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Coconut Oil and Alzheimer's Disease

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I always suggest that when considering whether or not to start a supplement for AD, it is best to consult with your doctor to ensure that there are no medication interactions, or any untoward effects of the supplement given one's own personal medical history. In addition, it is important to consider whether the data supporting the supplement is anecdotal or supported by results from a randomized, double-blind, placebo controlled trial. This is the best evidence, and the kind of evidence required by the FDA, for any substance to be prescribed for an indication.

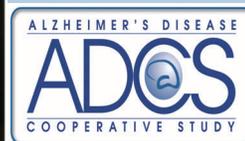
With that said, I would like to discuss the recent attention on coconut oil in the treatment of AD. Coconut oil has medium chain triglycerides, which are a good source of energy, in the form of ketone bodies. Ketones are byproducts of the breakdown of fats in the body; small amounts are normally produced. However, ketone levels rise when you fast (which can lead to a state called ketosis), in response to low glucose intake.

Another way to boost ketones in the body is to consume fats called medium-chain triglycerides (MCTs), of which coconut oil is a good source. MCTs are converted in the liver into ketones, which can be used by the brain as fuel; they are a more immediate source of energy than other fats and are not as readily stored as body fat. Ketones can provide energy to cells without the need for insulin, the hormone the body relies on to get glucose from the blood into cells. The theory behind coconut oil's potential use in AD is that ketones might provide an alternative energy source for brain cells that have lost their ability to use glucose as a result of Alzheimer's disease pathology.

However, there are no studies yet to support this.

In addition, it should be kept in mind that coconut oil is quite high in calories — 115 calories per tablespoon. That can add up when doses are 4 to 8 tablespoons or more a day. Large amounts can also cause diarrhea and other gastrointestinal problems

Unfortunately, there just isn't enough data to support the idea of using coconut oil to treat AD. It is impossible for us to know whether coconut oil has any beneficial effect in Alzheimer's disease until a randomized, double blind clinical trial is conducted.



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HIGH BLOOD CAFFEINE LEVELS IN OLDER ADULTS LINKED TO AVOIDANCE OF ALZHEIMER'S DISEASE

Tampa, FL (June 5, 2012) - Those cups of coffee that you drink every day to keep alert appear to have an extra perk – especially if you're an older adult. A recent study monitoring the memory and thinking processes of people older than 65 found that all those with higher blood caffeine levels avoided the onset of Alzheimer's disease in the two-to-four years of study follow-up. Moreover, coffee appeared to be the major or only source of caffeine for these individuals.

Researchers from the **University of South Florida** and the **University of Miami** say the case control study provides the first direct evidence that caffeine/coffee intake is associated with a reduced risk of dementia or delayed onset. Their findings appear in the online version of an article published June 5 in the ***Journal of Alzheimer's Disease***. The collaborative study involved 124 people, ages 65 to 88, in Tampa and Miami.

"These intriguing results suggest that older adults with mild memory impairment who drink moderate levels of coffee – about 3 cups a day – will not convert to Alzheimer's disease – or at least will experience a substantial delay before converting to Alzheimer's," said study lead author Dr. Chuanhai Cao, a neuroscientist at the **USF College of Pharmacy** and the **USF Health Byrd Alzheimer's Institute**. "The results from this study, along with our earlier studies in Alzheimer's mice, are very consistent in indicating that moderate daily caffeine/coffee intake throughout adulthood should appreciably protect against Alzheimer's disease later in life."

The study shows this protection probably occurs even in older people with early signs of the disease, called mild cognitive impairment, or MCI. Patients with MCI already experience some short-term memory loss and initial Alzheimer's pathology in their brains. Each year, about 15 percent of MCI patients progress to full-blown Alzheimer's disease. The researchers focused on study participants with MCI, because many were destined to develop Alzheimer's within a few years.

Blood caffeine levels at the study's onset were substantially lower (51 percent less) in participants diagnosed with MCI who progressed to dementia during the two-to-four year follow-up than in those whose mild cognitive impairment remained stable over the same period.

No one with MCI who later developed Alzheimer's had initial blood caffeine levels above a critical level of 1200 ng/ml – equivalent to drinking several cups of coffee a few hours before the blood sample was drawn. In contrast, many with stable MCI had blood caffeine levels higher than this critical level.

"We found that 100 percent of the MCI patients with plasma caffeine levels above the critical level experienced no conversion to Alzheimer's disease during the two-to-four year follow-up period," said study co-author Dr. Gary Arendash.

The researchers believe higher blood caffeine levels indicate habitually higher caffeine intake, most probably through coffee. Caffeinated coffee appeared to be the main, if not exclusive, source of caffeine in the memory-protected MCI patients, because they had the same profile of blood immune markers as Alzheimer's mice given caffeinated coffee. Alzheimer's mice given caffeine alone or decaffeinated coffee had a very different immune marker profile.

Since 2006, USF's Dr. Cao and Dr. Arendash have published several studies investigating the effects of caffeine/coffee administered to Alzheimer's mice. Most recently, they reported that caffeine interacts with a yet unidentified component of coffee to boost blood levels of a critical growth factor that seems to fight off the Alzheimer's disease process.

"We are not saying that moderate coffee consumption will completely protect people from Alzheimer's disease," Dr. Cao cautioned. "However, we firmly believe that moderate coffee consumption can appreciably reduce your risk of Alzheimer's or delay its onset." Alzheimer's pathology is a process in which plaques and tangles accumulate in the brain, killing nerve cells, destroying neural connections, and ultimately leading to progressive and irreversible memory loss. Since the neurodegenerative disease starts one or two decades before cognitive de-

Alzheimer’s disease,” Dr. Cao cautioned. “However, we firmly believe that moderate coffee consumption can appreciably reduce your risk of Alzheimer’s or delay its onset.”

Alzheimer’s pathology is a process in which plaques and tangles accumulate in the brain, killing nerve cells, destroying neural connections, and ultimately leading to progressive and irreversible memory loss. Since the neurodegenerative disease starts one or two decades before cognitive decline becomes apparent, the study authors point out, any intervention to cut the risk of Alzheimer’s should ideally begin that far in advance of symptoms.

“Moderate daily consumption of caffeinated coffee appears to be the best dietary option for long-term protection against Alzheimer’s memory loss,” Dr. Arendash said. “Coffee is inexpensive, readily available, easily gets into the brain, and has few side-effects for most of us. Moreover, our studies show that caffeine and coffee appear to directly attack the Alzheimer’s disease process.”

In addition to Alzheimer’s disease, moderate caffeine/coffee intake appears to reduce the risk of several other diseases of aging, including Parkinson’s disease, stroke, Type II diabetes, and breast cancer. However, supporting studies for these benefits have all been observational (uncontrolled), and controlled clinical trials are needed to definitively demonstrate therapeutic value.

A study tracking the health and coffee consumption of more than 400,000 older adults for 13 years, and published earlier this year in the *New England Journal of Medicine*, found that coffee drinkers reduced their risk of dying from heart disease, lung disease, pneumonia, stroke, diabetes, infections, and even injuries and accidents.

With new Alzheimer’s diagnostic guidelines encompassing the full continuum of the disease, approximately 10 million Americans now fall within one of three developmental stages of Alzheimer’s disease — Alzheimer’s disease brain pathology only, MCI, or diagnosed Alzheimer’s disease. That number is expected to climb even higher as the baby-boomer generation continues to enter older age, unless an effective and proven preventive measure is identified.

“If we could conduct a large cohort study to look into the mechanisms of how and why coffee and caffeine can delay or prevent Alzheimer’s disease, it might result in billions of dollars in savings each year in addition to improved quality of life,” Dr. Cao said.

The USF-UM study was funded by the NIH-designated Florida Alzheimer’s Disease Research Center and the State of Florida.

Reprinted courtesy of the University of South Florida.



Reflexes Reveal Changes in the Brain

**By Dr. Gregory Jicha
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New research presented this spring at the American Academy of Neurology indicates that studying reflexes during a neurological exam may prove to be a powerful indicator of subtle changes in memory and cognition. Changes in reflexes are known to appear with the development of Alzheimer's disease, but the idea that reflex testing may actually reveal early signs of cognitive impairment is novel.

Doctors have long known that several reflexes present in infants tend to reappear as one develops Alzheimer's. These primitive/regressive reflexes, also known as frontal release signs, are present at birth and are eventually suppressed by the maturing brain until they disappear. When the brain is injured, as occurs in Alzheimer's, these reflexes re-emerge. In order to test these reflexes, doctors go through what may appear to be a strange routine of tapping foreheads, touching lips and scratching the palms of patients' hands.

Perhaps the best known primitive/regressive reflex is the suckling or rooting reflex. This reflex allows newborns to suckle, gaining nourishment. When the lips are touched, the reflex is triggered. Another response, the glabellar reflex, is tested by tapping between the eyebrows. Most people quickly ignore the tap and stop blinking, while adults with brain injury continue to blink and cannot stop the response. The grasp reflex is an involuntary grasping of anything placed in the hand. This cannot be suppressed either when the brain is immature, or when it has been injured later in life. Finally, the palmomental reflex is observed by scratching the palm of the hand briskly. If the chin muscle contracts, the reflex has been triggered. Doctors have long known that these primitive/regressive reflexes reappear as an individual develops Alzheimer's disease, but now they are asking whether reflex testing may be used as an early diagnostic tool. Our recent work demonstrates that if a cognitively normal person displays two or more of these reflex signs, they tend to perform more poorly on tests of attention, memory and higher order thinking than do those who do not display two or more reflex signs. Looking for these reflexes may serve as one of the best ways to detect early Alzheimer's disease, years before memory tests decline and Alzheimer's disease becomes clinically apparent.

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Once an individual develops Alzheimer's disease, however, these reflexes are not significant in determining the degree of impairment. That can be evaluated through other physical and mental tests.

This represents an exciting development, as early intervention is key in Alzheimer's treatment. Although no cure presently exists, every advance that moves us closer to understanding brain changes in the earliest stages of mental impairment helps us to unravel a bit of the mystery of Alzheimer's. Early detection also provides the best outcomes for individuals with Alzheimer's given our currently available range of treatments.

So, the next time a neurologist begins tapping on your face or scratching at your hands, you'll know that they are checking important reflexes that can be key to understanding your health.

Having a 'Purpose in Life' May Help Shield You From Dementia

MONDAY, May 7 -- If you're looking for a way to keep dementia at bay, a new study suggests you can do so by developing a firm purpose in life.

The findings don't prove that having a purpose will make a difference, and it's possible that the researchers missed another important factor that's at play. Still, the study found that people who had more purpose -- as defined by the researchers -- seemed to be less affected by the brain-clogging gunk that's considered to be a cause of Alzheimer's disease.

"Somehow, having a purpose allows people to cope with the physical signs of Alzheimer's disease," said Patricia Boyle, an associate professor at the Rush Alzheimer's Disease Center at the Rush University Medical Center in Chicago.

Boyle and colleagues looked at tests given to 246 older people who later died and underwent autopsies that explored the state of their brains.

The researchers defined a purpose in life as "the tendency to find meaning from life experience, to be intentional and focused," Boyle said. "It's an indicator of well-being, that life is good and you are contributing to your life, you're making decisions."

To determine purpose in life, the researchers analyzed answers from a 10-item psychological test.

Among those who had a lot of brain gunk -- known as plaques and tangles -- the ones who had greater purpose in life appeared to be less affected by a decline in their mental (or "cognitive") powers. "The rate of cognitive decline was about 30 percent slower for someone with greater purpose in life, compared to someone with less purpose," Boyle said.

The researchers found that they were able to link a higher sense of purpose to better brain health even when they adjusted their statistics so they wouldn't be thrown off by high or low numbers of people with illness, signs of depression and other factors.

It's still not clear that purpose in life has anything to do with mental powers in old age. But if there is a connection, it may have something to do with the brain's capacity, said Dr. James Burke, director of the Memory Disorders Clinic at Duke University Medical Center.

Similarly, people who have more education seem to be better able to tolerate brain-clogging plaques and tangles without having as many cognitive problems, Burke said. "My own analogy is that if a city has more roads, it can tolerate more blocked roads while still allowing you to get to your destination. This is commonly used as the explanation, but difficult to prove."

The study is published in the May issue of the *Archives of General Psychiatry*.

In other Alzheimer's disease news, a small new study indicates that deep brain stimulation -- a treatment being tested to treat mental problems -- seems to help the brain work more efficiently in people who appear to have a mild form of the disease. (The disease can't be conclusively diagnosed until after death.)

The researchers, Gwenn Smith of the Johns Hopkins University School of Medicine and colleagues, examined four men and one woman who underwent the treatment for a year.

In deep brain stimulation, the brain is zapped with an electronic pulse that comes from a pacemaker-like device implanted in the chest.

The study, which was published online May 7 in the *Archives of Neurology*, was very small and "a very early look" at a new kind of treatment, noted Burke, who was not involved in the research. More research is needed, he added.

SOURCES: Patricia A. Boyle, Ph.D., associate professor, Rush Alzheimer's Disease Center, Rush University Medical Center, Chicago; James R. Burke, M.D., Ph.D., director, Memory Disorders Clinic, Duke University Medical Center, Durham, N.C.; May 2012, *Archives of General Psychiatry*; May 7, 2012, *Archives of Neurology*, online

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Guide Dogs for Dementia Care

By [Paula Spencer Scott](#), Caring.com senior editor

You've heard of guide dogs for the blind and the disabled. How about a trusty Golden Retriever or lab to guide your loved one with dementia? That's the premise being tested in Scotland.

Alzheimer's Scotland and Dogs for the Disabled are working together on a "guide dogs for the mind" experiment, which was conceived by design students at the Glasgow School of Art. The first dogs will be assigned to four couples in Scotland this September. In each couple, one of the pair has mild dementia.

"People in the early stages of dementia are still able to live a relatively normal life, and dogs help to maintain routine," Joyce Gray of Alzheimer's Scotland told [The Independent](#).

The dogs are trained to respond to sound triggers. The sounds prompt them to perform care tasks. For example, a dementia guide dog might wake the person with dementia up in the morning, deliver a bite-proof bag of medicine, or deliver notes to fix a meal, while leading the master to the proper cupboard.

Walking a dog provides both exercise and social benefits, too. In one test, developers found that someone with mild dementia who was walking a dog had far more community engagement, with people smiling at and talking to him than when he walked alone.

[Pet therapy for Alzheimer's patients](#) is an idea that's been around awhile. Animals have been found to lower anxiety and stress, encourage communication, improve mood, and lower blood pressure, for example. [Animals -- even robotic ones, along with furry toys and dolls](#) -- are often used in formal programs for Alzheimer's patients in the later stages. What's different with the dementia guide dogs is that they play an active role in patient care.

What gets trickier: What will happen to the dog-master relationship as Alzheimer's progresses and the person with dementia can no longer read notes, or risks eating the dog food instead of his own. (The test is being conducted with caregiving couples, for now.) But ideally, this innovative idea helps prolong independent living for those who have relatively mild cognitive deficits.

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Researchers Find Possible Role of Autoantibodies in Alzheimer's

Released: 5/22/2012 3:20 PM EDT

Source: University of Medicine and Dentistry of New Jersey (UMDNJ)

Study Also Suggests Autoimmune Component to Alzheimer's

Newswise — STRATFORD, NJ – New research by scientists at the University of Medicine and Dentistry of New Jersey-School of Osteopathic Medicine (UMDNJ-SOM) demonstrates how dying or damaged brain cells release debris into the bloodstream and give rise to specific autoantibodies that appear to be reliable biomarkers for early diagnosis of Alzheimer's and other neurodegenerative diseases. The researchers also identify a key mechanism in the development of Alzheimer's that mirrors a process that is common in such autoimmune disorders as rheumatoid arthritis. The study appears online in the *Journal of Autoimmunity*.

"Our earlier research showed that human blood contains perhaps thousands of autoantibodies for clearing cellular debris, and that some of these autoantibodies can potentially be used to accurately diagnose neurodegenerative diseases like Alzheimer's and Parkinson's," said Robert Nagele, PhD, a professor of medicine at the New Jersey Institute for Successful Aging at UMDNJ-SOM and the study's corresponding author. "Here, we found that the release of damaged proteins from dying neurons triggers the production of specific brain-reactive autoantibodies that are directed against this protein debris, a response similar to that seen in some autoimmune disorders."

The researchers focused on the role of enzymes, called PADs, in citrullination, a process that converts one type of amino acid into another (amino acids are the building blocks of proteins). After examining postmortem human brain tissue from individuals with Alzheimer's disease and healthy controls, the researchers found that neurons located in the area of the brain first affected by Alzheimer's disease accumulate both citrullinated proteins and a PAD enzyme. In addition, they demonstrated that a specific type of protein, PTCD2, which has been shown to be a potent biomarker for Alzheimer's, was present in citrullinated form in the neuron cells of the Alzheimer's disease brain samples.

Their results suggest that when neuron cells die, they release their contents into the fluid that surrounds the brain. The cellular remains then enter the bloodstream and their presence generates the production of specific autoantibodies that target this neuronal debris. This same protein citrullination process has been linked to the development of autoantibodies in rheumatoid arthritis, one of the most common forms of autoimmune disease.

"Our previous studies provided evidence that some of these autoantibodies may be able to return to the brain through breaches in the blood-brain barrier," said lead author Nimish Acharya of the UMDNJ-Graduate School of Biomedical Sciences and the New Jersey Institute for Successful Aging. "Once there, they selectively bind to the surfaces of neurons, disrupting the function of the brain cells and accelerating the accumulation of beta amyloid deposits."

This chronic cycle of protein-debris-generating autoantibodies that can then seep through the blood-brain barrier helps explain the long-term, progressive degeneration that results from Alzheimer's disease."

ADCS Trials Enrolling...

ADNI II Study

The goal of the Alzheimer's Disease Neuroimaging Initiative Study is to learn how to stop the progression of mild cognitive impairment (MCI) and Alzheimer's disease in future generations. Information from the study might, in the future, lead to new treatments.

<http://adcs.org/Studies/ImagineADNI.aspx>.



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