Reversing Alzheimer's Disease and Mild Cognitive Impairment is Now a Reality *Part 2*

By Scott Vander Wielen, DC, DABCI

Until recently, there was little that could be done to reverse Alzheimer's disease. Now that Dr. Dale Bredesen and the Institute for Functional have teamed up, the prognosis for an individual with Alzheimer's disease or mild cognitive impairment is about to change. My article last month focused on the three main types of Alzheimer's disease that Dr. Bredesen has characterized. The purpose of this month's article is to review the process underway inside the brain that identifies Alzheimer's disease or mild cognitive impairment.

Your body has a high IQ and innately knows how to care for itself. Interestingly, the process of Alzheimer's disease or mild cognitive impairment is actually a process to contain a bacteria or virus. Amyloid-β, a major player in Alzheimer's disease and a term you already may be familiar with, is really an antimicrobial protein. The amyloid-β entraps the bacteria or virus threatening the brain's cell membranes, resulting in more synaptic destruction than synaptic creation. In fact, successfully reducing the amyloid-β protein in clinical trials had adverse outcomes. Knowing the reason behind the presence of amyloid-β is a clue to discovering the root cause.

What causes an overproduction of amyloid-β? There is a protein in the brain called amyloid Precurser Protein (APP), which is made by neurons and sticks out of neurons, especially near synapses. There are molecular scissors called proteases that will cut the amyloid precursor protein. It can either be cut at one distinct site or at three spots along its length. If it is cut at one spot along its length the result is just two peptides, and synaptic connections are maintained, and neurons are nourished and preserved. However, if the amyloid precursor protein is cut along three spots along its length, then four peptides are produced, and one of them is amyloid-β. The result of this is a loss of brain synapses, a shriveling up of the neuron that extends out to connect to other neurons, and the activation of the neuron's programmed cell death. Everyone with Alzheimer's disease or mild cognitive impairment is on the wrong side of this very crucial balance.

As if that weren't enough, a characteristic of amyloid-β is it is prionic, which means amyloid-β can get more of itself without the need for more genetic material. It is a continuous loop. So the sense of urgency enters the picture because the process can be described as a feed forward process with an over-production of amyloid-β.

The good news is that amyloid precursor protein is a dependent receptor. This means how amyloid precursor protein gets cut depends on the molecule it grabs. We want that molecule to be netrin-1. Interestingly, netrin-1 in Sanskrit means, "one who guides." Part of a successful strategy will involve creating a healthy internal environment within the brain where netrin-1, the guiding molecule, is not removed.

The factors involved in creating a healthy internal environment for the brain are multi-layered. In order to make sure the netrin-1 molecule is not removed, small dense LDL, fasting insulin, homocysteine, hormones, vitamin D, hemoglobin A1c, mercury, copper, zinc, vitamin E, B6, B12 (and other factors) must be measured and tested. This is why it is a personalized treatment plan. Reversing Alzheimer's disease or mild cognitive impairment is not a one-size-fits-all solution. A detailed history and physical exam along with the right labs are necessary in order to make the right changes in lifestyle choices and effective clinical nutrition.

The bottom line: there is hope for anyone who suffers from Alzheimer's disease or mild cognitive impairment. No single compound can patch the holes in the roof that contribute to Alzheimer's disease, but the right combination can patch them all.

References: "The End of Alzheimer's: the First Program to Prevent and Reverse Cognitive Decline." D Bredesen. Avery. 2017.

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