The ALZHEIMER'S ANTIDOTE

A Comprehensive Nutrition and Lifestyle Strategy to Fight Alzheimer's Disease, Memory Loss and Cognitive Decline

Amy Berger, MS, NTP
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**Appendix A:** The Alzheimer’s Antidote

Appendix A: Recommended Reading

Appendix B: Alzheimer’s Disease as Type 3 Diabetes and the Potential Therapeutic Role of a Reduced-Carbohydrate Diet
Disclaimer

The information contained herein is intended for educational purposes only. It is not intended to replace guidance from a licensed healthcare professional.

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About the Author

Amy Berger, MS, NTP, has a master’s degree in Human Nutrition and is a certified Nutritional Therapy Practitioner. A proud U.S. Air Force veteran, Amy spent years doing what nutrition and health experts claimed were “all the right things” to lose weight and maintain optimal health, but failed to experience the expected results. Wanting to understand why the conventional advice about low-calorie, low-fat dieting and exercise did not lead to the promised outcomes, she began researching physiology and biochemistry and came to learn that much of what we currently believe about “healthy diets” is misguided, and, in many cases, downright incorrect.

Having learned these lessons the hard way, she has dedicated her career to showing others that vibrant health does not require starvation, deprivation, or living at the gym. Men and women cannot live by lettuce alone. Real people need real food!

You can read her blog and find more of her work at http://www.tuitnutrition.com.

Amy would like to thank Gary Taubes for introducing her to the idea that Alzheimer’s disease is linked to insulin and glucose handling problems in the brain. His book, Good Calories, Bad Calories, is where she first learned of this connection, and it inspired her graduate thesis, which was the seed that eventually grew into this book.

Special thanks also to Ellen Davis, MS, for assistance with editing and design. Ellen is the mind behind Ketogenic Diet Resource, the single best one-stop-shop for information and guidance on ketogenic diets for a variety of health concerns.

Amy would also like to thank the researchers, authors, clinicians, physicians, bloggers, and podcasters, who, over the years, have deepened and enriched her knowledge on the subjects discussed herein. You will find links to their work throughout this book, and in Appendix A, Recommended Reading.
Introduction: Why Did I Write This Book?

In the current landscape of conventional medicine and pharmaceutical drugs, a diagnosis of Alzheimer’s disease is essentially a death sentence. Pharmaceutical treatments developed to date have been woefully ineffective, and modern medicine has little else to offer in the fight against this debilitating condition. The best doctors and therapists have to recommend is to keep the mind active, such as by taking up new hobbies or learning foreign languages. To imply that something as devastating as Alzheimer’s disease can be prevented by crosswords and Sudoku puzzles is irresponsible and downright insulting. The lack of progress regarding Alzheimer’s treatment is unacceptable, given the emotional, psychological, and financial tolls this disease exacts from its victims and their caregivers.

Cognitive decline is not inevitable as we age, and if it does occur, we do not have to sit idly by and wait helplessly while it progresses and worsens. Based on the theory of the etiology of Alzheimer’s as outlined in this book, there may be ways to prevent, delay, and possibly even reverse the course of this devastating degenerative disease.

These strategies aren’t being widely discussed because many people don’t know about them. Even many physicians—including neurologists and geriatric specialists, the experts who should be the most knowledgeable on these issues—are unfamiliar with this extremely promising therapeutic avenue. We cannot blame them for this lapse in knowledge, however. The strategies discussed in this book are unconventional, and, in some ways, they’re relatively new. They don’t have decades of “gold standard” randomized, double-blind, placebo-controlled studies backing them up. But as they say in scientific circles, “Absence of evidence does not imply evidence of absence.” The reason we don’t have piles upon piles of scientific evidence proving the efficacy of the methods discussed here is not because they don’t work, but because they’re unconventional, and very few doctors have the courage to step outside the normal standards of care and accepted courses of action to try something different, even though these same-old courses of action will get us the same-old results: namely, no results. No improvement for the Alzheimer’s sufferers, and no relief for their caregivers.

This is heartbreaking—and absolutely unnecessary. A review of the medical literature to date makes a strong case that Alzheimer’s disease is largely a metabolic problem—one whose most strongly affected target is the brain. And if Alzheimer’s disease is a metabolic problem, then the most promising avenue for addressing the root cause of the condition—and therefore potentially slowing and reversing it—is a metabolic solution. Specifically, this relates to a dietary overhaul and
lifestyle modifications as they affect fuel metabolism on a cellular level throughout the body, but in particular, in the brain.

If you have been fighting the ravages of this disease yourself, or if you are a caregiver watching a loved one’s painful transformation into someone unrecognizable, I present this information to you with the sincerest wishes that it helps you reclaim what has been so devastatingly taken from you.

There is hope.

There is a way out of the fog.

Continue reading, come to understand the science and the logic behind the recommendations in this book, have the courage to implement them, and start making your way out, now.

I wish you the best on your journey.

Amy Berger
PART 1: The Metabolic Origins of Alzheimer’s Disease

In this section, we will explore the metabolic origins of Alzheimer’s, and make connections between our modern diet and lifestyle and the development of this condition. We’ll address key factors related to Alzheimer’s, including neuron structure, beta-amyloid plaques, and the ApoE4 genotype. We’ll also explore the logic behind why a low-carbohydrate nutritional plan stands the best chance of stemming the tide on memory loss and cognitive decline.
Chapter 1: The Origins of Alzheimer’s and a Strategy to Fight It

This section will give you a good general overview of the metabolic origins of Alzheimer’s disease, and why a low-carbohydrate diet is recommended as the most logical and promising nutritional strategy for combating this illness. For a more in-depth exploration of the biochemical processes involved and their physiological implications, please refer to Appendix B. That is the full research review upon which this basic summary is based, and it provides more substantial scientific information and addresses additional dietary and physiological factors not covered in this brief overview.

From aluminum to pesticides to genetically modified foods, several possible causes of Alzheimer’s disease (AD) have been put forward, many of which involve potentially toxic substances entering the body from the outside. And many different strategies have been recommended to keep the mind active and healthy: 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 also be a metabolic one, and no amount of word games or memorizing foreign idioms would be likely to help. It is important, of course, to keep cognitive function robust and active as we age, but to imply that Alzheimer’s is mostly a result of letting one’s mind get “lazy” is reprehensible. Something else is at work—something that affects brain function at the most basic level.

Identifying the fundamental causes of AD is imperative and grows more critical every day. Financial costs for AD-related healthcare 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. It is also of primary 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 the condition have failed to demonstrate beneficial effects. In fact, some initially promising drugs have actually made the signs and symptoms of AD worse.

A dive into the scientific literature regarding the causes of AD reveals a wealth of information indicating that the condition is, in fact, a result of metabolic abnormalities that start outside the brain. This affects 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 and live independently.

The research is unambiguous. AD results primarily from a failure of the brain to properly use glucose as a fuel. The connection between glucose handling, insulin signaling, and AD is so strong that many study authors now refer to AD as “diabetes of the brain,” or “type three diabetes.” Although type 2 diabetes and AD are closely associated, we mustn’t be fooled into believing that type 2 diabetes causes AD. Many type 2 diabetics will never go on to develop AD, and many AD 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 imbalances, but manifest differently depending on which parts of the body are affected. In type 2 diabetes, disturbed carbohydrate metabolism affects the muscles, organs, and periphery (the body outside the brain and central nervous system); in AD, damage is mostly localized to the brain.

**The Role of the Modern Diet**

If AD is ultimately the result of a metabolic disturbance similar to that 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 are likely to be behind AD. And the most likely of these causes is a diet that is mismatched to basic human physiology. What has become the “modern Western diet” or “standard American diet” is very different from the one on which our human ancestors are theorized to have evolved. Although the current commonly accepted dietary recommendations from government health agencies and medical organizations are slowly shifting, over sixty years of fear-mongering regarding saturated fats and cholesterol have led to a modern industrialized diet that is low in total fat and cholesterol, with an emphasis on carbohydrates—grains, specifically (wheat, corn, rice, etc.)—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, and we have been cautioned away from the more stable saturated fats from animal foods and tropical plants (such as coconut and palm oils). Our modern diet is also generally lower in phytonutrient- 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 modern conditions as diverse as obesity, heart disease, acne, poor eyesight, polycystic ovarian syndrome (PCOS), and cancer. When the physiological and biochemical effects of these foods, coupled with a lack of micronutrient-rich vegetables and whole, unprocessed, naturally occurring animal 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 obesity, hypertension, diabetes, heart disease, and the 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 the brain typically accounts for just 2% of total body weight, it uses around 20% of the body’s glucose and oxygen. 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 (the ability to comprehend and communicate information).

The metabolic syndrome (MetSy) is an especially important piece of this puzzle. MetSy is a conglomerate of markers that indicate the body is improperly handling carbohydrate. (Meaning that a person’s body responds with abnormally high levels of blood glucose and/or insulin 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 LDL particles; reduced HDL; elevated fasting blood glucose and insulin levels; hypertension (high blood pressure); and elevated hemoglobin A1c (a long-term measurement of blood glucose levels). 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 list.

Most, if not all, of the features of MetSy can be ameliorated by reducing the amount of carbohydrate in the diet. This is because MetSy is the long-term result of insulin resistance secondary to overconsumption of refined carbohydrates, combined with the relentless stress of modern life, and insufficient sleep and physical activity, all of which leads to a breakdown in the body’s ability to process carbohydrates and other fuels. It is important to note here that being diagnosed with metabolic syndrome is not required for a subsequent diagnosis of Alzheimer’s disease. Due to genetics, environmental factors, or just simply the way the chips fall, cognitive impairment and/or Alzheimer’s disease may be the only observable manifestation of carbohydrate intolerance. (Therefore, even if all the numbers on one’s bloodwork are in the “normal” ranges, the possibility of problems with carbohydrate handling should not be dismissed out of hand. 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 may have been present for years, in fact, but the signs were missed because clinicians were looking for them only from the perspective of weight loss, heart disease, or diabetes, and not from the perspective of a connection to brain function and cognitive decline.)

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 brain cells), the brain is especially vulnerable to the detrimental effects of the modern diet.
If we look to type 2 diabetes as a model for energy usage in a body that has lost the ability to properly metabolize carbohydrates, we see that not only can the body no longer be fueled effectively by carbohydrates, but 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. Type-2 diabetics 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 are actually starving. The same idea is at work in the Alzheimer’s brain:

*At its heart, AD is the result of the widespread starvation and death of brain cells secondary to hyperinsulinemia (excessive amounts of insulin in the blood), insulin resistance, and the lost ability to metabolize glucose.*

What is the Evidence?

Alzheimer’s disease doesn’t develop overnight. Both measurable and subjective signs and symptoms appear years before a diagnosis is made. Cognitive function declines by degrees. (In fact, “mild cognitive impairment” usually 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 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% reductions in CMRglu, with some authors claiming that this is *the predominant abnormality* in AD. 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, taste, pick things up, or otherwise move around. Positron Emission Tomography (PET) scans of people at risk for developing AD show that this decline occurs 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. It is noteworthy that subjects tested in younger years are cognitively normal; they show no signs of AD. 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 their twenties and thirties, decades before overt manifestation of AD, which makes dietary intervention a lifelong concern. Because the brain can spend years compensating for the lack of fuel, cognition remains normal in these earlier years, so there’s certainly...
Promising Progress

Researchers are beginning to amass evidence that the nutritional and lifestyle strategies suggested here are, in fact, effective for reversing cognitive impairment and improving Alzheimer’s disease. I encourage you to look at the following account of a multi-pronged approach that includes many of the interventions just discussed: reduced carbohydrate intake, ketogenesis, stress reduction, exercise, elimination of processed foods, and targeted nutritional supplementation, to include omega-3 fats and medium-chain triglycerides:


For additional reading on Alzheimer’s disease as “type 3 diabetes,” as well as low-carbohydrate and/or ketogenic diets as therapy for Alzheimer’s disease, and the negative impacts of high-carbohydrate, low-cholesterol diets upon brain function, you are invited to explore the following resources:

**Scientific papers:**

- **Nutrition and Alzheimer's disease: The detrimental role of a high carbohydrate diet.** Seneff, Wainright, Mascitelli. Eur J Intern Med. 2011 Apr;22(2):134-40. (*Most highly recommended. It is very scientifically detailed, but if you are of a mind to read and understand it, it is extremely educational.)


- **Type 3 diabetes is sporadic Alzheimer's disease: mini-review.** de la Monte SM. Eur Neuropsychopharmacol. 2014 Dec;24(12):1954-60.


Better geared for a lay audience:

• *Alzheimer’s* – The Charlie Foundation

• *Stop Alzheimer's Now,* by Bruce Fife, ND

• *Alzheimer's Disease: What if There Was a Cure?* By Mary Newport, MD

**Sources**


