The Role of Foodborne Infections in Chronic Neuropsychiatric Disorders

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Abstract

Foodborne infections have been correlated to gastrointestinal infections, but recent studies claims that certain pathogens like some foodborne pathogens also have major impact on neuropsychiatric disorders like salmonellosis, they have some association with brain and neural disorders. Disturbance in normal human microbial homeostasis can cause Crohn's, Alzheimer's and Parkinson's disease. Gut-Brain-Axis is a two-way communication system that coordinates gastrointestinal tract (GIT) and central nervous system (CNS). Thus it enables brain and gut to communicate and is a way for microbes to get access to the brain and cause damage. These disorders may include Schizophrenia, Autism spectrum disorder, Anxiety disorder, Bipolar disorder, and major depressive disorder, which affects functioning of CNS. Burden of neurological disorders, caused by foodborne pathogens has increased from 1990 to 2019. They affect the metabolism of organism after consuming contaminated food and cause diarrhea and cholera, which are highly related to neuropsychiatric disorders. Dysbiosis leads to the activation of inflammation and chronic inflammation may lead to breakdown of nervous tissues, causing various neurological and neurodegenerative disorders. This chapter explores the mechanisms used by pathogens impacting brain function, discusses role of these pathogens in neuropsychiatric disorders and their association with foodborne infections, highlighting the need of restraining consumption of contaminated food and need of further research to understand this complex interaction and its consequences on public health.

Keywords: Foodborne Pathogens, Dysbiosis, Gut brain axis, Neuropsychiatric disorders

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Introduction

Infectious diseases affect both animals and humans, generally caused by microbes (like bacteria, fungi, viruses and parasites). These infections can be air- and vector-borne (like lumpy skin disease and sheep pox infection caused by *capripoxvirus* and *phelobovirus* in animals respectively) or water- and foodborne (like cholera) infections (Trivellone et al., 2022; Qamar et al., 2023).

Ingesting pathogenic microbes (bacteria, viruses, fungi, and parasites) or their toxins (for bacteria and fungi) cause diseases, called foodborne diseases (Girma & Aemiro, 2022). Foodborne diseases affect both humans and animals (Lee & Yoon, 2021). Their short-term symptoms include nausea, vomiting, and diarrhea while their long-term symptoms include tissue damage, brain and neural disorders, and liver and kidney failure. These infections may include gastrointestinal parasitic infections (caused by microbes like *Entamoeba histolytica*, as described in table 1, or *Giardia lamblia*) or Salmonellosis or shigellosis (Girma & Aemiro, 2022). 600 million cases of foodborne diseases are reported due to the consumption of contaminated food with 420,000 deaths annually (Organization, 2024).

Foodborne infections can affect CNS, the consumption of contaminated food may also affect brain and its functioning (Healy et al., 2022). Conditions influenced by disturbances in brain-body communication or homeostatic feedback loop affecting motions and mental health is known to be neuropsychiatric disorder. These disorders may include Schizophrenia, Autism spectrum disorder, Anxiety disorder, Bipolar disorder, and major depressive disorder, which affects the functioning of CNS (Hashimoto, 2023). Burden of neurological disorders has increased from 1990 to 2019 (Huang et al., 2023).

A widely diverse ecosystem of human system includes bacteria, fungi and viruses, known as microbiota. There is a potential link between microbiota and neuropsychiatric disorders. Each organ possesses its own microbiota and has some influence over the neuropsychiatric disorders. Neurodegenerative disorders like AD, and Parkinson's disease are largely related to the variations in the gut microbiota (Hashimoto, 2023). Consumption of contaminated food leads to foodborne infections. These foodborne infections like listeria, cholera, has impact on neuropsychiatric disorders. Eating foods especially seafood, contaminated with mercury can cause psychological symptoms including tremors,

motor/cognitive dysfunction and memory loss. According to WHO, 600 million people, 1 in 10, fall sick due to the consumption of contaminated food and 420, 000 deaths have been reported annually. Eating contaminated food leads to the prevalence of symptoms including severe diarrhea, nausea, vomiting with abdominal pain. Food should be thoroughly cooked to prevent foodborne infections. There should be proper sanitation and hygiene to prevent prevalence of such foodborne infections (Qamar et al., 2023).

Aim of this chapter is to access the potential role foodborne infections in the development of neuropsychiatric disorders, the role of gut-brain axis in neuropsychiatric disorders caused by these pathogens, their fundamental biological mechanisms, including neurotransmitter alternations, neuroinflammation and immune modulation, providing the understanding of how these foodborne infections caused by alternations in gut microbiota and infections of pathogens like *Firmicutes*, *Bacteroidetes*, *Proteobacteria*, *Actinobacteria*, *Verrucomicrobia*, and *Fusobacteria* and how they contribute to mental health disorders (Quaglio et al., 2022). In addition, this chapter highlights the importance of food hygiene and further need of interdisciplinary research to establish links, ensuring public health.

1. Gut Brain Axis and Neural Inflammation

Microbial diversity colonizes usually every part of human body encompassing the skin as well as gastrointestinal, genitourinary and respiratory tract. The association of microbial communities inhabiting a specific biological domain is known as microbiota. It comprises of fungi, viruses, protozoa and prevalently bacteria (Matijašić et al., 2020). Although some are noxious but most are safe and even beneficial for human health. The human microbiota assists in developing immune system and protection against pathogens by providing a physical barrier. However, when there is a disturbance in microbial homeostasis, various diseases can occur like inflammatory bowel disease (IBD), Crohn's disease (CD), Alzheimer's disease (AD) and Parkinson's disease (Kandpal et al., 2022).

Majority of microbes resides in gastrointestinal tract (GIT) as it has abundant nutrients and large surface area, so it serves as an ideal habitat for these microbes. GIT holds more than 100 trillion microbes (Swer et al., 2023). The human gut microbes weigh almost 2.1kg and they play vital roles in body like gut permeability, nutrient metabolism, production of vitamins like vitamin K and vitamin B complexes, protection from pathogens attack and modulating immune response (Kandpal et al., 2022).

Firmicutes, *Bacteroidetes*, *Proteobacteria*, *Actinobacteria*, *Verrucomicrobia*, and *Fusobacteria* makes the most of intestinal microbial diversity. Among them almost 90% of gut microbes belong to *Fermicutes , Bacteriodetes , Proteobacteria*, or *Actinobacteria* phyla. These microbes are categorized as beneficial such as Lactobacillus*, Bifidobacterium, Enterococci*, or opportunistic bacteria such as *Clostridia, Bacteriodes*, *Bacilli and Staphylococci and Streptococci* (Quaglio et al., 2022).

The gut microbes produce metabolites which are usually aromatic amino acids, short chain fatty acids (SCFA) and Trimethylamine-N-oxide (TMAO). These metabolites enter host circulation by crossing gut barrier and have both beneficial and noxious effects. SCFA produced by anaerobic bacteria have a role in regulating gut barrier and modulating inflammatory response by activating regulatory T cells. They also regulate several pro-inflammatory cytokine levels thus inducing neuroinflammation. Aromatic amino acids like tryptophan, tyrosine and phenylalanine serve as starting product of several secondary metabolites, which work as neurotransmitters like phenylalanine is precursor of dopamine, epinephrine and nor-epinephrine while tryptophan is the starting product of 5-HT, serotonin, vitamin B13 and redox cofactors such as NAD(P)+. These metabolites have significant functions like serotonin works as a neurotransmitter that controls mood, cognition pattern, sleep, and appetite and pain perception. More than half of serotonin is formed in gut by gut mucosal cells and thus stimulates several enteric receptors and immune cells (Swer et al., 2023).

Kynurenine a derivative of tryptophan has role in immune response regulation but elevated levels of kynurenine decrease natural killer cells and dendritic cells, influencing immune response during inflammation or illness, schizophrenia and depression are also associated with imbalance in kyureninene metabolites. TMAO produced by gut microbes from dietary components such as choline and L-choline promotes CD68 expression which is indicator of dementia and neuronal aging. It also causes atherosclerosis which may also lead to dementia and cardiovascular diseases. It is a contributing factor in disturbing mitochondrial function and causing oxidative stress. Indole, a tryptophan derivative has neuroactive characteristics causing depression and influencing normal emotional state. Other metabolites such as Urolithin has anti-inflammatory and anti-oxidant properties, Anthocyanin decreases neuroinflammation and precludes oxidative stress. *Bifidobacterium* and *Lactobacillus* produces GABA (gamma-amino butyric acid) an inhibitory neurotransmitter, which has calming neural activity. Imidazole propionate causes disruption in insulin signals and causes type II diabetes which has negative impact on cognitive brain physiology and it poses a threat of Alzheimer's disease (Swer et al., 2023).

Gut- Brain-Axis is a two-way communication system that coordinates gastrointestinal tract (GIT) and central nervous system (CNS). Thus it enables brain and gut to exchange and transfer information. This interconnection allows brain to impact gut microbiota and vice versa. Gut influences brain through its microbial secretions, by regulating neuroendocrine responses, neuroplasticity associated mechanisms (neurogenesis), microglial response and by impacting overall brain health. How gut and brain are interconnected? In our intestine, the epithelial layer of villi has different types of cells, one of these enteroendocrine cells, are neuropod cells which makes synapse with vagus nerve, tenth cranial nerve, that joins visceral body organs with brain and act as a core for gut brain communication (Barton et al., 2023). This concept is actually fascinating that how gut brain axis transfer information from food to feelings. Thus through this vagus nerve, interaction takes place although there are some other immunological and endocrine paths for gut brain connection but route via vagus nerve is far more easy and direct. This route has also a potential portal for gut microbes to access brain (Zou et al., 2024).

But the normal microbiota, if disturbed can cause major problems. The imbalance of gut microbiome is usually known as dysbiosis which is usually characterized by loss of useful bacteria and abundance of harmful bacteria. It can be due to multiple factors like diet, which is low in fiber and high in sugar, lifestyle habits, hygiene, foodborne pathogens (as described in table 1,2 and 3), use of antibiotics, drugs and food additives. Several diseases which are not associated with gut can also disturb gut microbiota like SARS-COV-2 also causes dysbiosis by leaking virus into gut and binding with specific receptors (Hrncir, 2022).

1.1. Dysbiosis and Neuroinflammation

Dysbiosis is the key factor for increasing gut permeability leading to a condition known as "leaky gut". Due to this condition, toxins, metabolites and undigested food particles get a free entry into blood circulation. This activates immune system and pro-inflammatory cytokines and other immune cells are released to activate inflammatory response. These cytokines aids in brain's glial cells (microglia and astrocytes) activation via vagus nerve and blood brain barrier (BBB). Glial cells secrete neurotoxins and inflammatory mediators which leads to neuroinflammation. Inflammation is basically a protective mechanism of immune system but chronic inflammation has detrimental effects, as it might lead to continuous glial cell activation and breakdown of nervous tissue thus causing various neurological and neurodegenerative disorders (Adamu et al., 2024).

2. Mechanisms of Food-Borne Pathogens-Mediated Chronic Neuropsychiatric Disorders

Foodborne pathogens have a detrimental effect on the metabolism of an organism after consumption of contaminated food. Pathogens like bacteria, viruses, and a few parasites are considered the means of diseases like cholera or diarrhea, which can also result in neuropsychiatric disorders (Rajanna et al., 2023). Here there is an overview of mechanism

2.1. Mechanisms of Food-Borne Bacteria

i. Through Direct Brain Invasion

Listeriosis occurs through a foodborne infection called *Listeria monocytogenes. L. monocytogenes* is considered the pathogen that causes serious illness in humans. It has also been identified in domestic animals, more specifically in ruminants. This pathogen infects the CNS by crossing the blood-brain barrier when it is free in the blood. It uses internalin proteins (InIA & InIB), which bind at specific receptors (E-cadherin) of the host's cell. It causes meningitis and meningoencephalitis. Additionally, this infection can also lead to rhombencephalitis and brain abscessation (Osek & Wieczorek, 2022).

ii. Through Nueroinflamation

Helicobacter pylori is a bacterium that is mostly established in the stomach. It typically enters the human body through the oro-fecal route via contaminated food or water. Ongoing research has shown a link between H. pylori infection and neuroinflammation or neurological disorders as described in table 3, which can disturb gut microbiota and CNS through the gut-brain axis. Chronic H. pylori activates the immune system, which increases inflammation with pro-inflammatory cytokines. These cytokines damage neurons after reaching the brain. This inflammation may be associated with diseases like Parkinson's disease (Pădureanu et al., 2024).

Salmonella is involved in foodborne diarrheal diseases as described in table 4. As *Salmonella* has LPS in their cell wall. During infection, LPS leaks into the bloodstream which can provoke anxiety and depression due to the leakage of LPS (Zou et al., 2024). The LPS of *salmonella* binds with receptors of immune cells (monocytes, lymphocytes, and vascular endothelial cells), which induces the release of vascular cytokines such as TNF-α and IL-6. This quick effect of LPS can trigger septic shock both in humans and animals (Mikołajczyk et al., 2024).

The Vagus nerve is an important communication pathway between the gut and brain, which transmits signals between them. Researches have shown that gastrointestinal infection can cause anxiety-like behavior changes. A study has shown that vagotomy can reduce gut microbiota-induced anxiety in the depressive mice. The relation of the vagus nerve in linking gut inflammation and anxiety-like behavior caused by *Salmonella* is not fully understood. GABA (Gamma-Aminobutyric acid) maintains the gastrointestinal balance. Study shows the relation between *Salmonella*-induced anxiety and the vagus nerve. Vagotomy is done in research to confirm the impact of vagus nerve in depression and anxiety. Results also shows that vagotomy reduces anxiety and promotes that GABA synthesis, which would reduce the communication between gut and brain (Zou et al., 2024).

2.2. Mechanisms in Virus

i. Direct Neuroinvasion

Hepatitis E virus is a foodborne virus that is particularly involved in neurological diseases. Recent studies have shown that HEV can cross the BBB (blood-brain barrier) both in vitro and in vivo. Research found that both quasi-enveloped and non-enveloped HEV can directly invade into the brain. These studies are also found in pigs. This shows the capability of HEV to cross the BBB and can do replication in the CNS. Research found that HEV-infected Mongolian gerbils activate NLRP3-infected brain tissue, its high level leads to the damage of mitochondria and the breakdown of tight junctions in human brain cells. HEV-induced neuroinflammation and brain cell damage may contribute to neurological complications and potentially affect mental health (Wei et al., 2022).

ii. Immune activation and Neuroinflammation

Enterovirus A17 primarily spreads through oral-fecal route, including foodborne transmission (Solomon et al., 2010). First line of host defense is educed against virus, the innate immune system of host, detects the virus by PRRs, which induces Toll-like receptors (TLRs), RIG-l-like receptors (RKRs), NOD-like receptors (NLRs). These receptors trigger the immune pathways to produce inflammatory Cytokines. Unregulated immune responses such as Cytokines storm, may play crucial role in EV-A71 pathogenesis. In severe cases, this infection can also cause diseases like meningitis (Chen & Ling, 2019). Neuroinflammation triggered by EV-A71 in CNS involves negative impacts on neurodevelopment, particularly emotional and cognitive regulation as described in table 4. Studies indicate that children with severe EV-A71 CNS involvement score lower on intelligence tests compared to those with mild involvement, highlighting the long-term effects of viral-induced neuroinflammation on brain function (Lin et al., 2022).

2.3. Mechanisms in Parasites

i. Neurotransmitter Modulation

Toxoplasma gondii is a foodborne parasite that is known for causing toxoplasmosis. It can be disseminated through the ingestion of contaminated or raw food. Current research links latent toxoplasmosis infection with abnormalities related to cognitive function as described in table 3. Immune response to *T. gondii* infection triggers the production of gamma interferon, which induces astrocytes (brain cells) to release indoleamine 2, 3-dioxygenase. This enzyme can degrade tryptophan, an amino acid required for growth of *T. gondii*. This mechanism leads towards the release of neurotoxin, which can directly enhance dopamine production, which is associated with causing schizophrenia, autism, and OCD. *T.* gondii also expresses two enzymes that elevates dopamine production, which can cause hallucinations and delusion in the patient of toxoplasmosis (Dardona et al., 2023).

ii. Direct Brain Invasion

Taenia solium is the foodborne parasite that leads to many deaths, and has a very complex life cycle. Both humans and pigs are its intermediate hosts, but the definite host is human. Taeniasis occurs when a person eats undercooked pork, infected by the *Taenia solium* and contains cysticerci. The larvae develop into the adult, and its egg is released through the feces. Humans can ingest these eggs through contaminated food and water. It will cause cysticercosis, which can migrate through bloodstream towards CNS and cause neurocysticercosis. Cysticerci remain viable yet dormant in the brain for many years, their excystation triggers immune response and cause astrocytic gliosis, edema and neuronal degeneration. This leads to neurological and Psychiatric symptoms as described in table 3. These parasites evade immunity by blocking complement system and secreting cytokines. Neurocysticercosis can develop anxiety, depression, perspective sensory change and personality disorders (El-kady et al., 2021).

3. Human Foodborne Infections and Neuropsychiatric Infections

Human infection of pathogens with foodborne are generally associated with physical symptoms, such as diarrhea, vomiting, and fever. But the impact of foodborne pathogens on mental health is usually overlooked. Mental illness includes depression, anxiety, dementia, schizophrenia, personality disorder, and autism. Gut bacteria had both positive and negative impacts on mental health. They cause the release of proinflammatory cytokines such as interleukin IL-1 and IL-6, which can cause depression and other mental disorders. Foodborne parasites, including protozoa, flukes, nematodes, and cestodes, vary in their effects. Parasites in neural tissue have a larger chance of directly affecting mental health (Bolton & Robertson, 2016).

Table 1: Human Foodborne Pathogens causing Neuropsychiatric Diseases

Sr. No.	Pathogen name	Pathogen	Effects	Citation
01.	Prion Variant Creutzfeldt-Jakob disease (vCJD)	Midfolded proteins	It causes neuropsychiatric disease, showing behavioral and psychiatric symptoms such as psychotic features, agitation, and mood disorders.	(Thompson et al., 2014).
02.	*Entamoeba histolytica*	Protozoan	*E. Histolytica* may contribute to Attention deficit hyperactivity disorder i.e. ADHD.	(Elmehy et al., 2023).
03.	*Streptococcus spp.*(PANDA -related)	Bacteria	PANDA (pediatric autoimmune neuropsychiatric disorders) is a condition, caused by group A β-hemolytic Streptococci causing sinusitis and scarlet fever, characterized by OCD, tics, anxiety, depression and irritability aggression.	(Dore et al., 2024).
04.	Poliovirus	Virus	Prenatal exposure to poliovirus infection during gestation period, can elevate the risk of developing schizophrenia later in life.	(Zimmer et al., 2021).
05.	*Shigella dysenteriae*	Bacteria	*Shigella dysenteriae* causes short-term neurological disease like seizure and encephalopathy. And risk of attention deficit hyperactivity disorder i.e; ADHD.	(Sadaka et al., 2021).

Table 2: Animal foodborne infections and neuropsychiatric disorders

Sr. No.	Name of pathogen	Pathogen	Animal	Effects	Reference
1.	Avian reovirus	Virus	Chicken, birds	Arthritis, lameness, tenosynovitis, neurological and immunosuppression signs.	(Egana-Labrin & Broadbent, 2023).
2.	*Porcine circovirus*	Virus	Swine	Damage of brain glial cells and macrophages, neurological abnormalities, necrotic lesions in brain, dyspnea, coma and high mortality rate.	(Song et al., 2021).
3.	*Neospora caninum*	Protozoa	Cattle, canids	Abortion, lack of balance, nerve death, partial spinal cord distortion, triggering of an inflammatory response and neurological impairment.	(Del'Arco et al., 2022; Wei et al., 2022).

4. Diagnosis and Preventive Measures

Foodborne infections that can affect the neuropsychiatry of humans can be diagnosed by several means, it can be serological testing, to detect specific antibodies (like TES antibodies in case of *Toxocara* infections) like ELISA or western blotting. *Toxocara* is basically soil-transmitted parasite with potential foodborne transmission (Healy et al., 2022). Symptoms may include less pronounced neurological symptom and predominant gastrointestinal symptoms. Identification of toxins in serum, vomitus or stool can confirm the diagnosis of foodborne botulism. Growth and identification may require multiple days for detection (De Vet et al., 2023). Currently, molecular based culture-independent diagnostics tests (CIDTs) are used more often (Ruzante et al., 2021). Foodborne diseases are caused by contaminated food with infections pathogens. Probiotics offer biological control by enhancing immune response, and make strong intestinal barrier, which can inhibit the growth of GIT pathogens (Hassan et al., 2022). Antibiotics are used for combating with these infectious pathogens but their overuse can

lead towards the drug resistance. Now there is growing interest to use probiotics as an alternative to antibiotics for improving gastrointestinal health (Baskar et al., 2024).

Food borne pathogens cause deleterious effects on global health thus ensuring food safety is foremost concern at every level of production (Younes et al., 2024). Therefore, ensuring hygiene practices and implementing food safety measures is crucial for better livelihood and economic development. Foodborne pathogens put a burden on a country by influencing their economy and tourism. For example, there is an estimation that low and middle income countries loss USD 95 billion because of unsafe food each year. Food manufacturers and government should ensure following of control and preventive measures. A lot of preventive measures exist. Food handlers and consumers are the main focus of these interventions. These interventions are basically dependent on food safety systems, like the *Codex Alimentarius* (or "Food Code") and the Hazard Analysis and Critical Control Points (HACCP) (Levy et al., 2022). Awareness of vendors is also necessary to keep food safe and pathogen free. Studies shows that a considerable number of vendors are usually illiterate and no one ever had any food safety training and health certificate. Also wet market which is usually dirty and unhygienic should adopt preventive measures like cleaning and sanitizing utensils and wearing PPE to prevent the risk of food borne pathogens (Siddiky et al., 2022). Studies showed that food safety measures ensure reduction of microbial levels by 28.6% (Levy et al., 2022).

Table 3: Food borne Pathogens found in both human and animals causing Neuropsychiatric disorders.

Sr. No.	Pathogen name	Pathogen	Effects	Reference
1	*Toxoplasma gondii*	Protozoan parasite	Associated with worse cognitive function, and certain pathologies such as schizophrenia and bipolar disorder.	(de Haan et al., 2021; Del Pino & Zanón-Moreno, 2024).
2	*Salmonella spp.*	Bacterium	Reduce GABA levels thus cause Anxiety , depression, behavioral changes, and cognitive impairment	(Wu et al., 2022).
3	*Taenia solium*	Cestoda	Epilepsy, seizures, hallucinations, hydrocephalus, depression, social with drawl, bipolar disorder, Parkinson's disease, dementia, loss in cognitive function like attention, memory, and visual perception.	(Gale & Hedges, 2024).
4	*Helicobacter pylori*	Bacterium	Cognitive decline, trigger neuronal damage, dementia, might exacerbate atherosclerosis, thus promoting the development of cognitive decline in patients with vascular dementia.	(Wang et al., 2023).
5	*Campylobacter jejuni*	Bacterium	Can cause brain permeability, anxiety like behavior, memory impairment and inflammation.	(Wu et al., 2022).

Table 4: Treatment and preventive measures for of foodborne mediated neuropsychiatric disorders

Pathogen	Neuropsychiatric Symptoms	Treatment	Reference
Enterovirus	Seizures, Meningoencephalitis ,Guillain-Barre Syndrome (GBS)	The treatment involved intravenous immunoglobulin (IVIG), plasmapheresis, and steroids and Ribavirin is also sometime used.	(Jha et al., 2021)
Salmonella typhi	Sadness, low mood, lack of focus, feeling worthless, talking in confused manner.	Chlorpromazine	(Ukwaja, 2010).
Clostridium botulinum	Causes Parkinson's disorder, anxiety, depression and tension	Botulinum toxin A (BoNT/A) was explicitly used as a treatment for neuropsychiatric symptoms in Parkinson's disease patients.	(Zhu et al., 2024)
Listeria monocytogenes	Causes seizure, alteration of consciousness and meningitis.	Ampicillin, Amoxicillin, Linezolid, Quinolones, Cotrimoxazole.	(Pagliano et al., 2017)

Conclusion

Mental health disorders create significant burden on individuals, families, communities and global finances. Despite this, mental health disorders often gain less attention than physical health illnesses and their underlined cause usually remains unclear because they arise from mix of genetic and environmental factors. This chapter consolidates the association between foodborne pathogens and their effect on mental health. Although mechanism of action for some pathogens is clear but mechanism for some pathogens is ambiguous. Thus more future researches are needed in this area. Psychotic disorders receives very less attention than physical disorders despite their huge impact on individuals, communities, societies and global finances. The complexity of genetics, environmental and biological factors are the somehow underlying causes of neuropsychiatric disorders. This chapter highlights the evidence of link of foodborne pathogens and neuropsychiatric disorders. Studying these pathogen-induces neurological effects might open ner routes for prevention, diagnosis and treatment of mental disorders, thus there is a need of more research to improve public health policies.

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