LEARNING OBJECTIVES

15.1 Summarize the major cognitive and brain changes in older adulthood.

15.2 Describe the characteristics, prevalence, and causes of delirium.

15.3 Describe the characteristics, prevalence, and diagnosis of Alzheimer’s disease.

15.4 Describe the characteristics, prevalence, and diagnosis of other mild and major neurocognitive disorders.

15.5 Identify methods of prevention, treatment, and support for individuals with neurocognitive disorders.
November 5, 1994

My fellow Americans, I have recently been told that I am one of the millions of Americans who will be afflicted with Alzheimer’s disease.

Upon learning this news, Nancy and I had to decide whether as private citizens we would keep this a private matter or whether we would make this news known in a public way. In the past, Nancy suffered from breast cancer and I had my cancer surgeries. We found through our open disclosures we were able to raise public awareness. We were happy that as a result, many more people underwent testing. They were treated in early stages and able to return to normal, healthy lives.

So now we feel it is important to share it with you. In opening our hearts, we hope this might promote greater awareness of this condition. Perhaps it will encourage a clearer understanding of the individuals and families who are affected by it.

At the moment I feel just fine. I intend to live the remainder of the years God gives me on this Earth doing the things I have always done. I will continue to share life’s journey with my beloved Nancy and my family. I plan to enjoy the great outdoors and stay in touch with my friends and supporters.

Unfortunately, as Alzheimer’s disease progresses, the family often bears a heavy burden. I only wish there was some way I could spare Nancy from this painful experience. When the time comes, I am confident that with your help she will face it with faith and courage.

In closing, let me thank you, the American people, for giving me the great honor of allowing me to serve as your president. When the Lord calls me home, whenever that day may be, I will leave with the greatest love for this country of ours and eternal optimism for its future.

I now begin the journey that will lead me into the sunset of my life. I know that for America there will always be a bright dawn ahead.

Thank you, my friends. May God always bless you.

Sincerely,
Ronald Reagan

During the drive home from the hospital (after 11 weeks), I saw that I did not recognize the route home, even though it was a route quite familiar to me. At that point, I realized that some of the geography of the city was lost to me. But I did recognize my home and the neighborhood when we arrived there. My house was familiar to me, but I didn't remember where some things were kept and how some things were used. I had to relearn how to play the stereo, set the alarm clock, use the calculator, change a razor blade, etc. All of these things were relearned, but because of the short-term memory problem, it often took several trials to relearn and keep these things in mind.

So, many things I had learned and that had made me feel like a competent person seemed to have been lost, and I wondered if I could be an adequate husband, father, or worker again. Combined with this, I felt to some extent that I had lost my identity. This was not total or extreme, but there were some questions in my mind about beliefs, values, and purposes in life. In addition, I felt that I had lost some of my cultural background when I had difficulty remembering some of the customs, traditions, and beliefs of the groups to which I belonged. This produces a feeling of being somewhat alone.


We all age. As we age, our mental and physical abilities change. Some of these changes happen gradually and appear to be related to genetic differences. Huntington’s disease or Alzheimer’s disease are examples of such genetic disorders. The letter from Ronald Reagan shows that he understood that he was experiencing gradual memory changes. Other changes happen more quickly. Events such as a stroke or heart attack may lead to not only physical changes but also changes in mental processing. The description of Malcolm Meltzer, who was both the author and the subject of the case report, resulted from a heart attack that influenced brain function and was a sudden event. This chapter emphasizes neurocognitive disorders typically seen in older individuals. These disorders represent a loss of cognitive abilities not related to normal aging. Previously, these disorders were referred to as dementias.

We all know individuals or relatives who as they age show problems with memory or physical activities. Most of us also know individuals who continue to be productive well into their eighties and nineties. This has come to be called successful aging (Rowe & Kahn, 1987). The characteristics of successful aging include the following:

1. Freedom from disability and disease
2. High cognitive and physical functioning
3. Social activity including both having friends and being productive

The idea of successful aging emphasizes that life is more than just living a long time. It also includes a sense of connectedness and a close interaction with one’s environment and self. For example,
Abnormal Psychology

many of the aging performers associated with 1960s and 1970s music, such as Bob Dylan; Keith Richards; Carole King; Rod Stewart; Cher; and the group Crosby, Stills, and Nash, still perform today.

The aging of each of us is related to a number of factors. These include the genes we received from our parents. These also include the experiences that happen to us, including the stresses of everyday life as well as the positive events in our lives. Aging also involves choices we make in terms of what we eat, how we exercise, and the type of work we do. Scientists are also asking what one can do to increase brain health (Brem & Sensi, 2018). To place neurocognitive changes in perspective, it is important to have an understanding of normal aging. This chapter begins by noting the worldwide changes in aging. This is followed by research considerations of factors that may prevent or delay neurocognitive disorders. After a section on delirium, the major neurocognitive disorders are described, with special attention given to Alzheimer's disease.

Normal Cognitive Changes Related to Aging

Throughout the world, better public health conditions, such as clean water and sanitation as well as medical and disease-prevention procedures, have led to an increasing life expectancy. In the United States, individuals age 65 and older went from less than 2% of the population in 1900 to over 12% of the population in 2010 (see Figure 15.1). It is estimated that by 2060, over 20% of the U.S. population will be over 65 (see Figure 15.2). Aging worldwide is discussed in the cultural LENS in this chapter.

With better public health conditions, it is noncommunicable diseases such as cardiovascular problems and cancer that are the major causes of death among older individuals in both developed and developing countries. With individuals living longer, there is also a greater chance of developing neurocognitive disorders such as Alzheimer's and Parkinson's. Within the same age group, mortality is higher for people with neurocognitive disorders than for those without. Figure 15.5 shows the incidence of mild and moderate neurocognitive disorders for Europe, East Asia, and the United States. From this figure, one can note that the increase in neurocognitive disorders over the life span is similar throughout the world.

Do Cognitive Abilities Change With Age?

Cognitive abilities do change as one ages. In order to understand the changes seen in neurocognitive disorders, it is important to examine the normal changes in these cognitive abilities.
CULTURAL LENS

Aging Around the World

How old will you be in 2050 or 2060? You may be in your fifties or sixties or even older. With better access to clean water, fresh food, sanitation, and improved health care, individuals worldwide are living longer. In fact, it is estimated that by around 2020, there will be more people over 65 than children under 5 worldwide (see Figure 15.3). Data show that in North America, Europe, and Russia, over 11% of the population are 65 and older (see Figure 15.4). In fact, in Japan, Italy, and Germany, over 20% of their populations are 65 and older. This data can be influenced by a number of factors including birth rate and immigration as well as better health practices.

With the expectation of an increasing percentage of the population being older, many countries are asking what facilities and resources will be needed for an older population. A large number of cities in more than 37 countries are participating in the World Health Organization (WHO) Global Network of Age-Friendly Cities and Communities. In addition, AARP (formerly the American Association of Retired Persons) has developed reports on how different countries are planning and developing programs for older individuals (http://arc.aarpinternational.org/home).

Since we know that an active life with social contacts can help to prevent cognitive decline (Ballesteros, Kraft, Santana, & Tziraki, 2015; I. E. M. Evans et al., 2018; I. E. M. Evans, Martyr, Collins, Brayne, & Clare, 2018), communities in many countries have focused on healthy aging. One important component is accessibility in terms of transportation, access to and within buildings, and affordable housing. Another important component is social engagement. This includes the creation of community centers as well as providing

**FIGURE 15.3** When Will the Population of the World Have More Individuals Over 65 Years Than Those Under 5 Years of Age?

It is estimated that this will happen before 2020. This figure shows young children and older people as a percentage of the global population.

Source: He, Goodkind, & Kowal (2016).

**FIGURE 15.4** Percentage of Population Age 65 and Over: 2015 and Projected to 2050

Source: He et al. (2016).

(Continued)
Abnormal Psychology

across the life span. Timothy Salthouse (2004, 2011) combined data from 33 separate studies with 6,832 individuals to follow cognitive changes in terms of five major categories. Although memory loss is often seen as a problem of aging, other abilities are also affected. As can be seen in the graphs (Figures 15.6 through 15.10), there is a consistent picture of change over the life span for each category with the sole exception of vocabulary ability, which does not decrease over the life span.

The first category examined by Salthouse (2004) is vocabulary, based on measures in which the individual provided definitions of words, or named an object in a picture, or selected antonyms or synonyms. As can be seen in Figure 15.6, vocabulary ability remains fairly constant across the life span, and even increases slightly as individuals approach their sixties.

The second ability is perceptual speed. This reflects the ability to quickly compare patterns of letters or match symbols. As can be seen in Figure 15.7, this ability declines steadily from age 30 on.

The third factor is episodic memory. Episodic memory in these studies refers to the ability to recall information from stories or other stimulus items. As can be seen from Figure 15.8, these types of memory ability remain fairly stable until around 60 years of age, and then they begin to drop off.

The fourth category is spatial visualization. Tests of spatial visualization require someone to move between a two-dimensional and a three-dimensional figure or determine shapes to fill a larger shape or imagine how a pattern would appear in a folded piece of paper. As can be
FIGURE 15.5 Does Where You Live Influence the Development of Neurocognitive Disorders?

The development of neurocognitive disorders increases with age at similar rates in Europe, the United States, and Asia. This figure shows the incidence of mild and moderate neurocognitive disorders for Europe, East Asia, and the United States.

Note: CFAS is a study from England and Wales. Eurodem is results from Europe. Jorm is based on research in Europe, the United States, and Asia.


FIGURE 15.6 Does Your Vocabulary Increase or Decrease as You Age?

It tends to increase until your sixties. This figure shows vocabulary abilities across the life span based on the Wechsler Adult Intelligence Scale (WAIS) vocabulary, picture vocabulary, and synonym and antonym measures.

Source: Salthouse (2004, p. 553), with permission from Elsevier.

FIGURE 15.7 How Does Aging Affect Your Processing Speed?

After 20 years of age, the perceptual speed across the life span decreases. This figure is based on three different measures.

Source: Salthouse (2004, p. 553), with permission from Elsevier.

FIGURE 15.8 Does Your Episodic Memory Increase or Decrease as You Age?

It stays about the same until age 60 and then decreases. This figure shows episodic memory across the life span based on measures of free recall, logical memory, and paired associates.

Source: Salthouse (2004, p. 553), with permission from Elsevier.
Abnormal Psychology

seen from Figure 15.9, this ability is best in a person's twenties and thirties. It decreases in the thirties and then remains stable through a person's forties to sixties. It then decreases rapidly.

The fifth category is reasoning. This includes determining geometric patterns needed to complete a sequence or completing word or letter patterns. As can be seen from Figure 15.10, the ability to determine the next symbol in a set of geometric patterns is best in individuals in their twenties and then decreases gradually into their eighties.

**RESEARCH TERMS TO KNOW**

**Z-Score**

When a researcher has more than one measure of a particular concept, he or she needs a way to show the different measures on the same scale. In the case of cognitive abilities across the life span (Figures 15.6 through 15.10), each graph is based on three or four different measures of the same cognitive ability. Since each measure has a different mean and standard deviation, plotting each measure could give a confusing picture. However, if you converted these to standard scores such that each had the same mean, then you could plot them. This is what a z-score does. Every measure when converted to a z-score has a mean of zero. What you plot in these graphs is the standard deviation or differences from the mean. The actual formula for a z-score is the difference between a particular score and the mean of the scores divided by the standard deviation. Simply put, z-scores allow for the comparison of different measures on the same scale.

**How the Brain Changes With Age**

Two consistent findings are that older adults, generally defined as 65 years of age or older, show changes in brain structure and that they use their brains in different ways from younger...
adults (Mather, 2016; Park & Reuter-Lorenz, 2009; Reuter-Lorenz & Park, 2010). In terms of brain volume, reduction is seen in the hippocampus, cerebellum, dorsolateral prefrontal cortex (dlPFC), and caudate nucleus, which are areas related to executive control and memory. Along with hippocampus changes, reductions of dopamine with age also play a role in the inability to remember events (Leal & Yassa, 2015).

Areas involved in emotional processes such as the amygdala and ventromedial prefrontal cortex show little structural and functional decline compared with other areas. Likewise, the visual cortex and the entorhinal cortex show little reduction in volume with age. The entorhinal cortex is located in the temporal lobe and serves as a hub that connects the hippocampus and the neocortex. It is involved in memory and spatial navigation. It is one of the first areas affected in Alzheimer’s disease.

In order to solve problems, older individuals use their brains differently. Even when younger and older adults both perform a memory task successfully, the older adults recruit more brain regions than do younger adults. One interpretation is that older adults need additional executive resources to perform the same task. This is referred to as compensation. That is, in order to optimize their performance, older adults perform the same task using additional neural circuitry (see Figure 15.11). When older adults use just the brain areas that are activated in younger individuals, they do not perform the tasks as well as younger adults.

As you are sitting and doing nothing, the default network in your brain turns on. When you start performing a task, more task-related networks are activated and the default network is inhibited. In younger individuals, the same pattern of activity in the frontal lobes, the parietal lobes, the temporal lobes, and the cingulate is seen across a variety of tasks in numerous studies (Beason-Held, 2011). In older individuals, the number of brain areas involved in the default network is larger, especially in the frontal lobes. Older adults also have a more difficult time turning off the default network. It is assumed that this is related to the problems some older adults have in shifting cortical resources to new tasks.

One interesting study followed a group of individuals in Scotland who were given an IQ test at age 11 and then again at age 79 (www.lothianbirthcohort.ed.ac.uk/). Beginning at age 79, a subset of the original participants were also followed until they were 87. Overall, a number of behavioral factors such as not smoking and being more physically active and fit were associated with better mental aging. Also, eating fresh fruits and vegetables was associated with better health (Corley, Kyle, Starr, McNeill, & Deary, 2015). The effect of alcohol consumption on cognitive abilities in aging was related to one’s ability to metabolize alcohol (Ritchie et al., 2014). Individuals with higher genetic ability to process alcohol showed relative improvements in cognitive ability with more consumption, whereas those with low processing capacity showed a negative relationship between cognitive change and alcohol consumption. In terms of personality and cognitive abilities, low levels of neuroticism were associated with better cognitive aging (Zammit, Starr, Johnson, & Deary, 2014). It was also found that those individuals who had stimulating lifestyles including complex work environments showed better cognitive abilities in later life (Smart, Gow, & Deary, 2014). Although reduced, this association continued even when the age 11 IQ was considered.

A person’s genetics was also related to cognitive functioning (Cacciaglia et al., 2019). What the researchers discovered was that those individuals with a variant of the APOE gene, known as the APOE4 allele, showed more cognitive decline even in the absence of a neurocognitive disorder. Figure 15.12 shows that at age 11, there was little difference in the
IQ scores of those with and without the APOE4 allele. However, by age 79, those with the APOE4 allele showed a lower IQ score, whereas those without this allele showed a similar IQ score to that seen when they were age 11. On the logical memory task, those with the APOE4 allele showed a decline over the next 8 years.

**Delirium**

Delirium is a short-term state of confusion. This short-term condition is characterized by a change in cognitive processing such as an inability to focus attention or problems with language, memory, or orientation. The idea of delirium dates back to at least the time of Hippocrates (Caraceni & Grassi, 2011; Lindesay, Rockwood, & Macdonald, 2002). In the nineteenth century, the term confusion was often used in relation to delirium. Physical illness, toxins including alcohol, and infections were historically seen as causes of delirium. The modern concept of delirium is based largely on the work of Lipowski (1980), who brought together a spectrum of acute cognitive problems that influence consciousness and are associated with medical illness. The fact that signs and symptoms associated with delirium fluctuate and are seen to be reversible has been used to differentiate it from more stable neurocognitive disorders.

**Characteristics, Prevalence, and Causes of Delirium**

The fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM–5)* (APA, 2013) describes delirium as a disturbance in attention and awareness. That is, the person is less aware of his or her environment and has a reduced ability to direct and change the focus of attention. Additional cognitive disturbances such as memory, language, or perceptual disorders are seen. Further, delirium develops over a short period of time and shows fluctuations in severity during the day. In general, delirium develops in relation to another medical condition, including medication side effects or toxins.
Delirium occurs in up to 56% of older adults in general hospital populations (R. Jones, Kiely, & Marcantonio, 2010). It is seen in 15% to 53% of older individuals after a medical operation and in 70% to 87% of those in intensive care (APA, 2013). In a community sample, the prevalence of delirium is low (1%–2%) but increases with age to where the prevalence is 14% among those over 85. Overall, delirium is seen as a disturbance of consciousness that cannot be accounted for by neurocognitive disorders. Its onset is typically abrupt and lasts for only a few hours or days. It can be caused by a number of underlying physiological disturbances. Those who show weakened connection between brain areas may be more at risk for delirium (Van Montfort et al., 2019). As seen in the prevalence data, it is a frequent complication of hospitalization for older populations.

Delirium can manifest in a number of ways and be related to a number of causes. Problems with memory of recent events is one common manifestation of delirium. However, memory of older experiences also occurs. Language problems are such that the person sounds incoherent, disorganized, and rambling. Writing problems may also be present. The person may also appear disoriented. Although delirium more often occurs in older individuals, it can also be seen in young children. The following is the case study of Bobby Baldwin (not his real name).

**CONCEPT CHECK**

- What are the primary characteristics of successful aging? What can individuals and communities do to promote successful aging, and why is that important?
- What is the relationship between cognitive changes in older adults and mental illness?

**CASE OF BOBBY BALDWIN**

**Delirium**

Bobby Baldwin is a 7-year-old Caucasian male. He was taken to the hospital because he appeared to have ingested insecticide. While at the hospital, he was interviewed by a health care professional. When asked his address, he answered correctly but then spelled the name of the city as the word *house*. When asked about his birthday, he gave the wrong year for when he was born. He then said, “Jerry, get up and stand on this. Who said you could wear a blue jacket?” When asked about his hospital experience, he gave the wrong year for when he was born. He then said, “I was in the hospital 2 or 3 days.” When asked about what he last ate, he said, “Today is Thursday—it’s April—A B C. That is the alphabet. Jerry doesn’t know the alphabet.” He misperceived the 6’4”, 200-pound male health care worker as his mother. When identifying common objects, he answered all incorrectly—that is, a pen clip was a cross, a pen point was a cross and then a tent, and to the remaining objects he said, “How many hamburgers did you get?” He was unable to draw the geometric objects. Bobby also lost track in the middle of some of the tasks, such as counting backward.

When reexamined some 3 days later, Bobby was alert. He gave his correct birthday and address and knew what day it was. He could remember what he had last eaten. He could also draw geometric objects. He did complain of fatigue during the examination. The health care professional who examined Bobby concluded that there were no longer signs of delirium. Bobby did, however, show signs of mild central nervous system problems related to having ingested the insecticide.

Mild and Major Neurocognitive Disorders

Neurocognitive disorders represent a condition in which a person shows cognitive deficits that are greater than those experienced with normal aging. This section will cover the typical characteristics, prevalence, DSM–5 criteria, and various types of neurocognitive disorders.

Characteristics, Prevalence, and Diagnosis of Neurocognitive Disorders

Neurocognitive declines are typically shown in memory-related processes. Some older individuals call mild memory problems “senior moments,” in which they cannot remember someone’s name or a particular word. Some individuals show a progression in which it becomes more difficult to encode recent information or retrieve it from long-term memory. It is not uncommon for people with memory problems in old age to be able to remember events in great detail from their distant past while showing real problems remembering recent events.

In addition to memory problems, there can also be problems in a number of other cognitive processes. Complex attentional tasks in which the individual must divide her attention or pay attention to more than one process at a time may be difficult. Problems in executive functions that require planning, mental flexibility, and learning from mistakes can also be compromised. Declines in other tasks involving language and spatial abilities also occur in neurocognitive disorders. In addition to cognitive tasks, the person may show declines in social processes such as recognizing the intentions or emotions of others and being able to regulate one’s own behavior.

As noted previously, a number of cognitive abilities are shown to decline with age. However, in many older individuals, these do not interfere with living a normal life. They may rely more on lists or friends to help them live independently. When cognitive or social deficits are greater than those seen with normal aging, this decline is diagnosed as a mild neurocognitive disorder (see Table 15.1). If the declines are severe and interfere with one’s ability to function independently, then this is diagnosed as a major neurocognitive disorder (see Table 15.2). In DSM–5, the diagnosis of major neurocognitive disorder replaced the term dementia.

### TABLE 15.1 DSM–5 Diagnostic Criteria for Mild Neurocognitive Disorder

<table>
<thead>
<tr>
<th>A. Evidence of modest cognitive decline from a previous level of performance in one or more cognitive domains (complex attention, executive function, learning and memory, language, perceptual motor, or social cognition) based on:</th>
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<tbody>
<tr>
<td>1. Concern of the individual, a knowledgeable informant, or the clinician that there has been a mild decline in cognitive function; and</td>
</tr>
<tr>
<td>2. A modest impairment in cognitive performance, preferably documented by standardized neuropsychological testing or, in its absence, another quantified clinical assessment.</td>
</tr>
<tr>
<td>B. The cognitive deficits do not interfere with capacity for independence in everyday activities (i.e., complex instrumental activities of daily living such as paying bills or managing medications are preserved, but greater effort, compensatory strategies, or accommodation may be required).</td>
</tr>
<tr>
<td>C. The cognitive deficits do not occur exclusively in the context of a delirium.</td>
</tr>
<tr>
<td>D. The cognitive deficits are not better explained by another mental disorder (e.g., major depressive disorder, schizophrenia).</td>
</tr>
</tbody>
</table>

Source: Reprinted with permission from the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (Copyright 2013), American Psychiatric Association. All Rights Reserved.

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The diagnosis of a neurocognitive disorder is a two-step process. The first step is to determine if the person is showing normal changes related to aging, a mild neurocognitive disorder, or a major neurocognitive disorder. This initially can be accomplished by using the Mental Status Exam as described in Chapter 4. With mild neurocognitive disorder, the person is still able to function independently and perform activities such as paying bills and taking medications on his or her own (see Table 15.1). When the person is no longer able to function independently, the diagnosis is major neurocognitive disorder. The second step is to determine what caused the neurocognitive disorder. This includes Alzheimer's disease, frontotemporal lobar degeneration, Lewy body disease, vascular disease, substance/medication use, human immunodeficiency virus (HIV) infection, prion disease, Parkinson's disease, Huntington's disease, or another medical condition. These major causes of neurocognitive disorders will be described in the next section. Let us begin with an extended discussion of Alzheimer's disease.

It has been estimated that some type of neurocognitive disorder will impact around 15% of all individuals over age 65 and up to 45% of those over age 80. Since more extreme cases will require extensive care in a facility such as a nursing home, neurocognitive disorders require a large expenditure of resources on the part of the individual, his or her family, and society as a whole. Family members and caregivers in turn are at risk for a number of psychological problems including depression, anxiety, and stress. It has been estimated that about 10% of all health care costs are used for neurocognitive disorders. Costs are expected to increase in the future. However, the actual prevalence of neurocognitive disorders has decreased 44% from the 1980s to the 2010s in one large-scale U.S. study (Satizabal et al., 2016). In the United Kingdom, a 20% drop in these disorders was seen since the 1990s (Matthews et al., 2016). This suggests that neurocognitive disorders may be reduced over time. Although both studies only looked at prevalence, the U.S. study also showed a reduction in cardiovascular disorders suggesting that exercise, diet, or health habits could help to explain these results.

There is also a complex relationship between cognitive changes in older adults and mental illness (O'Hara, 2012). One clear example is depression in older adults. With depression in this age group, memory and executive function are generally impaired. Similar findings are seen in anxiety in older populations. They show problems in cognitive performance, the ability to divide their attention, and memory recall. In one study looking at women 85 years of age and older, the researchers measured depressive symptoms and performed a battery of neurocognitive tests. The participants then retook the tests 5 years later. The researchers found that depressive symptoms were associated with cognitive impairment over that time (Spira, Rebok, Stone, Kramer, & Yaffe, 2012). In fact, these individuals had 3 times the risk of developing mild neurocognitive impairment. Other studies have shown an increased

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**TABLE 15.2 DSM-5 Diagnostic Criteria for Major Neurocognitive Disorder**

A. Evidence of significant cognitive decline from a previous level of performance in one or more of the cognitive domains (complex attention, executive function, learning and memory, language, perceptual-motor, or social cognition) based on:
   1. Concerns of the individual, a knowledgeable informant, or the clinician that there has been a substantial decline in cognitive function; and
   2. A substantial impairment in cognitive performance, preferably documented by standardized neuropsychological testing or, in its absence, another quantified clinical assessment.

B. The cognitive deficits interfere with independence in everyday activities (i.e., at a minimum, requiring assistance with complex instrumental activities of daily living such as paying bills or managing medications).

C. The cognitive deficits do not occur exclusively in the context of a delirium.

D. The cognitive deficits are not better explained by another mental disorder (e.g., major depressive disorder, schizophrenia).

prevalence of symptoms of mental illness in those with a mild neurocognitive disorder compared with similar-age adults with normal cognitive processes (Teng, Tassniyom, & Lu, 2012). These symptoms of mental illness actually decreased the person's quality of life more than decreases in their cognitive abilities did.

**CONCEPT CHECK**
- What are the characteristics of each of the following in terms of triggering event, symptoms, treatment, and prevalence?
  - Delirium
  - Mild neurocognitive disorder
  - Major neurocognitive disorder

**Neurocognitive Disorder Due to Alzheimer's Disease**

Neurocognitive disorder due to Alzheimer's disease is a progressive disorder characterized by problems with memory (Selkoe, Mandelkow, & Holtzman, 2012). It is associated with a loss of neurons and disruption of cortical networks, which result in cognitive problems.

**CHARACTERISTICS, PREVALENCE, AND DIAGNOSIS OF ALZHEIMER'S DISEASE**

Initial memory problems may include forgetting names, misplacing household items, and forgetting the task one was about to undertake. As the disorder progresses, the person has more problems with finding words and may not be able to follow a familiar path from one location to another. The individual may also not undertake new tasks and may withdraw socially. The health care professional may also see the person looking to his family for answers to personal questions that should be part of his own personal knowledge. In the later stages of the disorder, motor problems become apparent. This includes urinary incontinence. The person may also spend time in bed without acknowledging other people, including family, or speaking to those around him. Delusions and hallucinations are seen in a subset of individuals. On average, the full course of the disorder encompasses 10 to 20 years. Currently, research is focusing on procedures that postpone or reduce the symptoms of Alzheimer's disease (Reiman et al., 2016). The artist William Utermohlen drew self-portraits as his Alzheimer's disease developed. Look over the series, which provides a vivid illustration of the artist's gradual decline due to the disease.

As noted in President Reagan's letter to the American public, reprinted at the beginning of this chapter, it is the family who experiences the greatest toll from the disorder, as the person with Alzheimer's disease loses his memories and sense of self. At one point, his wife Nancy said, “Ronnie's long journey has finally taken him to a distant place where I can no longer reach him” (Spitz, 2018). It is also hard for the children who had a relationship with their parent to lose this connection as well as watch it slowly disappear.

Alzheimer's disease is the most common neurodegenerative disorder in the world, and its prevalence is...
fairly similar worldwide (Hebert, Weuve, Scherr, & Evans, 2013; Prince et al., 2013). It was estimated that the prevalence of Alzheimer’s in 2015 climed from 4% in those 65 years or younger to 15% in those 65 to 74 years of age to 43% in those 75 to 84 years of age. After 85, the prevalence drops to 38%. It affects women about 3 times as often as men. Overall, it was estimated that more than 5.3 million people in the United States had Alzheimer’s disease in 2015, where Alzheimer’s ranks sixth overall as the cause of death. At present, it is the only disorder in the top 10 causes of death that cannot be prevented, cured, or even slowed in its progression.

The disorder was first described by Alois Alzheimer in his report of a 51-year-old woman who displayed progressive memory loss and disorientation (Alzheimer, 1907; see Alzheimer, Stelzmann, Schnitzlein, & Murtagh, 1995, for English translation). Alzheimer had followed this woman for a number of years. After she died, Alzheimer examined her brain and identified two major factors that came to be seen as hallmarks of Alzheimer’s disease. These are neurofibrillary tangles and neuritic plaques (see Figure 15.13).

![Figure 15.13](image)

**FIGURE 15.13 What Are the Brain Changes With Alzheimer’s Disease?**

These include neurofibrillary tangles and neuritic plaques.

Source: A.D.A.M., Inc.

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**NEUROFIBRILLARY TANGLES AND NEURITIC PLAQUES**

The neurofibrillary tangles are found within the neuron, whereas the neuritic plaques are extracellular deposits. Today, we know that β-amyloid (beta amyloid) is the core protein involved in extracellular amyloid plaques and that tau is the core protein of intracellular neurofibrillary tangles (Edwards, 2019; Iaccarino et al., 2018; Iqbal, Liu, & Gong, 2016; Y. Wang & Mandelkow, 2016). Using brain imaging techniques such as PET, β-amyloid and tau buildup can be seen in individuals with Alzheimer’s disease (see Figure 15.14).

Alzheimer’s disease is also associated with widespread synaptic and neuronal loss (Nath et al., 2012). During the progression of the disorder, the development of tangles follows a fixed pattern. It begins in an area in the temporal lobe and serves as a hub that connects the hippocampus and the neocortex, the entorhinal cortex. This then progresses to the hippocampus, which is associated with memory, and then to other cortical areas along anatomical connections. The development of neuritic plaques does not follow a fixed pattern. The development...
of tangles is better correlated with cognitive decline than is the development of plaques. Presently, Alzheimer’s disease can only be diagnosed with certainty from brain studies after death, although a number of imaging studies are suggesting alternatives to diagnosis for those who are living. See Table 15.3 for the DSM–5 diagnostic criteria for Alzheimer’s disease.

**GENES AND ALZHEIMER’S DISEASE**

Family studies of those with Alzheimer’s disease suggest that first-degree relatives (parents, siblings, and children) are at a greater risk for developing Alzheimer’s.

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**TABLE 15.3 DSM–5 Diagnostic Criteria for 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 from 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:

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, mental, 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.

Source: Reprinted with permission from the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (Copyright 2013). American Psychiatric Association. All Rights Reserved.
In fact, they have about 3 times the risk of developing the disorder. The photo of the DeMoe family from North Dakota shows a family in which all but one of the individuals pictured have a genetic makeup associated with early onset Alzheimer’s.

Genetic studies also distinguish between those who show signs of the disorder before age 65 (early onset) and those who show signs of the disorder after age 65 (Rademakers & Rovelet-Lecrux, 2009). Individuals with early onset Alzheimer’s show a stronger familial risk than the others. Early onset is associated with mutations in genes involved in encoding amyloid, called APP and β-amyloid (beta amyloid) processing, especially the genes PSEN1 and PSEN2. That is, the presence of the β-amyloid protein is the primary component in the development of plaques (van Norden et al., 2012).

Late onset Alzheimer’s has been consistently associated with an allele of the APOE gene. Individuals with one APOE4 allele have an increased risk that is about 2 to 3 times higher than individuals without it. If an individual has two APOE4 alleles, the risk increases to between 7 and 15 times. By contrast, the APOE2 gene is associated with a longer life and not having Alzheimer’s disease. The gene APOE is involved with removing β-amyloid. One current theory of Alzheimer’s disease is that the substances involved in the development and removal of plaques are not functioning correctly, allowing for their buildup. Current drug treatments for Alzheimer’s disease seek to lower the production of β-amyloid. It has also been shown that those who engage in cognitively stimulating activities in their early and middle life show fewer problems associated with β-amyloid (Landau et al., 2012).

One surprising finding is that healthy older adults who do not show cognitive problems may also have abundant plaques. In addition, plaques and tangles are found in individuals who do not show the loss of neurons seen in Alzheimer’s disease. Thus, the presence of plaques and tangles is not related to dementia in all cases. Cognitive reserve and the brain’s ability to reorganize networks to compensate may play a role in these individuals. At this point, the role of plaques and tangles in Alzheimer’s disease is still being worked out, although all individuals with Alzheimer’s disease show tangles and plaques and a loss of neurons (van Norden et al., 2012).

**NEUROIMAGING OF ALZHEIMER’ S DISEASE**

There would be an advantage to knowing who will develop Alzheimer’s disease in the future (Koch et al., 2012; Mevel, Chételat, Eustache, & Desgranges, 2011). One promising approach is to study brain metabolism. In one study, it was found that those areas that correspond to the default network in young adults corresponded to those areas with amyloid deposits in elderly individuals with Alzheimer’s (see Figure 15.15). In addition, the connection between the posterior cingulate and the hippocampus appears to be impaired in Alzheimer’s disease. Individuals with Alzheimer’s show less activation of the default network than healthy elderly controls. It is assumed that the deposit of amyloid plaques in these areas is related to lower glucose
metabolism as well as atrophy. Thus, changes in the default network could be an important biomarker of those at risk for Alzheimer’s disease.

**CONCEPT CHECK**

- What are the primary changes in the brain related to Alzheimer’s disease?
- What is the characteristic progression of symptoms in Alzheimer’s disease?
- What is the genetic risk for Alzheimer’s disease?

**Other Neurocognitive Disorders**

In addition to Alzheimer’s disease, there are other types of neurocognitive disorders. This section will briefly note these other disorders and describe their characteristics.

**VASCULAR NEUROCOGNITIVE DISORDER**

Vascular problems such as strokes can lead to a neurocognitive disorder. It can be one large stroke or a series of smaller ones. Approximately 8% of individuals who have a stroke go on to develop a neurocognitive disorder. Vascular neurocognitive disorder is the second most frequent cause of neurocognitive problems after Alzheimer’s disease. Problems in cognitive performance are usually seen as abrupt changes following the stroke. These abrupt changes are one characteristic that differentiates vascular neurocognitive disorder from Alzheimer’s disease, although the symptoms may appear similar.

In the late 1800s, the disorder was referred to as arteriosclerotic dementia (Erkinjuntti, 2005). With increased research related to blood flow in the brain, it became apparent that a number of factors influence how blood is delivered to the brain and converted for cognitive and motor processes. Without blood flow, there is a lack of oxygen, which can lead to brain damage. The term vascular neurocognitive disorder reflects the role of blood flow in cognitive performance.

**FRONTOTEMPORAL NEUROCOGNITIVE DISORDER**

Frontotemporal neurocognitive disorder was originally known as Pick’s disease, since Arthur Pick, a professor of psychiatry in Prague, first described brain changes in those with this disorder in 1892. The disorder is characterized by a reduction of the anterior lobes of the frontal and temporal areas. The parietal and occipital lobes do not show this reduction. Prevalence rates are about 2 to 3 per 100,000 people (Neary, 2005). This disorder may be seen as early as the third decade of life.

Frontotemporal neurocognitive disorder is seen in different variants depending on the brain areas involved. Anne Adams, who will be introduced in the next section, had a variant that shows a loss of language abilities. Another variant gives a pattern of impairment not unlike that seen in Phineas Gage, the railroad worker who received a rod through his brain (see Chapter 3). It is not so much cognitive impairment that is seen, but rather, behavioral and personality changes that come to the forefront. These include a lack of social awareness, a lack of insight, indifference, inappropriate behaviors, stereotyped behaviors, aggression, and a loss of inhibition. Some of these characteristics may be manifested in terms of eating. That is, the person will eat indiscriminately and may even take food from others’ plates. The person may also show repetitive motor behaviors such as hand rubbing or foot tapping. He or she may repeat the same phrase or do the same activity at the same time each day. Visuospatial skills tend not to be impaired, as you will see in the extended case study of Anne Adams (her real name).

**THE DEVELOPMENT OF FRONTOTEMPORAL NEUROCOGNITIVE DISORDER IN A SCIENTIST AND ARTIST**

Let’s look at an extended case study in which an individual with frontotemporal neurocognitive disorder shows deficits in cognitive abilities, especially language. However, as
she experienced these deficits, other areas of the brain became more active and increased her creativity. Dr. Anne Adams graduated from college with honors degrees in physics and chemistry. She taught college chemistry before taking time off to raise her children. After returning to work, she received a PhD in cell biology and taught for the next 4 years. At that point, her son was involved in a life-threatening motor vehicle accident. While taking care of her son, she went back to an old interest in art and began to paint. Surprisingly, her son recovered in 7 weeks but rather than return to teaching, she continued to paint, and would spend all day in her studio.

Unknown to her, she was developing a neurocognitive disorder, which changed the relationship of networks in her brain. Around age 60, she began to have language difficulties and was evaluated at the University of California, San Francisco. William Seeley and his colleagues (2008) followed these changes in her brain processes and in her art and published their findings in the journal Brain. Using structural and functional imaging, these researchers were able to show that as Anne Adams lost brain processing in the frontal and temporal areas, she was able to create enhanced connections in the right posterior areas including the right parietal, which is involved in spatial relationships as would be necessary for creativity in art. One intriguing scientific question relates to the manner in which loss in one area may increase abilities in another. In this case, the loss of spoken language was associated with an increase in creativity and artistic abilities. Fortunately, before her difficulty with spoken language, Anne Adams kept extensive notes on the nature of her paintings, which detailed the manner in which she related her paintings to music and mathematics.

In a strange coincidence, Anne Adams became interested in the French composer Maurice Ravel (1875–1937), best known for his work Boléro, a piece of music that is highly structured. The strange coincidence is that Ravel at the time in his life that he was writing Boléro may have begun to develop the same neurocognitive disorder that was to be manifested in Anne Adams a century later. At this point, at age 53, she turned the music of Boléro into art in the form of a visual analysis of the piece (see Figure 15.16). Each of the vertical panels represents a specific bar in the music. This was some 7 years before any of her symptoms appeared. At age 58, she moved from painting music to more abstract patterns such as those found in mathematics. One of Anne Adams’s paintings with this theme is called Pi (see Figure 15.17). This was 2 years before her symptoms appeared. There is an episode of the podcast Radiolab describing her case and Ravel (https://www.wnycstudios.org/story/unraveling-bolero).

Anne Adams’s symptoms began to appear around age 60 with difficulties in speech. However, her comprehension remained intact. By age 64, she was nearly mute, able to speak only 3- or 4-word phrases at best. With the development of her symptoms, she also shifted her painting style to one of realism with high surface fidelity. Her paintings at this time were very symmetrical.

During the next 4 years, her paintings emphasized a certain type of realism (see Figure 15.19), including surfaces of buildings (see Figure 15.20). Figure 15.21 shows the loss of brain areas in Anne Adams relative to the painting she was producing at the time.

The case of Dr. Anne Adams portrays a person with frontotemporal neurocognitive disorder who shows deterioration in language abilities over time. However, as she was losing language abilities, she began to display greater creativity according to those who worked with her. Thus, she showed compensation and increased reliance on other areas of the brain related to creativity. Unlike the artist William Utermohlen, for whom you could see the deterioration of his artistic abilities in his painting due to Alzheimer’s disease, Anne Adams showed shifts in the types of images that she focused on and the levels of detail that she emphasized (Seeley et al., 2008). As she was losing her speech, she emphasized a spatial language in her paintings (see Figure 15.18).
Figure 15.16  Unraveling Boléro.  
Painting by Anne Adams.  
Source: Courtesy of Robert A. Adams.

Figure 15.17  Pi.  
Painting by Anne Adams.  
Source: Courtesy of Robert A. Adams.

Figure 15.18  Examples From Anne Adams’s ABC Book of Invertebrates  
Source: Courtesy of Robert A. Adams.

Figure 15.19  Arbutus Leaves.  
Painting by Anne Adams  
Source: Courtesy of Robert A. Adams.

Figure 15.20  Amsterdam.  
Painting by Anne Adams  
Source: Courtesy of Robert A. Adams.

Figure 15.21  Magnetic Resonance Imaging With Paintings  
Source: Seeley et al. (2008, p. 44), by permission of Oxford University Press.
NEUROCOGNITIVE DISORDER DUE TO TRAUMATIC BRAIN INJURY

Traumatic brain injuries (TBIs) are seen across the life span (Silver, McAllister, & Yudofsky, 2011). Acceleration and deceleration forces on the brain as it impacts with the skull commonly lead to injuries, which often produce diffuse microstructural injury. The severity of these injuries can range from mild to severe. Mild TBIs are often referred to as concussions. Common sources of TBIs are sports, transportation accidents, and falls by the elderly. Some are one-time-only events and others are repeated, such as concussions in contact sports. Boxers show a syndrome referred to as “punch drunk.” This is seen in aging boxers and characterized by slowed thought as well as changes in emotional processing. The National Football League (NFL) and college football associations in the United States have recently begun a number of studies to determine the long-term effects of concussions on the players. The U.S. military has also noted an increase in TBIs, at times along with post-traumatic stress disorder (PTSD), in soldiers involved in the conflicts in the Middle East. Worldwide, TBIs are a critical public health problem that can lead to a variety of neurocognitive and psychological problems. For example, one meta-analysis involving over 700,000 individuals suggests that concussion and mild TBIs are associated with a higher risk of suicide, suicide attempts, and suicidal ideation (Brenner & Bahraini, 2018; Fralick et al., 2019).

These problems can include loss of consciousness, cognitive deficits, depression, and—at a later period—the onset of neurocognitive disorder. For example, chronic traumatic encephalopathy (CTE), which is a degenerative brain disease, has been found in athletes, soldiers, and others with a history of brain trauma. The types of deficits seen in individuals with TBI vary in terms of the areas of the brain affected by the injury. It is estimated by the Centers for Disease Control and Prevention (CDC) that in the United States, some 1.7 million occurrences of TBI happen each year, with about 2% of the population having TBI-related disabilities (Faul, Xu, Wald, & Coronado, 2010; https://www.cdc.gov/traumaticbraininjury/). LENS: The Silent Epidemic of Concussion in Sports describes what has been called a silent epidemic: the presence of concussion in amateur and professional sports.

LENS

The Silent Epidemic of Concussion in Sports

For years, few people paid attention to the potential dangers of hard-hitting encounters that occur frequently in contact sports. One of the most common injuries—concussion—has been referred to as a “silent epidemic.” It was once assumed that there were no long-term consequences of concussion. However, this has recently changed as more individuals have come forward to discuss the effects of having played such sports as football years ago. Ted Johnson, a former New England Patriots linebacker, had multiple concussions that resulted in significant memory and emotional problems throughout his thirties. Previously, athletes just “played through the pain.” The former Denver Broncos and Washington Redskins

(Continued)
Abnormal Psychology

running back Clinton Portis described his experiences as follows:

The truth is I had a lot of concussions... It was just the way things were at the time. I’d get hit hard and be woozy. I’d be dizzy. I’d take a play off and then go back in. Sometimes when I went back into the game, I still couldn’t see straight. This happened all the time. Sometimes once or twice a game. (Davenport, 2013)

It is now known that a person is most susceptible to another concussion for about 10 days after the first. Research has suggested that older, retired professional football players experience neurocognitive disorders at 5 times the national rate. It is becoming more apparent that repetitive blows to the head affect the brain in negative ways, which can lead to neurocognitive disorders. Often, it is not contact with another person but a person’s head hitting the ground that produces the concussion. This is not limited to men; women who play such sports as field hockey and soccer are also at risk. The symptoms of concussion in men and women are very similar. Even a fall from a bicycle can result in a concussion.

In response to a new recognition of the effects of concussion, a number of groups have changed their approach. The NFL now supports studies of the long-term effects of concussion in professional athletes. A number of older athletes such as the Hall of Fame professional quarterback Ken Stabler, who played for the Oakland Raiders, developed chronic traumatic encephalopathy, or CTE, a degenerative brain disease believed to be caused by repeated blows to the head (Montenigro, Corp, Stein, Cantu, & Stern, 2015).

Many universities have established centers for the study of concussion, and all U.S. states have some form of legislation related to concussion assessment and management in high school athletics as well as return-to-play guidelines. High school athletes are particularly at risk, since surveys suggest that this group believes there is not a problem playing sports with a concussion. Returning to play before the concussion has been fully resolved can increase long-term injuries.

Since adolescence is a time in which an individual’s brain goes through a series of cortical reorganizations, brain insults at this time put the adolescent at greater risk for serious injury. Further, as risk takers, adolescent athletes may even deny there is a problem so they can continue to play. For college and professional athletes, different pressures may cause them to ignore information concerning the effects of concussion. Overall, this can lead to a lack of candor when athletes at all levels describe their symptoms.

The CDC now offers a number of programs directed at athletes at all levels for the care and prevention of concussion and also works with the National Collegiate Athletic Association (NCAA) (www.cdc.gov/headsup/index.html). Professional football now has individuals in the press box who look for potential concussions on the field and immediately report this to team physicians. Many college programs engage in intensive baseline neuropsychological and neuroscience testing, which can be compared with a person’s performance after a concussion to help determine when the person should return to play. There is also a best practices guideline for athletic trainers (Broglio et al., 2014). It is no longer the case that a concussion should be seen as unimportant.

Thought Question: Since young people are at particular risk for serious injury from concussions while playing sports, what recommendations would you offer to make it safer?


NEUROCOGNITIVE DISORDER DUE TO LEWY BODY DEMENTIA

Lewy bodies are substances found in the neuron (see Figure 15.22). When these Lewy bodies build up, symptoms of a neurocognitive disorder, called Lewy body dementia, become apparent. The symptoms include changes in alertness and attention, which can result in drowsiness or staring into space. Other symptoms include visual hallucinations and Parkinson’s-like symptoms, which start a year after the cognitive impairment begins. The prevalence of this disorder is estimated to be less than 5% of the elderly population...
(APA, 2013). When autopsies have been conducted, Lewy bodies are seen in 20% to 35% of those with a neurocognitive disorder. The comedian Robin Williams is known to have had Lewy body dementia at the end of his life. His condition was described by his wife in an editorial in the journal Neurology (S. Williams, 2016).

**NEUROCOGNITIVE DISORDER DUE TO PARKINSON’S DISEASE**

Parkinson’s disease is a neurological condition that affects the motor system. The symptoms generally include tremors, which may involve the hands, arms, legs, jaw, and face. In addition, the person shows a slowness of movement and stiffness in his limbs. Other motor problems such as poor balance and coordination may be present. In addition, there may be problems with sleep patterns. As the disorder progresses, the ability to walk may be lost and a wheelchair is required. Parkinson’s disease typically begins after the age of 60 and affects about 1% of people over age 60 and 3% over age 85. It affects men more often than women.

In Parkinson's disease, damaged nerve cells are found in the part of the brain stem called the substantia nigra. There are two types of problems seen in this area. The first is a loss of neurons that create dopamine. Parkinson’s disease does not become apparent until 60% of the dopamine neurons in the substantia nigra are lost or dopamine levels in the basal ganglia fall by 80%. Treatment of Parkinson's disease typically involves drugs that replace the lost dopamine. The second type of problem is the presence of Lewy bodies in the substantia nigra and locus coeruleus in the brain stem.

About a third of the individuals with Parkinson’s disease continue to develop a neurocognitive disorder. Neurocognitive symptoms are generally not seen in the early stage of Parkinson’s, but are found in about 40% of 70-year-olds. The cognitive characteristics are similar to those found in neurocognitive disorder due to Lewy body dementia. These include inflexibility and problems with executive functions. It is suggested that the progression of the neurocognitive disorder is related to the migration of the Lewy bodies from the motor areas to the cortex. Hallucinations may also be seen, but this may be related to the increase of dopamine in the brain from medications. These hallucinations tend to be of a visual rather than auditory nature.

**NEUROCOGNITIVE DISORDER DUE TO HIV INFECTION**

The human immunodeficiency virus (HIV) can be passed on by coming in contact with the bodily fluids of an infected person. Common means of contact include unprotected sex, sharing a needle for drug use, or tainted blood such as from a transfusion. It can also be seen in infants born to infected women. The virus affects the person's immune system in a negative manner. In the later stages, this is referred to as acquired immune deficiency syndrome (AIDS). About a third of the individuals with AIDS will also show neurocognitive problems such as slowing in both cognitive and motor functions. These include memory problems, confusion, depression, and difficulty with fine motor tasks. Prior to the development of effective AIDS drugs, many individuals died of disorders related to a compromised immune system from HIV. Today, drug treatments have successfully increased the life span and reduced neurocognitive problems of those with HIV/AIDS.
SUBSTANCE-INDUCED NEUROCOGNITIVE DISORDER
The abuse of drugs over a period of time can lead to neurocognitive deficits, known as substance-induced neurocognitive disorder. These drugs can include illegal substances as well as medications. Toxins such as lead, mercury, and carbon monoxide are also included as potential causes of neurocognitive problems. One common substance that can lead to cognitive changes is alcohol. This is especially the case when combined with poor nutrition. There is also some suggestion that the toxic effects of alcohol make the person more susceptible to the negative effects of a head injury. Alcohol is seen as the third leading cause of neurocognitive disorder and affects more women than men.

NEUROCOGNITIVE DISORDER DUE TO HUNTINGTON’S DISEASE
Huntington’s disease is a genetic disorder that causes a degeneration of neurons in the brain (Ahveninen, Stout, Georgiou-Karistianis, Lorenzetti, & Glikmann-Johnston, 2018; Gusella & MacDonald, 2006). It is one of the few disorders that has a single cause—a gene on chromosome 4—in all people who are diagnosed with it. The gene that is associated with this disorder is dominant. Thus, a child of an individual with Huntington’s disease has a 50–50 chance of inheriting the gene that produces the disorder. Typically, this disorder does not become apparent until around age 40, which is after the primary childbearing years. The loss of brain cells results in cognitive, emotional, and motor disturbances. The cognitive deficits include problems with executive function, memory, arithmetic, and spatial ability. There are fewer problems with language functions. Emotional disturbances include mood swings and depression. Motor problems include both voluntary movements in which the person may appear clumsy and involuntary movements such as jerking of the body.

Prions are infectious pathogens that are different in structure—and the diseases they cause are different—from other pathogens such as bacteria, fungi, parasites, and viruses (L. C. Walker & Jucker, 2015). They produce tiny holes in the brain, which give it a spongy appearance and result in neurocognitive disorder due to prion disease. Prion proteins occur in their natural form in all brains and are harmless. The general public became aware of prions with the advent of “mad cow disease,” also called bovine spongiform encephalopathy, which spread through the food chain to humans in the United Kingdom in the 1990s. Prion disease is not infectious in the usual sense of the word but is spread through eating the brain tissue of a diseased organism. A similar disorder was found in an isolated tribe in Papua New Guinea whose members ate brains as a part of funeral rites.

A related disease that also causes brain degeneration is Creutzfeldt-Jakob disease, a disorder described by German neuroscientists Creutzfeldt and Jakob in the 1920s. This disorder can also have a genetic component that is estimated to be related to 10% of the occurrences of the disorder. Overall, these disorders affect about 1 in a million people. Creutzfeldt-Jakob disease typically occurs in individuals over 60 with about 90% dying within a year. During this time, there are cognitive impairments including memory loss, motor problems, personality changes, and impaired judgment. Although the cognitive problems may be similar to other neurocognitive disorders, it has a more rapid course of development and can be distinguished on autopsy by the sponge-like changes in the brain.

CONCEPT CHECK

• What are the defining characteristics of each of these neurocognitive disorders, including triggering event, time of onset, symptoms, and treatment?
  o Vascular neurocognitive disorder
  o Frontotemporal neurocognitive disorder
  o Neurocognitive disorder due to TBI
CHAPTER 15  Neurocognitive Disorders

- Neurocognitive disorder due to Lewy body dementia
- Neurocognitive disorder due to Parkinson’s disease
- Neurocognitive disorder due to HIV infection
- Substance-induced neurocognitive disorder
- Neurocognitive disorder due to Huntington’s disease
- Neurocognitive disorder due to prion disease

Prevention, Treatment, and Support

As our life span has increased, with many of us living into our eighties, nineties, and even beyond, it is becoming clear that not all older individuals develop neurocognitive disorders. This has led scientists to search for the characteristics of those who do and do not develop neurocognitive disorders. This research has helped us to better understand the environmental factors and genetic predispositions that are involved. This section will describe both prevention and treatment approaches to neurocognitive disorders. There will also be examples of support networks that have been developed to help those with the disorder live successfully in their communities.

Prevention of Neurocognitive Disorders

Although many people assume that these disorders are a normal part of old age, it is suggested that perhaps 50% of neurocognitive disorders could be prevented (Brayne, 2007). Some of the prevention factors are related to lifestyle. Such life changes as exercising, eating better, and not using tobacco have been shown to be related to a reduction in both physical and mental health problems.

Cognitive challenges and social relations have also been shown to help reduce cognitive decline (Ballesteros et al., 2015). One large-scale cognitive training program (the Advanced Cognitive Training for Independent and Vital Elderly [ACTIVE]) was directed at 2,832 individuals age 65 and older who lived independently (Rebok et al., 2014). These individuals received 10 training sessions for memory, reasoning, or speed of processing. Cognitive measures and self-reported daily abilities showed beneficial effects of the training, especially during the period 3–5 years after training. In comparison to control individuals, those who received training showed benefits after 10 years in reasoning and processing speed but not memory. These results suggest that cognitive training can delay the onset of cognitive decline.

Other prevention factors can be found through medical checkups. For example, studies that have examined blood pressure readings in childhood, midlife, and later life have shown that those with a higher blood pressure when young will have higher pressure throughout their life. High blood pressure at an earlier age, in turn, puts one at greater risk for neurocognitive disorders through strokes.

Can an Individual’s Activities Be Protective in Brain Changes?

The answer to this question is yes. It was first noticed that not all individuals showed the same changes from similar neurocognitive disorders or brain injury. From this observation, the concept of reserve was developed. That is, high-functioning individuals tend to show less loss of cognitive abilities in relation to neurocognitive disorders. The concept of reserve suggests that the brain can compensate for problems in neural functioning. This is
illustrated by the case in which the brains of older individuals expand their networks to solve problems as shown earlier in Figure 15.10. High functioning or intelligence is often associated with greater reserve.

Additional research has shown a role for exercise and social support. Exercise is thought to play an important role in aging by promoting healthy cardiovascular function. That is, exercise increases blood flow to the entire brain. Exercise has also been shown to slow the expression of Alzheimer’s-like disorders in a mouse model. In a review of literature from different areas, Kramer and Erickson (2007) suggested that exercise provides multiple routes to enhancing cognitive vitality across the life span. These include the reduction of disease risk as well as improvement in molecular and cellular structures of the brain. This, in turn, increases brain function. Further, aerobic exercise has been demonstrated to affect executive function more than other cognitive processes. Exercise has also been associated with lowering the risk for future Parkinson’s disease (Xu et al., 2010).

Following more than 700 older individuals without neurocognitive disorders for several years, Aron Buchman and his colleagues (2012) found that daily physical activity slowed cognitive decline. Exercise was also associated with a lower risk for developing Alzheimer’s disease. Although performing various types of cognitive tasks such as crossword puzzles or speaking a second language are also associated with successful aging, these brain effects appear to be more localized in those areas of the brain related to the specific task.

In order to better articulate the causal role of exercise, Lindsay Nagamatsu and her colleagues (2013) randomly assigned older individuals who were beginning to show mild cognitive impairment to one of three groups. The first group received resistance training and lifted weights. The second group received aerobic training and walked outdoors at levels that increased their heart rate. The third group received balance and stretching exercises. The third group served as the control group. After 6 months of twice-weekly exercise, the first two groups showed improvement in memory functions. This was seen more strongly on a difficult spatial memory test. The aerobic group also improved performance on the verbal memory test. The important point of this study is that 6 months of exercise can improve memory in 70-year-olds.

Social support has also been associated with a reduced risk for neurocognitive disorders and better physiological functioning (Yang et al., 2016). Two of these factors are the size of one’s network of friends and whether one is married or not. As suggested in studies of the social brain, understanding and maintaining social networks of friends require a variety of cognitive resources, which in turn offer a reserve for dealing with brain pathologies. One study followed 16,638 individuals over the age of 50 for 6 years. Those individuals who were more socially integrated and active showed less memory loss during the 6-year period (Ertel, Glymour, & Berkman, 2008).

One comprehensive study of aging is The 90+ Study (http://www.mind.uci.edu/research/90plus-study/). Initial members of this study started as part of another study of aging, which began in 1981 in Orange County, California. The 90+ Study was begun in 2003 to study the oldest of the old. By studying 14,000 individuals of the original study, the researchers were able to ask, what allows people to live to age 90 and beyond? Participants in The 90+ Study are visited by researchers every 6 months. A comprehensive battery of information including diet, activities, medical history, and medications is collected. In addition, neurological, cognitive, and neuropsychological tests are administered.

Researchers from The 90+ Study have published many scientific papers (e.g., Bilousova et al., 2016; Kawas, 2008; Kawas et al., 2015; J. L. Robinson et al., 2018). Some of the major findings are as follows: People who drank moderate amounts of alcohol or coffee lived longer than those who abstained. People who were overweight in their seventies lived longer than normal or underweight people did. Over 40% of people aged 90 and older suffer from dementia, while almost 80% are disabled. Both are more common in women than men.
About half of people with dementia over age 90 do not have sufficient neuropathology in their brain to explain their cognitive loss. People aged 90 and older with an APOE2 gene are less likely to have clinical Alzheimer’s dementia, but are much more likely to have Alzheimer’s neuropathology in their brains.

**Treatment of and Support for Those With Neurocognitive Disorders**

In the same way that cognitive training has been used to delay the negative effects of aging, it has been used to help individuals with neurocognitive disorders recover cognitive functions that can be restored. Most of these training programs seek to help individuals maximize their strengths while bypassing their weaknesses. An example of such a training program is CogSmart (Cognitive Symptom Management and Rehabilitation Therapy), developed by Elizabeth Twamley at the University of California, San Diego (Twamley, Jak, Delis, Bondi, & Lohr, 2014). This is a manualized, empirically supported approach that helps the individual with a neurocognitive disorder recover cognitive functioning and compensate for cognitive difficulties. For example, in one’s daily life, the use of cell phones, calendars, and lists can be used to remember meetings, grocery products, and tasks to do. Likewise, when talking with others, eye contact, reducing distractions, and paraphrasing what was said can improve the accuracy of conversations. The approach also includes formal steps in solving problems and learning new information. An additional advantage of these types of approaches is that they have been shown to reduce the burden on caregivers of those with dementia (Germain et al., 2018).

In addition to psychological approaches, medications are used for the treatment of neurocognitive disorders (Gatchel, Wright, Falk, & Trinh, 2016). The neurocognitive disorders described in this chapter, except for delirium, cannot be cured. However, sometimes the symptoms can be reduced. There are also secondary symptoms such as anxiety and depression that can be treated separately. Further, at times, it is difficult to distinguish depression from dementia. Also, it should be noted that with aging, medications can influence the body differently from how they influence younger or middle-aged individuals. That is, medications can be absorbed and distributed in the body differently. Also, there can be more adverse side effects with older individuals.

Different medications are used for different neurocognitive disorders. In Alzheimer’s disease, drugs such as cholinesterase inhibitors are used to increase memory and other cognitive functions by increasing concentrations of ACh in the hippocampus. Although useful, the memory effects are modest. Since Parkinson’s disease is the result of dopamine neurons not functioning correctly, treatment of Parkinson’s disease typically involves drugs that replace the lost dopamine such as L-dopa. However, it should be noted that dopamine replacement drugs are not always effective. There are currently no medications directed at the other neurocognitive disorders such as frontotemporal neurocognitive disorder and Lewy body disease.

A different approach for Parkinson’s disease is deep brain stimulation (DBS), which was approved by the FDA in 2002. Deep brain stimulation for depression was described in Chapter 6. DBS involves placing an electrode in the areas of the brain related to movement, usually the basal ganglia. This in turn is connected to a pulse generator that is placed under the person’s skin, usually near the collarbone. The pulse generator can be programmed to stimulate the brain in a number of ways related to the individual. Although not a treatment that is recommended for all who have Parkinson’s disease, it has proven useful for improving movement in a number of individuals.

While the search continues to discover more effective treatments for those with neurocognitive disorders, LENS: Dementia-Friendly Communities describes a movement to make cities and towns more livable for those with dementia.
As people around the world live longer, there is more opportunity for an individual to develop a neurocognitive disorder. Alzheimer’s is the most common of these disorders. In 2015, it was estimated that the prevalence of Alzheimer’s was 4% in those 65 years or younger, 15% in those 65 to 74 years of age, and 43% of those 75 to 84 years of age. After 85 years of age, the prevalence drops to 38%. Often, a person with Alzheimer’s disease will require living in a nursing home or assisted living facility. However, a number of individuals with milder forms of dementia are still able to interact with those in their town or community. In fact, being able to shop for food, attend church, and be with others is important for the health of many of these individuals.

Many retirement communities have trained staff on how to interact with those who have cognitive difficulties. This type of training is now moving to the larger community. There is currently a movement in a number of states such as Minnesota to help communities become more dementia-friendly. The Minnesota plan began as part of a 2009 legislative mandate aimed at addressing the problems of aging. Some 30 communities in Minnesota are now implementing the plan. In Paynesville, Minnesota, a small town of 2,400 people, there are twice-monthly “Fridays and Friends” events for those with dementia and other townspeople designed for socializing. Volunteers also help those with cognitive difficulties with various basic tasks such as buying groceries at the local market (see Figure 15.23).

Other U.S. states and nations including Canada and the United Kingdom are also seeking to make communities more dementia-friendly. In Watertown, Wisconsin, there are programs to train businesses in the community to institute minor changes in their daily operations to make life easier for those with cognitive difficulties. For example, servers who work in coffee shops or restaurants are taught to ask yes-or-no questions rather than naming all of the specials or menu items for the day. Cashiers are taught to slow down the transactions if the person finds it difficult to count his or her money. Even more complicated transactions such as estate planning or money management can be presented in shorter, more understandable segments of a larger presentation. In some other towns, local shops have created special areas of their store where those with dementia can feel calm, comfortable, and in control. Building designers are also considering how features such as lighting, sound, and pathways can make an environment more user friendly.
Thought Question: Think about your hometown or school environment. What specific changes would you recommend to make it more dementia-friendly? How would those changes also help other segments of your local population?

**CONCEPT CHECK**

- It is suggested that perhaps 50% of neurocognitive disorders could be prevented. What are some specific examples of each of the following areas of prevention?
  - Cognitive challenges
  - Social relations
  - Exercise
  - Lifestyle
- Describe the concept of reserve. What role does it play in protecting individuals from neurocognitive disorders?
- What are some strategies used to help an individual with a neurocognitive disorder recover cognitive functionality or compensate for cognitive losses?
- Medications can’t cure neurocognitive disorders, but what are some of the ways they can be useful in treating some of them?
As we age, our mental and physical abilities change. Some of these changes happen gradually and appear to be related to genetic differences. Other changes happen more quickly. They may involve not only physical changes but also changes in mental processing, including neurocognitive disorders. This contrasts with what has come to be called normal successful aging. Perhaps 50% of neurocognitive disorders could be prevented through lifestyle changes and preventive medical care. In order to understand the changes seen in neurocognitive disorders, it is important to examine the normal changes in cognitive abilities across the life span, including (1) vocabulary, (2) perceptual speed, (3) episodic memory, (4) spatial visualization, and (5) reasoning. Two consistent findings are that older adults show changes in brain structure and that they use their brains in different ways from younger adults, using compensation to improve their performance. High functioning or intelligence, exercise, and social support are all associated with a greater reserve and overall brain health to provide for compensation.

There is a complex relationship between cognitive changes in older adults and mental illness. Studies have shown that symptoms of mental illness actually decreased the person’s quality of life more than did decreases in their cognitive abilities. Delirium is a short-term condition characterized by a change in cognitive processing and can be caused by a number of underlying physiological disturbances. When cognitive or social deficits are greater than those seen with normal aging, this decline can be described in terms of a mild neurocognitive disorder. If the declines are severe and interfere with one’s ability to function independently, then these can be described in terms of major neurocognitive disorders. DSM–5 defines a variety of neurocognitive disorders.

Neurocognitive disorder due to Alzheimer’s disease is a progressive disorder characterized by problems with memory. It is associated with a loss of neurons and disruption of cortical networks, which result in cognitive problems. Alzheimer’s disease is the most common neurodegenerative disorder in the world, and its prevalence is fairly similar worldwide. Neurofibrillary tangles and neuritic plaques as well as widespread synaptic and neuronal loss in the brain are the hallmarks of Alzheimer’s disease. There is a genetic risk for developing Alzheimer’s. Changes in the default network, as shown in neuroimaging studies, could prove to be an important biomarker or predictor of those at risk for Alzheimer’s disease.

Vascular neurocognitive disorder can result from vascular problems such as strokes—either one large stroke or a series of smaller ones. Frontotemporal neurocognitive disorder has different variants depending on the brain areas involved. It can present as cognitive impairment or behavioral and personality changes. Neurocognitive disorder due to traumatic brain injury (TBI) occurs across the life span as a result of a one-time-only traumatic event or a series of smaller injuries. It results in slowed thought as well as changes in emotional processing.

Neurocognitive disorder due to Lewy body dementia results in a buildup of Lewy bodies in the neuron. Symptoms include changes in alertness and attention. Parkinson’s disease is a condition that affects the motor system. About a third of the individuals with Parkinson’s disease continue to develop a neurocognitive disorder. The cognitive characteristics are similar to those found in the neurocognitive disorder due to Lewy body dementia. About a third of individuals with AIDS will also show neurocognitive problems, referred to as neurocognitive disorder due to HIV infection, such as slowing in both cognitive and motor functions. The abuse of drugs or alcohol over a period of time can lead to neurocognitive deficits, referred to as substance-induced neurocognitive disorder.

Huntington’s disease is a genetic disorder that causes a degeneration of neurons in the brain, referred to as neurocognitive disorder due to Huntington’s disease, resulting in cognitive, emotional, and motor disturbances. It is one of the few disorders that has a single cause—a gene—in all people who have it. Prions are infectious pathogens that produce tiny holes in the brain, which give it a spongy appearance and result in neurocognitive disorder due to prion disease. A related disease that also causes brain degeneration is Creutzfeldt-Jakob disease, which can have a genetic component. These two diseases have a rapid course of development, and cognitive impairments include memory loss, motor problems, personality changes, and impaired judgment.

The treatment approaches for neurocognitive disorders are basically designed to slow the progression of the disorder and to offer the person support to live with the disorder. The neurocognitive disorders described in this chapter, except for delirium, cannot be cured. At times, the symptoms can be reduced using medication such as drugs that attempt to increase dopamine in those with Parkinson’s disease or memory abilities in other dementias. Deep brain stimulation has also been used to reduce the tremors in Parkinson’s disease. Psychological disorders such as anxiety and depression have been seen with dementia, and these have been treated using traditional approaches. There is also a worldwide movement to make communities more dementia-friendly.
CHAPTER 15  Neurocognitive Disorders

STUDY RESOURCES

REVIEW QUESTIONS

1. This chapter on neurocognitive disorders devotes a considerable amount of space to describing the process of aging and the diseases experienced (typically) by older adults. Does that mean it has no personal meaning for you or your friends—at least for a few more years? Present an argument that this information is vitally important for young people to know. What evidence would you cite to support your position and convince them that they should care?

2. Alzheimer’s disease is the most common neurodegenerative disorder in the world, and currently, it is the only disorder in the top 10 causes of death that cannot be prevented, cured, or even slowed in its progression. What would be your focus of research in each of these three important areas: prevention, cure, and slowing progression? Which one would you start with and why?

3. “One intriguing question relates to the manner in which loss in one area may increase abilities in another.” This question arises in relation to the case of Anne Adams, whose creativity and artistic abilities increased at the same time she was developing frontotemporal neurocognitive disorder. Using this example, as well as the concepts of brain reserve and compensation presented at the beginning of the chapter, what can we say about the plasticity of the brain, that is, the brain’s ability to reorganize and repurpose itself to be able to function?

FOR FURTHER READING


KEY TERMS AND CONCEPTS

acquired immune deficiency syndrome (AIDS) 577
delirium 564
dementias 557
frontotemporal neurocognitive disorder 572
human immunodeficiency virus (HIV) 577
Huntington’s disease 578

Lewy body dementia 576
major neurocognitive disorder 566
mild neurocognitive disorder 566
neuritic plaques 569
neurocognitive disorder due to Alzheimer’s disease 568
neurocognitive disorder due to prion disease 578
neurocognitive disorders 557
neurofibrillary tangles 569
Parkinson’s disease 577
reserve 579
substance-induced neurocognitive disorder 578
successful aging 557
traumatic brain injuries (TBIs) 575
vascular neurocognitive disorder 572

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