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AVOIDING ALZHEIMER'S DISEASE

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It is frightening to think of living with Alzheimer's disease (**AD**)—a progressive, degenerative disorder affecting brain cells, leading to disruptions in thinking, judgment, memory, personality and behavior. It's also scary that the exact cause is not known and there is no pat treatment or cure for it. Like all forms of dementia, it is not a normal part of aging. [1] Between 2000 and 2010, the death rate from AD in the US increased by 39% from 18.1 to 25.1 per 100,000 people. In 2011, AD became the 6th leading cause of death across all ages, 5th for those aged 65 and older. [2] Previously it was diagnosed mainly in younger people, usually in their 50s (and as young as 30), in which it developed progressively and rapidly to death. It was referred to as "pre-senile dementia." In the late 1990s the diagnosis changed to include almost all age groups. [3] Research has taken many dead-end directions. Hoped-for solutions have not panned out. Many people are misdiagnosed. Autopsies show that about half of the elderly people diagnosed with AD don't really have the disease. [4] In 2011, new criteria was released for diagnosing AD that reflects the growing understanding that brain damage begins years, even decades, before symptoms appear. [5] This means there is time for taking action to help prevent it.

AD is characterized by markers in the brain, including: **Amyloid plaques**, clumps of abnormal proteins and nerve cell fragments that develop in tissues between nerve cells. They are mostly composed of **beta-amyloid**, an excess of otherwise beneficial protein fragments. **Neurofibrillary tangles** are structural abnormalities of twisted, hair-like threads composed mostly of an irregular form of protein referred to as **tau**. These abnormalities spread within nerve networks by moving between linked brain cells (neurons). Though theories abound, scientists don't know what causes these abnormalities. [6] "The key to stopping the killing of neurons is figuring out what causes otherwise innocuous proteins to show their Mr. Hyde side, and discovering why the proteins flock together once they've turned." [7] Most experts agree that AD develops as a result of multiple factors, "through multiple pathways," rather than one cause. [8]

PROPOSED CAUSES: **Genes**. Though a number of gene variants have been accused, four are now favored, especially in apoE (apolipoprotein-E). Approximately 20% of the US population has the apoE genetic variant, yet most of them don't get AD. And most people who get AD don't have the variants. [9,10] If genes are involved, it may be epigenetic—a genetic susceptibility activated by environmental factors including lifestyle. Research shows that any number of things can turn a gene on or off and alter its influence. [11] Normally apoE participates in cholesterol transport and lipoprotein metabolism. This led to the notion that AD is caused by cholesterol. Elevated cholesterol has been linked to AD but not everyone with high cholesterol develops AD. Since cholesterol levels rise when repair is going on in blood vessel walls, couldn't the same be true for brain cells? Cholesterol is essential to brain cells, especially the membranes. ApoE helps to spur nerve cell growth and clear up debris; it can be involved in repair and remodeling of damaged brain cells. Cholesterol shouldn't be blamed simply because it is present. When both parents have AD, children have an increased risk. [12] Is it hereditary or because children share the same environment, eat off the same table, have the same deficiencies, are exposed to the same toxins, develop a similar lifestyle? The incidence of AD is rising fast. This can't be due to a genetic cause since the human genome doesn't change rapidly enough.

Blood vessels. The health of the brain and the cardiovascular system are closely linked. A network of blood vessels must nourish the brain with oxygen and nutrients. The heart needs to pump enough blood through those blood vessels. [1] Studies that focus on cardiovascular risk factors in the development of AD have mixed results. One reason is the endeavor to show that cholesterol is a problem when it is actually needed for brain cell health. Higher total- and LDL-cholesterol have been found in people with AD. [13] But this can indicate increased attempts by the body to repair damage, not cause damage. Some studies link high blood pressure to increased AD risk, but other studies found that chronically low blood pressure ups the risk. [14] Of course, if the normal flow of blood to the brain is reduced or disturbed, the health and function of brain cells are affected. [15] Atherosclerosis and AD are "independent but convergent disease processes"—separate problems that can appear together. [16] Damage to blood vessels in the brain—found in AD—increase risk of brain cell injury. [17]

Glucose and insulin. Reduced insulin and insulin activity in the brain are associated with AD. People with type 2 diabetes have double the risk of developing AD compared to people with normal blood sugar levels. [18] Nearly half of people with AD have impaired glucose tolerance and insulin resistance—pre-diabetes. [19] Insulin-related processes can alter brain cell survival, the expression of genes, and the ability of nerve cells to recover following injury. These processes may influence or be influenced by damage to brain proteins, plaque formation and premature death of brain cells. [20] Unlike other cells, brain cells don't need insulin to absorb glucose; insulin protects brain cells from damaging proteins. The brains of people with AD don't have high blood sugar levels, so diabetes is not necessary to develop insulin resistance in the brain. [18] The brain constitutes only 2% of total body weight, yet it consumes about 20% of total energy. High-energy requirements create reliance on proper function of mitochondria in brain cells. A decline in mitochondrial energy production can severely impact brain health. Unlike muscle and other non-nerve tissues, the brain doesn't store meaningful levels of glucose. It relies on its own production of insulin and insulin-like growth factors for a constant supply of glucose. When these and other related factors decline, it stresses mitochondria, impairs energy metabolism, and leads to brain cell damage. [21] Diets high in refined carbohydrates can inhibit lipid metabolism, reduce production of cholesterol in brain cells and interfere with delivery of essential fatty acids to the brain. This alters the composition of brain cell membranes, decreasing function of proteins such as glucose transporters and the amyloid precursor—which leads to glucose deficits and accumulation of amyloid. [22]

Homocysteine. Elevated blood levels of homocysteine occur in AD, indicating a deficiency of B vitamins, particularly B_{12}, B_6 and the associated folate. [23] Low levels of these nutrients are found in people with AD. [24] Supplying the entire B complex is much more effective than separated or, worse, synthetic versions of only a few. All B vitamins along with their affiliated food factors are essential for brain/ nerve cells.

Inflammation. Severe head trauma, head injury, another serious injury or a major immune-function problem may promote the development of AD. In its early stages, COX-2 levels in the brain greatly increase. Cells produce COX-2 as part of the inflammatory response to injury. Inflammation indicates an attempt to repair insult or injury. [1,25] Inflammation is often construed as a cause of the problem rather than a potential solution. [26] People with AD have elevated markers for *a*-amyloid and RAGE (receptor for advanced glycation end products), a peptide in the blood that attracts cell byproducts and removes them from the body—functions to get rid of wastes and abnormal substances. [27] "[I]t has become clear that the immune system itself may have beneficial effects in nervous system diseases considered neurodegenerative." [28]

Hormones. Low levels of estrogen in women or testosterone in men can adversely affect the brain. But these hormone concentrations are not always low in people with AD. As people age these hormones will lessen, but that doesn't mean AD will develop. Hormone replacement therapy (drugs) did not halt AD progression. [29]

Metals vs minerals. Inorganic minerals can be toxic; they differ from organic food-based minerals. **Aluminum**, found in brains of people with AD, was considered a cause of AD but is now labeled as merely a marker. [1] Corporate interests are involved. Tiny amounts of *organic* aluminum salts are present in natural foods and pass harmlessly out of the body. [30] But *inorganic* aluminum is toxic to the brain. Small amounts are in some antacids, buffered aspirin, antiperspirants, aluminum cans, pans, dental amalgams, cosmetics. Accumulation can be significant. Municipal water treatment can include the addition of aluminum to tanks to make bacteria settle out; inorganic aluminum in water is easily absorbed. Vaccines contain aluminum; injected aluminum is easily absorbed. Exposure to aluminum is aggravated by deficits of organic zinc, silica, calcium, copper and other nutrients. [31,,32,33] Sodium fluoride in water and toothpaste increases aluminum absorption. Commercial salt may contain sodium aluminosilicate. Some processed foods and drugs contain aluminum additives such as sodium aluminum phosphate. [34] Kidney failure patients used to receive dialysis that exposed them to aluminum-containing compounds; they had brain changes the same as in AD. [35] **Copper** toxicity can contribute to brain cell degeneration. **Inorganic** forms are implicated, such as copper leached from copper plumbing—corrosion in pipes depending on age, stagnation time, water quality, pH, type of disinfectants and phosphate inhibitors used to treat water. Organic copper is not a problem; it occurs naturally in foods, is metabolized by the liver and is safe. Inorganic copper bypasses the liver and is not easily handled or excreted. Copper is significantly elevated in people with AD. Japan and most developing countries have low rates of AD; copper plumbing is not used in these areas. Inorganic copper is used in many supplements; organic copper complexes are found in real food supplements. All molecules involved in the brain pathology of AD are binders of copper. **Zinc** deficiency can contribute to the problem; people with AD have very low zinc levels. This

mineral plays important roles—some which protect against brain cell damage. Meat with harmful residues (inorganic copper, dioxins, arsenic, drugs, pesticides) is commonly consumed. In 2008 Mexican authorities rejected a shipment of US beef because it contained copper in excess of their limits. The US allows unregulated residues of copper sulfate as a bactericide/fungicide on meat, fat and meat byproducts. It is also used as a fungicide for growing crops and on raw foods after harvest. [36,37] Toxic metals can denature (unwrap) amino acids, including those in the brain. Inorganic copper reduces the activity of neurotransmitter receptors. Such receptors and connections between brain cells are crucial to memory, learning, controlling behavior and recovering from injury. Inhibition of memory formation at the molecular level by copper has been established but the exact mechanism is not yet totally understood. The AD-affected brain has metal-trafficking fatigue causing redistribution of metals into inappropriate compartments. Fluoride in water combined with chlorine worsens the leaching of lead (another neurotoxin) and copper from brass plumbing fixtures. [38]

Drugs and other toxins. Medications with anticholinergic effects can contribute to memory loss and other mental dysfunctions. These include drugs used for hypertension, heart disease and lung problems; certain antidepressants, incontinence medications, narcotic pain relievers; and over-the-counter drugs for insomnia, poor digestion and antihistamines. The drugs block acetylcholine, a neurotransmitter. [39] Various forms of air pollution including particulates from traffic can impair cognitive function and cause neurodegeneration. [38] Even low levels of mercury exposure produce amyloid plaques and neurofibrillary tangles; enzymes are inhibited as found in AD brain samples. [40] Exposure to lead can result in lowered cognitive function. [41] Repeated exposure to pesticides is associated with increased AD risk. [42] General anesthesia may damage the brain and lead to the onset of AD. Heavy smoking in midlife is linked to an increased risk of AD. [43]

MEDICAL TREATMENTS. So far, no medical treatment slows or stops deterioration of brain cells. A few drugs temporarily slow worsening of symptoms for 6 to 12 months in only about half of patients. [1] Many drugs were tried. Neither non-steroidal anti-inflammatory drugs nor COX-2 inhibitors reduced risk; they may contribute to the AD process. [44] Drugs that prevent acetylcholine breakdown gave just a temporary memory boost. [45] Anti-hypertension drugs only slightly reduced the incidence and progression of AD. Statins to lower cholesterol didn't prevent AD. [46] Antidepressants didn't benefit and increased risk of adverse events. Sedatives and antipsychotics accelerated AD development. [47] Amyloid-*B* peptide-lowering agents didn't slow cognitive decline or loss of activities. [48] Antihistamines failed to show significant benefits. [49] Cholinesterase inhibitor drugs show limited, if any, improvement plus they have serious potential side effects including death. These drugs, similar to some pesticides, worsen brain chemistry. For example, Aricept increases risk of seizures. [50] A clinical trial for a vaccine was stopped early when some volunteers developed symptoms of aseptic meningoencephalitis. After modifications, the vaccine reduced plaques but not tangles, and didn't prevent neurodegeneration. Findings indicate that amyloid *B* in plaque is **not** a cause of AD; plaque is simply a biomarker. [51]

PREVENTIVES. People who get plenty of nutrients and consume a healthful diet are less likely to develop AD. [52] Sherry A Rogers, MD, refers to AD as a "total load disease" because heavy metals like mercury, aluminum and lead plus chemicals like plasticizers as well as deficiencies of a number of nutrients can all contribute to AD development. [53] Even preservatives added to cured meats, bacon and ground beef have been linked to AD. [54] Avoiding toxins and periodically using a detoxification program may lessen total load. Measures should be taken to avoid, reduce or overcome obesity, insulin resistance, metabolic syndrome and diabetes. Poor nutrition increases risk. Healthful diets of real whole foods, quitting tobacco, and exercising both body and brain have been shown to decrease risk by 50% or more. [55,56] Sedentary older adults have the lowest levels of cognitive function and fastest rate of cognitive decline. People over 65 who exercise regularly are less likely to develop AD. People with early AD who engage in regular physical activity are less prone to brain atrophy or shrinkage. [57] Brain-stimulating activities are also important including reading, writing, music, learning a new hobby, doing crosswords, a mentally challenging job, social interactions and the like can help prevent AD. [58,59]

Saturated **fats** have been implicated as a cause, but this is usually when studies bunch both saturated fats and trans fats (partially hydrogenated oils) together. Trans fats can contribute to brain deterioration, but real-food sources of saturated fats and other natural fats are essential to brain cells, particularly their membranes. Trans fats are being eliminated from processed foods but they're often replaced with other damaging contrived fats. Unaltered polyunsaturated and monounsaturated fatty acids can reduce AD risk. [60] Fatty acids make up about

50-60% of the brain's dry weight. Omega-3s, particularly DHA, are the most abundant fatty acids in cell membranes of the brain's gray matter. Getting omega-3s from fish significantly lowers AD risk and can decrease the rate of cognitive decline. Older adults who regularly ate foods rich in omega-3s plus vitamins C, D, E, and the B vitamins scored higher on cognitive tests than those whose diets were low in these nutrients. Frequent consumption of fruit, vegetables, fish, and omega-3-rich oils were shown to decrease AD risk. Fish is also rich in protein and choline, both of which play important roles in brain function. [61] Arachidonic acid (an omega-6 fatty acid) plus DHA improved memory and attention in people with mild cognitive impairment. [62] Consuming high amounts of processed nonfoods, altered or refined fats, and insufficient amounts of natural fats can change the chemistry of the brain. After searching for ways to curb the progression of AD in her husband, Mary T Newport, MD, gave him 2 tablespoons of virgin coconut oil per day as a source of **MCTs** (medium chain triglycerides). Within a few days there was noticeable improvement in his gait, ability to converse, memory, activities and sense of humor. During digestion MCTs are broken down into medium chain fatty acids, some of which are converted into ketones. Nerve cells, including those in the brain, rely on glucose for energy but can also convert ketones into energy. When adequate glucose isn't available—such as during a fast or very-low-carbohydrate diet—the body converts fat into ketones which the brain can use for energy. Another way to boost ketones is to consume MCTs; coconut oil is a rich source. The theory is that ketones may provide an alternative energy source for brain cells that have lost their ability to use glucose as a result of AD. This has not yet been proven and it's not known if MCTs can prevent AD, but the worst it can do is help. [63]

People who consume the most vitamin-**E**-rich foods decrease their risk of AD by 70% compared to those who eat the least. Those who took vitamin E supplements (d-alpha tocopherol, an isolated fraction of vitamin E complex) but did not eat vitamin E-rich foods didn't get the same protection. Foods rich in vitamin E complex include whole grains, egg yolks, nuts, seeds, legumes, avocados, and dark leafy greens. Adding foods rich in vitamin **C** complex gave even more protection. [64] More than 13,000 women aged 70 to 81 were assessed for changes over time in memory and attention. The women who in the past consumed the most vegetables (especially leafy greens and cruciferous types) had significantly less cognitive decline than women who ate the fewest. [65] Fruit and vegetables (apples, blueberries, celery, peppers, beets, others) have protective effects. Beets, for example, help lower homocysteine and improve circulation. Blueberries have immune-supporting, vasoprotective effects and may enhance brain cell function. [66] Vitamin C, flavonoids, carotenoids, folate, B_6, B_{12} and other B vitamins from foods are protective. [67] Some studies showed that eating a Mediterranean-type diet helps protect blood vessels in the brain. This type of diet focuses on fresh rather than processed, whole rather than refined; it is rich in fruits, vegetables, whole grains, legumes, nuts and seeds, olive oil, fish, poultry, moderate amounts of wine and some dairy. It contains minimal amounts of red meat, saturated fat, processed foods and sweets. Red meat and saturated fats are not 'bad,' though commercial types contain a higher amount of toxins than do other foods. People following a natural, whole foods eating plan have a much lower risk of AD than people eating a typical Western (processed nonfoods) diet. [68] Dietary **patterns** rather than individual nutrients are the best way to help ward off AD. Boosting the body's production of nitric oxide (NO) helps relax blood vessels and increase blood flow to get more nutrients to the brain. Lower levels of NO are found in people with AD than in people without cognitive impairment. Dark green leafy vegetables, beets and pomegranate are among the foods that increase NO levels. [69] According to David Perlmutter, MD, genetically engineered grains, gluten sensitivity, other food intolerances, altered fats, and various nutritional deficiencies can all contribute to AD development. [70] Organic butter, olives, unrefined oils, nuts, free-range eggs, pasture-raised meats, ocean fish, avocados and other sources of natural, unfooled-with fats are all good brain foods.

SPECIFIC NUTRIENTS. **Omega-3** fatty acids are beneficial in preventing AD and improving mild to moderate AD. Supplementation with isolated DHA and EPA had minimal or no effect on cognitive decline over time. Food sources (fish, cod liver oil, flaxseeds, walnuts, etc.) have a markedly better effect. Not only do people with AD have lowered levels of DHA in their brains, but the low levels are related to a defect in the liver's ability to convert DHA to the utilizable form that requires a certain protein. People with AD may have lost the ability to make the protein. [71] This may relate to toxic overload that can adversely affect liver function. Acetyl-L-carnitine (**ALC**) in the brain helps turn fat into energy. ALC is structurally related to **acetylcholine**, a neurotransmitter involved in memory and proper brain function. In AD, acetylcholine is not used properly. ALC mimics acetyl-choline function, stabilizes cell membranes and improves energy production. [72] **Choline,** a precursor of acetyl-choline, helps form brain cell membranes and is essential in transmission of nerve impulses. Brains of people

with AD have 40% to 50% lower levels of choline than normal brain tissue. Several amino acids affect the availability of neurotransmitter precursors. Abnormalities in amino acids (including phenylalanine, arginine and tryptophan) are found in persons with AD. [73] Food sources of ALC include red meat, dairy, fish, poultry, wheat, asparagus, avocados, peanuts. Choline is found in liver, egg yolk, cheese, nuts, oatmeal, lecithin. For years phosphatidylserine (**PS**) has been used in Europe to treat dementias and other brain disorders. It may help slow the rate of deterioration of early AD. PS is a complex, fat-soluble phospholipid molecule made by the body. In foods it is found particularly in green leafy vegetables, fish and lecithin. [74] **Alpha-lipoic acid** may slow the progression of AD. It activates a choline enzyme, helps protect neurons against toxins, increases glutathione, stimulates glucose uptake and use, activates compromised cerebral blood flow, and more. [75] Good sources include red meat, organ meats (liver, kidney, heart, etc.), nutritional yeast and quality whole milk.

Numerous **vitamins** are linked to AD. Higher serum vitamin D3 levels are associated with better cognitive performance in people with AD. People with higher levels of vitamin E "components" (tocopherols, tocotrienols) are less likely to develop AD. [76] Vitamin C, beta-carotene, other carotenes (zeaxanthin, lutein, lycopene, *B*-cryptoxanthin, *a*-carotene), vitamin A, superoxide dismutase and glutathione peroxidase all lower risk and are often deficient. Many of these nutrients are considered antioxidants; when people are given separated or synthetic versions in the hope of staving off AD, their risk is not really lowered. After a time (up to two years), serum antioxidant capacity (AOC) has no association with cognitive decline. Antioxidants are not the answer. [77] Real nutrient complexes do far more than act as antioxidants which are thought to protect cells from damaging effects of free radicals (which may be results rather than causes). Nutrients are best obtained from real foods in their natural complex, multi-ingredient form. Vitamin **B complex** is a brain essential. Since homocysteine was connected with AD, B_{12}, B_6 and folate were particularly studied because they reduce homocysteine. These nutrients aid cognitive functioning in people with preclinical AD and may help slow the atrophy of specific brain regions. B_{12} deficiency is a common cause of dementia, but is not well absorbed by the elderly (often due to a deficit of intrinsic factor). B_{12} is found in animal-based foods such as liver, fish and milk products. Folate occurs especially in vegetables, fruit, legumes, whole grains, poultry and shellfish. However, high-doses of B_{12}, B_6 and folic acid as synthetic fractions don't really slow cognitive decline in people with mild to moderate AD even though they have deficiencies. Low levels of folate increase risk of AD 200% and the lower the folate, the more the brain shrinkage, degeneration and permanent amyloid deposits. [78] A deficiency of niacinamide (B_3) is often present in dementia; increasing intake protects against AD development. Niacinamide participates in the production of acetylcholine. People with the lowest niacinamide intake were 80% more likely to be diagnosed with AD than those with the highest intake. Deficiency of thiamine (B_1) is often found in AD. Supplementation with a synthetic isolate has limited benefit. Food sources would no doubt bring about far more benefit. Other B vitamins and nutrients associated with the Bs (like choline and inositol) have favorable effects. [79]

A number of **minerals** can help prevent or slow degeneration of AD. Zinc deficiency may contribute to AD. Zinc plays a key role in creating and holding proteins in their correct shape. When proteins lose their shape, they stop working and can clump together—as in AD. Many enzymes become dysfunctional or non-functional when zinc is displaced or deficient. Excess copper displaces or creates a relative deficit of zinc. Conflicting study results report that copper intake may either increase or decrease the risk of AD; one factor is whether it is inorganic or organic copper. Organic zinc and copper are found in vegetables, legumes, nuts, grains, fruits, avocado, shellfish and meat organs (like liver). Clinical trials have shown that zinc has cognitive benefits and the addition of vitamins A and D further increase plasma zinc concentrations. [80] Maintaining sufficient levels of potassium can help prevent AD. Magnesium depletion (associated with high aluminum in brain cells) may be a factor in the development of AD. Iron, as a part of hemoglobin, is essential for transporting oxygen in the blood. The brain consumes up to 20% of all the body's oxygen. When it doesn't get sufficient amounts, brain cells can deteriorate. Older adults with low levels of iron have a 41% higher risk of dementia. Lithium may reduce the risk of AD and help alleviate symptoms of AD by preventing formation of neurofibrillary tangles. People with AD may lack sufficient silica. Silica in drinking water may reduce the risk of developing AD. [81] Many **herbs** may aid in preventing or slowing progression of AD. Several studies found promising benefits from ginkgo biloba for prevention and may have an impact on cognition and improved circulation for those who already have dementia. [82] Curcumin from turmeric may bind to *B*-amyloid, interfere with plaque formation and degrade existing plaques. [83] Many spices and herbs—cinnamon; lemon balm; saffron; American, Panax and Korean ginsengs; Bacopa; rosemary; lemon balm; sage; green tea plus others—have shown potential benefits. [84]

Many of the causes that contribute to AD development are preventable or correctable. The following supplements may be considered to support brain health and function in an effort to avoid AD:

Just Before Two Meals:
1 Neurotrophin PMG (chew)—nerve cell support
2 Cataplex B (chew)—B vitamins
2 Cataplex G (chew—B vitamins
1 Soybean Lecithin—choline, phosphatidylserine

After Two Meals:
2 SP Green Food—vitamins and minerals
1 Cod Liver Oil—A, D, omega-3s
1 Cataplex C (chew)—vitamin C complex
1 Betafood (chew)—helps increase nitric oxide

With One Meal:
1 Folic Acid-B12 (with instrinsic factor) and 1 SP Ginkgo Synergy

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