display unstable moods with the less stigmatizing categories of ADHD or depression. Others use the label of BP liberally in young children, often based solely on the presence of mood swings, irritability, and aggression, leading to concerns about overdiagnosis. Thus, clinicians presented with identical diagnostic information vary widely in their assessment of BP in children, from 0% risk to 100% risk (Jenkins et al., 2010). A focal point of this

debate is whether BP looks the same in young children as in adults. If it does, then mania in children should be diagnosed narrowly, with distinct episodes that are a clear departure from the youngster's usual behavior and functioning, and should have the same symptoms as those for adults (e.g., euphoria, grandiosity). If not, then a broader definition that includes early childhood onset of chronic emotion dysregulation with symptoms of ADHD, ODD, and other comorbidities, as well as severe irritability, extended outbursts of rage, and modified symptoms of mania would be sufficient to make the diagnosis (Carlson & Klein, 2014). How the BP label is used with young children has important implications for treatment, given the few available medications for stabilizing mood in children and the greater risks of using these medications for children than for adults (Ghaemi & Martin, 2007).

Young people who meet DSM-5 diagnostic criteria for BP display significant impairment in functioning, including previous hospitalization, MDD, treatment with medications, and co-occurring disruptive behavior and anxiety disorders. A history of psychotic symptoms and suicidal ideation and suicide attempts are also common (Axelson et al., 2006). They show severe and cyclical mood changes and outbursts. Thus, in its fully developed state, this condition is clearly different from a child's usual behavior (Carlson, 2002). During a manic episode, youths with BP may display intense symptoms, such as irritability and rage. Or they may show silly, giddy, overexcited, overtalkative behavior coupled with expansive, grandiose beliefs (e.g., a teen who feels she has a special connection to God). It is normal for children to pretend to have special powers or abilities, but a youngster with BP, during a manic episode, will actually believe he is the X-Man Wolverine and that he is indestructible and all-powerful. He might believe he can walk on water, control traffic, or jump off buildings without hurting himself—but he does not believe he might kill himself in the process.

Restlessness, agitation, and sleeplessness are also typical of youngsters with BP. Sexual disinhibition (like Ben's propositioning his teacher) may also occur when the youth becomes uncharacteristically preoccupied with sexual themes, sexually touching others, or "talking dirty." Youths with BP may experience unrealistic elevations in self-esteem (believing they are "the chosen one") and vast surges of energy; they may go with little or no sleep for days without feeling tired. They may be able to concentrate for hours on one activity that interests them, such as drawing or becoming engrossed in a mentally demanding fantasy game. At the same time, however, they may be highly distractible, constantly jumping from one thing to another (Geller & Luby, 1997).

The elated mood of youths with mania may (erroneously) give them the appearance of being happy and cheerful. Like Ben, they may say, "I feel absolutely terrific." It is difficult to recognize that a laughing, happy youngster also has a history of misery and distress. For this reason, evaluating a youth's current mood in relation to his or her developmental history is essential, particularly when there is an inconsistency between the child's elated mood and his or her history of trouble at home or school (Youngstrom, 2007).

Current research suggests that BP with an onset prior to age 18 is essentially the same disorder that occurs in adults, although possible differences in long-term outcomes and associated characteristics are not known (Carlson et al., 2004). Although not without problems, the diagnosis of BP can be made in children and adolescents using the same DSM-5 criteria used for adults. There are four primary types of BP: bipolar I disorder; bipolar II disorder; cyclothymic disorder; and other specified bipolar disorder. These different types are related to whether the youngster displays a manic or hypomanic episode. A *manic episode,* which is the hallmark feature of BP, involves a discrete period of a week or more during which the youngster displays an ongoing, pervasive, and unusually elevated or irritable mood and persistently increased goal-directed activity or energy. This episode is accompanied by the types of symptoms we have been describing such as an exaggerated self-esteem, a reduced need for sleep, racing thoughts, rapid and frenzied speech, attention to irrelevant details, increased activity, or overinvolvement in pleasurable but often reckless and risky behaviors. In addition, the youngster does not meet criteria for a depressive episode during the period of mood disturbance and increased energy and activity; the mood disturbance is not due to substance use or abuse or to a medical condition; and the disturbance causes significant impairment in usual activities or requires hospitalization in order to prevent the child or others from harm. A *hypomanic episode* has features that resemble a manic episode in quality but are less intense—the mood disturbance and increased activity or energy are less severe, of shorter duration, and produce less impairment in functioning than a manic episode.

A diagnosis of *bipolar I disorder* requires evidence for a manic episode and one or more major depressive episodes; a *bipolar II disorder* requires a hypomanic episode in combination with one or more major depressive episodes; a *cyclothymic disorder* describes children or adolescents who display numerous and persistent hypomanic and depressive symptoms for a year or more that cause considerable distress and impairment in functioning, but do not meet criteria for a manic episode or for a major depressive disorder

(Van Meter et al., 2013); and an *other specified bipolar disorder* describes individuals who display characteristic symptoms of BP that cause significant functional impairment but do not meet criteria for any of the other types of bipolar disorder. DSM-5 also includes a specifier of "*with mixed features,*" which can be used when a current manic or hypomanic episode includes subthreshold symptoms of depression or dysthymia or when an episode of MDD includes subthreshold symptoms of mania or hypomania.

Atypical symptom presentation makes it difficult to use DSM-5 criteria for BP to diagnose youngsters with mania (Youngstrom, 2007). Changes in mood, psychomotor agitation, and mental excitation are often volatile and erratic rather than persistent. Irritability, belligerence, and mixed manic–depressive features occur more frequently than euphoria. Unlike for adults, developmental limitations and the social environment place constraints on children's reckless behaviors, which typically involve school failure, fighting, dangerous play, and inappropriate sexual conduct. Thus, classic manic symptoms of grandiosity, psychomotor agitation, and reckless behavior must be differentiated from manic symptoms of common childhood disorders, such as ADHD and conduct problems, and from typical childhood behaviors, such as bragging, imaginary play, overactivity, and youthful blunders (American Academy of Child and Adolescent Psychiatry [AACAP], 2007c).

How are some of the more notable symptoms of mania expressed in youngsters with BP? When in a manic state, youngsters show great conviction about the correctness or importance of their ideas. Adolescents with BP may show grand delusions—illogical and strong beliefs that lead to poor judgment and impulsive behavior (Jamison, 1997). For example, they may badger their teachers about how to teach. This badgering may become so intense that teachers contact the parents, pleading with them to ask their children to cease. Youngsters with BP may intentionally fail subjects, acting on their illogical belief that children can choose what to pass or fail because they believe they are not being taught correctly. They may steal expensive items and be unresponsive to efforts by police or parents to explain that their actions are wrong and illegal. Although these youngsters know that stealing is illegal for others, they believe they are above the law. They may believe that they will achieve great fame, for example, as a brain surgeon, even though they are failing all of their classes at school. Similarly, a youngster with BP who is short, clumsy, and lacks any athletic ability may practice basketball with great fervor and strongly believe that he will become the next LeBron James. Another child may feel she has the ability to change the weather at will.

In contrast to youngsters with depression, who cannot fall asleep and may lie in bed for hours fretting and brooding, those with mania show high levels of activity at bedtime, spend very little time in bed, and require very little sleep. A child with mania might spend several hours at bedtime rearranging clothes in a dresser or closet, or an adolescent may wait until his or her parents are asleep and then sneak out of the house to go a party.

For children with mania, their words, thoughts, and actions occur in fast motion. Increased verbal production with puns, word plays, and incessant speech are common. At all ages, children with mania show *pressured speech*—they talk too much and too fast, change topics too quickly, and cannot be interrupted. They also have *racing thoughts* that they may describe in concrete terms—for example, by saying they can't do their schoolwork because their thoughts keep interrupting. In the words of one teen, "I wish I had a switch on my forehead so I could turn off my racing thoughts" (Geller & Luby, 1997). Like an adult with BP, a child with the disorder also shows a *flight of ideas,* which is an illogical jump from one idea to another. For example, in reply to the question, "Do you live in Los Angeles?" the child may reply, "Some people like to swim in the ocean. Do you have a dog?"

For children with mania of all ages, even slight changes in their surroundings can lead to significant distractibility. Heightened psychomotor agitation and goal-directed actions resemble normal activities carried out in excess, with a seemingly endless supply of energy. During a brief period of time, a manic youngster might draw several pictures, read a book, work on the computer, prepare a snack, make multiple phone calls, write a letter, and vacuum the house.

Accepting dares is common for youngsters with BP. In older adolescents, this may appear as a pattern of reckless driving that results in multiple tickets for speeding or driving under the influence. In preadolescents, it may be expressed as grandiose delusions of being able to jump out the window because they believe they can fly. They also may push the limits on usual childhood climbing on things, based on the strong belief that they are above the possibility of danger (Geller & Luby, 1997). In extreme cases, they may experience violent agitation with delusional thinking as well as visual and auditory hallucinations.

## Prevalence

Lifetime prevalence estimates of BP in community samples of youths 7 to 21 years of age range from about 0.5% to 2.5% worldwide (Merikangas et al., 2012; Van Meter, Moreira, & Youngstrom, 2011). In light

of the complicated presentation of symptoms and the difficulties in making an accurate diagnosis, it is possible that BP in young people is more common and occurs at a younger age than previously thought (Luby & Belden, 2006; Youngstrom, 2007).

The duration of manic symptoms in young people often does not meet the DSM-5's distinct 1-week duration requirement to be a manic episode. In an epidemiological study of 8- to 19-year-olds, it was found that recurrent episodes of mania or hypomania that met the DSM-5 criteria for episode duration were extremely rare (<0.3%), and restricted to 16- to 19-year-olds (Stringaris et al., 2010). Children with BP are also likely to present with rapid cycling episodes (at least four episodes of a mood disturbance over a 1-year period), with about 80% of children showing this course (Geller et al., 1995).

In light of these findings, the most common diagnoses are the milder bipolar II disorder, cyclothymic disorder, and other specified BP rather than bipolar I disorder (Lewinsohn, Klein, & Seeley, 1995). Children with a diagnosis of cyclothymic disorder or other specified BP are quite similar in terms of their current symptom severity and functional impairment to those with bipolar I disorder, suggesting that these disorders are on the same bipolar spectrum (Hafeman et al., 2013). It is of interest to note that studies of community samples have reported a stable rate of BP in young people over the past two decades, which is in contrast to the large increase in BP diagnoses in clinical samples over this period (Youngstrom & Algorta, 2014). This suggests a growing awareness and recognition of BP symptoms rather than an actual increase in rates, similar to what we described in Chapter 6 for increasing rates of ASD diagnoses.

Despite accounts of the onset of mania in children as young as 5 or 6 years old, the incidence of BP prior to puberty is extremely rare, but it increases during adolescence and is nearly as high as it is for adults (Lewinsohn, Klein, & Seeley, 2000). In sharp contrast to the effects of depression, BP affects boys and girls equally. However, symptoms may be expressed differently, with boys showing more manic moods and girls more depressed moods (Duax et al., 2007). In studies of youngsters with early-onset BP, boys seem to be affected more often than girls, especially when the age of onset is younger than 13 years. Rates of BP have not been found to differ by ethnicity or culture, but few studies have investigated this issue in children and adolescents (AACAP, 2007c). Given the many different definitions and methods that have been use to assess the prevalence of BP in young people, the most consistent results that emerge are that prevalence rates are higher in: clinic versus community samples; older versus younger participants; and samples that define BP more broadly and include participants with cyclothymic disorder and other specified BP (Youngstrom & Algorta, 2014).

## Comorbidity

Co-occurring disorders are extremely common in youngsters with BP. They include anxiety disorders, ADHD, ODD, CD, substance-use problems, and suicidal ideation and suicide attempts (Goldstein et al., 2012; Hauser, Galling, & Correll, 2013; Sala et al., 2010). These disorders share many overlapping symptoms with BP, possibly related to shared underlying processes for what are currently assumed to be distinct disorders (Youngstrom & Algorta, 2014). In addition to these disorders, sleep disturbances, disrupted relationships with family and peers, risk-taking behaviors, and medical problems such as overweight status or obesity, cardiovascular and metabolic disorders, epilepsy, and migraine headaches are also common in youngsters with BP, which further complicates how it is managed (Goldstein et al., 2013; Sheffer & Linden, 2007; Youngstrom & Algorta, 2014).

More than 60% of youngsters with BP display one or more comorbid anxiety disorders, and as many as 50% have two or more anxiety disorders. In addition to their frequency, comorbid anxiety disorders have been found to adversely affect the course of BP in young people, suggesting the need for early recognition and treatment (Sala et al. 2014). Many youngsters with BP display co-occurring symptoms of ADHD, such as poor judgment, distractibility, inattention, irritability, hyperactivity, anger, poor impulse control, demanding behaviors, and the tendency to jump from one topic or activity to another. For youngsters first seen because of symptoms of BP, about 60% to 90% of prepubertal children and 30% of adolescents also have ADHD (Geller & Luby, 1997).

ODD and CD occur in as many as 80% of children and adolescents with BP (Leibenluft & Rich, 2008). Symptoms of grandiosity, mania, and poor judgment in BP may be confused with symptoms of conduct problems. For example, one 11-year-old boy with BP, who believed he would be a famous rock star, stole several hundred dollars' worth of music and was totally unaffected when questioned by the police. CD overlaps with BP on symptoms such as running away, driving under the influence, substance abuse, sexual promiscuity, and stealing. Similarly, the flight of ideas and/or pressured speech associated with mania may be mistaken for a language disorder (Carlson, 2002; Geller & Luby, 1997).

In trying to differentiate the symptoms of BP from those of comorbid conditions, if symptoms occur or

worsen only during a mood episode, they may indicate mania. However, if they are chronic, occur between episodes, and represent the child's typical level of behavior, the presentation would be more consistent with ODD, ADHD, or anxiety disorder (Leibenluft & Rich, 2008).

## Onset, Course, and Outcome

About 60% of patients with BP experience their first episode prior to the age of 19 years, with a peak age of onset between 15 and 19 years (Merikangas et al., 2007; Post et al., 2008). Although BP in preschool children may be identified using age-adjusted mania symptoms (Luby, Tandon, & Belden, 2009), onset prior to age 10 is extremely rare. Younger children in clinical samples display higher overall levels of manic symptoms, whereas adolescents display higher rates of depressive symptoms. However, the core symptoms of depression and mania appear to be consistent across age groups (Youngstrom & Algorta, 2014). Youngsters with BP may first present with either depressive or manic episodes, although most report that their first mood episode was major depression. This is consistent with the reported high rates of switching from depression to mania (Geller & Luby, 1997).

Risk factors for eventual mania include a major depressive episode (characterized by rapid onset, psychomotor retardation, disrupted sleep patterns, and psychotic features) and a family history of mood disorders, especially BP (AACAP, 2007c). When a young person presents with a first episode of obvious mania, further manic episodes will very likely follow. Bipolar episodes are generally shorter than major depressive episodes, lasting from 4 to 6 months if left untreated. About 70% of adolescents recover from their initial episode within 6 months, but 50% will have at least one recurrent episode (Birmaher et al., 2006).

Adolescents with mania often have complex presentations that include psychotic symptoms such as hallucinations, paranoia, and thought disorder. They also have unstable moods with mixed manic and depressive features, and severe deterioration in behavior. These diverse forms of presentation may result in an underdiagnosis of BP in teens and may be misdiagnosed as schizophrenia.

Because it is difficult to recognize symptoms of BP in young people, the symptoms are commonly noticed well before a youngster is treated or hospitalized, but they are not labeled as BP (Youngstrom et al., 2005). A look back at the histories of adults with BP symptoms often shows that mood swings began around puberty; however, there is frequently a 5- to 10-year lag between the onset of symptoms and display of the disorder serious enough to be recognized and treated (Carlson, 1994).

The early onset and the course of BP make it chronic and resistant to treatment, with a poor long-term prognosis similar to that in adults (AACAP, 2007c). In a 5-year prospective follow-up study of adolescents with BP, nearly 50% had a relapsing course or never achieved complete remission (Strober et al., 1995). As compared with adults, adolescents with BP may have a more prolonged early course and a poorer response to treatment. However, long-term prognosis appears to be similar to that for adults, with most patients continuing to experience significant symptoms and functional impairment (DelBello et al., 2007).

## Causes

**JESSI**

### Runs in the Family

"Jessi's father had been an alcoholic and a manic depressive," says her mother, "probably since he was an adolescent. He died of dehydration that occurred during a manic episode. His illness had been a mystery to us. Growing up, Jessi knew her father was ill, and when she was older, she began to worry about what his sickness might mean for her. I worried too," says Jessi's mother. "By the time Jessi was in her early twenties, something was clearly wrong. At first, I noticed only that she had become less reliable—forgetting things, arriving late, and occasionally missing appointments with me. Frequently, she complained of fatigue, a cold, flu, or a stomachache. Increasingly, her responses were brief, perfunctory. Though we didn't know it then, Jessi was experiencing a huge mood shift that was taking months to complete itself. Jessi had MDD, without the manic swings of the bipolar disorder her father had suffered from."

Adapted from Dowling, 1992.

Relatively few studies have examined the causes of BP in young people, although research with adults, and more recently with children, indicates that BP is one of the most heritable forms of mental disorder (McInnes et al., 2003). Findings from family and gene studies indicate that BP is the result of a genetic vulnerability combined with environmental factors, such as life stress or a negative family climate. When an identical twin has BP, there is only a 65% chance that the other twin will have it too, suggesting that in addition to genes, other factors are important. Although BP can affect anyone, it has definitely been shown to run in families (Oquendo et al., 2013). Offspring of mothers with BP are more likely to exhibit greater physiological dysregulation in response to stress than controls at

as young as 6 months of age; this is a possible early vulnerability factor for later mood disorders (Johnson et al., 2014). If one or both parents have BP, the chances are about 5 times greater that their children will also develop BP or often, like Jessi, another recurrent mood disorder (Hodgins et al., 2002; Mesman et al., 2013).

Besides mood disorder, children at risk for BP by virtue of having parents with the disorder also display a wide range of psychopathology, particularly conduct problems and ADHD, as well as social and academic difficulties (Singh et al., 2007). Relatives of youngsters with BP also have a higher incidence of the disorder. Family incidence and risk for a broad range of psychiatric problems are highest in cases of early-onset BP, with lifetime prevalence rates of about 15% in first-degree relatives (AACAP, 2007c; Rende et al., 2007). This rate is 15 times greater than the prevalence of the disorder in the general population.

Increasing evidence suggests that BP arises from multiple genes, and some studies have identified several chromosomal regions and susceptibility genes (Alsabban, Rivera, & McGuffin, 2011; McInnis et al., 2003). As we have found for other disorders, specific genes that have been identified contribute only a small amount to the risk for BP. In addition, several of these genes have also been identified for youths with depression, anxiety, ADHD, or psychosis. There is likely a complex mode of inheritance rather than a single dominant gene. Individuals with a genetic predisposition do not necessarily develop BP, since environmental factors also play an important role in determining how genes are expressed (Geller & Luby, 1997).

A number of nonspecific risk factors that raise the risk for BP include poor maternal health or nutrition during pregnancy, substance use during pregnancy, a stressful early environment, exposure to traumatic events, and parental mood disorders (Youngstrom & Algorta, 2014). The ways in which environmental factors play a role are not well understood. However, one study suggests that parental BP may create a negative family climate, including problem-solving and communication deficits, which predict family conflict, which in turn predicts childhood BP (Du Rocher et al., 2008).

Brain scans of children identified as being at risk for BP that were taken before and after the onset of a manic episode have shown changes in the brain that reflect a pattern of emotion dysregulation in general, rather than one that is specific to BP onset (Gogtay et al., 2007). Generally, mood fluctuations in BP have been related to abnormalities in the structure and function of the amygdala, prefrontal and anterior cingulate cortex, hippocampus, thalamus, and basal ganglia, but findings have not always been consistent with respect to the types of abnormalities (Garrett & Chang, 2008; Gogtay et al., 2007). Such inconsistencies may be related to ongoing brain changes that are occurring in young people and the point in development at which brain structure and function are assessed.

Some studies have found that BP in adolescents is related to reduced volumes of the amygdala and hippocampus (Beardon et al., 2007; Blumberg et al., 2003). As you may recall, we discussed the importance of the amygdala for recognizing and regulating emotions in relation to depression. Research has found that youngsters with BP misread neutral facial expressions as hostile and in doing so show heightened activation of the amygdala and its connectivity to other parts of the brain involved in processing facial information (Perlman et al., 2013; Rich et al., 2008). These and other findings suggest that youths at risk for and those with BP may display unique neural correlates and deficits in facial emotion processing and dysregulation in brain regions associated with emotion regulation (Brotman et al., 2010; Garrett et al., 2012). Such deficits may be related to the poor social skills, aggression, and irritability that characterize youngsters with BP. In addition, adolescents with BP also show abnormal activation of prefrontal and subcortical areas of the brain during the anticipation of and response to monetary gains and loss, which suggest problems in reward processing, motivation, and goal pursuit (Singh et al., 2012).

## Treatment

Treatment of BP in children and adolescents is receiving increasing attention. Although there is currently no cure for BP, in most cases treatment can stabilize mood and allow for management and control of symptoms. Treatment of BP generally requires a multimodal plan that includes close monitoring of symptoms, educating the patient and the family about the illness, matching treatments to individuals, administering medications such as lithium or atypical antipsychotics to stabilize mood, and performing psychotherapeutic interventions to address the youngster's symptoms and related psychosocial impairments (AACAP, 2007c; Kowatch et al., 2005). The general goals of treatment are to decrease the child's symptoms and to prevent relapse, while also reducing long-term illness and enhancing the youngster's normal health and development (Geller & Delbello, 2008).

### *Medications*

Multiple medications have been used to treat youngsters with BP (Goldstein, Sassi, & Diler, 2012). The FDA has approved lithium for use in children as young as 12 years of age. However, there are currently no drugs that are FDA-approved for the treatment of BP

in children younger than this (AACAP, 2007c). Medications are typically used to address manic or mixed symptoms and depressive symptoms or to prevent relapse. Clinical trials of medication have had some success, and controlled studies of medication treatment for adolescents with BP are rapidly increasing in number, although few studies have evaluated effects in children younger than age 10 years of age (Goldstein et al., 2012; Liu et al., 2011). Until very recently, recommended treatments were based on findings with adults; however, as we saw with tricyclic antidepressants, such an extrapolation may not be warranted (Geller et al., 1998). Hence, mood-stabilizing medications need to be used with caution and conservatively with young people with BP, particularly in those who do not fit the classic presentation of symptoms seen in adults with bipolar I disorder; these youngsters may constitute as many as 75% of cases of BP (Horst, 2009; Merikangas & Pato, 2009).

Based on its use with adults with BP, lithium has been the agent of first choice in the treatment of youths with BP, and its efficacy has been demonstrated in a number of controlled studies (Goldstein et al., 2012). Lithium is a common salt that is widely present in the natural environment—for example, in drinking water—usually in amounts too small to have any effects. However, the side effects of therapeutic doses of lithium can be serious, especially when used in combination with other medications; side effects may include toxicity (poisoning), renal and thyroid problems, and substantial weight gain (Gracious et al., 2004). It can be given to young people when used with the same safety precautions and similar careful monitoring used for adults. However, lithium cannot be given to children in chaotic families or to children who are unable to keep the multiple appointments needed for monitoring potentially dangerous side effects (Carlson, 1994; Geller & Luby, 1997). In addition, one study found that only 35% of adolescents with BP reported full adherence with the medication regimen (DelBello et al., 2007). Some studies have found atypical antipsychotic agents to be more efficacious than lithium in treating acute manic and mixed episodes in young people with BP, suggesting that these medications may be a preferred option for many youths with BP (Goldstein et al., 2012). However, they too have many metabolic side effects. In addition, adjunctive medications to treat secondary symptoms such as ADHD, depression, and anxiety have also been used (AACAP, 2007c; Sanchez & Soares, 2011).

### *Psychosocial Treatments*

A focus on biological causes and pharmacological interventions for BP has resulted in a relative lack of attention to psychosocial treatments, although this situation is changing (Fristad & MacPherson, 2014). There is also a pressing need for studies on prevention, targeted interventions to delay or prevent progression to full manic or depressive episodes, and approaches that focus on possible environmental moderators of risk (Youngstrom & Algorta, 2014).

Medications may decrease symptoms of BP, but they do not help with the associated functional impairments or preexisting or co-occurring substance-use disorders, learning and behavior problems, and family- and peer-related issues. Nonadherence to medication regimens has been shown to be a major contributor to relapse. Thus, the family must be educated about the negative effects of nonadherence and to recognize possible symptoms of relapse. Psychosocial interventions focus on providing information to the child and family about the disorder, symptoms and course, possible impact on family functioning, and heritability of the disorder. Youths and parents are also taught ways of coping with symptoms and preventing relapse by using problem-solving, communication, emotion regulation, and cognitive—behavioral skills (Fristad, Goldberg Arnold, & Leffler, 2011; Fristad & MacPherson, 2014). Controlled research on psychosocial treatments for youngsters with BP is beginning to appear (Fristad et al., 2009; Goldstein et al., 2007; Miklowitz et al., 2011). This has resulted in several promising new early interventions for high-risk youths using family-focused therapy, CBT, and combined treatments to reduce symptoms of BP (Fristad & MacPherson, 2014; Miklowitz et al., 2013). Further efforts to identify young children at risk for developing BP are needed to enhance opportunities for both psychosocial and pharmacological preventive interventions (Howes & Falkenberg, 2011; Luby & Navsaria, 2010).

## Section Summary

### Bipolar Disorder

- A recent surge in interest in the diagnosis of bipolar disorder (BP) in children and adolescents has generated considerable controversy surrounding difficulties in identifying the disorder in young people.
- Youngsters with BP show periods of abnormally and persistently elevated, expansive, and/or irritable mood.
- They may display symptoms such as an inflated self-esteem, decreased need for sleep, pressured speech, flight of ideas, distractibility, and reckless behavior.
- BP is far less common than MDD in young people, with lifetime prevalence estimates of 0.5% to 2.5% worldwide.
- BP has a peak age at onset in late adolescence and, unlike depression, affects males and females about equally.

(*continues*)

## Section Summary (*continued*)

- The most common accompanying disorders are ADHD, anxiety disorders, conduct problems, and substance abuse.
- Very few studies have examined the causes of BP in children and adolescents. Family and gene studies with adults indicate that BP is the result of a genetic vulnerability in combination with environmental factors, such as life stress or disturbances in the family.
- Brain-imaging studies of youngsters with BP point to abnormalities in regions of the brain involved in emotion regulation, including the amygdala and anterior cingulate cortex.
- BP in young people requires a multimodal treatment plan with education of the patient and the family about the illness, medication, and psychosocial interventions to address the youngster's symptoms and related psychosocial impairments.

# Study Resources

### SECTION SUMMARIES



### KEY TERMS

# 11

# Anxiety and Obsessive—Compulsive Disorders

*It is hard to be brave, when you're only a very small animal.*

—Piglet (Pooh's Little Instruction Book, 1995)

## CHAPTER PREVIEW

***Separation Anxiety***: Brad is terrified of being separated from his mother. He follows her around the house constantly, always needing to know where she is.

***Social Anxiety***: Li-Ming is very preoccupied with what others think of her. She doesn't interact with anyone at school, and feels completely isolated.

***Panic Disorder***: Claudia describes her sudden attack of overwhelming anxiety. "My heart started pumping so fast I thought it would explode. I thought I was going to die."

***Generalized Anxiety***: Jared "worries about everything"—how he is doing in school, events in the news, and family finances.

***Obsessive–Compulsive Disorder***: Georgina can't stop thinking about not being able to sleep. Every night before bedtime she goes through the same routine of counting and grouping all the clothes and shoes in her bedroom closet and opening and closing the closet door.

ALL CHILDREN EXPERIENCE FEAR, worry, or anxiety as a normal part of growing up, but each child in our examples suffers from an anxiety or related disorder that is excessive and debilitating. An anxiety disorder is one of the most common mental health problems in young people, with lifetime prevalence estimates between 8% and 30% (Kessler et al., 2012a; Merikangas et al., 2010). Estimates vary widely with the child's age, type of anxiety disorder, and whether impaired functioning is part of the diagnosis. Conservatively, at least one child in every elementary school classroom is likely to have an anxiety disorder (Cartwright-Hatton, McNicol, & Doubleday, 2006). Despite their early onset, high frequency, persistence, and associated problems, anxiety disorders in children often go unnoticed and untreated (Gregory et al., 2007). Fewer than 20% of youngsters with anxiety disorders receive services for their problem, as compared with about 45% to 60% of those with conduct or attention disorders (Merikangas et al., 2011). This may be due to the frequent occurrence of fears and anxiety during normal development, the invisible nature of many symptoms (e.g., a knot in the stomach), and the fact that anxiety is not nearly as damaging to other people or property as are conduct problems (Higa-McMillan, Francis, & Chorpita, 2014).

For a long time, anxiety in children was thought to be a mild and transitory disturbance that would fade over time with normal life experiences. However, we now know that many children who experience anxiety display impairments in their school, social, and family/home functioning and will continue to display anxiety and other problems into adolescence and adulthood (Bittner et al., 2007; Langley et al., 2014). Having an anxiety disorder in childhood or adolescence is also one of the strongest predictors of most other later mental disorders (Kessler et al., 2012c). Although isolated symptoms of fear and anxiety are usually short-lived, anxiety disorders have a more chronic and stable course (Carballo et al., 2010). In fact, nearly half of those affected have an illness duration of 8 years or longer (Keller et al., 1992), and parents' reports of their child's anxiety symptoms predict anxiety disorders 24 years later (Reef et al., 2010). The societal costs for clinically anxious youths are also substantial, with estimated costs (e.g., health care, child care, missed work or school days) about 20 times higher for families with an anxious child versus those from the general population (Bodden, Dirksen, & Bögels, 2008). Thus, anxiety disorders in children are common, distressing, long-lasting, and costly (Rapee, Schneiring, & Hudson, 2009).

We begin our discussion with anxiety disorders, the primary focus of this chapter. We also discuss obsessive–compulsive disorder (OCD), a closely related disorder that was considered to be one of the anxiety disorders in previous versions of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM). OCD is now included in a separate chapter of DSM-5, with related disorders such as hoarding, hair-pulling, skin-picking, and body dysmorphic disorder (i.e., perceived defects or flaws in physical appearance). As we shall discuss, there is a close relationship between the anxiety disorders and some of these disorders (especially OCD), with respect to their behavioral and cognitive symptoms and other features.

## DESCRIPTION OF ANXIETY DISORDERS

**Anxiety** is a mood state characterized by strong negative emotion and bodily symptoms of tension in which the child apprehensively anticipates future danger or misfortune (Barlow, 2002). This definition captures two key features of anxiety—strong negative emotion and an element of fear. Children who experience excessive and debilitating anxieties are said to have **anxiety disorders.** These disorders occur in many forms. Some children, like Brad, feel anxious whenever they are separated from their mother or are away from home. Others feel anxious only in certain situations, such as when they have to travel by airplane or, like Li-Ming, when they have to give a talk in class. Some youngsters, like Claudia, have unpredictable bouts of such sudden and intense anxiety that they become terrified and immobilized. Others, like Jared, worry about almost everything and feel anxious most of the time for no apparent reason. Some children, like Georgina, experience repeated, intrusive, and unwanted thoughts that produce anxiety, and they spend hours in ritualized behavior in an effort to alleviate that anxiety.

Many youngsters with anxiety disorders suffer from more than one type, either simultaneously or at separate times during their development (Costello, Egger, & Angold, 2005b). In view of the substantial overlap among these disorders, we begin this chapter by discussing the general features and mechanisms of anxiety that apply across all types. The common occurrence of fears and anxieties in childhood and adolescence requires that we also consider the role of these emotions in normal development. We then examine each anxiety disorder and what makes it unique.

## Experiencing Anxiety

> When Isabella saw a dog running loose in front of her house, she became pale, sweaty, cold, and trembly. Her thoughts raced so fast that she couldn't think. She froze. Her heart pounded, she felt tense, and she found it difficult to breathe.

Isabella is experiencing anxiety in response to an event she sees as potentially threatening or dangerous. As humans, we are programmed to detect and react to signs of anxiety in ourselves and in others. In fact, anxiety is both expected and normal at certain ages and in certain situations. One-year-old infants become distressed when separated from their mothers, and almost all young children have short-lived specific fears—of the dark, for example. The child's world can be a strange and menacing place, full of unknown dangers—some real, others imagined. Although no one likes to feel anxious, not feeling anxious when the situation calls for it is far worse.

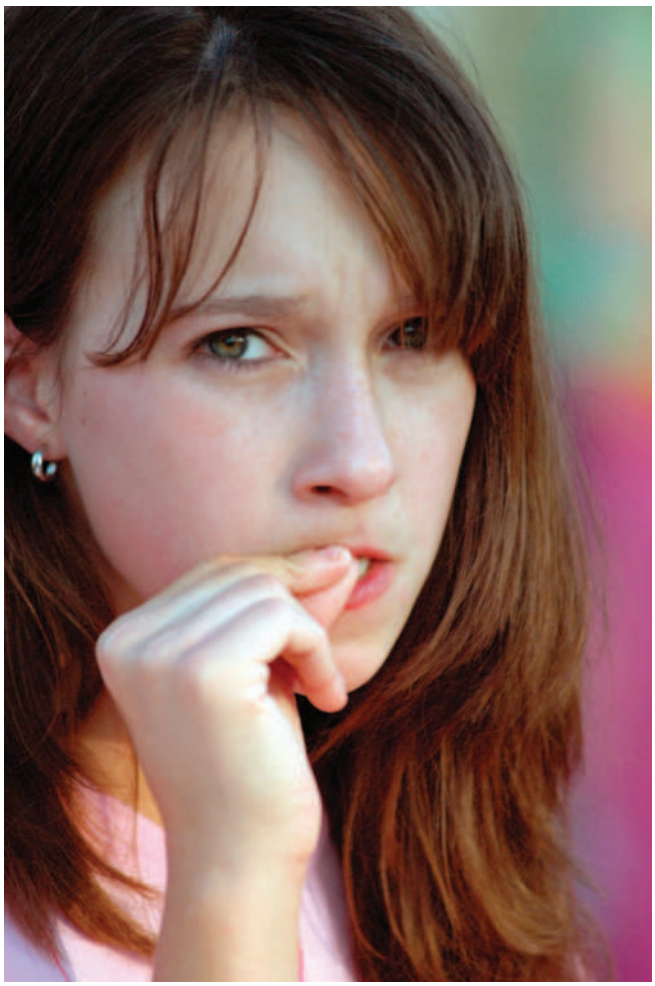
Anxiety often hits us when we do something important, and in moderate doses it helps us think and act more effectively. You will probably be better prepared for your next exam if you're just a little bit nervous about taking it. Similarly, some anxiety may help a child prepare harder for an upcoming oral report or athletic event. In this sense, anxiety is an adaptive emotion that readies children both physically and psychologically for coping with people, objects, or events that could be dangerous to their safety or well-being.

Although some anxiety is good, too much is not. Excessive, uncontrollable anxiety can be debilitating. A child may fail a test because she spends too much time thinking about how awful it would be to fail, making it nearly impossible to think about anything else (e.g., how to solve a math problem). In children with anxiety disorders, this normally useful emotion works against them.

When children experience fears beyond a certain age, in situations that pose no real threat or danger, and to an extent that seriously interferes with daily activities, anxiety is a serious problem. Even if the child knows there is little to be afraid of, he or she is still terrified and does everything possible to escape or avoid the situation. This pattern of self-defeating behavior, known as the **neurotic paradox** (Mowrer, 1950), can become self-perpetuating—much like Sisyphus repeatedly pushing the rock up the hill, only to have it roll back down each time.

First and foremost, anxiety involves an immediate reaction to *perceived* danger or threat—a reaction known as the **fight/flight response.** All of its effects are aimed at escaping potential harm, either by confronting the source of danger (fight) or by evading it (flight). If you look up to see a grand piano about to fall in your direction and experience no anxiety whatsoever, you will pay serious consequences. To avoid such a fate, your fight/flight response would kick into overdrive and you would jump out of harm's way.

Think of a recent situation that made you anxious. What was it about the situation that made you anxious? What physical symptoms did you notice? What were you thinking? What did you do? Describing what it's like to be anxious is not easy, because anxiety is a complex reaction with many symptoms, as shown in Table 11.1.



Robert W. Ginn/PhotoEdit

Youngsters with anxiety experience strong negative emotion and physical tension, and anticipate future danger.

**TABLE 11.1 | The Many Symptoms of Anxiety**

| | | |
|---|---|---|
| **Physical** | | |
| Increased heart rate | Dizziness | Blushing |
| Fatigue | Blurred vision | Vomiting |
| Increased respiration | Dry mouth | Numbness |
| Nausea | Muscle tension | Sweating |
| Stomach upset | Heart palpitation | |
| **Cognitive** | | |
| Thoughts of being scared or hurt | Thoughts of incompetence or inadequacy | Thoughts of bodily injury |
| Thoughts or images of monsters or wild animals | Difficulty concentrating | Images of harm to loved ones |
| Self-deprecatory or self-critical thoughts | Blanking out or forgetfulness | Thoughts of going crazy |
| | Thoughts of appearing foolish | Thoughts of contamination |
| **Behavioral** | | |
| Avoidance | Trembling lip | Avoidance of eye contact |
| Crying or screaming | Swallowing | Physical proximity |
| Nail biting | Immobility | Clenched jaw |
| Trembling voice | Twitching | Fidgeting |
| Stuttering | Thumb sucking | |

Based on Fears and Anxieties, by B. A. Barrios and D. P. Hartmann, 1997, p. 235. In E. J. Mash and L. G. Terdal (Eds.), Assessment of Childhood Disorders, 3rd ed.

How many of these symptoms did you experience? What do these many symptoms have in common?

The symptoms of anxiety are expressed through three interrelated response systems: the *physical system*, the *cognitive system*, and the *behavioral system*. It is essential to know how the three sets of symptoms work, since more than one may be evident in different children with the same anxiety disorder. Also, as we will discuss, different response systems are more dominant in certain anxiety disorders. Let's take a closer look at how each response system works.

### Physical System

When a person perceives or anticipates danger, the brain sends messages to the sympathetic nervous system, which produces the fight/flight response. The activation of this system produces many important chemical and physical effects that mobilize the body for action:

- *Chemical effects*. Adrenaline and noradrenaline are released from the adrenal glands.
- *Cardiovascular effects*. Heart rate and strength of the heart beat increase, readying the body for action by speeding up blood flow and improving delivery of oxygen to the tissues.
- *Respiratory effects*. Speed and depth of breathing increase, which brings oxygen to the tissues and removes waste. This may produce feelings of breathlessness, choking or smothering, or chest pains.
- *Sweat gland effects*. Sweating increases, which cools the body and makes the skin slippery.
- *Other physical effects*. The pupils widen to let in more light, which may lead to blurred vision or spots in front of the eyes. Salivation decreases, resulting in a dry mouth. Decreased activity in the digestive system may lead to nausea and a heavy feeling in the stomach. Muscles tense in readiness for fight or flight, leading to subjective feelings of tension, aches and pains, and trembling.

These physical symptoms are familiar signs of anxiety. Overall, the fight/flight response produces general activation of the entire metabolism. As a result, the individual may feel hot and flushed and, because this activation takes a lot of energy, he or she feels tired and drained afterward.

### Cognitive System

Since the main purpose of the fight/flight system is to signal possible danger, its activation produces an immediate search for a potential threat. For children with anxiety disorders, it is difficult to focus on everyday tasks because their attention is consumed by a constant search for threat or danger. When these children can't find proof of danger, they may turn their search inward: "If nothing is out there to make me feel anxious, then something must be wrong with me." Or they may distort the situation: "Even though I can't find it, I know there's something to be afraid of." Or they may do both. Children with anxiety disorders will invent explanations for their anxiety: "I must be a real jerk." "Everyone will think I'm a dummy if I say something." "Even though I can't see them, there are germs all over the place." Activation of the cognitive system often leads to subjective feelings of apprehension, nervousness, difficulty concentrating, and panic.

### Behavioral System

The overwhelming urges that accompany the fight/flight response are aggression and a desire to escape

the threatening situation, but social constraints may prevent fulfilling either impulse. For example, just before a final exam you may feel like attacking your professor or not showing up at all, but fortunately for your professor and your need to pass the course, you are likely to inhibit these urges! However, they may show up as foot tapping, fidgeting, or irritability (consider the number of teeth marks in pencils) or as escape or avoidance by getting a doctor's note, requesting a deferral, or even faking illness. Unfortunately, avoidance perpetuates anxiety, despite the temporary feeling of relief. Avoidance behaviors are negatively reinforced; that is, they are strengthened when they are followed by a rapid reduction in anxiety. As a result, each time a child is confronted with an anxiety-producing situation, the faster she or he gets out of it, the faster the anxiety drops off—so the more the child avoids such situations. As children with anxiety disorders engage in more and more avoidance, carrying out everyday activities becomes exceedingly difficult.

**CHANTELLE**

### The Terror of Being Home Alone

When Chantelle, age 14, realized she was at home alone, she was terrified. Her thoughts raced so fast it was impossible to think clearly. She forgot all the right things to do. Her heart pounded and she tensed up. She felt like she couldn't breathe, and she began to sob. She wanted to run but felt completely immobilized. (Based on authors' case material.)

Chantelle's reactions show how the three response systems of anxiety interact and feed off one another. Physically, Chantelle's heart pounded, she tensed, and she had difficulty breathing. Cognitively, she could not think clearly. Behaviorally, she was completely immobilized.

## Anxiety versus Fear and Panic

It is important to distinguish anxiety from two closely related emotions—fear and panic. **Fear** is an immediate alarm reaction to current danger or life-threatening emergencies. Although fear and anxiety have much in common, the fear reaction differs both psychologically and biologically from the emotion of anxiety. Fear is a *present-oriented* emotional reaction to current danger marked by a strong escape tendency and an all-out surge in the sympathetic nervous system. The overriding message is alarm: "If I don't do something right now, I might not make it at all." In contrast, anxiety is a *future-oriented* emotion characterized by feelings of apprehension and lack of control over upcoming events that might be threatening. Fear and anxiety both warn of danger or distress. However, only anxiety is frequently felt when no danger is actually present (Barlow, 2002).

**Panic** is a group of physical symptoms of the fight/flight response that unexpectedly occur in the absence of any obvious threat or danger. With no explanation for physical symptoms such as a pounding heart, the child may invent one: "I'm dying." The sensations themselves can feel threatening and may trigger further fear, apprehension, anxiety, and panic (Barlow, 2002).

## Normal Fears, Anxieties, Worries, and Rituals

Since fear and anxiety in moderate doses are adaptive, it is not surprising that emotions and rituals that increase feelings of control are common during childhood and adolescence. It is only when the emotions and rituals become excessive, occur in a developmentally inappropriate context, or lead to impairment in functioning such as an inability to go to school, make friends, complete academic tasks, or meet other developmental goals that they are of concern.

### *Normal Fears*

Since young people and their environments constantly change, fears that are normal at one age can be debilitating a few years later. For example, fear of strangers may serve a protective function for infants and young children, but when it persists beyond a certain age it can seriously interfere with the development of peer relations (Brooker et al., 2013). Whether or not a specific fear is normal also depends on its effect on the child and how long it lasts. If a fear has little impact on the child's daily life or lasts only a few weeks, it is likely a part of normal development.

The number and types of common childhood fears change over time, with a general age-related decline in number (Gullone, 1999). Even so, specific fears are common in older children, and many teens report that their fears cause them considerable distress and significantly interfere with daily activities (Ollendick & King, 1994). Girls tend to have more fears than boys at almost every age; they also rate themselves as more fearful and report fears that are more intense and disabling than do boys. Although fears show a general decline with age, some, such as school-related fears,

remain stable; others, such as social fears, may increase (Muris, 2007). Common fears and anxieties of infants, children, and adolescents are shown in Table 11.2. Also shown are possible relevant symptoms and corresponding DSM-5 anxiety disorders that may develop in relation to these symptoms.

**TABLE 11.2** | **Common Fears and Anxieties of Infancy, Childhood, and Adolescence; Possible Symptoms; and Corresponding DSM-5 Diagnoses**

| Developmental Period | Age | Common Fears and Anxieties | Possible Symptoms | Corresponding DSM-5 Anxiety Disorder |
|---|---|---|---|---|
| **Early Infancy** | Within first weeks | Loss of physical support, loss of physical contact with caregiver | — | — |
| | 0–6 months | Intense sensory stimuli (loud noises) | — | — |
| **Late Infancy** | 6–8 months | Shyness/anxiety with stranger, sudden, unexpected, or looming objects | — | Separation anxiety disorder |
| **Toddlerhood** | 12–18 months | Separation from parent, injury, toileting, strangers | Sleep disturbances, nocturnal panic attacks, oppositional defiant behavior | Separation anxiety disorder, panic attacks |
| | 2–3 years | Fears of thunder and lightning, fire, water, darkness, nightmares | Crying, clinging, withdrawal, freezing, avoidance of salient stimuli (e.g., turning the light on), night terrors, enuresis | Specific phobias (natural environment), panic attacks |
| | | Fears of animals | — | Specific phobias (animal) |
| **Early Childhood** | 4–5 years | Separation from parents, fear of death or dead people | Excessive need for reassurance | Separation anxiety disorder, generalized anxiety disorder, panic attacks |
| **Primary/Elementary School Age** | 5–7 years | Fear of specific objects (animals, monsters, ghosts) | — | Specific phobias |
| | | Fear of germs or of getting a serious illness | — | Obsessive–compulsive disorder (OCD) |
| | | Fear of natural disasters, fear of traumatic events (e.g., getting burned, being hit by a car or truck) | — | Specific phobias (natural environment), acute stress disorder, post-traumatic stress disorder, generalized anxiety disorder |
| | 5–11 years | School anxiety, performance anxiety, physical appearance, social concerns | Withdrawal, timidity, extreme shyness with unfamiliar adults and peers, feelings of shame | Social anxiety disorder (social phobia) |
| **Adolescence** | 12–18 years | Personal relations, rejection from peers, personal appearance, future, natural disasters, safety | Fear of negative evaluation | Social anxiety disorder (social phobia) |

Based on Beesdo, Knappe, & Pine, 2009.

Jacqueline Veissid/Photodisc/Getty Images

All children experience some fear, anxiety, and worry as a normal part of growing up.

### Normal Anxieties

Like fears, anxieties are very common during childhood and adolescence. Various types of anxiety are evident by age 4 (Eley, Lichenstein, & Moffitt, 2003), and about 25% of parents report that their child is too nervous, fearful, or anxious (Achenbach, 1991a). The most frequent symptoms in samples of children with normal anxieties are separation anxiety, test anxiety, overconcern about competence, excessive need for reassurance, and anxiety about harm to a parent (Barrios & Hartmann, 1997).

Younger children generally experience more anxiety symptoms than do older children, primarily about separation from parents. Girls display more anxiety than boys, but they generally experience similar types of symptoms. Although some specific anxieties decrease with age, such as separation anxiety and anxiety about school, nervous and anxious symptoms may not show the age-related decline observed for many specific fears (Hale et al., 2008). Anxious symptoms may reflect a stable trait that predisposes children to develop excessive fears related to their stage of development. Thus, the disposition to be anxious may remain stable over time, even though the objects of children's fears change.

### Normal Worries

If worrying about the future is so unproductive, why do we do so much of it? Part of the reason seems to be that the process of worry—thinking about all possible negative outcomes—serves an extremely useful function in normal development. In moderate doses, worry can help children prepare for the future—for example, by checking their homework before they hand it in or by rehearsing for an upcoming class play. Worry is a central feature of anxiety, and anxiety is related to the number of children's worries and to their intensity (Silverman, La Greca, & Wasserstein, 1995). Children of all ages worry, but the forms and expressions change. Older children report a greater variety and complexity of worries and are better able to describe them than are younger children (Chorpita et al., 1997).

### Normal Rituals and Repetitive Behavior

Ritualistic, repetitive activity is extremely common in young children (Peleg-Popko & Dar, 2003). A familiar example is the bedtime ritual of saying good night—addressing people in a certain order or giving a certain number of hugs and kisses. Normal ritualistic behaviors in young children include preferences for sameness in the environment (e.g., watching the same DVD over and over again), rigid likes and dislikes, preferences for symmetry (e.g., carrying a toy in each hand), awareness of minute details or imperfections in toys or clothes (e.g., being bothered by a minuscule thread on a jacket sleeve), and arranging things so they are "just right" (e.g., insisting that different foods not touch each other on the plate). Rituals help young children gain control and mastery over their social and physical environments and make their world more predictable and safer (Evans et al., 1997). Any parent who has violated these rituals and paid the price can appreciate how important they are to the young child.

Many common routines of young children fall into two distinct categories: repetitive behaviors and doing things "just right." These categories are strikingly similar to those found for older individuals with OCD and related disorders, which we discuss later in the chapter. It is not known whether OCD is an extreme point on a continuum of normal developmental rituals or an entirely different problem (Evans, Gray, & Leckman, 1999). However, research suggests that the neuropsychological mechanisms underlying compulsive, ritualistic behavior in normal development and those in OCD may be similar (Pietrefesa & Evans, 2007).

## Anxiety Disorders According to DSM-5

Anxiety disorders in DSM-5 are divided into seven categories that closely define the focus of the child's anxiety and the types of reaction and avoidance. To give you an overall picture, these disorders are described briefly in A Closer Look 11.1. The number of youths with multiple anxiety disorders increases with age. Keep in mind that significant associations exist between nearly all anxiety disorders. These associations are best explained by a model that

A CLOSER **LOOK 11.1**

## Main Features of Seven DSM-5 Anxiety Disorders

**Separation Anxiety Disorder (SAD)**
Characterized by excessive worry regarding separation from home or parents. Youths may show signs of distress and physical symptoms on separation, experience unrealistic worries about harm to self or others when separated, and display an unwillingness to be alone.

**Specific Phobia**
Characterized by severe and unreasonable fears and avoidance of a specific object or situation, for example, dogs, spiders, darkness, or riding on a bus.

**Social Anxiety Disorder (SOC) (Social Phobia)**
Characterized by a severe and unreasonable fear of being embarrassed or humiliated when doing something in front of peers or adults.

**Selective Mutism**
Characterized by a consistent failure to speak in specific social situations in which there is an expectation for speaking (e.g., school), even though the child may speak loudly and frequently at home or in other settings.

**Panic Disorder (PD)**
Characterized by recurrent, unexpected and severe panic attacks. These attacks may consist of an accelerated heart rate, shortness of breath, sweating, upset stomach, dizziness, fear of dying, and others. The individual also experiences a persistent concern or worry about additional panic attacks or their consequences, or displays a significant maladaptive change in behavior to avoid having panic attacks (e.g., avoidance of exercise or new situations).

**Agoraphobia**
Characterized by fear or anxiety about two or more situations such as using public transportation, being in open spaces (e.g., parking lots, marketplaces), being in enclosed spaces (e.g., theaters), being in a crowd, or being outside of the home alone. The fear or anxiety about these situations occurs because the individual thinks that escape might be difficult or help not available if they were to develop panic-like or other incapacitating symptoms.

**Generalized Anxiety Disorder (GAD)**
Characterized by ongoing and excessive worry about many events and activities. Youths may worry about their grades in school, their relations with peers, and their own or others' safety. They may constantly seek comfort or approval from others to help reduce their worry.

specifies multiple distinct anxiety syndromes that are related to a higher-order factor (e.g., negative affect) that is common to most if not all anxiety disorders, as well as depression (Higa-McMillan et al., 2014).

## Section Summary

### Description of Anxiety Disorders

- Anxiety disorders are among the most common mental health problems in children and adolescents, but they often go unnoticed and untreated.
- Anxiety is an adaptive emotion that prepares youngsters to cope with potentially threatening people, objects, or events. Strong negative emotions, physical tension, and apprehensive anticipation of future danger or misfortune characterize it.
- The symptoms of anxiety are expressed through three interrelated response systems: physical, cognitive, and behavioral.
- Fear is a present-oriented emotional reaction to current danger. In contrast, anxiety is a future-oriented emotion characterized by feelings of apprehension and a lack of control over upcoming events that might be threatening.
- Fears, anxieties, worries, and rituals in children are common, change with age, and follow a predictable developmental pattern with respect to type.
- DSM-5 specifies several types of anxiety and related disorders based on types of reaction and avoidance.

## SEPARATION ANXIETY DISORDER

### BRAD

### "Don't Leave Me!"

Brad, age 9, is unable to enter any situation that requires separation from his parents—playing in the backyard, going to other children's homes, or staying with a babysitter. When forcibly separated from his parents, Brad cries or throws a full-blown tantrum. When his mother plans to leave the house, he runs through all the horrible things that might happen to her, in an endless series

of what-if questions. When she becomes frustrated and angry, Brad becomes even more anxious. The more anxious he gets, the more he argues with his mother, and the angrier she gets. Brad has also threatened to hurt himself if forced to go to school.

Brad's separation problems began about a year ago, when his father was drinking too much and was frequently absent for long periods. Brad's problem gradually worsened over the course of the year, until he completely refused to go to school. Help was sought, but Brad continued to get worse. He developed significant depressive symptoms, including sadness, guilt about his problems, and occasional wishes to die.

Adapted from Last, 1988.

Separation anxiety is important for the young child's survival and is normal at certain ages. From about age 7 months through the preschool years, almost all children fuss when they are separated from their parents or others to whom they are close. In fact, a lack of separation anxiety at this age may suggest insecure attachment or other problems. Unfortunately, like Brad, some children continue to display such anxiety long after the age at which it is typical or expected. When anxiety persists for at least 4 weeks and is severe enough to interfere with normal daily routines such as going to school or participating in recreational activities, the child may have a separation anxiety disorder. The DSM-5 criteria are presented in Table 11.3.

Children with **separation anxiety disorder** (**SAD**) display age-inappropriate, excessive, and disabling distress related to separation from their parents or other major attachment figures and fear of being alone (Cooper-Vince et al., 2014). Young children with SAD may have vague feelings of anxiety or repeated nightmares about being kidnapped or killed or about the death of a parent. They frequently display excessive demands for parental attention by clinging to their parents and shadowing their every move. Often, they are reluctant to sleep separated from their parents, and they try to climb into their parents' bed at night or sleep on the floor just outside their parents' bedroom door (Allen et al., 2010). Older children with SAD may have difficulty being alone in a room during the day, sleeping alone even at home, running errands, going to school, or going to camp. They may also have specific fantasies of illness, accidents, kidnapping, or physical harm.

Children with SAD fear new situations and may display physical symptoms. To avoid separation, they may fuss, cry, scream, or threaten suicide if the parent leaves (although serious suicide attempts are rare); physical symptoms may include rapid heartbeat, dizziness, headaches, stomachaches, and nausea. Not surprisingly, parents, especially mothers, become highly distressed. Over time, as we saw with Brad, children

**TABLE 11.3** | **Diagnostic Criteria for Separation Anxiety Disorder (SAD)**

**DSM-5**

**(A)** Developmentally inappropriate and excessive fear or anxiety concerning separation from those to whom the individual is attached, as evidenced by at least three of the following:

- **(1)** Recurrent excessive distress when anticipating or experiencing separation from home or from major attachment figures.
- **(2)** Persistent or excessive worry about losing major attachment figures or about possible harm to them, such as illness, injury, disasters, or death.
- **(3)** Persistent and excessive worry about experiencing an untoward event (e.g., getting lost, being kidnapped, having an accident, becoming ill) that causes separation from a major attachment figure.
- **(4)** Persistent reluctance or refusal to go out, away from home, to school, to work, or elsewhere because of fear of separation.
- **(5)** Persistent and excessive fear of or reluctance about being alone or without major attachment figures at home or in other settings.
- **(6)** Persistent reluctance or refusal to sleep away from home or to go to sleep without being near a major attachment figure.
- **(7)** Repeated nightmares involving the theme of separation.
- **(8)** Repeated complaints of physical symptoms (e.g., headaches, stomachaches, nausea, vomiting) when separation from major attachment figures occurs or is anticipated.

**(B)** The fear, anxiety, or avoidance is persistent, lasting at least 4 weeks in children and adolescents and typically 6 months or more in adults.

**(C)** The disturbance causes clinically significant distress or impairment in social, academic, occupational, or other important areas of functioning.

**(D)** The disturbance is not better explained by another mental disorder, such as refusing to leave home because of excessive resistance to change in autism spectrum disorder; delusions or hallucinations concerning separation in psychotic disorders; refusal to go outside without a trusted companion in agoraphobia; worries about ill health or other harm befalling significant others in generalized anxiety disorder; or concerns about having an illness in illness anxiety disorder.

**(E)** criteria and Specify if should be deleted in their entirety. No substitutions for either.

*Source:* Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, American Psychiatric Association.

with SAD may become increasingly withdrawn, apathetic, and depressed, and are at risk for developing a variety of other anxiety disorders during adolescence (Lewinsohn et al., 2008).

## Prevalence and Comorbidity

SAD is one of the two most common anxiety disorders to occur during childhood (the other is specific phobia), and it is found in about 4% to 10% of all children (Merikangas et al., 2010). It is common in both boys and girls, although it is more prevalent in girls. About two-thirds of children with SAD have another anxiety disorder, and about half develop a depressive disorder following the onset of SAD. They may also display specific fears of getting lost or of the dark. School reluctance or refusal is also quite common in older children with SAD (Albano, Chorpita, & Barlow, 2003).

## Onset, Course, and Outcome

Of children referred for anxiety disorders, SAD has the earliest reported age at onset (7 to 8 years) and the youngest age at referral (Shear et al., 2006). SAD generally progresses from mild to severe. It may begin with harmless requests or with symptoms such as restless sleep or nightmares, which progress to the child sleeping nightly in his or her parents' bed. Similarly, school mornings may evoke physical symptoms and an occasional absence from school, which escalates into daily tantrums about leaving for school and outright refusal. The child may become increasingly concerned about the parents' daily routine and whereabouts (Albano et al., 2003).

Often, SAD occurs after a child has experienced major stress, such as moving to a new neighborhood, entering a new school, death or illness in the family, or an extended vacation. Brad's SAD emerged after his father developed a problem with alcohol and subsequently left home. The symptoms of SAD may also fluctuate over the years as a function of stress and transitions in the child's life. Although they may lose friends as a result of their repeated refusal to participate in activities away from home, children with SAD are reasonably skilled socially and get along with others. However, their school performance may suffer as a result of frequent school absences. The child may require special assignments just to keep up; in extreme cases, they may have to repeat the school year (Albano et al., 2003).

SAD persists into adulthood for more than one-third of children and adolescents. As adults, these individuals are more likely than others to experience relationship difficulties (e.g., never marry or become separated or divorced), other anxiety disorders and mental health problems (particularly panic disorder and depression), and functional impairment in their social and personal lives (Milrod et al., 2014; Shear et al., 2006).

School reluctance and refusal are quite common in youngsters with SAD.

ERIC

### Won't Go to School

Eric, age 12, was referred by a school psychologist and his parents for his intense school refusal behavior. On entering seventh grade and a new school, he began to experience a variety of negative symptoms, such as hyperventilation, anxiety, sad mood, and somatic symptoms. Although attendance was not a problem at first, by mid-September Eric began to report severe headaches on school mornings. School attendance then became intermittent. By late September, his aversion to school had worsened and he was staying at home on most days.

Adapted from Kearney, 1995.

## School Reluctance and Refusal

Although starting school is exciting and enjoyable for most children, many are reluctant to go to school and—for a few—school may create so much fear and anxiety that they will not go. These children can become literally sick with worry, let minor physical symptoms keep them at home, or pretend to be ill. **School refusal behavior** is defined as the refusal to attend classes or difficulty remaining in school for an entire day. It includes youngsters who resist going to school in the morning but eventually attend, those who go to school but leave at some point during the day, those who attend with great dread that leads to future pleas for nonattendance, and those who miss the entire day (Kearney, 2007).

School refusal is equally common in boys and girls, and it occurs most often between the ages of 5 and 11 years. Excessive and unreasonable fears of school usually first occur during preschool, kindergarten, or first grade and peak during the second grade. However, school refusal can occur at any time and may have a sudden onset at a later age, as happened with Eric. Children who refuse school may complain of a headache, upset stomach, or sore throat just before it's time to leave for school, then begin to "feel better" when permitted to stay at home, only to feel "sick" again the next morning. As the time for school draws near, the child may plead, cry, and refuse to leave the house and may even have a full-blown panic reaction. School refusal often follows a period at home during which the child has spent more time than usual with a parent (e.g., brief illness, holiday break, or summer vacation). At

other times, school refusal may follow a stressful event such as a change of schools (as happened with Eric), an accident, or the death of a relative or family pet.

For many children, fear of school is really a fear of leaving their parents—separation anxiety. However, school reluctance and refusal can occur for many reasons (Kearney & Albano, 2004). Most children who refuse to go to school have average or above-average intelligence, suggesting that it is not a difficulty with academics that leads to this problem. A fear of school may be associated with submitting for the first time to authority and rules outside the home, being compared with unfamiliar children, and experiencing the threat of failure. Some children fear school because they are afraid of being ridiculed, teased, or bullied by other children or being criticized or disciplined by their teachers. In other cases, the child's fear may result from an excessive or irrational fear of being socially evaluated or embarrassed when having to recite in class or undress in front of unfamiliar people in a gym class. Eric was extremely anxious about meeting new people, being late for class, moving from class to class, taking classes involving public speaking, and participating in gym class. He refused to attend school mainly to escape being socially evaluated and, to a lesser extent, to gain attention from his parents (Kearney & Silverman, 1996).



School reluctance and refusal are common problems related to anxiety.

The possible long-term consequences are serious for a child who displays a persistent pattern of school refusal behavior and does not receive help. Academic or social problems may develop as a result of missed instruction and peer interaction. Treatment usually emphasizes an immediate return to school and other routines and must take into account the specific functions being served by school refusal behaviors (Kearney & Albano, 2007).

## Section Summary

### Separation Anxiety Disorder

- Children with SAD display age-inappropriate, excessive, and disabling distress related to separation from and fear of being alone without their parents or other major attachment figures.
- SAD is one of the most common anxiety disorders of childhood, with the earliest reported age at onset and the youngest age at referral.
- School refusal behavior is defined as the refusal to attend classes or difficulty remaining in school for an entire day.

# SPECIFIC PHOBIA

### CHARLOTTE

### Arachnophobia

For 2 years, Charlotte, age 8, has complained of an intense fear of spiders. "Spiders are disgusting," she says. "I'm scared to death that one will crawl on me, especially when I'm sleeping. When I see a spider, even a little one, my heart pounds, my hands feel cold and sweaty, and I start to shake." Charlotte's mother says that her daughter goes completely pale when she sees a spider, even at a distance, and tries to avoid any situation where she thinks there might be one. Charlotte's fear is beginning to interfere with her daily activities. For example, she won't play in the backyard and refuses to go on class or family outings where she might encounter a spider. She is afraid to go to sleep at night because she thinks a spider might crawl on her. (Based on authors' case material.)

Many children have specific fears that are mildly troubling, come and go rapidly until about age 10, and rarely require special attention. However, if the child's fear occurs at an inappropriate age, persists, is irrational or

exaggerated, leads to avoidance of the object or event, and causes impairment in normal routines, it is called a **specific phobia.** Like Charlotte, children with a specific phobia display a marked fear or anxiety about specific objects or situations (e.g., animals, heights) for at least 6 months. The DSM-5 criteria for specific phobia are shown in Table 11.4.

Children with a specific phobia show an extreme and disabling fear about objects or situations that in reality pose little or no danger or threat; these children go to great lengths to avoid these objects or situations. They experience extreme fear or dread, physiological arousal to the feared stimulus, and fearful anticipation and avoidance when confronted with the object of their fear. Their fear or anxiety may be expressed by crying, tantrums, freezing, or clinging. Their thinking usually focuses on threats to their personal safety, such as being stung by a bee or struck by lightning. Anticipatory anxiety is also common. For example, a child with a phobia of dogs may think: "What if a big dog is running loose on my way to school and I get attacked and bitten in the face?" These worries cause distress severe enough to disrupt everyday activities. The children are constantly on the lookout for the feared stimulus and, as we saw with Charlotte, go to great lengths to avoid contact.

Children's beliefs regarding the danger of the feared stimulus are likely to persist despite evidence that no danger exists or despite efforts to reason with them. Unlike most adults with a specific phobia, children often do not recognize that their fears are extreme and unreasonable. If the feared object is rarely encountered, the phobia may not lead to serious impairment. However, if it is encountered regularly or if the fear causes significant distress or seriously interferes with important life events, the child's phobia can become a serious problem (Albano et al., 2003).

The phobias that can develop in children and adolescents seem limitless; they include fears of telephones, water, menstruation, newspapers, mathematics, haircuts, and bowel movements, to name just a few. Although it is possible to develop a phobia about almost any object, situation, or event—ranging from A (apiphobia, a fear of bees) to Z (zemmiphobia, a fear of the great mole rat)—children are much more likely to develop certain fears than others (Depla et al., 2008).

**TABLE 11.4** | **Diagnostic Criteria for Specific Phobia**

**DSM-5**

**(A)** Marked fear or anxiety about a specific object or situation (e.g., flying, heights, animals, receiving an injection, seeing blood).

**Note:** In children, the fear or anxiety may be expressed by crying, tantrums, freezing, or clinging.

**(B)** The phobic object or situation almost always provokes immediate fear or anxiety.

**(C)** The phobic object or situation is actively avoided or endured with intense fear or anxiety.

**(D)** The fear or anxiety is out of proportion to the actual danger posed by the specific object or situation and to the sociocultural context.

**(E)** The fear, anxiety, or avoidance is persistent, typically lasting 6 months or more.

**(F)** The fear, anxiety or avoidance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

**(G)** The disturbance is not better accounted for by another mental disorder, including fear, anxiety, and avoidance of situations associated with panic-like symptoms or other incapacitating symptoms (as in agoraphobia); objects or situations related to obsessions (as in obsessive–compulsive disorder); reminders of traumatic events (as in post-traumatic stress disorder); separation from home or attachment figures (as in separation anxiety disorder); or social situations (as in social anxiety disorder).

*Specify if (code based on the phobic stimulus):*

**Animal** (e.g., spiders, insects, dogs)

**Natural** environment (e.g., heights, storms, water)

**Blood**, injection, injury (e.g., needles, invasive medical procedures)

**Situational** (e.g., airplanes, elevators, enclosed places)

**Other** (e.g., situations that may lead to choking or vomiting; in children, loud sounds or costumed characters)

*Source:* Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, American Psychiatric Association.

Bonnie Kamin/PhotoEdit

Fear of animals is one of the most common and heritable specific phobias.

According to evolutionary theory, human infants are biologically predisposed as a result of natural selection to learn certain fears (Seligman, 1971). The sources of most children's phobias can be traced to the natural dangers encountered during human evolution—snakes, the dark, predators, heights, blood, loud noises, and unfamiliar places. For example, when listening to evolutionary fear-relevant sounds (e.g., snake hissing), infants as young as 9 months of age display heart rate slowing, an increased eye-blink startle response, and more visual orienting, as compared to when they listen to modern fear-relevant sounds (e.g., siren wailing) or pleasant sounds (e.g., crowd cheering) (Erlich, Lipp, & Slaughter, 2013). These fears are adaptive in an evolutionary sense because they alert the individual to possible sources of danger, thereby increasing the likelihood of survival. It is not only by chance that the most common and most heritable specific phobias in children are a fear of events in the natural environment (e.g., heights, thunder) and a fear of animals, particularly dogs, snakes, insects, and mice (Essau, Conradt, & Petermann, 2000). Although evolutionary theory explains a readiness to acquire specific types of fears, it does not explain why children differ in their fearfulness or why some children develop extreme and disabling anxiety.

As specified in DSM-5, common types of specific phobias in young people include fears of animals or insects (e.g., dogs or spiders); fears of natural events (e.g., heights or thunderstorms); fears of blood, injuries, or medical procedures (e.g., seeing blood or receiving an injection); and fears of specific situations (e.g., flying in airplanes, riding on a bus). Both similarities (e.g., age at onset, gender, treatment response) and differences (e.g., focus of fear, physiological reaction, neural response patterns, impairment, comorbidity) have been found across these types, with natural environment and animal phobias having the most in common with other types, and blood, injury, and injection phobias the least (LeBeau et al., 2010; Lueken et al., 2011).

## Prevalence and Comorbidity

About 20% of all youngsters experience specific phobias at some time in their lives, and those with this disorder tend to have multiple phobias (Kessler et al., 2012a; Merikangas et al., 2010). However, very few of these children are referred for treatment, suggesting that most parents do not view specific phobias as significantly harmful. There does seem to be a family vulnerability for particular types of phobias—children are at increased risk for the phobic disorder exhibited by their parent (LeBeau et al., 2010). Family risk can be attributed to both genetic and environmental factors. Specific phobias, particularly blood phobia, are more common in girls than boys (Essau et al., 2000). The most common co-occurring disorders for youngsters with a specific phobia are another anxiety disorder and depressive disorders (Leyfer et al., 2013). Although comorbidity is frequent for children with specific phobias, it tends to be lower than for other anxiety disorders (LeBeau et al., 2010).

## Onset, Course, and Outcome

Phobias involving animals, darkness, insects, blood, and injury typically have their onset at 7 to 9 years of age, which is similar to normal development. However, even though fears and phobias decline with age, clinical phobias are more likely to persist over time than are normal fears. Specific phobias can occur at any age but seem to peak between 10 and 13 years of age (LeBeau et al., 2010).

### Section Summary

#### Specific Phobia

- Children with a specific phobia exhibit an extreme and disabling fear of particular objects or situations that in reality pose little or no danger.
- Evolutionary theory contends that human infants are biologically predisposed to learn certain fears that alert them to possible sources of danger. This may explain why the most common specific phobia in children is a fear of animals, such as dogs, snakes, and insects.
- DSM-5 categorizes specific phobias into five subtypes based on the focus of the phobic reaction and avoidance: animal; natural environment; blood, injection, injury; situational; and other.
- About 4% to 10% of children experience specific phobias, but only a very few are referred for treatment. Specific phobias can occur at any age, but seem to peak between 10 and 13 years of age.

# SOCIAL ANXIETY DISORDER (SOCIAL PHOBIA)

*To understand the world one must not be worrying about one's self.*

—Albert Einstein (1879–1955)

- Kaylie is terrified to use the phone because, she says, she doesn't know how to have a conversation and would be embarrassed by the long periods of silence.
- Eugene is too embarrassed to use a public restroom.
- Li-Ming is terrified of speaking in front of her class—she's afraid of being humiliated.

Each of these youngsters has a **social anxiety disorder** (**SOC** [for "social"]) or **social phobia**—a marked and persistent fear of social or performance requirements that expose them to scrutiny and possible embarrassment (Knappe, Beesdo-Baum, & Wittchen, 2010). They go to great lengths to avoid these situations, or they may face the challenge with great effort, wearing a mask of fearlessness. Long after the age at which a fear of strangers is considered normal, children with SOC continue to shrink from people they do not know. When in the presence of other children or adults, they may blush, fall silent, cling to their parents, or try to hide. To be classified as SOC, their anxiety must occur in peer settings, not just when interacting with adults. The DSM criteria for SOC are shown in Table 11.5.

In addition to their extreme anxiety in social situations that make many people anxious, youngsters with SOC may feel anxious about the most mundane activities—handing out papers in class, buttoning their coat in front of others, or ordering a Big Mac and fries at McDonalds. Their most common fear is doing something in front of other people. They fear that if they speak in public, they may stumble over their words; if they ask a question, they may sound stupid; if they enter a room, they may trip and look awkward. One teenage girl was so fearful of being the focus of attention during meals that she spent every lunch period during her first year in high school sitting in a bathroom stall (Albano et al., 2003, p. 287).

Youngsters with SOC are more likely than other children to be highly emotional, socially fearful, and inhibited, sad, and lonely. They frequently experience socially distressing events with which they are unable to cope effectively, in part related to a lack of social skills (Beidel, Turner, & Morris, 1999). These children want to be liked by other people. However, their fear of acting in a way that may invite humiliation is so intense and pervasive that it often leads to loneliness and suffering because they cannot form the relationships they desire (La Greca & Lopez, 1998). If other people attempt to push them into social situations they may cry, have a tantrum, freeze, or withdraw even further. They fear most social situations, are afraid to meet or talk with new people, avoid contact with anyone outside their family, and find it extremely difficult to attend school, participate in recreational activities, or socialize (Beidel et al., 2007; Bernstein et al., 2008). Current evidence supports the view of SOC as existing on a continuum of severity from lesser to greater as a function of the number of social situations that are feared and/or avoided (Bögels et al., 2010).

The anxiety associated with SOC can be so severe that it produces stammering, sweating, upset stomach, rapid heartbeat, or a full-scale panic attack. Adolescents with SOC frequently believe that their visible physical reactions will expose their hidden feelings of inadequacy, which makes them even more anxious. In a repeating cycle, children with SOC anticipate their awkwardness and poor performance, which triggers further anxiety as they approach the feared situation, and further increases their nervousness and physical symptoms. As a result, they avoid social activities such as calling a classmate for missed homework, asking the

**TABLE 11.5** | **Diagnostic Criteria for Social Anxiety Disorder (Social Phobia)**

**DSM-5**

**(A)** Marked fear or anxiety about one or more social situations in which the individual is exposed to possible scrutiny by others. Examples include social interactions (e.g., having a conversation, meeting unfamiliar people), being observed (e.g., eating or drinking), or performing in front of others (e.g., giving a speech).

**Note:** In children, the anxiety must occur in peer settings and not just during interactions with adults.

**(B)** The individual fears that he or she will act in a way or show anxiety symptoms that will be negatively evaluated (i.e., will be humiliating or embarrassing; will lead to rejection or offend others).

**(C)** The social situations almost always provoke fear or anxiety.

**Note:** In children, the fear or anxiety may be expressed by crying, tantrums, freezing, clinging, shrinking away, or failing to speak in social situations.

**(D)** The social situations are avoided or endured with intense fear or anxiety.

**(E)** The fear or anxiety is out of proportion to the actual danger posed by the social situation and to the sociocultural context.

**(F)** The fear, anxiety, or avoidance is persistent, typically lasting for 6 months or more.

**(G)** The fear, anxiety, and avoidance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

**(H)** The fear, anxiety, and avoidance is not attributable to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.

**(I)** The fear, anxiety, or avoidance is not better explained by the symptoms of another mental disorder, such as panic disorder, body dysmorphic disorder, or autism spectrum disorder.

**(J)** If another medical condition (e.g., Parkinson's disease, obesity, disfigurement from burns or injury) is present, the fear, anxiety, or avoidance is clearly unrelated or is excessive.

*Specify if:*

***Performance only:*** If the fear is restricted to speaking or performing in public.

*Source:* Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, American Psychiatric Association.

teacher to explain something, answering the telephone, going to parties, and dating (Albano, 1995).

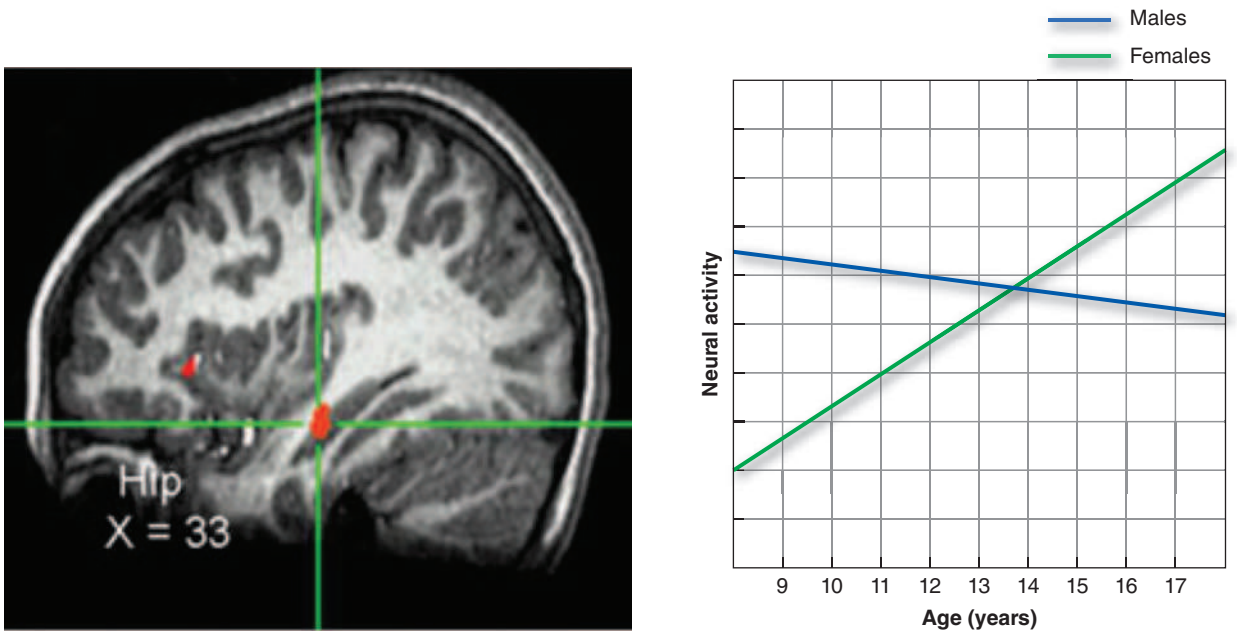
SOC encompasses a variety of social fears, including fear of performance situations, such as speaking in front of others, and fear of interaction situations, such as talking to others at a party. Some research has suggested that performance- and interaction-related social fears may differ from one another in their risk factors and clinical characteristics and that identifying children with SOC who differ in their types of social fears may help to further our understanding of this disorder (Knappe et al., 2011). To this end, DSM-5 provides a "performance only" specifier to identify youngsters with SOC whose anxiety is restricted to performance situations such as speaking in front of others or performing in public (e.g., at a sporting event or musical recital).

## Prevalence, Comorbidity, and Course

SOC is common; it has a lifetime prevalence of 6% to 12% and affects nearly twice as many girls as boys (Knappe et al., 2010). Girls may experience greater social anxiety because they are more concerned with social competence than are boys and attach greater importance to interpersonal relationships and evaluation by peers (Inderbitzen-Nolan & Walters, 2000). Some support for this hypothesis comes from a brain functional magnetic resonance imaging study into the neural correlates of anticipated evaluation by peers in 9- to 17-year-old male and female adolescents (Guyer et al., 2009). Females who thought that a peer they wished to interact with was evaluating them showed age-related increases in activation of brain regions (e.g., hippocampus) associated with processing emotional information. These increases did not occur in males. Representative findings from this study are shown in ● Figure 11.1 and suggest that, relative to males, adolescent females may have an increasing biological sensitivity to being evaluated by peers, which may, over time, increase their vulnerability to developing SOC.

Among children and adolescents referred for treatment for anxiety disorders, about one-third have SOC as their primary diagnosis. It is also the most common secondary diagnosis for children referred for other anxiety disorders (Leyfer et al., 2013). Even so, many cases of SOC are overlooked because shyness is common and because these children are not likely to call attention to their problem even when they are severely distressed (Essau, Conradt, & Petermann, 1999).

Two-thirds of children and adolescents with SOC have another anxiety disorder—most commonly, generalized anxiety disorder (Bernstein et al., 2008; Leyfer et al., 2013). Other common comorbid anxiety disorders are SAD and specific phobias (Beidel et al., 2007). About 20% of adolescents with SOC also suffer from major depression. They may also use alcohol and other drugs as a




● **FIGURE 11.1** | (a) Increased neural activity as detected in the hippocampus while participants appraised how they thought preferred peers would evaluate them. (b) As age increased, neural activity in the hippocampus increased in females but did not change in males.

(a) © 2009, Amanda E. Guyer, Erin B. McClure-Tone, Nina D. Shiffrin, Daniel S. Pine, Eric E. Nelson; "Probing the Neural Correlates of Anticipated Peer Evaluation in Adolescence" *Child Development,* © 2009, Society for Research in Child Development, Inc. (b) From Probing the neural correlates of anticipated peer evaluation in adolescence by Guyer et al. *Child Development, 80,* 1000–1015. Journal Compilation © 2009, Society for Research in Child Development, Inc. John Wiley and Sons.

form of self-medication to reduce their anxiety in social situations and are at risk for later substance-use problems (Albano et al., 2003; Buckner et al., 2008).

SOC is extremely rare in children under the age of 10, and it generally develops after puberty, with the most common age at onset in early- to mid-adolescence (Wittchen, Stein, & Kessler, 1999). However, given the nature of the disorder, individuals with SOC are often the most reluctant to seek treatment following the onset of their problem. The prevalence of SOC appears to increase with age, with considerable persistence and fluctuations in symptom severity over time (Beesdo-Baum et al., 2012). As adults, individuals with SOC may experience significant impairment in role functioning, including relationship problems, educational difficulties, and poorer overall quality of life. The average duration of symptoms of social anxiety is about 20 to 25 years—thus, it is not a short-lived condition of adolescence and young adulthood. In the absence of effective treatment, the likelihood of a complete and long-lasting remission for SOC is the lowest for all anxiety disorders (Knappe et al., 2010).

### Section Summary

#### Social Anxiety Disorder (SOC) (Social Phobia)

- Children with SOC fear being the focus of attention or scrutiny or of doing something in public that will be intensely humiliating.
- SOC is common, with a lifetime prevalence of 6% to 12%, and affecting nearly twice as many girls as boys.
- SOC generally develops after puberty, at a time when most teens experience heightened self-consciousness and worries about what others think of them.

## SELECTIVE MUTISM

**KEISHA**

### Mum's the Word

Keisha, age 6, doesn't speak at kindergarten to teachers or peers and did not do so during her 2 years in preschool. Two years ago she had difficulties being left at preschool and it took about 2 months before she could be left without crying. Although she doesn't talk to other children, she interacts with them and participates in school activities. Keisha speaks openly to all family members at home but does not speak to them in public if others might hear her. She says that she does not know why she doesn't talk, but has told her mother that she feels scared. Her mother says Keisha is shy and is a worrier.

Adapted from Leonard & Dow, 1995.

Children with **selective mutism** fail to speak in specific social situations in which there is an expectation to speak (e.g., at school), even though they may speak loudly and frequently at home or in other settings (Viana, Beidel, & Rabian, 2009). The DSM-5 criteria also require that the child's disturbance interferes with educational or work achievement or with social communication, that it is present for at least 1 month, that it is not limited to a lack of knowledge or discomfort with the spoken language required in the social situation, that it is not better explained by a communication disorder, and that it does not occur only during the course of autism spectrum disorder, schizophrenia, or another psychotic disorder (APA, 2013).

### Prevalence, Comorbidity, and Course

Selective mutism is rare, estimated to occur in about 0.7% of all children in community samples (Bergman, Piacentini, & McKracken, 2002). Prevalence does not seem to vary by sex or race/ethnicity. The most common co-occurring disorders are other anxiety disorders, particularly SOC and specific phobia. Oppositional behaviors may also occur, but these may be limited to situations in which the child is required to speak (Cunningham, McHolm, & Boyle, 2006). The average age at onset is about 3 to 4 years; however, there is often a considerable lag between onset and referral, possibly because the child's mutism may not occur at home. With school entry and the associated increase in social interaction and tasks (e.g., reading aloud), the child is more likely to be identified and referred. The persistence of selective mutism is variable, although many children seem to "outgrow" the disorder. However, research in this area is limited, and the long-term course of the disorder is not known (APA, 2013).

Although it was not previously included as a diagnosable disorder in DSM-IV, selective mutism has many features in common with the anxiety disorders (Leonard & Dow, 1995), which resulted in its inclusion as an anxiety disorder in DSM-5. For example, about 45% to 75% of children with selective mutism meet diagnostic criteria for SOC in ways other than their reluctance to speak (Viana et al., 2009), and nearly 40% of their parents have also been diagnosed with SOC during their lifetime (Chavira et al., 2007). Other common comorbidities include communication, elimination, and oppositional disorders (Cohan et al., 2008).

Based on the similarities between selective mutism and SOC, it has been suggested that selective mutism may be a developmentally specific variant of SOC in young children or an early precursor to SOC, rather than a unique disorder (Bergman et al., 2002; Dummit et al., 1997). However, there are also differences between

the two disorders—for example, nonverbal social engagement and oppositional features occur in selective mutism, but less so in SOC (Yaganeh, Beidel, & Turner, 2006). Relatedly, one study identified three subgroups of children with selective mutism: anxious—mildly oppositional; (2) anxious—communication delayed; and (3) exclusively anxious (Cohan & Chavira, 2008). Anecdotal accounts from adults who suffered from selective mutism as children also suggest that in some cases trauma played a role and not talking was a self-protective response (Omdal, 2007). For example, Dr. Maya Angelou, a celebrated African-American poet and writer who captivated audiences with her words, was mute for nearly 5 years after suffering the trauma of being raped at 8 years old.

## Section Summary

### Selective Mutism

- Children with selective mutism fail to talk in specific social situations where there is an expectation to do so, even though they may speak in other settings.
- Selective mutism is a rare disorder occurring in about 0.7% of all children. Its prevalence does not seem to vary by sex or race/ethnicity.
- The most common co-occurring disorders are other anxiety disorders, particularly SOC and specific phobia.
- The average age at onset is about 3 to 4 years, but there is often a considerable lag between onset and referral.
- Selective mutism and SOC are currently viewed as distinct but strongly related disorders.

# PANIC DISORDER AND AGORAPHOBIA

## CLAUDIA

### An Attack Out of Nowhere

Claudia, age 16, was watching TV after a noneventful day at school. She suddenly felt overwhelmed by an intense feeling of light-headedness and a smothering sensation, as if she couldn't get any air to breathe. Her heart started to pound rapidly, as if it would explode. The attack came on so fast and was so intense that Claudia panicked and thought she was having a heart attack that would kill her. She began to sweat and tremble, and she felt the room was spinning. These feelings reached a peak within 2 minutes … but this was the seventh attack that Claudia had experienced this month. She frantically ran to her mother and pleaded to be taken to the hospital emergency room—again. (Based on authors' case material,)

Prior to DSM-5, panic disorder and agoraphobia were connected because diagnosing panic disorder included the designation "with" or "without" agoraphobia (i.e., a marked fear or avoidance of certain situations in which the individual thinks that escape may be difficult, or help not available, if they were to experience panic-like or other incapacitating symptoms). However, in DSM-5, panic disorder and agoraphobia are now separate disorders with different diagnostic criteria. This change was based on research suggesting that a number of adolescents and adults experience agoraphobia without panic symptoms. However, to date, most studies of young people have considered the two conditions together (Higa-McMillan et al., 2014). Therefore, we discuss both disorders in this section.

Adolescents like Claudia with a **panic disorder** (**PD**) display recurrent unexpected panic attacks followed by at least 1 month of persistent concern or worry about having another attack and its consequences or a significant change in their behavior related to the attacks in order to avoid having them. The DSM-5 diagnostic criteria for Panic Disorder are presented in Table 11.6.

A **panic attack** is a sudden and overwhelming period of intense fear or discomfort that is accompanied by four or more physical and cognitive symptoms characteristic of the fight/flight response (see Table 11.6). Usually, a panic attack is short, with symptoms reaching maximal intensity in 10 minutes or less and then diminishing slowly over the next 30 minutes or the next few hours. Panic attacks are accompanied by an overwhelming sense of imminent danger or impending doom, and

Chris Collins/Cusp/Corbis

Panic

**TABLE 11.6 | Diagnostic Criteria for Panic Disorder**

**DSM-5**

**(A)** Recurrent unexpected panic attacks. A panic attack is an abrupt surge of intense fear or intense discomfort that reaches a peak within minutes and during which time four (or more) of the following symptoms occur:

**Note:** The abrupt surge can occur from a calm state or an anxious state.

**(1)** Palpitations, pounding heart, or accelerated heart rate.
**(2)** Sweating.
**(3)** Trembling or shaking.
**(4)** Sensations or shortness of break or smothering.
**(5)** Feelings of choking.
**(6)** Chest pain or discomfort.
**(7)** Nausea or abdominal distress.
**(8)** Feeling dizzy, unsteady, light-headed, or faint.
**(9)** Chills or heat sensations.
**(10)** Paresthesias (numbness or tingling sensations).
**(11)** Derealization (feelings of unreality) or depersonalization (being detached from oneself).
**(12)** Fear of losing control or "going crazy."
**(13)** Fear of dying.

**Note:** Culture-specific symptoms (e.g., tinnitus, neck soreness, headache, uncontrollable screaming or crying) may be seen. Such symptoms should not count as one of the four required symptoms.

**(B)** At least one of the attacks has been followed by 1 month (or more) of one or both of the following:
**(1)** Persistent concern or worry about additional panic attacks or their consequences (e.g., losing control, having a heart attack, "going crazy")
**(2)** Significant maladaptive change in behavior related to the attacks (e.g., behaviors designed to avoid having panic attacks, such as avoidance of exercise or unfamiliar situations).

**(C)** The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition (e.g., hyperthyroidism, cardiopulmonary disorders).

**(D)** The disturbance is not better accounted for by another mental disorder.

*Source:* Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, American Psychiatric Association.

by an urge to escape. Although they are brief, they can occur several times a week or month. It is important to remember that although the symptoms are dramatic, they are not physically harmful or dangerous.

Panic attacks are easily identified in adults, but some controversy exists over how often they occur in children and adolescents. Although panic attacks are extremely rare in young children, they are common in adolescents (Mattis & Ollendick, 2002). One explanation is that young children lack the cognitive ability to make the catastrophic misinterpretations (e.g., "my heart is beating rapidly and I'm sitting here watching TV like I always do—I must be going crazy") that usually accompany panic attacks (Nelles & Barlow, 1988). However, research suggests that young children may in fact be capable of such misinterpretations (Mattis & Ollendick, 1997).

If limited cognitive capacity is not the primary reason that panic attacks are so rare in young children, what is? In a revealing study, the relationship between the occurrence of panic attacks and pubertal stage was assessed in 754 girls in the sixth and seventh grades. Importantly, increasing rates of panic were related to pubertal development, not to increasing age (Hayward et al., 1992). The significance of pubertal development and anxiety disorders in females has received general support (Reardon, Leen-Feldner, & Hayward, 2009). For example, in one study, sixth- to eighth-grade females who developed internalizing symptoms were on average 5 months earlier in their pubertal development than females who did not develop symptoms (Hayward et al., 1997). Given that spontaneous panic attacks are rare before puberty and are related to pubertal stage, and that adolescence is the peak time for the onset of the disorder, the physical changes that take place around puberty seem critical to the occurrence of panic.

Why do the physical symptoms of the fight/flight response occur if an adolescent is not initially frightened? One possibility is that things other than fear can produce these symptoms. A youngster may be distressed for a particular reason and that stress may increase production of adrenaline and other chemicals that produce physical symptoms of panic. Increased adrenaline may be chemically maintained in the body even after the stress is no longer present. Another possibility is that the youngster may breathe a little too fast (subtle hyperventilation), which also can produce symptoms. Because the over-breathing is very slight, the child gets used to it and does not realize that he or she is hyperventilating. A third possibility is that some youngsters are experiencing normal bodily changes but, because they are constantly monitoring their bodies (as adolescents are prone to do), they notice these sensations far more readily (Barlow, 2002).

Adolescents with PD may avoid locations where they've had a previous panic attack or situations or activities in which they fear an attack might occur. An adolescent with PD might think: "It would be bad enough to have an attack at all, but it would be really dangerous if I had one while riding my bike to school. I'd be totally preoccupied with the attack and would

have an accident. I'd probably destroy my bike and wind up seriously hurting myself or someone else in the process!" The youth's avoidance of riding a bike to school could be misinterpreted as a fear of bike riding, when it is actually a fear of having a panic attack while riding the bike.

If not recognized and treated, PD and its complications can seriously interfere with relationships at home and at school and with school performance. Some adolescents with PD may be reluctant to go to school or be separated from their parents. In severe cases, the tendency to avoid everyday life circumstances may increase and generalize, to the point at which the older adolescent with PD becomes terrified to leave the house at all.

**Agoraphobia** is characterized by marked fear or anxiety in certain places or situations (i.e., being in a crowd, being outside the home alone; see Table 11.7). The individual fears or avoids these situations because of thoughts that escape might be difficult, or help might not be available, if they were to experience panic-like or other incapacitating symptoms (e.g., fear of falling in an elderly person) (Craske et al., 2010; Wittchen et al., 2010). An older adolescent with agoraphobia who dares to venture into a feared situation does so only with great distress or when accompanied by a family member or a friend.

Agoraphobia is a distinct disorder that can be conceptualized independently from both panic attacks and panic disorder. Support for this comes from a 10-year longitudinal follow-up study of a normative sample of 3,000 individuals 14 to 24 years of age at the outset of the study (Wittchen et al., 2008). Sex and age differences in incidence and age at onset were observed between those with agoraphobia and those with panic attacks and panic disorder. The course and stability of agoraphobia also differed from that of panic disorder, and panic attacks did not reliably predict the onset of agoraphobia. Thus, rather than being an outcome of panic disorder, consistent with DSM-5, agoraphobia appears to be an anxiety disorder in its own right (Higa-McMillan et al., 2014).

**TABLE 11.7** | **Diagnostic Criteria for Agoraphobia**

**DSM-5**

**(A)** Marked fear or anxiety about two (or more) of the following five situations:

- **(1)** Using public transportation (e.g., automobiles, buses, trains, ships, planes).
- **(2)** Being in open spaces (e.g., parking lots, marketplaces, bridges).
- **(3)** Being in enclosed spaces (e.g., shops, theatres, cinemas).
- **(4)** Standing in line or being in a crowd.
- **(5)** Being outside of the home alone.

**(B)** The individual fears or avoids these situations because of thoughts that escape might be difficult or help might not be available in the event of developing panic-like symptoms or other incapacitating or embarrassing symptoms (e.g., fear of falling in the elderly; fear of incontinence).

**(C)** The agoraphobic situations almost always provoke fear or anxiety.

**(D)** The agoraphobic situations are actively avoided, require the presence of a companion, or are endured with intense fear or anxiety.

**(E)** The fear or anxiety is out of proportion to the actual danger posed by the agoraphobic situations and to the sociocultural context.

**(F)** The fear, anxiety, or avoidance is persistent, typically lasting for 6 months or more.

**(G)** The fear, anxiety, or avoidance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

**(H)** If another medical condition (e.g., inflammatory bowel disease, Parkinson's disease) is present, the fear, anxiety, or avoidance is clearly excessive.

**(I)** The fear, anxiety, or avoidance is not better explained by the symptoms of another mental disorder.

**Note:** Agoraphobia is diagnosed irrespective of the presence of panic disorder. If an individual's presentation meets criteria for panic disorder and agoraphobia, both diagnoses should be assigned.

*Source:* Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition. American Psychiatric Association.

## Prevalence and Comorbidity

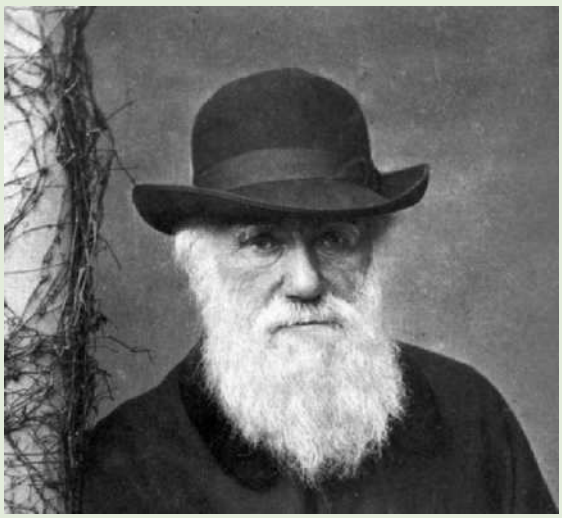
Panic attacks are common among nonreferred adolescents, affecting about 16% of teens (Mattis & Ollendick, 2002). PD and agoraphobia are much less common, with an estimated lifetime prevalence for both of about 2.5% for youths 13 to 17 years of age (Merikangas et al., 2010). Adolescent females are about twice as likely as adolescent males to experience panic attacks, and a fairly consistent association has been found between panic attacks and stressful life events (King, Ollendick, & Mattis, 1994). Most referred adolescents with PD have one or more other disorders, most commonly an additional anxiety disorder (particularly generalized anxiety disorder or SAD) and major depressive disorder. Other comorbid conditions include mania and hypomania, attention-deficit/hyperactivity disorder (ADHD), and oppositional defiant disorder (ODD) (Doerfler et al., 2007). The most common comorbidities for agoraphobia are other anxiety disorders (e.g., PD, specific phobias, and SOC), major depressive disorder, post-traumatic stress disorder, and alcohol-use disorder (APA, 2013).

## A CLOSER **LOOK 11.2**

### Did Darwin Have a Panic Disorder?

Charles Darwin (1809–1882) was a gregarious and daring traveler and outdoorsman in his college days. However, in his late 20s—just a year after returning to England after a 5-year voyage to South America and the Pacific aboard the HMS *Beagle*—he started to have an "uncomfortable palpitation of the heart." The symptoms arose shortly after he began keeping a secret notebook that, 22 years later, would become his book-length elaboration of the theory of evolution, *On the Origin of Species*. Over the years, his affliction was described as a case of bad nerves, a tropical disease, intellectual exhaustion, arsenic poisoning, suppressed gout, and a host of other symptoms. However, in his journal Darwin described his malady as a "sensation of fear … accompanied by troubled beating of the heart, sweat, trembling of muscles."

From Desmond & Moore, 1991.



Bettmann/Corbis

The relation between SAD and PD has received considerable attention, to see whether separation experiences during childhood contribute to the development of later PD or whether SAD is a childhood form of adult panic disorder (Craske et al., 2010). Findings generally support SAD as a strong predictor of PD. However, since SAD also predicts other anxiety disorders (but not depressive or substance-use disorders), it may be an early marker for anxiety disorders in general, rather than a specific risk factor for PD (Kossowsky et al., 2013).

After months or years of unrelenting panic attacks and the restricted lifestyle that results from avoidance behavior, adolescents and young adults with PD and agoraphobia may develop severe depression and may be at risk for suicidal behavior. Others may begin to use alcohol or drugs as a way of alleviating their anxiety (Higa-McMillan et al., 2014).

## Onset, Course, and Outcome

Although PD has been found to occur in young children, few cases have been reported (Higa-McMillan et al., 2014). The average age at onset for a first panic attack in adolescents with PD is 15 to 19 years, and 95% of adolescents with the disorder are postpubertal (Bernstein, Borchardt, & Perwien, 1996). PD occurs in otherwise emotionally healthy youngsters about half the time. The most frequent prior disturbance, if one exists, is a depressive disorder (Last & Strauss, 1989). Unfortunately, PD and agoraphobia are stable over time and have one of the lowest complete remission rates for any of the anxiety disorders (Wittchen et al., 2000). Individuals with PD and those with PD and agoraphobia with an early onset are more likely to experience comorbid disorders and a recurrence of symptoms following a period of remission than those with a later onset, indicating that early onset PD and agoraphobia are particularly serious disorders (Ramsawh et al., 2011). In the absence of treatment, these disorders are likely to have a persistent and chronic course.

## Section Summary

### Panic Disorder and Agoraphobia

- A panic attack is a sudden and overwhelming period of intense fear or discomfort accompanied by physical and cognitive symptoms.
- Adolescents with PD display recurrent unexpected panic attacks followed by persistent concern about having another attack, constant worry about the consequences, or a significant maladaptive change in their behavior related to the attacks, designed to avoid having additional attacks.
- Agoraphobia is characterized by marked fear or anxiety in certain situations. The individual fears or avoids these situations because of thoughts that escape might be difficult or help might not be available if they were to experience panic-like or other incapacitating symptoms.
- Many postpubertal adolescents experience panic attacks, but PD and agoraphobia are much less common, affecting about 2.5% of teens, and females about twice as often as males. Average age at onset for a first panic attack in adolescents with PD is 15 to 19 years.
- PD and agoraphobia are associated with many other disorders, most commonly other anxiety disorders.