

Optional Reading: Neurocognitive Disorders

Historically, the term dementia was used to refer to an individual experiencing difficulties with memory, language, abstract thinking, reasoning, decision making, and problem-solving (Erber & Szuchman, 2015). However, in the latest edition of the Diagnostic and Statistical Manual of Mental Disorders Fifth Edition (DSM-5) (American Psychiatric Association, 2013) the term dementia has been replaced by neurocognitive disorder. A **major neurocognitive disorder** is diagnosed as a significant cognitive decline from a previous level of performance in one or more cognitive domains and interferes with independent functioning, while a **minor neurocognitive disorder** is diagnosed as a modest cognitive decline from a previous level of performance in one or more cognitive domains that does not interfere with independent functioning. Several different neurocognitive disorders commonly emerge in late adulthood. It can be difficult to determine the exact type because the symptoms may overlap with each other. Diagnosis often includes a medical history, physical exam, laboratory tests, and changes noted in behavior. Alzheimer's disease, vascular neurocognitive disorder and neurocognitive disorder with Lewy bodies will be discussed below.

Alzheimer's Disease. Probably the most well-known and most common neurocognitive disorder for older individuals is Alzheimer's disease. In 2016 an estimated 5.4 million Americans were diagnosed with Alzheimer's disease (Alzheimer's Association, 2016), which was approximately one in nine aged 65 and over. If there are no medical breakthroughs to prevent or cure the disease, by 2050 the number of people age 65 and older with Alzheimer's disease is projected to be 13.8 million. Alzheimer's disease is the 6th leading cause of death in the United States, but the 5th leading cause for those 65 and older. Among the top 10 causes of death in America, Alzheimer's disease is the only one that cannot be prevented, cured, or even slowed. Current estimates indicate that Alzheimer disease affects approximately 50% of those identified with a neurocognitive disorder (Cohen & Eisdorfer, 2011).

Alzheimer's disease has a gradual onset with subtle personality changes and memory loss that differs from normal age-related memory problems. Confusion, difficulty with change, and deterioration in language, problem-solving skills, and personality become evident next. In the later stages, the individual loses physical coordination and is unable to complete everyday tasks, including self-care and personal hygiene (Erber & Szuchman, 2015). Lastly, individuals lose the ability to respond to their environment, to carry on a conversation, and eventually to control movement (Alzheimer's Association, 2016). On average, people diagnosed with Alzheimer's survive eight years, but some may live up to 20 years. The disease course often depends on the individual's age and whether they have other health conditions.

The greatest risk factor for Alzheimer's disease is age, but there are genetic and environmental factors that can also contribute. Some forms of Alzheimer's are hereditary, and with the early onset type, several rare genes have been identified that directly cause Alzheimer's. People who inherit these genes tend to develop symptoms in their 30s, 40s, or 50s. Five percent of those identified with Alzheimer's disease are younger than age 65. When Alzheimer's disease is caused by deterministic genes, it is called familial Alzheimer's disease (Alzheimer's Association, 2016). Traumatic brain injury is also a risk factor, as well as obesity, hypertension, high cholesterol, and diabetes (Carlson, 2011).

Beta Amyloid and Tau. According to Erber and Szuchman (2015) the problems that occur with Alzheimer's disease are due to the "death of neurons, the breakdown of connections between them, and the extensive formation of plaques and tau, which interfere with neuron functioning and neuron survival" (p. 50). Plaques are abnormal formations of protein pieces called beta-amyloid. Beta-amyloid comes from a larger protein found in the fatty membrane surrounding nerve cells. Because beta-amyloid is sticky, it builds up into plaques (Alzheimer's Association, 2016). These plaques appear to block cell communication and may also trigger an inflammatory response in the immune system, which leads to further neuronal death.

Tau is an important protein that helps maintain the brain's transport system. When tau malfunctions, it changes into twisted strands called tangles that disrupt the transport system. Consequently, nutrients and other supplies cannot move through the cells and they eventually die. The death of neurons lead to the brain shrinking and affecting all aspects of brain functioning. For example, the hippocampus is involved in learning and memory, and the brain cells in this region are often the first to be damaged. This is why memory loss is often one of the earliest symptoms of Alzheimer's disease.

Washington University School of Medicine (2019) reported that researchers associated with the School of Medicine discovered that failing immune cells, known as microglia, appear to be the link between amyloid and tau, which are the two damaging proteins of Alzheimer's disease. Amyloid plaques, which appear first, do not cause Alzheimer's, but the presence of amyloid leads to the formation of tau tangles, which are responsible for the memory loss and cognitive deficits seen in those with Alzheimer's disease. It appears that weakening microglia cause the amyloid plaques to injure nearby neurons, thus creating a toxic environment that increases the formation and spread of tau tangles. These findings could lead to a new approach for developing therapies for Alzheimer's.

Sleep Deprivation and Alzheimer's. Studies suggest that sleep plays a role in clearing both beta-amyloid and tau out of the brain. Shokri-Kojori et al. (2018) scanned participants' brains after getting a full night's rest and after 31 hours without sleep. Beta-amyloid increased about 5% in the participants' brains after losing a night of sleep. These changes occurred in brain regions that included the thalamus and hippocampus, which are associated with the early stages of Alzheimer's disease. Shokri-Kojori et al. also found that participants with the largest increases in beta-amyloid reported the worst mood after sleep deprivation. These findings support other studies that have found that the hippocampus and thalamus are involved in mood disorders.

Additionally, Holth et al. (2019) found that healthy adults who remained awake all day and night, had tau levels that were elevated by about 50 percent. Once tau begins to accumulate in brain tissue, the protein can spread from one brain area to the next along neural connections. Holth et al. also found that older people who had more tau tangles in their brains by PET scanning had less slow-wave, deep sleep. Holth et al. concluded that good sleep habits and/or treatments designed to encourage plenty of high-quality sleep might play an important role in slowing Alzheimer's disease. In contrast, poor sleep might worsen the condition and serve as an early warning sign of Alzheimer's disease.

Healthy Lifestyle Combats Alzheimer's. Dhana and colleagues with the Rush University Medical Center in Chicago examined whether healthy lifestyle mitigates the risk of Alzheimer's disease (Natanson, 2019). Researchers followed a diverse group of 2765 participants for 9 years and focused on five low-risk lifestyle factors: healthy diet, at least 150 minutes/week of moderate to vigorous physical activity, not smoking, light to moderate alcohol intake, and engaging in cognitively stimulating activities. Results indicated that those who adopted four or five low-risk lifestyle factors had a 60% lower risk of Alzheimer's disease when compared with participants who did not follow any or only one of the low-risk factors. The authors concluded that incorporating these lifestyle changes can have a positive effect on one's brain functioning and lower the risk for Alzheimer's disease.

Vascular Neurocognitive Disorder is the second most common neurocognitive disorder affecting 0.2% in the 65-70 years age group and 16% of individuals 80 years and older (American Psychiatric Association, 2013). Vascular neurocognitive disorder is caused by a *blockage of cerebral blood vessels that affects one part of the brain* rather than a general loss of brain cells seen with Alzheimer's disease. Personality is not as affected in vascular neurocognitive disorder, and more males are diagnosed than females (Erber and Szuchman, 2015). It also comes on more abruptly than Alzheimer's disease and has a shorter course before death. Risk factors include smoking, diabetes, heart disease, hypertension, or a history of strokes.

Neurocognitive Disorder with Lewy bodies. According to the National Institute on Aging (2015a), *Lewy bodies are microscopic protein deposits found in neurons* seen postmortem. They affect chemicals in the brain that can lead to *difficulties in thinking, movement, behavior and mood*. Neurocognitive Disorder with Lewy bodies is the third most common form of dementia and affects more than 1 million Americans. It usually begins at age 50 or older and appears to affect slightly more men than women. The disease typically lasts 5 to 7 years from the time of diagnosis to death but can range from 2 to 20 years depending on the individual's age, health, and severity of symptoms.

Lewy bodies can occur in both the cortex and brain stem which results in cognitive as well as motor symptoms (Erber & Szuchman, 2015). The movement symptoms are similar to those with Parkinson's disease and include tremors and muscle rigidity. However, motor disturbances occur at the same time as cognitive symptoms, whereas with Parkinson's disease the cognitive symptoms first appear well after the onset of motor symptoms. Individuals diagnosed with Neurocognitive Disorder with Lewy bodies also experience sleep disturbances, recurrent visual hallucinations, and are at risk for falling.