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The Impact of the Gut-brain Axis on Alzheimer’s Disease

ELISSA WAKIM

ANT 6030: Biomedical Review Article Independent Study

Nominated by: Dr. Christopher Wyatt

Elissa Wakim, a graduate student at Wright State University, is pursuing a Master's Degree in Immunology and Microbiology with the aim of becoming a doctor. Having completed her undergraduate studies in Physiology & Neuroscience with a minor in Psychology at the same institution, she demonstrates a strong academic foundation. Amidst the challenges posed by the COVID-19 pandemic, Elissa committed herself to obtaining licensure as a certified phlebotomist, actively participating in plasma procurement for immunocompromised patients. Elissa envisions herself as a compassionate and innovative physician, leveraging her diverse background to advance medical science and improve patient care.

Student note:
In pursuing my vision of becoming a doctor, I have found my passion lies in addressing the significant knowledge gaps impacting countless individuals worldwide, particularly within the realm of neuroscience and Alzheimer's Disease. Focused on mitigating the devastating effects of this illness, I have directed my research towards understanding and improving the gut microbiome as a means of reducing the onset of Alzheimer's Disease. This approach not only targets an underrepresented group of individuals who have not yet reached a concerning stage of the disease but also offers hope to those who may not have access to preventative treatments.

Faculty note:
Elissa’s review for the Graduate Biomedical Review Article class was one of the best I have ever seen. It focuses on the links between the gastrointestinal tract and the brain; the gut-brain axis and the development of Alzheimer's disease. As a student in the Microbiology and Immunology Masters Program Elissa was particularly interested in the gut microbiota and their connection to neurodegenerative disease. She tidily reviewed the literature and wrote a fascinating and compelling piece of work.
The Impact of the Gut-brain Axis on Alzheimer’s Disease

Introduction

Alzheimer’s disease is a global health crisis that affects millions of individuals and their families daily. We all have encountered this neurodegenerative disease one way or another, and it is expected to increase dramatically within the next couple of decades. Despite rigorous research efforts, the exact etiology of Alzheimer’s disease remains unconcluded, and effective treatments are limited. Within the last few years, there has been increasing evidence that the complex and bidirectional communication between the brain and the gut (Gut-brain axis) plays a crucial role in the pathogenesis of Alzheimer’s disease. The gut-brain axis consists of a very complex system of signaling pathways that involve the enteric nervous system, the central nervous system [CNS], and the gut microbiota. Modification of the gut microbiota composition (dysbiosis) can alter brain function, promoting the development/progression of Alzheimer’s disease. Imbalances in the gut microbiota lead to increased permeability in the gut, increased systemic inflammation, and increased production of neuroactive substances. As a result, neuroinflammation, accumulation of protein aggregates in the brain and synaptic dysfunction are observed. All of these are hallmark characteristics of Alzheimer’s disease.

Understanding the complex interplay between the gut microbiota and Alzheimer’s disease offers immense hope to new research of varying mechanisms with potential paths to better therapeutics. This research paper aims to further explore the multidimensional impact the gut-brain axis has on Alzheimer’s disease, understand the underlying mechanisms, and discuss possible therapeutic strategies capable of targeting this communication system.

Increased research would not only be of scientific interest but also of ethical interest, as it offers the possibility to improve the lives of millions of diagnosed patients and their loved ones.

Alzheimer’s Disease

Alzheimer's disease [AD] is a progressive neurodegenerative disorder that primarily affects cognitive functions and leads to memory loss, impaired reasoning, and a decline in overall cognitive abilities. It is a complex and multifactorial disorder that is characterized by the progressive degeneration of brain cells, particularly neurons. This degeneration results in a gradual loss of cognitive function, memory, and the ability to perform daily activities. It is known to be the most common cause of dementia, accounting for approximately 60-70% of all cases.

Alzheimer’s disease can be broadly classified into two main types: early-onset AD [EOAD] and late-onset AD [LOAD]. Early onset AD typically manifests before the age of 65 and is often associated with genetic mutations, while late-onset AD occurs after the age of 65 and is believed to have more complex etiology involving both genetic and environmental factors. Additionally, Alzheimer's disease can be classified into sporadic familial forms, with the latter being linked to specific mutations in genes like APP, PSEN1, and PSEN2 (Alzheimer’s Association, 2021). These genes play an important role in producing the amyloid-beta plaques. Late-onset Alzheimer’s disease is mainly influenced by genetic and environmental factors.
Dr. Alois Alzheimer, a German psychiatrist and neurologist, was the first person to describe the disease in the year 1906. He documented a case of a patient named Auguste Deter, who exhibited severe memory loss, language problems, and changes in behavior. Upon her death, Dr. Alzheimer examined her brain and discovered abnormal protein deposits [known as amyloid plaques] and tangled bundles of fibers [neurofibrillary tangles], which are now recognized as hallmarks of the disease (Maurer et al., 1997). Since Dr. Alzheimer’s groundbreaking work, significant progress has been made in understanding the disease. Researchers have identified genetic risk factors, developed advanced imaging techniques, and uncovered intricate details of the underlying pathology. Despite these advances, many aspects of Alzheimer’s disease remain elusive, and effective treatments are still lacking.

The amyloid hypothesis is one of the leading theories regarding the pathophysiology of Alzheimer's disease. It postulates that the accumulation of beta-amyloid protein fragments in the brain plays a key role in disease pathophysiology development (Selkoe & Hardy, 2016). The abnormal processing of the gene AAP [amyloid precursor protein] leads to the production of beta-amyloid peptides. These beta-amyloid fragments form plaques that disrupt neuronal communication and trigger inflammation, ultimately leading to cell death (Hardy & Higgins, 1992). The amyloid-beta 42 variant of the beta-amyloid peptides has neurotoxic effects instituted by differing mechanisms including synaptic dysfunction, calcium homeostasis disruption, and tau hyperphosphorylation induction, leading to the formation of neurofibrillary tangles (Heneka et al., 2015).

Another critical aspect of Alzheimer's disease is the aggregation of tau protein into neurofibrillary tangles within neurons. Tau is a microtubule-associated protein that is mostly found in neurons. It plays an important role in stabilizing the microtubules which are involved in intracellular transport and structural integrity. In Alzheimer’s disease, these tau proteins hyperphosphorylate and undergo a conformational change. This causes them to detach from the microtubules and provoke them into neurofibrillary tangles that are insoluble. These tangles interfere with cellular function, causing neurons to malfunction and die (Ballatore et al., 2007; Iqbal et al., 2010). The exact relationship between beta-amyloid and tau pathology is an active area of research, but it is known that the spread of tau pathology through the brain contributes to the progression of cognitive decline in Alzheimer’s disease (Braak & Braak, 1991).

Oxidative stress is another key mechanism that has been implicated in the pathophysiology of AD. Oxidative stress occurs when there is an imbalance between the production of reactive oxygen species [ROS], also known as free radicals, and the ability of the body’s antioxidant defenses to neutralize them. In the context of Alzheimer’s disease, oxidative stress plays a significant role in disease progression through several mechanisms. One of which is the accumulation of amyloid-beta plaques in the brain promoting the generation of reactive oxygen species and diminishing the function of antioxidant enzymes (Butterfield & Halliwell, 2019). Tau proteins forming neurofibrillary tangles also exacerbate oxidative stress, leading to damaged cells, dysfunction, and cognitive decline as seen in Alzheimer’s disease patients (Wang et al., 2020).

Alzheimer's is a global health crisis that affects millions of individuals worldwide. According to the World Health Organization (WHO), approximately 50 million people had dementia in 2020, and this number is projected to triple by 2050, largely due to population aging. The economic burden of AD is substantial, with costs related to healthcare, long-term care, and lost productivity totaling hundreds of billions of dollars annually in the United States alone. Families of individuals with Alzheimer’s disease also bear a significant financial and emotional burden. Social impacts,
alongside economic impacts, also play a big role in AD prevalence. These impacts mainly include the caregivers and healthcare system. AD profoundly affects not only those diagnosed but also their families and caregivers. Providing care for individuals with AD is emotionally and physically demanding, often resulting in caregiver burnout and strain on family relationships. Meanwhile, the increasing prevalence of AD poses challenges to healthcare systems worldwide. Facilities and resources dedicated to dementia care are often insufficient, leading to delays in diagnosis, limited access to specialized care, and a need for innovative approaches to managing the disease.

Even though AD research has come a long way since 1906, there are still many challenges to face including lack of effective treatments, early diagnosis, complexity of the disease, and ethical/societal issues. One of the most pressing challenges is the absence of disease-modifying treatments. While several medications can temporarily alleviate symptoms, none have been shown to halt or reverse the progression of the disease. Early diagnosis is crucial for effective intervention, yet identifying Alzheimer's disease in its early stages remains challenging. Biomarkers and advanced imaging techniques are being used to enhance early detection. AD is a very complex disorder with multiple contributing factors, making it difficult to develop targeted therapies. Researchers are exploring various approaches, including immunotherapy and gene therapy, to address the diverse mechanisms involved. Lastly, as research progresses, ethical concerns related to genetic testing, consent, and data privacy become more prominent. Striking a balance between advancing scientific knowledge and protecting individuals’ rights is an ongoing challenge.

Alzheimer's disease is a devastating condition that robs individuals of their cognitive abilities and places a heavy burden on society. Despite significant advancements in research, numerous challenges remain, including the absence of disease-modifying treatments, the complexity of the disease, and ethical concerns. Continued research and investments in AD are essential to improve the lives of those affected and eventually find a cure for this debilitating neurodegenerative disorder.

Recent studies have shed new light on the complex relationship between Alzheimer’s disease and the gut microbiota. A relatively recent study led by Cryan et al. 2019 drew attention to a bidirectional communication system that linked the central nervous system and the gut to the pathology of Alzheimer’s disease. It has then been investigated that alterations to the large quantity of microbes located in the digestive tract [gut microbiome] influence the neuroinflammation, accumulation of amyloid-beta in the brain, and the oxidative stress seen in Alzheimer's disease patients. This relation allows researchers to explore the extent of influence one has on the other. This may open new avenues for research and therapeutics regarding this disease.

**Gut Microbiota// Composition & Function**

The human Gut shelters a dynamic and complex ecosystem of microorganisms commonly known as the gut microbiota. This system plays a pivotal part in human health and disease. The gut microbiota has trillions of microorganisms including fungi, bacteria, viruses, and archaea (Sender et al., 2016). The composition and diversity of gut microbiota can vary widely from person to person and can be influenced by various factors, including diet, genetics, age, and environmental exposures. Emerging research has highlighted the pivotal role of the gut microbiota in not only aiding in digestion but also in modulating the immune system, synthesizing essential nutrients, and influencing various aspects of human physiology, making it a subject of great interest in the fields of medicine, nutrition, and biology. Understanding and harnessing the power of the gut microbiota has the potential to revolutionize healthcare and enhance our knowledge of the intricate interplay between microbes and their human hosts.
The composition of the gut microbiota is a complicated and intricate ecosystem that is influenced by many different factors. Diet is one of the most potent determinants of the gut microbiota composition. A study by David et al. (2014) showed that shifts in people’s dietary patterns significantly changed the gut microbiota within a few days. These dietary shifts included the consumption of fiber-rich plant-based foods rather than high-fat, low-fiber diets. Fiber-rich diets promote the growth of beneficial bacteria such as Bifidobacterium and Lactobacillus, whereas high-fat diets are correlated to increasing the number of harmful bacteria like Firmicutes while decreasing Bacteroidetes. In addition to shifting dietary patterns, greater dietary diversity results in a richer and more resilient microbiota.

Apart from diet, host genetics play a crucial role in shaping the gut microbiota. A landmark twin study done by Goodrich et al. (2016) revealed that genetics can influence an abundance of specific bacterial taxa. While the overall structure of the microbiota is more influenced by environmental factors, certain genetic traits can predispose individuals to home-specific microbial communities. For instance, genetic variations in mucin production or immune system genes can affect the adhesion and interaction of microbes within the gut. These genetic factors, in conjunction with environmental influences, underline the personalized nature of the gut microbiota and its response to various external stimuli, contributing to the ongoing research efforts to better understand and manipulate this complex ecosystem for improved health outcomes.

**Figure 1:**

The gut microbiota plays a role in the breakdown of dietary components and in the control of the body's metabolic processes. The health and disease status, characterized by the balance between beneficial and harmful bacteria, is influenced by the production of various microbial metabolites, which in turn depends on the nutrients provided.
Because the gut microbiota is mainly located in the small and large intestines, it has a significant role in both digestion and metabolism. These two mechanisms participate in breaking down and using the dietary components acquired from our food. One main function of the gut microbiota is the fermentation of complex carbohydrates that escape digestion of the small intestine. This leads to the production of short-chain fatty acids, also known as SCFAs, like acetate, butyrate, and propionate which are taken up by the host and function as important energy sources. SCFAs have great effects on the overall health of the gut. For example, Butyrate has been associated with maintaining the integrity and function of the epithelial cells found in the colon. They do so by providing a source of nourishment to the cells which then contribute to the preservation of the barrier of the gut (Koh et al., 2016). Eubiotic microbiota provides health benefits, and dysbiotic microbiota causes harm [Figure 1]. Research has shown that adhering to a Mediterranean diet for instance is more favorable in terms of a healthy gut. This diet is rich in fruits, vegetables, whole grains, and olive oil. Mediterranean diets have been linked to lower levels of inflammatory markers like C-reactive protein and interleukin-6 (Garcia-Molina et al., 2015). Other healthy promoters include GABA, Vitamins, polyphenols, and indoles. Polyphenols are anti-inflammatory and contribute to immunomodulatory effects, thus reducing the risk of potential inflammation (Calder et al., 2011). Western diets, on the other hand, are high in saturated fats, sugars, and processed foods. All of which cause inflammation and lead to an impaired immune function (Ghanim et al., 2009). Disease promoters include amines, polyamines, H2S, and methyl phenols.

Another large process that the gut microbiota is involved in is the breakdown of indigestible plant polysaccharides and dietary fibers that are undigestible. Microbes localized in the colon carry many enzymes that can break down complex molecules into more simple forms, which are able to be absorbed and utilized by their host. The gut microbiota can convert lignans from our diet into enterolignans, which act as antioxidants and positively affect our health (Kuijsten et al., 2005). Together, these microbial activities involved in digestion increase the host's ability to obtain nutrients and bioactive compounds from the diet. These factors highlight the importance of the gut microbiota in advancing nutrient utilization and maintenance.

The relationship between the gut microbiota and the immune system is complex and begins developing early on in life, continuing to grow throughout an individual's lifespan. Being able to understand it allows us to better understand immune homeostasis. The gut is consistently being exposed to a large array of microbes, thus it’s crucial for the immune system to be able to differentiate between commensal (beneficial) and pathogenic (harmful) microorganisms. The gut microbiota aids in educating the immune system, helping its proper function and development. Studies examining germ-free animals showed that the absence of a microbiota leads to an abnormal immune system, like underdeveloped lymphoid tissues, and a compromised immune response (Smith et al., 2007).

An important mechanism that highlights the influence of the gut microbiota on immunity is the shaping of the immune cell composition in the gut-associated lymphoid tissue [GALT]. There is a large population of immune cells like B cells, T cells, and antigen-presenting cells in the gut. They are influenced by signals sent from the gut microbiota, which helps it maintain a balance between the anti-inflammatory and pro-inflammatory immune response. For instance, some gut microbiotas induce the production of regulatory T cells [Tregs]. These cells function to suppress excessive immune responses and prevent autoimmune reactions (Belkaid and Hand, 2014), which are important in preventing chronic inflammatory conditions. Commensal bacteria is able to induce the
production of anti-inflammatory cytokines, like interleukin-10 [IL-10], and weaken the proinflammatory production (Round and Mazmanian, 2009). This intricate balance of cytokines helps maintain the immune tolerance to gut microbiota and prevents unnecessary immune activation. In addition, the microbiota is also involved in producing antimicrobial peptides and immunoglobulin A [IgA]. These components are essential parts of the mucosal immune defense system (Belkaid and Harrison, 2017).

Researching the gut microbiome reveals a very complicated web of connections that goes beyond topics related to just digestion and metabolism. Microbes hold significant power of not only our physical health but also our mental health. The emerging field has allowed researchers to further study the interconnectedness, especially relating to our cognitive and emotional wellbeing. These functions are seen within the gut-brain axis. It highlights the influence the gastrointestinal system exerts on our emotional and mental states.

**Gut-Brain Axis**

The Gut-brain axis is a very intricate bidirectional system that links the central nervous system [CNS] with the gut. The central nervous system includes the brain and the spinal cord. This complex system has a large array of biochemical signaling pathways that are mediated by many components, including hormonal, neuronal, and immune mechanisms. Most relatively, the gut-brain axis permits constant information to be exchanged between the gut and the brain, which allows them to work together to regulate a host of physiological and psychological processes (Mayer, 2011).

The gut microbiota, consisting of trillions of microorganisms, plays an important role in this dialogue. These microorganisms interchange information with the gut lining, serving as the physical barrier between the gut components and the bloodstream, resulting in the production of signaling and metabolite molecules. Some of these substances can cross the gut lining, entering the bloodstream, and influencing our brain and behavior. Because of this, we would be able to see changes in our cognitive and emotional processes (Cryan et al., 2019). This interaction is fundamentally important not only in understanding digestive health but also in understanding neurological and psychiatric conditions, which continue to shed light on the implications, opening the door to a wide array of diseases and disorders.

Research has shown that signals originating from the gut can influence brain function and behavior. For example, microbial metabolites, like short-chain fatty acids and neurotransmitters, influence mood and cognition (Cryan & Dinan, 2012). Contrarily, the brain also can exert an intense influence on the gut, modulating secretion, motility, and permeability. This bidirectional interplay emphasizes the holistic nature of our health, where the brain and the gut are intricately linked together, and their actions result in alterations affecting our thoughts and behavior and leading to diseases, like Alzheimer’s disease.

The vagus nerve is a very significant component of the gut-brain axis (figure 2). It functions to mediate the bidirectional communication between the gut and the central nervous system. The vagus nerve is also labeled the tenth cranial nerve and is a major parasympathetic nerve that stimulates many organs in the body, one of which being the gastrointestinal tract. The nerve can relay sensory information from the gut to the brain using the afferent fibers. It provides feedback on the gut’s condition and signals, when in the presence of microbial metabolites, nutrients, and immune responses. The information is then sent to many different brain regions including the
nucleus of the solitary tract. This tract is responsible for regulating mood, appetite, and autonomic regulation. It can integrate the signals resulting in brain function alterations (Bonaz et al, 2016).

Figure 2 shows there are different paths of communication available between the brain and the gut microbiota, including the vagus nerve, the immune system, short-chain fatty acids, and tryptophan (the precursor of serotonin).
The gut-brain axis relies on a complex communication system of neurotransmitters and hormones that regulate the connection of the gut and the central nervous system. Serotonin (5-HT), for example, is a neurotransmitter that is primarily known for its role in regulating our mood. It is abundantly found in the gastrointestinal tract and plays a central role in the gut-brain signaling (figure 3). Enterochromaffin cells, found in the gut, release serotonin as a response to being stimulated and influence gut secretion, motility, and signaling to the brain (Mawe & Hoffman, 2013). In addition, the gut normally produces a multitude of neuropeptides and hormones such as leptin, ghrelin, and peptide YY, all of which modulate energy and appetite. Leptin is produced by adipose tissue and functions to relay information about energy storage to the brain. This aids in regulating feeding behavior and the amount of energy we can spend (Friedman & Halaas, 1998).

The gut microbiota can also affect the production and regulation of neuroactive molecules like gamma-aminobutyric acid [GABA] as well as brain-derived neurotrophic factor [BDNF]. Both molecules are also involved in mood and cognitive function (Cryan & Dinan, 2012).

**Figure 3:**

![Brain-gut interactions](image)

Figure 3 shows the bidirectional brain-gut interactions that are related to serotonin signaling. In green, the Enterochromaffin cells consist of more than 90% of the serotonin that is produced in the body. This serotonin is labeled as 5-HT, and its synthesis is regulated by SCFAs and secondary bile acid (2 Bas) that are made by spore-forming clostridiales, resulting in increased stimulation and levels of tryptophan (TPH1). Enteroendocrine cells (EECs) signal to the afferent nerve fibers via synapse-like connections.
As we expand our research of the complex mechanisms involved in the gut-brain axis, a clear intersection emerges between the gut microbiota and the shaping of its balance and communication within the axis. We have long established that the gut microbiota has influence beyond just gastrointestinal health and that it holds important implications for neurological wellbeing. This connection highlights the need to look deeper into the relationship between the health of our gut microbiota and our neurological state and condition. One condition of significant importance is Alzheimer’s Disease. With the disease projected to increase dramatically within the next few decades, research is continuing to shed light on how dysbiosis of the gut microbiota may influence the pathogenesis and progression of Alzheimer’s disease.

**Gut Microbiota Dysbiosis in Alzheimer’s Disease**

Emerging research has focused on the complicated relationship between the gut microbiota and the pathogenesis of Alzheimer’s Disease. There have been many studies linking gut dysbiosis to its pathogenicity. Dysbiosis, in our case, is the state of imbalance in the function and the composition of the gut. Studies have shown that modifications in the amount of specific microbial taxa, like Lactobacillus and Bifidobacterium (beneficial bacteria), increase proinflammatory microbes. These compositional alterations in the gut microbiota are associated with increased permeability of the gut epithelial barrier. This leads to the translocation of bacterial products like liposaccharides into the blood. The translocation activates systemic inflammation and can contribute to neuroinflammatory processes seen in the progression and development of Alzheimer’s disease (Cattaneo et al., 2017; Vogt et al., 2017).

Recent research suggests that the gut microbiota influences Alzheimer’s disease via the gut-brain axis as well. Dysbiosis is shown to lead to the production of neuroactive metabolites, such as short-chain fatty acids, which can control microglial activity in the brain, as well as promote neuroinflammation. As a result, these neuroinflammatory processes aggravate and lead to the accumulation of pathological protein aggregates (amyloid-beta and tau), which are commonly known to be hallmarks of Alzheimer’s disease.

The dysbiosis of the gut microbiota in Alzheimer’s disease patients is increasingly being recognized as a contributing factor of the pathogenesis of this disease. It is being highlighted in the understanding of the gut microbiome as a potential therapeutic avenue in the management of Alzheimer’s disease (Sampson et al., 2016; Vogt et al., 2017).

**Therapeutic Approaches Targeting the Gut-Brain Axis in Alzheimer's Disease**

Therapeutic approaches targeting the gut-brain axis in Alzheimer’s disease have gathered significant attention in the last few decades as a possible avenue of disease management. These approaches aim to regulate the gut microbiota activity as well as the composition to decrease neuroinflammation and its downstream effects on the pathology of Alzheimer’s disease.

One approach uses prebiotics and probiotics to regain the balance of the gut microbiota. Probiotics are live microorganisms that positively influence the composition of the gut microbiota, while prebiotics are non-digestible compounds associated with the promotion of beneficial gut bacteria growth. Research has shown that live microorganisms play a central role in stimulating a healthy gut microbiota and enhancing overall health. Multiple studies have focused on the
Advantages of probiotics in maintaining and restoring the gut microbiota such as that conducted by Goodoory et al. in 2023. This study, published in the Journal of Gastroenterology, showed that administering a specific probiotic strain led to significant enhancement in the symptoms of irritable bowel syndrome, such as abdominal pain and bloating. The Journal of Clinical Gastroenterology also published an important study that focused on probiotic use in patients with antibiotic-associated diarrhea. The research showed that there is certain probiotic supplementation which can reduce the risk of developing antibiotic-associated diarrhea. This supports the claim that probiotics serve as preventative measures against disturbance in the gut microbiota. Another study published in the Journal of Psychiatric Research investigated the potential connection between the use of probiotics with mood enhancement. It suggested that implementing probiotics in one’s diet may play a major role in modulating the gut-brain axis and positively influencing the mental well-being of patients. One main point that all the research highlighted was that the effectiveness of probiotics is strain-specific, which means that not all probiotic supplements will produce the same results.

Fecal microbiota transplantation [FMT] is a method that is more direct. It is a procedure that manually transplants beneficial gut bacteria from a donor stool to a patient with a damaged microbiota composition (Xu et al., 2021). Research has shown that fecal microbiota transplantations obtained from a healthy donor led to improvements in cognition and a decline in neuroinflammation (Tian et al., 2019).

Dietary interventions and nutrition have also been studied as possible therapeutic approaches to targeting the gut-brain axis in Alzheimer’s disease. Research has shown that diets rich in polyphenols, omega-3 fatty acids, and fiber have been shown to promote the growth of beneficial gut bacteria as well as reducing inflammation of the gut and brain (Bordalo et al., 2019). These dietary modifications enhance the function of our gut barrier and can decrease the translocation of proinflammatory molecules found in the bloodstream. Such approaches highlight the many strategies that can be placed to address the interplay between the gut microbiota and Alzheimer's disease. This potentially can lead to the development of new therapies in the fight against this neurodegenerative disease (Swanson et al., 2020).

Conclusion

In conclusion, this paper has highlighted the important role of the gut-brain axis in Alzheimer’s disease and focused on its profound involvement in understanding the pathogenesis and possible therapeutic interventions for this tragic neurodegenerative disorder. Current research, as exemplified by studies such as those studies by Cryan and Dinan (2019) and Meyer (2011), has shown the complex bidirectional communication between the central nervous system and the gut, highlighting the major influence the gut microbiota has on the development and the progression of Alzheimer’s disease. Dysbiosis of the gut microbiota, as discussed in studies by Goodrich et al. (2016) and Vernochi et al. (2020), is seen when there is an imbalance of microorganisms, which leads to altered microbial composition and function. Effects of this imbalance that contribute to the neuroinflammatory processes that underlie Alzheimer’s disease include an increase in systematic inflammation, gut permeability, and translocation of neuroinflammatory molecules (Belkaid et al., 2014 & Heneka et al., 2015). Many therapeutic approaches, supported by research such as that by Wang et al. (2020) and Ghanim et al. (2009), are available to target the gut-brain axis including prebiotics, probiotics, diet changes, and fecal microbiota transplantation.
Recognizing the gut-brain axis as an important player in Alzheimer’s disease allows multiple paths of research to emerge and therapeutic developments to evolve. New prospects and holistic approaches may come forth as researchers continue to better understand this complex disease. The ability to harness the microbiota’s influence on Alzheimer’s disease opens doors to a new perspective in battling this disease. While research has been ongoing and lengthy, there is still so much to discover and explore in this field with a high possibility in ultimately strengthening our ability to delay or even prevent the progression of it. This research would be able to bring so much hope to people suffering from the disease and to their families as well.
References


