

DEMENTIA AND PC

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Through the generations, health fears change. Long ago, people feared consumption, the old name for pulmonary tuberculosis. Oh, you thought tuberculosis applied only to the lungs. It's a bacterium, a Mycobacterium, that can attack bones, kidneys, skin and even the heart. In 1900, pulmonary diseases, including pneumonia, were the leading cause of death in the United States, and life expectancy was around forty-seven years. By the early 1930's, life expectancy increased to sixty, and heart disease became the biggest fear. Of course, a longer life span gives enough time for pathologies, like clogged arteries, to develop and cause problems. In the early 1700's, interest in cancer epidemiology was aroused. Tobacco was culpable even then. Believe it or not, cancer surgery was performed in the early 19th century, some time after 17th-century autopsies identified cancer as an operable condition. Added to the collection of fearsome medical issues is dementia, first making the list in the 18th century as a state of intellectual deficiency, later restricted to those with disrupted cognitive ability. Back in time, syphilis was considered as the common cause of dementia, at least until 1906, when Alzheimer's was tagged as the major culprit.

Dementia may have a number of causes, but generally cites the degeneration of neurons or disturbances in body systems that affect neurons' functions. Though Alzheimer's is a common cause, vascular dementia is a contributing factor. Here, reduced blood flow to the brain is to blame. Stroke may be responsible, but a heart

attack can also reduce blood supply for a time. Vascular dementia has an identifiable cause; Alzheimer's does not. The Alzheimer's Association estimates that sixty to eighty percent of dementia diagnoses rest on that disease.

Neurodegeneration means that neurons gradually lose their ability to function as designed. As they fade, neuron-to-neuron connections—synapses—are lost and messages cannot be transmitted in the brain. A range of dysfunction results. Alzheimer's and vascular dementia are not alone in this situation. Parkinson's disease may be accompanied by Lewy body dementia, medications and chronic alcoholism may cause aberrations, and, of course, tumors may incite disturbances. To our dismay, there are no single tests to confirm a diagnosis. Medical history, physical exam, lab tests, and symptom overview can help, yet not be definitive.

Certainly, we're interested in ways to prevent the onset of any dementia. Lifestyle seems to play so important a role that it is believed to be involved in more than a third of reported cases (Rosenberg, 2019) (Kivipelto, 2018). Some of the risk factors include lack of education in childhood, hypertension and obesity at midlife, hearing loss, diabetes, physical inactivity, smoking and social isolation. Addressing these issues may reduce risk by more than twenty percent (Frankish, 2017). Researchers found that early education adds to a person's "cognitive reserves," helping them to function despite the initial symptoms of dementia, thus delaying onset and progress of the condition. As far as obesity goes, extra fat relates to an insulin response that incites inflammation to impair cognitions (Yamamoto, 2019). After arthritis and hypertension, hearing loss is the third most prevalent chronic medical condition among those older than sixty-five (Wingfield, 2012). Hidden at first are the effects on cognitions and neural integrity. According to at least one study, hearing aids do little to improve cognitive prognosis (vanHooren, 2005). Trusted interventions for Alzheimer's prevention include exercise (walking), nutrition counseling, cognitive and social stimulation and management of medical conditions.

Although we have no control over one's willingness to exercise, to spend time with peers or family in stimulating social engagements, or to work crossword puzzles or other mind games, we can offer nutritional interventions to mitigate circumstances and to ease the fear of dementia risks. There are a few particular foods held to reduce the risk of memory loss. Berries are well-known for their anti-inflammatory and anti-oxidant properties, being able to prevent cell damage in the brain. The MIND Diet, designed by Dr. Martha Clare Morris, from Rush University Medical Center in Chicago, suggests two servings of berries a week. The anthocyanins in blueberries are associated with increased neuronal signaling and glucose disposal (Krikorian, 2010).

Like berries, leafy greens are high in anti-oxidants. The combination

of lutein, folate, beta-carotene and phylloquinone slow memory loss when eaten regularly (Morris, 2018). Carrots, sweet potatoes and cantaloupe share a common color, but also beta-carotene, shown to improve cognition scores and verbal memory outcomes in a study that encompassed four thousand subjects of middle and advanced ages (Grodstein, 2007). Whole grains, especially rich in B vitamins, work to reduce brain inflammation, a sad state of affairs in developed countries where the populace is too busy to consume a balanced diet. Even as supplements, administration of the entire group of vitamins B at doses that exceed government recommendations is deemed a rational approach for preserving brain health (Kennedy, 2016). Despite the vitamin content, some grains and cereals have been the subject of mycotoxicity by virtue of exposure to damp conditions during harvest and storage. Compensatory at providing B vitamins would be lean meats and poultry. Never to be omitted from a conversation about the brain are omega-3 fatty acids, sourced from fish as well as from seeds and nuts (Külzow, 2016) (Witte, 2014).

Omega-3 fats from foods, including seeds and nuts, provide the mother fatty acid, alpha-linolenic acid (ALA). In a healthy adult over thirty or thirty-five years old, conversion to the downstream fatty acids, EPA and DHA, is an inefficient process, especially in males. Therefore, supplementation with fish oil or other marine oil is a prudent undertaking. The Karolinska Institute, a research-based medical university in Sweden, discovered that n-3 fats in dietary supplements can cross the blood-brain barrier (BBB) in Alzheimer's disease, affecting markers for both the disease itself and for inflammation (Freund-Levi, 2014). N-3 fats accumulate in the central nervous system (CNS) during gestation. It had been assumed that these fats are constantly replaced throughout life, but little was known about dietary influences on the transport of fatty acids across the BBB, whose function protects the brain from harmful chemicals in the blood, but also blocks drug delivery to the brain.

More than one problem can affect the fatty acid profile of the brain. In those with Alzheimer's disease, low concentrations of DHA have been noted. That aroused interest in DHA supplementation in an attempt to determine whether DHA repletion could have a positive effect on mild forms of that insult. Freund-Levi and her team found that levels of DHA showed a direct correlation to the degree of change in Alzheimer's (Ibid.).

To embellish the positive action of DHA on brain health, the merging with phosphatidylcholine was seen to make a difference in assimilation. It's well understood that DHA synthesis in the brain is considerably low, so uptake depends on a circulating lipid pool to maintain homeostatic values. Once DHA passes the BBB, it enrolls into a series of pathways that mingle with membrane phospholipids to become distributed throughout the brain, where it becomes a bioactive mediator to regulate signaling pathways

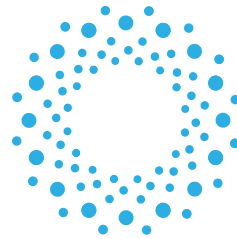
important to synapse manufacture / stability and the control of neuroinflammation (Lacombe, 2018). This is especially good news for people who carry the APO E4 gene, an occasional marker for Alzheimer's risk, but not necessarily a predictor of active disease.

Admittedly, APO E4 is the most prevalent common determinant for Alzheimer's presentation, but gender, age, lifestyle and nutrition are factors that modulate the gene's supremacy. Here, too, higher DHA levels are associated with lower risk of AD. The confounding news, though, is that APO E4 carriers are less responsive to DHA than they need to be. Either higher doses or combination with phosphatidylcholine can resolve the issue in the carrier's favor (Pontifex, 2018) (Patrick, 2019). Notable is that carriers respond better to the DHA in fish than they do to that in supplements (Ibid.). Late in the last century, studies on demented lab animals showed that administration of PC improved memory and increased brain choline and acetylcholine concentrations, while it had little such effect on already-healthy animals (Chung, 1995) (Moriyama, 1996). BodyBio PC, a complex containing P-ethanolamine (PE) and P-inositol (PI), is a welcome adjunct to marine oil DHA. Its PE serves as a reservoir for conversion to P-serine, the signaling molecule that also plays a role in coagulation and macrophage activation. PE influences membrane topology and autophagy while attending to mitochondrial well-being and oxidative phosphorylation. Alone, PE is associated with improvement in Parkinson's disease, non-alcoholic liver disease and Alzheimer's. Unused PE reverts to PC (Calzada, 2016). In vascular dementia, it's been noted that phospholipid levels are drained, thus serving as biomarkers for severity of the condition, but also encouraging phospholipid replacement via exogenous supply (Sabogal-Guagueta, 2018).

Becoming a senior citizen does not mean you become a senile citizen, despite what you may have seen or heard about in others. One of the best ways to stave off the mental adversities that are associated with ageing is to get enough sleep. Not only does sleep offer the chance to lock in memories, but also helps to process what may become new skills. Sleeping more than nine hours a night, though, can backfire and cause the very conditions we are trying to avoid. Yes, there is a ton of studies that promote sleep as a tool to prevent dementia, but a recent study from the University of Miami Miller School of Medicine found that those who slept longer than nine hours a night showed a decline in memory and language skills (Ramos, 2019). An earlier study at Brigham and Women's Hospital, in Boston, indicated that sleep duration shorter or longer than the recommended seven-eight hours a night may increase risk of metabolic and cardiovascular diseases, as well as decreased cognitions (Devore, 2014).

Sleep deprivation has a substantiated ill effect on liver health by causing metabolic issues related to oxidative stress and phospholipid degradation. Here, liver phosphatidylcholine stores are diminished,

eventually affecting brain health as well as liver well-being (Chang, 2008) (Chua, 2015), since PC is a system-wide necessity. Here, once more, is a rationale for PC administration. By the way, PC is indicated in the Physician's Desk Reference for Nutritional Supplements to modulate liver function. But it has to be a liposomal formulation, like BodyBio PC, not the ersatz triple lecithin pretender.



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