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Turmeric – A Spice with Health Potential

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Turmeric is the spice that gives curry powder its piquant flavor. Epidemiologists have noted that cultures consuming the greatest amount of turmeric have low incidence of Alzheimer's disease (AD). India, for example, which uses this spice both in cooking and as an herbal medicine, has one of the lowest incidences of AD in the world. The rate of AD in India is less than one-quarter that of the US.

Examining The Effects of Turmeric

Of course this observation in itself does not prove a causal relationship between turmeric and the low incidence of AD. However, it did serve as a stimulus to probe for a possible connection that could be validated by scientists. This article summarizes the results of experiments examining the effects turmeric on the degenerative events to the brain that are associated with AD.

Turmeric is prepared from the root of the plant *Curcuma longa*. The characteristic dark yellow color is due to the presence of a group of related phenolic compounds known as curcuminoids, a major constituent of the plant. Studies in animals and in cell cultures are discovering that these compounds have profound biological effects, virtually all of which have positive health value. The effects include ulcer healing, cancer prevention, protection from free-radical oxidation, anti-inflammatory, anti-arthritic and many more. Curcumin, which constitutes about 4% of turmeric, is currently being developed as a cancer chemo-preventive agent in India. This article, however, will explore this curcuminoid as a potential agent for preventing or improving the symptoms of AD. The rate of Alzheimer's disease in India is less than one-quarter the of the U.S.

What causes Alzheimer's?

Alzheimer's disease is a chronic neurodegenerative disease that results in loss of brain function. Early symptoms are forgetfulness and impaired awareness of the events of daily life. The disease is more common in the elderly and affects on average



about 3.1% of Americans between 70-79 years, (vs. 0.7% in India). The percentage increases with age. The precise cause of AD is presently unknown. However, a specific protein, amyloid-beta, is ubiquitous in the brains of AD patients and is believed to play a major pathological role. In AD patients, this protein is produced in excessive amounts that collect in deposits called amyloid plaque. (The word "amyloid" describes any protein fragment that combines to form starch-like structures deposited in tissues, and reacts with a stain called Congo red.)

These amyloid-beta proteins are produced from a larger protein (amyloid protein precursor, APP) which is present in normal healthy cells and, in fact, is important to the health of our nerve cells. APP is a good guy when whole and produced at normal levels. However, it can be the source of toxic amyloid-beta when either too much APP is produced, or when APP is processed abnormally, resulting in an excess of amyloid-beta due to mutations or abnormalities in specific processing enzymes. The toxicity appears to be related to the propensity of these amyloid-beta protein fragments to stick to one another, and to form progressively larger aggregated structures.

The initial structure formed by these sticky proteins is known as a soluble oligomer, which appears to be the most toxic. This is followed by formation of structures known as fibrils, which finally are transformed into larger structures, called amyloid-beta aggregates. The aggregates are insoluble, and thus fall out of solution to form deposits (plaque) on the cells' exterior. Recent evidence indicates the presence of these oligomers, fibrils and aggregates activates the production of free radicals as well as inflammatory cells. The free radicals attack nervous tissue constituents, releasing oxidized and damaged cellular constituents (such as fragments of membrane lipids) which, in turn, exacerbate the condition by amplifying the immune-inflammatory response. The net result is severe damage to important structures of the brain. The accumulated damage ultimately is expressed as clinical manifestations of AD.

Back to Curcumin

Recent experimental information supports a role for curcumin in inhibiting the formation of amyloid-beta oligomers and fibrils, as well as potentially acting to remove amyloid plaque. These effects were observed in the test tube, in cell culture and in animals fed curcumin at non-toxic doses. Curcumin appears to disassemble amyloid-beta aggregates and prevents oligomer and fibril formation. It binds to amyloid-beta, and prevents the latter from binding to itself.

What is exciting about the potential for this compound as a successful agent to treat AD is the fact that it is well-tolerated even at oral doses between 4-8 g/day in humans. A dose equivalent in humans to just 1-2 grams/day has been



demonstrated in animal studies to produce a serum concentration that is effective in inhibiting amyloid-beta accumulation in brain tissue. In addition, this dose was demonstrated to inhibit the production by brain cells of inflammatory mediators believed to be partly responsible for brain cell destruction initiated by amyloid-beta. Furthermore, curcumin is a highly effective antioxidant (more potent than vitamin E), capable of neutralizing oxidants that escape the cells' antioxidant defense system, thus protecting the delicate nervous tissue from oxidant-induced damage.

Prevention is the best way to maintain health

Because amyloid begins to accumulate years before symptoms are evident, the use of anti-amyloid agents would be most effective if used prior to the appearance of symptoms. The symptoms from this damage may not appear for years, and the damage is difficult to reverse.

To be effective in preventing, postponing or treating AD, a substance – whether a drug, a food ingredient or a dietary supplement – must meet three criteria. It must be bioavailable, which means it must be able to reach the area of interest, the brain, at an effective concentration after oral ingestion. Second, it must be non-toxic at a dose required to reach an effective concentration at the location of interest in the body. Curcumin satisfies these criteria.

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The only missing piece to this story is the third criterion: evidence that curcumin is effective in treating AD in humans. This is currently under intense investigation, and the prospects are promising. The results should be available in the next few years.

