The Bidirectional Relationship Between the Gut Microbiome and Cytokines and their effect on the Etiology of ASD

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Autism Spectrum Disorder (ASD) is a complex neurodevelopmental condition that affects how individuals communicate, behave, and interact socially. It impacts about one in thirty-six children in the United States. While genetic factors are well-known contributors, growing research suggests that abnormalities in immune system signaling—especially involving inflammatory proteins called cytokines—and imbalances in the gut microbiome may also play key roles in ASD development. This paper reviews the connection between these two systems and how their interaction may influence ASD. To explore this relationship, a narrative review of existing research was conducted using databases like Google Scholar and PubMed. Studies were selected based on relevance to cytokines, gut microbiota, and their role in brain development and ASD. Both human and animal studies were included to provide a broad understanding of the topic. The findings suggest that high levels of pro-inflammatory cytokines such as Interleukin-6 (IL-6) and Interleukin-1 (IL-1) during pregnancy can lead to placental inflammation and disruption of the blood-brain barrier, possibly affecting fetal brain development. At the same time, gut microbial imbalances—known as dysbiosis—may alter the production of neurotransmitters like serotonin and serotonin and gamma-aminobutyric acid (GABA), which are important for mood and behavior. These systems also appear to influence each other in a two-way cycle that may further impact brain function. Understanding this bidirectional relationship could offer new ideas for preventing or managing ASD through immune or gut-targeted strategies.

1 Introduction

Autism Spectrum Disorder (ASD) is a complex, lifelong neurodevelopmental condition that affects social interaction, communication, and behavior. Over the past few decades, the prevalence of ASD diagnoses has risen significantly, with current estimates suggesting that about one in thirty-six children in the United States are affected. While genetic factors play a central role in ASD, they do not fully explain its increasing prevalence or the wide range of behavioral and neurological symptoms seen in individuals. As a result, researchers have begun exploring additional biological systems that may contribute to ASD development—particularly the immune system and the gut microbiome.

One prominent hypothesis is maternal immune activation (MIA), which occurs when a pregnant mother's immune system is triggered by infection, stress, or environmental toxins. This immune response includes the release of pro-inflammatory cytokines, such as interleukin(IL)-6 , which help regulate immune responses but may also interfere with fetal brain development when present at elevated levels. Studies have reported unusually high levels of IL-6 in individuals with ASD and have linked such cytokine exposure during pregnancy to abnormal placental and neurological development [1–3].

At the same time, scientists have observed consistent differences in the gut microbiome—the diverse community of microorganisms living in the digestive tract—between people with ASD and those without. These microbes play essential roles not only in digestion but also in the production of brain-signaling molecules such as GABA, which influence mood, behavior, and cognitive function [4–6]. Many individuals with ASD experience gastrointestinal issues, suggesting that changes in gut health may be more than coincidental.

Importantly, recent research suggests that these two systems—the immune response and the gut microbiome—are deeply connected. Cytokines can alter the composition of the gut microbiota, while imbalances in gut microbes (known as dysbiosis) can trigger or amplify immune responses. This bidirectional relationship may create a cycle of inflammation and microbial imbalance that disrupts early brain development and contributes to the onset or severity of ASD symptoms.

The primary goal of this paper is to explore how cytokines and the gut microbiome influence the etiology of ASD, both independently and through their interactions. This narrative review synthesizes existing research across immunology, neuroscience, and microbiology to build a clearer picture of these complex biological relationships.

The scope of this review focuses on human and animal studies that describe mechanisms involving cytokines (particularly IL-6 and IL-1*beta*) and microbial signaling, especially during prena-

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tal development and early childhood. While this review does not include a formal meta-analysis or PRISMA-based systematic review, it incorporates high-quality, peer-reviewed studies from databases such as Google Scholar and PubMed.

By integrating findings from multiple disciplines, this paper aims to clarify a lesser-understood pathway in ASD development. Understanding how immune signaling and microbiota interact may help guide future research and inform early interventions—potentially improving long-term outcomes for individuals with ASD.

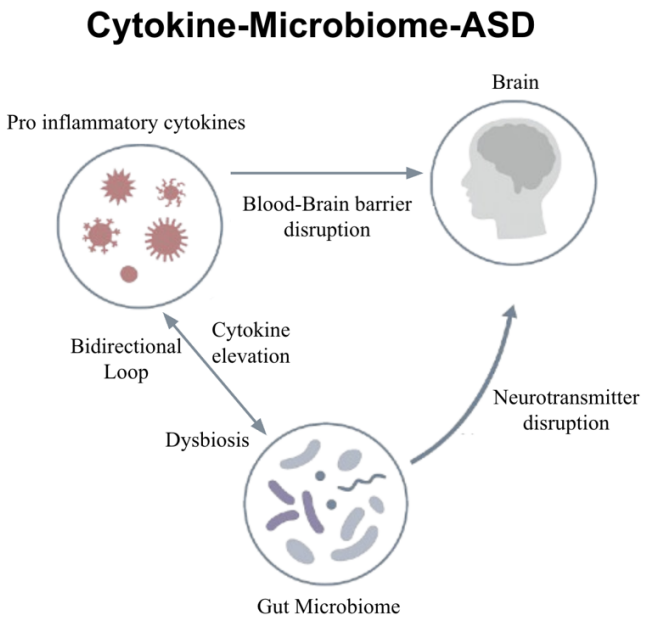

Fig. 1 Represents the Conceptual model of the bidirectional relationship between pro-inflammatory cytokines, gut microbiome dysbiosis, and neurodevelopmental disruptions associated with Autism Spectrum Disorder (ASD).

2 Methodology

To explore how the gut microbiome and cytokines might contribute to Autism Spectrum Disorder (ASD), this review employed a structured literature search and narrative synthesis of peer-reviewed research studies.

A comprehensive literature search was conducted using two primary databases: Google Scholar and PubMed. The search was performed between June 2024 and Aug 2024, using combinations of the following keywords: ”autism and cytokines”, ”gut microbiome and ASD”, ”IL-6 and brain development”, ”maternal immune activation”, ”placental inflammation and ASD”, and ”gut-brain axis and neurodevelopment.” Boolean operators such as AND and OR were used to refine the results and identify studies relevant to both cytokines and microbiota in the context of ASD.

Studies were included in this review if they met several specific criteria. First, they had to be published in peer-reviewed journals between the years 2000 and 2024. Only studies that focused on the role of cytokines or the gut microbiota in the development of Autism Spectrum Disorder (ASD) were considered. The review included both human and animal studies, as long as the animal models provided insights that could be relevant to human biology. All selected studies were written in English, had full-text availability, and presented clear findings, discussions, or hypotheses related to the biological mechanisms involved in ASD.

Exclusion criteria included are preprints without peer review, articles unrelated to ASD or microbiome-immunology interactions, and studies focusing exclusively on adult ASD without developmental context.

For each study that was included in the review, several important pieces of information were recorded. This included the author names and the year the study was published, as well as the type of study—such as whether it was experimental, a case study, a review article, or a meta-analysis. Specific biological mechanisms or cytokines being discussed, like IL-6 or IL-1*beta*, and any microbial species or microbiota characteristics that were studied were also noted. Most importantly, key findings related to ASD, immune signaling, and gut-brain interactions were captured. After gathering this information, it was organized into categorized notes and grouped the content by major topics, such as immune dysregulation, placental inflammation, gut dysbiosis, and bidirectional feedback mechanisms.

This review used a narrative synthesis approach to organize findings from various fields, including immunology, neuroscience, and microbiology. The results were grouped into four main themes (a) Maternal cytokines and placental inflammation (b) Cytokines and brain immune dysregulation (c) Gut microbiome dysbiosis in ASD and (d) Bidirectional mechanisms between cytokines and microbiota

This structure allowed for clear thematic discussion of how each system—immune and microbial—contributes to ASD, and how their interaction may influence outcomes.

Although no formal quality scoring tool (such as GRADE or CASP) was used, all studies were evaluated based on source credibility (peer-reviewed journals), clarity of methodology, sample size, and alignment with current scientific consensus. Preference was given to studies with experimental data or systematic reviews from established journals. Articles from government or institutional sources (e.g., NIH, PMC) were prioritized over blog posts or opinion papers to minimize selection bias.

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3 Results

This section presents the key findings from research studies examining how cytokines and the gut microbiome—individually and together—may influence the development of Autism Spectrum Disorder (ASD). These findings are organized into four major themes.

3.1 Maternal Cytokines and Placental Inflammation

Cytokines are signaling proteins that regulate immune responses and help maintain a healthy pregnancy[1]. However, when cytokine levels—especially IL-6 and IL-1*beta*—become too high, they can lead to inflammation in the placenta and disrupt fetal brain development. IL-6 supports placental growth, while IL-1*beta* is more active in early pregnancy. Studies have found that inflammation in the placenta is associated with an increased risk of ASD[7]. The placenta also produces serotonin, a key neurotransmitter for brain development, which may be affected by inflammation[8].

Factors that elevate cytokines during pregnancy include obesity, asthma, smoking, infections, and gestational diabetes[2,3]. For example, obesity causes chronic inflammation and raises IL-6 levels[9]. Asthma has been linked to a 40% higher chance of ASD in offspring[10], and smoking increases white blood cell production, which also boosts IL-6[11]. Infections during pregnancy can dramatically increase maternal IL-6 levels[4]. Since both IL-6 and IL-1*beta* are involved in placental development, abnormally high levels may interfere with fetal neurodevelopment and raise ASD risk[7,12].

3.2 Cytokines and Brain Immune Dysregulation

Once in the brain, cytokines play key roles in neuron and glial cell development. Excess cytokines can weaken the blood-brain barrier (BBB), a protective membrane that keeps harmful substances out of the brain[13]. IL-4, IL-6, IL-8, and IL-17 are especially known to increase BBB permeability[5]. When the barrier is compromised, immune cells like T-cells and B-cells can enter the cerebrospinal fluid and release more inflammatory cytokines[14]. Immune imbalance is common in ASD—only about 30% of individuals have normal immune profiles[14].

Cytokines also influence gene expression. For example, the major histocompatibility complex (MHC), particularly MHC class I (MHC-I), helps shape axons and dendrites and is regulated by cytokines. Some MHC-I genes, such as HLA-A2, Death Receptor 4, and Death Receptor 11, are associated with ASD and can alter T-cell behavior. CD4+ T-cells, which are often elevated in people with ASD, may also play a role in immune dysregulation[14].

3.3 Gut Microbiome Dysbiosis in ASD

Many individuals with ASD report gastrointestinal (GI) problems such as diarrhea and constipation. These symptoms are about seven times more common in individuals with ASD than in neurotypical individuals. One possible reason is dysbiosis—an imbalance of gut bacteria—which can result from poor diet, antibiotics, asthma, infections, or stress[4,5]. Dysbiosis typically lowers microbial diversity and allows harmful bacteria to dominate.

The maternal gut microbiome may affect fetal neurodevelopment by transferring microbes to the placenta[15]. The gut-brain axis, which includes the enteric nervous system and vagus nerve, allows microbes to influence brain activity. The gut microbiome produces neurotransmitters like serotonin and short-chain fatty acids (SCFAs), which affect mood and behavior[4].

Loss of helpful bacteria, such as through antibiotics or poor nutrition, can disrupt neurotransmitter production and increase ASD risk[15]. One mouse study showed that males exposed to Bacteroides fragilis developed ASD-like behaviors, while females did not—possibly reflecting the higher rate of ASD in males[16,17]. Research also shows higher levels of Firmicutes and lower levels of helpful bacteria like Bifidobacterium, Prevotella, Coprococcus, and Veillonellaceae in ASD[18,19].

Harmful bacteria like Desulfovibrio are more common in children with ASD, especially those with frequent antibiotic use for ear infections[20]. This genus produces hydrogen sulfide and acetic acid, both of which may disrupt immune function or act as neurotoxins[5,21]**?** .

Clostridium species, which are also more common in ASD, produce harmful chemicals like phenols and p-cresol that may damage the nervous system[6,22]. Although some behavioral improvements have been observed after treatment with vancomycin, they are often temporary due to the bacteria's spore-forming resistance[23].

3.4 Bidirectional Mechanisms Between Cytokines and Microbiota

Cytokines and gut microbes influence each other in a feedback loop. When dysbiosis occurs, the intestinal lining may become "leaky," allowing bacteria and toxins to enter the bloodstream—a phenomenon linked to elevated IL-6 and IL-1*beta* levels[24–26]. These cytokines can further damage the intestinal barrier and worsen inflammation.

SCFAs produced by beneficial bacteria also influence immune responses and brain development[27]. Hsiao et al. found that mouse models of ASD had weaker intestinal barriers, higher IL-6 levels, and altered microbiomes[28] . Additionally, inflammation from asthma, obesity, or smoking may shift microbial communities, reinforcing dysbiosis[2,3,10]. Some microbes flourish in inflammatory environments, while others decline, leading to more gut imbalance and behavioral changes[29,30].

One rat study showed that early-life stress (such as maternal separation) elevated IL-6 and disrupted the gut microbiome[4]. Since the fetal microbiome starts forming in the placenta—where cytokines like IL-6 are active—these early interactions may play a crucial role in the origins of ASD[1,31].

4 Discussion

This literature review suggests that high levels of pro-inflammatory cytokines—particularly interleukin-6 (IL-6)—during pregnancy may increase the risk of Autism Spectrum Disorder (ASD) by disrupting brain development. Elevated IL-6 levels have been linked to a weakened blood-brain barrier (BBB), which normally protects the brain from harmful substances11. When this barrier is compromised, it may allow immune cells and cytokines to enter the brain and interfere with neuron and glial cell development. In the future, treatments that aim to strengthen the BBB or reduce inflammation might help lower ASD risk. Some genes related to immune function, such as Death Receptor 4 and HLA-A2, have also been linked to ASD, and tools like CRISPR gene editing may eventually be used to study or modify these genetic pathways[14]. However, more research is needed to confirm the safety and effectiveness of such methods.

The findings also suggest that the gut microbiome plays a significant role in ASD. Several studies have shown that people with ASD often have higher levels of Bacteroides and Firmicutes, and lower levels of beneficial microbes such as Bifidobacterium, Coprococcus, and Veillonellaceae[15,16,18,19]. These microbial imbalances—or dysbiosis—may contribute to gastrointestinal symptoms and behavioral issues. Approaches such as using probiotics, fermented foods, or diets rich in fiber might help rebalance the microbiome and reduce symptoms[32]. Anti-inflammatory treatments have already shown promise in reducing irritability, GI distress, and social challenges[33], and improvements in digestion have even been associated with better communication skills and eye contact[34].

Several bacteria appear especially promising for future treatment options. Bifidobacterium can increase GABA, a neurotransmitter that helps calm brain activity[6]. Coprococcus helps produce butyrate, a short-chain fatty acid that supports immune balance[22]. Lactobacillus may increase oxytocin, a hormone related to bonding and social behavior[35]. These microbes have also been linked to reductions in anxiety and depression symptoms[36].

However, not all microbes have positive effects. Desulfovibrio, for instance, can produce hydrogen sulfide, which may harm the immune system or act as a neurotoxin[21,37]. Similarly, Clostridium produces p-cresol and phenols, which can disrupt the nervous system[6,22]. Although antibiotics like vancomycin have led to short-term improvements in some children, long-term results have been limited by bacterial resistance[23].

Importantly, this review highlights the two-way relationship between cytokines and gut bacteria. Inflammatory cytokines can alter the gut microbiome, and microbiome imbalances can increase intestinal permeability, leading to more cytokine production. This cycle may begin as early as pregnancy, when maternal immune activation and microbial transmission first influence fetal development[1,31].

The first two years of life are a critical period for both brain and gut development. Early intervention strategies that support a healthy gut and immune system during this time—such as improving maternal health, reducing stress, using prebiotics or probiotics, and limiting unnecessary antibiotic use—may reduce ASD risk or severity.

Together, these insights suggest that ASD is not caused by a single factor, but rather by the interaction between immune signals and gut microbes. A better understanding of this complex relationship may help develop more personalized and effective strategies for ASD management and prevention.

5 Conclusion

This review highlights that both cytokines and the gut microbiome play important and interconnected roles in brain development and the possible emergence of Autism Spectrum Disorder (ASD). Elevated levels of pro-inflammatory cytokines such as interleukin-6 (IL-6) and interleukin-1*beta* (IL-1*beta*) have been associated with placental inflammation, immune dysregulation, and compromised blood-brain barrier function—all of which can disrupt early neurodevelopment. At the same time, microbial dysbiosis, or an imbalance in the gut microbiota, may influence behavior and brain function by altering the gut-brain axis and the production of important neurotransmitters and immune signals.

One of the most important insights from this review is the bidirectional relationship between these two systems. Inflammatory cytokines can alter the composition of the gut microbiome, while dysbiosis can further promote inflammation by increasing intestinal permeability. This feedback loop may be a key mechanism in the development or worsening of ASD symptoms.

This study aimed to explore how cytokine signaling and microbiome health may interact to influence ASD, and the findings support this connection. While direct causation is still uncertain, the evidence from both human and animal studies suggests that early disruptions in immune balance and microbial composition can have lasting effects on brain development.

The implications of this work are both theoretical and practical. Understanding how immune and microbial factors interact opens the door to new approaches for ASD prevention and management. Interventions that target inflammation, restore microbial balance, or strengthen the gut-brain axis—especially during pregnancy or early childhood—may offer promising directions for future research.

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This review also has limitations. As a narrative review, it does not include a formal quality assessment of all included studies, and some evidence is still preliminary or based on animal models. Further longitudinal and clinical studies are needed to confirm these relationships and to identify safe, personalized treatment strategies.

Ultimately, this research underscores the importance of seeing ASD not just as a genetic or neurological condition, but as a complex disorder shaped by multiple biological systems. With continued research and collaboration across fields like immunology, neuroscience, and microbiology, we may be able to uncover more effective ways to support individuals with ASD and their families.

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