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Abstract.

A gut-brain axis (GBA) has a long history of conceptual development. Intestinal dysbiosis has now been recognized as a key player in the development of adult neurodevelopmental disorders, obesity, and inflammatory bowel disease. Recent developments in metagenomics suggest those nutrition and gut microbiotas (GM) are important regulators of the gut-brain communication pathways that cause neurodevelopmental and psychiatric problems in adulthood. Intestinal dysbiosis and neurodevelopmental disease outcomes in preterm newborns are being linked by recent research. Recent clinical investigations demonstrate that in critical care units, intestinal dysbiosis occurs before late-onset newborn sepsis and necrotizing enterocolitis. Strong epidemiologic data also shows a connection between necrotizing enterocolitis and extremely low birth weight babies long-term psychomotor impairments and late-onset neonatal sepsis. The GBA theory suggests that intestinal bacteria may indirectly affect preterm newborns developing brains. In this review, we emphasize the structure and function of the GBA and discuss how immune-microbial dysfunction in the gut affects the transmission of stress signals to the brain. Preterm babies who are exposed to these signals develop neurologic disorders. Understanding neuronal and humoral communication through the GBA may provide insight into therapeutic and nutritional strategies that may enhance the results of very low-birth-weight babies.

Key words. Neurological Disorders (ND), Immune Function (IF), Gut Microbiotas (GM), Gut-Brain Axis (GBA), Mental Health.

Introduction.

GBA refers to the two-way communication system between the gastrointestinal (GI) tract, and central nervous system (NS), involving neural, immunological pathways, and hormonal. This communication plays a crucial role in various aspects of human health, including neurological disorders (ND), mental health, and immune function (IF) [1]. In ND, Emerging research suggests that disruptions in the GBA may give to the enlargement and progression of ND. For example, studies have found associations between gut dysbiosis (imbalance in the gut microbial community) and multiple sclerosis [2]. The GM can produce and regulate various neurotransmitters, including dopamine, serotonin, and gamma-aminobutyric acid (GABA), which are involved in ND functioning [3]. In Mental Health, The GBA has profound implications for mental health conditions such as depression, anxiety, and stress-related disorders [4]. The GM influences the production of neurotransmitters and neuroactive compounds that impact mood and behavior [5]. In IF, The IF growth and operation are significantly influenced by the GM. A significant fraction of IF is nearby the gut-associated lymphoid tissue (GALT), which is continually exposed to antigens produced from the GM. Interactions between the GM and the IF help establish IF tolerance and prevent the development of autoimmune diseases. Disruptions in the GM composition, such as dysbiosis or increased intestinal permeability (leaky gut), can lead to IF dysregulation and inflammation, which have been implicated in various diseases, including autoimmune disorders and allergic conditions [6-8]. The GBA refers to the two-way statement system connecting the gastrointestinal tract (the gut) and intelligence. This axis involves a complex network of connections between the gut, the autonomic NS, the enteric NS, and the central NS [9]. The gut and the brain are constantly exchanging signals and information through various pathways, including neural, hormonal, and IF pathways. This communication stage is essential to position in maintaining the overall physical condition and has implications for ND, mental health, and IF [10]. The GM communicates through the mind in the course of the vagus nerve, a major pathway for bidirectional signaling. This connection enables the GM to influence brain function and emotional states. Studies have shown that manipulating GM through interventions like probiotics and fecal microbiota transplantation can have beneficial effects on mental health symptoms [11]. Moreover, GM can modulate the resistant reaction by influencing the production and function of resistant cells and resistant molecules. This IF modulation can have far-reaching effects on overall health and may influence the expansion and sequence of immune-related disorders [12]. The GBA is a complex and dynamic system that plays a crucial role in ND, mental health conditions, and IF [13]. Accepting the connections between the gut & the brain can provide insights into the underlying mechanisms of these conditions and potentially lead to novel therapeutic approaches targeting the GM and the GBA [14]. In the study [15], the complicated interactions of the microbiota GBS are examined to better understand the pathogenesis caused by the microbiota, the potential for noninvasive prognosis, and treatment options that take advantage of modulations of the microbiota GBA. Furthermore, they analyze the shortcomings of present strategies and give insights into the continuing shift from the bench to the bedside. Higher fiber diets have been shown to increase the variety and abundance of beneficial taxa in GM as well as butyrate synthesis, which has been shown to
have neuroprotective effects and improve the plasticity of neurons. The article [16] provided insight into how gender variations in the GBA may be responsible for the disparities involving male and female prevalence of psychiatric, neurodevelopmental, and neurodegenerative illnesses. They concentrate on conditions including anxiety disorders, autism spectrum disorder, psychotic disorders, stress, and Parkinson's disease. Study [17] examined the GBA through the lens of stress and how stress may affect a person's physiology from the time of conception until maturity. There are several mechanisms through which microbiome changes generate shifts in behavior. Inflammation in the stomach has effects on the brain as well, and a comparable systemic reaction is seen. Study [18] described the pathophysiological processes resulting from disturbance of the MGB axis and potential treatment strategies, with a particular emphasis on the complex network's GM. The development of novel medicines for enhancing the therapeutic care of these patients may be aided by an understanding of the processes governing the GM and its mutual connections by the resistant and other systems. Additionally, the blow of psychiatric medications on top of the GM presently being utilized in patients as well as potential therapeutic techniques focusing on this environment will be taken into consideration. Natural polysaccharides are critical for preserving the regular state of gut flora, according to the research that has been conducted so far. The study's findings, which include the modulatory role of plant polysaccharides on gut flora and the two-way communication between the GM and the brain, provide fresh insights into how to prevent and cure neurodegenerative illnesses [19]. Article [20] discussed the changes in GM that take place during depressive states, the relationship between these changes and depressive-like behavior, the possible apparatus connecting disruption in GM to depression, and the earliest attempts to use GM intervention for the treatment of depression. Study [21] provided a general overview of GM, paying close attention to mast cells and ND. Mast cells serve as sensors and effectors of cytokines and neurotransmitters, which have important roles in neuroimmune communication. Probiotic use in this situation may be a good therapeutic strategy to use in conjunction with conventional treatments. To determine if the gut bacteria cause ND or whether such conditions affect the bacterial composition, further research must be done. In Study [22], they summarise the most recent evidence that the protected organization and GM take part in important roles in the conservation of brain performance and the emergence of ND. Additionally, they present the latest developments in the GBA idea for treating ND using probiotics and faecal microbiota transplants. In a study [23], they evaluated molecular networks through which the microbiota, gut, and GBA control brain growth and purpose, and the potential roles for these networks in ND associated with immunological dysfunction. Although several studies have looked into the microbiota-GBA, there are still difficulties in applying the results of these studies to people because of the intricate interactions between the GM and the brain. Study [24] focused on the two-way announcement among the stomach and brain, its effects & functions in the regulation of different ND, such as anxiety, depression, and schizophrenia, and makes an effort to investigate the fundamental apparatus on behalf of the equal. Animal models for studying the masterpiece of the intestinal microbiota, research on the impact of bacteria spreading from faces, and germ-free mice Study [25] evaluated the search of computational analysis approaches that can help to comprehend the relationship between the microbiome, the gut, and the brain, as well as the causes and development of neurological disorders, using a systems ecology perspective. A shotgun approach and high-throughput sequencing of healthy and neurological condition specimens recorded in biological databases generated large amounts of microbiological 16S ribosomal RNA sequence data that provided here together with the bioinformatics instruments employed to determine such relationships. The study [26] provided the disciplines of gastroenterology and neuroscience with the purpose of demonstrating that the gut-brain relation can lead to the establishment of a number of neurological diseases. They discussed the most recent information on how microbial imbalance contributes to neurodegeneration and neuroinflammatory disorders like Alzheimer's, Parkinson's, and multiple sclerosis.

The gut-brain axis.

The GBA is defined as the communication pathway between the motor and sensory components of the gastrointestinal tract and the central NS. Figure 1 demonstrates the intricacy, moderators, and multiorgan linkages of GBA-related signaling pathways that affect the balance between health and sickness in a preterm baby.

The brain, which connects the hypothalamic-pituitary-adrenal axis, limbic system, brain stem, and cerebral cortex, is the main part of the GBA. The limbic cortex controls smell and unifies sensory and motor processes. Other parts of the brain that control a variety of actions provide information to the limbic system. Studies on the effects of pain and maternal deprivation on newborn mice have shown that the limbic system is crucial to the hippocampus' development. Damage to the hippocampus in premature babies without cerebral hemorrhage is presumably the source of neurobehavioral problems documented in these children throughout childhood. When studying limbic system impairments in human preterm babies, researchers must account for gender differences in the impact of stress. Through the sympathetic, autonomic, and enteric nervous systems (ENS), the peripheral parts of the GBA connect with the brain. The ENS, via which gut bacteria may influence brain growth and function, has been shown to impact neuronal signals. The ENS is an intricate network of nerves and cells that lines the interior of the gut wall. Diseases of the newborn that affect the ENS are discussed in the context of GBA. The GBA includes peripheral components for the autonomic NS, the sympathetic NS, and the hypothalamic-pituitary axis. A significant retrograde communication pathway from the stomach to the brain is the afferent vagus nerve. The intestinal epithelial barrier function may be lost as a consequence of this inflammation, allowing for bacterial invasion. Increased intestinal permeability leads to gastric inflammation by simultaneously activating the immune. Changes in brain function and illness are brought on by signals that the GBA transmits to the intestinal and systemic IF. The ENS, the autonomic NS, and the hypothalamic-pituitary
axis all provide messages to the limbic system and the brain about nutrition and microorganisms in the intestine. The brain stems and higher cortical areas like the limbic system, which is involved in rewards, are connected reciprocally through the hypothalamus. Hormone and neuropeptide production in the stomach during a stressful situation eventually triggers the gland through signals sent to the brain. Apparatus of the GBA linked include peptide YY, pancreatic polypeptide, glucagon-like peptide-1, and oxyntomodulin, whereas fasting increases ghrelin production. With the help of neuropeptide signals, the GBA also affects intestinal IF. Dendritic cells' and T cells' respective roles in the brain, in secondary lymphoid organs like Peyer's patch, and the intestinal wall are all influenced by neuropeptides like vasoactive intestinal peptide and norepinephrine. Depending on the ratio of "neuropeptides and other immunomodulatory" substances, immune cells may either induce an inflammatory response to microbial and nutritional, hence guiding the formation of distinct effectors lymphocyte populations. NS has been linked to changes in the composition of the gut microbial community, which in turn has been linked to an impairment of barricade purpose and an increase in intestinal permeability.

**Diseases associated with GBA disorders in the embryo and newborn.**

Coordination of neuronal, intestinal motor and absorptive, and immunologic systems throughout pre- and postnatal life ensures a strong newborn baby and fosters continuing growth and development throughout immaturity and babyhood. Multiple interaction cells and organs contribute to a normal vs. dysfunctional GBA, which results in either health or illness (Figure 1). Pediatricians are aware of morphologic ENS deficiencies. Hirschsprung's disease, which increases the risk of enterocolitis, is brought on by ENS congenital abnormalities. Megalocystis, megacolon, and malrotation are all symptoms of the extremely deadly developmental disorder of the ENS known as intestinal neuronal dysplasia. "Myelomeningocele and acquired adult disorders" like acute spinal cord injury are linked to gut motor dysfunction. A nonanatomic, neurogenic intestinal obstruction condition is brought on by congenital or acquired disorders of the spinal cord that change ENS function. There are other things than congenital abnormalities that may happen to a fetus that influence how the ENS develops. When used during human pregnancy, selective serotonin reuptake
inhibitors (SSRIs) have sparked worries about the ENS developing abnormally. Investigations of infants who were exposed to SSRIs while they were still in the womb have shown eating problems, impaired motor function, and behavioral abnormalities; these results seem connected to anomalies in the autonomic NS. The signs of the baby being exposed to selective serotonin reuptake inhibitors during pregnancy include:

(i) Delayed infantile speech development milestones
(ii) Behavior issues that began after childhood.

We must find out more about the "defects" that pregnancy-related medications like valproic acid and SSRIs may introduce into the human fetal ENS. The pathophysiology of autism spectrum disorders (ASD) may be influenced by selective serotonin reuptake inhibitors used during pregnancy, albeit the mechanisms by which selective serotonin reuptake inhibitors alter NS development are currently poorly understood. Olanzapine, an antipsychotic medicine that balances the brain, was administered to rats. These outcomes included changed fecal microbiota composition, recruitment of macrophages into adipose tissue, and higher plasma stage of proinflammatory cytokines. This study discusses the link between prenatal selective serotonin re-uptake inhibitors exposure and autism spectrum disorders.

Interactions of GBA with gut microbiota, epidermis, and immune cells.

The physiological role of the GBA is examined concerning interactions between intestinal epithelia, GM, and the IF. Given their closeness, GM can activate and control intestinal IF and gut epithelia, and the ENS. Although there is a lack of primary research on this issue, a review explains the crucial involvement of intestinal microbiota in the postnatal increase of gastrointestinal processes in premature newborns. Recent research indicates an association between postnatal events, such as the use of antibiotics, and childhood obesity in infants and children. Obesity and GM are related because the latter tends to accumulate more energy. Research on identical twins has shown phylum-level differences in the GM between lean and obese individuals. These differences include lower bacterial diversity, altered gene representation in the microbiome, and abnormal metabolic pathways. Diet-related microbial indicators in the feces of obese people are associated with altered microbiota and poor metabolic health. Microbes in the gut might set the stage for efficient dietary energy salvage. One such bacterium is Faecalibacterium prausnitzii, which is often found in the intestines of overweight children in southern India and is known to enhance epithelial health. Beyond affecting obesity's pathophysiology, gut microorganisms also have an impact on the prevalence of diseases including type 2 diabetes, demyelinating disorders, mental illness, behavioral abnormalities, and irritable bowel syndrome. How dendritic cells, intestinal epithelia, and other underlying IF recognize commensal and pathogenic bacteria and how these cells communicate with one another. Dendritic cells process and display antigens as M cells cover Peyer's patches during the development of tolerance. Secretory IgA prevents intestinal epithelial invasion by binding to invading microorganisms in intestinal fluid and the mucin layer. In addition, the mucin layer contains antimicrobial peptides generated by enterocytes that prevent microbial penetration of the intestinal barrier. Intestinal lip polysaccharide binds Toll-like receptor-4 on enterocytes, which is produced by the cell walls of Enterobacteriaceae. Intestinal IF cells are activated to create cytokines in response to pathogen-associated molecular pattern recognition, which in turn triggers ND transmissions through the vagus nerve. Toxoplasma gondii infection causes ileitis in mice via activating Toll-like receptor-9 in the intestinal cells. The animals lacked cerebral, resulting in increased amounts of proinflammatory cytokines in their brain tissue. The findings of this research provide compelling evidence that pathogen-specific infection and inflammation in the gastrointestinal tract might trigger immune-mediated inflammation in the central NS. According to recent research, the inflammasome helps the gut's innate IF cells identify signals connected to bacteria and other types of harm. By acting on the hypothalamic-pituitary axis, the GBA may control the activation of gut innate IF cells through the inflammasome, therefore reducing inflammation. In numerous animal species, it has been shown that GM may transmit inflammation to the gut-brain axis. While interactions between Escherichia coli and the gut-brain axis convey an inflammatory reaction, F. prausnitzii, generates an anti-inflammatory reaction. Mucin synthesis by intestinal epithelia, a crucial human defense against microbial invasion, may be boosted by commensal flora to induce protective responses. In addition, B. thetaiotaomicron up-regulates the production of angiogenin4 in crypt Paneth cells, which serves as both an “antimicrobial peptide and an angiogenic” factor to promote the development and condition of the intestinal villi. Consequently, beneficial bacterium maintain health and balance in the growing digestive tract. Intestinal dysbiosis and intestinal inflammation define necrotizing enterocolitis (NEC), a disease that mostly affects premature infants. There is an increased risk for significant long-term neurodevelopmental problems when NEC worsens the hospital stay. Negative effects on brain function are mediated through the GBA due to the stress caused by NEC. Long-term psychomotor and intestinal impairments are associated with infections and intestinal damage lead to the release of microbial toxins and pro-inflammatory cytokines into following NEC. In NEC, oxidant stress and other mediators of brain damage are proportional to the severity of the intestinal injury. More study in newborns is needed to determine the involvement of the gut-brain axis in the etiology of cerebral palsy and autism spectrum disorders. However, intestinal inflammation during pregnancy may be triggered by both viral and noninfectious disorders. Infection and inflammation of the fetal intestines presuppose that germs are ingested by the baby before birth. Swallowing does not begin until about 29–31 weeks of pregnancy. Since premature labor is linked to the presence of the bacteria Ureaplasma parvum and Ureaplasma urealyticum, the general premise is accurate. Ureaplasma colonization of the respiratory tract in extremely premature newborns increases the risk of necrotizing enterocolitis. Changes in the intestinal microbiota and the resulting "dysbiosis" likely play a central role in the pathogenesis of NEC, although they occur after birth. Between 7 days and 72hr before the commencement of NEC cases, a
bloom or significant rise in the phylum Proteobacteria has been found in fecal samples. However, the research did not find a correlation between any specific genus or species and NEC in newborns. In extremely premature neonates, intestinal dysbiosis is a risk factor for the development of late-onset neonatal sepsis. Metagenomics has revealed these previously unknown aspects of the GM and the etiology of late-onset neonatal sepsis or NEC in extremely preterm newborns. The field of metagenomics examines the genes and metabolites produced by bacteria in a specific setting, such as the digestive tract. To determine the molecular fingerprints of bacteria in a given environment, analytical tools are used in metagenomic approaches. Software programs are used to analyze these signals, and their results define the microbial ecology of the samples’ environments. Neonatal intensive care unit nurses and doctors may not be acquainted with the analytical tools, molecular procedures, bioinformatics, and data formats related to metagenomic research. Microbes in human newborn organs may be identified using culture-independent approaches, as described in a recent review of metagenomics. Interstitial cells of Cajal operate as the intestinal pacemaker, stimulating the muscularis mucosae at regular intervals. Pathogenic bacteria in the intestinal lumen may be eliminated with the aid of gut motility and feces. When a very premature newborn develops ileus, doctors should be on the lookout for signs that toxins in the gut are blocking the action of the interstitial cells of Cajal. The accompanying justification outlines the physiology, however, most neonatologists already know that an ileus is a warning sign of NEC. For proper formation and operation of both the gastrointestinal and systemic IF systems, GM is crucial. Pregnancy continuation and healthy development of the fetus are linked to a Th2:Th1 lymphocyte bias. The Th2 bias seen in newborns is likely a carryover from immunosuppression found in pregnant women. Researchers have shown that newborn mice are more vulnerable to infection because of the immunologic environment. Intestinal microbial colonization after birth is linked to a reduction in the risk of infection and an end to the neonatal Th2:Th1 imbalance. In light of the GBA theory, this work reviews a variety of neurologic repercussions of common intestinal illnesses; therefore, the list of stressors is by no means complete. We chose prenatal and postnatal stress conditions in the gut based on their relevance to GBA pathogenesis. The study of the laws that govern psychological events and human behavior is the field of psychology. To make matters worse, it seems that the more we learn about human psychology, the more we discover how little we understand. Until very recently, there was not a single mental disease for which a reliable physiological, biochemical, or genetic biomarker had been developed. There is a noticeable gap in the field of psychology’s practical application, and mental diseases continue to constitute significant medical obstacles. Table 1, 2, 3 and 4 depicts the dramatic rise in the healthcare burden caused by the rising prevalence of individuals with mental and neurological problems over the last several decades. Mental problems account for about 20% of all medical costs, while treatment and recovery rates are far lower than for other diseases. All of these results point to the idea that humans are super organisms, which has been overlooked by previous studies. Neurological and mental disorders increased medical expenses (DALYs and Collaborators). Figure 2 displays the disability-adjusted life years (DALYs) brought on, respectively, by mental and neurological disorders.

### Table 1. Neurological and mental disorders increased medical expenses (DALYs and Collaborators).

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Start date</th>
<th>End date</th>
<th>Participants</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurological disorder</td>
<td>Jan 12, 2017</td>
<td>Mar 13, 2018</td>
<td>60000</td>
</tr>
<tr>
<td></td>
<td>Apr 10, 2018</td>
<td>June 21, 2019</td>
<td>70000</td>
</tr>
<tr>
<td></td>
<td>July 14, 2019</td>
<td>Aug 13, 2020</td>
<td>80000</td>
</tr>
<tr>
<td></td>
<td>Sep 25, 2020</td>
<td>Apr 18, 2021</td>
<td>81000</td>
</tr>
<tr>
<td></td>
<td>June 20, 2021</td>
<td>Mar 19, 2022</td>
<td>90000</td>
</tr>
<tr>
<td>Mental and Substance use disorders</td>
<td>Jan 16, 2017</td>
<td>Apr 14, 2018</td>
<td>90000</td>
</tr>
<tr>
<td></td>
<td>Apr 20, 2018</td>
<td>July 25, 2019</td>
<td>300000</td>
</tr>
<tr>
<td></td>
<td>July 28, 2019</td>
<td>Aug 10, 2020</td>
<td>500000</td>
</tr>
<tr>
<td></td>
<td>Sep 18, 2020</td>
<td>Apr 3, 2021</td>
<td>140000</td>
</tr>
<tr>
<td></td>
<td>June 15, 2021</td>
<td>Mar 22, 2022</td>
<td>140000</td>
</tr>
</tbody>
</table>

### Table 2. Disability-adjusted life years (DALYs) brought on, respectively, by mental and neurological disorders.

<table>
<thead>
<tr>
<th>Year</th>
<th>Mental and substance use disorders</th>
<th>Neurological disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td>2018</td>
<td>4.8</td>
<td>3.2</td>
</tr>
<tr>
<td>2019</td>
<td>5.5</td>
<td>3.5</td>
</tr>
<tr>
<td>2020</td>
<td>5.3</td>
<td>3.3</td>
</tr>
<tr>
<td>2021</td>
<td>5.4</td>
<td>3.6</td>
</tr>
<tr>
<td>2022</td>
<td>5.6</td>
<td>3.8</td>
</tr>
</tbody>
</table>

### Table 3. The number of DALYs in 2018, 2020, and 2022 brought on by different diseases.

<table>
<thead>
<tr>
<th>Disease</th>
<th>2018</th>
<th>2020</th>
<th>2022</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADHD</td>
<td>1000</td>
<td>1000</td>
<td>1000</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
<td>2000</td>
<td>2000</td>
<td>2000</td>
</tr>
<tr>
<td>Eating disorders</td>
<td>3000</td>
<td>3000</td>
<td>3000</td>
</tr>
<tr>
<td>Parkinson's disease</td>
<td>4000</td>
<td>4000</td>
<td>4000</td>
</tr>
<tr>
<td>Conduct disorder</td>
<td>6000</td>
<td>6000</td>
<td>6000</td>
</tr>
<tr>
<td>Tension-type headache</td>
<td>8000</td>
<td>8000</td>
<td>8000</td>
</tr>
<tr>
<td>Bipolar disorder</td>
<td>9000</td>
<td>9000</td>
<td>9000</td>
</tr>
<tr>
<td>Autistic spectrum disorders</td>
<td>10000</td>
<td>10000</td>
<td>10000</td>
</tr>
</tbody>
</table>

### Table 4. The DALYs brought on by different diseases in 2018, 2020, and 2022.

<table>
<thead>
<tr>
<th>Disease</th>
<th>2018</th>
<th>2020</th>
<th>2022</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenia</td>
<td>10000</td>
<td>10000</td>
<td>10000</td>
</tr>
<tr>
<td>Alcohol use disorders</td>
<td>12000</td>
<td>12000</td>
<td>12000</td>
</tr>
<tr>
<td>Drug use disorders</td>
<td>20000</td>
<td>20000</td>
<td>20000</td>
</tr>
<tr>
<td>Alzheimer's and other dementias</td>
<td>30000</td>
<td>30000</td>
<td>30000</td>
</tr>
<tr>
<td>Anxiety disorders</td>
<td>28000</td>
<td>28000</td>
<td>28000</td>
</tr>
<tr>
<td>Depressive disorders</td>
<td>41000</td>
<td>41000</td>
<td>41000</td>
</tr>
<tr>
<td>Migraine</td>
<td>42000</td>
<td>42000</td>
<td>42000</td>
</tr>
</tbody>
</table>

Though beneficial for the IF system, microbial colonization of the gut also works in tandem with food to promote healthy brain development. Many beneficial bio elements for health...
Figure 2. Neurological and mental disorders increased medical expenses (DALYs and Collaborators).

Figure 3. The DALYs caused by various illnesses in 2018, 2020, and 2022.

Figure 4. Increased permeability and loss of the protective intestinal barrier.
...and brain development are found in human milk. Human milk's immunological qualities are preserved better by freezing than during pasteurization. NEC may be avoided because of the presence of lactoferrin, lysozyme, and other antimicrobial proteins and immunomodulatory substances in fresh colostrums and milk. NEC may be avoided in a rat newborn model by administering disialyllacto-N-tetraose, an oligosaccharide found in human milk. Human milk oligosaccharides help the developing digestive tract of premature newborns via many methods. Premature newborns that suffer from NEC, an intestine disorder, often have severe inflammation and GBA-mediated indirect brain damage. For these reasons, we recommend giving colostrums immediately after delivery and breast milk that has been newly produced throughout the neonatal intensive care unit stay. Ingesting fresh mother's milk encourages the growth of beneficial commensal bacteria in the digestive tract, including probiotic bacteria that thrive on the oligosaccharides found in human milk. Because of this, human breast milk helps babies grow up with fully functional brains and NS. Instead of utilizing a baby formula, neonatal caregivers advocate for the use of donor human milk when a mother is unable to provide her own. The immunomodulatory and antibacterial capabilities of milk proteins are greatly diminished when human donor milk is extensively pasteurized before use. Donor human milk loses some of its antibacterial and immunomodulatory properties after being pasteurized and frozen and thawed. The composition of human milk oligosaccharides differed significantly between donor and maternal milk, suggesting a further investigation into the antimicrobial advantages offered by donor milk.

In light of these studies, we decided to investigate whether or whether the food of extremely low-birth-weight preterm babies affected their risk of developing GBA. To avoid NEC and promote optimum growth and outcomes, nothing beats fresh, pure mother's milk. Friel showed that preterm newborns fed mother's milk fortified at greater than fifty percent had elevated levels of the biomarker urine F2-isoprostane, which indicates oxidant stress. In this study, preterm babies fed either conventional or high-density caloric formulas did not have their urine isoprostanes measured. Researchers found that exposing neutrophils, endothelial, and intestinal epithelial cells assimilate the formula, but not human breast milk, caused cell death in vitro. The study's authors argued that the findings shed light on the causes of NEC. When infants are fed supplemented human milk or cow milk-based formula at a premature stage, they experience a stress response that may interfere with brain development by sending harmful signals through the GBA. Nutritional, microbial, and immunological events linked to brain damage in premature newborns are shown in Figure 4. We underline that these variables may also affect intestinal barrier function, allowing pathogenic microorganisms to invade and translocate, which ultimately results in late-onset newborn sepsis and NEC.

Very premature newborns that were given probiotics had a lower rate of necrotizing enterocolitis. Anti-TNF-alpha and -kappa B pathway inhibitors are produced by probiotic bacteria, according to recent research. Both are inhibited by probiotic bacteria, as was recently discovered. Scientists think probiotics protect the brain by obstructing the GBA, where harmful macromolecules are transported. Preterm newborns have a threefold increased chance of having ASD, and this remains true even if the most common causes of brain damage in preterm infants—intracranial hemorrhage and ischemia are ruled out. An animal model of autism spectrum disorder was shown to benefit from Bacteroides fragilis therapy by oral administration to progeny. The findings of this study imply that an altered GM contributes to the etiology of ASD. In newborn mice given enteral B. fragilis, an anti-inflammatory milieu (interleukin-10 production) may be the protective strategy.

White matter pathways in the brain are often the site of central NS injuries in premature newborns. Therefore, there is a lot of focus on imaging methods that may characterize connectome damage. Preterm newborns likely suffer brain damage due to harmful substances or neurotransmissions entering the brain through the GBA. Functional magnetic resonance imaging, Magneto, Encephalography, and positron emission tomography are all examples of cutting-edge imaging methods used to probe neural circuits. Researchers will undoubtedly reveal damage instigators connected with the GBA if metagenomic methods are functional toward the GM in tandem with intelligence representation, and the results resolve correspond with human health and illness.

**Conclusion.**

Because unfavorable environmental impacts may be the origins of ND found in childhood and adulthood, pediatric scientists must concentrate on the gut-brain axis of the fetus or preterm neonate. In this discussion, we have used autism and obesity as examples of GBA-related diseases with developmental origins. Future studies should focus on ND caused by GBA anomalies and try to pin out the underlying processes at work at a young age. Infant feces were collected and studied as part of the Human Microbiome Project. Association with the emergence of autism, cerebral palsy, or other ND in children born with very low birth weights is required to validate the molecular results linked to gut microorganisms in those investigations.

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**Conflict of interest statement.**

The author declares no conflict of interest.

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