Aging and Nutrition in Older Adults and the Elderly: The Relationship between Nutritional Factors on Cognitive Impairment

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As people age, they experience many changes in their health and strive to stay healthy as long as possible. Today, people are living longer. Some changes are a part of normal aging, but other changes are due to environmental factors and genetics such as cognitive decline (Moorthy et al., 2012) Psychological damage is not a part of normal aging (Brown, 2011). Cognitive disorders describe a wide range of mental malfunctions such as mild cognitive impairment and dementia associated with Alzheimer's, Huntington's, and Parkinson's. Alzheimer's is the most common cause of dementia which is defined as a "condition of progressive cognitive decline, typically characterized by impaired thinking, memory decision-making, and linguistic ability" (Brown, 2011, 507). According to Brown, 14% of older adults (70 or older) have dementia and 10% have Alzheimer's disease (Brown, 2011). Why is dementia common in aging adults? Most likely it is the result of traumatic psychological changes which increases as one ages. Sometimes dementia can be treated, but most of the time, no treatment will cure dementia. Therefore, what can prevent cognitive decline? Modifying environmental factors such as dietary habits have been discovered to prevent cognitive decline (Brown, 2011 and Moorthy et al., 2012). Many studies show from extensive research from professional nutrition journals that cognitive impairment is associated with vitamin B-12 and folate, vitamin D, copper, fatty acids, and dehydration.

A commonly studied nutritional factor associated with cognitive decline is vitamin B-12 and folate. Vitamin B-12 and folate are related to the amount of homocysteine in the blood (Brown, 2011). Homocysteine, an amino acid which is associated with cognitive decline, increases with deficiencies of vitamin B-12 and folate (Brown, 201; Moorthy et al., 2012). According to Moorthy, in 90 of 100 studies with 46, 881 men and women as test subjects have shown an association with B-12 and homocysteine with poor cognition. Research has concluded that

homocysteine has been associated with brain atrophy, especially high concentrations of homocysteine (Moorthy et al., 2012).

Vitamin B-12 and folate are essential for converting homocysteine to methionine, an amino acid which contributes to the synthesis S-adenosylmethionine, used in the central nervous system. Since methylation reactions in the central nervous system cannot occur, adequate levels of B-12 and folate are important to prevent the excess of homocysteine (Brown, 2011).

A 2007 study from the American Journal of Clinical Nutrition agrees with this relationship. The objective of the study was to find the relations between folate and B-12 related to anemia, macrocystosis, and cognitive impairment. Participants in the study were seniors from the 1999-2002 National Health and Nutritional Exam Survey. The study concluded that participants with low B12 status, the high serum folate levels were associated with cognitive impairment and participants with normal B12 status, the high serum folate levels were associated with protection against cognitive impairment. This study agrees with the previous hypothesis that adequate B-12 and folate is important in preventing cognitive impairment (Morris, Jaques, Rosenburg, & Selhub, 2007).

In another study, which looked at the affect of gene differentiation in ethnic groups on cognitive impairment, found that low vitamin B-12 was associated with poorer scores on a mental status exam (Moorthy et al., 2012). This evidence agrees with the previous evidence, but also did not find that folate or homocysteine was associated with cognition. This does not rule out the agreement of other professionals and researchers of the affect of folate and homocysteine on cognitive development, but gives light that more research may be needed to accurately say the affect of homocysteine. Perhaps, even though the researchers were aware of the evidence of the affect of folate and homocysteine, they were more focused on the genetic factors associated with

certain ethnic groups. Therefore, B-12 and folate has a significant association with cognitive decline in older adults.

Vitamin D has been discovered to be associated with mild cognitive function. According to Annweiler in a 2012 study from the European Journal of Neurolgy, low vitamin D concentrations are associated with mild cognitive impairment in the adult population that live in the community and do not have dementia. The participants in the study were 95 white, non-demented community members with subjective memory complaints, but without diagnosed dementia. The results showed that an increase in vitamin D was associated with lower risks of mild cognitive impairment and patients with mild cognitive impairment, had lower vitamin D levels. Therefore this study shows evidence of the importance on adequate vitamin D to prevent cognitive impairment. But since this study is recent and before this study, the associated between vitamin D and mild cognitive impairment, not dementia, more evidence may be needed to conclude this reasoning (Annweiler, Fanitino, Schott, Krolak-Salmon, Allali, & Beauchet, 2012)

Toxicity of copper has been hypothesized as a cause of mild cognitive impairment and Alheizmer's. According to Brewer in a 2009 study published in the Journal of American College of Nutrition, he believes that mild cognitive impairment and Alzheimer's is due to the intake of too much copper. This hypothesis is still yet to be widely approved by other professionals, but in other studies such as an Italy study by Squitti and colleagues, tests done on animals, and a study by Morris and colleagues, Brewer is convinced that copper does affect cognitive impairment. In the study by Morris and colleagues, they found that the participants with the highest copper intake and the highest fat diet, lost 19 years of cognition which was determined over a 6 year period. This loss is more than three times the normal rate which is huge.

Brewer proposes that all molecules involved with Alzheimer's disease bind copper which contribute to cognitive decline. Homocysteine as previously determined to be associated with cognitive decline interacts with copper. Since homocysteine levels increase and the risk of Alheimer's increases, copper plays a role in homocysteine levels. Copper is essential, but only in trace amounts and free copper is toxic. Free copper is found in tap water and supplements. Brewer advises that water should be tested for levels of copper and supplements should not have copper in them. It has been found that many multi vitamins have a high copper level which is not necessary and toxic. (Brewer, 2012).

As previously studied, dietary fatty acids may play a role in cognitive decline. Studies have shown that high levels of saturated fatty acids increase the risk of cognitive impairment. In contrast, high levels of unsaturated and polyunsaturated fatty acids have been shown to lower the risk of cognitive impairment. According to a 2010 study from Italy, studies on polyunsaturated fatty acids are limited and the few studies on polyunsaturated fatty acids have contrasting findings (Panza, Frisardi, Seripa, Pilotto, & Solfrizzi, 2010).

But other interest has been in the findings of a gene (APOE genotype) which is proposed to be associated with fatty acids and cognitive impairment. This gene may affect the difference of the effect that dietary fatty acids have on lipid protein metabolism. This contributes the contrasting evidence of the effect of dietary fatty acids. Other studies have proposed that people with this gene do not have any protective effects or benefits from eating fish. Also, these people are more at risk of environmental factors such as alcohol and low B-12 influences. Therefore, since people with this carrier have poor repair mechanisms, this gene is associated with a predisposition for decline in cognitive function. This study shows no evidence for any difference with this gene in relation to cognitive decline, except for the association with attention.

Therefore, polyunsaturated fatty acids may help lower the risk of cognitive decline in people without the carrier (APOE). (Panza, Frisardi, Seripa, Pilotto, & Solfrizzi, 2010).

Contrasting evidence is a study on the relationship of cheese on cognitive impairment. Cheese, made up of mostly saturated fatty acids, has a protective effect of cognitive decline. Evidence from a study of 1056 elders from Alabama's elderly population, who were mostly female and white, showed that when cheese was eaten at least once a week, 40% lower risk of cognitive decline. No well evidence backs up this reasoning. Perhaps, saturated fatty acids counter balance the monounsaturated and unsaturated fatty acids, but in this study they did not consider the gene (APOE). Other inconsistent or contradictory studies include that higher monounsaturated fatty acids provide protection against cognitive decline and other studies show that lower monounsaturated fatty acids are associated with better cognitive function (Rahman, Baker, Allman, Zarmrini, 2007).

This conflicting evidence may have to do with not associating the predisposition gene with this affects on cognitive function. Perhaps, if more studies focused on the effect of the fatty acids related to this gene, more concrete reasoning would be found.

Another association with cognitive decline is dehydration. Dehydration is a serious condition as well as more risky for the elderly. Dehydration, even as little as 2 %, has been proven to be associated with impaired mental performance, confusion, and disorder. It affects the brain from functioning properly because of the imbalance of homeostatic functions. This imbalance affects the amount of electrolytes in the blood, therefore decreasing blood flow and altering brain activity (Adana, 2011).

Researchers have proposed that dehydration may lead to dementia. In accordance with this proposal, they have found that the same test for healthy adults and older adults is not accurate

(Brown, 2011). This shows researchers that dehydration may have a more detrimental or serious affect on the elderly and may need more research. Even though, more research is needed to accurately understand the affect of dehydration on cognitive performance, dehydration has been shown to affect cognitive decline.

In conclusion, all these studies are important to preventing and decreasing the risk of cognitive impairment in the aging population. Vitamin B-12 and folate has sufficient evidence that deficiencies in these nutrients lead to increase levels of homocysteine. A high level of homcysteine is not good for the body and should be avoided. Older adults and the elderly should meet the RDA for vitamin B-12 and folate to help prevent cognitive decline. Second, adequate vitamin D is important for cognitive function. Older adults and seniors should also meet the RDA for vitamin D and even though more evidence is needed, vitamin D is still an essential vitamin for other reasons too. Third, high levels of copper have been proposed to affect cognitive function. Some researchers may refute this hypothesis and more evidence is needed. But older adults should still not take copper in their multi-vitamins or supplements and check their water for copper. Even if more evidence is needed, the body does not need more copper than what it already makes. Fourth, studies have concluded that unsaturated and polyunsaturated fatty acids help prevent cognitive impairment whereas saturated fatty acids impair cognitive function. Conflicting and inconsistent studies of the affect of fatty acids on cognition show that more evidence is needed. One reason for this is a predisposed gene which may affect why people have different affects from fatty acids on cognition. This still needs to be explored. Lastly, dehydration affects cognitive function, therefore the elderly should be advised to drink lots of fluids and fluid intake should be tracked in nursing homes. Nutritional factors do play a

role in cognitive function, but much more evidence is necessary to prevent dementia and Alzheimer's disease.