

AMY BERGER, MS, CNS, NTP

Foreword by David Perlmutter, MD



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THE

Alzheimer's ANTIDOTE

Using a Low-Carb, High-Fat Diet to
Fight Alzheimer's Disease, Memory Loss,
and Cognitive Decline

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Chelsea Green Publishing
White River Junction, Vermont

The Origins of Alzheimer's and a Strategy to Fight It

From aluminum to pesticides, environmental toxins, and genetically modified foods, several possible causes of Alzheimer's disease (AD) have been put forward, many of which involve potentially harmful substances entering the body from the outside and negatively affecting cognitive function. And many different strategies have been recommended to keep the mind active and healthy, such as crossword puzzles, learning a musical instrument or a new language, or taking up hobbies that encourage the formation of new neural pathways. But what if the true underlying cause of AD is a systemic metabolic problem coming from the inside? If that were the case, then the solution would be a metabolic one—a multifaceted strategy that alters several biochemical pathways in the body and, in particular, restores proper fuel metabolism in the brain—and no amount of word games or memorizing foreign idioms would be likely to have a significant impact. It is important, of course, to keep cognitive function robust and active as we age and to challenge ourselves to keep learning, but to imply that Alzheimer's is mostly a result of letting one's mind get "lazy" is scientifically irresponsible and, frankly, a cop-out. Something else is at work—something that affects cognitive function and neuronal impulse transmission in the brain at the most basic level.

Identifying the fundamental causes of AD is imperative and grows more critical every day. Financial costs for health care related to AD are expected to reach into the trillions of dollars by mid-century, and this economic shock pales in comparison to the emotional toll this debilitating disease exacts from its victims and their loved ones and caregivers.¹ It is also of paramount importance that we uncover the causes of AD because

addressing the problem at its source is the only hope we have of preventing, slowing the progression of, and possibly even reversing this frightening form of neurodegeneration. And because we have not yet been able to address the root cause, the vast majority of pharmaceutical drugs targeting individual symptoms of the condition piecemeal have failed to demonstrate beneficial effects. In fact, some initially promising drugs have actually made the signs and symptoms of AD *worse*.²

A dive into the scientific literature regarding the causes of AD reveals a wealth of information indicating that the condition results from metabolic abnormalities that start outside the brain. These abnormalities affect the entire body, but the signs are often missed—or worse, ignored—until damage to the brain is so deep and widespread that it begins to cause cognitive decline that interferes with everyday living and renders formerly strong, independent, capable people unable to care for themselves.

The research is unambiguous: AD results primarily from a failure of parts of the brain to harness sufficient energy from glucose. As a consequence of this insufficient fueling, neurons in the affected brain regions degrade and degenerate, leading to a loss of communication among them. This breakdown in neuronal communication results in the confusion, memory loss, and behavioral changes characteristic of Alzheimer's disease. The connection between glucose handling, insulin signaling, and AD is so strong that many researchers now refer to AD as “diabetes of the brain,” or “type 3 diabetes.”³ Although type 2 diabetes and AD are closely associated, we must not be fooled into believing that type 2 diabetes causes AD. Many people with type 2 diabetes will never go on to develop AD, and many Alzheimer's patients are not diagnosed diabetics. The relationship between the two is more like that of physiological cousins; that is, they result from the same underlying metabolic disturbances, but they manifest differently depending on which parts of the body are affected. In type 2 diabetes, for example, insulin resistance and disturbed carbohydrate metabolism affect the muscles, organs, and periphery (the rest of the body aside from the brain and central nervous system); in Alzheimer's disease, damage is mostly localized to the brain.

The Role of the Modern Diet

If Alzheimer's is ultimately the result of metabolic disturbances similar to those seen in type 2 diabetes—namely, insulin resistance and hyperinsulinemia (elevated levels of insulin in the bloodstream for extended periods of

time)—then the same causes as are seen in type 2 diabetes are likely to be behind AD. While there are many factors that contribute to dysregulated insulin signaling, one of the most powerful is a diet that is mismatched to basic human physiology.

The pattern of eating that has become the “standard American diet” and that has morphed and spread into the “modern Western diet” in many other parts of the world, is very different from the one on which our human ancestors are theorized to have evolved.⁴ Although the current commonly accepted dietary recommendations from government health agencies and medical organizations are slowly shifting, over a half-century of fear-mongering regarding saturated fats and dietary cholesterol in the modern industrialized world has led to recommendations to consume a diet low in total fat and cholesterol, with an emphasis on carbohydrates—specifically, grains, such as wheat, corn, and rice—as the primary source of calories. The few fats that are recommended are vegetable oils (such as soybean and corn oil), which are high in fragile, easily oxidized polyunsaturated fatty acids; we have been cautioned away from the saturated fats found predominantly in animal foods and tropical plants (such as butter, coconut, and palm oils), which are more chemically stable and better suited for cooking.⁵

The modern industrial diet is also generally lower in phytonutrients and antioxidant-rich dark green and brightly colored vegetables and fruits than the diet our robust, healthy ancestors likely consumed. The majority of the plant foods we now consume are starchy carbohydrate sources, such as wheat, potatoes, and corn. This evolutionarily discordant diet has been linked to conditions as diverse as heart disease, acne, obesity, poor eyesight, polycystic ovarian syndrome (PCOS), and cancer.⁶ When the physiological and biochemical effects of these foods, coupled with a lack of micronutrient-rich vegetables and whole, unprocessed, naturally occurring fats start affecting cognitive function later in life, we can add Alzheimer's disease to the list of conditions likely caused by this dietary derailment.

With epidemics of hypertension, diabetes, heart disease, and metabolic syndrome threatening human health on a global scale, the effects of this highly refined diet so poor in vitamins, minerals, and naturally occurring fats upon the physical body are undeniable. But the physiological insults of this diet don't stop at the boundary that separates the brain from the rest of the body (called the “blood-brain barrier”). The brain is an extremely energy-hungry organ: Although it typically accounts for just 2 percent of

total body weight, the brain uses around 20 percent of the body's glucose and oxygen.⁷ Considering the brain's disproportionate consumption of fuel, anything that interferes with fuel delivery or processing in the brain will have dramatic effects on memory, emotions, behavior, and cognition.

Metabolic syndrome (MetSy) is an especially important piece of this puzzle. MetSy is a conglomeration of markers that indicate the body is improperly handling carbohydrates.⁸ (A person's body responds with abnormally high levels of insulin or blood glucose for a prolonged period upon consumption of starchy and sugary foods.) These markers include abdominal obesity (the apple shape of an enlarged midsection with relatively thinner arms and legs); elevated triglycerides (fats in the blood); elevated numbers of small, dense low-density lipoprotein (LDL) particles; reduced high-density lipoproteins (HDLs); elevated fasting blood glucose and insulin levels; hypertension (high blood pressure); and elevated hemoglobin A1c (a long-term measurement of blood glucose levels).⁹ Many of these conditions go hand in hand with type 2 diabetes, and there is reason to suspect that mild cognitive impairment—the precursor to AD—could well be added to the diseases they lead to.

Most, if not all, of the features of MetSy can be ameliorated by reducing the amount of carbohydrate in the diet.¹⁰ This is because MetSy is the result of long-term insulin resistance secondary to overconsumption of total food—refined carbohydrates, in particular—compounded by the relentless stress of modern life, poor quality and quantity of sleep, and insufficient physical activity, all of which contribute to a breakdown in the body's ability to properly process carbohydrates and other fuels. Other lifestyle and dietary factors beyond carbohydrate intake contribute to insulin resistance and MetSy, but excessive carbohydrate consumption is one of the most powerful drivers.

It is important to note here that being diagnosed with MetSy or type 2 diabetes is not required for a subsequent diagnosis of Alzheimer's disease. (We will explore this in more detail in chapter 2.) Due to genetics, environmental factors, or just the way things play out in the body, cognitive impairment or Alzheimer's disease might be the only observable manifestation of insulin resistance and carbohydrate intolerance. Therefore, even if all the numbers on one's bloodwork are in the "normal" ranges, the possibility of problems with carbohydrate handling and elevated insulin should not be dismissed outright. And it is much more likely that at least some of

the features of MetSy *will* be present when the labwork is evaluated more closely. They might have been present for years, in fact, but the signs were missed because clinicians were looking for them mainly from the perspective of weight loss, heart disease, or diabetes, and not from the perspective of a connection to brain health and cognitive function.

The scientific literature shows that the brain is no more protected from metabolic and environmental assaults than the rest of the body. In fact, there is reason to believe that, due to its high energy demands, accelerated oxygen consumption, high concentration of long-chain polyunsaturated fatty acids (which are susceptible to damage by oxidation), and decreased capacity for regeneration (ability to create new cells), the brain is especially vulnerable to the detrimental effects of the modern diet and lifestyle.

If we look to type 2 diabetes as a model for energy usage in a body that has lost the ability to handle carbohydrates properly, we see that not only can the body no longer be fueled effectively by carbohydrates but also chronically elevated insulin levels prevent the body's other premier fuel sources—fats and ketones—from reaching high enough levels in the bloodstream to sustain the body. People with type 2 diabetes often experience problems with fatigue, chronic pain, and poor energy levels. This is because, despite often (but not always) being overweight, at a cellular level, they're actually starving. The same idea is at work in the Alzheimer's brain: At its heart, AD is a fuel shortage in the brain. It is the result of the widespread starvation and death of neurons secondary to hyperinsulinemia (excessive amounts of insulin in the blood), insulin resistance, and a reduced capacity to metabolize glucose.

What Is the Evidence?

Like any other modern chronic illness, Alzheimer's disease doesn't develop overnight. Measurable and subjective signs and symptoms appear years before a diagnosis is made. Cognitive function declines by degrees. (In fact, as I've said, "mild cognitive impairment" often precedes full-blown Alzheimer's.) What we consider the normal foibles and forgetfulness of older age might well be the earliest signs that the brain is struggling to fuel itself.

One of the primary hallmarks of AD is a reduction in the rate at which the brain uses glucose (called the cerebral metabolic rate of glucose, or CMRglu). Compared to healthy people, AD patients have shown up to 45 percent reductions in CMRglu, with some researchers claiming that this

is the *predominant abnormality* in AD.¹¹ Notably, this reduced fuel usage is localized to regions of the brain involved in memory processing and learning, while areas dedicated to visual and sensorimotor processing are unaffected—meaning that cognitive function is affected, but not a person's ability to walk, see, pick things up, or otherwise move around. Positron emitting tomography (PET) scans of people at risk for developing AD show that this decline begins in younger years, long before symptoms of AD are present, and it seems to be the very first step in a long chain of events whose eventual end is AD. This drop in glucose usage as a triggering factor is particularly insidious because there are no overt signs that the change is occurring. The brain might spend decades compensating for and overcoming this fuel shortage before it has progressed to the point where signs and symptoms become evident. It is noteworthy that subjects tested in younger years are cognitively normal; they show no signs of Alzheimer's disease. Therefore, this slow decline in CMRglu can be seen as a kind of canary in the coal mine—preclinical evidence that something has gone awry long before damage has progressed to the point of overt signs and symptoms.

The decline in brain glucose metabolism can be detected in those at risk (based on genetic type or family history) as young as in their twenties and thirties, decades before noticeable manifestation of AD. This makes dietary and lifestyle interventions a lifelong concern, and not just something to tack onto an Alzheimer's diagnosis at age eighty, in desperation. The brain might be able to compensate for and overcome this suboptimal fuel delivery for years, which allows cognitive function to remain normal. And when cognitive function is normal in individuals in their forties or fifties, there's no reason to seek a PET scan to measure the brain's glucose usage. However, the occasional fuzzy-headedness and "brain fog" we tend to associate with normal aging—*Where did I leave my keys? Don't I have an appointment somewhere on Thursday?*—might be the brain's way of letting us know it is beginning to lose the ability to harness energy from glucose effectively. We can joke about having "senior moments," and we all have times when we walk into a room and forget why we went there, but as these things happen more frequently and in more disturbing ways as we age, they are no laughing matter.

At one time, Alzheimer's disease was flippantly referred to as "old timer's disease," because it typically struck the elderly. Now, however, individuals ever younger are being diagnosed with MCI and AD. No longer is cognitive

impairment limited to those in their twilight years. Moreover, we might expect that a certain degree of memory loss and confusion is normal in people of very advanced age. But what are we to make of things when people in their fifties and sixties—or younger—begin to show the signs and symptoms of cognitive decline?

A decline in cerebral glucose metabolism has obvious ramifications. In the context of a standard diet containing the three main types of fuel sources (called *macronutrients*—proteins, fats, and carbohydrates), glucose (which derives predominantly from carbohydrates) serves as the brain's primary fuel. Therefore, if the brain's ability to use this fuel is compromised, neuronal cells will struggle to perform their functions and might eventually starve. To emphasize again: At its core, AD is the deterioration and death of brain cells via starvation.

Another piece of the puzzle linking AD to chronically elevated insulin levels is what is known as beta-amyloid (A β) plaques in the brain. (We'll cover A β in more detail in chapter 6.) A β plaques are protein fragments that accumulate in the brain, solidify, and interfere with cells' ability to communicate with each other. Aside from the reduced utilization of glucose, these plaques are one of the defining signatures of AD. The appearance of A β protein fragments is a normal process that occurs even in healthy people, but their formation into larger, insoluble masses represents a quintessential feature of AD.¹²

A β is found in healthy human brains, but in AD patients it accumulates far beyond the levels seen in healthy people.¹³ This is noteworthy because, at low levels, the body can easily clear away A β proteins. But at higher levels they coalesce into plaques. Think of it this way: Everyday household trash isn't a problem as long as the sanitation crew comes by regularly to haul it away. But if the sanitation workers go on strike, the trash will accumulate and eventually build up to levels that will make the neighborhood intolerable and unlivable. This is what happens when too much A β builds up in the Alzheimer's brain and isn't cleared away.

If the low levels of A β found in healthy brains don't interfere with cognitive function, then something is causing A β to build up to dangerous levels in AD patients. There are two possible reasons for this: One is that AD patients are producing more of it; the second is that they are producing normal amounts of it, but it is not being broken down and cleared away as it should be—that is, the sanitation crew is on strike. Research indicates it is the latter.

The main way that A β is cleared out is with insulin-degrading enzyme—the same enzyme the body uses to clear away insulin after it has done its job of stopping the liver from releasing stored glucose into the bloodstream (as it does between meals) and helping to move glucose and amino acids out of the bloodstream and into cells. Enzymes are proteins that act as helpers and catalysts to make biochemical reactions happen more quickly and efficiently. I like to think of it this way: Parents of more than one child always claim they don't have a favorite child. Enzymes are not like this; they do choose favorites. In scientific terms, enzymes have higher affinities for certain targets of action (called substrates) than others. Insulin-degrading enzyme has both insulin and A β as its targets, but its affinity for insulin is much higher than for A β . (Insulin is the “favorite child.”) Therefore, when both insulin and A β need to be broken down and cleared away, insulin takes precedence. This means that even when just small amounts of insulin are present, insulin-degrading enzyme (the sanitation crew) will focus its attention on clearing away the insulin, leaving the A β to accumulate.¹⁴

So when insulin levels are chronically elevated—as they often are in people consuming a diet high in refined carbohydrates, particularly when this is combined with being sedentary, chronically sleep deficient, and under a lot of stress (all aspects of the modern diet and lifestyle that can contribute to insulin resistance)—the enzyme is occupied with clearing the insulin, thus allowing the A β to build up and form plaques. This might be one explanation for why the highest risk for Alzheimer's disease is among people of a certain genetic makeup with type 2 diabetes and who are treated with insulin.¹⁵ The higher the amount of insulin in the bloodstream, the more A β will build up, and the more it builds up without being cleared away, the more likely it is to form plaques.

How to Fuel a Struggling Brain

If AD is, at its heart, the result of specific brain regions becoming unable to properly metabolize glucose, coupled with a buildup of amyloid plaques and other neuronal structural changes, secondary to long-term chronically elevated insulin, fatty acid imbalance in the brain, and key micronutrient insufficiencies, then any dietary intervention aimed at improving or preventing this condition should seek to correct the metabolic and structural abnormalities via the following methods: reducing insulin levels; transitioning the body and brain to fuels other than glucose; and providing

a rich supply of protective nutrients; in particular, omega-3 fatty acids, vitamin B₁₂, zinc, and other brain-critical vitamins and minerals.

As a model to guide therapeutic intervention, we can look to what happens during fasting or simple carbohydrate restriction to see how the body sustains itself when it is deprived of glucose in the diet. So if Alzheimer's is ultimately the result of neurons that are starving because they can no longer use glucose properly, then the first and most important step is to provide these neurons with a different source of fuel—one they *can* use.

Glucose Versus Ketones as Fuel for the Brain

The major switch that occurs when the body receives very little carbohydrate is that it switches from running on glucose as its primary fuel to instead using fats, another type of fuel called *ketones*, and small amounts of glucose derived from noncarbohydrate sources.¹⁶ (The latter is a process called *gluconeogenesis*, and we will discuss it in detail in chapter 2.) Ketones are produced when insulin levels are very low. They are by-products of the body breaking down fat—from stored body fat as well as dietary fat in the foods we eat. Ketones themselves also serve as fuel, and the brain is particularly well equipped to thrive on ketones. There are a few different ways to elevate ketone levels, which we will explore in chapter 2, but for now it suffices to know that keeping insulin levels low via dramatically reducing carbohydrate intake is effective for most people.

It is often claimed that glucose is the brain's only fuel, or that the brain requires 120–140 grams of glucose per day. This is untrue and oversimplifies human physiology. Glucose is regularly cited as the “preferred” fuel for the body and brain. However, it is only preferred in the sense that it will generally be used first. It is neither more efficient nor physiologically “safer” than two of the other fuels the body and brain can run on: fats and ketones. In the absence of dietary carbohydrates, ketones can provide as much as 40–60 percent of the brain's energy, thus dramatically reducing the amount of glucose required.¹⁷ Moreover, the brain's remaining requirement for glucose does not automatically imply a need for dietary carbohydrate. The human body is the ultimate reuse and recycle machine; it can convert other substances—such as amino acids (from protein) and glycerol (from fats)—into glucose.

Conventional medicine sometimes contends that ketones are harmful, but this is not the case. They are a completely normal part of human

metabolism that preferentially fuel the brain and central nervous system while the rest of the body runs on fats during times of very low carbohydrate intake.¹⁸ (The benign state of nutritional ketosis achieved via a very low-carbohydrate diet is not the same thing as the acutely dangerous state known as *diabetic ketoacidosis*. This is further clarified in chapter 2.)

The question you might be asking yourself now is, if ketones are such a useful fuel for the brain, and the Alzheimer's brain is struggling to fuel itself, then why doesn't the brain automatically and immediately shift to using ketones instead of glucose? The answer is: A sufficient supply of ketones isn't available. The body doesn't generate high amounts of ketones on a regular basis. Generally speaking, ketone production only occurs when insulin levels are very low. In fact, levels of ketones sufficient to fuel the brain are generally only produced when carbohydrate intake and resulting insulin levels are low enough to flip the metabolic switch that causes the body to make a wholesale shift away from glucose and toward fats as its primary fuel source. Put very simply, the body only generates high amounts of ketones when it needs to—for example, when carbohydrate intake and glucose availability are low enough that the body *must* shift to using a different source of fuel. Therefore, the most effective way to raise blood ketones and begin providing the brain with a fuel it can use properly is to dramatically reduce dietary carbohydrates. Other dietary and lifestyle factors affect insulin levels, and these will be addressed in subsequent chapters, but greatly reducing carbohydrate intake is among the simplest and easiest strategies to implement right off the bat.

People vary widely with regard to their individual level of carbohydrate tolerance and the precise amount of carbohydrate reduction their bodies require in order to make the transition from running mostly on glucose to running mostly on fat and ketones. However, generally speaking, in order for this to happen, carbohydrate intake needs to be much, much lower than it typically is on the starch- and grain-heavy standard American or Western diet.

A Dietary Path out of the Fog

If Alzheimer's disease is, in fact, another of the modern "diseases of civilization" primarily caused by a diet and lifestyle at odds with human physiology, then returning to a diet more congruent with the one on which our species is believed to have evolved is a reasonable starting point in the

battle against this debilitating condition. This might resemble a Paleolithic diet—one made up of relatively high amounts of animal fat and protein; abundant nonstarchy vegetables; and moderate amounts of fruit, nuts, and seeds; and devoid of high-glycemic cereal grains, refined sugars, and chemically manipulated processed foods high in vegetable oils.

This type of diet—combined with appropriate amounts of physical activity, adequate sleep, stress reduction, and exposure to fresh air and daylight in order to support the body's natural circadian rhythm—might help maintain lifelong insulin sensitivity, resulting in vibrant function of the body and brain well into old age. Thus, a physiologically appropriate diet might help to prevent cognitive decline.

However, in order to potentially slow the progression of AD that has already taken hold, or possibly even reverse some of the existing cerebral damage and metabolic derangement observed in AD patients, carbohydrate reduction is a powerful first step. This reduction includes avoiding or greatly limiting otherwise wholesome, unprocessed foods that are high in starch or sugar, such as potatoes, yams, beets, beans, high sugar fruits (such as grapes, bananas, and apples), and other starchy tubers and root vegetables. These foods, which healthy, robust populations have been consuming for millennia, are not detrimental for health, *per se*. I am not suggesting that these foods are not nutritious, nor that they are in any way a cause of disease. Metabolically fit, healthy individuals need not avoid them. But for someone experiencing the ravages of AD or another form of cognitive decline or impairment—someone whose brain has lost the ability to harness sufficient fuel from glucose—providing the body with large amounts of glucose in the form of dietary carbohydrate will likely not be conducive to healing. It is only in the relative absence of dietary carbohydrates—and this includes even the wholesome, nutritious ones—that insulin levels will be low enough for the body to make the shift away from glucose and toward using fats for fuel and will therefore generate enough ketones to provide the brain with nourishment, the severe lack of which is primarily responsible for the signs and symptoms of AD in the first place. The therapeutic and neuroprotective effects of ketones are so impressive, in fact, that one of the premier researchers studying ketones and brain health has suggested that a drawback of the modern, carbohydrate-heavy diet is that it is “keto-deficient.”¹⁹

Very low-carbohydrate ketogenic diets have a long history of efficacy for disorders of the central nervous system, and they seem especially

promising for AD and other neurological conditions.²⁰ If ketones are the brain's primary fuel source under conditions of reduced glucose availability, then AD patients should show improvement in cognitive function on a ketogenic diet or with administration of ketones via an outside source. This has been demonstrated in "gold standard" randomized, double-blind, placebo-controlled studies. Oral administration of ketones has resulted in improved performance on cognition tests compared to placebo.²¹

In a study involving dietary ketosis via a very low-carbohydrate diet (less than 10 percent of total calories coming from carbs) for MCI patients, the low-carbohydrate subjects had better performance on memory tests compared to subjects on a 50 percent carbohydrate diet, with higher scores correlated to higher blood ketone levels.²² (In other words, the higher the level of ketones in the blood, the better the subjects performed on the tests.) A significant reduction in insulin levels was observed for the low-carb group but not for the higher carb group, meaning that the reduced carbohydrate intake was successful at lowering insulin levels, while there was no significant change in insulin in subjects consuming half their total calories as carbohydrates. The authors speculated that the improved memory might have resulted from a combination of the brain's use of ketones and its improved insulin sensitivity, the latter of which might help it use glucose better.

Classical ketogenic diets have been used for almost a century for epilepsy treatment. These classical ketogenic diets call for upward of 80–90 percent of total calories coming from fat. That's quite a departure from the high-carbohydrate diet that has become the norm in the modern Western world. The good news is, something this drastic and difficult to maintain might not be necessary as a nutritional therapy for AD. Classical ketogenic diets restrict carbohydrates as well as proteins, because high-protein intakes might stimulate insulin secretion, which would undermine the purpose of a diet intended to generate an elevated level of ketones and limit the amount of glucose in the bloodstream. (This restriction on the amount of both carbohydrates and proteins explains why a classical ketogenic diet is so high in fat: there are only three macronutrients, so when we limit intake of two of them, only one is left to fill the gap. Calories and nourishment have to come from somewhere, and on a ketogenic diet, with reductions in carbohydrates and proteins, they come mostly from fat, in the form of stored body fat as well as nourishing fats from wholesome foods, such as grass-fed and pastured meats, wild-caught fish, avocados, nuts, and seeds.)

Rather than a very strict ketogenic diet as a dietary strategy for Alzheimer's, simply lowering carbohydrate intake to a point where some ketones are generated and excessive insulin levels are corrected could potentially have positive effects just by easing the metabolic burden on the brain. Of course, individual insulin sensitivity is a factor, as is an individual's ability to generate elevated levels of ketones. Some people's bodies simply generate higher ketone levels more readily than others, but ketone levels would be expected to rise at least somewhat in anyone following a very low-carbohydrate and higher fat diet.

Moreover, unlike a classical ketogenic diet, a very low-carbohydrate diet (which still generates some ketones) allows for consumption of a wider array of low glycemic load vegetables and fruits, which are typically richer in micronutrients, antioxidants, and phytochemicals than refined grains and sugars, which carry a high glycemic index and load and would be prohibited on such a diet.²³ Therefore, a very low-carb diet as a primary avenue for therapy is more practical, since the difficulty with sticking to classical ketogenic diets is that they're extremely restrictive, and some people might find them unpalatable for the long term. The difficulty of staying on a traditional ketogenic diet for an extended period of time might also explain why much of the research involving ketones as therapy for AD is limited to ketone drink mixtures rather than dietary overhauls. (More on these interesting compounds in chapter 2.) There is also likely trepidation on the part of the medical community regarding such a high fat intake—particularly saturated fat—despite mounting evidence that saturated fat intake is *not* associated with increased risk for cardiovascular disease and that reductions in dietary *carbohydrate*, in fact, can improve multiple markers for heart disease.²⁴ This wonderfully promising avenue for research in dietary therapy is being hindered by an outdated nutritional school of thought.

Other Factors: Supplements and Lifestyle

The damage observed in the Alzheimer's brain is complex and multifactorial. Therefore, any intervention intended to delay or possibly reverse this damage should be a multifaceted strategy that addresses the root cause as well as ancillary and downstream effects. The majority of these potentially helpful practices are nutritional in nature, but others are alterations in lifestyle practices. Obviously, the foundation of what might be considered an “anti-Alzheimer's strategy” is a diet very low in carbohydrates and high in

fats and overall nutrient density. Beyond that, there are nutritional supplements that might be beneficial based on their biochemical effects, and there are also lifestyle interventions that might be effective due to their influence on reducing insulin levels, enhancing overall metabolic efficiency in the body, and directly facilitating better cognitive function by stimulating the brain to form new neuronal connections. We will explore each of these in more detail in parts three and four.

The Takeaway: There Is a Solution

Researchers are beginning to amass evidence that the nutritional and lifestyle strategies introduced here and discussed in more detail throughout this book are, in fact, effective for reversing cognitive impairment and Alzheimer's disease. Dale Bredesen, a researcher and physician at the forefront of this research, has developed a multipronged intervention that has yielded extremely promising results.²⁶ While the intervention calls for adjusting multiple biochemical and physiological levers via diet and lifestyle, it should come as no surprise that the foundation of this approach is

AD is not a mysterious, untreatable brain disease—it is a reversible, metabolic/toxic, usually systemic illness with a relatively large window for treatment.

—Dale Bredesen²⁵

a switch to what Dr. Bredesen calls a *lipid-based metabolism*—that is, following a diet that transitions the body from being fueled primarily by glucose to being fueled primarily by fats and ketones.

Some of Dr. Bredesen's patients, whose cognitive function was so severely impaired that they had to leave their professions, are now back at work and leading their normal lives. He has achieved fascinating improvements in patients with mild cognitive impairment as well as full-blown Alzheimer's, and the positive effects were even achieved among individuals who were carriers of the ApoE4 genotype, which is the strongest genetic risk factor for AD. (More on this in chapter 7.)

Dr. Bredesen's program—called MEND, for metabolic enhancement for neurodegeneration—hammers home the point that, with the possible exception of damage caused by physical trauma to the head, skull, or brain, cognitive impairment and dementia are metabolic problems. As such, they

require metabolic therapies. There might come a time when pharmaceutical medications help augment these metabolic therapies, but treating the symptoms piecemeal will never be as effective a solution as addressing the root causes.

Other aspects of Dr. Bredesen's program involve just the sort of lifestyle practices we'll explore in part three: good quantity and quality of sleep, brief periods of fasting, stress management, exercise, restoration of vitamin and mineral sufficiency, and more. That these dietary and lifestyle factors are entirely within our control should give us hope that we can have a positive impact on a disease process for which pharmaceutical treatments developed to date have been so disappointing and ineffective.

4 Lifestyle Tips for a Healthy Brain

Exercise* : *Every little bit helps!*

Exercise isn't just good for the physical body. It's essential for supporting lifelong brain health and cognitive function¹. Exercise helps maintain good blood sugar regulation, and with Alzheimer's disease (AD) now called "type 3 diabetes," controlling insulin & blood glucose levels is one of the most important things you can do to protect your brain². If you're already active, *stay active*, and if you're not engaging in regular physical activity, get started to whatever extent you can: Walking, biking, swimming, golf, gardening, water aerobics, senior stretching—*it all counts!* Find something you enjoy, and keep active regularly. If you are able, *intense* activity and weightlifting are especially beneficial for improving whole-body insulin sensitivity, and energy use *in the brain*. Walking is fantastic: no special equipment required: Just put your shoes on, grab a friend, and *go!*



Fasting* : *Our bodies are wired for feast & famine, not feast, feast, feast*

There's a reason many cultures have a tradition of periodic fasting, be it Lent, Passover, Rosh Hashanah, Ramadan, or other observances. Abstaining from certain foods—or from eating altogether—does wonders for the body and brain. People who fast periodically often report feelings of extreme clear-headedness and emotional well-being. Some even feel a sense of "euphoria." They usually attribute this to achieving some kind of spiritual enlightenment, but the truth is far more scientific than that: *it's ketones!* During fasting, insulin levels remain low, and the body shifts to using fat (instead of carbohydrates) as its primary fuel source. A byproduct of this is higher levels of ketones, which are a "superfood" for the brain³. The primary malfunction leading to cognitive impairment and AD is the brain's loss of ability to burn glucose⁴. When ketone levels are higher—achievable through fasting or a low-carbohydrate diet—ketones become the primary fuel for the brain, leading to clearer thinking and sharper memory.



Sleep: *It's an essential nutrient—Vitamin S*

Sleep is when the brain "cleans house." It clears away old, worn out cells and other debris, including the amyloid plaques associated with Alzheimer's⁵. Ben Franklin was right: "Early to bed and early to rise makes a man healthy, wealthy, and wise!" (We can't guarantee the "wealthy" part, but healthy and wise, for sure!) Plus, insufficient sleep results in poor blood sugar & insulin control—factors intimately associated with the pathology of Alzheimer's and cognitive impairment⁶. This isn't rocket science: you already know you think more clearly after a good night's sleep. Think of sleep as "fasting for the brain"—a time for the brain to *rest and reset*.



Stress Reduction & Relaxation: *Cool the flame in the brain*

In their golden years, no one ever looks back and says they wish they'd spent more time at work, or more time stressed out! Stress makes the body's "fight or flight" hormones rise, and these can wreak havoc with blood sugar control. "Brain fog" and fuzzy thinking can be signs of blood sugar highs & lows. Taking time to rest & relax helps you stay off this rollercoaster. Think of stress as setting the brain on fire with inflammation & oxidation. Relaxing calms and cools things down.



*Always check with your physician or other licensed healthcare provider before starting an exercise program or fasting regimen.

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Top 5 Foods for the Brain

Eggs: *With the yolks!*

Eggs are an excellent source of *choline*, a nutrient required for synthesis of acetylcholine—a neurotransmitter involved in memory processing and learning. Eggs are also a fantastic source of cholesterol. Cholesterol? *Yes! This is a good thing!* Cholesterol is an absolutely essential building block for all cell membranes and *the myelin sheath that surrounds and protects neurons*—the cells in the brain that “talk to each other” and enable healthy cognitive function. **Cholesterol is a struggling brain’s best friend!** In fact, *higher* cholesterol levels in older people may be protective against dementia & Alzheimer’s disease.¹ ***Ditch the egg white omelets, folks. These critical nutrients are found only in the yolks!***



Shellfish: *Oysters, mussels, clams, lobster, shrimp*

Shellfish and crustaceans are loaded with vitamin B₁₂. B₁₂ is required for proper formation of the myelin sheath. In fact, B₁₂ deficiency results in memory loss—especially in older folks, who typically consume fewer B₁₂-rich foods.² Shellfish is also a great source of selenium, copper, iron, and other minerals, *especially zinc*. Zinc is an essential cofactor for the enzyme that breaks down the amyloid plaques associated with Alzheimer’s disease.³

Fatty Coldwater Fish: *Salmon, mackerel, sardines*

These marine foods are outstanding sources of EPA and DHA, omega-3 fats that are building blocks for brain cells. Without these essential fats, the brain cannot be structurally sound. Think of them like brick & mortar for building a house: without them, the house falls apart. DHA is required for proper brain function.⁴ And remember: these nutrients are *in the fat!* This means eating the skin! No more boneless, skinless cans of seafood. You *want* the fat in & under the skin.



Organ Meat: *Especially liver*

Forget acai berries, pomegranate juice, and whatever other faddish “miracle fruits” appear on the scene. The tried-and-true superfood of yesteryear—and *today*—is liver! Liver’s nutrient profile is off the charts! This nutritional powerhouse is chock-full of critically important zinc, selenium, copper, iron, vitamin A, and *all* the B-vitamins, *especially B₁₂*. Also a great source of complete protein & brain-boosting cholesterol. Hate liver? You’re not alone! Make pâté, or sneak it into chili & meatloaf. You’ll never know it’s in there! ☺

Coconut Products: *Coconut oil, coconut milk, dried coconut (unsweetened)*

Coconut contains a unique type of fat, called *medium chain triglycerides* (MCTs). These special fats don’t follow the same digestive process as other dietary fats. They are absorbed quickly and provide the body—and *brain!*—with a ready source of energy, known as ketones. One of the problems in Alzheimer’s disease and cognitive impairment is that the brain has lost the ability to fuel itself efficiently with glucose. Ketones are an alternative fuel source for the brain, and they’ve been shown to enhance memory and cognitive function in Alzheimer’s patients.⁵ Again, you *want* the fat. Use *full-fat* coconut milk, NOT light!



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PRAISE FOR

THE Alzheimer's ANTIDOTE

“Magnificent. . . . *The Alzheimer's Antidote* harvests our most highly regarded scientific research to create an empowering, user-friendly game plan that rewrites our health destiny as it relates to the brain. And this is a program for everyone, whether already diagnosed, at high risk, or even if there is no family history of this disease. . . . In these pages are your highly empowering tools that will allow you to gain control over your genetic and cognitive destiny.”

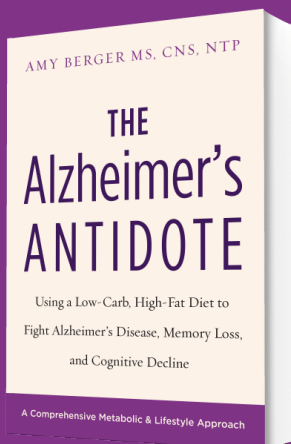
—David Perlmutter, MD, author of *Grain Brain*, #1 *New York Times* bestseller

“There are few things people fear more than cancer, with the possible exception of neurodegenerative diseases such as Alzheimer's disease (AD). Not only does AD ultimately cut lives short, it effectively steals who the person 'is' long before they die. Traditional treatment methods have been lackluster at best, but there is hope. The Alzheimer's Antidote is a scientifically sound method of nutrition and lifestyle that combats AD at a molecular level. If you or someone you know suffers from AD, I highly recommend this book.”

—Rob Wolf, *New York Times* bestselling author of *The Paleo Solution* and *Wired to Eat*

“Amy Berger delves deep into Alzheimer's as a complex metabolic disease, one that can be greatly reduced, and likely avoided completely, with the right combination of lifestyle modifications within our control. Berger offers comprehensive treatment approaches that go way beyond what most patients are told by their physicians. This book is long overdue and a must-read for health care providers and laypeople alike.”

—David M. Brady, ND, CCN, DACBN, author of Amazon bestseller *The Fibro Fix*



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