Alzheimers.

Hi Sherrie,

I've read some pretty discouraging reviews on Alzheimers Disease lately.

Some experts are claiming that there's not much you can do to prevent it.

They say that exercise, diet, weight loss, Ginkgo Biloba, B vitamins and antioxidants have not been proven to be effective in decreasing the risk or slowing the onset of Alzheimers Disease and other forms of dementia.

That's pretty grim news for those of us who like to be proactive when it comes to protecting our health & well being. It almost makes you say "Why bother?".

But, the problem is that the studies that those experts are referring to were looking at one variable at a time. Some of those studies show benefits, but others don't. So it all seems pretty murky.

But what if you tried a more holistic approach? What if you looked at more than one variable at a time?

Well a group headed by Dr. Nikolaos Scarmeas did just that (JAMA, 302: 627-637, 2009). They looked at the effect of both diet and exercise on the risk of developing Alzheimers.

They enrolled 1880 elderly subjects from Northern Manhattan with an average age of 77 in their study. All of the subjects were healthy and free of dementia at the start of the study.

They found that those subjects who most closely followed a Mediterranean diet were 40% less likely to develop Alzheimers over the next five and a half years than the subjects who were consuming a typical American diet.

And those subjects who were physically very active were 33% less likely to develop Alzheimers than the couch potatoes.

But those subjects who closely followed the Mediterranean diet and were physically active were a whopping 60% less likely to develop Alzheimers than the junk food eating couch potatoes.

In a smaller study the effect of diet alone or exercise alone might not have been large enough to approach statistical significance - which explains the ambiguous results obtained from many of those studies that have focused on only one variable at a time.

But, the effect of combining both diet and exercise is of obvious benefit.

So, if you'd like to prevent Alzheimers and other dementias as you age, my best advice would be to double up - follow a good diet and get plenty of exercise.

But why stop there? Why not try adding Ginkgo Biloba, B vitamins and antioxidant supplements for a triple play?

What the heck - Why not just pile on and aim for ideal body weight as well?

There is some evidence that each of those approaches reduces the risk of Alzheimers.

The evidence may not be good enough to recommend any one of them by itself, but they should all be considered as part of a holistic approach to reducing the risk of developing Alzheimers and other age-related dementias.

To Your Health!
Dr. Stephen G Chaney

Dr. Stephen Chaney
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From a Dr Painter talk

A recent guideline on the pharmaceutical treatment of Alzheimer's dementia was published in the Annals of Internal Medicine (Mar 2008). It reviewed outcomes from 59 different, randomized clinical trials, performed during the last 19 years. The news was not good.

The 5 drugs currently approved by the FDA to treat dementia all failed to provide clinically important improvements for the patients who took them.
It advised providers that: Clinicians must take into account the Adverse effects of these drugs, and balance potential harm against the modest potential benefits.

Dear Sherrie,

Hold on, I'll explain the big words in a moment...

Two new articles about Omega-3 fatty acids and Alzheimer's disease illustrate why it has been so difficult to unambiguously prove the value of diet and supplementation on health outcomes in the past.

The new frontier in nutrition research is called nutrigenomics. For years we have known that individual needs for nutrients vary tremendously, but have not known how to determine what those needs were except by trial and error.

Simply put, nutrigenomics is the application of our ever-expanding understanding of individual differences in the human genome to both nutritional needs and health outcomes.

These new studies illustrate the value of nutrigenomics. First, a bit of background:

A particular gene variant called apoE4 is associated with increased risk of Alzheimer's. However, this is just one of many gene variants that increase the risk of Alzheimer's.

While the apoE4 variant puts people at increased risk for Alzheimer's disease, most people with Alzheimer's disease have acquired it for reasons other than inheriting the apoE4 variant.

This past June researchers at Karolinska Institute and Uppsala University examined whether omega-3 supplementation had any effect on the psychiatric symptoms associated with Alzheimer's disease. Specifically they looked at the agitation and depression often associated with Alzheimer's disease.

When they looked at all patients with Alzheimer's disease, they found no effect of omega-3 supplementation on symptoms of Alzheimer's compared to the placebo group. However, when they separated the patients based on whether or not they carried the apoE4 variant, they found that carriers of the apoE4 gene variant responded positively to the omega-3 fatty acids with regard to agitation symptoms, while non-bearers of the gene variant showed an improvement in depressive symptoms.

A second study reported by French scientists in the November 13th 2007 issue of Neurology found that people who ate fish at least once a week had a 35-percent lower risk of Alzheimer's

disease and 40-percent lower risk of dementia, but only if they did not carry the ApoE4 gene variant.

These kinds of studies represent what I call the "new science" for nutritional studies, whereas studies that simply look at the effect of nutrient intake on Alzheimer's or any other disease as a whole represent "old science".

Both of the studies described above would have concluded that omega-3 fatty acids had no effect on Alzheimer's disease if the scientists had not looked at the benefits of omega-3 fatty acids in people with different genetic backgrounds. That would have been "old science".

But, because they looked specifically at the effect of omega-3 fatty acids on people with different genetic backgrounds, the scientists conducting these studies were able to clearly demonstrate the benefits of omega-3 fatty acids on Alzheimer's. That's "new science".

Nutrigenomic studies of the type I described above will be the "gold standard" of the future. Unfortunately, at present many nutritional studies are still being carried out the old-fashioned way and lack the power to adequately determine the effect of nutrition on disease risks in individuals.

So the next time you see a study saying that a particular nutrient has no effect on some disease, look at the study itself and ask whether it represents "old science" or "new science".

To your health,
Steve Chaney

"My Dad was showing signs of pretty severe Alzheimer's/senility, he was 80 and wasn't actually diagnosed but it was very obvious. He had just retired as a Podiatrist at age 78 and was always very sharp, but shortly got to the point where he couldn't remember from one minute to the next and would get really disoriented. He had to stop driving as he wouldn't remember where he was. One time he couldn't even remember his birthday. Another time, I dropped him off next door where he lived and we took his car over to our house to check it over, and that quickly he was wondering around looking for his car.

I started him on Shaklee Gingko (increases blood supply and activity in the brain) 3 -5 per day (divided in two doses) and 4- 400 IU of Vit E (circulation) per day plus a full supplement program. Within a month- 6 weeks he was AMAZINGLY back to himself. I esp. Would notice how sharp he would get in a few hours after his morning dose of supplements."

If you want the rest of the story, let me know and I will send it to you. We will not be here next until next Monday.

Have a great week, Judy

Article on Hormone – replacement therapy

More bad news for hormone-replacement therapy (HRT). One of the claims had been that HRT might help prevent Alzheimers. This study suggests that even that supposed benefit probably doesn't occur!

Long-Term Estrogen Replacement Therapy In Postmenopausal Women With Alzheimer's Disease May Worsen Memory Loss, Study Suggests

October 28, 2002WASHINGTON (The American Psychological Association) -- Postmenopausal women with Alzheimer's disease who undergo long-term estrogen replacement therapy (ERT) may make their memory loss worse, according to a new study from researchers at the University of Arizona.

The study in the October issue of Behavioral Neuroscience, a journal published by the American Psychological Association (APA), used female rats to study the effect of ERT on memory. The findings are transferable to humans because the conditions reproduced in the study are analogous to that of postmenopausal women who have existing brain inflammation caused by a neurodegenerative illness like Alzheimer's or by head trauma and then choose to undergo long-term ERT.

G. L. Wenk, Ph.D., and colleagues at the Arizona Research Laboratories at the University of Arizona had 40 rats perform a water maze task to look at the interaction of two conditions known to exist within the brains of female Alzheimer's patients, 1) the presence of chronic neuroinflammation, and 2) having too much or not enough estrogen. Both of these conditions are likely to precede the onset of symptoms associated with Alzheimer's. As part of the experiment, some of the rats were ovariectomized (had their

ovaries surgically removed) to mimic the changes seen in postmenopausal women. Aged rats do not undergo an ovarian failure but ovariectomized rats experience both the ovarian failure and the alterations in gene expression within the hypothalamus that appear in women in menopause.

The researchers found that the removal of the rats' ovaries was not enough to impair performance in the water maze task. However, the introduction of either sustained estrogen replacement therapy or chronic brain inflammation did impair memory performance in the ovariectomized rats. Furthermore, the combined occurrence of both conditions (sustained estrogen replacement therapy and longer term brain inflammation) significantly worsened cognitive performance beyond that produced by either condition alone.

"A therapy designed to mimic the natural cycle of hormone fluctuation may provide a more effective therapy to slow the progression of Alzheimer's disease in postmenopausal women," according to the researchers. They add that their findings were confirmed by a 2000 study in the Journal of the American Medical Association (JAMA) involving a long term, placebo-controlled study that examined the effects of estrogen replacement therapy on cognitive function in a large groups of women with mild to moderate Alzheimer's. The effects of ERT were initially beneficial, but the performance of women receiving sustained ERT declined more than that of women receiving the placebo treatment.

"When considered together, the results of this and other clinical trials suggest a pattern of beneficial effects on cognitive function after relatively short-term ERT; however, this beneficial effect is attenuated, and possibly reversed, after much longer treatment regimens," say the authors. "Although a comparison between humans and rodents must be made with caution, it is interesting that continuous long-term estrogen therapy immediately after ovariectomy in the present study parallels the detrimental cognitive effect seen in postmenopausal Alzheimer's disease women who receive continuous, long-term ERT decades after the onset of menopause."

Alzheimer's Disease - Why everyone should take B-Complex!

Here is a very good reason why everyone should be taking Shaklee's B-complex!

According to the Doctor Gott Column, featured in many area newspapers, there "appears to be newly discovered relation between the level of homocysteine in the blood and Alzheimer's disease, as reported in the New England Journal of Medicine (Feb. 14, 2002)

For years, homocysteine has been known to have a direct toxic effect on cells lining the body's arteries. The compound is clearly related to coronary artery disease, stroke, peripheral vascular disease and the aging of the brain.

The new study indicates that the higher the homocysteine level, the more likely a person will develop Alzheimer's. This finding is doubly important because homocysteine blood levels can be significantly reduced by supplemental folic acid, a vitamin, in dosages of one to two milligrams a day. The relation of homocysteine to vascular diseases is so compelling that the United States government now mandates folic acid fortification of the food supply.

High homocysteine levels precede the development of Alzheimer's disease by several years. Therefore, early detection coupled with folic acid therapy could, in theory, prevent the disastrous consequences of
homocysteine-induced vascular disorders. Here is an instance where inexpensive treatment can be truly useful."

Doctor Gott Column
March 25, 2002
Warsaw Times Union

Subject: FW: INCREASING LOW LEVELS OF FOLIC ACID POSSIBLY A KEY FACTOR IN ALZHEIMER'S DISEASE PREVENTION (Low levels of Folic Acid may also contribute to Parkinson's Disease)

Dear friends,
This is valuable information worth passing along from the National Institute of Health....
PLEASE READ

SHAKLEE'S B-COMPLEX IS AN EXCELLENT SOURCE OF FOLIC ACID AND THE VITA LEA ALSO SUPPLIES YOUR RDA.
Please read on... and notify us if you would like more information.
----- Original Message -----
From: Linda <mailto:lindasarto@mindspring.com> Sartore

Subject: FOLIC ACID POSSIBLY A KEY FACTOR IN ALZHEIMER'S DISEASE PREVENTION

NATIONAL INSTITUTES OF HEALTH
National Institute on Aging
NIH NEWS RELEASE

FOLIC ACID POSSIBLY A KEY FACTOR IN ALZHEIMER'S DISEASE PREVENTION

Mouse experiments suggest that folic acid could play an essential role in protecting the brain against the ravages of Alzheimer's disease and other neurodegenerative disorders, according to scientists at the National Institute on Aging. This animal study* could help researchers unravel the underlying biochemical mechanisms involved in another recent finding that concluded people with high blood levels of homocysteine have nearly twice the risk of developing the disease.**

In the study, published in the March 1, 2002 issue of the "Journal of Neuroscience", the investigators fed one group of mice with Alzheimer's-like plaques in their brains a diet that included normal amounts of folate, while a second group was fed a diet deficient in this vitamin. These mice are transgenic, meaning they were bred with mutant genes that cause AD in people. They develop AD-like plaques in their brains that kill neurons.

The NIA team counted neurons in the hippocampus, a brain region critical for learning and memory that is destroyed as plaques accumulate during Alzheimer's disease. The investigators found a decreased number of neurons in the mice fed the folic acid deficient diet.

The scientists also discovered that mice with low amounts of dietary folic acid had elevated levels of homocysteine, an amino acid, in the blood and brain. They suspect that increased

levels of homocysteine in the brain caused damage to the DNA of nerve cells in the hippocampus. In transgenic mice fed an adequate amount of folate, nerve cells in this brain region were able to repair damage to their DNA. But in the transgenic mice fed a folate-deficient diet, nerve cells were unable to repair this DNA damage.

"These new findings establish a possible cause-effect relationship between elevated homocysteine levels and degeneration of nerve cells involved in learning and memory in a mouse model of Alzheimer's disease," said Mark Mattson, Ph.D., chief of the NIA's Laboratory of Neurosciences and the study's principal investigator.

People who have Alzheimer's disease often have low levels of folic acid in their blood, but it is not clear whether this is a result of the disease or if they are simply malnourished due to their illness. But based on emerging research, Dr. Mattson speculates consuming adequate amounts of folic acid -- either in the diet or by supplementation -- could be beneficial to the aging brain and help protect it against Alzheimer's and other neurodegenerative diseases.

Green leafy vegetables, citrus fruits and juices, whole wheat bread and dry beans are good sources of the vitamin. Since 1998, the Food and Drug Administration has required the addition of folic acid to enriched breads, cereals, flours, corn meals, pastas, rice, and other grain products. However, because it can take a long time for the symptoms of Alzheimer's disease to surface, researchers speculate it will be many years before folate supplementation in food could affect the incidence of dementia in the United States. A human clinical trial is being planned.

In AD, plaques develop first in areas of the brain used for memory and other cognitive functions. They consist of largely insoluble deposits of a protein called beta- amyloid. Although researchers still do not know whether amyloid plaques themselves cause AD or whether they are by- products of the AD process, there is evidence that amyloid deposition may be a central process in the disease. But unlike human brain cells, the brain cells in laboratory mice are not killed by the progressive accumulation of beta amyloid. This finding led Dr. Mattson and his research team to suspect folic acid or some other component of the mouse diet might help these nerve cells resist beta amyloid damage. In earlier work, Dr. Mattson found evidence suggesting folic acid deficiency can increase the brain's susceptibility to Parkinson's disease.

The NIA leads the Federal effort to support and conduct basic, clinical, and social and behavioral studies on aging and AD. It supports the Alzheimer's Disease Education and Referral (ADEAR) Center, which provides information on AD research, including clinical trials, to the public, health professionals, and the media. ADEAR can be contacted toll free at 1-800-438-

4380 weekdays or by visiting the website www.alzheimers.org. Press releases, fact sheets, and other materials about aging and aging research can be viewed at the NIA's general information website, www.nia.nih.gov.

New Research Connects Mercury to Alzheimer's Disease Scientists Connect Alzheimer's Disease To Mercury

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Research Conducted at the University of Calgary Faculty of Medicine has demonstrated that trace amounts of mercury can cause the type of damage to nerves that is characteristic of the damage found in Alzheimer's Disease. The level of mercury exposure is consistent with those levels found in humans with mercury / silver amalgam dental fillings. The exposure to mercury caused the formation of "neurofibrillar tangles", which are one of the two diagnostic markers for Alzheimer's Disease. The scientists found that other metals, including aluminum, did not cause the damage. Previous research has shown that mercury can cause the formation of the other Alzheimer's Disease diagnostic marker, "amyloid plaques."

The research, published in a peer-reviewed medical journal, is accompanied by video visual presentation of the effect. Utilizing digital time-lapse photography, this video shows rapid damage to the nerve cells after introduction of minute amounts of mercury. Funding for this video was provided by the International Academy of Oral Medicine and Toxicology (IAOMT).

Alzheimer Disease is Added to the Growing List of Diet-Related Chronic Conditions; Folic Acid is Essential for Health Throughout the Lifespan, says the ASNS/ASCN

BETHESDA, Md., March 23 /PRNewswire/ -- Folic acid, also called folate, has been shown to reduce the risk of disease throughout the lifespan, preventing birth defects, warding off coronary heart disease, stroke, peripheral vascular disease, atherosclerosis, and possibly reducing the risk of breast and colon cancer, dementia and Down syndrome. Recent research provides evidence that folic acid may also help prevent brain degeneration that causes Alzheimer disease.

In a study of elderly Catholic nuns, low serum folate levels in blood samples collected in 1993 was strongly associated with atrophy of the cerebral cortex in women who had a significant number of Alzheimer lesions

in the brain when they died a few years later.

Says Dr. David A. Snowdon, Associate Professor at the University of Kentucky and Director of the Nun Study, ``The goal of the Nun Study is to determine the causes of Alzheimer disease, other brain diseases, and the mental and physical disability associated with old age. Our recent findings suggest that folic acid is important in the development of the human nervous system during pregnancy, and also may also play an important role in maintaining the integrity of the brain in late life.''

Folic acid is a B vitamin found naturally in leafy dark green vegetables like spinach and asparagus, and in beans, peas, legumes, liver, orange juice. In 1998, the United States Food and Drug Administration mandated that folic acid be added to enriched grain products, such as bread and breakfast cereals, in order to increase the folate levels of the population. The current Recommended Dietary Allowance (RDA) for folic acid is 400 micrograms. This level of intake should be adequate to prevent elevated serum homocysteine concentrations, which have been linked to risk of chronic diseases, such as coronary heart disease and Alzheimer disease.

The Nun Study was completed before folic acid fortification became mandatory in the United States. It remains to be seen whether folic acid fortification will result in a lower incidence of Alzheimer disease. Meanwhile, Alzheimer disease should be included in the list of potentially diet-related chronic conditions.

For more information about the Nun Study, visit www.coa.uky.edu/nunnet/