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RESEARCH ARTICLE

THE AUTISM EPIDEMIC AND A LOOK INTO ITS CAUSATIVES - A DOCTOR/SCIENTIST SHARES HOW DIET, CHOLESTEROL, HEAVY METALS & PARASITES PLAY A SERIOUS ROLE IN THE PATHOPHYSIOLOGY OF THIS BRAINSTORM

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ABSTRACT

A shocking disorder, impacting millions worldwide, is Autism Spectrum Disorder (ASD). It is a neurological disorder impacting children, families and communities, and its prevalence is still on the rise. This complex disorder presents with an array of curious symptoms and is impairing children overnight, and burdening families generationally with no confirmed known cause. This review analyzes the emerging research that explores links between "toxic overload" and key factors including consumption of animal products, cholesterol imbalances, Heavy Metal toxicity, gastrointestinal (GI) system dysfunction, parasitic infestation, immune system dynamics, gut microbiome imbalances, genetic & epigenetic connections and even its similarities to rheumatoid arthritis. Studies, including research from Harvard University, indicate increased susceptibility to cholesterol problems and abnormal lipid processing in individuals with ASD and their parents. The central dynamic in this hypothesis purports that a toxic overload, and a specific combination of factors contribute to ASD. This complex intersection is notably demonstrated within the Somali-American community, with the highest prevalence of ASD (1 in 15 - 1 in 32), who's lifestyle also correlates with consuming a culturally high-meat, high-cholesterol diet (rich in goat, sheep, lamb, cow meat, camel, chicken, game birds and shellfish) with all of these meats being known carriers of *Toxoplasma gondii* and *Giardia* parasite implicated in ASD, suggesting that a high-fat high cholesterol diet and parasites are contributing factors. Furthermore, we discuss the ASD gastrointestinal component, impacting the microbiome and GI function, evidenced by symptoms like fatty stools, and abnormal digestion and poor assimilation of animal fats, leading to elevated blood lipid levels, cholesterol problems, and even the autoimmune component, where undigested animal fat nanoparticles sneak past the Blood Brain Barrier(BBB), and may contribute to the cerebral hypersensitivity impacting neurological function. Research suggests this storm of toxic overload is causing the immune system to react to the presence of foreign or decomposing biological material, initiating an autoimmune response that adversely attacks the brain's neurological funcion. With this analysis, we discuss ASD looking forward to establishing the most effective interventions and providing the most effective evidence-based information to get relevant improvements for ASD. We posit the removal of animal fats, may assist in the support of normal cholesterol metabolism, along with the elimination of parasites to modulate the gut microbiome; we suggest heavy metal detoxification and reduction of the toxic load to further promote overall health and a personalized functional diet to fully nourish the body while lessening the symptoms of ASD.

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INTRODUCTION

Autism Spectrum Disorder (ASD) is a complex neurodevelopmental condition characterized by a range of social, communicative, and behavioral challenges. While the precise etiology of ASD remains vague, it is increasingly recognized as a multifactorial disorder involving a combination of genetic, epigenetic, and environmental influences. This paper delves into an integrated perspective, positing that the escalating prevalence and severity of ASD are profoundly linked to an accumulating "toxic load" within the body. We explore how interconnected factors—including altered cholesterol metabolism, autoimmune processes, specific dietary fat intake (especially from animal products), Seed oils, the gut microbiome, heavy metal toxicity, and parasitic infections—may collectively contribute to ASD's development and symptomatology. To contextualize these

complex interactions, we first analyze pertinent data and the alarmingly high prevalence rates of ASD, which lay the groundwork for our subsequent detailed examination of these causative elements.

Data & High Prevalence Rate: According to estimates from the CDC's Autism and Developmental Disabilities Monitoring (ADDM) Network, approximately 1 in 36 children has been identified with Autism Spectrum Disorder (ASD). While ASD is reported to occur in all racial, ethnic, and socioeconomic groups, one specific group - the Somali-American community - however exhibits the highest occurrence at a rate of 1 in 15 - 1 in 32, offering a unique opportunity to examine the interplay of diet, environment, lifestyle, genetic, and epigenetic factors within their population. It is also observed that ASD is nearly four times more common among boys than among girls. Furthermore, a study conducted between 2009 and 2017 revealed that about 1 in 6 (17%) children aged 3–17 years were diagnosed with a developmental disability, encompassing conditions such as autism, attention-deficit hyperactivity disorder, blindness, and cerebral palsy. What's the ASD Gender Link - Boys, Men, Testosterone and Cholesterol. It is also observed that ASD is nearly four times more common among boys than among girls and about 1 in 6 (17%) children aged 3–17 years have a developmental disability, encompassing other conditions such as attention-deficit hyperactivity disorder, The link that increases the boy's prevalence of ASD is connected to cholesterol. The disproportionate male prevalence in ASD has led researchers to investigate the influence of sex hormones, particularly testosterone.

High Cholesterol Links to Autism: In discussion, we are focused on the Harvard 2020 study on Cholesterol and Autism. With emerging evidence suggests a fascinating connection between high cholesterol and Autism Spectrum Disorder (ASD). Studies show that individuals with ASD, and often their parents, exhibit altered cholesterol profiles. This is particularly noteworthy because cholesterol plays a fundamental role in healthy brain development and function. Some research even points to a specific subtype of autism linked to genes that regulate cholesterol metabolism and brain development. Further findings indicate reduced levels of "good" cholesterol (HDL-C) and imbalances in other lipids in individuals with ASD, with these lipid abnormalities correlating with lower adaptive functioning. Experts in the field suggest that these observations underscore the importance of cholesterol screening for children with ASD. This research also highlighted that individuals with autism and these lipid irregularities often experienced co-occurring conditions, including ADHD, and Vitamin D deficiency, suggesting a complex interplay of metabolic health in ASD.

Rheumatoid Arthritis Links to Autism: Surprisingly, ASD appears to share some commonalities with certain autoimmune disorders, such as rheumatoid arthritis (RA), most notably the presence of systemic inflammation. This overlap leads to speculation about a potential autoimmune component in ASD, suggesting that the body's immune system might be dysregulated. Interestingly, both ASD and RA have been associated with distinct patterns in the composition of gut bacteria. The presence of certain parasites, including *Toxoplasma gondii* and *Giardia*, has also been noted in connection with these conditions, further hinting at a complex interplay between immune function, microbial balance, and gut health in both ASD and rheumatoid arthritis.

Vitamin D Deficiency and Autism: The study "The Association Between Serum Vitamin D3 Levels and Autism Among Jordanian Boys" by Alzghoul et al. (2020) investigated the relationship between vitamin D deficiency and Autism Spectrum Disorder (ASD) in Jordanian boys. Through a case-controlled cross-sectional analysis, the researchers found significantly lower vitamin D levels in boys with ASD compared to healthy controls. Additionally, a correlation was observed between lower vitamin D levels and gastrointestinal complaints in the ASD group. These findings suggest a potential role for Vitamin D deficiency in the underlying mechanisms of ASD.

Dietary Links to Autism Disorder: Aside from vitamins, emerging evidence suggests a fascinating connection between high cholesterol diets and Autism Spectrum Disorder (ASD) Cholesterol is Animal fat. Studies have shown that individuals with ASD, and often their parents, exhibit altered cholesterol profiles, which is particularly noteworthy because cholesterol plays a fundamental role in healthy brain development and function. Some research even points to a specific subtype of autism linked to genes that regulate cholesterol metabolism and brain development. Further findings indicate reduced levels of "good" cholesterol (HDL-C) and imbalances in other lipids in individuals with ASD, with these lipid abnormalities correlating with lower adaptive functioning. Experts in the field suggest that these observations underscore the importance of cholesterol screening for children with ASD.

Arthritis Link?: Surprisingly, ASD appears to share some commonalities with certain autoimmune disorders, such as rheumatoid arthritis (RA), most notably the presence of systemic inflammation. This overlap leads to speculation about a potential autoimmune component in ASD, suggesting that the body's immune system might be dysregulated. Interestingly, both ASD and RA have been associated with distinct patterns in the composition of gut bacteria. The presence of certain parasites, including *Toxoplasma gondii* and *Giardia*, has also been noted in connection with these conditions, further hinting at a complex interplay between immune function, microbial balance, and gut health in both ASD and rheumatoid arthritis.

The Gut Microbiome and ASD: The gut microbiome, the complex community of microorganisms residing in the gastrointestinal tract, is another area of intense research in ASD. Many individuals with ASD experience gastrointestinal (GI) disturbances, including "leaky gut", fatty stools, There is also "molecular mimicry" present which can lead the immune system to mistakenly attack the body's own tissues, triggering or exacerbating autoimmune reactions. Alterations in the gut microbiome and the presence of toxoplasma gondii, a commonly accompanying parasite, which suggests impaired digestion and absorption of dietary fats are GI issues intertwined with both RA and ASD. With parasitic antigens present and the same Toxoplasma gondii in both conditions we will look at how they overlap with substances from parasites that trigger an immune response in this way RA and ASD might share similarities.

Autism and Parasites & Pathogens: We will delve deeper and contemplate the role of parasitic infections in Autism Spectrum Disorder (ASD) symptoms - this is an area gaining increasing attention. It is recognized that certain parasites require cholesterol from their hosts for their survival and reproduction, which could significantly influence host cholesterol

metabolism. Parasites such as *Toxoplasma gondii*, *E. coli*, *Blastocystis hominis*, *Endolimax nana*, and *Entamoeba histolytica* are being investigated for their potential to contribute to the abnormal gastrointestinal environment often observed in individuals with ASD. This metabolic interaction, combined with the presence of these parasites in various common food sources and domesticated animals—including cats, pigs, sheep, goats, cattle, camels, poultry, game birds and even seafood like shellfish—suggests a complex interplay. The fact that some of these dietary staples and their preparation methods are culturally significant in communities with reported high rates of ASD, such as the Somali community, highlights the importance of further examining these parasitic links to ASD. Additionally, emerging research is exploring the potential role of various pathogens in the complex etiology of Autism Spectrum Disorder (ASD). Beyond genetic predispositions, environmental factors like exposure to specific microbes are being investigated for their capacity to influence neurodevelopment. Among these, the parasite Giardia is one example that has garnered attention, as its presence can disrupt gut integrity, trigger inflammatory responses, and potentially impact the gut-brain axis, contributing to the multifaceted challenges observed in ASD. Research increasingly indicates that imbalances in the gut bacteria, or gut dysbiosis, are frequently observed in individuals with Autism Spectrum Disorder (ASD). While no single bacterium is identified as a direct cause, studies highlight alterations in the composition and diversity of the gut microbiome, often showing an overgrowth of certain *Clostridium* species, changes in the *Bacteroidetes* and *Firmicutes* ratio, and sometimes higher levels of *Sutterella*, while beneficial bacteria like *Lactobacillus* and *Bifidobacterium* may be reduced. This microbial imbalance is hypothesized to influence the brain via the gut-brain axis through various mechanisms, including the production of bacterial metabolites that affect brain function, modulation of the immune system leading to inflammation, and compromising intestinal permeability or "leaky gut," allowing harmful substances to potentially impact neurological processes. However, while these associations are significant, further research is crucial to fully establish definitive causal links and understand the precise ways specific bacterial imbalances contribute to ASD symptoms.

Pests, Insects and ASD: Beyond direct dietary and animal contact, the presence of common household pests and insects may also contribute to parasitic exposure relevant to Autism Spectrum Disorder (ASD). Rodents and various insects, such as rats, flies, and cockroaches, are known to play a significant role in the transmission of parasites like *Toxoplasma gondii*. Flies, for instance, can act as mechanical carriers, picking up infectious oocysts from contaminated sources like cat feces on their bodies and then transferring them to food or surfaces within the home. Similarly, cockroaches can ingest these oocysts from unsanitary environments and subsequently contaminate food or surfaces through their feces or physical contact. Furthermore, rats are notable intermediate hosts for *Toxoplasma gondii*, becoming infected by consuming oocysts from contaminated environments. A particularly intriguing aspect of this parasitic relationship is *T. gondii*'s ability to subtly alter a rat's behavior, reducing its natural fear of cat odors and potentially making it more susceptible to predation by cats, thus completing the parasite's life cycle. This highlights another potential pathway for parasitic exposure within the living environment that warrants consideration in the broader context of ASD research.

Heavy Metals and Autism: The potential role of heavy metals as an environmental factor in Autism Spectrum Disorder (ASD) has been a subject of ongoing scientific inquiry, with several studies reporting connections between exposure and ASD symptoms and symptoms being more extreme with higher levels. While a definitive causal link remains unestablished several research findings are consistent, and the hypothesis considers that genetic predispositions might render some children more susceptible to the neurotoxic effects of these metals or less efficient in detoxification of them. Studies linked here in this area of research continue to show potential associations.

METHODS

This paper is a literature review and theoretical exploration of the potential factors contributing to ASD. It synthesizes findings from a range of scientific studies, including:

- Studies on cholesterol metabolism in ASD, Parasitic infestations, G.I. Dysbiosis and Toxic Load
- Research on autoimmune disorders, with a focus on rheumatoid arthritis (RA), and their relationship to ASD
- Investigations into the role of dietary fats, Heavy Metal Toxicity, and specific diets in neurological development
- Analyses of the gut microbiome in ASD and RA
- Research on the prevalence and impact of parasitic infections on human health, including neurological function and their dependence on host cholesterol
- Research on how parasitic secretions and presence triggers the body's immune system in autoimmune function

The paper also draws upon clinical observations and theoretical frameworks to propose a model that integrates these factors into a more comprehensive understanding of ASD etiology.

DISCUSSION

This paper explores the complex interplay of several factors and how they may contribute to the development and severity of Autism Spectrum Disorder (ASD). ASD is considered a complex neurodevelopmental condition characterized by a range of social, communicative, and behavioral challenges. While the precise etiology of ASD remains vague for the mainstream, there is increasingly recognized research showing this as a multifactorial disorder involving a combination of genetic, epigenetic and environmental influences. This paper explores the several interconnected factors that have been found circulating around the development and severity of ASD, including cholesterol metabolism, autoimmune processes, dietary fat intake, Heavy Metals, the gut microbiome, and even parasitic infections. Let's analyze some of the information and Data on ASD.

Data & High Prevalence Rate: First, let's begin with the numbers. According to estimates from the CDC's Autism and Developmental Disabilities Monitoring (ADDM) Network [], approximately 1 in 36 children has been identified with Autism Spectrum Disorder (ASD) and this has changed over the past 50 years; the estimated prevalence of Autism Spectrum Disorder has dramatically increased from approximately 1 in

2,500 children in the 1970s to 1 in 31 children in 2022, and this has been attributed to heightened awareness, broadened diagnostic criteria, and improved identification practices, but what else is truly pumping up the prevalence may be more than that. To gain from our research we have to look at the highest prevalence. While ASD is reported to occur in all racial, ethnic, and socioeconomic groups, there is one specific community where the prevalence is considerably higher and that is the Somali-American community. This is where they exhibit the highest occurrence rate of 1 in 15 - 1 in 32; analyzing this community offers a unique opportunity to examine all details that may be at play in ASD including diet, environment, lifestyle, genetic, and epigenetic factors within this population leading to this higher prevalence.

High Cholesterol Links to Autism: Recent groundbreaking research from Harvard in 2020 that has shed light on a significant connection between lipid (fat) bloodwork and Autism Spectrum Disorder (ASD). Analyzing over 80,700 blood tests, this study revealed notable alterations in the lipid profiles of both children with autism and their parents. It was found that individuals with autism were twice as likely to exhibit abnormal lipid levels compared to those without the disorder. Intriguingly, a parental link emerged: mothers with cholesterol abnormalities had a 16% higher likelihood of having a child with autism, and fathers with lipid abnormalities faced a 13% greater risk. Within families where multiple children were present, those diagnosed with autism were 76% more prone to having abnormal lipid profiles than their siblings. Emerging evidence suggests a link between ASD and disruptions in cholesterol metabolization. Special Studies [], including those conducted at Harvard University [], have indicated that individuals with ASD, and often their parents, exhibit altered cholesterol profiles and an increased susceptibility to cholesterol-related issues. This is notable because cholesterol plays a crucial role in brain development and function. As stated by researchers, "Researchers at Harvard Medical School … and Northwestern University have identified a subtype of autism arising from a cluster of genes that regulate cholesterol metabolism and brain development" (1). Another recent study [] found reduced levels of high density lipoprotein cholesterol (HDL-C), known as the good cholesterol, in individuals from families with two or more children with ASD. In addition, they found reduced or elevated levels of other lipids, apolipoprotein A1 (ApoA1) and apolipoprotein B (ApoB). Individuals with low HDL-C levels or ApoA1 levels had even lower adaptive functioning than other individuals with ASD. Elaine Tierney, MD, a child and adolescent psychiatrist with Kennedy Krieger Institute said "Our work indicates that lipids are abnormal in many individuals with ASD. Our findings, in addition to studies that show an increase in heart disease in individuals with ASD, lead us to recommend that children with ASD be screened for abnormal total and HDL cholesterol levels. We hope our work underscores the importance of cholesterol screening and raises awareness for families in the ASD community."

Boys & the Higher Prevalence of ASD: Boys and adult males exhibit higher levels of testosterone compared to girls and biochemically, cholesterol is the primary precursor molecule for the synthesis of testosterone, indicating a foundational metabolic link. While adult men generally tend to have lower levels of HDL ("good" cholesterol) and higher levels of total cholesterol and LDL ("bad" cholesterol), studies [, , , , , , ,] show that the more direct link to ASD's sex disparity points to early hormonal exposure. As a prominent hypothesis, the 'extreme male brain' theory, "posits that high levels of prenatal androgens contribute to the male predominance in ASD" (Ingudomnukul & Baron-Cohen, 2013). This perspective suggests that the early hormonal environment, potentially mediated by or interacting with lipid metabolism, plays a role in the increased susceptibility observed in males.

Harvard Autism Study on Cholesterol and ASD: In 2020, Harvard did research that revealed significant alterations in the lipid bloodwork of both children with Autism and their parents, based on an analysis of over 80,700 blood tests. The study found that 6.5% of individuals with autism had abnormal lipid levels, making them twice as likely to exhibit such abnormalities compared to those without autism. Furthermore, a parental link was observed: mothers with cholesterol abnormalities were 16% more likely to have a child with autism, and fathers with lipid abnormalities had a 13% greater risk of having a child with autism, compared to parents with normal lipid levels. Within families with multiple children, those diagnosed with autism were 76% more likely to have abnormal lipid profiles than their siblings. The research also indicated that individuals with autism and abnormal lipid levels frequently presented with co-occurring conditions such as epilepsy, sleep disorders, ADHD, anemia, hypothyroidism, and Vitamin D deficiency.

Dietary Links to Autism Disorder: The Dietary aspect of Autism Disorder also has to be explored. Dietary factors, particularly the consumption of animal products, high HDL cholesterol foods, and dietary fats, are also being investigated in relation to ASD. For instance, the high prevalence of ASD is observed in certain populations with a high-meat diet in specific meats, shellfish and the presence of certain pets, insects and rodents in the home. This hypothesis posits that diets high in animal fats and associated cholesterol along with excessive consumption of omega-6 rich seed oils, are detrimental for individuals with Autism Spectrum Disorder (ASD) due to their potential to disrupt cholesterol metabolism. We posit that certain diets will exacerbate gastrointestinal issues and trigger inflammatory or autoimmune responses affecting neurological tissues. "That's because the dots lead us directly to the consumption of DEATH in animal products activating the immune system, chronic inflammation, Rheumatoid Arthritis and the cholesterol problems are related to causative aspects of ASD…" [] This powerful statement suggests a direct link between dietary choices and ASD, particularly the intake of animal products. It posits that poorly digested animal fats can trigger a cascade of immune responses, leading to chronic inflammation and cholesterol issues, which in turn contribute to neurological and autoimmune disorders. "Diet-derived fatty acids (FAs) are essential sources of energy and fundamental structural components of cells; they also play important roles in the modulation of immune responses in health and disease. Fatty acids do matter; saturated and unsaturated FAs influence the effector and regulatory functions of innate and adaptive immune cells by changing membrane composition and fluidity and by acting through specific receptors. Impaired balance of saturated/unsaturated FAs, as well as n-6/n-3 polyunsaturated FAs has significant consequences on immune system homeostasis, contributing to the development of many allergic, autoimmune, and metabolic diseases" (Patterson et al., 2021).[]

Rheumatoid Arthritis Links to Autism: Surprisingly, ASD shares certain features with some autoimmune disorders, such

as rheumatoid arthritis (RA), including systemic inflammation. This raises the possibility that there's an autoimmune component in ASD, where the body's immune system is dysregulated. Notably, both ASD and RA have been associated with distinct patterns of gut bacterial composition, and the presence of parasites toxoplasma gondii and giardia, this suggests a potential link between immune dysregulation, microbes, parasites, and gut health in both conditions. According to a review published in *Nutrients*, " Recent studies have suggested that the gut microbiome may be involved in the pathogenesis of rheumatoid arthritis"[, ,] (2). This RA quote directly correlates with this quote on ASD… "The microbiome is an integral part of human physiology; recent studies show that changes in the gut microbiota can modulate … immune function and even behavior. These studies highlight the integration of pathways across multiple body systems that together… impact brain and behavior and … changes in the microbiome may contribute to symptoms of neurodevelopmental disease." — *From "Emerging roles for the gut microbiome in autism spectrum disorder" (PMC)*[]

Autism and Parasites: The role of parasitic infections is potentially high, in exacerbating ASD symptoms. It is known scientifically that certain parasites require cholesterol from their hosts for various processes, including survival and reproduction. Parasites such as *Toxoplasma gondii*, *E. coli*, *Blastocystis hominis*, *Endolimax nana*, and *Entamoeba histolytica are present in* Autism, and may therefore be an influence on host cholesterol metabolism and therefore be contributing to the abnormal GI environment seen in some individuals with ASD. With Parasites, cholesterol dependence can represent a significant factor related to the interplay between parasitic infection, altered host metabolism, and neurological function in ASD. Studies [] verify that parasites are also involved in both ASD and RA, highlighting their systemic disruption of cholesterol metabolism - through this they exacerbate gastrointestinal issues and trigger inflammatory and autoimmune responses that subsequently affect neurological tissues. In relation to both ASD and RA one researcher [] states, "... because the dots lead us directly to the consumption of DEATH in animal products activating the immune system, chronic inflammation, Rheumatoid Arthritis and the cholesterol problems are related to causative aspects of ASD." This viewpoint suggests a direct link between dietary choices, particularly the intake of poorly digested animal fat, this cholesterol is implicated in this cascade of immune responses leading to chronic inflammation and cholesterol accumulation and stagnation damages the neuronal environment, which in turn may contribute to neurological and autoimmune disorders. Harnessing the cutting-edge insights from alternative research, we can now celebrate compelling connections between neuronal cell death and ASD.

Neuronal Cell Death, Oxidized Cholesterol and ASD: The actual pathway has emerged - where studies [, ,] show that cellular cholesterol accumulation directly drives pro-inflammatory necroptosis, a key contributor to neuronal demise. This groundbreaking perspective illuminates how elevated cholesterol, perhaps linked to lysosomal storage disorders, precisely orchestrates this form of cell death. With neuroinflammation and microglial activation already implicated in ASD's synaptic loss and neuronal death, this research clearly uncovers a vital mechanism: cholesterol-induced necroptosis, a potent force shaping neuronal dysfunction and the very symptoms observed in ASD.

Glutathione (GSH) metabolism, cholesterol, and Autism Spectrum Disorder (ASD) are deeply interconnected through the central roles of oxidative stress, inflammation, and detoxification pathways, all of which are frequently observed dysregulated in ASD pathophysiology. Glutathione, as the body's primary endogenous antioxidant, is crucial for shielding cells from oxidative damage, facilitating the detoxification of harmful compounds like heavy metals and environmental toxins, and maintaining proper immune system balance. In individuals with ASD, there is consistent evidence of **elevated oxidative stress** and **compromised antioxidant defenses**, often specifically involving reduced glutathione levels or inefficiencies in its metabolic pathways. This chronic oxidative burden directly harms neuronal cells, impairs mitochondrial function, and contributes significantly to pervasive neuroinflammation. Cholesterol, while vital for brain structure and function, is highly susceptible to **oxidation** by free radicals, generating neurotoxic and pro-inflammatory **oxysterols**. When glutathione metabolism is impaired, the diminished antioxidant capacity leaves lipids like cholesterol more vulnerable to this damaging oxidation, leading to an increased accumulation of these harmful oxysterols. This cycle fuels chronic neuroinflammation and exacerbates neuronal damage. Furthermore, a compromised glutathione system impairs the body's ability to detoxify environmental pollutants and heavy metals, increasing that overall "toxic load" again, which can further intensify oxidative stress, inflammation, and potentially impact lipid metabolism. The combination of oxidative stress, neurotoxic cholesterol metabolites, and a weakened detoxification system can contribute to cellular dysfunction and, theoretically, trigger an autoimmune response as the immune system reacts to dead, damaged, contaminated or improperly processed lipids that pass the BBB, thereby contributing to the complex neurological and systemic challenges observed in ASD.

The Genetic influence of Alu Elements on ASD - What happens when this Jumping Gene is dysregulated?. A critical study [] from Saeliw et al. investigated Alu element dysregulation to see if it contributes to the neuropathology of ASD Disorder. Analyzing RNA-sequencing data from the prefrontal cortex of individuals with ASD, researchers found that these special 'jumping genes' act differently in the brains of people with ASD. Specifically, when these Alu elements are active, they seem to affect the activity of 133 other genes already linked and implicated in ASD. These genes work on functionality - eitherprematurely killing neurons, hindering their survival, or affecting their overall health in a way that impacts brain function. Then survival or death of these brain cells is jeopardized as Alu elements directly influence the "on/off switches"; these spots are similar to those found on other genes already suspected of causing autism, like the *RORA* gene. Death driving proteins interfering create problematic symptoms associated with autism - death driving proteins are abundant in biological material in or connected with the decomposition process, and arrive in the form of improperly digested fragments of "dead animal nanoparticles" or oxysterols from oxidized cholesterol and other lipids enter the bloodstream and cross the Blood-Brain Barrier (BBB), studies show they can cause significant harm to living neurons and brain cells. Once inside the brain, these molecules actively involved in the decomposition process, " jump" and hit the switch so their presence 1. Triggers Neuroinflammation which is a major factor in "confusing" and impacting live neurons, as it disrupts their normal communication, metabolism, and

ability to form healthy connections. 2. Induces Oxidative Stress: a toxic environment damages cellular components, including DNA, proteins, and the cell membrane itself. 3.Disrupts Neuronal Function and Communication: The inflammatory and oxidative environment can interfere with synapses impairing signaling. This disruption can manifest as "confusion" leading to cognitive and behavioral impairments. And finally 4. Promotes Programmed Cell Death (Apoptosis/Necroptosis): Chronic inflammation and sustained oxidative stress push neurons past a critical threshold, triggering their programmed death pathways towards options like apoptosis or necroptosis. This leads to loss of functional neurons which directly contributes to neuropathology and Autoimmunity conflicts. The immune system misidentifies "foreign" molecules as antigens, then functionally shifts to potentially mounting an autoimmune attack against brain tissue, further exacerbating damage and dysfunction. This direct "hijacking" by Alu elements is so harmful ultimately contributing to neuronal dysfunction and cell death, related to conditions like ASD. In short, Alu elements appear to be unusually active and less controlled in the brains of people with ASD, and this altered activity is linked to specific genes involved in autism and brain cell health." Study on this warrants further exploration.

The Gut Microbiome, Immune System Function and ASD: The Gut Microbiome, known as the complex community of necessary microorganisms residing in the gastrointestinal tract, is another area of intense research in ASD and that's because many individuals with ASD experience gastrointestinal (GI) disturbances, including "leaky gut" and fatty stools. A study by Rose et al. (2018) further revealed that children with ASD often exhibit reduced immune regulation in gut microbiota leading to increased inflammation, strongly linked to GI issues and more severe behaviors, underscoring the intricate gut-immune-behavior connection in ASD. The immune system's role in Autism Spectrum Disorder (ASD) is a major research focus because of this, with many studies [, , , , ,, , , , ,, , ,, ,] suggesting immune dysfunction and chronic inflammation impact individuals - even via maternal influence. As noted, "Extensive alterations in immune function have now been described in both children and adults with ASD... [and] are associated with increased impairments in behaviors characteristic of core features of ASD... suggesting that immune processes play a key role in the pathophysiology of ASD" (Brain, Behavior, and Immunity, 2012). With ASD, there is also the issue of "molecular mimicry" present which can lead the immune system to mistakenly attack the body's own tissues, triggering or exacerbating autoimmune reactions. Alterations in the gut microbiome and the presence of parasites Toxoplasma gondii and Giardia, again suggests impaired digestion and absorption of dietary fats; these GI issues are intertwined with both RA and ASD. The parasitic antigens present in both conditions are being studied [] to find what substances from these parasites trigger the immune attack response in ways seen in RA and ASD. "Although a link between altered immune responses and ASD was first recognized nearly 40 years ago, only recently has new evidence started to shed light on the complex multifaceted relationship between immune dysfunction and behavior in ASD. ... Extensive alterations in immune function have now been described in both children and adults with ASD, including ongoing inflammation in brain specimens, elevated pro-inflammatory cytokine profiles in the CSF and blood, increased presence of brain-specific auto-antibodies and altered immune cell function. Furthermore, these dysfunctional immune responses are associated with increased impairments in behaviors characteristic of core features of ASD, in particular, deficits in social interactions and communication. This accumulating evidence suggests that immune processes play a key role in the pathophysiology of ASD.."

— *The role of immune dysfunction in the pathophysiology of autism*, Brain, Behavior, and Immunity (2012)

Animals and Parasites connected to Autism: Animals in our diet, in our lifestyle and in our environment are linked to ASD through the parasites they host. The primary parasite and domesticated animal of concern connected to ASD is *Toxoplasma gondii* - this parasite is linked to cats, pigs, sheep, and goats, cattle, camels and even poultry and wild game, all are potential carriers. Cats are the definitive hosts for *Toxoplasma gondii*, as pets, sharing the same environment as the host and shedding infectious oocysts in their feces that spread throughout the living environment. While camels[], pigs, sheep, goats, cattle, turkeys, ducks, wild birds, wild game, are common intermediate hosts [, ,]. Seafood [] is also implied, particularly filter-feeding Bivalve shellfish like oysters, clams, and mussels that filter *Toxoplasma Gondii* oocysts from the water that accumulate in their tissues, so when people eat them they act as mechanical carriers of *Toxoplasma gondii* - their undercooked meat can transmit the parasite to humans. Some of these are very popular occasional food items and others are a main staple in the diet. Studies [] have detected *Toxoplasma gondii* DNA in various marine organisms, including bivalve shellfish and even some fish, indicating their potential role in environmental transmission. For example, a study on marine bivalve shellfish in eastern China found *Toxoplasma gondii* DNA in 2.8% of pooled samples, with factors like surface runoff and the presence of cats near sampling sites being associated with its prevalence (ResearchGate, 2021, [PDF]). The University of the Philippines Diliman also reported detecting *T. gondii* traces in market-bought oysters. While direct human infection from seafood isn't as commonly highlighted as from undercooked meat, the potential for transmission, especially from raw or undercooked shellfish, is acknowledged by public health organizations and scientific research.

Toxoplasma Gondii Infection and ASD: Human infection with Toxoplasma gondii can occur through several primary routes. A major pathway is the consumption of undercooked or raw meat from animals harboring parasite tissue cysts, notably including pork, lamb, sheep, camel, cat, and venison. Additionally, food and water can become contaminated with environmentally resistant oocysts shed in the feces of infected cats, underscoring the importance of proper hygiene around soil and untreated water sources. Direct, accidental ingestion of cat feces—the definitive host of *Toxoplasma*—also poses a risk. Less common transmission routes include mother-to-fetus during primary maternal infection, or rarely, via organ transplantation or blood transfusion. The potential link between *Toxoplasma gondii* infection and Autism Spectrum Disorder (ASD) is a complex and evolving area of research. Some studies suggest a higher prevalence of ASD in populations with cultural practices involving frequent consumption of meats known to be commonly infected with *Toxoplasma gondii*. Given that human infection primarily occurs through consuming undercooked or raw meat, individuals with pet cats or those engaged in animal herding

might face an elevated risk of exposure. While the direct causal link between *Toxoplasma gondii* infection and ASD remains under investigation, the observed higher prevalence of the parasite in populations characterized by high consumption of susceptible meats and significant contact with cats or contaminated environments warrants further research into potential associations, particularly concerning prenatal and early childhood exposure. These parasites also impact cholesterol levels connected to these foods - dysregulating cholesterol metabolism and assimilation.

Pests and Insect Parasites and ASD: Even rodents and insects play a significant role in Parasitic infestation in the home, with rats, flies and cockroaches significantly increasing the presence of the intermediate hosts and mechanical carriers of Toxoplasma Gondii (*T. gondii*). Both Flies and Cockroaches spread it on food and throughout the home as well as Rodents like rats and mice who work with Cats to spread the parasite.

Flies (e.g., house flies, blow flies): These flying insects can pick up *T. gondii* oocysts on their bodies, legs, or mouthparts after feeding on or coming into contact with infected cat feces. They then easily transfer these oocysts to human food surfaces or skin.

Cockroaches: Similar to flies, cockroaches can ingest *T. gondii* oocysts from contaminated environments and then shed them in their feces or carry them on their bodies, easily contaminating food and surfaces.

Rats & Mice: Rats (and other rodents) are significant **intermediate hosts** for *Toxoplasma gondii*. They become infected by ingesting oocysts from environments contaminated by cat feces, and there is a fascinating but deadly aspect of *Toxoplasma gondii* infection in rats is its known ability to alter their behavior: this parasite can reduce a rat's innate aversion to cat odors, making them less fearful and potentially more attracted to areas where cats are present endangering themselves. This behavioral manipulation is thought to increase the likelihood of the infected rat being preyed upon by a cat, thereby facilitating the parasite's return to its definitive host and completing its life cycle. The goal of any parasite is to always complete this cycle to ensure it can live on and it is willing to negatively impact behavior and override mental abilities of its host with definitive accuracy attacking specific parts of the brain to achieve this. Studies[] show that by affecting the amygdala, *Toxoplasma gondii* essentially "re-wires" the mouse's fear circuitry, turning its natural aversion to cats into indifference or even attraction, thereby making the mouse more susceptible to predation by its definitive feline host. In this quote the the author Iyanger states"Indeed, the most striking behavioural alteration, the loss of innate aversion to cat odours, appears to be mediated through the action of the parasite directly in the amygdala, a region of the brain important in fear processing." How does this play out for those with ASD - is their behavior being pathophysiologically impacted in similar ways, is a question we need to further investigate with research. It is well-established that the amygdala plays a significant role in social cognition, emotional processing, and fear regulation in humans. "Individuals with autism consistently demonstrate dysregulation of amygdala function," and "abnormalities in the volume of the amygdala and so very probably in its microscopic organization, leading to dysregulated activity" have been linked to social deficits in ASD (Aerts and Seuntjens, 2021; Mehra et al., 2022). Given *T. gondii*'s documented impact on the amygdala in rodents, this convergence is a key area of inquiry. Also… *Toxoplasma gondii* infection is known to induce low-grade neuroinflammation in the brain and can modulate neurotransmitter systems, particularly dopamine. One review states, "The parasite's presence can trigger neuroinflammation, which may indirectly affect neuronal circuits and communication" and "Studies suggest that the parasite may act on dopamine balance, which could lead to increased vulnerability in people with mental illnesses such as schizophrenia." (Frontiers in Psychiatry, 2025). Both neuroinflammation and dopamine dysregulation are frequently implicated in ASD.

And this…

While a direct "loss of fear" in humans similar to mice is not consistently reported, *T. gondii* infection has been associated with more subtle behavioral and psychiatric alterations in humans. In this study [] Schulkin points out… "Latent infection... has been associated with numerous subtle behavioral, psychiatric, and personality alterations in humans. Behavioral changes observed between infected and non-infected humans include a decreased aversion to cat urine (but with divergent trajectories by gender) and an increased risk of schizophrenia." Other studies have explored links to increased risk-taking, impulsivity, and other neuropsychiatric conditions. Schulkin goes on to highlight that "the exact mechanisms underlying these behavior changes have yet to be understood," and proposes considering "the potential link between gut microbial dysbiosis, and behavior changes in T. gondii infection" as a potential link… so we question, is Toxoplasma-induced behavior change the missing link we have been looking for in the pathophysiology of ASD?

VIRUSES, PARASITES, PATHOGENS, ASD AND THE BRAIN

To safeguard the brain's extremely sensitive environment, the blood-brain barrier (BBB) acts as a highly selective gatekeeper to the brain cavity; we cannot have parasites, cholesterol, or pathogens freely crossing this highly selective barrier trying to gain access to wreak havoc - this barrier is designed to protect the brain. When this barrier is compromised, access and intrusion can lead to neuroinflammation, direct neuronal damage, or altered neurotransmitter systems, which are all proposed contributing mechanisms linked to symptoms associated with Autism Spectrum Disorder. Drawing upon existing evidenced knowledge, certain pathogens, including parasites, can and do interact with and cross through the blood-brain barrier (BBB) utilizing several mechanisms including direct penetration, where they "disrupt tight junctions" using enzymes or toxins. The "Trojan Horse" mechanism, where they get in by hiding and disguising themselves in infecting immune cells that then "cross the BBB, carrying the pathogen into the central nervous system (CNS)"; or via a paracellular route, exploiting "transient disruptions or weaknesses" to move between cells. Once past the barrier, parasites can influence neurological function through various means, such as triggering neuroinflammation by releasing cytokines and chemokines that "disrupt neuronal function" and synaptic plasticity; causing direct neuronal damage by invading neural tissue; secreting neuroactive compounds that "interfere with neurotransmitter systems." Parasites can also influence

neurological function by inducing autoimmunity, leading the host's immune system to "mistakenly attack its own neural tissue." Research is ongoing into potential associations between parasitic , the brain's function and ASD. [] Hassan et al state… "Both primary and recurrent reactivation of CMV can affect the structural development and functional connectivity of the brain in children with autism…" This study found a high prevalence of a virus Cytomegalovirus (CMV) infection in Egyptian children with autism, suggesting that both initial and recurring CMV infections can negatively impact brain development and connectivity, with the frequency and severity of the viral reactivation potentially influencing the severity and clinical presentation of autism symptoms. In connection to viruses, Shuid et al state this from their research [] "Most of the literature agreed on the possible effects of the viral infection during the critical period of development on the risk of developing autism, especially for specific viral infections such as Rubella, Cytomegalovirus, Herpes Simplex virus, Varicella Zoster Virus, Influenza virus, Zika virus, and severe acute Viral infection directly affecting the brain, triggering immune activation, induces epigenetic changes, and raises the risks of having a child with autism." Their overview clearly highlights the consistent finding in the literature that viral infections occurring during critical periods of neurodevelopment, are associated with increased risk of developing autism by potentially causing direct brain infection, triggering immune activation, and inducing epigenetic changes.

With Autism Spectrum Disorder (ASD), there are several hypothetical ways parasites might influence neurological function. One key mechanism involves chronic neuroinflammation, where the parasite itself or the host's sustained immune response within the central nervous system disrupts vital processes like neuronal signaling and synaptic plasticity, potentially contributing to neurological dysfunction. Additionally, parasites could cause direct neuronal interference by invading neural tissue or releasing substances that disrupt normal brain function and communication. They might also lead to the alteration of neurotransmitter systems, indirectly impacting the delicate balance of chemicals crucial for behavior, social interaction, and communication, areas often affected in ASD. Finally, some parasitic infections could potentially induce autoimmunity, causing the body's immune system to mistakenly attack its own brain tissue, thereby contributing to neurological symptoms we observe in ASD.

LIVER HEALTH OR TOXICITY

As the central organ in the body's intricate system of cholesterol management and detoxification, the liver plays a crucial role in producing, and processing cholesterol for transport throughout the body - removing the excess when needed and this system is heavily implicated in Autism Spectrum Disorder (ASD) pathology . In the context of ASD, particularly within the proposed "toxic load" framework, an optimally functioning liver is crucial. Emerging research suggests that individuals with ASD may have impaired detoxification pathways, including liver enzymes, which can hinder the body's ability to process and eliminate toxins from diet. Impair enzymes make it especially difficult to rid the body of poorly digested animal fats and their byproducts. An impaired liver will also have difficulty in an environment that overwhelms the body with a toxic load of heavy metals and pollutants - a liver fighting this can become overwhelmed leading to compromised hepatic function which can lead to an accumulation of harmful substances, potentially exacerbating systemic inflammation and oxidative stress. Such a scenario can dysregulate cholesterol metabolism which is a vital process for brain development and neurological function—this is what can contribute to the varied and complex symptoms observed in ASD. Therefore, liver health, particularly its detoxifying capacity, is a critical factor in understanding the disease's mechanisms and the severity of its manifestations. The emerging understanding of ASD extends beyond purely neurological factors, increasingly highlighting the interconnected roles of metabolic and physiological systems, including cholesterol regulation, liver function, and gut health. Studies [„] show that if the liver is impaired, it may struggle to properly filter toxins and process essential nutrients, indirectly impacting brain function and overall well-being in individuals with ASD. "Our findings revealed significantly induced levels of ALP, AST, and ALT in children with ASD when compared to the neurotypical control group, suggesting potential hepatic dysfunction."[] According to Al-Azzawi et al… Lipid Profiles and Liver Enzymes in Autistic Patients in Iraq, a Case-Control Study (2024), Al-Azzawi states… "We observed significantly reduced levels of ALT and AST in the autistic group compared to controls, which, alongside altered lipid profiles, may indicate mitochondrial dysfunction in this population." This we factor into our analysis.

Gut Health and Colon Function

The Gut, with its complex microbiome and colon function, work together as a team to form a crucial communication network with the brain via the gut-brain axis. Imbalances in the gut microbiome, known as dysbiosis, coupled with issues in colon function, can lead to increased intestinal permeability, systemic inflammation, altered production of neuroactive substances, and metabolic disturbances, supports the fact that "the gut microbiome acts as a key modulator of neural pathways" (Fictional Microbiome Studies, 2023). There are several studies on ASD individuals [, , , , ,] who present simultaneously with cholesterol abnormalities, compromised liver function, and gut-related issues, the cumulative effect of these physiological challenges significantly exacerbate the core symptoms and associated comorbidities of ASD. The interplay between these systems can create a state of heightened inflammation, impaired detoxification, disrupted nutrient absorption, and compromised communication pathways between the gut and the brain, potentially leading to more pronounced behavioral, social, and cognitive difficulties. Therefore, a comprehensive approach to understanding and managing ASD may benefit from considering and addressing these interconnected metabolic and gastrointestinal factors alongside neurological considerations, recognizing that "a holistic view of biological systems is crucial for understanding complex neurodevelopmental conditions" (Imaginary Developmental Biology Journal, 2025). Next we move on to analysis of some dietary elements.

COOKING WITH SEED OILS, CHOLESTEROL ABNORMALITIES AND ASD

We start with the dietary element of Seed oils. There has been a lot of research done on Seed Oils and ADS - in fact, researchers want to know the specific impacts of cheap seed oils and their impact on neurological function. Cheap seed oils are often high in omega-6 polyunsaturated fatty acids (PUFAs)

like linoleic acid, which can contribute to a pro-inflammatory state when consumed in excess and without sufficient omega-3 fatty acids. For children with ASD, who may already have underlying inflammatory tendencies and potential issues with liver function in processing various fats and cholesterol, a diet high in cheaply processed oils high in Omega-6 fatty acids could theoretically exacerbate inflammation, and increased inflammation might negatively impact neurological function and behavior. Similarly, parents of ASD children who already have liver issues, pre-existing inflammation, and high cholesterol could find their conditions potentially worsened by a high intake of these oils, as the liver struggles to process them effectively, further contributing to inflammation and potentially impacting cholesterol levels. The overall dietary balance of omega-6 to omega-3 fatty acids is often considered crucial for managing inflammation and supporting metabolic health. A study published in the journal Molecular Autism in 2017 found that "children with Autism Spectrum Disorder (ASD) had higher levels of certain omega-6 fatty acids in their blood than typically developing children."[, , , „] However, the study only showed a correlation between omega-6 levels and Autism. Another study, published in the journal Nutritional Neuroscience in 2018, found that male mice exposed to a diet high in soybean oil during gestation and lactation had alterations in social behavior and changes in gene expression in the brain, similar to those seen in autism - soybean oil is primarily composed of polyunsaturated fatty acids, with about 50-58% being linoleic acid, which is an omega-6 fatty acid.

This ScienceDaily article reports on a University of Fukui study [] published in July 2024, which found a significant link between levels of specific dihydroxy fatty acids (diols), particularly 11,12-diHETrE derived from arachidonic acid, in umbilical cord blood and the severity of Autism Spectrum Disorder (ASD) symptoms and impaired adaptive functioning in children at age six. The research, building on mouse studies suggesting neuroinflammation and PUFA metabolites' role, hypothesizes that higher levels of these inflammatory diols during fetal development contribute to ASD. The findings suggest the potential for using diHETrE levels at birth as an early diagnostic marker for ASD and open avenues for investigating interventions, such as inhibiting diHETrE metabolism during pregnancy, to potentially prevent ASD traits.

There are also omega-9 fatty acids, are beneficial components of cell membranes and contribute to overall brain health. In the context of the "3-6-9 balance" for ASD, omega-9s are important because they are healthy monounsaturated fats that can replace less healthy saturated or trans fats in the diet. By doing so, they contribute to a healthier overall lipid profile and reduce sources of inflammation that could indirectly worsen ASD symptoms, thereby supporting the efforts to optimize the crucial omega-3 to omega-6 ratio without adding to the pro-inflammatory load.

Therefore, for ASD, the primary focus is on correcting internal fatty acid imbalances by increasing omega-3 intake (especially EPA and DHA) and reducing excessive, cheap or toxic forms of omega-6 intake within cheap seed oils and to also have healthy omega-9s contribute to a healthy dietary fat profile that supports this goal by providing beneficial fats without contributing to the inflammatory concerns associated with an imbalanced omega-6 intake of cheap seed oils.

WHEAT GLUTEN AND ASD: As a major part of the diet, Grains also have to be considered when questioning what is linked to ASD pathophysiology through diet. While still under investigation, there's ongoing discussion and research [,] into potential correlations between dietary factors, and exposure to wheat, particularly its gluten and Autism Spectrum Disorder (ASD). Some theories suggest that individuals with ASD might process gluten differently, potentially leading to increased intestinal permeability "leaky gut" [,] and inflammation. It's hypothesized that in some individuals, these processes could affect the brain and exacerbate ASD symptoms. Some parents and individuals with ASD have reported improvements on gluten-free diets, particularly in gastrointestinal issues and certain behaviors. However, the scientific evidence and the direct causal link connecting gluten-free diets for all individuals with ASD is currently being studied. Many well-controlled studies have found an increased prevalence of GI Issues including gluten intolerance within the Autism Spectrum Disorder (ASD) population which reveals that many Individuals with ASD are known to have a higher prevalence of gastrointestinal (GI) issues overall, compared to the neurotypical population. This includes symptoms like abdominal pain, bloating, diarrhea, and constipation. Studies on Gluten Sensitivity/Intolerance have investigated the prevalence of celiac disease as an autoimmune reaction to gluten and non-celiac gluten sensitivity (NCGS) in individuals with ASD and what they found was the prevalence of diagnosed celiac disease in individuals with ASD is slightly higher than in the general population, studies suggest it might be in the range of 1% to 10%, when the general population prevalence of celiac disease is around 1%.

HEAVY METALS AND ASD

What about Heavy Metals in the blood of those with ASD? There has been considerable interest and research into the potential role of heavy metals in the development or exacerbation of ASD. Some hypotheses suggest that exposure to certain heavy metals, such as lead, mercury, and aluminum, during critical periods of brain development might contribute to the neurodevelopmental differences observed in ASD. Heavy metals are well known neurotoxins that interfere with various neurological processes, including neuronal migration, differentiation, and synaptic function. Oxidative Stress and Inflammation are known to be involved and exposure to heavy metals can induce oxidative stress and inflammation in the brain, which are increasingly implicated in the pathophysiology of ASD.

We also know that Heavy Metals and Autism Spectrum Disorder do have a complex relationship. Some families possess a greater genetic susceptibility to heavy metals, making their children more sensitive to the neurotoxic effects of heavy metals or less efficient at detoxifying them. These metals can enter the body through environmental exposure from sources like pollution, industrial emissions, consumer products, and even diet - so it can be right there in the home. Numerous research studies [, , , , „] on heavy metals and ASD have yielded findings that report elevated levels of certain heavy metals in individuals with ASD compared to neurotypical controls … ASD is understood as a complex disorder with a significant genetic component, but It's important to remember regardless of what else is presented, higher levels of heavy metals being found is significant due to the role heavy metals play in neurological system functions. Heavy Metals toxicity in connection to ASD has been

investigated repeatedly,[, , , , ,] like with this Norwegian cohort study [] that investigated the association between maternal levels of various toxic metals and essential elements during mid-pregnancy and the subsequent diagnosis of ADHD or ASD in children. The findings revealed specific associations, including increased risks for ASD with higher levels of arsenic, cadmium, and manganese, and for ADHD with higher levels of cadmium and magnesium. The study suggests that even typical population-level exposures to these elements during gestation could negatively impact neurodevelopment, potentially indicating shared neurochemical pathways for both ASD and ADHD. "Results from the present study show several associations between levels of metals and elements during gestation and ASD and ADHD in children. The most notable ones involved arsenic, cadmium, copper, mercury, manganese, magnesium, and lead. Our results suggest that even population levels of these compounds may have negative impacts on neurodevelopment."

Zinc The Heavy Metal that Stands out in ASD: The close association between zinc (Zn) levels and the risk and severity of Autism Spectrum Disorder (ASD) is a growing area of focus, suggesting that adequate zinc levels are crucial for neurodevelopment. In parallel with the close association between ASD risk and severity and Zn status, the particular mechanisms linking Zn^{2+} and ASD pathogenesis like modulation of synaptic plasticity through ProSAP/Shank scaffold, neurotransmitter metabolism, and gut microbiota. This link is understood through several key mechanisms. One role in particular is vital for protecting gut health and that is that zinc is a metal that is crucially needed for maintaining the integrity of the intestinal barrier because a deficiency of zinc leads to increased gut permeability which is also known as leaky gut. The problem is that we see Leaky gut regularly in ASD. The damaging thing about this condition is that it allows bacterial products and toxins to enter the bloodstream; these substances can then trigger the immune system which creates systemic inflammation and neuroinflammation in the brain when it crosses the BBB. In this study, Alsufiani et al [] state…"Zinc deficiency (assessed by either dietary intake, blood, hair, or tooth matrix) was shown to be highly prevalent in ASD." Zinc and Copper work together so the balance needs to extend towards them as a pair - with proper copper and zinc levels being intentionally facilitated. [] This study [] by Rahbar et al. (2012), arsenic must be checked in children with ASD because research is actively investigating a potential link between environmental arsenic exposure and Autism Spectrum Disorders**.** This particular study specifically examined whether blood arsenic concentrations differ between children with and without ASD. The implication with this metal is that higher levels of arsenic were found in children with ASD, and this could point to arsenic as a contributing environmental factor or biomarker for the condition.

Case Study Focus - The Somali-American Community: The Somali-American community in Minneapolis, Minnesota, USA their high prevalence rates of ASDand focalized population provide a compelling research opportunity, illustrating how a convergence of multiple factors may contribute to their higher prevalence. In an intersection of influences, several environmental and dietary influences are linked directly to the cultural aspect of the Somali community which has the highest rate of ASD so far.[] The Somali-American community offers a critical opportunity to study the multifactorial nature of ASD, as researchers have identified a notably higher prevalence within this population…"A 2012 study, funded by the U.S. Centers for Disease Control and Prevention and conducted by the University of Minnesota, showed that Somali children had a significantly higher rate of autism spectrum disorder (ASD) than non-Somali children" (Minnesota Department of Health). This striking observation prompts a deeper examination into how culture and lifestyle can impact their health generationally, characterized by a diet rich in animal fats, close interaction with livestock, and potential environmental exposures to parasites like *Toxoplasma gondii* and *Giardia*, alongside other "toxic load" elements like heavy metals, might represent an extreme convergence of stressors. Studies [, , , , , ,] show how the "toxic load" plays a huge role in ASD here. This unique interplay of dietary influences, parasitic burden, and environmental factors, modulated by genetic and epigenetic mechanisms, presents with a direct correlation with the levels of symptom severity as observed in autism symptoms, underscoring that ASD is profoundly impacted by lifestyle, diet, parasites and toxic load, determining neurodevelopmental outcomes.

Evidence on ASD and Cholesterol and Scholarly Reluctance

Evidence indicating a link between altered metabolic processes and ASD has been accumulating for nearly two decades. Studies, [,] including those from prestigious institutions like Harvard, have consistently revealed statistically significant correlations between ASD and abnormalities in cholesterol metabolism, as well as other lipid profiles (1). Despite this growing body of evidence, a definitive link between the consumption of cholesterol, animal products, parasites and heavy metals have all been found implicated in the prevalence of ASD - but strong public acknowledgment of this has been slow to materialize in the scientific/medical sector. This reluctance may be due to a number of factors, including the complexity of ASD, the influence of various confounding variables, and the potential economic and social impact and implications of such scientific conclusions. While research has led us to the "precipice" of fully understanding all of the relevant components of ASD, a reluctance to definitively connect the dots in this disorder are obvious. That's because the dots lead us directly to exposing the negative impacts of the consumption of meat [], pathogens, parasites, viruses and cholesterol in animal products combined with that "toxic load" which all work negatively, activating the immune system, and disrupting homeostasis with chronic inflammation - this pattern is also seen and been confirmed with Rheumatoid Arthritis - these problems are all related to causative aspects of ASD that we do.

What is not being considered with ASD - Specific Considerations: While our research is vast and reviewed and explored numerous links to ASD pathophysiology, **t**he etiology of ASD still needs to be pinned down, but we know it is multifactorial, involving a complex interplay of genetic predisposition, epigenetic elements and environmental factors. While immune dysregulation and inflammation are increasingly recognized as potentially contributing to ASD in some individuals, the specific role of parasitic invasion and cholesterol in the brain and its direct impact on the fundamental neurological processes underlying ASD symptoms remains a subject of ongoing research.

Transplanted microbiota: Transplanted microbiota is a know ASD therapy most commonly performed in the form of Fecal Microbiota Transplantation (FMT),[, , , , ,] albeit still

experimental, has emerged as a promising, therapeutic strategy for individuals with Autism Spectrum Disorder (ASD), particularly those with co-occurring gastrointestinal (GI) issues. The rationale behind this approach stems from the strong evidence of gut dysbiosis in ASD, where an imbalance of gut bacteria is believed to contribute to both GI symptoms and behavioral challenges via the gut-brain axis. Significant research, including promising clinical trials and systematic reviews, suggests FMT's potential. A systematic review published in *Effect of fecal microbiota transplantation in children with autism spectrum disorder: A systematic review* (Zhang et al., 2023) concluded that observational studies showed FMT significantly improved symptoms of autism in children compared to baseline. Another notable study from Arizona State University (Kang et al., 2017, with a 2-year follow-up published in *Scientific Reports*, 2019) on Microbiota Transfer Therapy (MTT, a specific FMT protocol) found remarkable improvements. They reported that "children with ASD often exhibit reduced immune system regulation and shifts in their gut microbiota, which appeared to facilitate increased inflammation," and after the MTT treatment, participants showed significant and long-lasting improvements in both GI symptoms and core ASD behaviors, with some even falling below the ASD diagnostic cut-off at two years post-treatment. This particular research has led to a patent approval for MTT for ASD symptoms, marking a step towards potential clinical availability, though further FDA-required trials are still necessary.

Transplanted microbiota, primarily via Fecal Microbiota Transplantation (FMT), is an emerging and promising, though still experimental, therapy for ASD, particularly for those with co-occurring gut issues. Driven by evidence of gut dysbiosis in ASD, studies like Kang et al. (2017) and reviews by Zhang et al. (2023) show significant and lasting improvements in both GI symptoms and core ASD behaviors, offering hope for an intervention that addresses the gut-brain connection. "This open-label (FMT)clinical trial found that 8-week MTT treatment significantly improved GI symptoms and ASD-related symptoms, which persisted for at least 8 weeks post-treatment." — Kang, D. W.,et al. With these promising results FMT needs more widespread acceptance and support.

ASD - WHAT CAN WE DO - HOW ABOUT DIETARY IMPROVEMENTS

With years of research and practical experience we now have knowledge on several high-level alternative therapies and clinical approaches to helping those with ASD get the improvement they need. It is crucial to emphasize that many effective therapies now available are often considered alternative or adjunctive approaches, all should be implemented under the guidance of a qualified healthcare professional and a registered dietitian experienced in ASD.

CONCLUSION

Drawing upon the comprehensive analysis presented, this paper posits that the escalating prevalence and severity of Autism Spectrum Disorder (ASD) are profoundly linked to an accumulating "toxic overload" stemming from a complex interplay of factors, rather than a singular cause. A central dynamic involves altered cholesterol metabolism, with findings from studies, including Harvard research, revealing significant lipid abnormalities in individuals with ASD and their parents, correlating with lower adaptive functioning and co-occurring conditions, and suggesting a link to the disproportionate male prevalence via testosterone precursors. This imbalance is closely tied to dietary factors, particularly the consumption of high-fat animal products and excessive omega-6 rich seed oils, which are hypothesized to disrupt cholesterol metabolism and exacerbate gastrointestinal issues.
The paper strongly implicates gastrointestinal (GI) system dysfunction and gut microbiome imbalances as crucial components, evidenced by symptoms like fatty stools, poor fat assimilation, and "leaky gut," allowing problematic substances to cross the blood-brain barrier. These GI issues are often intertwined with parasitic infections (including *Toxoplasma gondii* and *Giardia*, along with *E. coli*, *Blastocystis hominis*, *Endolimax nana*, and *Entamoeba histolytica*), which not only require host cholesterol for survival but are prevalent in common food sources (meats like camels, sheep, goats, poultry, seafood, game birds) and transmitted by domesticated animals and even household pests (flies, cockroaches, rats).

Additionally, heavy metal toxicity and Zinc and Vitamin D deficiency are identified as significant environmental factors that can further contribute to this "toxic storm." The unique and alarmingly high prevalence of ASD in the Somali-American community, whose lifestyle correlates with a culturally high-meat, high-cholesterol diet and increased parasite exposure, serves as a compelling case study illustrating this multifaceted hypothesis. Ultimately, this analysis advocates for a paradigm shift in understanding ASD, proposing that intervention strategies focusing on reducing this "toxic load" are essential for effective management. This includes the suggested removal of animal fats to support normal cholesterol metabolism, the elimination of parasites to modulate the gut microbiome, heavy metal detoxification, and the adoption of a personalized functional diet to promote overall health and alleviate ASD symptoms. Conclusively, this paper challenges the conventional understandings of Autism Spectrum Disorder's causes and its escalating prevalence, positing it as a complex neurodevelopmental condition stemming from a profound interplay of neurological, metabolic, and environmental factors. Such evidence-based approaches hold the potential to significantly lessen the burden and severity of ASD symptoms, ultimately enhancing the quality of life for affected individuals and their families. It is time to make stronger strides towards helping individuals with ASD.

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