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File: ■ Green Tea (*Camellia sinensis*)
■ Neurodegeneration
■ Brain Aging

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RE: Chronic Green Tea Consumption Exerts Protective Effects on Age-related Neurodegeneration

Andrade JP, Assunção M. Protective effects of chronic green tea consumption on age-related neurodegeneration. *Curr Pharm Des*. 2012;18(1):4-14.

The cause of aging is unknown, but one theory is the "free radical theory." The theory is that mitochondria in cells simultaneously produce free radicals (which cause oxidative damage) and are, themselves, a preferential target of reactive oxygen species. The oxidative damage causes loss of function and efficiency, and more reactive oxygen molecules are released, thereby continuing the cycle. Neurons in the brain are particularly susceptible to this cycle. Green tea (*Camellia sinensis*) is an excellent source of antioxidants (such as catechins and other polyphenols). The purpose of this review article was to compile recent studies evaluating the effects of green tea or its catechins on brain aging at the biochemical or behavioral level. This is not a systematic review; the authors do not list any search criteria. The report is just an overall review of the topic.

The authors state that there is a lack of systematic clinical trials that evaluate the actions of green tea on the brain. They cite epidemiological data that green tea consumption is associated with a decrease in the prevalence of cognitive impairment in Japanese people, as well as a reduced risk of Parkinson's disease. There is debate as to whether plant polyphenols or their metabolites can cross the blood brain barrier in humans. While only minimal concentrations of flavanols (e.g., quercetin) have been found in vivo in rat brains, flavonoids (e.g., catechins) have been shown in some in vivo studies to accumulate in certain brain regions.

Green tea polyphenols are proposed to protect lipids and proteins in the hippocampus (an area of the brain responsible for memory and learning). In aged rats, green tea consumption increased levels of superoxide dismutase (SOD, a scavenger of superoxide radicals) in the hippocampus. Also, in a human neuroblastoma cell line, (-)-epigallocatechin-3-gallate (EGCG) increased SOD. In an in vivo stroke model, EGCG delayed hippocampal cell death. Green tea polyphenols can also protect against reactive nitrogen species. For example, EGCG protected against nitric oxide (NO)-induced hippocampal cell damage. Despite all of these in vivo and in vitro findings, many authors

report that the antioxidant activity of polyphenols cannot occur in humans because green tea polyphenols minimally enter brain cells. However, some authors believe that green tea polyphenols may act via a signaling pathway rather than by directly acting on the brain to benefit brain aging. For example, some in vitro studies show that green tea polyphenols suppress nuclear factor-kappaB (NF-κB). The activation of NF-κB occurs with aging and many age-related neurodegenerative diseases. Also, EGCG inhibits the expression of pro-apoptotic (cell death) genes in vitro.

In vivo studies demonstrated that chronic green tea intake or epicatechin intake prevented age-associated loss of the ability to perform certain memory tasks, and increased working memory-related learning. Also, in mice, oral consumption of EGCG for 4 weeks increased the number of cells in memory regions of the brain. Other studies in mice reported an increase in neurite outgrowth, potentiation of neuritogenesis, and a promotion of synaptic plasticity.

The authors conclude that altogether the global findings confirm the neuroprotective abilities of green tea and its polyphenolic compounds in antioxidant defense, neuronal survival, and neurogenesis associated with brain aging. Polyphenols from sources other than green tea may also provide benefits. Nevertheless, clinical trials are necessary to establish any benefit, particularly since there may be limited direct access to brain cells.

—Heather S. Oliff, PhD