# Alzheimer's disease and holistic wellness: Will counseling and medicine find common ground?

In my classes on gerontological mental health, I often characterize elders as time capsules with physical and psychological artifacts reflecting a lifetime of interplay between bodies, minds and the social world. Among the array of late-life mental health concerns with apparent connections to mind-body-social context interactions, perhaps none is more frightening than Alzheimer's disease (AD).

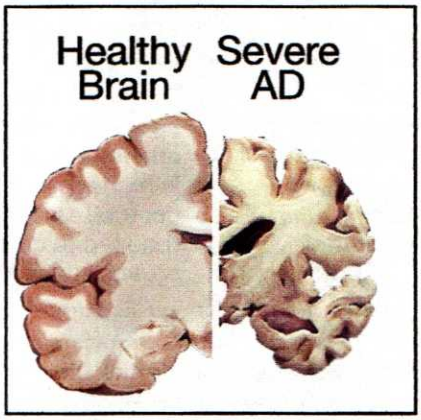
With AD, a lifetime of memories — loved ones, careers, passions — are swept away through the scourge of an unforgiving process of decline. Days can become filled with anxiety, depression, disorientation, disruptive behaviors, delirium, delusions, hallucinations and dependence on caregivers to assist with simple tasks such as dressing, grooming, cooking and shopping. The ability to express and receive language, process visual stimuli and carry out routine motor functions becomes an impenetrable mystery, with death as the inevitable outcome.

In this world of aging baby boomers, the statistics are becoming all too familiar. The Alzheimer's Association widely publishes that upward of 5.1 million Americans may currently have AD. With the inexorable march of time causing 10,000 more Americans to turn 65 on any given day, this number is likely to triple by 2050. Payments for AD-related health care were $226 billion in 2015. Without significant progress in strategies for prevention or cure, the 2050 price tag promises to be astronomical. And, as many counselors know, the dramatic impact on caregiving families — lost wages, extreme physical and mental stress, family strain, exhaustion, anger, resentment, grief — can linger for years after the sad passing of the loved one receiving care.

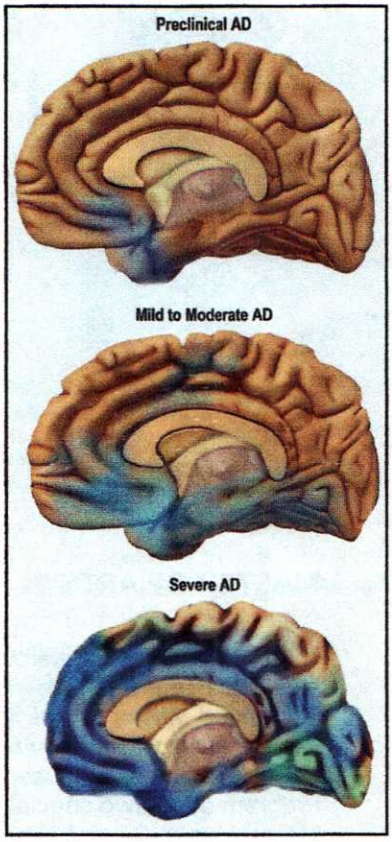
## Changes in brain anatomy, physiology

Many of the psychological struggles that impact our clients, such as anxiety and depression, often show subtle changes in brain anatomy and physiology, but the mark left by AD is dramatic. Over time, the brains of individuals with AD show progressive "shrinkage." In reality, this is the gross manifestation of massive cell death.

This shrinkage targets several key areas including, most predominantly, the hippocampus, an area associated with memory where the disease process begins, and parts of the cerebral cortex.




The cell death in the hippocampus early in the AD process explains why one of the first manifestations of the disease involves memory loss. Cortical destruction (destruction of the cerebral cortex) explains issues that emerge with language, executive function, routine motor tasks and sociability — in short, many of the functions we associate with being human.

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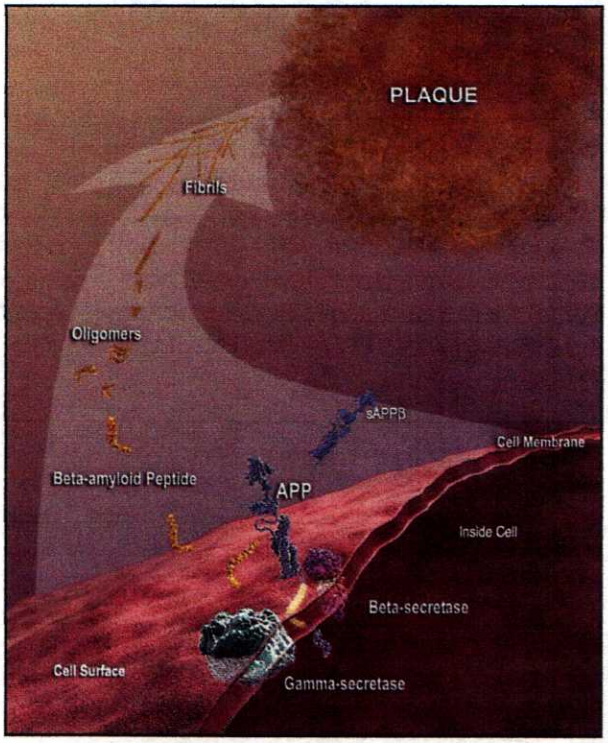
Several other brain structures also show cell loss, including the locus coeruleus and dorsal raphe nuclei. These areas are particularly significant because of their association with neurotransmitter production of norepinephrine and serotonin, respectively. This may explain why those afflicted with AD are often plagued with depression that sometimes begins before the actual diagnosis. Studies have demonstrated that this depression represents a neurophysiological disease process that is separate from other non-AD-related depression.

Sadly, individuals who struggle with AD depression are, relative to those without AD depression, plagued with a litany of challenges. These include decreased ability to carry out basic functions such as feeding, toileting, grooming and dressing (called activities of daily living); increased physical aggression (behavior that in a long-term-care setting can lead to physical or chemical restraints such as antipsychotic medications); higher risk of being discharged from assisted living with earlier entry into long-term care; increased mortality; and increased risk of suicide.

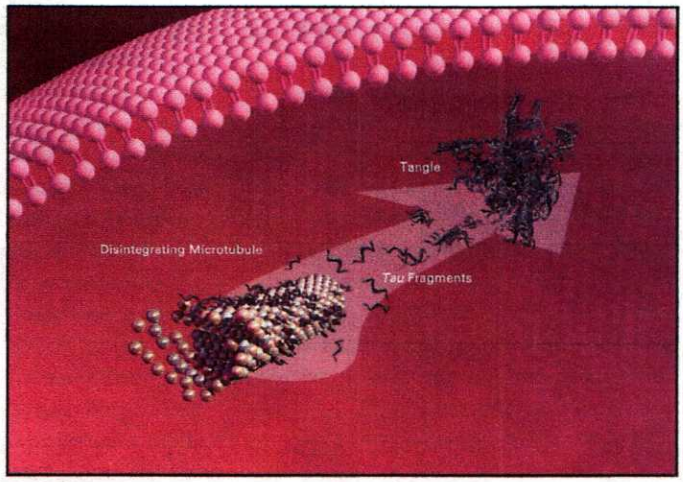
Translational advancements in AD science throughout the 20th century were slow in coming. By the end of the millennium, AD treatments could do little to slow and nothing to stop the steady drumbeat of disease progression. Definitive diagnosis of AD was possible only by inspection of brain tissue samples in which amyloid plaques and neurofibrillary tangles were found to be present. More commonly called *plaques* and *tangles*, these microanatomical features of AD brain tissue destruction gave pathologists a close-range view of the mechanism wreaking havoc on brain structure and function. They also provided possible avenues for research efforts aimed at understanding the etiology of the disease and finding a cure.




The plaques and tangles likely start to form upward of a decade before AD symptoms appear. The plaques, known more specifically as beta-amyloid plaques, are large clumps of protein and cellular components held together by a sticky substance called beta-amyloid. These plaques situate themselves between brain neurons in afflicted areas, and their formation is related to interrupted neural function. Ironically, the beta-amyloid is generated by a process that would actually assist in healthy neuron function in normal tissue.

A molecule called amyloid precursor protein (APP), which is planted in the cell membrane of the neuron, is normally cleaved in two places by enzymes called alpha secretase and gamma secretase. The APP fragments that are released through this normal cleavage process enter the spaces between the neurons and are important in healthy neural growth and survival. When the alpha secretase is replaced in this process by a close relative called beta secretase, the APP is cleaved in a slightly different manner that can have grave consequences for individuals afflicted with AD.

The product of this beta secretase cleavage pathway is the "sticky" beta-amyloid, which is released into the spaces between cells. After being released, the beta-amyloid goes through a series of steps in which individual beta-amyloids adhere to one another. Gradually, they build to a point where they form fibrils that then latch on to other proteins and cellular components to form the large insoluble clumps that we recognize as amyloid plaques. In the stage before fibril formation, the smaller collections of beta-amyloid may stick to the surfaces of other neurons in the area and impair cell function.




The neurofibrillary tangles also represent clusters of protein gone awry, but these clusters form inside, rather than between, neurons. Tangles are composed of a protein called tau that is, under normal conditions, a key component in stabilizing intracellular structures known as microtubules. Microtubules form a network of tubular structures that play a central role in transporting nutrients and other important cellular components down the axon of the neuron.

In those with active AD, the tau protein unites with an abnormal number of phosphate molecules, causing it to strip away from the microtubule. The unleashed tau fibers then associate with one another, ultimately amassing enough volume to form the neurofibrillary tangles. Without the support of the normal tau protein, the microtubules collapse. Cell-to-cell communication is impaired, and cell death ensues. For an excellent video from the National Institute on Aging that depicts plaque and tangle formation, see *nia.nih.gov/alzheimers/alzheimers-disease-video*.

## The Nun Study

The pathological changes in brain tissue paint an undeniably horrific picture. But beginning with research launched in 1991 came a most curious finding that, along with other research, laid the groundwork for complex models of disease etiology, progression and severity.

David Snowdon, an epidemiologist from the University of Minnesota (who later moved his research to the

University of Kentucky), persuaded 678 nuns from the School Sisters of Notre Dame congregation to participate in a longitudinal study of AD, commonly known as the Nun Study. This population was ideal because the nuns, ages 75-101, shared so many lifestyle factors, including diet, spiritual practices, social environment and access to resources such as education, health care, green space, exercise equipment and print materials. Thus, many confounding variables were eliminated from the study.

All of the sisters agreed, upon their deaths, to donate their brains to the study. Their generosity allowed Snowdon and his team to compare variables such as brain weight and the number and patterning of plaques and tangles with a wide array of factors over time, including cognition tests, autobiographical writings the sisters completed upon first entering the order as young women and cardiovascular, cerebrovascular and other health-related factors.

Among the many findings emerging from the study was one that was very surprising. Several of the nuns, who spent most of their days living lives that were socially engaged and intellectually stimulating, and who had outcomes on cognition tests that were well within normal ranges until the time they died, also had brains that, upon autopsy, showed plaques, tangles and volume reduction that were consistent with AD.

Snowdon's study also showed that the autobiographies the nuns produced when entering the order in early adulthood helped to predict late-life cognitive decline. Nuns whose autobiographies were filled with rich, complex prose (a measure that Snowdon termed "idea density") were less likely to show cognitive decline in late life than were their counterparts who exhibited low idea density. This finding suggested that something related to verbal development helped to ward off the devastation of AD.

Another important outcome of the study underscored the importance of vascular health, pointing to the need for attention to diet, exercise and other heart- and brain-healthy practices.

## A role for counseling

Snowdon's work, and other work like his, spawned a movement aimed at slowing or preventing AD-related cognitive decline through lifestyle management. The basic logic behind this movement is that any lifestyle choice that slows the deterioration of cognition can serve as an antidote to suffering — be it the suffering of AD victims or their caregivers.

The movement has been informed by a balance between "science and sense." The science, which is logistically quite challenging, has made slow and steady progress but has yet to make enough headway so that we can speak definitively about prevention or postponement of AD. From what we know about risk factors and patterns revealed through epidemiological and other studies, however, there is much that can cautiously be said using "sense." Absent a cure for AD, the ultimate goal would be to reduce its rate of cognitive decline and postpone associated impairment long enough that death actually precedes impairment. It is within this realm of postponement that a role for counseling has begun to emerge.

Of the three most predominant and best-studied risk factors associated with AD — age, genetics and family history — age is the least amenable to prevention strategies. At age 65, 1 person in every 9 has AD, whereas at age 85, the number rises to 1 in 3. We cannot halt the march of time.

The story regarding genetics has two divergent paths. One path leads to a very powerful, somewhat deterministic genetic component that is responsible for a special type of AD known as "early onset," which appears before age 65. After age 65, however, the genetic components are less deterministic. A gene known as APOE4 increases vulnerability, but the outcome is far from certain. This uncertain outcome may offer an opening for exploration of environmental factors that can be manipulated to stall the onset of cognitive decline. I will describe some of those possible factors below. For many of those factors, a role for counseling exists.

Being from a family in which any other family member has AD increases risk, but whether this is a genetic or lifestyle outcome is uncertain. Of course, there is the chance that both genes and environment/lifestyle are playing a role, again opening the door to possible forestalling measures through manipulation of the environment and changes in lifestyle. Once more, this suggests a beneficial role for counseling.

Outside of these three factors, there are others that appear to be fruitful targets in the battle against cognitive decline. Interestingly, many of these targets are related to general brain health and can be tied to systemic social justice issues. They also have a relationship to mental health intervention and wellness counseling. It is well-known, for example, that stroke exacerbates loss of cognition among AD victims, so it stands to reason that anything that compromises the vascular health of brain tissue, including heart health, hypertension and diabetes, would be suspect in a comprehensive understanding of disease progression. When we combine this information about vascular health with the fact that AD, stroke, cardiac problems, diabetes and hypertension are more common among African Americans and Latinos than other racial/ethnic groups, it raises questions about possible systemic environmental causes.

The reason or reasons for these race/ethnicity associations are speculative, but it does open the door to thoughts regarding the role of chronic stress and trauma stemming from economic, racial and other social injustice. For an extended discussion on the role of epigenetics and psychoneuroimmunology in transforming experiences of marginality and social injustice into vascular and other health and mental health problems, see the Neurocounseling columns from the March and September 2015 issues of *Counseling Today*. For now, suffice it to say that issues related to socioeconomic status, such as affordability and accessibility of whole-food sources, lack of green space, unsafe neighborhoods and a preponderance of fast-food options, all potentially foster weight problems, metabolic syndrome (which includes prediabetic physiological changes), heart disease, cerebrovascular disease and hypertension. In this case, helping clients to participate as fully as possible in stress reduction, foster social supports, access services and engage in healthy lifestyle choices related to diet and exercise may hold promise for future brain health.

Another risk factor directly within the purview of counseling practice is depression. There is some evidence that years before the emergence of AD cognitive decline, damage done by non-AD-related depression can set the stage for a tougher battle with cognitive loss. With higher rates of depression and AD among women, recognizing and treating depression in midlife should be part of a comprehensive public health strategy.

Depression also has a negative impact on two other factors important to brain health: exercise and social engagement. In animal studies, exercise has been shown to foster increased vascularization of brain tissue and increased volume of neuron-to-neuron connection in older mice and rats. Epigenetic studies have shown that exercise increases hippocampal levels of a substance essential for nerve growth (see the September 2014 Neurocounseling column). This and other evidence has been the impetus for major campaigns to engage seniors in regular exercise. Likewise, regular positive social engagement is considered essential for brain health and is more likely to be pursued in the absence of depression.

Finally, another social justice issue that emerges in many studies relates to education. The protective value of education is undeniable, and it is an affordance increasingly associated with financial means. Helping clients to articulate and reach educational and vocational goals, and to access resources to assist them in reaching those goals, could be part of a long-term strategy for brain health.

Jane Myers, Thomas Sweeney and Melvin Witmer's Wheel of Wellness, a cornerstone of counseling practice, touches on a number of areas germane to brain health. As a model that considers intellectual, spiritual, social, emotional and physical wellness, it has the potential to inform a lifelong strategy for late-life brain health.

Attempting to look ahead through the lens of early 21st-century medical research, it seems likely that the philosophical underpinnings of the counseling profession will comport with emerging best practices as science achieves a better understanding of pathways to delay or even prevent AD. Advances that have already occurred in diagnostic techniques through imaging and testing of cerebrospinal fluid may encourage physicians and mental health professionals to more fully engage in concerted and intentional early intervention and prevention strategies. ❖

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