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Environmental Microbiology and Neuroscience: The Nexus

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ABSTRACT
The brain has long been studied as a self-contained organ, and environmental microbes have long been studied as agents of ecosystem function and disease. A growing body of researches now reveal the intersection between both fields. Microbial communities in soil, water, air, and the built environment seed and reshape the human microbiota, which in turn modulates immune responses, endocrine stress responses, neural signaling, and the development and ageing of the central nervous system. Environmental microorganisms and their metabolites can also act on neural tissue directly, either as neurotoxins or as neuroinvasive pathogens, and anthropogenic pressures such as antibiotic resistance, agrochemicals, microplastics, and polluted air alter these relationships. This review integrates evidence from environmental microbiology, microbial ecology, immunology, and neuroscience. It; maps the environmental reservoirs and exposure routes that feed the human microbiome, summarizes the mechanistic pathways connecting microbial exposure to brain function, evaluates the biodiversity and "Old Friends" hypotheses, examines neurotoxic and neuroinvasive environmental taxa, considers pollution and antimicrobial resistance as modifiers, and identifies methodological weaknesses and research priorities. Most mechanistic evidence were derived from animal models, while, human data were largely associative. A well-controlled, longitudinal, and geographically diverse studies are needed before environmental microbial exposure can be treated as a modifiable determinant of brain health.

Keywords: Antimicrobial resistance, Environmental microbiology, Microbiota–gut–brain axis, Neuroinflammation, Neurotoxins, One Health

INTRODUCTION

For the most part of the twentieth century, neuroscience and environmental microbiology have developed along separate lines. Neuroscience concentrated on neurons, glia, circuits, and behavior, while, Microbiology, on the microbial communities that drive biogeochemical cycles and on the pathogens that inhabit and move through soil, water, and air. Experimental work over the past two decades has established that the gut microbiota influences brain development, stress physiology, and behaviour (Cryan & Dinan, 2012; Mayer *et al*., 2015; Sharon *et al*., 2016), and that these effects are relevant to neurodevelopmental, psychiatric, and neurodegenerative conditions (Wang & Kasper, 2014; Morais *et al*., 2021).

What is often left implicit in the gut–brain literature is the origin of the microorganisms involved. Human microbiota are not self-generating, they are assembled from maternal, dietary, social, and environmental sources throughout life (Pannaraj *et al*., 2017; Rothschild *et al*., 2018). Soil, in particular, is among the most diverse microbial habitats on Earth (Delgado-Baquerizo *et al*., 2018), and its relationship to human health is increasingly framed within a One Health perspective that links soil, plant, animal, and human microbiomes (Wall *et al*., 2015; Banerjee & van der Heijden, 2023). Water, aerosols, and indoor surfaces serve as other sources of exposure (Kembel *et al*., 2012). Consequently, the quality, diversity, and contamination status of environmental microbial communities may constitute upstream determinants of neural health (Atukunda, 2025; Mandal *et al*., 2026).

This review uses the term "nexus" to describe three distinct but overlapping relationships. First, environmental microbes act as sources for the human microbiota and thereby shape the microbiota–gut–brain axis indirectly. Second, some environmental microorganisms and their products act directly on the nervous system, as neurotoxins, neuroinvasive pathogens, or amyloid-promoting factors. Third, human activities that alter environmental microbial communities, including antibiotic release, agrochemical use, and plastic and particulate pollution, may modify both of these relationships. Existing literatures on this topic has developed along four largely separate lines. Mechanistic neuroscience and microbiology have established that the gut microbiota communicates with the brain through immune, neural, endocrine, and metabolic routes (Cryan & Dinan, 2012; Sharon *et al*., 2016; Morais *et al*., 2021). Immunological and epidemiological researches on the hygiene, Old Friends, and biodiversity hypotheses has linked reduced environmental microbial contact to immune dysregulation and, more tentatively, to mental ill health (Rook *et al*., 2013; Haahtela, 2019; Wong & Osborne, 2022), and other researchers have begun to test whether enriching children's surroundings with biodiverse material changes immune and microbial endpoints (Roslund *et al*., 2020). Toxicological and clinical microbiology have characterized environmental microbes and products that act directly on the nervous system, including cyanobacterial toxins and neuroinvasive pathogens (Cox *et al*., 2005; Disson & Lecuit, 2012; Siddiqui *et al*., 2016). Finally, environmental microbiology has documented soil, water, and effluent reservoirs of antibiotic resistance (Forsberg *et al*., 2012; Ndukwe & Ibrahim, 2024), including the effect of abattoir discharges on the physicochemical and microbial quality of a Nigerian river (Adeniyi *et al*., 2019).

Several gaps limit the value of these separate literatures. First, gut–brain reviews generalize the microbiota as a host-intrinsic feature and report insufficiently about the environmental communities from which they are assembled, while environmental studies scarcely measure neural or behavioural outcomes (Berman *et al*., 2019). Second, evidence for protective environmental exposure rests mainly on immune and allergy endpoints and on animal models, with few human studies measuring brain-relevant outcomes (Roslund *et al*., 2020; Wong & Osborne, 2022). Third, neurotoxic and neuroinvasive environmental microorganisms are usually discussed as isolated toxicological or infectious problems rather than alongside the beneficial and indirect pathways. Fourth, anthropogenic pressures such as antimicrobial resistance, agrochemicals, microplastics, and hydrocarbons are rarely considered as modifiers of the environment–microbiota–brain relationship. Fifth, the available reports come mainly from high-income, temperate regions, and settings with heavy effluent, agrochemical, and petroleum-related pollution, such as parts of the Niger Delta, are under-represented. In the literature search described in Section 2, a previous review that integrates these components together in one framework that also considers pollution and antimicrobial resistance was not identified.

Individuals are exposed simultaneously to beneficial, harmful, and pollution-modified environmental microbial communities, and the consequences for the brain can be evaluated only by examining these pathways together. Urbanization, loss of biodiversity, nutrient-driven cyanobacterial blooms (Paerl & Otten, 2013), and the spread of antimicrobial resistance are changing these exposures, giving the review or question practical significance for environmental management and public health. This review therefore aims to: highlight the environmental reservoirs and exposure routes that supply the human microbiota; summarize the mechanistic pathways connecting microbial exposure to brain function; evaluate the biodiversity and Old Friends hypotheses; describe neurotoxic and neuroinvasive environmental microorganisms; examine antimicrobial resistance and pollutants as modifiers of these relationships, using abattoir effluent-impacted soil in the Niger Delta as a regional case; and identify methodological gaps and research priorities. The search and selection procedures are described in Section 2.

Strategy for Literature Search and Selection Criteria

Review Design

This is a narrative review. It brings together evidence from environmental microbiology, immunology, toxicology, and neuroscience, whose study designs are too different to be combined statistically. The review was organized following the Scale for the Assessment of Narrative Review Articles (SANRA), a guideline for reporting narrative reviews (Baethge *et al*., 2019).

Literature Sources and Search Strategy

Relevant articles were sourced from PubMed, Scopus, Web of Science Core Collection and Google Scholar, up to November, 2026, and supplemented with regional sources, including FUDMA Journal of Sciences and African Journals Online, to capture studies from Nigeria and other parts of sub-Saharan Africa. The searches combined three groups of keywords: environmental terms (such as, soil, water, biodiversity, abattoir effluent); microbial terms (such as, microbiome, probiotics, cyanobacteria, antimicrobial resistance); and brain-related terms (such as, brain, depression, anxiety, Parkinson's disease, Alzheimer's disease). Terms for pollutants (pesticides, microplastics, hydrocarbons, antibiotics) were added for the section on pollution. The reference lists of key articles were also checked for additional sources.

Eligibility Criteria

Inclusion criteria: Articles were included if they were peer-reviewed (original studies, systematic reviews, meta-analyses, or major reviews), were written in English, and dealt with at least one of the following: (i) environmental microbes and their effects on the human microbiota, immunity, or the brain; (ii) environmental microbes or their toxins that act directly on the nervous system; or (iii) antimicrobial resistance or pollutants that change these relationships. Landmark older papers were kept regardless of date, and recent studies, controlled trials, and mechanistic experiments were preferred.

Exclusion criteria: Preprints, conference abstracts, theses, non-peer-reviewed web pages, duplicate or retracted papers, papers not in English, and gut–brain studies with no environmental link (unless needed to explain a mechanism) were excluded.

Environmental Reservoirs and Routes of Microbial Exposure

Soil, Water, Air, and the Built Environment

A global survey of topsoil showed that a comparatively small number of bacterial phylotypes dominate soils worldwide, while overall diversity remains very high (Delgado-Baquerizo *et al*., 2018). Soil communities provide a continuous supply of organisms and metabolites that humans encounter through food, dust, hands, and recreation (Thiele-Bruhn, 2021). Whether soil contributes substantially to the adult gut microbiome remains debated; a critical synthesis concluded that possible transfer routes exist, but that the quantitative contribution is uncertain and probably depends on lifestyle, age, and the degree of soil contact (Blum *et al*., 2019). Surface and drinking waters carry bacteria, protists, and cyanobacteria whose composition shifts with nutrient loading and temperature, while aerosols transport bacterial and fungal spores across scales that range from a room to a continent (Thiele-Bruhn, 2021). Indoor environments, which now account for most human time, host microbial assemblages that reflect building design, ventilation, and occupancy (Kembel *et al*., 2012).

Exposure Routes and the Concept of Microbiome Rewilding

Microorganisms reach the host by ingestion, inhalation, and skin contact (Thiele-Bruhn, 2021; Atukunda, 2025). Each route engages different immune compartments, which matters because many of the proposed neural effects are mediated by immune signaling. "Experimental greening" of urban sites has been proposed as a way to restore microbial exposure in settings where it has been depleted. This idea has been called "microbiome rewilding" (Mills *et al*., 2017). Experimental greening involves the temporary installation of plant life (vegetation) or modification of green infrastructure, in order to assess or measure psychological, environmental or structural impacts/benefits, prior permanent installation/implementation (Bogerd *et al*., 2020; Loh *et al*., 2025). Field data are consistent with the proposal that exposure to urban green space was followed by detectable transfer of environmental taxa to the skin and respiratory tract of participants (Selway *et al*., 2020). Evidence that this transfer persists, colonizes, or alters brain-relevant physiology in people is still lacking, and the gap between

taxon transfer and functional consequence should be kept in view throughout this review.

Mechanistic Pathways Linking Microbes to the Brain

Five partly independent pathways have been described as presented in Table 1 below. In practice, these independent pathways interact, and few experimental designs can separate them clearly.

Immune Regulation and Neuroinflammation

Peripheral inflammation is a recognized contributor to depression and other neuropsychiatric conditions, via cytokine signaling to the brain and via effects on neurotransmitter metabolism (Miller & Raison, 2016). Microbial exposure early in life tunes the development of regulatory immune circuits, and loss of such exposure has been argued to promote chronic low-grade inflammation and stress vulnerability (Raison *et al*., 2010; Rook *et al*., 2013). Within the brain, resident microglia depend on microbial signals for maturation. Germ-free mice showed immature microglia with an impaired response to challenge, a deficit partly reversed by short-chain fatty acids (Erny *et al*., 2015). The discovery of functional lymphatic vessels in the meninges has further clarified how immune cells and soluble factors can communicate between the periphery and the central nervous system (Louveau *et al*., 2015).

Neural and Neuroendocrine Signaling

Some of the behavioural effects of gut bacteria require an intact vagus nerve (Siopi *et al*., 2023). In mice, chronic ingestion of a *Lactobacillus* strain (a probiotic treatment) altered anxiety and depression-related behaviour and central gamma-aminobutyric acid (GABA) receptor expression, and these changes were abolished by vagotomy (Bravo *et al*., 2011). Similar findings were reported by Liu *et al*. (2021), that *L. rhamnosus* administration to male mice after vagotomy, reduced anxiety-like behavior. The hypothalamic–pituitary–adrenal (HPA) axis is another target (Liu *et al*. (2021). Germ-free mice showed an incraesed stress response that is normalized by early, but not late, colonization, which suggests a developmental window of sensitivity (Sudo *et al*., 2004). Germ-free animals also show altered anxiety-like behaviour and neurochemistry (Neufeld *et al*., 2011) and altered expression of synaptic genes (Diaz Heijtz *et al*., 2011).

Microbial Metabolites and Neuroactive Compounds

Microorganisms act as biochemical factories whose products enter host circulation, engage receptors on enteric neurons, immune cells, and the vagus nerve, and in some cases reach the brain itself (Forsythe & Kunze, 2013). Recent syntheses describe these metabolites as the principal medium of host–microbe signaling and as mediators of neuroimmune cross-talk (Ouyang *et al*., 2025; Zhou *et al*., 2025). Since, the human microbiota is assembled partly from environmental sources such as soil, water, and diet (Blum *et al*., 2019), the chemistry of environmental microbial communities is a possible upstream influence on neural function.

Short-Chain Fatty Acids (SCFAs)

Acetate, propionate, and butyrate are produced by fermentation of dietary fibre and signal through free fatty acid receptors, histone deacetylase inhibition, vagal afferents, and immune cells (Dalile *et al*., 2019; Silva *et al*., 2020). Bacterial fermentation yields short-chain fatty acids that influence gut barrier function, immune cells, and, through several routes, brain physiology (Silva *et al*., 2020).

In germ-free mice, microglia are immature and respond poorly to challenge, a defect partly corrected by SCFA supplementation (Erny *et al*., 2015). Brain concentrations of SCFAs are low and effects depend on dose and cell type, so extrapolation from rodent data should be cautious.

Tryptophan Metabolites

Dietary tryptophan is metabolized along three branches: serotonin synthesis, the kynurenine pathway, and microbial conversion to indoles such as indole-3-propionic acid and indole-3-aldehyde, which are ligands of the aryl hydrocarbon receptor and influence barrier function and immune tolerance (Agus *et al*., 2018). Specific indigenous bacteria (spore-forming) promote host serotonin biosynthesis through metabolites that act on enterochromaffin cells (Yano *et al*., 2015). Most gut serotonin do not cross the blood–brain barrier, its influence on mood is likely indirect, through enteric, vagal, and immune routes, and through the availability of precursors.

Microbial Neurotransmitters and other Neuroactive Molecules

Gut bacteria synthesize or modify GABA, dopamine and noradrenaline precursors, and acetylcholine, although the transfer of these compounds to the central nervous system remains debated (Chen *et al*., 2021). Human gut bacteria (*Bacteroides* strains) that produce GABA have been isolated, and their relative abundance was inversely related to signatures of brain activity linked to depression (Strandwitz *et al*., 2019). Butyrate-producing *Faecalibacterium* and *Coprococcus* were associated with better quality of life, and *Coprococcus* and *Dialister* were depleted in depression independently of antidepressant use (Valles-Colomer *et al*., 2019). A tyrosine-derived microbial metabolite, 4-ethylphenylsulfate, increased anxiety-like behaviour and altered oligodendrocyte maturation in mice (Needham *et al*., 2022), showing that metabolites with no classical neurotransmitter role can also reach the brain and change its function. The term “microbial endocrinology” captures the fact that microbes both synthesize and respond to host hormones and neurochemicals, a reciprocity that complicates simple cause-and-effect models (Lyte, 2013).

Environmental microorganisms also produce neuroactive or neurotoxic compounds that reach humans directly, as with the cyanobacterial amino acid β-N-methylamino-L-alanine (BMAA) (Cox *et al*., 2005). In addition, environmental exposures such as diet, antibiotics, and pollutants reshape the community and therefore its metabolic output (Ma *et al*., 2025; Zhao *et al*., 2025; Zhao *et al*., 2026) (Figure 1). Pollution-driven shifts in microbial metabolism, for instance in antibiotic-resistant or effluent-impacted communities, are possible but insufficiently tested modifiers of this axis.

Barrier Integrity

Germ-free mice exhibit a more permeable blood–brain barrier, linked to reduced expression of tight-junction proteins, and colonization with short-chain-fatty-acid-producing bacteria restores barrier function (Braniste *et al*., 2014). Barrier integrity is therefore an additional route through which microbial community composition could modulate the vulnerability of the brain to circulating toxins and inflammatory mediators.

Table 1: Principal Pathways Linking Environmental and Host-Associated Microbes to the Nervous System

Pathway	Principal Mediators	Citation
Immune regulation	Regulatory T cells, cytokine balance, low-grade inflammation	Rook *et al*. (2013); Roslund *et al*. (2020); Miller & Raison (2016)
Neural (vagal)	Vagal afferents, enteric signaling	Bravo *et al*. (2011); Liu *et al*. (2021); Siopi *et al*. (2023)
Neuroendocrine	HPA-axis programming, stress reactivity	Sudo *et al*. (2004); Reber *et al*. (2016)
Microbial metabolites	Short-chain fatty acids, tryptophan–serotonin pathway, GABA and other neuroactive compounds	Yano *et al*. (2015); Strandwitz (2018); Silva *et al*. (2020);
Barrier and glial	Tight junctions of the blood–brain barrier, microglial maturation, meningeal lymphatics	Braniste *et al*. (2014); Erny *et al*. (2015); Louveau *et al*. (2015)
Direct neurotoxicity or invasion	BMAA, domoic acid, neuroinvasive pathogens, bacterial amyloids	Cox *et al*. (2005); Disson & Lecuit (2012); Chen *et al*. (2016)

*BMAA = β-N-methylamino-L-alanine; GABA = γ-aminobutyric acid; HPA = hypothalamic–pituitary–adrenal

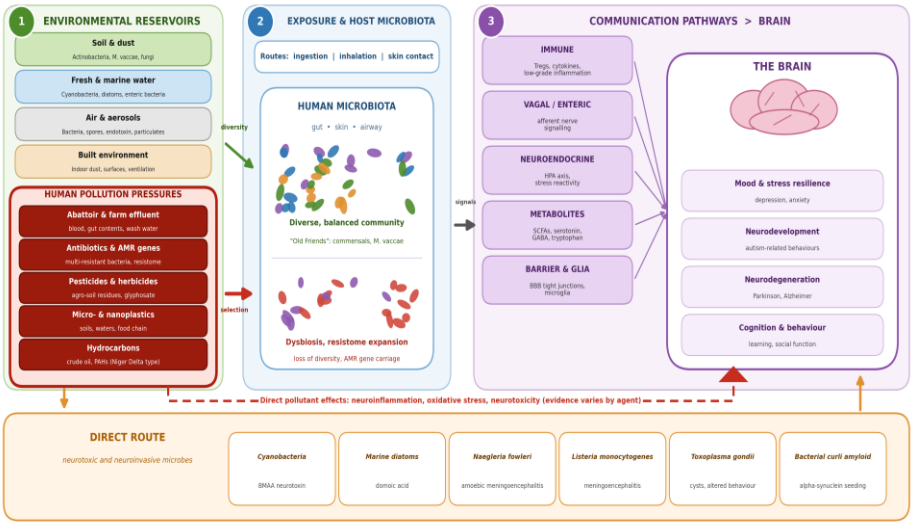


Figure 1: Communication Pathways between Environmental Microbial Communities and the Brain

(1). Soil, water, air, and built environments supply microbes that seed the human microbiota. Diversity-promoting exposure (green arrow) favours a balanced community, while, anthropogenic pressures (red box)—abattoir and farm effluent, antibiotics and antimicrobial resistance (AMR) genes, pesticides and herbicides, micro and nanoplastics, and hydrocarbons such as crude oil and polycyclic aromatic hydrocarbons (PAHs), select for dysbiosis and an expanded resistome. (2). The resulting microbiota communicates with the brain through immune, vagal, neuroendocrine, metabolite, and barrier/glial pathways (3), with implications for mood, neurodevelopment, neurodegeneration, and cognition. The dashed red route indicates possible direct pollutant effects on the nervous system; the strength of evidence differs among agents and is weakest for hydrocarbons in this context. The orange route indicates neurotoxic or neuroinvasive environmental microbes. BBB = blood–brain barrier; BMAA = β-N-methylamino-L-alanine; GABA = γ-aminobutyric acid; HPA = hypothalamic–pituitary–adrenal; SCFAs = short-chain fatty acids; Tregs = regulatory T cells.

Biodiversity, "Old Friends," and Brain Health

The "hygiene hypothesis" originated in an epidemiological observation that Hay fever was less frequent among children from larger households, which was interpreted as protection by early-life infections (Strachan, 1989). It has since been reformulated. The "Old Friends" hypothesis proposes that the immune system evolved in constant contact with harmless or commensal organisms from soil, water, animals, and plants, and that reduced contact in industrialized settings weakens immunoregulation (Rook *et al*., 2013; Rook, 2013). The "biodiversity hypothesis" extends the argument to the ecosystem level, proposing that reduced environmental biodiversity leads to reduced microbial diversity on and in the human body, with consequences for immune-mediated disease (Haahtela, 2019). Since, inflammation is implicated in mood disorders, the framework has been applied to mental health, with the suggestion that modern/urban, sanitized life may raise the risk of inflammatory-type depression (Raison *et al*., 2010).

Evidence from Epidemiology and Trials

Immune-related associations are majorly documented. Among adolescents in Finland, the diversity of environmental surroundings correlated with the composition of skin bacteria and with atopic sensitization (Hanski *et al*., 2012), and green cover around homes was associated with lower sensitization in children (Ruokolainen *et al*., 2015). Farm children in two communities with contrasting farming practices differed in innate immune profiles and in asthma prevalence (Stein *et al*., 2016), an observation consistent with the idea that microbial exposure shapes immune phenotype.

Majority of the findings that connects impact of environmental microbial communities directly to the brain come from animal studies and from population studies of green space. In mice, a soil bacterium, *Mycobacterium vaccae*, activated serotonergic neurons in a brain region that has been linked to emotional behaviour (Lowry *et al*., 2007), and its ingestion reduced anxiety-related behaviour and improved maze teaching (Matthews & Jenks, 2013).

Immunization with a heat-killed preparation of the same organism promoted stress resilience in mice and prevented stress-induced colitis (Reber *et al*., 2016). At the human level, a Danish register-based study found that children who grew up with the lowest levels of residential green space had a substantially higher subsequent risk of psychiatric disorders than those with the highest, with a dose–response pattern (Engemann *et al*., 2019). A randomized, controlled biodiversity intervention in urban day-care centres, in which play areas were enriched with forest floor and other vegetation, increased immune regulation markers and shifted the skin and gut microbiota of children relative to controls (Roslund *et al*., 2020). A review of the literature on biodiversity and mental health via microbiota concluded that the supporting evidence is still thin, with few studies directly linking biodiversity to neuropsychiatric outcomes (Wong & Osborne, 2022).

Three (3) limitations are however emphasized; 1. Green-space studies cannot separate microbial exposure from physical activity, social contact, air quality, noise, and socioeconomic position (Sandifer *et al*., 2015; Aerts *et al*., 2018). 2. the animal experiments used single microorganisms at relatively high doses and cannot be assumed to reflect the complex, low-dose mixtures individuals actually meet. 3. Trials such as that of Roslund *et al*. (2020) measured immune and microbial endpoints rather than psychiatric ones, so an inferential step remains between the surrogate and the clinical outcome.

Environmental Microorganisms as Neurotoxic or Neuroinvasive Agents

Not all environmental microbial exposures are beneficial. Several taxa act on the nervous system directly, and their ecology is driven by the same environmental variables that environmental microbiologists study.

Cyanobacterial Toxins

Diverse cyanobacteria, in aquatic and terrestrial habitats, produce the non-protein amino acid β-N-methylamino-L-alanine (BMAA) (Cox *et al*., 2005). BMAA has been proposed as an environmental contributor to amyotrophic lateral sclerosis and other neurodegenerative disorders (Bradley & Mash, 2009), and it can be mis-incorporated into proteins in place of serine in human cell cultures, which may promote misfolding and aggregation (Dunlop *et al*., 2013). The hypothesis remains contested, in part because analytical methods have differed in specificity and because epidemiological evidence in humans is limited, but it illustrates how a microbial product in water or food could participate in neurodegeneration. Bloom-forming cyanobacteria are favoured by nutrient enrichment and warming, which infers that eutrophication and climate change may increase exposure (Paerl & Otten, 2013).

Algal Neurotoxins

Domoic acid, produced by certain marine diatoms and accumulated in shellfish, is an excitotoxic glutamate analogue that causes amnesic shellfish poisoning and hippocampal injury (Pulido, 2008). These eukaryotic microalgae are part of the aquatic microbial community and are commonly covered in environmental microbiology.

Neuroinvasive Pathogens

The free-living amoeba *Naegleria fowleri*, found in warm freshwater, causes primary amoebic meningoencephalitis after nasal exposure, with very high case fatality (Siddiqui *et al*., 2016). *Listeria monocytogenes*, a food-borne and environmentally persistent bacterium, crosses the intestinal, placental, and blood–brain barriers to cause meningoencephalitis (Disson & Lecuit, 2012). Also, an apparent host behavioural manipulation by an environmentally transmitted parasite is that of *Toxoplasma gondii*. *Toxoplasma gondii* selectively alters predator- related risk perception, leaving general health and other baseline behaviours largely intact. Berdoy *et al*. (2000) demonstrated that rats infected with *Toxoplasma gondii* lose their caution against cats and avoidance of the scent of cats' urine or odour, instead, showed attraction or decreased aversion to areas marked by scent of cats. This specific behavioural manipulation consequently increases the likelihood of the rat to be preyed upon by a cat, allowing *Toxoplasma gondii* to complete its sexual life cycle in the cat's gut.

Bacterial Amyloids

Gut and environmental bacteria produce functional amyloids, such as curli, that can seed misfolding of host proteins (Chen *et al*., 2016; Van Gerven *et al*., 2018). Exposure of aged rats and nematodes to curli-producing *Escherichia coli*, enhanced alpha-synuclein aggregation in the gut and brain (Chen *et al*., 2016), providing a mechanistic hypothesis connecting microbial protein products to synucleinopathy, and other neurodegenerative diseases (Cisse *et al*., 2026).

Anthropogenic Pressures: Antimicrobial Resistance and Other Pollutants

Antimicrobial Resistance and the Environmental Resistome

Soil bacteria harbour a vast reservoir of resistance genes, and a functional metagenomic study showed that identical resistance genes occur in soil bacteria and in clinical pathogens, which demonstrates that the environmental and clinical resistomes are connected (Forsberg *et al*., 2012; Majumdar *et al*., 2026). Freshwater systems are likewise recognized as reservoirs of resistance genes whose clinical relevance depends on their transfer to pathogenic bacteria (Ndukwe & Ibrahim, 2024). Effluent from abattoirs, farms, hospitals, and municipal sources can enrich resistant organisms and resistance determinants in receiving soils and waters, and human contact with these matrices provides an exposure route (Nass & Zaher, 2025). The neuroscience relevance operates mainly through antibiotics and the microbiome. Short courses of antibiotics can leave long-lasting changes in the composition of the gut microbiota (Jernberg *et al*., 2010), and in a mouse model of Alzheimer's disease, antibiotic-induced microbial perturbation altered neuroinflammation and amyloid deposition (Minter *et al*., 2016). Zou *et al*. (2026) induced gut microbiota dysbiosis with a 14 days administration of ciprofloxacin, in mice, and observed that the microbiota dysbiosis was linked with increased susceptibility to seizure, and disruption of the integrity of intestinal and blood-brain barrier (BBB), resulting in exacerbated systemic and neuroinflammation. Antibiotic treatment in early life stage (post-weaning) of mice, impacted brain signaling pathways, specifically reducing neuroreceptor gene expression, leading to social impairment, anxiety and depression-like behaviors in mice (Johnson & Burnet, 2020). Also, drug-resistant *Streptococcus pneumoniae* and *S. agalactiae* have been shown to trigger pain-sensing neurons (nociceptors) in the meninges, thereby releasing a neuropeptide [calcitonin gene-related peptide (CGRP)] which binds to receptors on immune cells (macrophages) in the meninges, and shuts down their defense activity. This in turn allowed resistant pathogens to proliferate and cause conditions like bacterial meningitis (Harris, 2023). Thus, environmental antibiotic residues and resistant microbial communities deserves to be studied as potential neuro-

relevant exposures, although direct human evidence is currently lacking.

A Regional Case: Abattoir Effluent-Impacted Soil in the Niger Delta

Abattoir effluent illustrates how an environmental source can couple microbial resistance, and potentially neurobiological concerns. Blood, gut contents, and wash water discharged onto soil deliver enteric and skin-associated bacteria together with the selective pressure of veterinary antimicrobials and disinfectants. A phenotypic study of abattoir effluent-impacted soil in Yenagoa, Bayelsa State, isolates identified as *Salmonella* sp., *Staphylococcus* sp., *Proteus mirabilis*, and *Kluyvera cryocrescens* showed multiple antibiotic resistance (MAR) indices of 0.36 to 0.63 (Ajuzieogu, 2026). Since, a MAR index above 0.2 is conventionally taken to indicate a high-risk source of contamination (Krumperman, 1983), these values place such soils among environments where resistant organisms are likely to be abundant and where human contact, through handling, runoff, crops, and dust, is probable. Consistent with this, abattoir effluent discharged into a Nigerian river was reported to alter the river's physicochemical properties and microbial diversity (Adeniyi *et al*., 2019).

The link to neuroscience is, at present, a set of testable hypotheses rather than demonstrated effects. First, resistant enteric bacteria and resistance determinants in soil may be acquired by exposed workers and nearby residents, and the forced use of broad-spectrum antibiotics to treat the resulting infections could perturb the gut microbiota in the manner described in Section 6.1 (Jernberg *et al*., 2010; Minter *et al*., 2016; Zou *et al*., 2026). Zhao *et al*. (2025) demonstrated that Azithromycin significantly altered short-chain fatty acid (SCFA) profiles and restructured the microbe-host interactions in mice on long-term exposure to environmentally relevant doses of the antibiotic via drinking water. Second, soil is a recognized reservoir of resistance genes shared with pathogens (Forsberg *et al*., 2012), so effluent-impacted soils may broaden that reservoir, with consequences for the treatability of invasive infections, including central nervous system infections, in resource-limited health systems. Third, effluent-impacted soils in the region may also carry co-contaminants or non-antibiotic compounds such as hydrocarbons, metals, and agrochemicals, which co-selects for resistance (Samreen *et al*., 2021). Whether these independently affect neural health (Rueda-Ruzafa *et al*., 2019) has not been studied in this setting. Pairing phenotypic profiling of antibiotic resistance with metagenomic resistome profiling and microbiome and cognitive or mood measures in exposed and unexposed human groups would convert these hypotheses into data.

Agrochemicals, Microplastics, and Particulate Pollution

Herbicides and pesticides alter soil and gut microbial communities, and reviews of glyphosate suggest possible neurological consequences mediated through the gut microbiota, although the evidence base is preliminary and includes substantial uncertainty about exposure levels (Rueda-Ruzafa *et al*., 2019). Micro- and nanoplastics, which are now ubiquitous in soils and waters, can reach neural tissue in experimental systems and have been linked to oxidative stress, inflammation, and neurotoxicity (Prüst *et al*., 2020). Air pollution is associated with neuroinflammation and central nervous system disease through mechanisms that include microglial activation (Block & Calderón-Garcidueñas, 2009). Whether such pollutants act partly by restructuring airborne, soil, or gut microbial communities, rather than by direct toxicity alone, is a question that is only beginning to be addressed (Ma *et al*., 2026; Zhu *et al*., 2026). Settings with heavy loading from effluent, hydrocarbons, and agrochemicals, which include many rapidly urbanizing regions of sub-Saharan Africa, are particularly under-represented (Lapworth *et al*., 2017) in this literature and offer an opportunity for integrated studies.

Implications for Neurodevelopmental, Psychiatric, and Neurodegenerative Disorders

Neurodevelopment

In a mouse model of maternal immune activation, treatment with the commensal *Bacteroides fragilis* corrected gut permeability, altered microbial composition, and improved several autism-related behaviours (Hsiao *et al*., 2013). These findings point to the perinatal period as a critical window in which environmental and maternal microbial exposures could matter, consistent with the developmental sensitivity seen in hypothalamic–pituitary–adrenal (HPA)-axis studies (Sudo *et al*., 2004). Also, Skrabulyte-Barbulescu *et al*. (2026) reported that the traditional fermented food (Ogi) as a biologically plausible class of environmental inputs that positively calibrates perinatal glial programming through SCFA and tryptophan-derived metabolites -mediated epigenetic mechanisms, with probable downstream consequences for brain development (Abey, 2026).

Mood disorders: In mice, chronic ingestion of *Lactobacillus rhamnosus* JB-1 reduced anxiety- and depression-related behaviour and altered central GABA receptor expression through the vagus nerve (Bravo *et al*., 2011), while, transfer of gut microbiota from depressed patients to microbiota-depleted rats and mice induced depression-like behaviour and metabolic alterations in the recipients (Kelly *et al*., 2016; Zheng *et al*., 2016). Clinical translation is at an earlier stage. A fermented milk product with probiotics altered activity in brain regions that process emotion and sensation in healthy women (Tillisch *et al*., 2013), and a combination of two probiotic strains (*L. helveticus* R0052 and *B. longum* R0175) reduced psychological distress in healthy volunteers in a small trial (Messaoudi *et al*., 2011). Proposed routes include immune signaling, the hypothalamic–pituitary–adrenal axis, vagal and enteric pathways, and microbial metabolites (Dinan *et al*., 2013; Sarkar *et al*., 2016).

The concept of psychobiotics (live organisms that confer mental-health benefit), has been proposed to describe such interventions (Dinan *et al*., 2013), although effect sizes and reproducibility remain open questions.

Psychobiotics are the subset of probiotics shown to benefit mood, stress, anxiety, or cognition (Dinan *et al*., 2013). The term was later widened to include prebiotics, which are substrates selectively used by host microorganisms to confer a benefit (Sarkar *et al*., 2016; Gibson *et al*., 2017), and it also covers synbiotics (combinations). All psychobiotics are therefore probiotics or prebiotics, but not all probiotics are psychobiotics. The label has to be earned strain by strain through evidence of a mental-health effect, and results cannot be assumed to transfer from one strain, dose, or formulation to another. Interest in the area stems from the limits of current treatments and the microbiota–gut–brain axis (Rosas-Sánchez *et al*., 2025).

Clinical Evidence

An eight-week trial of a multi-strain probiotic in patients with major depressive disorder lowered depression scores and inflammatory and metabolic markers relative to placebo (Akkasheh *et al*., 2016), and an adjunctive probiotic given to depressed inpatients on anti-depressants was accompanied by

greater symptom improvement together with gut microbial and neural changes (Schaub *et al*., 2022). Meta-analyses summarized in Table 2, broadly favour probiotics over placebo for depression and anxiety, although pooled effects vary from small in earlier syntheses to moderate or large in recent ones restricted to diagnosed patients. Strain-level work suggests that *Bifidobacterium*-containing preparations improve depressive symptoms (Huang & Liu, 2024), and sleep quality also improved (Zhang *et al*., 2025). An umbrella review of 30 meta-analyses concluded that probiotics show a consistent benefit in major depression but inconsistent effects on anxiety, while evidence for prebiotics and synbiotics remains limited or insufficient (Sá Filho *et al*., 2026). Trial evidence in other disorders, such as schizophrenia, bipolar disorder, and autism spectrum disorder, remains sparse and preliminary.

Table 2: Meta-Analyses of Probiotic, Prebiotic, and Synbiotic Trials for Depression and Anxiety

Meta-analysis	Scope	Key finding
Sá Filho *et al*. (2026)	Umbrella review of 30 meta-analyses of randomized controlled trials (RCTs)	Probiotics: consistent benefit in major depression (Standardized Mean Difference (SMD) = −0.50), inconsistent for anxiety; prebiotics limited; synbiotics scarce
Liu *et al*. (2019)	34 controlled trials	Probiotics: small effects on depression (d = −0.24) and anxiety (d = −0.10); prebiotics no different from placebo
Zhang *et al*. (2023)	13 RCTs; 786 patients with depression	Benefit confined to probiotic-containing agents; effective in mild and moderate depression
Asad *et al*. (2025)	20 RCTs; clinically diagnosed samples	Probiotics: depression SMD = −0.96, anxiety SMD = −0.59; prebiotics n.s.; high heterogeneity
Zhang *et al*. (2025)	72 RCTs	Versus placebo: depression SMD = −0.53, anxiety SMD = −0.44, sleep SMD = −0.39

Neurodegeneration: Patients with Parkinson's disease differ from controls in faecal microbiota composition, and some taxa correlate with motor phenotype (Scheperjans *et al*., 2015). In mice over-expressing alpha-synuclein, the microbiota was required for motor deficits and neuroinflammation, and colonization with microbiota from patients' exacerbated symptoms (Sampson *et al*., 2016). Altered gut microbiota have also been reported in people with Alzheimer's disease (Vogt *et al*., 2017). All of these results are correlational in humans, and the direction of causation is uncertain, as disease-related changes in diet, motility, and medication can reshape the microbiota. The environmental dimension, specifically whether lifetime exposure to environmental microbes affects risk, has not been tested directly.

Methodological Challenges and Knowledge Gaps

Several recurring problems limit inference. (i) *Causality*: Most human studies are cross-sectional or observational, whereas causal evidence relies on germ-free or antibiotic-treated rodents whose physiology differs from that of humans (Morais *et al*., 2021). (ii) *Resolution*: Sequencing of marker genes describes taxa but not strain-level function, viability, or metabolite output; moving to shotgun metagenomics, metatranscriptomics, and metabolomics is essential (Gilbert *et al*., 2018). (iii) *Exposure assessment*: Metrics such as distance to green space or land-cover class are poor proxies for microbial dose. Direct environmental sampling combined with personal sampling is rarely done. (iv) *Confounding*: Diet, socioeconomic status, urbanicity, pollutant exposure, and medication all co-vary with both microbial exposure and brain outcomes. (v) *Timing*: Developmental windows for effects of exposure are probable but have been mapped only in animal models. (vi) *Net benefit versus* risk: The same soils and waters that provide commensal organisms also carry pathogens and toxins (Section 6), so any exposure-based intervention requires a safety framework. (vii) *Representation:* Most human cohorts are from high-income temperate regions, which limits generalization to tropical, low-income, and heavily polluted environments.

Future Directions

Integrated studies that treat the environment, the host microbiota, and the nervous system as one system is encouraged.

i. Birth and early-childhood cohorts should pair repeated environmental sampling (soil, dust, water, air) with host multi-omics and neurodevelopmental assessment.
ii. Randomized exposure trials of the type reported by Roslund *et al*. (2020) should be extended to include validated behavioural, cognitive, and mental-health outcomes and longer follow-up.
iii. Laboratory experiment should replace single-species challenges with defined or natural environmental consortia and low-dose, realistic exposure regimes.
iv. The neurotoxic and neuroinvasive taxa described in Section 5 should be incorporated into environmental monitoring programmes, with attention to how nutrient loading, warming, and pollution alter their distribution (Paerl & Otten, 2013).
v. Resistome surveillance (Forsberg *et al*., 2012) should be coupled with assessment of gut-microbiota and neurological outcomes in exposed populations, for example, around abattoirs and other effluent sources.
vi. Restoration practices, including soil remediation and the use of beneficial bacterial inoculants in agriculture, should be evaluated for unintended effects on microbial exposure relevant to human health, which are currently unknown.
vii. Urban planning and building design that deliberately support microbial diversity (Kembel *et al*., 2012; Mills *et al*., 2017) should be assessed through health-impact studies rather than assumed to be beneficial.

CONCLUSION

The nexus between environmental microbiology and neuroscience is supported by converging lines of mechanistic, ecological, and epidemiological evidence, but the strength of support differs greatly among its components. The microbiota–gut–brain axis is firmly established in experimental systems. The dependence of the human microbiota on environmental sources is substantial and partly demonstrated. The specific proposal that environmental microbial diversity protects mental and neurological health is a promising but insufficiently tested hypothesis, while, the

direct neurotoxic role of certain environmental microbes is well documented for some agents and yet to be determined for others. A rigorous, geographically inclusive, One Health programme that connects environmental sampling to neurobiological outcomes would transform this relationship into evidence usable by clinicians, environmental managers, and policymakers.

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