Alzheimer's Disease

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* Dementia
* Donepezil
* Galantamine
* Naproxen
* Rofecoxib

We examined the efficacy of a vitamin/nutriceutical formulation (folate, vitamin B6, alpha-tocopherol, S-adenosyl methionine, N-acetyl cysteine, and acetyl-L-carnitine) in a 12-month, open-label trial with 14 community-dwelling individuals with early-stage Alzheimer's disease. Participants improved in the Dementia Rating Scale and Clock-drawing tests (Clin 1 and 2). Family caregivers reported improvement in multiple domains of the Neuropsychiatric Inventory (NPI) and maintenance of performance in the Alzheimer's Disease Cooperative Study-Activities of Daily Living (ADL). Sustained performance was reported by caregivers for those participants who continued in an 16-month extension. Performance on the NPI was equivalent to published findings at 3 to 6 months for donepezil and exceeded that of galantamine and their historical placebos. Participants demonstrated superior performance for more than 12 months in NPI and ADL versus those receiving naproxen and rofecoxib or their placebo group. This formulation holds promise for treatment of early-stage Alzheimer's disease prior to and/or as a supplement for pharmacological approaches. A larger, placebo-controlled trial is warranted.

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Testosterone helps protect Neurons - May combat Alzheimer's

August 17, 2001; MONTREAL - A study that found testosterone has a protective effect on human neurons could be the first step toward preventing neurodegenerative diseases such as Alzheimer's through testosterone-replacement therapy, the study's lead clinical investigator said yesterday.

"This is the beginning," said Morrie Gelfand, an obstetrician-gynecologist at the Jewish General Hospital in Montreal and professor of obstetrics and gynecology at McGill University. "Alzheimer's is a big jigsaw puzzle -- these are some of the basic pieces. If cells are being protected by these hormones, that's a very important issue."

Scientists have been investigating for some years the effectiveness of estrogen in preventing Alzheimer's disease in women.
But a McGill-based team of researchers led by Gelfand and molecular biologist Andrea LeBlanc has become the first to show testosterone protects neurons against cell death, suggesting the male sex hormone may also have potential as a preventative therapy for both men and women. The results of the study were recently published in the Journal of Neurochemistry.

Gelfand has long been interested in testosterone replacement therapy, which has been used for more than 30 years in Canada to treat memory loss, depression and anxiety in men -- symptoms linked to the decline of testosterone that comes with age -- and improve energy, well-being and libido in women.

Ongoing research on estrogen as a potential therapy for Alzheimer's disease in women led Gelfand to wonder whether the testosterone he was prescribing in combination with estrogen might have similar potential.

The researchers found that bathing human fetal neuron cells in physiological amounts of testosterone (amounts that were similar to those that naturally occur in the body) eliminated apoptosis, or cell death, for up to 48 hours and significantly inhibited cell death for up to 96 hours.

The next step for Gelfand is to determine how many of the patients he has treated with combination hormone-replacement therapy have developed neurodegenerative diseases. For LeBlanc, it will be further research into the mechanism through which testosterone protects neurons against death.

**Amino Acid Linked to Alzheimer's**

**Folic acid may help forestall the disease**

Jennifer Thomas: HealthScoutNews Reporter

**May 28 (HealthScoutNews) -- Heard of Homocysteine?**

If not, you will soon, health experts say. New research is finding homocysteine (ho-mo-SIS-teen), an amino acid, may play a role in the onset of dementia.

The good news is that vitamins B6, B12 and folic acid may reduce the levels of homocysteine in the blood, says Dr. James Toole, a professor of neurology and public health science at Wake Forest University School of Medicine.

He believes people will soon have their homocysteine levels checked as routinely as cholesterol.

"Researchers have found high homocysteine levels are associated with Alzheimer's disease and brain atrophy," Toole says. "This is big time news."

The latest research appears in today's issue of the journal Neurology. Two studies show that people with elevated levels of homocysteine are more likely to have brain atrophy and vascular disease, which are related to the development of dementia. Alzheimer's disease is one form of dementia.

In one of the studies, researchers tested the blood homocysteine levels of 36 healthy, elderly people, and then used brain scans to measure the amount of brain atrophy, or loss of brain cells and volume.

The study found those who had the highest levels of brain atrophy were twice as likely to have high homocysteine levels as those with less atrophy.

Previous research has shown mild elevations of homocysteine in about 5 percent to 7 percent of the population, says Toole, who wrote an editorial in the journal on the research.
In the second study, researchers did similar tests on 43 people with Alzheimer's and 37 healthy people. They found people with high homocysteine levels were 10 times more likely to have vascular disease.

The study also found Alzheimer's patients were 12 times more likely to have low levels of vitamin B6 than the healthy people.

"The finding will need to be confirmed by other studies, but it is interesting," says Joshua W. Miller, author of the second study and an assistant professor of medical pathology at University of California, Davis Medical Center. "Vitamin B6 has been shown to play a role in brain function and memory, so it's possible that taking B6 supplements could help Alzheimer's patients."

Earlier studies have linked Alzheimer's and elevated homocysteine, Miller says. His research didn't find that same correlation, but in studies that did find a link, the dementia attributed to Alzheimer's could be made worse by vascular disease.

Homocysteine is an amino acid that's formed when the body breaks down methionine, which is found in protein rich foods, Miller says. Previous research has found folic acid counteracts the homocysteine by converting it into a non-toxic form.

However, in people who don't have enough folic acid, the level of homocysteine rises and becomes toxic. Homocysteine is suspected of irritating the lining of blood vessels, accelerating atherosclerosis and contributing to blockages, Toole says.

If folic acid -- also known as folate -- keeps homocysteine down, shouldn't everyone take a supplement?

Not exactly, Toole says.

Folic acid deficiencies have been linked to certain birth defects. In 1998, the U.S. Food and Drug Administration mandated that grain producers fortify their product with folic acid. That means that bread, cereals and pastas -- anything made with grain -- contain added folic acid, Toole says. So, it's very possible you get plenty of folic acid by eating a balanced diet.

Also, no one has established the optimum level of folic acid people need. So theoretically, you could take too much, Toole says.

Vitamins B12 and B6 may also lower homocysteine levels, Miller says.

In addition to grains, citrus fruits, tomatoes and vegetables are good sources of folic acid. You can get vitamin B6 from meat, poultry, fish, fruits, vegetables and grain products. Major sources of B12 include meat, poultry, fish and milk, Toole says.

Toole does recommend that everyone over age 60, or people at high risk of heart and vascular disease, ask to have their homocysteine levels checked by their doctor. This can be done with a simple blood test.

**What To Do**

Read more about homocysteine at familydoctor.org. To learn more about the possible link between homocysteine levels and Alzheimer's, see this National Institutes of Health report.
**SOURCES:** James Toole, M.D., professor, neurology and public health science, Wake Forest University School of Medicine, Winston-Salem, N.C; Joshua W. Miller, Ph.D., assistant professor, medical pathology, University of California, Davis Medical Center; May 28, 2002, Neurology

**DGNews**

New Study Supports Use of Positron Emission Tomography Scans in Early Diagnosis of Alzheimer's Disease

RESTON, VA -- March 12, 2004 -- Early diagnosis of Alzheimer's disease (AD) has become vitally important now that drugs are available that may help slow the otherwise unremitting course of the disease. But an accurate diagnosis is critical when initiating early therapy since some of the most promising treatments have been shown to exacerbate other forms of dementia.

Recent studies have demonstrated that Positron Emission Tomography (PET) scans may be the most accurate method of diagnosing AD, particularly in its early stages. Given the potential benefits of early diagnosis, the issue of insurance coverage for PET diagnosis of AD has become a focal point of discussion among legislators, researchers in the nuclear medicine field, other physicians, and the general public.

A study appearing in the March issue of The Journal of Nuclear Medicine identifies a new PET technique that may increase the already high accuracy of PET in diagnosing AD at a very early stage. Researchers reported that AD-related processes leading to altered brain connections between the entorhinal cortex (EC) and both hemispheres of the brain can be clearly identified with 18F-FDG PET.

The EC is a small area located deep in the brain that plays a central role in memory functions, and is an early site for neuronal damage resulting in memory impairment in AD. The EC is normally connected to other areas of the brain that constitute the so-called "neo-cortex", i.e. the shell of the brain, in both hemispheres.

"This study shows that most of these connections between the hemispheres are destroyed at a very early stage of AD. For example, when brain metabolism is reduced in the right EC, a parallel reduction can be found in the right neocortical areas. Such a pattern of coupled metabolic reductions between the deep and surface brain may make PET even more accurate at differentiating AD from other forms of dementia," said lead author Lisa Mosconi, MD, of the Department of Clinical Pathophysiology Nuclear Medicine Unit of the University of Florence (Italy).

"Our results confirm the importance of studying the connections between the EC and the neocortex to get a more complete picture of the functional alterations that occur very early in AD, and suggest that the clinical picture of AD could be better defined by using FDG-PET measurements" said coauthor Alberto Pupi, MD, also of the Department of Clinical Pathophysiology Nuclear Medicine Unit. f PET for the diagnosis of AD." "This, of course, could help promoting the use of FDG-PET in clinical trials of new drugs to treat AD, which could in turn accelerate the approval process for insurance reimbursement of PET for the diagnosis of AD."

"Vitamin Supplement Use May Reduce Effects Of Alzheimer's Disease"

BETHESDA, MD -- January 20, 2004 -- Antioxidant vitamin supplements, particularly vitamins E and C, may protect the aging brain against damage associated with the pathological changes of Alzheimer's disease, according to a study conducted by the Johns Hopkins Bloomberg School of Public Health and other institutions. The researchers believe antioxidant vitamin supplements may be an ideal prevention strategy for our aging population as they are relatively nontoxic and are thought to have wide-ranging health benefits.
The study, "Reduced Risk of Alzheimer's Disease in Users of Antioxidant Vitamin Supplements" is published in the January 2004, issue of the journal Archives of Neurology. Peter P. Zandi, PhD, lead author of the study and an assistant professor in the School's Department of Mental Health, said, "These results are extremely exciting. Our study suggests that the regular use of vitamin E in nutritional supplement doses, especially in combination with vitamin C, may reduce the risk of developing Alzheimer's disease." The researchers examined data from the Cache County Study, which is a large, population-based investigation of the prevalence and incidence of Alzheimer's disease and other dementias. Residents who were 65 or older were assessed from 1996-1997 and again from 1998-2000. Study participants were asked at their first contact about vitamin usage.

The researchers then compared the subsequent risk of developing Alzheimer's disease over the study interval among supplement users versus nonusers to come to their conclusions. Approximately 17 percent of the study participants reported taking vitamin E or C supplements. These individuals were significantly more likely to be female, younger, better educated and reported better general health when compared to non-supplement users. In addition to those who took vitamin supplements, another 20 percent of study participants used multivitamins, but without a high dosage of vitamin E or C. The researchers found a trend towards reduced Alzheimer's disease with a combination of vitamin E and C supplements, even after controlling for age, sex, education and general health. However, there was no notable reduction in the risk of Alzheimer's disease with vitamin E or vitamin C alone or with multivitamins.

Multivitamins typically contain the recommended daily allowance of vitamin E (22 IU or 15 mg) and vitamin C (75-90 mg), while individual supplements contain doses up to 1,000 IU of vitamin E and 500-1,000 mg or more of vitamin C. The researchers explained that the use of vitamins E and C may offer protection against Alzheimer's disease when taken together in the higher doses available in individual supplements. In addition, there may be some protective effect with vitamin E when it is combined with the lower doses of vitamin C found in multivitamins. Dr. Zandi said, "Further study with randomized prevention trials is needed before drawing firm conclusions about the protective effects of these antioxidants.

Such trials should consider testing a regimen of vitamin E and C in combination. If effective, the use of these antioxidant vitamins may offer an attractive strategy for the prevention of Alzheimer's disease." The study was funded by grants from the National Institutes of Health and National Institute of Mental Health. The Bryan Alzheimer's Disease Center at Duke University completed the APOE genotyping. James C. Anthony, Ara S. Khachaturian, Stephanie V. Stone, Deborah Gustafson, JoAnn T. Tschanz, Maria C. Norton and John C. S. Breitner co-authored the study. SOURCE: Johns Hopkins University Bloomberg School of Public Health

S.P.E.C.T. Brain Scan

Increases Accuracy Of Alzheimer's Diagnosis

April 20, 2001

WESTPORT (Reuters Health) - Single photon emission computed tomography (SPECT) can increase the likelihood of an accurate diagnosis of Alzheimer's disease, according to a report in the April 10th issue of Neurology.

Dr. William Jagust, from the University of California Davis Medical Center, and colleagues prospectively collected clinical data and SPECT images on 70 patients with dementia, who were followed to autopsy. In addition, they collected data on 14 control subject subjects who were followed to autopsy and on 71 controls on whom no autopsies were performed.
Analysis revealed that among all the patients and controls, a clinical diagnosis of probable Alzheimer's disease was associated with an 84% likelihood of pathologic Alzheimer's disease.

Using SPECT imaging, the likelihood of Alzheimer's disease was raised to 92% when the SPECT scan was positive. If the scan was negative the likelihood of Alzheimer's disease was reduced to 70%, Dr. Jagust's group reports.

When the clinical diagnosis was possible Alzheimer's disease, the likelihood of Alzheimer's disease was 84% when SPECT imaging was positive and 52% when it was negative. Without SPECT the likelihood of Alzheimer's disease among these patients was 67%, the investigators note.

The use of SPECT to increase the likelihood of a correct diagnosis of Alzheimer's disease may be essential because "some potential therapeutic options (eg., antiamyloid agents, vaccination, or neural transplantation) might be associated with high risks, necessitating accurate diagnoses," Dr. Jagust and colleagues suggest.


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