

# Autism and Diet: Is There a Connection?

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**Autism spectrum disorder is a relatively common developmental brain disorder that typically presents in children under the age of two. Autism is characterized by a broad range of functional brain deficiencies centered in the area of emotional intelligence. There is little doubt that autism has been increasing in all countries, but the question arises why is this so and what can be done about it? Because little is known about the cause of autism, efforts to answer these important questions cause anguish for both medical communities and patients and their families who live with this challenging disorder every day.**

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## BACKGROUND

Autism spectrum disorder is a relatively common developmental brain disorder that typically presents in children under the age of two. Autism is characterized by a broad range of functional brain deficiencies centered in the area of emotional intelligence. There is little doubt that autism has been increasing in all countries,<sup>2,10,11</sup> but the question arises why is this so and what can be done about it? Because little is known about the cause of autism, efforts to answer these important questions cause anguish for both medical communities and patients and their families who live with this challenging disorder every day.

## THE AUTISM-DIET CONNECTION

The possible connection between diet and autism spectrum disorder has been explored over the years.<sup>3-6</sup> Performing controlled studies to show a connection between dietary elements and any disease process is challenging for several reasons. There are an almost endless number of dietary elements that might play a role in triggering autism, and these elements likely act over long periods of time. Biology is extremely complex and it is often challenging to focus on the most potent variables and then to decide on possible mechanisms of how these variables might adversely effect human biology.

This is where empiric or observational science comes into play. The concept of gravity never would have gotten off the ground if someone hadn't noticed that the apple always falls down. Empirical observations guide us on where to spend our efforts when it comes to controlled studies. Fortunately, little research has been done studying apples falling sideways! Patients with autism, their families and the healthcare providers who care for them have often noticed that certain

dietary changes seem to have either positive or negative often anecdotal and inconsistent. To make sense of this confusion, it is important to step back and imagine the various ways that autism and dietary factors might be connected. It is clear that autism is likely caused by a combination of hereditary factors<sup>1</sup> and unknown environmental triggers. There are three scenarios of how autism and diet might be connected and they are not necessarily mutually exclusive:

1. **Autism is at least partially triggered by certain dietary elements.** In this model nutritional factors would be considered primary triggers for some individuals—the disease simply couldn't develop without the presence of these dietary elements.
2. **Certain dietary elements act as secondary triggers of autism.** In this model dietary factors are capable of accelerating autism or making it worse, but these factors are not the primary trigger of the disease.
3. **Certain dietary factors are triggering a completely separate brain disorder that clinically overlaps with autism.** In this model dietary factors appear to be connected to autism, when in fact they are responsible for a separate disorder that shares certain symptoms with autism.

This paper will examine the existing evidence for each of these three possibilities and discuss the empirical observations that might direct our research efforts in the future. It will also discuss common clinical experiences with dietary changes and autism. Now let's examine each of these possible connections separately.

## DIET AS A PRIMARY TRIGGER OF AUTISM

Primary triggers are potent enough to be directly responsible for a majority of cases of a disease in genetically susceptible individuals. For example, cigarette smoking must be considered a primary trigger for most forms of lung cancer.

Although some cases of lung cancer occur in non-smokers, the majority of cases occur in people who have been exposed to cigarette smoke at some time in their lives. At the present time there is little direct evidence that autism is caused by any particular dietary pattern. If indeed a single environmental element were responsible for autism, it likely would have been already discovered. Autism occurs in cultures eating a broad range of different foods.<sup>2</sup> Therefore it seems unlikely that any particular diet will be shown to prevent or effectively treat all cases of autism. It has been suggested that a modern industrial diet may play a role in autism.<sup>3,4</sup> Some believe that autism may be an autoimmune disease.<sup>5</sup> Both gluten and casein have been targeted as possibly dietary triggers of autism in some individuals.<sup>6</sup> Low folic acid intake in the first trimester of pregnancy also appears to be associated with an increased risk of autism. Thus an inadequate intake of foods high in folate or foods fortified with folic acid might play a role in triggering autism in some individuals.<sup>7,8</sup>

### DIET AS A SECONDARY TRIGGER OF AUTISM

Secondary triggers are factors that are known to accelerate a disease process without actually playing a primary role in causing the disease. In the real world it is sometimes difficult to separate primary and secondary triggers. An environmental factor could be considered a secondary trigger if it is absent in many cases of a disease, but when present it seems to accelerate the disease process. Salt is considered a secondary trigger of hypertension. There is little evidence that excessive sodium causes hypertension, but once the disease is established, many studies have shown that a high salt diet accelerates the disease process.

It's important to remember that association between two factors doesn't necessarily mean there is a cause and effect relationship between them. For an element to be considered a true secondary trigger, there must be some evidence that removing the factor slows down or reverses the underlying disease process. Although a low salt diet may not prevent hypertension, it is often useful in controlling the disease along with other treatment modalities.

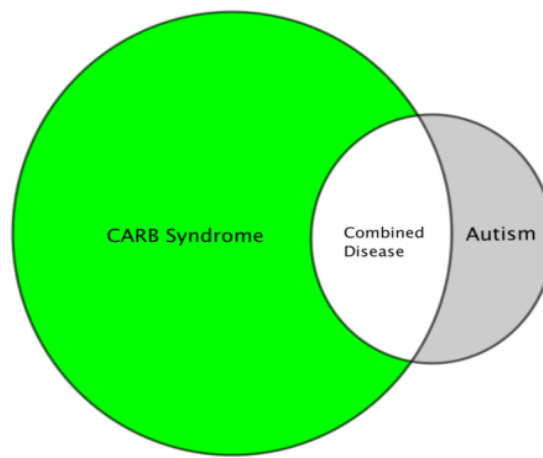
There is some evidence that dietary factors may play a role as secondary triggers of autism.<sup>3,4</sup> There is also a fair amount of empirical evidence of a diet-autism connection. Many patients with autism, their families and the providers who care for them have noted improvements or worsening of the symptoms of autism with the introduction or removal of various dietary elements.<sup>9,10</sup> Although such changes might be consistent in a given patient, it is challenging to identify dietary patterns that are consistent through a broad range of patients with autism. Because each patient with autism is both genotypically and phenotypically a unique individual, it is also likely that different dietary elements will have somewhat varying effects on each individual with the disease. The best that we can hope for is to look for patterns of eating that seem to benefit the majority of autistic patients. In the long run performing controlled studies is the best way to tease out the most potent secondary triggers, but we must

rely on our empirical observations to identify the most promising variables to study.

It's important to remember that we don't fully understand the pathology of most chronic diseases. In an ideal world we would understand the primary and secondary triggers of a disease and use this information to prevent the disease or reverse the pathological process in its early stages before the disease becomes irreversible. At this late stage the only viable option is treatment with medications to control the symptoms of the disease, which is not necessarily the best option for optimal health and functioning.

Consider type II diabetes. Years ago it was discovered that insulin is an effective treatment for diabetes. This discovery occurred after we had already developed a crude model of the pathological process of diabetes. Yet treatment with insulin or medications is far from ideal for patients with type II diabetes. We now understand enough about the pathology of this type of diabetes to prevent the disease in many people or reverse it in the early stages of the pathological process.<sup>11</sup> These exciting new approaches are rooted in making specific dietary changes. Because our current medical model tends to focus on medications rather than lifestyle changes, many patients have yet to benefit from these recent discoveries. Our understanding of autism and similar brain disorders is a long way from our current understanding of chronic diseases like type II diabetes. It's also interesting to note that autism has been tied to insulin signaling.<sup>12</sup>

Based on existing research and many years of clinical experience, it seems likely that there are dietary elements that are acting as secondary triggers of autism,<sup>4,8</sup> yet we have a lot of work ahead of us to identify these secondary triggers so that we can incorporate them into effective treatment protocols.



**Figure 1.** Relationship between CARB syndrome and Autism.

## DIETARY FACTORS DRIVING A SEPARATE DISEASE PROCESS

This area has received the least amount of research attention, yet it provides us with the greatest potential to help improve the lives of patients with autism today rather than some distant future. Is it possible that certain dietary factors are driving a separate brain disease process with symptoms that overlap with autism? If this disease has yet to be identified by the research community and medical profession, it certainly would create a great deal of clinical confusion. It might also help explain why the incidence of autism appears to be increasing at an alarming rate.<sup>13,14</sup> Cases of autism that in the past were subclinical might now appear as full-blown cases because autism is now combined with a disease that shares certain key symptoms. That is why I will focus my attention on this third possible scenario.

To gain insight about this “mystery disease”, we need to go back over a decade and look at the work of Hudson and Pope from McLean Hospital in Boston. In the 1990’s they published a series of papers suggesting that a common pathological process might be driving a group of common brain disorders, including major depression, ADHD, bulimia, cataplexy, dysthymic disorder, fibromyalgia, generalized anxiety disorder, irritable bowel syndrome, migraine, obsessive-compulsive disorder, panic disorder, PTSD, premenstrual dysphoric disorder, and social phobia. They named this condition Affective Spectrum Disorder.<sup>15</sup>

Long before reading their papers, I had also noticed that these and similar brain disorders seem to occur together in many patients. Because at the time I was measuring their body composition and had access to their laboratory studies, I also noticed that most of these patients also seem to have common metabolic problems like insulin resistance, obesity, metabolic syndrome and type II diabetes. As I followed these patients over years or decades, I also noticed that they tended to develop predictable brain dysfunction symptoms that overlap with common heredity conditions such as depression, anxiety disorders, ADHD and similar conditions, yet it seemed to fit the pattern of an entirely new disease. An example might be helpful. Years ago virtually all patients with major depression lost their appetite and lost weight. Weight loss was a defining characteristic of depression. In recent decades many patients with depression seem to have an increased appetite and weight gain.<sup>16</sup> It seems likely that this second group has the disease first described by Hudson and Pope, not true major depression.

Over decades of making empirical observations on thousands of patients, I have refined Hudson and Pope’s model and incorporated common metabolic disorders into the disease model. It appears that this disease is triggered by certain common dietary elements. I propose that the name of this new disease be changed to Carbohydrate Associated Reversible Brain syndrome or CARB syndrome. I have chosen this name because the primary triggers of the disease seem to be excessive fructose mainly from sugar and HFCS and high glycemic carbohydrates, especially from grains. Excessive omega 6 fatty acids from vegetable oils might also

be playing a role in this disease process.<sup>17</sup> Of course this triad of dietary elements describes modern processed foods.

A total of 22 predictable brain dysfunction symptoms<sup>1</sup> seem to be associated with CARB syndrome. Although not every patient has all of these symptoms, as with any disease process they seem to unfold in a predictable manner over time as the disease progresses. Some of these symptoms clearly overlap with some of the key symptoms of autism. Because CARB syndrome appears to be a very common disorder driven by relatively recent radical changes to our modern diet, it is almost inevitable that many patients with autism will also develop CARB syndrome. When they do, their clinical picture becomes much more complex and their ability to function in the world significantly declines. In a sense it’s like adding gas to a fire.

The diagnosis of CARB syndrome is relatively straightforward. Patients suspected of having CARB syndrome are surveyed concerning the 22 symptoms of the disease and virtually all patients will have a least a dozen or more of these symptoms. Early in the disease process only a few symptoms will be present, so it’s important to monitor patients over time for the development of additional symptoms of the disease. All patients with CARB syndrome will have the cardinal symptom of the disease—relatively intense cravings for sweet and starchy foods and beverages. Although such cravings are now fairly common, it is likely that their presence indicates some degree of CARB syndrome.<sup>19</sup> If someone with Autism has these cravings, then it is likely that they have two diseases affecting their brain rather than one. If their CARB syndrome is left untreated, the patient’s metabolic state and brain function will deteriorate over time, greatly interfering with their physical health and brain function, impairing their ability to function in the world.

The good news is that unlike autism, we have a fairly good understanding of the triggers and pathology of CARB syndrome. As the name suggests, the condition is largely reversible with appropriate therapy. Based on my years of clinical experience, the treatments derived from this model are effective at preventing or treating the condition. Because CARB syndrome is as yet an unproven disease model, I only recommend treatments with minimal risks. Although full details of treatment for CARB syndrome are beyond the scope of this article, the basics of treatment are fairly straightforward.

Because the triggers of CARB syndrome seem to be sources of excessive fructose from sugar and HFCS, high glycemic carbohydrates mainly from grains and excessive omega 6 fatty acids from vegetable oils, it makes sense to greatly reduce or eliminate these elements from the diet. To learn more about the metabolic dangers of excessive fructose, Richard Johnson’s books *The Sugar Fix*<sup>20</sup> and *The Fat Switch*<sup>21</sup> are excellent reference materials. A Paleo style diet outlined in Loren Cordain’s book *The Paleo Answer*<sup>22</sup> or Robb Wolf’s book *The Paleo Solution*<sup>23</sup> is anchored in evolutionary biology and eliminates these relatively new

elements of the human diet.

It is also important to pay careful attention to the cardinal symptom of CARB syndrome—cravings for sweet and starchy foods. These cravings have been identified in various brain disorders including depression.<sup>24</sup> If these cravings are left unsuppressed they will push patients to consume more of the very food and beverages that are driving their illness. Certain medications and targeted supplements seem to be effective at suppressing these cravings.<sup>25</sup> The most effective medications are combinations of low dose drugs that target both dopamine/norepinephrine and serotonin. Patients who benefit from these medications will also have other symptoms that suggest inadequate levels of these key monoamine neurotransmitters. Combining low dose bupropion and citalopram is one example of a combination that works well for some patients. There are many other combinations that may work well for selected patients. Fenfluramine, the notorious weight loss drug, was very effective for controlling serotonin deficiency symptoms in many patients even though it was only approved for weight loss, but it was withdrawn from the market because of heart valve problems.

The amino acid L-glutamine when given in doses of 1,000-2,000 mg three times a day between meals can also help suppress these cravings. L-glutamine is very safe and free of side effects. Dr. John Briffa also recommends using L-glutamine for this purpose in his book *Waist Disposal*.<sup>26</sup> Because of their importance in maintaining healthy brain function, taking omega 3 supplements for a total of 1,500-2,500 mg of DHA/EPA daily seems reasonable. This should be combined with an effort to reduce intake of omega 6 fatty acids from vegetable oils. There is some evidence that omega 3 fatty acids might also be helpful for autism.<sup>27</sup>

Because patients with CARB syndrome have symptoms reflecting low levels of monoamine neurotransmitters, using combination supplements containing the precursor amino acids L-tyrosine and 5-htp in a ratio of 10 to 1 can be helpful at relieving these symptoms. Studies have suggested that these precursors when taken as supplements can increase levels of the key monoamine neurotransmitters.<sup>16</sup> Doing so helps to not only relieve cravings for sweet and starchy foods, but also many of the other symptoms of CARB syndrome. These precursors can be safely taken with low dose medications and they seem to enhance the therapeutic effects of the drugs. Patients taking these precursors also seem to avoid drug withdrawal symptoms when the medications are abruptly discontinued.

Research has suggested that many patients with autism have elevated folate receptor autoantibodies leading to reduced levels of brain folic acid.<sup>28</sup> I also recommend measuring a homocysteine level. If it is above 10, I recommend taking a combination of vitamin B6, vitamin B12, trimethylglycine (betaine) and L-methylfolate.<sup>29</sup> L-methylfolate is the only form of folic acid to pass through the blood-brain barrier and it is critical for the formation of monoamine neurotransmitters. L-methylfolate has been approved by the

FDA under the brand name Deplin for the treatment of depression. It is also useful as adjunct therapy for many common brain disorders including autism when they are associated with an elevated homocysteine level.

If a patient presents with both Autism and CARB syndrome, the options for managing Autism are quite limited, but CARB syndrome almost always slowly but steadily responds to these treatments. Metabolic parameters improve, excessive body fat slowly declines and brain dysfunction symptoms gradually improve. When left with just autism, their ability to function both physically and mentally often dramatically improves because they now have only one disease rather than two. Because autism and CARB syndrome seem to feed some of the same brain dysfunction symptoms, these symptoms can be expected to improve when the CARB syndrome is effectively treated.

### SUPPORTING EVIDENCE

Although at this time CARB syndrome is an unproven theoretical model, it does seem to fit well with many of the patients commonly seen in primary care. The model has well defined symptoms and metabolic markers as well as a predictable clinical course. When looking at the relationship between CARB syndrome and autism, it is clear that there are three distinct groups: those with autism without signs of CARB syndrome, those with autism who do appear to have both diseases and a large group of patients who fit the CARB syndrome pattern but do not have autism.

Because both autism and CARB syndrome are defined by symptoms and there is no biological test for either disorder, a precise estimate of the number of patients who have both disorders will await the development of validated criteria for CARB syndrome. Currently efforts are underway to use the 22 symptoms associated with CARB syndrome as the basis for a diagnostic screening questionnaire. Future research based on this type of diagnostic screening should allow us to more precisely differentiate those individuals with autism and CARB syndrome from those who only have autism.

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### CONFLICT OF INTEREST

None.

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