11

Abnormal Behaviour Across the Lifespan

CHAPTER OUTLINE

Neurodevelopmental Disorder
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Depression in Childhood and Adolescence

Delirium
Clinical Picture
Diagnosis
Treatment and Outcomes

Dementia
Clinical Picture
Neuropathy
Causes and Diagnosis
Treatment and Outcomes

Did You Know That...

- Many behaviour patterns considered normal for children would be considered abnormal in adults?
- Maternal smoking during pregnancy may contribute to the development of attention-deficit/hyperactivity disorder (ADHD) in children?
- Some people can recall verbatim every story they read in a newspaper?
- Most children with ADHD are given stimulants to help calm them down?
- Major depression can occur in young children?
- A specific protein in spinal fluid can be used to diagnose Alzheimer's disease?
- In the elderly, delirium-like symptoms can sometimes occur as early signs of infection or heart problems, making accurate delirium diagnosis problematic?

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Psychological problems in childhood and adolescence often have a special poignancy. They affect children at ages when they have little capacity to cope. The same could be said for some diseases that are more likely to occur later in life. Contrary to images and messages we get from our current culture that aging is equated with disease and decline, most older people are physically and mentally healthy, living happy and productive lives. In fact, psychological disorders in general are lower among older people than younger people. Here we'll focus on the 10% to 20% of people over the age of 65 who have psychological problems that could be severe enough to warrant diagnosis and treatment, disorders such as delirium and dementia disorder. We'll come to these at the end of this chapter.

Some psychological problems in childhood and adolescence mirror the types of problems found in adults—problems such as mood and anxiety disorders. In some cases, such as separation anxiety, the problems are unique to childhood; in others, such as attention-deficit/hyperactivity disorder (ADHD), the problems manifest differently in childhood than in adulthood.

To determine what is normal and abnormal among children and adolescents, not only do we consider the criteria outlined in earlier chapters, we also weigh what is to be expected given a child's age, gender, and family and cultural background, as well as the sundry developmental transformations that are taking place. Many problems are first identified when a child enters school. They may have existed earlier but were tolerated or unrecognized as problematic in the home. Sometimes, the stress of starting school contributes to their onset. Keep in mind, however, that what is socially acceptable at one age, such as intense fear of strangers at about nine months, may be socially unacceptable at more advanced ages. Many behaviour patterns that would be considered abnormal among adults—such as intense fear of strangers and lack of bladder control—are perfectly normal for children at certain ages.

Just how common are mental health problems among Canada's children and adolescents? Although we lack nationwide statistics, University of British Columbia researchers reviewed studies from Ontario, Quebec, the United States, and the United Kingdom that indicated that about 14% of children and adolescents have a mental disorder (Waddell, McEwan, Shepherd, Offord, & Hua, 2005; Waddell & Shepherd, 2002). The four most common categories—anxiety disorders, conduct disorders, ADHD, and depressive disorders—account for nearly 90% of those mental disorders.

Despite the prevalence of psychological disorders among the young, fewer than a quarter of children and youth with mental disorders receive specialized clinical services (Waddell et al., 2005; Waddell, Offord, Shepherd, Hua, & McEwan, 2002). Children who have internalized problems, such as anxiety and depression, are at higher risk of going untreated than are children with externalized problems (problems involving acting out or aggressive behaviour), which tend to be disruptive or annoying to others.

In this chapter we first examine a number of psychological disorders affecting children and adolescents, and then focus on two of the most frequent cognitive disorders found among the elderly: delirium and dementia. Delirium develops quickly and can be described as a confused state, while dementia is the gradual worsening of memory and cognitive function. We examine the features of these disorders, their causes, and the treatments used.

NEURODEVELOPMENTAL DISORDER

Neurodevelopmental disorder involves markedly impaired behaviour or functioning in multiple areas of development. This disorder generally becomes evident in the first few years of life and is often associated with intellectual disability (APA, 2013). It was generally classified as a form of psychosis in early editions of the DSM, and was thought to reflect childhood forms of adult psychoses, such as schizophrenia, because it shared features such as social and emotional impairment, oddities of communication, and stereotyped motor behaviours. Research has shown that it is distinct from schizophrenia and other psychoses, however, and neurodevelopmental disorder is now classified separately from psychotic disorders. The label of schizophrenia with childhood onset is reserved for the relatively rare instances in which schizophrenia develops in childhood (Jacobsen et al., 1997).

McGill University child research psychiatrist Eric Fombonne (2005) estimates that the prevalence of neurodevelopmental disorder is 0.6% to 0.7%. The DSM (APA, 2013) includes the following neurodevelopmental disorders: autism spectrum disorder, intellectual disability, communication disorders, attention-deficit/hyperactivity disorder, specific learning disorder and motor disorders. The major type of neurodevelopmental disorder, and our focus here, is autism, or autism spectrum disorder.

Autism Spectrum Disorder

Autism Spectrum Disorder (ASD) is one of the severest disorders of childhood. It is a chronic, lifelong condition. Children with autism, like Mahin, who is described in the following case study, seem utterly alone in the world. Despite parental efforts to bridge the gulf that divides them, children with autism spectrum disorder remain in their private worlds.

Mahin nursed eagerly, sat, and walked at the expected ages. Yet some of his behaviour made us vaguely uneasy. He never put anything in his mouth. Not his fingers nor his toys—nothing. . . . More troubling was the fact that Mahin didn't look at us, or smile, and wouldn't play the games that seemed as much a part of babyhood as diapers. He rarely laughed, and when he did, it was at things that didn't seem funny to us. He didn't cuddle, but sat upright in my lap, even when I rocked him. But children differ and we were content to let Mahin be himself. We thought it hilarious when my brother, visiting us when Mahin was eight months old, observed "That kid has no social instincts whatsoever." Although Mahin was a first child, he was not isolated. I frequently put him in his playpen in front of the house, where the schoolchildren stopped to play with him as they passed. He ignored them, too.

It was Natasha, a personality kid, born two years later, whose responsiveness emphasized the degree of Mahin's difference. When I went into her room for the late feeding, her little head bobbed up and she greeted me with a smile that reached from her head to her toes. And the realization of that difference chilled me more than the wintry bedroom. Mahin's babbling had not turned into speech by the time he was three. His play was solitary and repetitious. He tore paper into long thin strips, bushel baskets of it every day. He spun the lids from my canning jars and became upset if we tried to divert him. Only rarely could I catch his eye, and then saw his focus change from me to the reflection in my glasses. . . .

[Mahin's] adventures in our suburban neighbourhood had been unhappy. He had disregarded the universal rule that sand is to be kept in sandboxes, and the children themselves had punished him. He walked around a sad and solitary figure, always carrying a toy airplane, a toy he never played with. At that time, I had not heard the word that was to dominate our lives, to hover over every conversation, to sit through every meal beside us. That word was autism.

Adapted from Eberhardy, 1967

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Autism spectrum disorder. One of the most severe childhood disorders, autism spectrum disorder is characterized by pervasive deficits in the ability to relate to and communicate with others, and by a restricted range of activities and interests. Children with autism spectrum disorder lack the ability to relate to others and seem to live in their own private worlds.

autistic thinking The tendency to view oneself as the centre of the universe, to believe that external events somehow refer to oneself.

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Autism derives from the Greek *autos*, meaning "self." The term *autism* was first used in 1906 by Swiss psychiatrist Eugen Bleuler to refer to a peculiar style of thinking among people with schizophrenia. (Autism is one of Bleuler's four As.) **Autistic thinking** is the tendency to view oneself as the centre of the universe, to believe that external events somehow refer to oneself. In 1943, another psychiatrist, Leo Kanner, applied the diagnosis "early infantile autism" to a group of disturbed children who seemed unable to relate to others, as if they lived in their own private worlds. Unlike children suffering from intellectual disability, children with **autism spectrum disorder** seemed to shut out any input from the outside world, creating a kind of "autistic aloneness" (Kanner, 1943).

ASD is a lifelong condition that spans all socioeconomic levels (Fombonne, 2005). It typically becomes evident in toddlers between 18 and 30 months of age (Rapin, 1997), and is four times more common in boys (Fombonne, 2005).

FEATURES OF AUTISM SPECTRUM DISORDER Perhaps the most poignant feature of autism is the child's utter aloneness (see Table 11.1). Other features include language and communication problems and ritualistic or stereotyped behaviour. The child may also be mute, or if some language skills are present, they may be characterized by peculiar usage, as in echolalia (parroting back what the child has heard in a high-pitched monotone); pronoun reversals (using "you" or "he" instead of "I"); use of words that have meaning only to those who have intimate knowledge of the child; and tendencies to raise the voice at the end of sentences, as if asking a question. Nonverbal communication may also be impaired or absent. For example, children with autism spectrum disorder may not engage in eye contact or display facial expressions. Although these children may be unresponsive to others, researchers find them to be capable of displaying strong emotions, especially strong negative emotions such as anger, sadness, and fear (Capps, Kasari, Yirmiya, & Sigman, 1993).

A primary feature of autism spectrum disorder is interminable, repeated, purposeless, stereotyped movements—twirling, flapping the hands, or rocking back and forth with the arms around the knees. Some children with autism spectrum disorder mutilate themselves, even as they cry out in pain. They may bang their head, slap their face, bite their hands and shoulders, or pull out their hair. They may also throw sudden tantrums or panics. Another feature of autism spectrum disorder is an aversion to environmental changes—a feature termed "preservation of sameness." When familiar objects are moved even slightly from their usual places, children may throw tantrums or cry continually until their placement is restored. They may also insist on eating the same food every day.

Children with ASD are bound by ritual. The teacher of one five-year-old girl with autism spectrum disorder learned to greet her every morning by saying, "Good morning, Lily, I am very, very glad to see you" (Diamond, Balvin, & Diamond, 1963). Although Lily would not respond to the greeting, she would shriek if the teacher omitted even one of the *very*s.

Children who develop autism spectrum disorder appear to have failed to develop a differentiated self-concept—a sense of themselves as distinct individuals (Toichi et al., 2002). Despite their unusual behaviour, children/subjects with autism spectrum disorder are often quite attractive and can have an "intelligent look" about them. However, as measured by scores on standardized tests, their intellectual development tends to lag below the norm. International studies indicate that 30% of ASD subjects have mild to moderate levels of intellectual disability and 40% have severe to profound levels (Fombonne, 2005). Even those who function at an average level of intelligence show deficits in activities requiring the ability to symbolize, such as recognize emotions, engage in symbolic play, and problem solve conceptually (Yirmiya & Sigman, 1991). They also display difficulty in attending to tasks that involve interacting with other people.

THEORETICAL PERSPECTIVES Early views of autism spectrum disorder focused on pathological family relationships. Kanner and his colleagues (e.g., Kanner & Eisenberg,

TABLE 11.1

Diagnostic Criteria for Autism Spectrum Disorder

A. Persistent deficits in social communication and social interaction across multiple contexts, as manifested by the following, currently or by history (examples are illustrative, not exhaustive; see text):
   1. Deficits in social-emotional reciprocity, ranging, for example, from abnormal social approach and failure of normal back-and-forth conversation; to reduced sharing of interests, emotions, or affect; to failure to initiate or respond to social interactions.
   2. Deficits in nonverbal communicative behaviors used for social interaction, ranging, for example, from poorly integrated verbal and nonverbal communication; to abnormalities in eye contact and body language or deficits in understanding and use of gestures; to a total lack of facial expressions and nonverbal communication.
   3. Deficits in developing, maintaining, and understanding relationships, ranging, for example, from difficulties adjusting behavior to suit various social contexts; to difficulties in sharing imaginative play or in making friends; to absence of interest in peers.

B. Restricted, repetitive patterns of behavior, interests, or activities, as manifested by at least two of the following, currently or by history (examples are illustrative, not exhaustive; see text):
   1. Stereotyped or repetitive motor movements, use of objects, or speech (e.g., simple motor stereotypies, lining up toys or flipping objects, echolalia, idiosyncratic phrases).
   2. Insistence on sameness, inflexible adherence to routines, or ritualized patterns of verbal or nonverbal behavior (e.g., extreme distress at small changes, difficulties with transitions, rigid thinking patterns, greeting rituals, need to take same route or eat same food every day).
   3. Highly restricted, fixated interests that are abnormal in intensity or focus (e.g., strong attachment to or preoccupation with unusual objects, excessively circumscribed or perseverative interests).
   4. Hyper- or hyporeactivity to sensory input or unusual interest in sensory aspects of the environment (e.g., apparent indifference to pain/temperature, adverse response to specific sounds or textures, excessive smelling or touching of objects, visual fascination with lights or movement).

C. Symptoms must be present in the early developmental period (but may not become fully manifest until social demands exceed limited capacities, or may be masked by learned strategies in later life).

D. Symptoms cause clinically significant impairment in social, occupational, or other important areas of current functioning.

E. These disturbances are not better explained by intellectual disability (intellectual developmental disorder) or global developmental delay. Intellectual disability and autism spectrum disorder frequently co-occur; to make comorbid diagnoses of autism spectrum disorder and intellectual disability, social communication should be below that expected for general developmental level.

Note: Individuals with a well-established DSM-IV diagnosis of autistic disorder, Asperger's disorder, or pervasive developmental disorder not otherwise specified should be given the diagnosis of autism spectrum disorder. Individuals who have marked deficits in social communication, but whose symptoms do not otherwise meet criteria for autism spectrum disorder, should be evaluated for social (pragmatic) communication disorder.

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1955) suggested that children with the disorder were reared by cold, detached parents who were dubbed "emotional refrigerators." Psychoanalyst Bruno Bettelheim (1967) also focused on the family by suggesting that extreme self-absorption is the child's defence against parental rejection. The parents rear the child in an emotionally and socially desolate atmosphere in which the child's efforts to develop language and social skills wither. The child surrenders efforts to develop mastery over the external world and withdraws into a world of fantasy. The pathological insistence on preservation of sameness represents the child's rigid, defensive efforts to impose order and predictability.

Research, however, has not supported the devastating assumption that parents of children with autism spectrum disorder are frosty and remote (Hoffmann & Prior, 1982). Of course, there is truth to the notion that children with autism spectrum disorder and their parents do not relate to one another very well, but causal connections are clouded. Rather than rejecting their children and thus fostering the disorder, parents may grow somewhat aloof because their efforts to relate to their children repeatedly meet with failure. Aloofness then becomes a result of the disorder, not a cause.

Psychologist O. Ivar Lovaas and his colleagues (Lovaas, Koegel, & Schreibman, 1979) offer a cognitive-behavioural perspective on autism spectrum disorder. They suggest that children with ASD have perceptual deficits that limit them to processing only one stimulus

at a time. As a result, they are slow to learn by means of classical conditioning (association of stimuli). From the learning theory perspective, children become attached to their primary caregivers because they are associated with primary reinforcers such as food and hugging. Children with autism spectrum disorder, however, attend either to the food or to the cuddling and do not connect it with the parent.

Cognitive theorists have focused on the kinds of cognitive deficits shown by children with ASD and the possible relationships among these deficits. Children with autism spectrum disorder appear to have difficulty integrating information from various senses (Rutter, 1983). At times, they seem hypersensitive to stimulation. At other times they are so insensitive that an observer might wonder whether they are deaf. Perceptual and cognitive deficits seem to diminish their capacity to make use of information—to comprehend and apply social rules. This may impede the development of what psychologists call a **theory of mind**. Theory of mind is the ability to appreciate that other people have a mental state that is different from one's own. Children with autism spectrum disorder show deficits in their ability to infer beliefs, intentions, and emotions in others (Baron-Cohen, 1995, 1998). Not being able to readily see the world from another person's perspective interferes with the normal give and take of social relationships.

theory of mind The ability to appreciate that other people have a mental state that is different from one's own.

But what is the basis of these perceptual and cognitive deficits? Canadian experts who have reviewed the research into the causes of autism spectrum disorder have concluded that the many impairments associated with autism spectrum disorder, including intellectual disability, communications deficits, repetitive bizarre motor behaviour, and even seizures, suggest an underlying neurobiological basis (Nicolson & Szatmari, 2003). MRI scans show that compared to normal children, children with autism spectrum disorder have a period of overgrowth of brain size early in postnatal development, especially in the frontal regions. This period is followed by significantly slowed growth resulting in a brain volume smaller than average for children aged 5 to 16. The brain tissue that connects the two halves of the brain, the corpus callosum, is smaller than normal in patients with autism spectrum disorder, which may affect **lateralization** of brain function. The area of the brain that regulates motor function, the cerebellum, also shows abnormal development in individuals with autism spectrum disorder.

lateralization The developmental process by which the left hemisphere specializes in verbal and analytic functions and the right hemisphere specializes in nonverbal, spatial functions.

Nicolson and Szatmari's (2003) review of the research indicates that there is substantial support for the suggestion that genetics plays a significant role in the neurodevelopment of children with autism spectrum disorder, with susceptible genes on chromosomes 2 and 7 in particular. It may be that multiple genes are involved and that they interact with other factors, possibly environmental or biological in origin, leading to autism spectrum disorder (McIntosh, 1999).

Recently, researchers have found that the mirror neurons—circuits that activate in similar ways when we perform actions or watch other people perform the same actions—in those with autism spectrum disorder are not as fully developed and may be the reason for social deficits (Bastiaansen et al., 2011). Also, brain scans are revealing the disorder's characteristic signature. Researchers from Yale University have identified three distinct neural signatures: trait markers, which are brain regions with reduced activity in children with autism spectrum disorder and their unaffected siblings; state markers, which are brain areas with reduced activity found only in children with autism spectrum disorder; and compensatory activity, which is enhanced activity seen only in unaffected siblings. The enhanced brain activity may reflect a developmental process by which these children overcome a genetic predisposition to develop autism spectrum disorder (Kaiser et al., 2010). This discovery may lead to earlier diagnosis of autism spectrum disorder. Early diagnosis tools are crucial to early intervention programs ("Children With Autism," 2011; "New Research Tool," 2008).

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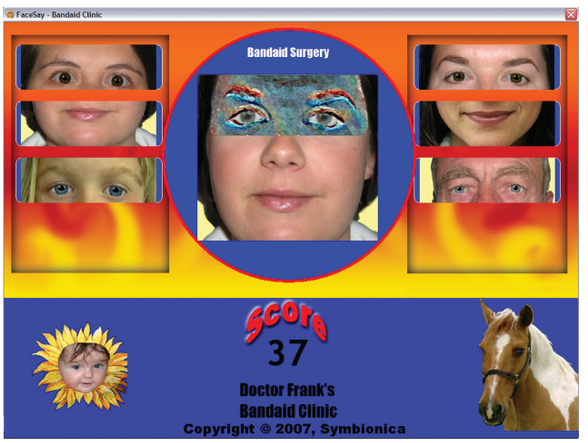
Establishing contact. One of the principal therapeutic tasks in working with children with autism spectrum disorder is the establishment of interpersonal contact. Behaviour therapists use reinforcers to increase adaptive social behaviours, such as paying attention to the therapist and playing with other children. Behaviour therapists may also use punishments to suppress self-mutilative behaviour.

The Early Autism Study, led by Mel Rutherford at McMaster University, is developing an early diagnosis tool using eye-tracker technology that measures eye direction while babies look at faces, eyes, and bouncing balls on a computer screen. These researchers are finding that they can distinguish between a group of siblings with the disorder from a group without the disorder at 9 months and 12 months ("New Research Tool," 2008).

Still, the cause of ASD remains unknown, and some recent controversial but scientifically plausible ideas are being examined. Some ideas point to the immune system, to viruses, and to an overload of hormones.



FaceSay images provided courtesy of Symbionica, LLC. All rights reserved.

Learning to recognize emotions through computer programs. An interactive computer game called FaceSay™, by Symbionica, helps children with autism spectrum disorder learn where to look for clues to emotions on faces and thereby learn how to recognize emotions in others.

TREATMENT Although there is no cure for autism spectrum disorder, structured treatment programs have yielded the best results. The most effective treatment programs focus on behavioural, educational, and communication deficits and are highly intensive and structured, offering a great deal of individual instruction (Rapin, 1997). In a classic study conducted by Lovaas (1987) at UCLA, children suffering from autism spectrum disorder received more than 40 hours of one-to-one behaviour modification each week for at least two years. Significant intellectual and educational gains were reported for 9 of the 19 children (47%) in the program. The children who improved achieved normal IQ scores and were able to succeed in grade 1. Only 2% of a control group that did not receive the intensive treatment achieved similar gains. Treatment gains were well maintained at the time of a follow-up when the children were 11 years old (McEachin, Smith, & Lovaas, 1993).

These psychological intervention programs are effective for many children with autism spectrum disorder, but the key point is that any intervention must begin early. Research continues to support the necessity of early intervention programs; the more intense or comprehensive the therapy, the better it is in terms of helping children improve social and communication skills. Those who received therapy, including behavioural, speech, and occupational therapy, had the best outcomes in a recent study of over 1000 children with autism spectrum disorder (Mazurek, Kanne, & Miles, 2012). Children who are better functioning at the start of treatment typically gain the most.

Biological approaches have had only limited impact in the treatment of autism spectrum disorder. One line of research has focused on drugs normally used to treat schizophrenia, such as Haldol, which blocks dopamine activity. Several controlled studies show Haldol to be helpful in many cases in reducing social withdrawal and repetitive motor behaviour (such as rocking), aggression, hyperactivity, and self-injurious behaviour (McCracken et al., 2002). We have not seen drugs lead to consistent improvement in cognitive and language development in children with autism spectrum disorder, however.

An interactive computer program called FaceSay™, by Symbionica, LLC, has been shown to improve the ability of children with autism spectrum disorder to recognize faces, facial expressions, and emotions, according to the results of a study conducted by psychologists at the University of Alabama at Birmingham (Hopkins et al., 2011). FaceSay™ features interactive games that let children with autism spectrum disorder practise recognizing the facial features and expressions of an avatar, or software "puppet." Specifically, the computer program teaches the children where to look for facial cues such as an eye gaze or a facial expression.

Traits in autism spectrum disorder generally continue into adulthood to one degree or another. Yet some children with autism spectrum disorder do go on to achieve college and university degrees and are able to function independently (Rapin, 1997). Others need continuing treatment throughout their lives, even institutionalized care. Even the highest-functioning adults with the disorder manifest deficient social and communication skills and a highly limited range of interests and activities (APA, 2013).

REVIEW IT

Austism Spectrum Disorder

- **What is autism spectrum disorder?** Autism spectrum disorder is characterized by pervasive deficits in the ability to relate to and communicate with others, and by a restricted range of activities and interests.
- **What are the clinical features of autism spectrum disorder?** Children with autism spectrum disorder shun affectionate behaviour, engage in stereotyped behaviour, attempt to preserve sameness, and tend to have peculiar speech habits such as echolalia, pronoun reversals, and idiosyncratic speech. The causes of the disorder remain unknown, but gains in academic and social functioning have been obtained through the use of early intensive behaviour therapy.

INTELLECTUAL DISABILITY (INTELLECTUAL DEVELOPMENTAL DISORDER)

Intellectual disability, also called intellectual developmental disorder, involves a broad delay in the development of cognitive and social functioning. The course of development of children with intellectual disability is variable. Many improve over time, especially if they receive support, guidance, and enriched educational opportunities. Children with intellectual disability who are reared in impoverished environments may fail to improve or may deteriorate further in relation to other children.

Intellectual disability is generally assessed by a combination of formal intelligence tests and observation of adaptive functioning. The DSM-5 uses three criteria in diagnosing mental intellectual disability: (1) deficits in intellectual functions as indicated by clinicians and standardized testing, (2) evidence of impaired functioning in adaptive behaviour, and (3) onset of the disorder in the developmental period. People whose behaviour is impaired fail to meet the standards of behaviour that are expected of someone of the same age within a given cultural setting. They do not develop comparable social and communication skills or become adequately independent and self-sufficient. For infants, task-related judgments of subaverage intellectual functioning may be used in place of IQ scores because tests of infant intelligence either do not yield reliable IQ scores or do not yield any IQ scores at all.

The DSM-5 classifies intellectual disability according to level of severity. Table 11.2 provides a description of the deficits and abilities associated with various degrees of intellectual

TABLE 11.2

Classifications of Developmental Delay

Classification	Range of IQ Scores	Adaptive Limitations	Percentage of Developmentally Delayed Population
Mild	55–70	Can reach grade 6 skill level. Capable with training of living independently and being self-supporting.	90%
Moderate	40–55	Can reach grade 2 skill level. Can work and live in sheltered environments with supervision.	6%
Severe	25–40	Can learn to talk and perform basic self-care but needs constant supervision.	3%
Profound	Below 25	Very limited ability to learn; may only be able to learn very simple tasks; poor language skills and limited self-care.	1%

Source: Ciccarelli, Harrigan, & Fritzley, 2010, p. 339. Reprinted with permission by Pearson Canada Inc.

disability. Children with mild intellectual disability are generally capable of meeting basic academic demands such as learning to read simple passages. As adults, they are generally capable of independent functioning, although they may require some guidance and support.

A 2002 study of adolescents conducted in the Niagara Region found results comparable to earlier Canadian and international studies (Bradley, Thompson, & Bryson, 2002). The researchers identified the overall prevalence of intellectual disability as 7.18 in 1000. Using broad criteria, they found that the prevalence for mild intellectual disability (IQ = 50–75) was 3.54 in 1000; for severe intellectual disability (IQ under 50) it was 3.64 in 1000. Although the overall rates of mild and severe intellectual disability in adolescents were nearly equivalent, the number of individuals with mild intellectual disability declined slightly with age, whereas the rates of severe intellectual disability remained constant. The authors caution that the lower-than-expected rate of mild intellectual disability may be related to Ontario's integration policy for education, which has made individuals with mild intellectual disability less visible as they adapt to the mainstream classroom, albeit with a modified program of study. Moreover, once formal schooling is completed, people with mild intellectual disability blend into the community much more readily than people with severe intellectual disability. Males in the study outnumbered females by a ratio of 1.3 to 1.0, but more females fell in the range of severe intellectual disability.

Not all systems of classification of intellectual disability are based on level of severity. The American Association on Intellectual and Developmental Disabilities, formerly the American Association on Mental Retardation (AAMR), an organization composed of leading professionals in the field, classifies intellectual disability according to the intensity of support needed by the individual in various areas of functioning (AAMR, 1992). Some individuals need only intermittent support that varies in intensity from time to time on an as-needed basis, whereas others require more constant or pervasive support involving extensive commitment of staff and resources. This system of classification attempts to match the level of support needed to the individual's ability to function in work, school, and home environments.

Down syndrome Condition caused by a chromosomal abnormality involving an extra chromosome on the 21st pair (trisomy 21); it is characterized by intellectual disability and various physical abnormalities. Formerly called *mongolism* and *Down's syndrome* in Canada.

Causes of Intellectual Disability

In many cases, intellectual disability can be traced to biological causes, including chromosomal and genetic disorders, infectious diseases, and brain damage. Specifically, where the intellectual disability is due to genes, geneticists have uncovered several genes that have been identified to cause this disorder. However, it is not one or two genes that cause intellectual disability but spontaneous mutations in the genes, as demonstrated in new research by Dutch geneticists. The majority of intellectual disability is caused by spontaneous mutations in paternal sperm or maternal egg cells (Vissers et al., 2010). To be clear, intellectual disability is not transmitted from one generation to the next but occurs through spontaneous genetic changes such as deletions and duplications (Webber et al., 2009). The gene in which the child has a defect still shows a normal function in both parents.

Unexplained cases might involve cultural or familial causes, such as being raised in an impoverished home environment, or perhaps they involve an interaction of environmental and genetic factors, the nature of which remains poorly understood (Thapar, Gottesman, Owen, O'Donovan, & McGuffin, 1994).

DOWN SYNDROME AND OTHER CHROMOSOMAL ABNORMALITIES The most common chromosomal abnormality resulting in intellectual disability is **Down syndrome**, which is characterized by an extra or third chromosome on the 21st pair of chromosomes, resulting in 47 chromosomes rather than the normal complement of 46. Down syndrome occurs in about 1 in 800 births. It usually occurs when the 21st pair of chromosomes in either the egg or the

CP Photo/Michael Yarish/Everett Collection

Lauren Potter. Lauren Potter plays Becky Jackson in the hit television series *Glee*.

sperm fails to divide normally, resulting in an extra chromosome. Chromosomal abnormalities become more likely as parents age, so expectant couples in their mid-30s or older often undergo prenatal genetic tests to detect Down syndrome and genetic abnormalities. Down syndrome can be traced to a defect in the mother's chromosomes in about 95% of cases (Antonarakis & Down Syndrome Collaborative Group, 1991), with the remainder attributable to defects in the father's sperm.

People with Down syndrome are recognizable by certain physical features, such as a round face; broad, flat nose; and small, downward-sloping folds of skin at the inside corners of the eyes that give the impression of slanted eyes. Children with Down syndrome are also characterized by a protruding tongue; small, squarish hands and short fingers; a curved fifth finger; and disproportionately small arms and legs in relation to their bodies. Nearly all of these children have intellectual disability, and many suffer from physical problems, such as malformations of the heart and respiratory difficulties. Sadly, most die by middle age. In their later years, they tend to suffer memory losses and experience childish emotions that represent a form of senility.

Children with Down syndrome suffer various deficits in learning and development. They tend to be unco-ordinated and to lack proper muscle tone, which makes it difficult for them to carry out physical tasks and engage in play activities like other children. Down syndrome children suffer memory deficits, especially for information presented verbally, which makes it difficult for them to learn in school. They also have difficulty following instructions from teachers and expressing their thoughts or needs clearly in speech. Despite their disabilities, most can learn to read, write, and perform simple arithmetic if they receive appropriate schooling and the right encouragement.

Although less common than Down syndrome, chromosomal abnormalities on the sex chromosome may also result in intellectual disability, such as in Klinefelter's syndrome and Turner's syndrome. Klinefelter's syndrome, which occurs only in males, is characterized by the presence of an extra X sex chromosome, resulting in an XXY sex chromosomal pattern rather than the XY pattern that men normally have. Estimates of the prevalence of Klinefelter's syndrome range from 1 in 500 to 1 in 1000 male births (Brody, 1993). Men with this XXY pattern fail to develop appropriate secondary sex characteristics, resulting in small, underdeveloped testes; low sperm production; enlarged breasts; poor muscular development; and infertility. Mild intellectual disability or learning disabilities frequently occur among these men. Men with Klinefelter's syndrome often don't discover they have the condition until they undergo tests for infertility.

Turner's syndrome is found exclusively in females and is characterized by the presence of a single X sex chromosome instead of the normal two. Although such girls develop normal external genitals, their ovaries remain poorly developed, producing reduced amounts of estrogen. As women, they tend to be shorter than average and infertile. They also tend to show evidence of mild intellectual disability, especially in skills relating to math and science.

FRAGILE X SYNDROME AND OTHER GENETIC ABNORMALITIES Fragile X syndrome is the most common type of inherited (genetic) intellectual disability. It is the second most common form of intellectual disability overall after Down syndrome ("Blood Test Can Detect Retardation," 1993; Plomin, Owen, & McGuffin, 1994). The disorder is believed to be caused by a mutated gene on the X sex chromosome (Huber, Gallagher, Warren, & Bear, 2002). The defective gene is located in an area of the chromosome that appears fragile, hence the name *fragile X syndrome*. Fragile X syndrome causes intellectual disability in about 1 in every 1000 to 1500 males and (generally less severe) intellectual disability in about 1 in every 2000 to 2500 females (Angier, 1991b; Rousseau et al., 1991). The effects of fragile X syndrome range from mild learning disabilities to intellectual disability so profound that those affected can hardly speak or function.

Females normally have two X sex chromosomes, whereas males have only one. For females, having two X sex chromosomes seems to provide some protection against the disorder if the defective gene turns up on one of the two chromosomes (Angier, 1991b). This may explain why the disorder usually has more profound effects on males than on females. Yet the mutation does not always manifest itself. Many males and females carry the fragile X mutation but show no clinical evidence of it. Nevertheless, they can pass along the syndrome to their offspring.

A genetic test can detect the presence of the mutation by direct DNA analysis and may help prospective parents who seek out genetic counselling. Prenatal testing of the fetus is also available. Although there is no treatment for fragile X syndrome, identifying the defective gene is the first step toward understanding how the protein produced by the gene functions to create the disability—which may lead to the development of treatments (Huber et al., 2002).

Phenylketonuria (PKU) is a genetic disorder that occurs in 1 in 10 000 births (Plomin et al., 1994). It is caused by a recessive gene that prevents the child from metabolizing the amino acid phenylalanine (Phe), which is found in many foods. Consequently, phenylalanine and its derivative, phenylpyruvic acid, accumulate in the body, causing damage to the central nervous system that results in intellectual disability and emotional disturbance. PKU can be detected in newborns by analyzing blood or urine samples. Although there is no cure for PKU, children with the disorder may suffer less damage or develop normally if they are placed on a diet low in phenylalanine soon after birth (Brody, 1990). Such children receive protein supplements that compensate for their nutritional loss.

phenylketonuria Genetic disorder that prevents the metabolization of phenylpyruvic acid, leading to intellectual disability. Abbreviated *PKU*.

University of Toronto researchers Mary Lou Smith and her colleagues tested children with PKU who had either high or low levels of Phe (M. L. Smith, Klim, & Hanley, 2000). They found that PKU children with low Phe levels were indistinguishable from non-PKU controls on several cognitive tasks. In contrast, however, the higher the levels of Phe in school-aged children with PKU, the greater the impairment in cognitive performance on specific problem-solving and verbal memory tasks.

Tay-Sachs disease is caused by recessive genes on chromosome 15. A fatal degenerative disease of the central nervous system, it mostly afflicts Jews of Eastern European ancestry and French Canadians of the Gaspé region of Quebec (Triggs-Raine, Richard, Wasel, Prence, & Natowicz, 1995). About 1 in 14 French Canadians in Eastern Quebec is a carrier of the recessive gene responsible for the disorder (Chodirker et al., 2001; Kaback et al., 1993). Children afflicted by Tay-Sachs suffer gradual loss of muscle control, deafness and blindness, intellectual disability, and paralysis, and usually die before the age of five.

Tay-Sachs disease Disease of lipid metabolism that is genetically transmitted and usually results in death in early childhood.

Today, various prenatal diagnostic tests can detect the presence of chromosomal abnormalities and genetic disorders. In amniocentesis, which is usually conducted about 14 to 15 weeks following conception, a sample of amniotic fluid is drawn with a syringe from the amniotic sac that contains the fetus. With chorionic villus sampling (CVS), cells are extracted from the placenta outside the sac where the fetus develops. CVS is best performed between 10 and 12 weeks into a woman's pregnancy. Depending upon the procedure, cells from the fetus or the placenta can then be examined for abnormalities, including Down syndrome, X-linked disorders, PKU, Smith-Lemli-Optiz syndrome, and Tay-Sachs.

In the future, it may be possible to control the impact of defective genes during prenatal development. For now, expectant couples rely on genetic counselling. It offers a complete and accurate view of the options available and can assist couples in making informed decisions about terminating a pregnancy or, alternatively, help them prepare for a baby who has congenital defects.

PRENATAL FACTORS Some cases of intellectual disability are caused by maternal infections or substance abuse during pregnancy. Rubella (German measles) in the mother, for example, can be passed along to the unborn child, causing brain damage that results in intellectual disability, and may play a role in autism spectrum disorder. Although the mother might experience only mild symptoms or none at all, the effects on the fetus can be tragic. Other maternal diseases that can cause intellectual disability in the child include syphilis, **cytomegalovirus**, and genital herpes.

cytomegalovirus Maternal disease of the herpes virus group that carries a risk of intellectual disability to the unborn child.

Widespread programs that immunize women against rubella before pregnancy and tests for syphilis during pregnancy have reduced the risk of transmission of these infections to children. Most children who contract genital herpes from their mothers do so during delivery by coming into contact with the herpes simplex virus that causes the disease in the birth canal. Caesarean sections (C-sections) reduce the risk of the baby's coming into contact with the virus during outbreaks.

Drugs the mother ingests during pregnancy are able to pass through the placenta to the child. Some can cause severe birth deformities and intellectual disability. Children whose mothers drink alcohol during pregnancy are often born with fetal alcohol syndrome (FAS). FAS is among the most prominent causes of intellectual disability. Maternal

smoking during pregnancy has also been linked to the development of attention-deficit/hyperactivity disorder in children (Milberger, Biederman, Faraone, Chen, & Jones, 1996).

Birth complications, such as oxygen deprivation or head injuries, place children at increased risk for neurological disorders, including intellectual disability. Prematurity also places children at risk of intellectual disability and other developmental problems. Brain infections, such as encephalitis and meningitis, or traumas during infancy and early childhood can cause intellectual disability and other health problems. Children who ingest toxins, such as paint chips containing lead, may also suffer brain damage that produces intellectual disability.

CULTURAL-FAMILIAL CAUSES Children with intellectual disability fall mainly into the mild range of severity, and there is no apparent biological cause or distinguishing physical feature that sets these children apart from other children. Psychosocial factors, such as an impoverished home, a social environment that is not intellectually stimulating, or parental neglect or abuse, may play a causal or contributing role in the development of intellectual disability in such children. Supporting a family linkage is evidence from a study in Atlanta in which mothers who failed to finish high school were four times more likely than better-educated mothers to have children with mild intellectual disability (Drews, Yeargin-Allsopp, Decouflé, & Murphy, 1995).

cultural-familial intellectual disability Milder form of intellectual disability that is believed to result, or at least be influenced by, impoverishment in the child's home environment.

These cases are considered **cultural-familial intellectual disability**. Children in impoverished families may lack toys, books, or opportunities to interact with adults in intellectually stimulating ways. Consequently, they may fail to develop appropriate language skills or become unmotivated to learn the skills that are valued in contemporary society. Economic burdens, such as the need to hold multiple jobs, may prevent the parents from spending time reading to them, talking to them at length, and exposing them to creative play or trips to museums and parks. Children may spend most of their days glued to the television set. The parents, most of whom were also reared in poverty, may lack the reading or communication skills to help shape the development of these skills in their children. A vicious cycle of poverty and impoverished intellectual development may be repeated from generation to generation.

Children with this form of developmental delay may respond dramatically when provided with enriched learning experiences, especially at earlier ages. For example, the Health Canada–funded Aboriginal Head Start (AHS) program was instituted in 114 urban centres and northern communities across Canada to promote education and school readiness, Aboriginal culture and language, parental involvement, health, nutrition, and social support (Health Canada, 2002a). To date, the AHS program has been highly successful and has exceeded program expectations as major gains have been achieved in all areas of children's development (Health Canada, 2000a; Indian and Northern Affairs, 2003; Ottawa Aboriginal Head Start Program, 1998).

Intervention

The services that children with intellectual disability require to meet the developmental challenges they face depend in part on the type of intellectual disability and the level of severity (Dykens & Hodapp, 1997; Snell, 1997). With appropriate training, children with mild intellectual disability may approach a grade 6 level of competence. They can acquire vocational skills that allow them to support themselves minimally through meaningful work. In Canada, most mildly intellectually disabled children are integrated into the regular classroom, typically with a modified curriculum. Children with more severe forms of intellectual disability, on the other hand, may be placed in special schools or classes if the caregiver prefers (Bradley et al., 2002).

mainstreaming The practice of having all students with disabilities included in the regular classroom. Also referred to as *integration* or *inclusion*.

Controversy remains over the **mainstreaming** of children with intellectual disability into regular classes. Although some children with mild intellectual disability may achieve better when they are mainstreamed, others may not do so well in regular classes. Some of these children find regular classes overwhelming and they withdraw from their schoolmates. There has also been a trend in Canada and the United States

A CLOSER LOOK

Savant Syndrome

Got a minute? Try the following:

1. Without referring to a calendar, calculate the day of the week that March 15, 2079, will fall on.
2. List the prime numbers between 1 and 1 billion. (Hint: The list starts 1, 2, 3, 5, 7, 11, 13, 17, . . .)
3. Repeat verbatim the newspaper stories you read over coffee this morning.
4. Sing accurately every note played by the first violin in Beethoven's Ninth Symphony.

These tasks are impossible for all but a very few. Ironically, people who are most likely to be able to accomplish these feats suffer from autism spectrum disorder, intellectual disability, or both. Such a person is commonly called an idiot savant. The term *savant* is derived from the French *savoir*, meaning "to know." The label *savant syndrome* is preferable to the pejorative *idiot savant* in referring to someone with severe mental deficiencies who possesses some remarkable mental abilities. The prevalence of savant syndrome among people with intellectual disability is estimated at about 0.06%, or about 1 case in 2000 (Hill, 1987). The emergence of savant syndrome is also closely linked to infantile autism (L. K. Miller, 1999). Most people with savant syndrome, like most people with autism spectrum disorder, are male (Treffert, 1988). Among a sample of 5400 people with autism spectrum disorder, 531 cases (9.8%) were reported by parents to have savant syndrome (Rimland, 1978). Because they want to think of their children as special, however, parents might overreport the incidence of savant syndrome.

The savant syndrome phenomenon occurs more frequently in males by a ratio of roughly 6 to 1. The special skills of people with savant syndrome tend to appear out of the blue and may disappear as suddenly. Some people with the syndrome engage in lightning calculations. Thomas Fuller, a 19th-century enslaved man in Virginia, "was able to calculate the number of seconds in 70 years, 17 days, and 12 hours in a minute and one half, taking into account the 17 leap years that would have occurred in the period" (S. B. Smith, 1983). There are also cases of persons with the syndrome who were blind but could play back any musical piece, no matter how complex, or repeat long passages of foreign languages without losing a syllable.

Various theories have been presented to explain savant syndrome (Treffert, 1988). Some experts believe that children with savant syndrome have unusually well-developed memories that allow them to record and scan vast amounts of information. It has been suggested that people with savant syndrome may inherit two sets of hereditary factors, one for intellectual disability and the other for special abilities. Perhaps it is coincidental that their special abilities and their mental handicaps were inherited in common. Other theorists suggest that the left and right hemispheres of their cerebral cortex are organized in an unusual way. This latter belief is supported by research suggesting that the special abilities these individuals possess often involve skills associated with right hemisphere functioning. Still other theorists suggest that they learn special skills to compensate for their lack of more general skills, perhaps as a means of coping with their environment or of earning social reinforcements. It could be that their skills in concrete functions, such as calculation, compensate for their lack of abstract thinking ability. Perhaps, as the neurologist Oliver Sacks speculates, the brain circuits of some people with savant syndrome are wired with a "deep arithmetic"—an innate structure for perceiving mathematical relationships that is analogous to the prewiring that allows people to perceive and produce language.

Some earlier research has pointed to possible gender-linked left hemisphere damage occurring prenatally or congenitally. Compensatory right hemisphere development might then take place, establishing specialized brain circuitry that processes concrete and narrowly defined kinds of information (Treffert, 1988). An environment that reinforces savant abilities and provides opportunities for practice and concentration would give further impetus to the development of these unusual abilities. As it stands, savant syndrome remains a mystery.

Stephen Vaughan/Globe Photos Inc.

Savant syndrome. Dustin Hoffman (left) won the Best Actor Oscar for his portrayal in the 1988 film *Rain Man* of a man with autism who showed a remarkable capacity for numerical calculation. Tom Cruise played his brother. Hoffman was able to capture a sense of emotional detachment and isolation in his character.

Moodboard_Images/iStockphoto

Inclusion. Across Canada, most children with special learning needs remain in regular classrooms and are provided with educational programs that meet their individual needs.

toward deinstitutionalization of people with more severe intellectual disability, a policy shift motivated in large part by public outrage over the appalling conditions that existed in many institutions serving this population.

People with intellectual disability who are capable of functioning in the community have the right to receive less restrictive care than is provided in large institutions. Many are capable of living outside an institution and have been placed in supervised group homes. Residents typically share household responsibilities and are encouraged to participate in meaningful daily activities, such as training programs or sheltered workshops. Others live with their families and attend structured day programs. Adults with intellectual disability often work in outside jobs and live in their own apartments or share apartments with other persons who have mild intellectual disability. Behavioural approaches can be used to teach persons with more severe intellectual disability such basic hygienic behaviours as toothbrushing, self-dressing, and hair combing. In demonstrating toothbrushing, the therapist might first define the component parts of the targeted behaviour (picking up the toothbrush, wetting the toothbrush, taking the cap off the tube, putting the paste on the brush, and so on) (Kissel, Whitman, & Reid, 1983). The therapist might then shape the desired behaviour by using such techniques as verbal instruction (e.g., "Jim, pick up the toothbrush"), physical guidance (physically guiding the client's hand in performing the desired response), and reward (use of positive verbal reinforcement) for successful completion of the desired response ("That's really good, Jim"). Such behavioural techniques have been shown to be effective in teaching a simple but remunerative vocational skill (for example, stamping return addresses on envelopes) to a group of adult women with such severe intellectual disability that they were essentially nonverbal (Schepis, Reid, & Fitzgerald, 1987). These techniques may also help people with severe intellectual disability develop adaptive capacities that can enable them to perform more productive roles.

Other behavioural treatment techniques include social-skills training, which focuses on increasing the individual's ability to relate co-operatively with others, and anger-management training to help individuals develop more effective ways of handling conflicts than aggressively acting out (Huang & Cuvo, 1997).

Children with intellectual disability stand perhaps a three to four times greater chance of developing other psychological disorders, such as attention-deficit/hyperactivity disorder (ADHD), depression, or anxiety disorders (Borthwick-Duffy, 1994). Mental health professionals have been slow to recognize the prevalence of mental health problems among people with intellectual disability, perhaps because of a long-held conceptual distinction between emotional impairment on the one hand and intellectual deficits on the other (Nezu, 1994). Many professionals even assumed (wrongly) that people with intellectual disability were worry free and somehow immune to psychological problems (Nezu, 1994). Given these commonly held beliefs, it is perhaps not surprising that many of the psychological problems of people with intellectual disability have gone unrecognized and untreated (Reiss & Valenti-Hein, 1994).

Children and adults with intellectual disability may need psychological counselling to help them adjust to life in the community. Many have difficulty making friends and may become socially isolated. Problems with self-esteem are also common, especially because people who have intellectual disability are often demeaned and ridiculed. Supportive counselling may be supplemented with behavioural techniques to help those with developmental disabilities acquire skills in areas such as personal hygiene, work, and social relationships.

REVIEW IT

Intellectual Disability

- **What is intellectual disability, and how is it assessed?** Intellectual disability is a general delay in the development of intellectual and adaptive abilities. It is assessed through evaluation of performance on intelligence tests and measures of functional ability. Most cases fall in the range of mildly intellectually disabled.
- **What are the causes of intellectual disability?** There are many causes of intellectual disability, including chromosomal abnormalities such as Down syndrome; genetic disorders such as fragile X syndrome, phenylketonuria, and Tay-Sachs disease; prenatal factors such as maternal diseases and alcohol (and other drug) use; and familial/cultural factors associated with intellectually impoverished home environments.

LEARNING DISORDERS

Many famous scientists, leaders, and celebrities have been thought to have what are now considered learning disorders. Among their number are Albert Einstein, Alexander Graham Bell, Winston Churchill, Agatha Christie, and Tom Cruise. These highly creative and successful people suffered from **dyslexia** and related disorders. The term *dyslexia* is derived from the Greek roots *dys-*, meaning "bad," and *lexikon*, meaning "of words." Dyslexia is the most common type of **learning disorder** (also called a *learning disability*) (Shaywitz, 1998). It accounts for roughly 80% of learning-disability cases. People with learning disorders have average or higher intelligence, and may even be gifted, but show inadequate development in reading, math, or writing skills that impairs school performance or daily activities. (See the Closer Look box on page 400 for a Canadian definition of *learning disabilities*.)

dyslexia Type of learning disorder characterized by impaired reading ability that may involve difficulty with the alphabet or spelling.

learning disorder Deficiency in a specific learning ability noteworthy because of the individual's general intelligence and exposure to learning opportunities.

About 12% of children in Canadian schools are identified as having a learning disorder (Lipps & Frank, 1997). Learning disorders tend to run a chronic course and are the most common long-term conditions of children up to age 14 (Cossette & Duclos, 2002). The more severe the problem is in childhood, the more likely it is to affect adult development (Spreen, 1988). Children with learning disorders tend to perform poorly in school; about half of all children who received remedial education were identified as having a learning disability (Lipps & Frank, 1997).

Specific Learning Disorders

DSM-5 classifies learning disorders as a single disorder with three subtypes: impairment in reading, impairment in written expression, and impairment in mathematics.

IMPAIRMENT IN MATHEMATICS Impairment in mathematics is a specific learning disorder that characterizes children with deficiencies in arithmetic skills. They may have problems understanding basic mathematical terms or operations, such as addition or subtraction; decoding mathematical symbols (+, =, etc.); or learning sequential facts, such as multiplication tables, by rote memory. The problem may become apparent as early as grade 1 (age 6) but is not generally recognized until about grade 3 (age 8).

IMPAIRMENT IN WRITTEN EXPRESSION Impairment in written expression—dysgraphia—characterizes children with grossly deficient writing skills and occurs regardless of the ability to read. It is not related to intellectual impairment. The deficiency may be characterized by errors in spelling, grammar, or punctuation or by difficulty in composing sentences and paragraphs. Severe writing difficulties generally become apparent by age 7 (grade 2), although milder cases may not be recognized until age 10 (grade 5) or later. Dysgraphia is known as one of the most common learning disabilities, with a prevalence of 4% to 10%. It often overlaps with other learning disabilities such as speech impairment, ADHD, or developmental co-ordination disorder (Nicolson & Fawcett, 2011; Nicolson & Szatmari, 2003).

A CLOSER LOOK

A Canadian Definition of Learning Disabilities

On January 30, 2002, after years of deliberation, the Learning Disabilities Association of Canada (LDAC) adopted an official definition of learning disabilities that is the culmination of a thorough review of learning disabilities research and input from hundreds of individuals in all provinces and territories by the LDAC National Legal Committee and the LDAC "Think Tank" (Learning Disabilities Association of Canada, 2002):

> Learning disabilities refer to a number of disorders that may affect the acquisition, organization, retention, understanding, or use of verbal or nonverbal information. These disorders affect learning in individuals who otherwise demonstrate at least average abilities essential for thinking and/or reasoning. As such, learning disabilities are distinct from global intellectual deficiency.
>
> Learning disabilities result from impairments in one or more processes related to perceiving, thinking, remembering, or learning. These include but are not limited to: language processing; phonological processing; visual spatial processing; processing speed; memory and attention; and executive functions (e.g., planning and decision-making).
>
> Learning disabilities range in severity and may interfere with the acquisition and use of one or more of the following:
>
> - oral language (e.g., listening, speaking, understanding);
> - reading (e.g., decoding, phonetic knowledge, word recognition, comprehension);
> - written language (e.g., spelling and written expression); and
> - mathematics (e.g., computation, problem solving).
>
> Learning disabilities may also involve difficulties with organizational skills, social perception, social interaction, and perspective taking.
>
> Learning disabilities are lifelong. The way in which they are expressed may vary over an individual's lifetime, depending on the interaction between the demands of the environment and the individual's strengths and needs. Learning disabilities are suggested by unexpected academic under-achievement or achievement that is maintained only by unusually high levels of effort and support.
>
> Learning disabilities [occur] due to genetic and/or neurobiological factors or injury that alters brain functioning in a manner that affects one or more processes related to learning. These disorders are not due primarily to hearing and/or vision problems, socio-economic factors, cultural or linguistic differences, lack of motivation, or ineffective teaching, although these factors may further complicate the challenges faced by individuals with learning disabilities. Learning disabilities may co-exist with various conditions including attentional, behavioural, and emotional disorders, sensory impairments, or other medical conditions.
>
> For success, individuals with learning disabilities require early identification and timely specialized assessments and interventions involving home, school, community, and workplace settings. The interventions need to be appropriate for each individual's learning disability subtype and, at a minimum, include the provision of:
>
> - specific skill instruction;
> - accommodations;
> - compensatory strategies; and
> - self-advocacy skills.

Source: Learning Disabilities Association of Canada, 2002. Reprinted with the kind permission of the Learning Disabilities Association of Canada.

IMPAIRMENT IN READING Impairment in reading—dyslexia—characterizes children who have poorly developed skills in recognizing letters and words and comprehending written text. Children with dyslexia may read slowly, with difficulty, and may distort, omit, or substitute words when reading aloud. They may have trouble decoding letters. They may perceive letters upside down (*w* for *m*) or reversed (*b* for *d*). Dyslexia is usually apparent by the age of 7, coinciding with grade 2, but it is sometimes recognized in 6-year-olds. Although it was earlier believed that the problem affected mostly boys, more recent studies find similar rates among boys and girls (APA, 2000a; Shaywitz, 1998). Yet boys with dyslexia are more likely than girls to exhibit disruptive behaviour and so are more likely to be referred for evaluation. Children and adolescents with dyslexia tend to be more prone than their peers to depression, to have lower self-worth and feelings of competence in their academic work, and to have signs of ADHD (Boetsch, Green, & Pennington, 1996).

THEORETICAL PERSPECTIVES Canadian neuropsychologists contend that learning disorders originate primarily from neurobiological factors (Fiedorowicz, 1999; Fiedorowicz et al., 1999, 2002). Many children with learning disorders have problems with visual or auditory sensation and perception. They may lack the capacity to copy words or to discriminate geometric shapes. Other children have short attention spans or show hyperactivity, which is also suggestive of an underlying brain abnormality.

Much of the research on learning disorders has focused on dyslexia. Mounting evidence points to underlying brain dysfunctions (Shaywitz et al., 1998). Cross-cultural language research has now shown that dyslexia is a universal neuroanatomical disorder that causes the same reading disabilities (Paulesu et al., 2001). There is evidence of impaired visual processing in people with dyslexia that would be consistent with a defect in a major visual relay station in the brain involved in sequencing the flow of visual information from the retina to the visual cortex (Livingstone, Rosen, Drislane, & Galaburda, 1991). Inspection of the autopsied brains of people who had dyslexia showed that this relay station was smaller and less well organized than in other people. As a result, the brains of people with dyslexia are not likely to be able to decipher a rapid succession of visual stimuli, such as those involved in decoding letters and words. Words may thus become blurry, fuse together, or seem to jump off the page—all problems reported by people with dyslexia (Blakeslee, 1991).

Peter Dazeley/Photographer's Choice/Getty Images Inc.

Dyslexia. Children with dyslexia have difficulty decoding words. Note the mispelled words in this person's message.

Dysfunctions in other sensory pathways involving the sense of hearing and even the sense of touch may also be involved in learning disorders. For example, research suggests that some forms of dyslexia may be traceable to an abnormality in the brain circuits responsible for processing rapidly flowing auditory information (Blakeslee, 1994). This flaw in brain circuitry may make it difficult to understand rapidly occurring speech sounds, such as the sounds corresponding to the letters *b* and *p* in syllables like *ba* and *pa*. Problems discerning the differences between many basic speech sounds can make it difficult for people with dyslexia to learn to speak correctly and later, perhaps, to learn to read. They continue to have problems distinguishing between words like *boy* and *toy* or *pet* and *bet* in rapid speech. If defects in brain circuitry responsible for relaying and processing sensory data are involved in learning disorders, as the evidence suggests, it may lead the way to the development of specialized treatment programs to help children adjust to their sensory capabilities.

Genetic factors appear to be involved in brain abnormalities associated with dyslexia (Fiedorowicz et al., 1999, 2002). People whose parents have dyslexia are at greater risk themselves (see Figure 11.1 on page 402) (Vogler, DeFries, & Decker, 1985). Moreover, higher rates of concordance (agreement) for dyslexia are found between identical (MZ) than fraternal (DZ) twins—70% versus 40% (Plomin et al., 1994). Suspicion has focused on the role that particular genes may play in causing subtle defects in the brain circuitry involved in reading.

Intervention

With the growing recognition of the neurobiological nature of learning disabilities, our approach to treatment in Canada now involves support and intervention strategies that focus on a child's information processing style and academic strengths. The support aspect focuses on bolstering the child's self-esteem and increasing motivation, developing close teacher–parent partnerships, and, in older children, developing effective self-advocacy skills. Once an **individual education plan** (IEP) is in place, intervention can be accomplished through language re-education; a variety of mixed-ability teaching methods; academic accommodations such as alternative learning and testing methods; development of compensatory skills; and the use of assistive technologies such as computers, spell checkers, and calculators (Learning Disabilities Association of Canada, 2003a, 2003b).

individual education plan A contractual document that contains learning and behavioural outcomes for a student, a description of how the outcomes will be achieved, and a description of how the outcomes will be evaluated. Abbreviated *IEP*.

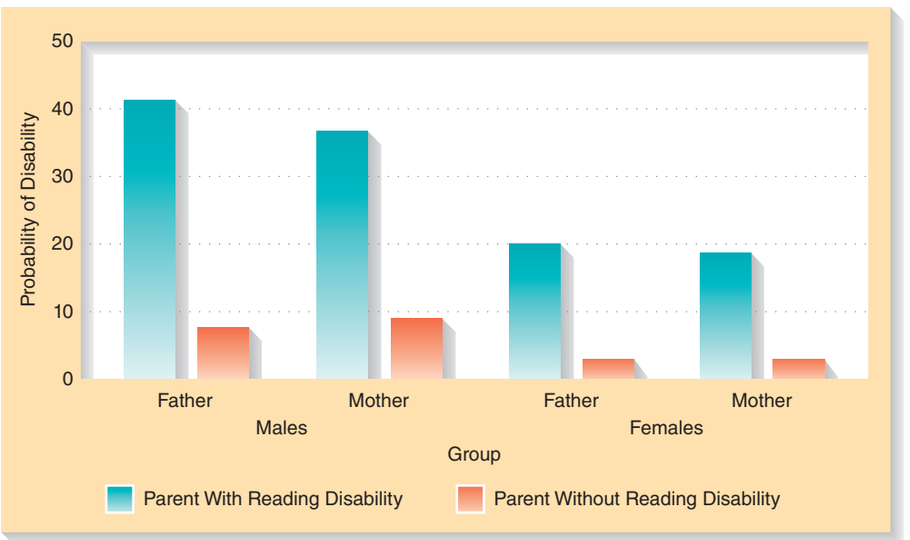


FIGURE 11.1 Familial risk of developmental reading disorder (dyslexia). Boys are at greater risk than girls of developing dyslexia, and children of both genders whose parents have dyslexia are at relatively greater risk. Although these data are consistent with a genetic explanation of the etiology of dyslexia, it is also possible that parents with dyslexia do not provide their children with the types of stimulation that foster reading skills, such as books and the reading of bedtime stories.

Source: Adapted from Vogler, DeFries, & Decker, 1985, pp. 419–421. Copyright Sage Publications. Reprinted with permission.

REVIEW IT

Learning Disorders

- **What are learning disorders?** Learning disorders (also called *learning disabilities*) are specific deficits in the development of arithmetic, writing, or reading skills.
- **What are the causes of learning disorders and approaches to treatment?** The causes remain under study but most probably involve underlying neurobiological brain dysfunctions that make it difficult to process or decode visual and auditory information. Intervention focuses mainly on the remediation and accommodation of specific skill deficits.

ATTENTION-DEFICIT AND DISRUPTIVE BEHAVIOUR DISORDERS

The category of attention-deficit and disruptive behaviour disorders encompasses a diverse range of problem behaviours, including attention-deficit/hyperactivity disorder (ADHD), conduct disorder (CD), and oppositional defiant disorder (ODD). These disorders are socially disruptive and usually more upsetting to other people than to the children who receive the diagnoses. Although there are differences among these disorders, the rate of comorbidity (co-occurrence) among them is very high (Jensen, Martin, & Cantwell, 1997).

Attention-Deficit/Hyperactivity Disorder

attention-deficit/hyperactivity disorder Behaviour disorder of childhood characterized by excessive motor activity and inability to focus one's attention. Abbreviated *ADHD*.

hyperactivity Abnormal behaviour pattern found most often in young boys that is characterized by extreme restlessness and difficulty maintaining attention.

Many parents believe their children are not attentive toward them—that they run around on whim and do things in their own way. Some inattention, especially in early childhood, is normal enough. In **attention-deficit/hyperactivity disorder** (ADHD), however, children display degrees of impulsivity, inattention, and **hyperactivity** that are considered inappropriate to their developmental levels.

ADHD is divided into three subtypes: a predominantly inattentive type, a predominantly hyperactive or impulsive type, and a combination type characterized by high levels of both inattention and hyperactivity-impulsivity (APA, 2013). The disorder is usually first diagnosed during elementary school, when problems with attention or hyperactivity-impulsivity make it difficult for the child to adjust to school. Although signs of hyperactivity are often observed earlier, many overactive toddlers do not go on to develop ADHD.

ADHD is far from rare. Canadian studies have found the prevalence rates of ADHD to be between 5% and 10% in children aged 6 to 14 (Romano, Baillargeon, & Tremblay, 2002). Boys were two to three times more likely than girls to be identified as having ADHD, and 6- to 8-year-olds had higher rates than 12- to 14-year-olds. Although inattention appears to be the basic problem, there are associated problems such as an inability to sit still for more than a few moments, bullying, temper tantrums, stubbornness, and failure to respond to punishment (see Table 11.3).

TABLE 11.3

Diagnostic Criteria for Attention-Deficit/Hyperactivity Disorder (ADHD)

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 a 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 or 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 and 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 uncomfortable being still for extended time, as in restaurants, meetings; may be experienced by others as being restless or difficult to keep up with).
   f. Often talks excessively.
   g. Often blurts out an answer before a question has been completed (e.g., completes people's sentences; cannot wait for 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 prior to age 12 years.

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

D. There is 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).

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NORMAL/ABNORMAL BEHAVIOUR

Attention Issues: No Disorder

Kai is a seven-year-old boy in grade 2. His parents describe him as an active and energetic boy who sometimes seems like he has "ants in his pants." He is especially excitable in new situations that are full of stimulation. For example, his teacher has had to talk to him a few times because of his excitability during gym class. He sometimes gets too rough when playing games with his classmates, though his intentions are never aggressive. When he's excited about something, Kai has trouble focusing on anything else. His mom says that it's virtually impossible to get his attention when he's waiting for a friend to come over, or just before a birthday party. Sometimes she worries about leaving him at a friend's house in case he gets out of hand. Luckily, this hasn't happened and Kai continues to be invited on playdates and to parties. Kai's teacher has remarked that he has the ability to focus on lots of things at school, especially activities where he has something to do with his hands. Kai's parents also note that he is able to sit and listen to stories or watch kids' movies.

Attention Issues: ADHD

Behzad is a nine-year-old boy who lives with his parents and two younger siblings. He is in grade 4 at school and is struggling. He has been identified by his teacher as having attention and behaviour problems at school. On a regular basis, Behzad is removed from the regular classroom due to being disruptive. He constantly interrupts the teacher and other children, he has trouble staying in his seat, and he cannot finish classroom assignments. Behzad's parents have had similar concerns at home. Behzad is very physical with his siblings and sometimes accidentally hurts them. He is especially aggressive when he's frustrated, and it seems that he is increasingly frustrated even when doing things he likes. He has trouble sitting through television shows and is not able to sit and read books. He can sometimes focus on a video game for a sustained period of time, but this seems to be one of the only activities that can consume his attention. Behzad's parents are very worried because his disruptive behaviour and trouble focusing appears to be having a negative impact on his achievement at school. They are also worried that the teachers are beginning to think of him as a "problem" and are removing him from the classroom rather than looking at ways of helping him focus.

Activity and restlessness impair the ability of children with ADHD to function in school. They seem incapable of sitting still. They fidget and squirm in their seats, butt into other children's games, have outbursts of temper, and may engage in dangerous behaviour, such as running into the street without looking. All in all, they can drive parents and teachers to despair.

Where does "normal" age-appropriate overactivity end and hyperactivity begin? Assessment of the degree of hyperactive behaviour is crucial, because many normal children are called "hyper" from time to time. Some critics of the ADHD diagnosis argue that it merely labels children who are difficult to control as mentally disordered or sick. Most children, especially boys, are highly active during the early school years. Proponents of the diagnosis counter that there is a difference in quality between normal overactivity and ADHD. Normally overactive children are goal directed and can exert voluntary control over their own behaviour. But children with ADHD appear hyperactive without reason and do not seem to be able to conform their behaviour to the demands of teachers and parents. Put it another way: most children can sit still and concentrate for a while when they want to; children with ADHD seemingly cannot.

Children with ADHD tend to do more poorly in school than their peers despite being, for the most part, of average or above average intelligence. They may fail to follow or remember instructions and complete assignments. They are more likely than their peers to have learning disabilities, to repeat grades, and to be placed in special-education classes (Faraone et al., 1993; Leibson, Katusic, Barbaresi, Ransom, & O'Brien, 2001). They also stand a greater risk of having mood disorders, anxiety disorders, and problems getting along with family members (Biederman et al., 1996). They are frequently disruptive in the classroom and tend to get into fights (especially the boys). Not surprisingly, they are frequently unpopular with their classmates. Although ADHD symptoms tend to decline with age, the disorder can persist into adolescence and adulthood (Asherson, Chen, Craddock, & Taylor, 2007; M. Weiss & Murray, 2003).

THEORETICAL PERSPECTIVES Although the causes of ADHD are not known, both biological and environmental influences are believed to be involved (Arnold, O'Leary, & Edward, 1997). Increasing evidence points to a complex genetic vulnerability to ADHD wherein multiple genes have a singularly small, albeit additive, impact (Faraone & Khan, 2006). Hereditary evidence comes from findings of higher concordance rates for ADHD among monozygotic (MZ) twins than dizygotic (DZ) twins, supporting a genetic linkage (Sherman, McGue, & Ianoco, 1997).

Neuropsychological testing, EEG studies, and MRI studies of children and adolescents with ADHD point to abnormalities in the areas of the brain involved in regulating the processes of attention, inhibition of motor (movement) behaviour, and executive control (i.e., the ability to focus, plan, and act) (Bush, Valera, & Seidman, 2005; Castellanos, Glaser, & Gerhardt, 2006; Seidman, Valera, & Makris, 2005; Serene, Ashtari, Szeszko, & Kumra, 2007). We shall also see that the effects of stimulants on children with ADHD offer some support to the hypothesis of organic causes. Despite evidence suggestive of biological factors, we lack a definitive biological explanation of ADHD. In fact, some theorists suggest that the mounting evidence from neuropsychological studies challenges the long-held belief that ADHD is a single coherent clinical entity (Stefanatos & Baron, 2007).

ADHD has also been linked with exposure to environmental toxins. Children who have ADHD were found to be 2.5 times more likely than other children to have had prenatal exposure to environmental tobacco smoke (ETS) (Braun, Khan, Froehlich, Auinger, & Lanphear, 2006). Moreover, a significant dose–response relationship was found: the greater the exposure to ETS, the higher the risk of ADHD, especially for girls. These researchers also found a significant dose–response relationship between higher levels of lead in the blood and ADHD.

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Attention-deficit/hyperactivity disorder (ADHD). ADHD is more common in boys than girls and is characterized by attentional difficulties, restlessness, impulsivity, excessive motor behaviour (continuous running around or climbing), and temper tantrums.

TREATMENT It seems odd that the drugs used to help ADHD children calm down and attend better in school belong to a class of stimulants that include Ritalin (methylphenidate) and longer-acting variants, such as Ritalin SR and Concerta. These stimulants have a paradoxical effect of calming down children with ADHD and increasing their attention spans. Although the use of stimulant medication is not without criticism, it is clear that these drugs can help many children with ADHD calm down and concentrate better on tasks and schoolwork, perhaps for the first time in their lives (L. S. Goldman, Genel, Bezman, & Slanetz, 1998). These drugs not only improve attention in ADHD children but also reduce impulsivity, overactivity, and disruptive, annoying, or aggressive behaviour (Gillberg et al., 1997; Hinshaw, 1992). Stimulant medication appears to be safe and effective when carefully monitored, and successful in helping about three out of four children with ADHD ("Attention Deficit Disorder," 1995; Hinshaw, 1992; Spencer et al., 1996). Improvements are noted at home as well as in school. The normal (voluntary) high activity levels shown in physical education classes and on weekends are not disrupted, however.

We do not know what accounts for the seemingly paradoxical effects of stimulants in calming children with ADHD, although it is suspected that these drugs work on neurotransmitter systems in the brain ("Attention Deficit Disorder," 1995). Although the precise mechanisms are not well understood, we know that these drugs heighten dopamine and norepinephrine activity in the prefrontal cortex of the brain, the area that regulates attention and control of impulsive behaviour (Faraone, 2003a, 2003b). Thus, the drugs may help ADHD children focus their attention and avoid acting out impulsively. Stimulant medication has become so popular that its use increased more than sevenfold during the 1990s (Gibbs, 1998). Currently, an estimated 82 in 1000 Canadian children are using these types of drugs as treatment for ADHD (Romano, Baillargeon, Fortier, et al., 2005). The

rate of usage climbs from 0.58% in 4- to 5-year-old boys to a peak of 6.31% in 10- to 11-year-old boys. By comparison, girls are much less likely to use these drugs; their highest usage is at age 8 to 9 (1.09%).

Although stimulant medication can help reduce restlessness and increase attention, it is hardly a panacea. Canadian pediatric researchers caution that although Ritalin has a significant short-term effect on the symptoms of ADHD, there is a lack of evidence that demonstrates its usefulness beyond four weeks of treatment. Research is needed to determine its long-term effectiveness (Schachter, Pham, King, Langford, & Moher, 2001). Moreover, no solid evidence has shown that stimulant medication improves academic performance or leads to better outcomes in adulthood.

Then there is the question of side effects. Although short-term side effects (e.g., loss of appetite or insomnia) usually subside within a few weeks of treatment or may be eliminated by lowering the dose, concerns have been raised about whether stimulants might retard a child's growth. Although these drugs do slow growth for a few years, researchers found that the drug delays but does not stunt a youngster's growth (Gorman, 1998). Recently, Health Canada (2006b) posted a drug advisory warning Canadians with high blood pressure, heart disease, and cardiovascular conditions or an overactive thyroid to avoid any stimulant drugs to manage symptoms of ADHD. In rare cases, these drugs can result in cardiac arrest, stroke, or sudden death. A subsequent Health Canada (2006c) information update also indicated the potential for adverse psychiatric effects, such as agitation and hallucinations in children who use ADHD drugs.

With so many children on Ritalin and similar drugs, critics claim we are too ready to seek a "quick fix" for problem behaviour in children rather than examining other factors contributing to the child's problem, such as dysfunctions in the family (Gibbs, 1998). As one pediatrician put it, "It takes time for parents and teachers to sit down and talk to kids. . . . It takes less time to get a child a pill" (Hancock, 1996, p. 52). Whatever the benefits of stimulant medication, medication alone typically fails to bring the social and academic behaviour of children with ADHD into a normal range (Hinshaw, 1992). Drugs cannot teach new skills. Thus, attention has focused on whether a combination of stimulant medication and behavioural or cognitive-behavioural techniques can produce greater benefits than either approach alone. Cognitive-behavioural treatment of ADHD combines behaviour modification, typically based on the use of reinforcement (e.g., a teacher praising the child with ADHD for sitting quietly) and cognitive modification (e.g., training the child to silently talk himself or herself through the steps involved in solving challenging academic problems). Thus far, the evidence favours a combination approach. A McMaster University–based review of 14 studies involving nearly 1400 participants found that a combination of medication and behavioural interventions yielded better outcomes than either type of treatment alone (Schachar et al., 2002).

Conduct Disorder

conduct disorder Pattern of abnormal behaviour in childhood characterized by disruptive, antisocial behaviour.

Although they both involve disruptive behaviour, **conduct disorder** differs in important ways from ADHD. Whereas children with ADHD seem literally incapable of controlling their behaviour, children with conduct disorders purposefully engage in patterns of antisocial behaviour that violate social norms and the rights of others. Whereas children with ADHD throw temper tantrums, children diagnosed as conduct disordered are intentionally aggressive and cruel. Like antisocial adults, many conduct-disordered children are callous and apparently do not experience guilt or remorse for their misdeeds.

The prevalence of conduct disorder in Canadian children and youth is estimated to be 3.3% (Waddell & Shepherd, 2002). Conduct disorders are much more common among boys than girls, especially the childhood-onset type in which characteristic features of the disorder appear before age 10 (APA, 2013). Conduct disorder typically takes a somewhat different form in boys than girls. In boys, it is more likely to be manifested in stealing, fighting, vandalism, or disciplinary problems at school, whereas in girls the disorder is more likely to involve lying, truancy, running away, substance use, and prostitution

(APA, 2013). Although there are differences between ADHD and conduct disorder, some children with conduct disorder also display a pattern of short attention span and hyperactivity that may justify a double diagnosis.

Conduct disorder is typically a chronic or persistent disorder (Lahey et al., 1995). Longitudinal studies show that Canadian elementary schoolchildren with conduct disorders are more likely than other children to engage in delinquent acts as early adolescents (Tremblay et al., 1992). Another form of conduct disorder may involve a cluster of personality traits that have different origins than antisocial behaviour (Wootton, Frick, Shelton, & Silverthorn, 1997). These traits include callousness (uncaring, mean, and cruel behaviour) and an unemotional way of relating to others.

Oppositional Defiant Disorder

oppositional defiant disorder Disorder in childhood or adolescence characterized by excessive oppositionality or tendencies to refuse requests from parents and others.

Debate continues among professionals over the issue of whether conduct disorder (CD) and **oppositional defiant disorder** (ODD) are separate disorders or variations of a common disruptive behaviour disorder (Rey, 1993). Or perhaps ODD is a precursor or milder form of conduct disorder (Abikoff & Klein, 1992; Biederman et al., 1996). Currently, the two disorders are conceptualized as related but separate disorders. ODD is more closely related to nondelinquent (negativistic) conduct disturbance, and conduct disorder involves more outright delinquent behaviour in the form of truancy, stealing, lying, and aggressiveness (Rey, 1993). University of British Columbia researchers have found a strong association between oppositional defiant disorder symptoms and generalized anxiety symptoms in preadolescent children (Garland & Garland, 2001). However, they noted reluctance on the part of clinicians to give a diagnosis of ODD to children with anxiety disorders. This reluctance can have an impact on treatment and outcomes.

Children with ODD tend to be negativistic or oppositional. They are defiant of authority, which is exhibited by their tendency to argue with parents and teachers and refuse to follow requests or directives from adults. They may deliberately annoy other people, become easily angered or lose their temper, become touchy or easily annoyed, blame others for their mistakes or misbehaviour, feel resentful toward others, or act in spiteful or vindictive ways toward others (Angold & Costello, 1996; APA, 2013). The disorder typically begins before age eight and develops gradually over a period of months or years. It typically starts in the home environment but may extend to other settings, such as school.

ODD is one of the most common diagnoses among children (Doll, 1996). Studies show that among children diagnosed with a psychological disorder, about one in three are judged to meet the criteria for ODD (Rey, 1993). An estimated 6% to 12% of school-aged children display ODD (Frick & Silverthorn, 2001). ODD is more

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Oppositional defiant disorder (ODD). A common childhood disorder that engenders a "no-win" situation for everyone concerned.

common overall among boys than girls. However, this overall effect masks a gender shift over age. Among children 12 years of age or younger, ODD appears to be more than twice as common among boys; yet among adolescents, a higher prevalence is reported in girls (Rey, 1993). By contrast, most studies find conduct disorder to be more common in boys than girls across all age groups.

THEORETICAL PERSPECTIVES The causal factors in ODD remain obscure. Some theorists believe that oppositionality is an expression of an underlying child temperament described as the "difficult-child" type (Rey, 1993). Learning theorists view oppositional behaviours as arising from parental use of inappropriate reinforcement strategies. In this view, parents may inappropriately reinforce oppositional behaviour by "giving in" to the child's demands whenever the child refuses to comply with the parents' wishes, which can become a pattern.

McMaster University researchers have found that family and parenting factors are implicated in the development of disruptive behaviour disorders such as oppositional defiant disorder and conduct disorder (Cunningham & Boyle, 2002). Some forms of disruptive behaviour disorders appear to be linked to unassertive and ineffective parenting styles, such as failure to provide positive reinforcement for appropriate behaviour and use of harsh and inconsistent discipline for misbehaviour. Families of children with CD tend to be characterized by negative, coercive interactions (Dadds, Sanders, Morrison, & Rebgetz, 1992). Children with CD are often very demanding and noncompliant in relating to their parents and other family members. Family members often reciprocate by using negative behaviours, such as threatening or yelling at the child or using physical means of coercion. Parental aggression against children with conduct behaviour problems is common, including pushing, grabbing, slapping, spanking, hitting, or kicking (Jouriles, Mehta, McDonald, & Francis, 1997). Parents of children with oppositional defiant disorders or severe conduct disorder display high rates of antisocial personality disorder and substance abuse (Frick et al., 1992). It's not too much of a stretch to speculate that parental modelling of antisocial behaviours can lead to antisocial conduct in their children.

Some investigations focus on the ways in which children with disruptive behaviour disorders process information. For example, children who are overly aggressive in their behaviour tend to be biased in their processing of social information: they may assume that others intend them ill when they do not (Lochman & Dodge, 1994). They usually blame others for the scrapes they get into. They believe they are misperceived and treated unfairly. They may believe that aggression will lead to favourable results (Dodge, Lochman, Hamish, Bates, & Pettit, 1997). They are also less able than their peers to generate alternative (nonviolent) responses to social conflicts (Lochman & Dodge, 1994).

Genetic factors may interact with family or other environmental factors in the development of conduct disorder in children and antisocial behaviour in adolescence (Slutske et al., 1997, 1998). Genetic factors may also be involved in the development of oppositional defiant disorder.

TREATMENT The treatment of conduct disorders remains a challenge. Although there is not an established pharmacological treatment approach, Toronto psychiatrist Lindley Bassarath (2003) reviewed recent studies that indicate that certain antipsychotic and stimulant drugs may be effective in reducing antisocial behaviour in CD children and adolescents. Psychotherapy has not generally been shown to help disruptive children change their behaviour. Placing children with conduct disorders in programs or treatment settings with explicit rules and clear rewards for obeying them may offer greater promise (Henggeler et al., 1986). Such programs usually rely on operant conditioning procedures that involve systematic use of rewards and punishments.

Many children with conduct disorders, especially boys, display aggressive behaviour and have problems controlling their anger. Many can benefit from programs designed to help them learn anger-coping skills that they can use to handle conflict situations without resorting to violent behaviour. Cognitive-behavioural therapy has been used to teach boys who engage in antisocial and aggressive behaviour to reconceptualize social provocations

as problems to be solved rather than as challenges to their manhood that must be answered with violence. They have been trained to use calming self-talk to inhibit impulsive behaviour and control anger whenever they experience social taunts or provocations and to generate and try out nonviolent solutions to social conflicts (Lochman & Lenhart, 1993). Other programs present child models on video demonstrating skills of anger control. The results of these programs appear promising (Kazdin & Weisz, 1998; Webster-Stratton & Hammond, 1997). Sometimes the disruptive child's parents are brought into the treatment process (Kazdin & Whitley, 2003).

The following example illustrates the involvement of the parents in the behavioural treatment of a case of oppositional defiant disorder:

Dimitry was a seven-year-old second-grader referred by his parents. The family relocated frequently because the father was in the navy. Dimitry usually behaved when his father was taking care of him, but he was noncompliant with his mother and yelled at her when she gave him instructions. His mother was incurring great stress in the effort to control Dimitry, especially when her husband was at sea.

Dimitry had become a problem at home and in school during grade 1. He ignored and violated rules in both settings. Dimitry failed to carry out his chores and frequently yelled at and hit his younger brother. When he acted up, his parents would restrict him to his room or the yard, take away privileges and toys, and spank him. But all of these measures were used inconsistently. He also played on the railroad tracks near his home and twice the police had brought him home after he had thrown rocks at cars.

A home observation showed that Dimitry's mother often gave him inappropriate commands. She interacted with him as little as possible and showed no verbal praise, physical closeness, smiles, or positive facial expressions or gestures. She paid attention to him only when he misbehaved. When Dimitry was noncompliant, she would yell back at him and then try to catch him to force him to comply. Dimitry would then laugh and run from her.

Dimitry's parents were informed that the child's behaviour was a product of inappropriate cueing techniques (poor directions), a lack of reinforcement for appropriate behaviour, and lack of consistent sanctions for misbehaviour. They were taught the appropriate use of reinforcement, punishment, and **time out**. The parents then charted Dimitry's problem behaviours to gain a clearer idea of what triggered and maintained them. They were shown how to reinforce acceptable behaviour and use time out as a contingent punishment for misbehaviour. Dimitry's mother was also taught relaxation training to help desensitize her to Dimitry's disruptions. Biofeedback was used to enhance the relaxation response.

During a 15-day baseline period, Dimitry behaved in a noncompliant manner about four times per day. When treatment was begun, Dimitry showed an immediate drop to about one instance of noncompliance every two days. Follow-up data showed that instances of noncompliance were maintained at a bearable level of about one per day. Fewer behavioural problems in school were also reported, even though they had not been addressed directly.

S. J. Kaplan, 1986, pp. 227–230. Reprinted with the kind permission of Springer Science+Business Media B.V.

time out Behavioural technique in which an individual who emits an undesired behaviour is removed from an environment in which reinforcers are available and placed in an unreinforcing environment for a period of time as a form of punishment. Time out is frequently used in behavioural programs for modifying behaviour problems in children, in combination with positive reinforcement for desirable behaviour.

In Canada, as in most other industrialized nations, the emphasis is more on treatment than prevention. This usually means that by the time a conduct-disordered youth gets into care, his or her problem behaviour is well established and therefore more resistant and more expensive to treat. A failure to implement effective prevention programs may have more to do with short-sighted political agendas and rigid service-delivery systems than it does with program costs or a lack of desire to serve long-term needs to prevent future conduct-disordered youth (Moretti et al., 1997).

REVIEW IT

Attention-Deficit and Disruptive Behaviour Disorders

- **What are attention-deficit and disruptive behaviour disorders?** This category includes attention-deficit/hyperactivity disorder (ADHD), conduct disorder, and oppositional defiant disorder. ADHD is characterized by impulsivity, inattention, and hyperactivity. Children with conduct disorders intentionally engage in antisocial behaviour. Children with ODD show negativistic or oppositional behaviour but not outright delinquent or antisocial behaviour characteristic of conduct disorder. However, ODD may represent an early stage of development of conduct disorder.
- **How are these disorders treated?** Stimulant medication is generally effective in reducing hyperactivity, but has not led to general academic gains. Behaviour therapy may help ADHD children adapt better to school. Behaviour therapy may also be helpful in modifying behaviours of children with conduct disorders and oppositional defiant disorder.

ANXIETY AND DEPRESSION IN CHILDHOOD AND ADOLESCENCE

Anxieties and fears are a normal feature of childhood, just as they are a normal feature of adult life. Childhood fears—of the dark or of small animals—are commonplace and are usually outgrown naturally. Anxiety is considered abnormal, however, when it is excessive and interferes with normal academic or social functioning or becomes troubling or persistent. Children, like adults, may suffer from different types of diagnosable anxiety disorders, including specific phobias, social phobias, and generalized anxiety disorder (GAD). Although these disorders may develop at any age, we will consider a type of anxiety disorder that typically develops during early childhood: separation anxiety disorder.

Children may also show a more general pattern of avoidance of social interactions that characterizes avoidant personality disorder. Although children who are socially avoidant or have social phobias may have warm relationships with family members, they tend to be shy and withdrawn around others. Their avoidance of people outside the family interferes with their development of social relationships with their peers. Their distress at being around other children at school can also impede their academic progress. Such problems tend to develop after normal fear of strangers fades, at age two and a half or later.

Despite the stereotype of a happy childhood, clinical depression is found in children and adolescents. Estimates indicate that slightly more than 2% of Canadian children experience a depressive disorder (Waddell & Shepherd, 2002). Major depression has even been found, although rarely, among preschoolers. Although there is no discernible gender difference in the risk of depression in childhood, a prominent gender difference appears after the age of 15, with adolescent girls becoming about twice as likely to become depressed as adolescent boys (Hankin et al., 1998). Nationwide surveys have revealed that, of Canadian youth aged 15 to 19 years, 12% of the females and 3% to 6% of the males have had a major depressive episode (Shaw & Grenier, 2001).

Separation Anxiety Disorder

separation anxiety disorder
Childhood disorder characterized by extreme fears of separation from parents or others on whom the child is dependent.

It is normal for children to show anxiety when they are separated from their caregivers (Ainsworth & Bowlby, 1991). Mary Ainsworth (1989), who has chronicled the development of attachment behaviours, notes that separation anxiety is a normal feature of the child–caregiver relationship and begins during the first year of life. The sense of security normally provided by bonds of attachment apparently encourages children to explore their environments and become progressively independent of their caregivers (Bowlby, 1988).

Separation anxiety disorder is diagnosed when separation anxiety is persistent and excessive or inappropriate for the child's developmental level. That is, three-year-olds

ought to be able to attend preschool without nausea and vomiting brought on by anxiety. Six-year-olds ought to be able to attend grade 1 without persistent dread that something awful will happen to themselves or their parents. Children with this disorder tend to cling to their parents and follow them around the house. They may voice concerns about death and dying and insist that someone stay with them while they are falling asleep. Other features of the disorder include nightmares, stomach aches, nausea and vomiting when separation is anticipated (as on school days), pleading with parents not to leave, or throwing tantrums when parents are about to depart. Children may refuse to attend school for fear that something will happen to their parents while they are away. The disorder affects about 4% of children and young adolescents and occurs more frequently, according to community-based studies, among females (APA, 2013). The disorder may persist into adulthood, leading to an exaggerated concern about the well-being of one's children and spouse and difficulty tolerating any separation from them.

The development of separation anxiety disorder frequently follows a stressful life event, such as illness, the death of a relative or pet, or a change of schools or homes. Richa's problems followed the death of her grandmother:

Richa's grandmother died when Richa was seven years old. Her parents decided to permit her request to view her grandmother in the open coffin. Richa took a tentative glance from her father's arms across the room, then asked to be taken out of the room. Her five-year-old sister took a leisurely close-up look, with no apparent distress.

Richa had been concerned about death for two or three years by this time, but her grandmother's passing brought on a new flurry of questions: "Will I die?" "Does everybody die?" and so on. Her parents tried to reassure her by saying, "Grandma was very, very old, and she also had a heart condition. You are very young and in perfect health. You have many, many years before you have to start thinking about death."

Richa also could not be alone in any room in her house. She pulled one of her parents or her sister along with her everywhere she went. She also reported nightmares about her grandmother and, within a couple of days, insisted on sleeping in the same room with her parents. Fortunately, Richa's fears did not extend to school. Her teacher reported that Richa spent some time talking about her grandmother, but her academic performance was apparently unimpaired.

Richa's parents decided to allow Richa time to "get over" the loss. Richa gradually talked less and less about death, and by the time three months had passed, she was able to go into any room in her house by herself. She wanted to continue to sleep in her parents' bedroom, however, so her parents "made a deal" with her. They would put off the return to her own bedroom until the school year had ended (a month away), if Richa would agree to return to her own bed at that time. As a further incentive, a parent would remain with her until she fell asleep for the first month. Richa overcame the anxiety problem in this fashion with no additional delays.

The Authors' Files

Perspectives on Anxiety Disorders in Childhood

Theoretical understandings of excessive anxiety in children to some degree parallel explanations of anxiety disorders in adults. Psychoanalytic theorists argue that childhood anxieties and fears, like their adult counterparts, symbolize unconscious conflicts. Cognitive theorists focus on the role of cognitive biases underlying anxiety reactions. In

support of the cognitive model, investigators find that highly anxious children show cognitive biases in processing information, such as interpreting ambiguous situations as threatening, expecting negative outcomes, thinking poorly of themselves and of their ability to cope, and engaging in negative self-talk (Weems, Costa, Watts, Taylor, & Cannon, 2007). Expecting the worst, combined with low self-confidence, encourages avoidance of feared activities—with friends, in school, and elsewhere. Negative expectations may also heighten feelings of anxiety to the point where they impede performance. Learning theorists suggest that the occurrence of generalized anxiety may touch on broad themes, such as fears of rejection or failure, that carry across situations. Underlying fears of rejection or self-perceptions of inadequacy may generalize to most areas of social interaction and achievement.

Depression in Childhood and Adolescence

The basic features of depression in children and adolescents are similar to those in adults (Kovacs, 1996). Depressed children and adolescents typically show a greater sense of hopelessness, display more cognitive errors and negative attributions (e.g., blaming themselves for negative events), have lower perceived competence or self-efficacy, and have lower self-esteem than do their nondepressed peers (Lewinsohn, Rohde, & Seeley, 1994). They often report episodes of sadness, crying, and apathy as well as insomnia, fatigue, and poor appetite. They may refuse to attend school, express fears of their parents' dying, and cling to their parents or retreat to their rooms. They may have suicidal thoughts or attempt suicide.

Moderate levels of depression may persist for years, severely impacting school performance and social functioning (Nolen-Hoeksema & Girgus, 1994). Adolescent depression is associated with an increased risk of future major depressive episodes and suicide attempts in adulthood (M. M. Weissman, Wolk, et al., 1999). About three out of four children who become depressed from age 8 to 13 have a recurrence later in life (Goleman, 1994).

cglade/iStockphoto

Is this child too young to be depressed? Although we tend to think of childhood as the happiest and most carefree time of life, depression is actually quite common among children and adolescents. Depressed children may report feelings of sadness and lack of interest in previously enjoyable activities. Many, however, do not report, or are not aware of, feelings of depression, even though they may look depressed to observers. Depression may also be masked by other problems such as conduct/school-related problems, physical complaints, and overactivity.

Children who experience depression may also lack skills in various domains, including academic performance, social acceptance by peers, and athletic performance (Seroczynski, Cole, & Maxwell, 1997). They may find it hard to concentrate in school and may suffer from impaired memory, making it difficult for them to keep their grades up (Goleman, 1994).

Childhood depression rarely occurs by itself (Hammen & Compas, 1994). Depressed children typically experience other psychological disorders, especially anxiety disorders and conduct or oppositional defiant disorders (Hammen & Compas, 1994). Eating disorders are also common among depressed adolescents, at least among females (Rohde, Lewinsohn, & Seeley, 1991). Overall, childhood depression increases the chances that a child will develop another psychological disorder by at least twentyfold (Angold & Costello, 1993).

We should recognize that depressed children or adolescents may fail to label what they are feeling as depression. Further, conduct disorders, academic problems, physical complaints, and even hyperactivity may stem now and then from unrecognized depression. Among adolescents, aggressive and sexual acting out may also be signs of underlying depression.

CORRELATES AND TREATMENT OF DEPRESSION IN CHILDHOOD AND ADOLESCENCE As children mature and their cognitive abilities increase, however, cognitive factors, such as attributional styles, appear to play a stronger role in the development of depression. Older children (in grades 6 and 7) who adopt a more helpless or pessimistic explanatory style (attributing negative events to internal, stable, and global causes and attributing positive events to external, unstable, and specific causes) are more likely than children with a more optimistic explanatory style to develop depression (Nolen-Hoeksema, Girgus, & Seligman, 1992). Researchers also find that adolescents who are depressed tend to hold more dysfunctional attitudes and to adopt a more helpless explanatory style than their nondepressed peers (Lewinsohn et al., 1994).

Genetic factors also appear to play a role in explaining depressive symptoms, at least among adolescents (O'Connor, McGuire, Reiss, Hetherington, & Plomin, 1998). The role of genetics in childhood depression requires further study, however (Kovacs, Devlin, Pollock, Richards, & Mukerji, 1997).

Adolescent girls may face a greater risk of depression because they tend to face more social challenges than boys during adolescence—challenges such as pressures to narrow their interests and pursue feminine-typed activities (Nolen-Hoeksema & Girgus, 1994). It may be that girls who adopt a more passive, ruminative style of coping as children may be at greatest risk of becoming depressed when they face socially restrictive attitudes that devalue their accomplishments and abilities in relation to those of boys, when they face restrictions placed on the social roles and activities deemed appropriate for their gender, and when they encounter sexual pressures or abuse.

Accumulating evidence supports the effectiveness of cognitive-behavioural therapy (CBT) in treating depression in childhood and adolescence (Braswell & Kendall, 2001; Lewinsohn & Clarke, 1999). Although individual approaches vary, CBT usually involves a coping-skills model in which children or adolescents receive social-skills training (e.g., learning how to start a conversation or make friends) to increase the likelihood of obtaining social reinforcement (Kazdin & Weisz, 1998). In addition, family therapy may be useful in helping families resolve underlying conflicts and reorganize their relationships in ways that members can become more supportive of each other.

The earlier generation of antidepressants failed to show superior results to placebos in treating childhood or adolescent depression (Rutter, 1997). We cannot assume drugs that may be effective with adults will work as well or be as safe when used with children (Vitello & Jensen, 1997). However, the drug Prozac, one of the new generation of SSRI antidepressants, has been shown in at least one study to produce better results than a placebo control in relieving severe and persistent depression in children and adolescents (Emslie et al., 1997). Still, the complete elimination of depressive symptoms was rare.

SUICIDE AMONG CHILDREN AND ADOLESCENTS Suicide is relatively uncommon among children under the age of 10 in Canada. However, for youth aged 10 to 19 years, Health Canada (1999a) reports that after traffic fatalities, suicide is the second leading cause of death, accounting for almost one in five deaths. The rates of suicide climb with age, rising from 2.0 in 100 000 between the ages of 10 and 14 years to 11.5 between the ages of 15 and 19. Males are about three and a half times as likely as females to complete a suicide. These official statistics account only for reported suicide; some apparently accidental deaths may be suicides as well.

Despite the commonly held view that children and adolescents who talk about suicide are only venting their feelings, young people who do intend to kill themselves may very well talk about it beforehand (Brody, 1992a). In fact, those who discuss their plans are the ones most likely to carry them out. Moreover, children and adolescents who have survived suicide attempts are most likely to try again (Brody, 1992a). Unfortunately, parents tend not to take their children's suicidal talk seriously. They often refuse treatment for their children or terminate treatment prematurely.

Several factors are associated with an increased risk of suicide among children and adolescents (Levy, Jurkovic, & Spirito, 1995; Lewinsohn et al., 1994; Neiger & Hopkins, 1988):

1. *Gender.* Girls, like women, are three times more likely than boys to attempt suicide. Boys, like men, are more likely to succeed, however, perhaps because boys, like

men, are more apt to use lethal means, such as guns. The presence of a loaded handgun in the house turns out to be the greatest risk factor for completed suicide among children, even those as young as five (Brody, 1992a).

2. *Age*. Young people in late adolescence or early adulthood (ages 15 to 24) are at greater risk than younger adolescents.
3. *Ethnicity*. The suicide rate for Canadian Aboriginal youth is three to four times higher than for youth in the general population (Health Canada, 1999a). The rates of suicide in some First Nations communities are among the highest in the world ("Federal Paternalism," 1999).
4. *Depression and hopelessness*. Depression and hopelessness, especially when combined with low self-esteem, are major risk factors for suicide among adolescents, as for adults.
5. *Previous suicidal behaviour*. A quarter of adolescents who attempt suicide are repeaters. More than 80% of adolescents who take their lives have talked about it before doing so. Suicidal teenagers may carry lethal weapons, talk about death, make suicide plans, or engage in risky or dangerous behaviour. A family history of suicide also increases risk of teenage suicide (Mann, Underwood, & Arango, 1996).
6. *Family problems*. Family problems are present among about 75% of adolescent suicide attempters. Problems include family instability and conflict, physical or sexual abuse, loss of a parent due to death or separation, and poor parent–child communication (Asarnow, Carlson, & Guthrie, 1987; Wagner, 1997).
7. *Stressful life events*. Many suicides among young people are directly preceded by stressful or traumatic events, such as the breakup of a relationship with a girlfriend or boyfriend, unwanted pregnancy, getting arrested, having problems at school, moving to a new school, or having to take an important test.
8. *Substance abuse*. Addiction in the adolescent's family or by the adolescent is a factor.
9. *Social contagion*. Adolescent suicides sometimes occur in clusters, especially when a suicide or a group of suicides receives widespread publicity (R. C. Kessler, Downey, Milavsky, & Stipp, 1988; Phillips & Carstensen, 1986). Adolescents may romanticize suicide as a heroic act of defiance. There are often suicides or attempts among the siblings, friends, parents, or adult relatives of suicidal adolescents. Note the case of Alexandra:

Alexandra was an exceptionally attractive 17-year-old who was hospitalized after cutting her wrists.

"Before we moved to [an upper-middle-class U.S. town in Westchester County]," she told the psychologist, "I was the brightest girl in the class. Teachers loved me. If we had had a yearbook, I'd have been the most likely to succeed. Then we moved, and suddenly I was hit with it. Everybody was bright, or tried to be. Suddenly I was just another ordinary student planning to go to college.

"Teachers were good to me, but I was no longer special, and that hurt. Then we all applied to college. Do you know that 90% of the kids in the high school go on to college? I mean four-year colleges? And we all knew—or suspected—that the good schools had quotas on kids from here. I mean you can't have 30 kids from our senior class going to Yale or Princeton or Wellesley, can you? You're better off applying from Utah.

"Then Carmen got her early-acceptance rejection from Brown. Carmen was number one in the class. Nobody could believe it. Her father had gone to Brown and Carmen had scored almost 1500 on her SATs. Carmen was out of commission for a few days—I mean she didn't come to school or anything—and then, boom, she was gone. She offed herself, kaput, no more, the end. Then Brian was rejected from Cornell. A few days later, he was gone, too. And I'm like, 'These kids were better than me.' I mean their grades and their SATs were higher than mine, and I was going to apply to Brown and Cornell. I'm like, 'What chance do I have? Why bother?'"

The Authors' Files

You can identify how catastrophizing cognitions can play a role in such tragic cases. Consistent with the literature on suicide among adults, suicidal children and adolescents make less use of active problem-solving strategies in handling stressful situations. They may see no other way out of their perceived failures or stresses. As with adults, one approach to working with suicidal children and adolescents involves helping them challenge distorted thinking and generate alternative strategies for handling the problems and stressors they face.

REVIEW IT

Anxiety and Depression

- **What types of anxiety disorders affect children?** Anxiety disorders that occur commonly among children and adolescents include specific phobias, social phobia, and generalized anxiety disorder. Children may also show separation anxiety disorder, which involves excessive anxiety at times when they are separated from their parents. Cognitive biases such as expecting negative outcomes, negative self-talk, and interpreting ambiguous situations as threatening figure prominently in anxiety disorders in children and adolescents, as they often do among adults.
- **What are the distinguishing features of depression in childhood and adolescence?** Depressed children, especially younger children, may not report or be aware of feeling depressed. Depression may also be masked by seemingly unrelated behaviours, such as conduct disorders. Depressed children also tend to show cognitive biases associated with depression in adulthood, such as adoption of a pessimistic explanatory style and distorted thinking. Although rare, suicide in children does occur and threats should be taken seriously. Risk factors for adolescent suicide include gender, age, geography, race, depression, past suicidal behaviour, strained family relationships, stress, substance abuse, and social contagion.

DELIRIUM

Perhaps you have experienced a state of delirium upon waking and not knowing where you were, or experienced strange hallucinations and weird symptoms during a high fever or after an injury. The following picture of delirium is much like that experience: a temporary state in which there is a clouding of consciousness and the person has a feeling of being unaware of what is happening around them. It's a frightening state for those experiencing it and for those observing. It is really a state of brain failure, in which one lies between normal wakefulness and stupor or coma.

Clinical Picture

Delirium is a commonly occurring syndrome among the elderly that typically involves confusion, problems with concentration, and cognitive dysfunction such as memory disturbances (see Table 11.4 on page 416). The elderly are particularly at risk due to the brain changes that are associated with normal aging (Trzepacz, Meagher, & Wise, 2002) and because older adults are more prone to falls and are more susceptible to adverse reactions from medications (Chan, Nicklason, & Vial, 2001), experiences that can provoke a state of delirium. Impaired attention is considered the core cognitive disturbance. Most patients also experience a variety of other symptoms, including problems with orientation, language skills, mood, thinking, perception, and motor behaviour and the disruption of sleep–wake cycles.

For each individual the symptoms vary, but the patterns of symptoms are common. There are three typical patterns seen: acute onset (sometimes abrupt, but often over hours or days), fluctuant course (symptoms tend to wax and wane over any 24-hour period and typically worsen at night), and transient nature (in most cases, delirium resolves within days or weeks). With so many symptoms, it can easily be mistaken for other psychiatric or medical disorders. For example, in the elderly, delirium and the corresponding changes in

TABLE 11.4

Diagnostic Criteria for Delirium

A. A disturbance in attention (i.e., reduced ability to direct, focus, sustain, and shift attention) and awareness (reduced orientation to the environment).

B. The disturbance develops over a short period of time (usually hours to a few days), represents a change from baseline attention and awareness, and tends to fluctuate in severity during the course of a day.

C. An additional disturbance in cognition (e.g., memory deficit, disorientation, language, visuospatial ability, or perception).

D. The disturbances in Criteria A and C are not better explained by another preexisting established, or evolving neurocognitive disorder and do not occur in the context of a severely reduced level of arousal, such as coma.

E. There is evidence from the history, physical examination, or laboratory findings that the disturbance is a direct physiological consequence of another medical condition, substance intoxication or withdrawal (i.e., due to a drug of abuse or to a medication), or exposure to a toxin, or is due to multiple etiologies.

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mental state are often the early signs of infection or heart problems (Wahlund & Bjorlin, 1999). Knowing the pattern of onset and symptoms can also help distinguish delirium from dementia.

Before delirium becomes acute, it almost always involves a prodromal phase over two to three days of discontent, restlessness, poor concentration, anxiety, irritability, sleep disturbance, and, for some, nightmares. The course of the disorder is not always typical but does seem to follow general trends such as rapid onset, development over a period of days, and brevity; rarely does it last for more than a month.

Psychomotor, or body movement, disturbances might be either hyperactive, manifesting in restless or agitated behaviour, or hypoactive, slowing down movements. For some there is a shift from one extreme to the other (APA, 2013).

Diagnosis

In order to properly diagnose delirium, typically several measures are used. Initially, cognitive impairment is identified with instruments such as the Mini–Mental State Examination (MMSE) (Folstein, Folstein, & McHugh, 1975) (see Table 11.5 for sample questions used in this exam). Following this, a rating scale is used to determine the actual symptoms experienced and the type of onset of these symptoms. Formal delirium diagnosis requires documentation of acute onset and fluctuant course. The Delirium Rating Scale (DRS) (Trzepacz, Baker, & Greenhouse, 1998) is the most widely used scale to date, and although it requires interpretation by a skilled clinician, it has the significant advantage of distinguishing between the disturbances of depression, delirium, and dementia (see Table 11.6 for further details on distinguishing delirium and dementia). The DRS is performed by a skilled clinician who then also applies the DSM or ICD criteria for the full picture of dysfunction.

Treatment and Outcomes

Since most cases of delirium are reversible, the underlying causes, whether medical or injury, must be identified immediately. Treatment typically involves medication, environment changes, and family support (APA, 2013). The medications most widely used are neuroleptics (Fricchione et al., 2008).

TABLE 11.5

Sample Items from the Mini Mental State Examination

Orientation to Time
"What is the date?"

Registration
"Listen carefully. I am going to say three words. You say them back after I stop.
Ready? Here they are . . .
APPLE (pause), PENNY (pause), TABLE (pause). Now repeat those words back to me." [Repeat up to 5 times, but score only the first trial.]

Naming
"What is this?" [Point to a pencil or pen.]

Reading
"Please read this and do what it says." [Show examinee the words on the stimulus form.]
CLOSE YOUR EYES

TABLE 11.6

Distinguishing Features of Depression, Dementia, and Delirium

Features	Depression	Delirium	Dementia
Onset	Weeks to months (rapid)	Hours to days (acute)	Months to years (slow, indefinite)
Duration	Short	Variable	Long/lifetime
Mood	Consistent	Labile	Fluctuation
Disabilities	Recognizes	New disabilities appear (acute)	May conceal deficits
Answers	"Don't know"	May be incoherent (acute)	Offers response but not correct, but may be close to correct
MMSE	Performance fluctuates	Acute fluctuations	Fairly stable with downward trajectory over time
Progression	Resolves with treatment	Resolves with treatment	Ongoing

Source: Insel & Badger, 2002, p. 363. Copyright John Wiley & Sons. Reprinted with permission.

In some cases the individual recovers naturally, without intervention. In others, treatment is effective or the individual develops a progressive neurological deficit or dies from an underlying physical condition.

REVIEW IT

Delirium

- **Cognitive problems are seen in anxiety and dementia. What are the differences between these types of symptoms in anxiety and dementia?** In anxiety, cognitive problems, such as catastrophizing or ruminating, and more subtle changes in thinking can play a role in causing and maintaining the disorder. In dementia, the cognitive problems are the main defining feature.
- **How are delirium and dementia different?** The onset, characteristic, and pattern of symptoms are different in delirium and dementia. With delirium, the onset is sudden, depending on the cause; with dementia, the onset is insidious, slow, and often unrecognized. In delirium, the symptoms typically include disorganized, distorted, or fragmented speech with possible delusions or hallucinations; with dementia, there is a general impoverishment of thoughts, with words difficult to find yet misperceptions or hallucinations usually absent.

DEMENTIA

dementia A form of cognitive impairment involving generalized progressive deficits in a person's memory and learning of new information, ability to communicate, judgment, and motor co-ordination.

While delirium has a sudden onset, with symptoms that come and go, **dementia** has quite a gradual onset and is a gradual decline from previous levels of functioning. Dementia is a form of cognitive impairment involving generalized progressive deficits in a person's memory and learning of new information, ability to communicate, judgment, and motor co-ordination (APA, 2013) (see Table 11.7). You can imagine that this would have a significant impact on how an individual functions in his or her environment and with other people.

TABLE 11.7

Diagnostic Criteria for Major or Mild Neurocognitive Disorder Due to Alzheimer's Disease

A. The criteria are met for major or mild neurocognitive disorder.

B. There is insidious onset and gradual progression of impairment in one or more cognitive domains (for major neurocognitive disorder, at least two domains must be impaired).

C. Criteria are met for either probable or possible Alzheimer's disease as follows:

For major neurocognitive disorder:

Probable Alzheimer's disease is diagnosed if either of the following is present; otherwise, **possible Alzheimer's disease** should be diagnosed.

1. Evidence of a causative Alzheimer's disease genetic mutation from family history or genetic testing.
2. All three of the following are present:
   a. Clear evidence of decline in memory and learning and at least one other cognitive domain (based on detailed history or serial neuropsychological testing).
   b. Steadily progressive, gradual decline in cognition, without extended plateaus.
   c. No evidence of mixed etiology (i.e., absence of other neurodegenerative or cerebrovascular disease, or another neurological, mental, or systemic disease or condition likely contributing to cognitive decline).

For mild neurocognitive disorder:

Probable Alzheimer's disease is diagnosed if there is evidence of a causative Alzheimer's disease genetic mutation from either genetic testing or family history.

Possible Alzheimer's disease is diagnosed if there is no evidence of a causative Alzheimer's disease genetic mutation from either genetic testing or family history, and all three of the following are present:

1. Clear evidence of decline in memory and learning.
2. Steadily progressive, gradual decline in cognition, without extended plateaus.
3. No evidence of mixed etiology (i.e., absence of other neurodegenerative or cerebrovascular disease, or another neurological or systemic disease or condition likely contributing to cognitive decline).

D. The disturbance is not better explained by cerebrovascular disease, another neurodegenerative disease, the effects of a substance, or another mental, neurological, or systemic disorder.

Coding note: For probable major neurocognitive disorder due to Alzheimer's disease, with behavioral disturbance, code first **331.0** (**G30.9**) Alzheimer's disease, followed by **294.11** (**F02.81**) major neurocognitive disorder due to Alzheimer's disease. For probable neurocognitive disorder due to Alzheimer's disease, without behavioral disturbance, code first **331.0** (**G30.9**) Alzheimer's disease, followed by **294.10** (**F02.80**) major neurocognitive disorder due to Alzheimer's disease, without behavioral disturbance.

For possible major neurocognitive disorder due to Alzheimer's disease, code **331.9** (**G31.9**) possible major neurocognitive disorder due to Alzheimer's disease. (**Note:** Do *not* use the additional code for Alzheimer's disease. Behavioral disturbance cannot be coded but should still be indicated in writing.)

For mild neurocognitive disorder due to Alzheimer's disease, code **331.83** (**G31.84**). (**Note:** Do *not* use the additional code for Alzheimer's disease. Behavioral disturbance cannot be coded but should still be indicated in writing.)

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Clinical Picture

Beginning with mild yet annoying symptoms like forgetfulness, the person's symptoms become increasingly obvious and distressing. As the condition progresses, their capacity to care for themselves and for staying in touch with their surroundings gradually worsens.

Memory loss and disturbance of executive function (abstract thinking, organizing, and planning) are two major losses with dementia. In fact, the first sign of dementia is memory loss. As the memory loss becomes more pronounced, individuals become unable to remember even basic facts about themselves and their lives.

Generally, deterioration continues downward over the course of months and years. A Canadian study confirmed that the average duration of life after the individual contacts a doctor for memory problems is about 3.3 years (Wolfson et al., 2001). This contradicts the duration of 7 to 10 years that was taught for many years.

Neuropathy

There are a number of brain abnormalities now known to be characteristic of dementia: amyloid plaques, neurofibrillary tangles, reduction in neurotransmitters involved in memory, and inflammation of the brain. Plaques and tangles are also found in healthy brains but are found in far greater numbers in people with Alzheimer's disease.

The **amyloid plaques** are made largely of protein, called *beta amyloid*, or *A-beta*, and are split from a much larger protein molecule known as APP. Both APP and A-beta are present in normal brains. The key problem in Alzheimer's disease is that abnormally high amounts of A-beta accumulate in the brain, overwhelming the enzymes and other molecules whose job it is to clear it away (Cummings, 2004). As well, the clearing away process itself appears to be defective.

The **neurofibrillary tangles** are made of a protein called *tau*, which, like amyloid, occurs in normal nerve cells. In Alzheimer's disease, tau becomes chemically altered and piles up as thread-like tangles, impairing the protein's key roles in nerve cells. One of these roles is in nerve sprouting, an important feature of self-repair in the nervous system.

The third very important alteration in the brain concerns the neurotransmitter **acetylcholine** (Ach), which is important in memory. It seems that one of the areas affected the earliest and most severely is in the base of the forebrain involved in Ach release. The depletion of Ach contributes significantly to the memory deficits (Schliebs & Arendt, 2006). Drugs that inhibit the breakdown of the already-reduced levels of Ach can be beneficial for patients.

Another common characteristic of the disease is inflammation of the brain. Whenever and wherever the body suffers trauma or is attacked by some kind of potentially threatening influence, such as an infection or a toxin, it defends itself in part by mounting an inflammatory response. This immune response also occurs in the Alzheimer's brain.

amyloid plaques Hallmarks of Alzheimer's disease: the accumulation of protein fragments, normally broken down in healthy brains, that accumulate to form hard, insoluble plaques between nerve cells (neurons) in the brain.

neurofibrillary tangles Pathological protein aggregates (or brain lesions) found within brain cells (in the cerebral cortex and hippocampus) in patients with Alzheimer's disease and thought to contribute to the degradation of neurons in the brain.

acetylcholine A neurotransmitter important in memory. Abbreviated *Ach*.

Causes and Diagnosis

The diagnosis of dementia involves a thorough physical and psychological assessment to evaluate symptoms such as aphasia (difficulty remembering words or being completely unable to speak, read, or write), apraxia (loss of the ability to execute or carry out learned purposeful movements), and other memory loss.

There are at least 50 disorders known to cause dementia (Bondi & Lange, 2001), including degenerative diseases such as Huntington's and Parkinson's. Other causes are severe head injury; inhalation of toxic substances; oxygen deprivation; strokes; infectious diseases such as syphilis, meningitis, and AIDS; intracranial tumours; and certain dietary deficiencies, especially of the B vitamins. The most common cause of

Alzheimer's disease Fatal neurogenerative disorder that accounts for the majority of dementia cases.

dementia, accounting for about 56% of all cases, is **Alzheimer's disease**, a fatal neurodegenerative disorder (Selkoe, 1993). The prevalence of Alzheimer's disease is often inaccurately reported; only 5% to 7% of the over-65 population suffer with this disorder (Hy & Keller, 2000).

People who have a mother with Alzheimer's disease appear to be at higher risk for getting the disease than those individuals whose fathers are afflicted. In a new study (Mosconi et al., 2010), researchers set out to evaluate Alzheimer's risk in healthy, cognitively normal individuals by measuring their cerebrospinal fluid proteins, which are known to be altered in Alzheimer's. They compared individuals with a maternal or paternal history of Alzheimer's to individuals with no family history. They found that only individuals whose mothers had Alzheimer's showed altered levels of a protein called amyloid, a major hallmark of Alzheimer's pathology. In contrast, individuals whose fathers had Alzheimer's and those with no family history had protein levels within normal range. The prevailing hypothesis is that the protein clumps together into plaques, which damage the brain's nerve cells, causing the characteristic symptoms of Alzheimer's. Thus, while a person might have a family history of Alzheimer's disease, which significantly increases the risk for developing this disorder, which of that person's parents has the disease is very important in determining the risk.

The diagnosis of Alzheimer's is often given only after all other potential causes of dementia are ruled out. Only in the past decade have reliable screening and diagnostic tools been available. Researchers have discovered a previously unknown substance in spinal fluid that can be used to diagnose Alzheimer's disease: a beta-amyloid protein called Abeta16 ("New Marker," 2009). Independent studies show that Alzheimer's patients have higher levels of the protein in their spinal fluid than do healthy individuals (Fukumoto et al., 2010). Researchers are proposing that the discovery of the new protein could be used to diagnose patients with Alzheimer's and also help determine which medications are most effective for the disease.

In fact, researchers at Purdue University are in the early stages of designing a molecule to stop the debilitating symptoms of Alzheimer's disease. It has been shown in its first phase of clinical trials to be safe and to reduce biomarkers, like the beta-amyloid protein, for the disease ("New Alzheimer's Treatment," 2008).

Detecting Alzheimer's disease early in the process is ideal. Tools such as brain scans and memory tests, like the neuropsychological memory test in Figure 11.2, currently appear to predict best whether a person with cognitive problems might develop Alzheimer's

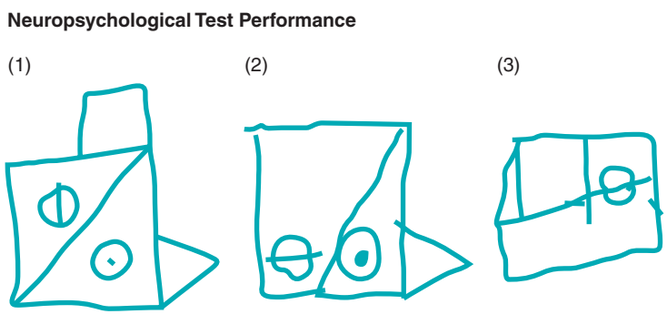

FIGURE 11.2 Neuropsychological test performance.
These drawings represent part of the neuropsychological test performance of a 59-year-old woman with a diagnosis of Alzheimer's disease. The figure at the left (1) was drawn by the psychologist, who then handed the piece of paper to the patient and asked her to make an exact copy of the figure next to the original. After the patient had completed her replica (2), the piece of paper was turned over and she was asked to draw the figure again, this time from memory. The figure that she drew based on memory is presented at the right (3).

Source: Oltmanns, Thomas F. & Emery, Robert E. *Abnormal Psychology,* 6th ed. © 2010. Reprinted and electronically reproduced by permission of Pearson Education, Inc., Upper Saddle River, New Jersey.

disease. In 2010, the American Academy of Neurology reported on tests performed on 85 people with mild cognitive impairment (MCI) who were part of a larger study called the Alzheimer's Disease Neuroimaging Initiative (Landau et al., 2010). Several techniques were used in assessing the subjects in the study, including MRI brain scans, to measure the size of a participant's hippocampus, the part of the brain responsible for learning and memory; measurement of amyloid proteins (such as those discussed above); and PET brain scans, to detect metabolic abnormalities in the brain that might signal Alzheimer's disease. People who showed abnormal results on both PET scans and episodic memory tests were nearly 12 times more likely to develop Alzheimer's disease than those who scored normally on both measures (Landau et al., 2010). In other words, the PET scans and the memory tests best predicted later development of Alzheimer's disease.

When Alzheimer's disease is diagnosed, the clinician also specifies any concurrent neurocognitive, neurological or systemic illness, and/or depressive disorder. (APA, 2013).

Treatment and Outcomes

Currently, there is no cure for Alzheimer's disease, but some medications can slow down the decline of memory, language, and thinking abilities. Aricept, Exelon, and Reminyl have been approved for use in Canada to treat symptoms in people with mild to moderate Alzheimer's disease. For those with moderate to advanced Alzheimer's disease, Ebixa (memantine hydrochloride) has been conditionally approved. All are cholinesterase inhibitors, drugs that inhibit the breakdown of acetylcholine.

Another line of treatment research is focused on developing vaccines that might help clear away any accumulated plaques.

REVIEW IT

Dementia

- **Is memory impairment the only indication that a person is developing dementia?** No, memory impairment is not the only sign. Decline in executive functioning is another sign that is often revealed through difficulty performing familiar tasks. There might also be problems with language and difficulties with orientation of time and space as well as decreased judgment and changes in mood or behaviour.

Define It

acetylcholine, 419
Alzheimer's disease, 420
amyloid plaques, 419
attention-deficit/hyperactivity disorder, 402
autism spectrum disorder, 388
autistic thinking, 388
conduct disorder, 406
cultural-familial intellectual disability, 396
cytomegalovirus, 395
dementia, 418
Down syndrome, 393
dyslexia, 399
hyperactivity, 402
individual education plan, 401
lateralization, 390
learning disorder, 399
mainstreaming, 396
neurofibrillary tangles, 419
oppositional defiant disorder, 407
phenylketonuria, 395
separation anxiety disorder, 410
Tay-Sachs disease, 395
theory of mind, 390
time out, 409

Think About It

- Have you known anyone with autism spectrum disorder or worked with children who have the disorder? What were the most prominent features of the disorder? What, if any, interventions were helpful?
- Do you think children with intellectual disabilities should be mainstreamed into regular classes? Why or why not?
- Do you think people with learning disorders should be given special consideration when given standardized tests (e.g., provincial achievement tests), such as having extra time? Why or why not?
- What are the risks and benefits of using stimulant medication such as Ritalin in treating ADHD in children?
- Do you know someone with Alzheimer's disease? Were there signs of memory loss early on? What interventions are helpful?

Weblinks

Autism Society Canada http://autismsocietycanada.ca
This website is a primary source for Canadian information about autism spectrum disorders, services, and resources.

Canadian Association for Community Living www.cacl.ca
This site provides information about Canadian community supports and services for persons of all ages who have intellectual disabilities.

Special Olympics Canada www.specialolympics.ca
Homepage for Special Olympics Canada, a nonprofit organization dedicated to enriching the lives of Canadians with intellectual disabilities.

Learning Disabilities Association of Canada (LDAC) www.ldac-taac.ca
This site is Canada's central repository for information about learning disabilities, resources, and supports.

Children and Adults with Attention Deficit Disorders (CHADD) www.chadd.org
This Canadian site is dedicated to the support, education, and betterment of the lives of children and adults with attention deficit disorders.

Children and Youth www.ontario.cmha.ca/children_and_youth.asp
This page from the Canadian Mental Health Association provides information about a wide range of mental and emotional problems of children and youth.

Alzheimer Society www.alzheimer.ca
The Alzheimer Society is a leading national not-for-profit health organization. Its aim is to improve the quality of life for Canadians affected by Alzheimer's disease and related dementias and advance the search for the cause and cure.

Canadian Dementia Action Network www.cdan.ca/aboutus.html
CDAN brings together Canada's world-class biomedical researchers and clinicians for the purpose of quickly identifying promising treatments for Alzheimer's disease and related dementias (ADRD).

Abnormal Behaviour Across the Lifespan

Neurodevelopmental Disorders

Autism Spectrum Disorder

One of the most severe disorders of childhood, autism is a chronic lifelong condition, the causes of which are mostly unknown.

Features

- Impaired social communications and social interactions
- Restricted, repetitive, and stereotyped behaviour patterns

Treatment

- Early intervention is important
- Intensive behavioural treatment (IBT)

Intellectual Disability

Broad delay in the development of cognitive and social functioning

Causes of Intellectual Disabilities

- Chromosomal and genetic disorders
- Prenatal infectious diseases and brain injuries
- Cultural or familial causes, such as being raised in an impoverished home environment

Intervention

- Mainstreaming: having all students with disabilities included in the regular classroom
- Behavioural techniques that help children and adults acquire skills in areas such as personal hygiene, work, and social relationships

Learning Disorders

Average or higher intelligence, but shows inadequate development in reading, math, or writing skills that impairs school performance or daily activities

Subtypes of Learning Disorders

- Impairment in mathematics
- Impairment in written expression
- Impairment in reading

Intervention

- Remediation and accommodation of specific skill deficits
- Target information processing style and academic strengths
- Individual education plan (IEP): learning and behaviour outcomes contract

Theoretical Perspectives

- Neurobiological problems with visual or auditory sensation and perception
- Genetic factors in dyslexia

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Anxiety and Depression

Separation Anxiety Disorder
- Characterized by extreme fears of separation from parents or others on whom the child is dependent

Perspectives on Anxiety Disorders in Childhood
- Psychoanalytic theorists: anxiety symbolizes unconscious conflicts
- Cognitive model: expecting negative outcomes, negative self-talk, and interpreting ambiguous situations as threatening
- Learning (anxiety-control) and cognitive-behavioural therapy

Depression in Childhood and Adolescence
- Sense of hopelessness, displays cognitive errors and negative attributions, lower perceived competence or self-efficacy, and lower self-esteem
- Episodes of sadness, crying, and apathy, as well as insomnia, fatigue, and poor appetite

Correlates and Treatment of Depression in Childhood and Adolescence
- Frequently related to family problems and conflicts
- Cognitive-behavioural and family therapy

Attention-Deficit and Disruptive Behaviour Disorders

Attention-Deficit/Hyperactivity Disorder (ADHD)

Characterized by excessive motor activity and inability to focus one's attention

Theoretical Perspectives
- Brain abnormalities involved in regulating the processes of attention, inhibition of motor (movement) behaviour, and executive control
- Neuropsychological studies challenge the long-held belief that ADHD is a single coherent clinical entity.

Intervention
- Cognitive-behavioural treatment combined with medication

Conduct Disorder

Pattern of purposeful antisocial behaviour that violates social norms and the rights of others

Oppositional Defiant Disorder

Characterized by excessive oppositionality or tendencies to refuse requests from parents and others

Theoretical Perspectives
- Family and parenting factors
- Biased processing of social information

Treatment
- Multisystemic therapy (MST) approach
- Cognitive-behavioural therapy

Cognitive Disorders of Aging

Delirium

Commonly occurring syndrome among the elderly that typically involves confusion, problems with concentration, and cognitive dysfunction such as memory disturbances

Clinical Picture
- Problems with orientation, language skills, mood, thinking, perception, and motor behaviour and the disruption of sleep–wake cycles
- Acute onset, fluctuant course, and transient

Treatment and Outcomes
- Medication (neuroleptics), environment changes, and family support

Dementia

A form of cognitive impairment involving generalized progressive deficits in a person's memory and learning of new information, ability to communicate, judgment, and motor co-ordination.

Clinical Picture
- Memory loss and disturbance of executive function (abstract thinking, organizing, and planning) are two major losses with dementia.

Alzheimer's Disease
- Fatal neurogenerative disorder that accounts for the majority of dementia cases