

# Elimination of Autism Symptoms Using the Specific Carbohydrate Diet: Literature Review and Case Report

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## Abstract

Numerous publications have documented the effectiveness of medical nutrition therapy in alleviating several autism symptoms. This case report reviews the literature and highlights the case of a 4-year-old boy with autism and his improvements following the adoption of the Specific Carbohydrate Diet (SCD) protocol, which eliminates sugars (except for honey and fruit), grains, lactose, and processed foods. After following the SCD for 17 months, he experienced the elimination of autism symptoms, and his individualized education program (IEP) was dissolved. Long-term

follow-up and diagnostic re-evaluation with the Autism Diagnostic Observation Schedule (ADOS) confirmed an ADOS score of zero. The young man is now 19 and is studying in college. While this case and the literature to date are promising, further investigation is warranted to understand the relationship between gastrointestinal issues, intestinal permeability, nutritional interventions, and autism spectrum disorders.

**Keywords:** autism, diet, nutrition, specific carbohydrate diet (SCD), gut and psychology syndrome (GAPS), paleo diet, gluten-free, literature review, case report

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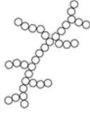
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## Introduction and Literature Review

Autism spectrum disorder (ASD) is a complex developmental disorder. ASD patients often exhibit impairments in social interaction, speech delay, restricted interests, and repetitive behavioral patterns.<sup>1</sup> Increasingly, clinicians and parents are recognizing the importance of the gut-brain connection.<sup>2</sup> A large proportion of children diagnosed with ASD (46–84%) have gastrointestinal (GI) dysfunction, suggested by symptoms including constipation, diarrhea, abdominal pain, and gastroesophageal reflux disease (GERD).<sup>3</sup> GI dysfunctions in children with ASD may be associated with low activity of disaccharidase enzymes;<sup>4</sup> sulfation deficiency in ingested phenolic compounds, including acetaminophen, salicylates (e.g., aspirin), and artificial food colorings;<sup>5,6</sup> generalized reduction in GI microbiome biodiversity leading to bacterial overgrowth;<sup>7,8</sup> and increased intestinal permeability (leaky gut).<sup>9</sup> All of these GI-related dysfunctions may lead to treatable nutritional deficiencies.<sup>10,11</sup>

A 2022 systematic review and meta-analysis found that dietary interventions were associated with significant symptom reduction in ASD patients, and that gluten-free diets were associated with improved social behavior.<sup>12</sup> Research has shown that patients with ASD may have heightened antibody production against gluten.<sup>13</sup> These gluten antibodies may be related to the formation of various nervous system autoantibodies (e.g., transglutaminase 6, synapsin I, and gangliosides).<sup>14</sup> Gluten protein is 70% gliadin, and gliadin peptides have been shown to induce proinflammatory cytokines, including IL-15, IFN- $\gamma$ , IL-6, TNF- $\alpha$ , IL-1 $\beta$ , CCL2, and CCL3.<sup>15</sup> Other published studies have shown that children with ASD following a gluten-free diet exhibit fewer colon cells producing TNF- $\alpha$ , as compared to ASD children eating a gluten-containing diet and producing greater amounts of TNF- $\alpha$ .<sup>16</sup> Autistic children with gastrointestinal complaints like diarrhea and constipation often show abnormal immune reactions to food (non-IgE mediated food hypersensitivities correlated with higher levels of TNF- $\alpha$  and IL-12).<sup>16,17</sup> Numerous case reports, open-label trials, and observational studies have noted behavioral improvements upon eliminating certain foods, and behavioral decline when those foods are reintroduced.<sup>18–22</sup> Studies have also shown improved behavior in ASD children who consume a gluten-free diet.<sup>23,24</sup> The ScanBrit trial, which included 72 participants conducted over 24 months, observed significant improvements after following a gluten-free diet as evaluated with three validated instruments: Gilliam Autism Rating Scale, Autism Diagnostic Observation Schedule (ADOS), and ADHD-IV Rating Scale.<sup>25</sup>

**Table 1. Saccharides**

Legal		Brush Border Glycohydrolase Enzyme Required	Structure
Monosaccharides (e.g., honey, fruits)	Glucose Fructose Galactose	None <sup>37</sup>	
Illegal			
Disaccharides (e.g., cane sugar, maple sugar, beet sugar, malt sugar, milk sugar)	Sucrose Isomaltose	Sucrase- Isomaltase (SI) <sup>38</sup>	
	Maltose	Maltase-Glucoamylase (MGAM) <sup>38</sup>	
	Lactose	Lactase-Phlorizin Hydrolase (LPH) <sup>39</sup>	
Polysaccharides (e.g., starches, grains, corn, rice)	Starch Polymer	Isomaltase (major role) <sup>38</sup>  Glucoamylase (auxiliary role) <sup>34,38,40</sup>	



**Note:** Each circle represents a single sugar molecule. Since monosaccharides are already single sugar molecules, they can be absorbed immediately by the small intestine. Monosaccharides are less likely to feed-up harmful microorganisms, thus are considered legal on the SCD.<sup>35</sup> Some individuals poorly digest disaccharides and polysaccharides, most likely due to damaged/blunted small intestinal villi/microvilli, which ought to be producing brush border digestive enzymes (but are not).<sup>34</sup> This maldigestion allows bacteria and yeast to overgrow as they feed on the unabsorbed complex sugars. These bacteria/yeast further damage the small intestinal villi/microvilli, which further inhibits carbohydrate metabolism, and creates a “vicious cycle” that continues to deteriorate.<sup>34</sup> (Image credit: Liz Paton)

Fasano et al. have shown that the root cause of many diseases is an underlying increase in intestinal permeability (leaky gut), in which large food molecules pass through the intestinal wall before being fully digested.<sup>26</sup> Gliadin peptides (contained in wheat and other grains) have been shown to increase intestinal permeability by upregulating zonulin release in patients with celiac disease and individuals with non-celiac gluten sensitivity (NCGS).<sup>27</sup> When incompletely digested peptides leak through a permeable intestinal epithelium, gluten and other food peptides may trigger an inflammatory IgG immune response.<sup>28</sup> Interestingly, titers of food-specific IgG antibodies and the intestinal permeability biomarkers, including anti-lipopolysaccharide (LPS) and anti-occludin IgG and IgA antibodies, were found to be correlated.<sup>28</sup> An additional side effect of gluten consumption is that gluteomorphins may have opiate-like characteristics in the brain.<sup>29,30</sup> Thus, removing IgG-trigger foods (including gluten) from the child’s diet may reduce inflammation, intestinal permeability, brain exposure to opioid-like peptides and subsequent behavioral perturbations,<sup>29</sup> and GI disturbances including pain and bloating.<sup>31,32</sup>

**What is the Specific Carbohydrate Diet?**

Dr. Sidney Haas initially published the Specific Carbohydrate Diet (SCD) as a treatment for celiac disease in 1951.<sup>33</sup> Biologist Elaine Gottschall explained the details of the SCD in her 1994 book titled “*Breaking the Vicious*

**Table 2. Amylose/Amylopectin**

Legal	Foods	Brush Border Enzyme Required	Bonds Broken	Structure
Amylose	asparagus black beans <sup>b</sup> (soaked) broccoli brussels sprouts cabbage carrots cauliflower celery cucumber eggplant kidney beans <sup>b</sup> (soaked) lentils <sup>a</sup> (soaked) lettuce navy beans <sup>b</sup> (soaked) onions peas peppers spinach squash <sup>34</sup>	None required, as Salivary Amylase and Pancreatic Amylase can break these bonds.  Although SI (major role) <sup>38</sup> and MGAM (auxiliary role) <sup>38</sup> will significantly contribute if they are present. <sup>37</sup>	α-1,4 glycosidic bonds form the linear segments of amylopectin	
	<b>Illegal</b>			
Amylopectin	black eyed peas chickpeas/garbanzos corn jicama mung beans okra potatoes sweet potatoes taro turnips unsoaked beans <sup>34</sup>	Isomaltase (major role) <sup>38</sup>  Glucoamylase (auxiliary role) <sup>38,40</sup>	α-1,6 glycosidic bonds at the branch points <sup>37</sup>	

<sup>a</sup>Only if symptom-free—more likely to be tolerated;  
<sup>b</sup>Only if symptom-free—less likely to be tolerated.

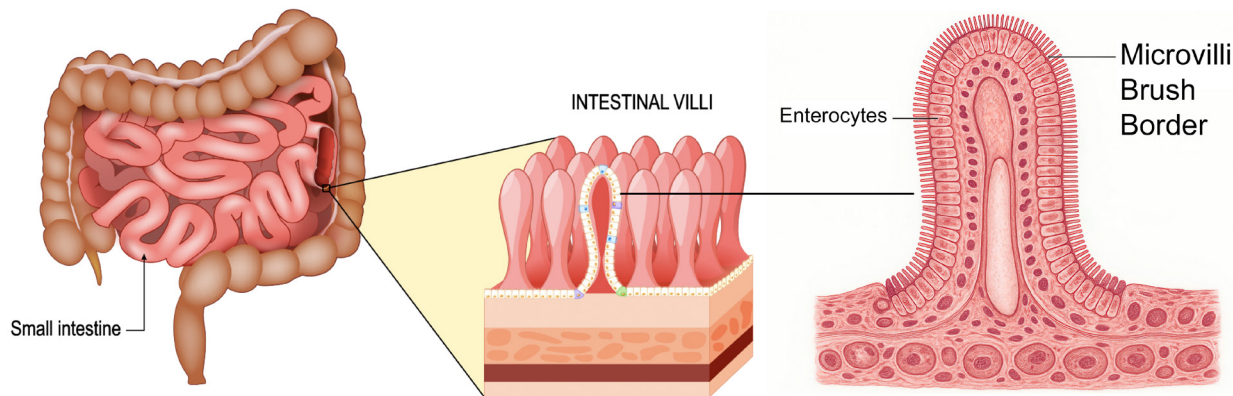
**Note:** Amylose is a linear chain of glucose units linked by α-1,4 glycosidic bonds.<sup>41</sup> Amylopectin contains α-1,4 glycosidic bonds, but also has α-1,6 glycosidic bonds at branch points.<sup>41</sup> The digestive enzymes salivary amylase and pancreatic amylase, function to break down α-1,4 linkages, leaving the branched α-1,6 bonds largely intact. The α-1,6 linkages require the brush border enzyme isomaltase to break them down effectively.<sup>42</sup> Patients with compromised small intestinal villi/microvilli may not produce adequate amounts of isomaltase to break down the α-1,6 bonds found in amylopectin.<sup>34</sup> (Image credit: Liz Paton)

*Cycle*<sup>34</sup> The SCD is an elimination diet that eliminates complex carbohydrates, which may serve as a food source for dysbiotic organisms in the gut microbiome. Within the SCD community, the customary nomenclature used to categorize foods is:<sup>34</sup>

- a. “Legal” for foods that are allowed and compliant with the diet’s restrictions.
- b. “Illegal” for foods that are eliminated and not allowed on the diet.

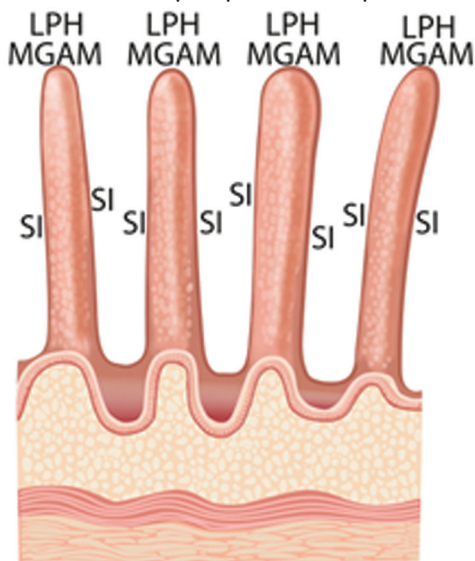
This terminology is widely used in resources, food lists, and discussions surrounding SCD. This diet allows the intake of nearly all fruits, vegetables containing more amylose than amylopectin, lactose-free dairy, meats, eggs, butter, oils, nuts, and nut-derived flours<sup>35</sup> (Tables 1 and 2). The diet permits carbohydrate-containing foods that are monosaccharides, while banning disaccharides and

**Figure 1. Intestinal Villi and Microvilli**



**Note:** The microvilli brush border is the location of brush border enzyme production.<sup>34</sup> (Image credit: DesignUA/Adobe Stock® and Rahasia/Adobe Stock®, used under Standard license)

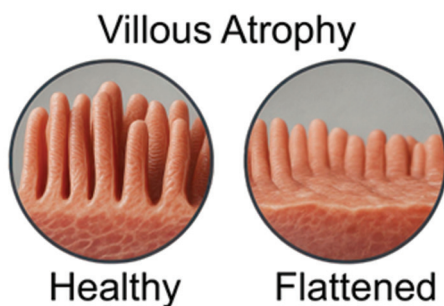
**Figure 2. Microvilli Brush Border - Production of Glycohydrolase Enzymes**



**Note:** These enzymes are required for the digestion of disaccharides, polysaccharides, and amylopectin.<sup>37,44,45</sup> (Image credit: Nahno Mariano Moyano)

**Abbreviations:** SI, Sucrase-Isomaltase; MGAM, Maltase-Glucoamylase; LPH, Lactase-Phlorizin Hydrolase.

**Figure 3. Villous Atrophy**



**Note:** Flattening of the villi/microvilli in the small intestine may be caused by gluten, toxins, and/or dysbiosis.<sup>34</sup> This can reduce the production of enzymes, including SI, MGAM, and LPH,<sup>46,47</sup> leading to an inability to digest disaccharides, polysaccharides, and amylopectin.<sup>34</sup> (Image credit: ©RyujinxRyuken/Adobe Stock, used under Standard license)

**Abbreviations:** SI, Sucrase-Isomaltase; MGAM, Maltase-Glucoamylase; LPH, Lactase-Phlorizin Hydrolase.

**Table 3.** Literature Review: A History of the SCD as Medical Nutrition Therapy for Autism

Date	Title	Summary
1985	Autism and lactic acidosis <sup>48</sup>	Researchers from Georgetown and Cornell shared data suggesting that autism may be associated with faulty “disorders of carbohydrate metabolism”.
1994	Breaking the Vicious Cycle - Intestinal Health through Diet <sup>44</sup>	Included a chapter entitled “The Autism Connection”, which outlined the theory that intestinal bacterial and fungal overgrowth are common in autism, and improvements in GI symptoms and behavior may be seen in patients following the SCD.
2004	Digestion-gut-autism connection: the Specific Carbohydrate Diet <sup>10</sup>	Expounded upon the theory that intestinal bacterial and fungal overgrowth are common in autism, and improvements in GI symptoms and behavior may be seen in patients following the SCD.
2004	Gut and Psychology Syndrome (GAPS) <sup>49</sup>	Focuses on an elimination diet based on the SCD. Theorizes that autism and many other psychological conditions are rooted in the patient’s gut health, including: leaky gut; passing of toxins into the bloodstream (including lipopolysaccharides - LPS); crossing of glutomorphins and casomorphins from the gut → bloodstream → Blood Brain Barrier (BBB); dysbiosis exacerbated by poor nutrition and antibiotic use.
2007	We Band of Mothers: Autism, My Son, and The Specific Carbohydrate Diet <sup>50</sup>	Book published by Autism Research Institute featuring the case of a 12-year-old boy who went from 12+ bowel movements per day (unable to leave the house) to 1-2 formed bowel movements per day, able to travel internationally, and showing vast improvements in receptive language.
2008	Nourishing Hope for Autism <sup>51</sup>	Comprehensive guide that explains the scientific rationale for dietary interventions in children with autism and provides practical, customizable steps for using special diets and nutrition to address symptoms and improve overall health.
2009	Parent ratings of behavioral effects of biomedical interventions <sup>52</sup>	Parent survey published by Autism Research Institute based upon compiled data from 27 000 parents, who rated the SCD as the #1 biomedical diet intervention (with 71% of ASD children showing improvement).
2012	The BrainFood Cookbook <sup>53</sup>	A combined SCD reference guide and SCD cookbook for Autism and Attention-Deficit/Hyperactivity Disorder (ADHD).
2012	GAPS Stories <sup>54</sup>	A collection of case reports, describing improvements in gastrointestinal and behavioral symptoms after instituting the GAPS/SCD diet.
2015	The SCD for Autism and ADHD <sup>55</sup>	A combined SCD reference guide and SCD cookbook (dairy-free).
2016	Diets for Autism Spectrum Disorder <sup>56</sup>	Suggested implementing a casein-free version of the SCD in children with ASD.
2017	Success story: Autism recovery with the SCD/GAPS diet <sup>57</sup>	This case report was initially presented as a short-form conference poster at the International Society for Nutritional Psychology Research conference.
2019	Caregiver perspective on use of the Specific Carbohydrate Diet In children with Autism Spectrum Disorder <sup>58</sup>	A caregiver survey of 256 ASD participants following the SCD reported significant improvements in GI pain, constipation, diarrhea, gastroesophageal reflux, gagging, vomiting, throat pain, anxiety, irritability, and toilet training, with 63% of participants reporting improved language after the SCD intervention.
2020	Brief Report: Implementation of a Specific Carbohydrate Diet for a Child with Autism Spectrum Disorder and Fragile X Syndrome <sup>59</sup>	The case report described good tolerance of the SCD in a 4-year-old boy with ASD, and subsequent improvements in gastrointestinal symptoms, nutrient status, and behavioral domains.
2021	Specific Carbohydrate Diet (SCD/ GAPS) and dietary supplements for children with autistic spectrum disorder <sup>60</sup>	The 3-month open-label pilot study with 10 children in the SCD group (7 in the control group) found 43% improvements in the SCD group vs. 14% improvement in controls. These improvement levels were in the context of imperfect adherence to the SCD diet, with SCD compliance in the treatment group estimated at 40-80%. This suggests that even with less-than-perfect adherence, the SCD may still lead to significant improvements in ASD symptoms.
2022	Diet in treatment of autism spectrum disorders <sup>61</sup>	The SCD was described in a review article of dietary interventions that may be helpful in the treatment of ASD.
2022	Efficacy and Safety of Diet Therapies in Children With Autism Spectrum Disorder <sup>12</sup>	Systematic review and meta-analysis found that dietary interventions were related to significant symptom reductions in ASD patients. Listed the SCD as one of the most used dietary interventions.
2025	The Personalized Autism Nutrition Plan <sup>62</sup>	Offers a personalized nutrition plan, based upon a child’s unique biochemical needs and symptoms, to holistically improve the well-being of children with ASD and other neurodevelopmental delays. Describes the content and implementation of the SCD.

polysaccharides. Therefore, it excludes sucrose, lactose, and cheeses containing high amounts of lactose, maltose, isomaltose, grains, grain-derived flours, corn, okra, potatoes, soy, and most food additives and preservatives. A comprehensive list of “legal vs illegal” foods<sup>36</sup> can be found at <https://breakingtheviciouscycle.info/legal%2Filllegal-list-pdf/>

Intestinal microvilli are the sites of brush border production of glycohydrolase enzymes sucrase-isomaltase (SI), maltase-glucoamylase (MGAM), and lactase-phlorizin hydrolase (LPH), which are needed for the digestion of disaccharides and polysaccharides<sup>42</sup> (Figures 1 and 2). When microvilli are damaged (e.g., by gluten, toxins, and/or dysbiosis), they can no longer produce sufficient brush border enzymes<sup>34</sup> (Figure 3). This leads to poor digestion, malabsorption, and dysbiosis, which continue to damage the microvilli in a vicious cycle.<sup>34</sup> Thus, it is hypothesized that by removing illegal foods for several months, we may modulate dysbiosis, reduce

mucosal inflammation, and allow villi/microvilli to recover.<sup>35,43</sup>

### A History of the SCD as Medical Nutrition Therapy for Autism

In 1985, researchers from Georgetown and Cornell shared data suggesting that autism may be associated with “disorders of carbohydrate metabolism”.<sup>48</sup> Since then, several researchers, practitioners, and caregivers have published books, case reports, and research on SCD as medical nutrition therapy for autism. Table 3 summarizes the scientific and lay literature from 1985 to 2025.

### Case Report

This case report describes the elimination of ASD symptoms and lifting of the individualized education program (IEP) following the SCD dietary approach. Long-term follow-up demonstrated sustained recovery from symptomatology as reflected in a re-evaluation Autism

**Table 4. Patient’s Timeline**

Age	Details
Birth	Vaginal birth, went into labor naturally. 3 days overdue. 15-hour labor, epidural, and Pitocin given. Birth weight: 8 pounds 3 ounces. Apgar Score = 9 Declined in-hospital vaccinations. Exclusively breastfed for 6 months, and continued breastfeeding until age 5.
2 months	First round vaccinations (IPV, DT/DTap, HiB/HepB, and PCV7, all on the same day).
4 months	Second round vaccinations (IPV, DT/DTap, HiB/HepB, and PCV7, all on the same day).
6 months	Third round vaccinations (IPV, DT/DTap, and PCV7, all on the same day).
12 months	Fourth round vaccinations (Hep B/HiB, MMR-V, and Fluzone #1 2006-2007 formulation, all on the same day). Triggered fever, which ran for 3 weeks (103 °F on several dates). Acetaminophen, several doses given daily for 3 weeks. Labs run 14 days post-vaccination (LabCorp): i. WBC = 19820 H (range 6000-17500) ii. Hemoglobin = 10.8 L (range 11.8-13.8) iii. Platelet count = 420 H (range 150-350) The child experienced night sweats for several years thereafter. Parents declined further vaccinations - medical waiver received.
14 months	Started walking. Frequent toe-walking was observed.
23 months	Expressive speech below 25% cutoff. Dx: Expressive Language Delay at University of Maryland School of Medicine, 737 W. Lombard St., Baltimore, MD.
24 months	Tympanometry indicated adequate middle ear mobility and pressure. Began Speech Therapy twice monthly for 30-minute sessions.
26 months	Lead blood test = negative
32 months	Dx: Autism via ADOS DSM-IV at Baltimore Infants and Toddlers Program, 10 W. Eager St., Baltimore, MD. Vaccine Titers (LabCorp): i. Rubella Antibodies IgG = 23 (Immune >9) ii. Rubeola (Measles) Ab, IgG, EIA = 5.12 H (Positive Immune >1.09) iii. Mumps Abs, IgG = 2.48 H (Positive Immune > 1.09) Food IgG testing done at LabCorp: range 0-5 (higher indicates greater adverse reaction): Wheat = 5; Corn = 4; Soybean = 4; Milk (cow) = 3 Began Gluten-free Diet: Stimming behaviors (hand flapping) reduced, but autism symptoms persisted.
34 months	Comprehensive Stool Analysis (Doctor’s Data, Saint Charles, IL): i. Lysozyme = 728 H (range < 600) (marker of GI inflammation) ii. Lactoferrin = 16.6 H (range < 7.3) (marker of GI inflammation) iii. Yeast = moderate (pathogen) iv. sIgA = 21 L (range 51-204; sIgA are protective GI antibodies. Low levels may indicate the gut has been fighting a chronic stressor, and can no longer mount an adequate response.) <sup>65</sup>
35 months	Home videos show poor speech, lack of response to own name, and poor eye contact. Confirmed Dx: Autism at Kennedy Krieger Institute, 707 N. Broadway, Baltimore, MD Repeated ADOS DSM-IV: Communication Total = 7 (Autism threshold ≥ 4) Social Interaction Total = 10 (Autism threshold ≥ 7) Stereotyped Behaviors and Restricted Interests Total = 3 (Autism threshold ≥ 2) <b>Began Specific Carbohydrate Diet (SCD)</b>
46 months	Puerto Rico trip (ate something SCD-illegal) Temporary regression: back to solo play, resumed humming, resumed “blowing raspberries” sounds.
52 months	<b>Elimination of Autism Symptoms</b> Home videos show excellent expressive speech, interpersonal skills, and eye contact. Individualized Education Program (IEP) services lifted (including speech therapy, OT).
Age 6	Graduated from Montessori Children’s House (ages 3-6).
Age 9	Graduated from Montessori Lower Elementary. Enrolled in a competitive Private Boys’ School. Transitioned to Gluten-Free Diet (GF grains and potatoes permitted).
Age 17	Attained Eagle Scout Ranking. Attained Kung Fu Black Belt.
Age 18	Graduated from a competitive Private Boys’ School. Enrolled in a competitive Engineering School. ADOS re-evaluation score = 0. Psychological Evaluation & Therapy, 1700 Reisterstown Road, Suite 231, Pikesville, MD
Age 19	Evaluation at Kennedy Krieger Institute: Stanford Binet score = 111 (above average abilities) NIH Toolbox Pattern Comparison Processing Speed Test > 95% of peers (above average abilities).

Diagnostic Observation Schedule (ADOS) score of 0, indicating no evidence of autism.

This case report was approved by the Institutional Review Board of Johns Hopkins University (HIRB00020122) and was aligned with the EQUATOR network’s CARE guidelines for case reports.<sup>62,63</sup> Written informed consent was obtained from the child and parent to publish this case report.

**Patient Timeline**

The patient was conceived naturally (timeline provided in Table 4), to parents who were 34 years old at the time of conception. During the third month of gestation, the mother noticed a bull’s-eye rash on her leg, was diagnosed with Lyme Disease, and was prescribed a 21-day course of antibiotics. The mother contracted an upper respiratory infection (common cold) in the third month of gestation, and took over-the-counter Robitussin

cough syrup (Guaifenesin), and prescription Tessalon Perles (Benzonatate) for coughing.

### Primary Concerns and Symptoms of the Patient

A 12-month-old boy received several vaccine injections at his 1-year-old physician visit. Immediately thereafter, he ran a 103°F fever for 3 weeks. Multiple doses of acetaminophen were administered daily over these 3 weeks. The dosing of acetaminophen was stopped after 3 weeks; thereafter the patient experienced ongoing night sweats for several months. (The phenomenon of night sweats [aka sleep hyperhidrosis] is when the normal circadian rhythm regulating body temperature is exaggerated, causing profuse sweating during the sleeping hours.<sup>66</sup> Sleep hyperhidrosis has been reported in 20% of autistic children vs 5% of controls.<sup>67</sup>)

At 23 months old, the boy was diagnosed with Expressive Language delay at the University of Maryland School of Medicine. The Baltimore City Infants and Toddlers Program provided twice-monthly in-home speech therapy for 30 minutes per session. He had a limited vocabulary of several nouns and one verb. He often exhibited echolalia: involuntary repetition of words or phrases that someone else has said, without understanding the meaning. Challenging behaviors included stimming (hand flapping), banging his head on the ground, humming like a train, screeching, and making “blowing raspberries” sounds. During this time the boy was eating a Standard American Diet, including gluten and pasteurized dairy products. The family also consumed a daily smoothie containing raw milk kefir - a fermented dairy product made from unpasteurized A2 milk.

### Initial Autism Testing and Diagnosis

At age 32 months, speech had not yet normalized, and psychological testing via the Autism Diagnostic Observation Schedule (ADOS, DSM-IV criteria) at the Baltimore City Infants and Toddlers Program revealed a diagnosis of autism. At age 35 months, Kennedy Krieger Institute in Baltimore confirmed the autism diagnosis by retesting using the Autism Diagnostic Observation Schedule (ADOS, DSM-IV criteria):

Communication domain = 7 (Autism threshold  $\geq$  4)

Social Interaction domain = 10 (Autism threshold  $\geq$  7)

Stereotyped Behaviors and Restricted Interests domain = 3 (Autism threshold  $\geq$  2)

### Therapeutic Intervention

Shortly after the autism diagnosis, a licensed physician prescribed a gluten-free, casein-free diet. All gluten-containing foods were removed from the house. The family adhered strictly to a gluten-free diet, but found that stopping kefir consumption was associated with an increased incidence of upper respiratory infections. A decision was made to avoid regular pasteurized milk, but to continue consuming fermented A2 casein raw milk (as

kefir). This strict gluten-free approach worked well to extinguish bothersome behaviors such as head banging and hand flapping. However, a single instance of a dietary infraction (grandmother offering a single spoonful of gluten-containing chicken soup broth) triggered a temporary return of stimming behaviors (hand flapping) for a few days. The gluten-free diet was followed strictly thereafter, with all foods made at home and packed lunches sent to preschool. Clear instructions were provided to relatives and to preschool staff of his gluten-free status, and special homemade cupcakes were kept in the school's freezer for party days. Despite following a strict gluten-free diet, the boy's low expressive speech persisted.

At age 35 months, the parents decided to embark upon the Specific Carbohydrate Diet (SCD). Gluten, all grains, lactose, sucrose, potatoes, food coloring, and food additives were eliminated. All foods were organic, and all animal products were grass-fed/pasture-raised. Lactose-free kefir (A2/raw/fermented for 24 hours) was continued. SCD-legal food lists were consulted from *Breaking the Vicious Cycle*<sup>34</sup> and *Gut and Psychology Syndrome*.<sup>49</sup> All SCD non-compliant foods were removed from the house. Packaged foods were avoided, and all meals were made from scratch in the home kitchen. The mother translated the family's recipes into SCD-compliant versions by eliminating wheat flour and sugar, and using nut flour, coconut flour, fruit, and honey instead. Several servings of fruits and vegetables were consumed daily, as well as animal proteins, and spring water. The boy continued to use the same speech therapist, who noted his expressive language and social skills improved significantly.

### Patient Outcomes

By age 4 years and 6 months, ASD symptoms were significantly reduced (17 months after starting the SCD), and the boy no longer needed special education services. The child subsequently graduated from Montessori Children's House at age 6; graduated from Montessori Lower Elementary School at age 9, and subsequently entered a competitive private school. The child's resolution of ASD symptoms was further reflected in attaining an Eagle Scout ranking at age 17, graduating from high school at age 18, and current enrollment in a competitive engineering college at the time of the composition of this manuscript. ADOS re-evaluation in 2024 revealed a score of 0, confirming the elimination of his autism symptoms.

### Discussion

This case report presents the case of a boy diagnosed with autism who exhibited dramatically improved speech and daily behavior after implementing the Specific Carbohydrate Diet (SCD). Before initiating the SCD, he was wearing diapers, socially isolated, did not point or make eye contact, had difficulty understanding commands or questions, often used echolalia when speaking, frequently jumped up and down, and frequently flapped his hands.

As shown in the timeline (Table 4), the boy received several vaccine injections at his 1 year physician visit, and ran a high fever for 3 weeks thereafter (103°F on several dates). Multiple doses of acetaminophen were administered daily over these 3 weeks. The metabolism of acetaminophen requires glutathione for phase 2 biotransformation in the liver.<sup>68,69</sup> The vaccines he received contained the adjuvants aluminum and thimerosal (flu vaccine).<sup>70,71</sup> Glutathione is also needed for phase 2 biotransformation of aluminum and mercury (thimerosal contains 50% mercury).<sup>70,72-74</sup> Therefore, we hypothesize that glutathione levels may have become depleted owing to repeated acetaminophen dosing,<sup>75</sup> thereby reducing glutathione availability to detoxify aluminum and mercury, both of which are potent neurotoxins.<sup>76,77</sup> Aluminum and mercury are strong inducers of gut dysbiosis and lipopolysaccharide (LPS).<sup>78,79</sup> Comprehensive stool analysis showed results suggesting dysbiosis (Table 4). A clinical hypothesis is that LPS, along with gluten consumption, may have caused the breakdown of tight junctions between gut epithelial cells.<sup>79</sup> Dysfunctional tight junctions may have led to intestinal paracellular permeability, resulting in leaky gut.<sup>80</sup> Increased paracellular transit of food-associated peptides may have triggered immune stimulation, production of food peptide antibodies, and inflammation.<sup>81</sup> Lab testing showed elevated food IgG antibody levels and GI inflammation (Table 4). This may have affected brain function, resulting in learning difficulties and erratic behavior.<sup>82</sup>

Additionally, we hypothesize that initiating the SCD elimination diet may have modulated dysbiosis and reduced mucosal inflammation, allowing microvilli to recover.<sup>34</sup> All these may have increased the production of intestinal brush border enzymes, improved gut-brain axis function, and reduced autism symptoms.<sup>34,49</sup> After 19 months on the SCD, he was toilet-trained, able to attend a neurotypical preschool, play with friends, engage in imaginative play, ask questions, and speak in complete sentences. Subsequently, the IEP team met and decided to remove his IEP, as he no longer required special education services. The elimination of autism symptoms remained solid (with no evidence of regression) and was verified via re-administration of the ADOS at age 18, with a score of 0 (Table 4).

### Parent Perspective

“My heart was broken when my son was diagnosed with autism at the age of 2. He would often exhibit ASD behaviors such as flapping his hands, toe-walking, and banging his head on the ground when frustrated. He would run around aimlessly in circles. He did not point at objects. Toilet training was not even a consideration. He would obsessively play with trains for hours. He had no interest in opening Christmas presents; he actually dropped them on the ground and walked away. He would play by himself, even in a roomful of children. He rarely looked people in the eye. A standardized speech test put

him down in the 6th percentile. When my husband and I spoke to him, he literally could not understand what we were saying. He also exhibited echolalia, repeating things that he had heard without understanding a word of it. He never asked any questions.

I was fortunate to get an appointment with a specialist who insisted we start the Gluten-Free/Casein-Free (GFCF) diet immediately. We drove directly from his office to the health food store and purchased an entirely new inventory for our kitchen. I put our whole family on the diet to ensure no cheating would occur. I was very pleased that “gluten-free” worked well to eliminate his stimming/flapping behaviors, but his general lack of comprehension persisted.

I had read some testimonials from other moms about how the Specific Carbohydrate Diet (SCD) had helped their children. In fact, the SCD was listed as the #1 most effective non-drug intervention according to a survey of 27 000 parents published by the Autism Research Institute. So, we started the SCD, and I cooked absolutely everything from scratch. Again, our whole family followed the diet to ensure compliance. I noticed that his eye contact, speech, comprehension, and behavior were improving. Within a year of starting the SCD, he was enrolled in a neurotypical preschool, toilet trained, playing with friends, asking questions, using pronouns correctly, and speaking in complete sentences.”

### Conclusion

Medical nutrition therapy has been shown to reduce the symptoms of autism in numerous publications. This case report demonstrated the elimination of autism diagnosis and individualized education program (IEP) dissolution for a 4-year-old boy with autism who had followed the Specific Carbohydrate Diet (SCD). While promising, more research is needed to explore the associations between GI dysfunction, intestinal permeability, IgG-mediated food sensitivities, medical nutrition therapy, and autism spectrum disorder.

### Declarations: Ethics Approval And Consent To Participate

This case report was approved by the Institutional Review Board of Johns Hopkins University (Approval number: HIRB00020122, Date: October 21, 2024). Written informed consent was obtained from the child and parent to publish this case report.

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### References

1. American Psychiatric Association. *DSM-5 Classification*. American Psychiatric Association; 2016. Accessed November 7, 2025. <https://play.google.com/store/books/details?id=dKTuzgEACAAJ>
2. Gershon MD, Margolis KG. The gut, its microbiome, and the brain: connections and communications. *J Clin Invest*. 2021;131(18):e143768. doi:10.1172/JCI143768
3. Holingue C, Newill C, Lee LC, Pasricha PJ, Daniele Fallin M. Gastrointestinal symptoms in autism spectrum disorder: A review of the literature on ascertainment and prevalence. *Autism Res*. 2018;11(1):24-36. doi:10.1002/aur.1854

4. Williams BL, Hornig M, Buie T, et al. Impaired carbohydrate digestion and transport and mucosal dysbiosis in the intestines of children with autism and gastrointestinal disturbances. *PLoS One*. 2011;6(9):e24585. doi:10.1371/journal.pone.0024585
5. Alberti A, Pirrone P, Elia M, Waring RH, Romano C. Sulphation deficit in "low-functioning" autistic children: a pilot study. *Biol Psychiatry*. 1999;46(3):420-424. doi:10.1016/S0006-3223(98)00337-0
6. de Oliveira ZB, Silva da Costa DV, da Silva Dos Santos AC, et al. Synthetic colors in food: A warning for children's health. *Int J Environ Res Public Health*. 2024;21(6):682. doi:10.3390/ijerph21060682
7. Kang DW, Park JG, Ilhan ZE, et al. Reduced incidence of Prevotella and other fermenters in intestinal microflora of autistic children. *PLoS One*. 2013;8(7):e68322. doi:10.1371/journal.pone.0068322
8. Wang L, Yu YM, Zhang YQ, Zhang J, Lu N, Liu N. Hydrogen breath test to detect small intestinal bacterial overgrowth: a prevalence case-control study in autism. *Eur Child Adolesc Psychiatry*. 2018;27(2):233-240. doi:10.1007/s00787-017-1039-2
9. D'Euferia P, Celli M, Finocchiaro R, et al. Abnormal intestinal permeability in children with autism. *Acta Paediatr*. 1996;85(9):1076-1079. doi:10.1111/j.1651-2227.1996.tb14220.x
10. Gottschall E. Digestion-gut-autism connection: the Specific Carbohydrate Diet. *Med Veritas J Med Truth*. 2004;1:261-271. doi:10.1588/medver.2004.01.00029
11. Hartman C, Eliakim R, Shamir R. Nutritional status and nutritional therapy in inflammatory bowel diseases. *World J Gastroenterol*. 2009;15(21):2570-2578. doi:10.3748/wjg.15.2570
12. Yu Y, Huang J, Chen X, et al. Efficacy and Safety of Diet Therapies in Children With Autism Spectrum Disorder: A Systematic Literature Review and Meta-Analysis. *Front Neurosci*. 2022;13:844117. doi:10.3389/fnec.2022.844117
13. Trajkovski V, Petlichkovski A, Efinska-Mladenovska O, et al. Higher plasma concentration of food-specific antibodies in persons with autistic disorder in comparison to their siblings. *Focus Autism Other Dev Disabl*. 2008;23(3):176-185. doi:10.1177/1088357608320413
14. Yu XB, Uhde M, Green PH, Alaedini A. Autoantibodies in the extraintestinal manifestations of celiac disease. *Nutrients*. 2018;10(8):1123. doi:10.3390/nu10081123
15. Lerner A, Benzvi C, Vojdani A. Gluten is a Proinflammatory Inducer of Autoimmunity. *J Transl Gastroenterol*. 2024;2(2):109-124. doi:10.14218/JTG.2023.00060
16. Jyonouchi H, Geng L, Ruby A, Reddy C, Zimmerman-Bier B. Evaluation of an association between gastrointestinal symptoms and cytokine production against common dietary proteins in children with autism spectrum disorders. *J Pediatr*. 2005;146(5):605-610. doi:10.1016/j.jpeds.2005.01.027
17. Jyonouchi H. Food allergy and autism spectrum disorders: is there a link? *Curr Allergy Asthma Rep*. 2009;9(3):194-201. doi:10.1007/s11882-009-0029-y
18. Lucarelli S, Frediani T, Zingoni AM, et al. Food allergy and infantile autism. *Panminerva Med*. 1995;37(3):137-141.
19. Herbert MR, Buckley JA. Autism and dietary therapy: case report and review of the literature. *J Child Neurol*. 2013;28(8):975-982. doi:10.1177/0883073813488668
20. Evangelidou A, Vlachonikolis I, Mihailidou H, et al. Application of a ketogenic diet in children with autistic behavior: pilot study. *J Child Neurol*. 2003;18(2):113-118. doi:10.1177/08830738030180020501
21. Blades M. Autism: an interesting dietary case history. *Nutr Food Sci*. 2000;30(3):137-140. doi:10.1108/00346650010319741
22. Knivsberg AM, Reichelt KL, Høien T, Nødland M. Effect of a Dietary Intervention on Autistic Behavior. *Focus Autism Other Dev Disabl*. 2003;18(4):248-257. doi:10.1177/10883576030180040601
23. Goin-Kochel RP, Mackintosh VH, Myers BJ. Parental reports on the efficacy of treatments and therapies for their children with autism spectrum disorders. *Res Autism Spectr Disord*. 2009;3(2):528-537. doi:10.1016/j.rasd.2008.11.001
24. Pennesi CM, Klein LC. Effectiveness of the gluten-free, casein-free diet for children diagnosed with autism spectrum disorder: based on parental report. *Nutr Neurosci*. 2012;15(2):85-91. doi:10.1179/1476830512Y.0000000003
25. Whiteley P, Haracopos D, Knivsberg AM, et al. The ScanBrit randomised, controlled, single-blind study of a gluten- and casein-free dietary intervention for children with autism spectrum disorders. *Nutr Neurosci*. 2010;13(2):87-100. doi:10.1179/147683010X12611460763922
26. Fasano A. All disease begins in the (leaky) gut: role of zonulin-mediated gut permeability in the pathogenesis of some chronic inflammatory diseases. *F1000Res*. 2020;9:F1000 Faculty Rev-69. doi:10.12688/f1000research.20510.1
27. Lammers KM, Lu R, Brownley J, et al. Gliadin induces an increase in intestinal permeability and zonulin release by binding to the chemokine receptor CXCR3. *Gastroenterology*. 2008;135(1):194-204.e3. doi:10.1053/j.gastro.2008.03.023
28. Vita AA, Zwickey H, Bradley R. Associations between food-specific IgG antibodies and intestinal permeability biomarkers. *Front Nutr*. 2022;9:962093. doi:10.3389/fnut.2022.962093
29. Loscalzo R. The role of gluten in the etiology of neurodevelopmental disorders: opioid and immunological mechanisms. *Nutritional Perspectives: Journal of the Council on Nutrition*. 2007;30(2):16.
30. Ulaş AŞ, Çakır A, Erbaş O. Gluten and casein: their roles in psychiatric disorders. *JEBMS*. 2022;3(1):13-21. doi:10.5606/jebms.2022.1003
31. Biesiekierski JR, Newnham ED, Irving PM, et al. Gluten causes gastrointestinal symptoms in subjects without celiac disease: a double-blind randomized placebo-controlled trial. *Am J Gastroenterol*. 2011;106(3):508-514. doi:10.1038/ajg.2010.487
32. Casini I, Fatighenti E, Giannantoni A, et al. Food-Specific IgG4 Antibody-Guided Exclusion Diet Improves Conditions of Patients with Chronic Pain. *Pain Ther*. 2022;11(3):873-906. doi:10.1007/s40122-022-00391-z
33. Haas SV, Haas MP. *Management of Celiac Disease*. Pickle Partners Publishing; 1951.
34. Gottschall E. *Breaking the Vicious Cycle - Intestinal Health through Diet*. Kirkton Press; 1994.
35. Suskind DL, Wahbeh G, Gregory N, Vendettuoli H, Christie D. Nutritional therapy in pediatric Crohn disease: the specific carbohydrate diet. *J Pediatr Gastroenterol Nutr*. 2014;58(1):87-91. [https://journals.lww.com/jpgn/fulltext/2014/01000/Nutritional\\_Therapy\\_in\\_Pediatric\\_Crohn\\_Disease\\_\\_22.aspx?fbclid=IwAR3uFnmYyZm3xwxy65wB9rgy8SHyNIE4aS3mbVvmbM-LqItMC6WY2vJrgg](https://journals.lww.com/jpgn/fulltext/2014/01000/Nutritional_Therapy_in_Pediatric_Crohn_Disease__22.aspx?fbclid=IwAR3uFnmYyZm3xwxy65wB9rgy8SHyNIE4aS3mbVvmbM-LqItMC6WY2vJrgg) doi:10.1097/MPG.00000000000000103
36. Breaking the Vicious Cycle. Legal and Illegal Foods List. Accessed November 7, 2025. <https://breakingtheviciouscycle.info/legal%2Fillegal-list-pdf>
37. Van Beers EH, Büller HA, Grand RJ, Einerhand AW, Dekker J. Intestinal brush border glycohydrolases: structure, function, and development. *Crit Rev Biochem Mol Biol*. 1995;30(3):197-262. doi:10.3109/10409239509085143
38. Tannous S, Stellbrinck T, Hoter A, Naim HY. Interaction between the  $\alpha$ -glucosidases, sucrose-isomaltase and maltase-glucoamylase, in human intestinal brush border membranes and its potential impact on disaccharide digestion. *Front Mol Biosci*. 2023;10:1160860. doi:10.3389/fmolb.2023.1160860
39. Amiri M, Naim HY. Characterization of mucosal disaccharidases from human intestine. *Nutrients*. 2017;9(10):1106. doi:10.3390/nu9101106
40. Ren L, Qin X, Cao X, et al. Structural insight into substrate specificity of human intestinal maltase-glucoamylase. *Protein Cell*. 2011;2(10):827-836. doi:10.1007/s12328-011-1105-3
41. Kong X. Fine Structure of Amylose and Amylopectin. In: *Starch Structure. Functionality and Application in Foods*. Springer Singapore; 2020:29-39, doi:10.1007/978-981-15-0622-2\_3.
42. Hooton D, Lentle R, Monro J, Wickham M, Simpson R. The secretion and action of brush border enzymes in the mammalian small intestine. *Rev Physiol Biochem Pharmacol*. 2015;168:59-118. doi:10.1007/112\_2015\_24
43. Kakodkar S, Farooqui AJ, Mikolaitis SL, Mutlu EA. The specific carbohydrate diet for inflammatory bowel disease: A case series. *J Acad Nutr Diet*. 2015;115(8):1226-1232. doi:10.1016/j.jand.2015.04.016
44. Goda T, Yasutake H, Tanaka T, Takase S. Lactase-phlorizin hydrolase and sucrose-isomaltase genes are expressed differently along the villus-crypt axis of rat jejunum. *J Nutr*. 1999;129(6):1107-1113. doi:10.1093/jn/129.6.1107
45. McConnell RE, Higginbotham JN, Shifrin DA Jr, Tabb DL, Coffey RJ, Tyska MJ. The enterocyte microvillus is a vesicle-generating organelle. *J Cell Biol*. 2009;185(7):1285-1298. doi:10.1083/jcb.200902147
46. Nieminen U, Kahri A, Savilahti E, Färkkilä MA. Duodenal disaccharidase activities in the follow-up of villous atrophy in coeliac disease. *Scand J Gastroenterol*. 2001;36(5):507-510. doi:10.1080/00365520120555
47. Nichols BL, Nichols VN, Putman M, et al. Contribution of villous atrophy to reduced intestinal maltase in infants with malnutrition. *J Pediatr Gastroenterol Nutr*. 2000;30(5):494-502. doi:10.1097/00005176-200005000-00007
48. Coleman M, Blass JP. Autism and lactic acidosis. *J Autism Dev Disord*. 1985;15(1):1-8. doi:10.1007/BF01837894
49. Campbell-McBride N. *Gut and Psychology Syndrome*. Medinform Publishing; 2004.
50. Chintz J. *We Band of Mothers: Autism, My Son and the Specific Carbohydrate Diet*. Autism Research Institute; 2007.
51. Matthews J. Nourishing hope for autism: nutrition and diet guide for healing our children. San Francisco: Healthful Living Media. Published online 2008.
52. Edelson S. Parent ratings of behavioral effects of biomedical interventions. Accessed November 7, 2025. Autism Research Institute. 2009;34. <https://autism.org/treatment-ratings-for-autism/>
53. Taylor A. *The BrainFood Cookbook - Autism & ADD Recovery Using the SCD/ GAPS/PALEO Diet*. Taylor Interactive; 2012.
54. Campbell-McBride N. GAPS Stories: Personal Accounts of Improvement and Recovery Through the GAPS Nutritional Protocol. Medinform Publishing; 2012. [https://play.google.com/store/books/details?id=3nx\\_DwAAQBAJ](https://play.google.com/store/books/details?id=3nx_DwAAQBAJ)
55. Ferro P, Prasad R, Moomchala N. *The SCD for Autism and ADHD: A Reference and Dairy-Free Cookbook for the Specific Carbohydrate Diet*. Swallowtail Press; 2015.
56. Ferro P, Prasad R. Diets for autism spectrum disorder: learning from IBD and IBS treatments. *Altern Complement Ther*. 2016;22(2):54-58. doi:10.1089/act.2016.29046.pfe
57. Taylor A. Success Story: Autism Recovery with the GAPS diet [Gut and Psychology Syndrome]. In: International Society for Nutritional Psychology Research; 2017.
58. Barnhill KM, Richardson W, Rodriguez L, et al. Caregiver perspective on use of the Specific Carbohydrate Diet In children with Autism Spectrum Disorder. *J Nutr Health*. 2019;(6).
59. Barnhill K, Devlin M, Moreno HT, et al. Brief Report: Implementation of a Specific Carbohydrate Diet for a Child with Autism Spectrum Disorder and Fragile X Syndrome. *J Autism Dev Disord*. 2020;50(5):1800-1808. doi:10.1007/s10803-018-3704-9

60. Ábele S, Meija L, Folkmanis V, Tzivian L. Specific Carbohydrate Diet (SCD/GAPS) and dietary supplements for children with autistic spectrum disorder. *Proc Latv Acad Sci B Nat Sci*. 2021;75(6):417-425. doi:10.2478/prolas-2021-0062
61. Alam S, Westmark CJ, McCullagh EA. Diet in treatment of autism spectrum disorders. *Front Neurosci*. 2023;16:1031016. doi:10.3389/fnins.2022.1031016
62. Matthews JS. *The Personalized Autism Nutrition Plan*. BenBella Books; 2025.
63. Riley DS, Barber MS, Kienle GS, et al. CARE guidelines for case reports: explanation and elaboration document. *J Clin Epidemiol*. 2017;89:218-235. doi:10.1016/j.jclinepi.2017.04.026
64. Gagnier JJ, Kienle G, Altman DG, Moher D, Sox H, Riley D; CARE Group\*. The CARE Guidelines: Consensus-based Clinical Case Reporting Guideline Development. *Glob Adv Health Med*. 2013;2(5):38-43. doi:10.7453/gahmj.2013.008
65. Lipski E. Digestive Wellness for Children: How to Strengthen the Immune System & Prevent Disease Through Healthy Digestion. Published online 2006. [https://books.google.com/books?hl=en&dr=&id=nAiay7osRhcC&oi=fnd&pg=PP11&dq=digestive+wellness+lipski&ots=h5rOecBaLT&sig=K\\_X0t7yf\\_TnQ8jzAsnwXbsjCpAY](https://books.google.com/books?hl=en&dr=&id=nAiay7osRhcC&oi=fnd&pg=PP11&dq=digestive+wellness+lipski&ots=h5rOecBaLT&sig=K_X0t7yf_TnQ8jzAsnwXbsjCpAY)
66. Dall L, Stanford JF. Fever, chills, and night sweats. *Clinical Methods: The History, Physical, and Laboratory Examinations* 3rd edition. Published online 1990. <https://www.ncbi.nlm.nih.gov/books/NBK324/>
67. da Silveira Cruz-Machado S, Guissoni Campos LM, Fadini CC, Anderson G, Markus RP, Pinato L. Disrupted nocturnal melatonin in autism: association with tumor necrosis factor and sleep disturbances. *J Pineal Res*. 2021;70(3):e12715. doi:10.1111/jpi.12715
68. Stahl SH, Yates JW, Nicholls AW, et al. Systems toxicology: modelling biomarkers of glutathione homeostasis and paracetamol metabolism. *Drug Discov Today Technol*. 2015;15:9-14. doi:10.1016/j.ddtec.2015.06.003
69. Yan M, Huo Y, Yin S, Hu H. Mechanisms of acetaminophen-induced liver injury and its implications for therapeutic interventions. *Redox Biol*. 2018;17:274-283. doi:10.1016/j.redox.2018.04.019
70. Tomljenovic L, Shaw CA. Aluminum vaccine adjuvants: are they safe? *Curr Med Chem*. 2011;18(17):2630-2637. doi:10.2174/092986711795933740
71. Lee-Wong M, Resnick D, Chong K. A generalized reaction to thimerosal from an influenza vaccine. *Ann Allergy Asthma Immunol*. 2005;94(1):90-94. doi:10.1016/S1081-1206(10)61292-6
72. Geier DA, King PG, Hooker BS, et al. Thimerosal: clinical, epidemiologic and biochemical studies. *Clin Chim Acta*. 2015;444:212-220. doi:10.1016/j.cca.2015.02.030
73. Teschke R. Aluminum, arsenic, beryllium, cadmium, chromium, cobalt, copper, iron, lead, mercury, molybdenum, nickel, platinum, thallium, titanium, vanadium, and zinc: molecular aspects in experimental liver injury. *Int J Mol Sci*. 2022;23(20):12213. doi:10.3390/ijms232012213
74. Rubino FM. Toxicity of glutathione-binding metals: A review of targets and mechanisms. *Toxics*. 2015;3(1):20-62. doi:10.3390/toxics3010020
75. Slattery JT, Wilson JM, Kalthorn TF, Nelson SD. Dose-dependent pharmacokinetics of acetaminophen: evidence of glutathione depletion in humans. *Clin Pharmacol Ther*. 1987;41(4):413-418. doi:10.1038/clpt.1987.50
76. Schofield K. The metal neurotoxins: an important role in current human neural epidemics? *Int J Environ Res Public Health*. 2017;14(12):1511. doi:10.3390/ijerph14121511
77. Alexandrov PN, Pogue AI, Lukiw WJ. Synergism in aluminum and mercury neurotoxicity. *Integr Food Nutr Metab*. 2018;5(3). doi:10.15761/IFNM.1000214
78. Alexandrov PN, Hill JM, Zhao Y, et al. Aluminum-induced generation of lipopolysaccharide (LPS) from the human gastrointestinal (GI)-tract microbiome-resident *Bacteroides fragilis*. *J Inorg Biochem*. 2020;203(110886):110886. doi:10.1016/j.jinorgbio.2019.110886
79. Afzal M, Altayar AE, Ghaboura N, et al. Effect of mercury on gut microbiome. In: *Gut Microbiome and Environmental Toxicants*. CRC Press; 2025:201-221. doi:10.1201/9781003489221-10.
80. Barbara G, Barbaro MR, Fuschi D, et al. Inflammatory and Microbiota-related regulation of the intestinal epithelial barrier. *Front Nutr*. 2021;8:718356. doi:10.3389/fnut.2021.718356
81. Fu L, Cherayil BJ, Shi H, Wang Y, Zhu Y. Food Allergy: From Molecular Mechanisms to Control Strategies. 2019th ed. Springer; 2019. doi:10.1007/978-981-13-6928-5
82. Vojdani A, Cooper E. Antibodies against CNS antigens in autism: Possible cross-reaction with dietary proteins and infectious agent antigens. *Neuropsychiatric Disorders and Infection*. Published online March 10, 2005:171-186. doi:10.3109/9780203007648-20