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In The **News**

SAME works when other anti-depressants fail

In a study published in the journal *Movement Disorders* (Nov 15, 2000), s-adenosyl-methionine (SAME) was administered to 13 depressed patients with Parkinson's disease. All patients had been previously treated with other antidepressant agents and had no significant benefit or had intolerable side effects. SAME was administered in doses of 800 to 3600 mg per day for a period of 10 weeks. Eleven patients completed the study, and 10 had at least a 50% improvement on the 17-point Hamilton Depression Scale. One patient did not improve. Two patients prematurely terminated participation in the study because of increased anxiety. One patient experienced mild nausea, and another two patients developed mild diarrhea, which resolved spontaneously. The mean improvement in depression scores from before to after treatment was approximately 64%.



Although this study was uncontrolled and preliminary, it suggests that SAME is well tolerated and may be a safe and effective alternative to the antidepressant agents currently used in patients with Parkinson's disease. Please note that some of these Parkinson's patients received very high doses of SAME, which could account for the few side effects observed. Previous clinical studies show that doses of 800 to 1600 mg a day of SAME produce remarkable anti-depressant benefits in otherwise healthy people without significant side effects.

Aspirin & Alzheimer's

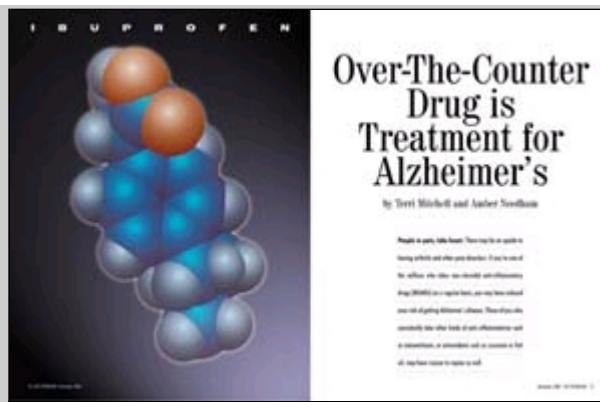
In the November 2000 issue of *Life Extension* we reported that ibuprofen, aspirin and other anti-inflammatories slow the progression of Alzheimer's disease (AD). People who chronically take anti-inflammatory drugs lower their risk of getting AD in the first place. Most studies have looked at people who take non-steroidal anti-inflammatory drugs (NSAIDs) for other conditions such as rheumatoid arthritis. Most of these people are taking moderate to high doses. A new study looks at whether or not lower doses of NSAIDs and other anti-inflammatories also reduce risk. It was found that they do, at least in people over 75. As in previous studies, aspirin seems to work about half as well as NSAIDs such as ibuprofen-but it still can reduce risk about 25%.

High doses are not needed for beneficial effects. Aspirin in doses less than 175 mg/day and NSAIDs in doses less than 500 mg/day provide protection against AD. This is good news for people worried about intestinal bleeding and potential kidney damage (the latter due to high-dose NSAIDs). The very group that is most at risk for AD (over age 75) is also more likely to get serious side effects from anti-inflammatories.

The fact that it only takes low doses of the drugs to get the effects means, also, that anti-inflammatories work by some mechanism other than reducing inflammation. The authors suggest that low levels of anti-inflammatories may work by inhibiting the release of b-amyloid from platelets and/or by inhibiting excess levels of cyclooxygenase (COX).

A surprising finding of the study was the strong showing of ACE-inhibitors. Taking these drugs also appears to lower the risk of Alzheimer's. ACE (which stands for "angiotensin converting enzyme") inhibitors are used to treat heart disease and lower blood pressure.

The study, conducted in Sydney, examined 78 patients with probable AD (as assessed by National Institute of Neurological and Communicative Disorders and Stroke-Alzheimer's Disease and Related Disorders Association criteria). It also looked at 45 people with possible AD and/or vascular dementia, and 40 people with other dementias. The effects of anti-inflammatories held up only for AD, not for dementias of other types. The study did not, however, look at duration. In other words, we don't know how long a person has to take anti-inflammatories to get the beneficial effects. Other studies suggest that a person has to take NSAIDs for years, while aspirin may start working immediately. (Two plus years of NSAID use in the Baltimore Longitudinal Study of Aging reduced the risk 60%). While this question is still up-in-the-air, it seems that the question of whether a person can get anti-Alzheimer's effects by taking low doses of anti-inflammatories has been answered in the affirmative for now.



Anthony JC, et al. 2000. Reduced prevalence of AD in users of NSAIDs and H2 receptor antagonists: the Cache County study. *Neurology* 54:2066-71.

Broe GA, et al. 2000. Anti-inflammatory drugs protect against Alzheimer [sic] disease at low doses. *Arch Neurol* 57:1586-91.

Stewart WF, et al. 1997. Risk of Alzheimer's disease and duration of NSAID use. *Neurology* 48:626-32.

Sugaya K, et al. 2000. New anti-inflammatory treatment strategy in Alzheimer's disease. *Jpn J Pharmacol* 82:85-94.

All in the Genes

Genetics' contribution to the risk of breast, colorectal and prostate cancer is 27%, 35% and 42% respectively, according to a study of 44,788 pairs of twins from Scandinavian registries, published in the *New England Journal of Medicine* last summer. But for most of more than 15 other anatomic sites, the study found little or no genetic contribution to risk.

This study sheds additional light on a controversy that has raged over the genetic vs. environmental contribution to cancer, Robert M. Hoover, M.D., of the National Cancer Institute wrote in an editorial accompanying the article. Most researchers agree that 80% to 90% of human cancer is environmentally caused. The findings, Hoover writes, are consistent with the literature behind this consensus, including studies of certain cancers among immigrant populations that come to match rates in the new homeland after several generations. Nonetheless, the discovery over the last 15 years of the genetic mechanisms underlying cancer has overshadowed environmental explanations in the minds of researchers, doctors, policymakers and the public, according to Hoover.

For cancer researchers, the most intriguing implication is that "there must be major gaps in our understanding of the genetic basis of colorectal, breast and prostate cancer." According to the authors, who were led by Paul Lichtenstein of the Karolinska Institute, Stockholm, the frequency of known high-risk mutations is insufficient to account for observed rates of these cancers in the study. Hoover adds that the nature/nurture debate obscures the fact that ". . . genes and environment interact to produce a risk greater than the sum of their independent effects," which can be reduced by advances in either area. For health-care professionals and individuals who wish to take charge of their health, the message is an optimistic one: genetics is not destiny.



-David Holtzman

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