Microbiome Throughout Life

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Introduction

The human body hosts trillions of microorganisms, including bacteria, archaea, fungi, and viruses, collectively constituting the 'microbiome'. The microbiome is the entire ecological community of microorganisms together with their genetic material that inhabit a defined environment within or on the body. The microbial communities actively participate in the immune responses, metabolic regulation, neurological signalling and protection against pathogens (Cryan et al., 2019) . The gut microbiome alone encodes a genetic repertoire which is estimated to exceed the human genome by a factor of approximately 150, reflecting the metabolic versatility that these communities bring to host biology (Dong & Gupta, 2019).

The microbial communities that characterise a newborn bear little resemblance to those of a healthy adult, and the microbiome of an octogenarian, a person who is between the ages of eighty and eighty-nine, differs significantly from that of a person in their mid-life. These transitions are shaped by birth mode, feeding practices, dietary change, hormonal shifts, immune maturation and the cellular process of ageing. Therefore understanding how the microbiome changes across the lifespan is therefore fundamental to developing rational, life stage appropriate strategies for disease prevention and health promotion (Ponsero *et al.*,2022).

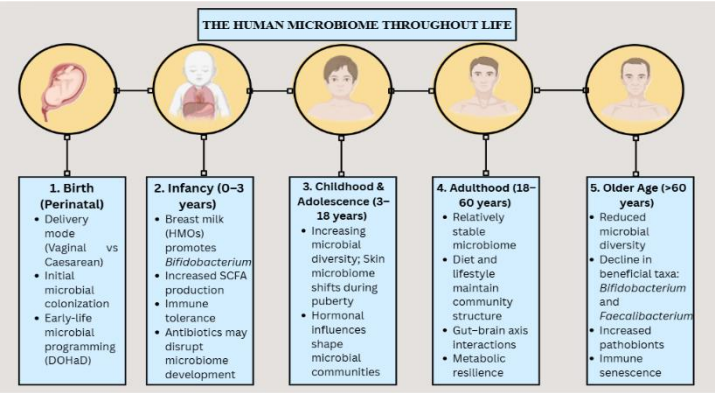

Fig. 1: Depicts the human microbiome throughout the stages of life.

1. Perinatal Colonisation and the DOHaD (Developmental Origins of Health and Disease) Framework

The question of when the human microbiome begins has been the subject of considerable scientific debate. As illustrated in Figure 1, this microbial relationship unfolds across five distinct life stages, beginning at birth and evolving through infancy, childhood, adulthood, and older age. For decades, the prevailing view held that the fetal environment was sterile and that microbial colonisation commenced only at birth. This 'sterile womb' hypothesis has since been challenged by sequencing-based studies detecting low-biomass microbial signals in placental tissue and in meconium (meconium refers to the initial dark, viscous substance present in the intestines of a developing fetus, and constitutes the newborn's first bowel movement, typically passed within 24 to 48 hours of birth) (StatPearls, 2023; Chang *et al.*, 2023). The consensus increasingly positions the intrapartum and neonatal periods as the decisive window for microbiome establishment. The term 'neonates' refers to newborn infants from birth up to 28 days of age, a period characterised by rapid physiological adaptation to the extrauterine environment (Klopp *et al.*, 2022).

The Developmental Origins of Health and Disease (DOHaD) framework, which suggests that early-life exposures programme long-term physiological trajectories, provides a compelling context for understanding the significance of perinatal microbial colonisation. During vaginal delivery, the neonate is inoculated with maternal vaginal and faecal microbiota, seeding the infant gut with beneficial taxa such as *Lactobacillus* species, *Bifidobacterium* species, and *Bacteroidetes* (Microbial Beginnings, 2024; Lyu *et al.*, 2021). In contrast, infants delivered by Caesarean section are colonised primarily by skin-associated and hospital-derived microbes, including *Staphylococcus* species, *Streptococcus* species, *Clostridium* species, and *Enterococcus* species, and exhibit reduced microbial diversity and delayed maturation compared to vaginally delivered infants (Pahirah *et al.*, 2024; Lyu *et al.*, 2021; Tian *et al.*, 2023).

Longitudinal studies have linked Caesarean delivery to heightened risks of asthma, atopic disease, and metabolic syndrome in later life, though disentangling delivery mode from the clinical indications necessitating it remains methodologically complex (Dong and Gupta, 2019). The environment plays a formative role as well. Neonates born in community settings, or those with siblings and pets, acquire more diverse microbial communities than infants raised in highly sanitised urban environments. The 'old friends' hypothesis extended the hygiene hypothesis to propose that reduced early-life exposure to commensal microorganisms, particularly those prevalent in traditional, rural, or farming environments, impairs immunological tolerance and predisposes to inflammatory and allergic conditions. These observations, now documented across multiple birth cohort studies, underscore the importance of viewing early microbial colonisation as a biological process with lifelong health implications rather than a passive event (Dogra *et al.*, 2020).

2. Infancy and Early Childhood: Diet, Immunity, and Microbiome Maturation

Following initial colonisation, the infant gut microbiome undergoes rapid and dramatic compositional shifts containing human milk oligosaccharides (HMOs), secretory immunoglobulin A, and its own microbial constituents, powerfully selects for *Bifidobacterium* dominant communities (Microbial Beginnings, 2024). HMOs are a group of complex carbohydrates present in human breast milk that cannot be digested by the infant directly, but instead serve as selective substrates for beneficial gut bacteria. Species such as *Bifidobacterium longum subsp. Infantis* possess unique genomic machinery for HMO catabolism, enabling them to outcompete potential pathogens in the early gut environment. Bifidobacterial dominance in breastfed infants is associated with increased short-chain fatty acid (SCFA) production, reduced intestinal pH, and induction of regulatory T-cell populations, collectively fostering immune tolerance (Dogra *et al.*, 2020).

Formula-fed infants harbour more diverse but less *Bifidobacterium*-enriched communities, with greater representation of *Clostridium* species, *Bacteroides* species, and *Enterococcus* species. These differences in early microbial ecology have been proposed as one mechanistic link between feeding mode and immune outcomes, although the magnitude and clinical significance of formula-feeding effects on downstream health are still being characterised (Dong and Gupta, 2019). The introduction of solid food around six months of age initiates another transformative phase. As dietary complexity increases, the gut microbiome diversifies substantially, with the proportional rise of fibre-degrading taxa including *Ruminococcus* species, *Faecalibacterium* prausnitzii, and *Bacteroides* species characteristic of the adult gut (Ponsero *et al.*, 2022).

Antibiotic exposure in early childhood constitutes one of the most significant perturbations to microbiome development. Broad-spectrum antibiotics administered during infancy have been associated with persistent reductions in microbial diversity, delayed restoration of *Bifidobacterium* populations, and increased susceptibility to dysbiosis-related outcomes including obesity, inflammatory bowel disease, and *Clostridioides* difficile infection (Dong and Gupta, 2019; Dogra *et al.*, 2020). The period from birth to approximately three years of age has been identified as a critical window during which microbiome configuration becomes relatively stable. It compositionally resembles the adult state, making early-life perturbations particularly consequential (Ponsero *et al.*, 2022).

3. Childhood, Adolescence, and the Adult Microbiome

3.1 The Adult Gut Microbiome: Diet, Lifestyle, and Resilience

The adult gut microbiome, in the absence of major perturbations, is characterised by relatively high alpha diversity and ecological stability. Alpha diversity refers to the richness and evenness of microbial species within a single sample or individual, and is widely used as a measure of microbiome health, with higher diversity generally associated with greater resilience and metabolic capacity. The dominant phyla in healthy adults are *Bacteroidota* (formerly *Bacteroidetes*) and *Firmicutes*, with contributions from *Actinobacteria*, *Proteobacteria*, and *Verrucomicrobia*. However, the notion of a universal healthy gut microbiome composition has been substantially revised; inter-individual variation in microbiome composition is enormous, and much of this variation is attributable to dietary patterns, geographic and cultural factors, host genetics, and prior antibiotic exposures (Bradley and Haran, 2021).

Diet remains the single most modifiable determinant of adult gut microbiome composition. Plant-based diets rich in fermentable dietary fibre promote the growth of short-chain fatty acid producing taxa including *Faecalibacterium prausnitzii*, *Roseburia intestinalis*, and *Akkermansia muciniphila*. *Faecalibacterium prausnitzii* is an obligate anaerobic bacterium that constitutes approximately 5% of the gut microbiota in healthy adults and is one of the most abundant butyrate producers in the human colon, contributing substantially to intestinal anti-inflammatory activity (Martín *et al.*, 2023). *Akkermansia muciniphila* is a mucin-degrading bacterium residing primarily in the intestinal mucosa that supports gut barrier integrity and is inversely associated with obesity, type 2 diabetes, and inflammatory conditions (Xu *et al.*, 2021; Zhou *et al.*, 2022). Both species are consistently associated with reduced systemic inflammation and improved metabolic health, and their enrichment has been linked to Mediterranean-pattern dietary adherence (Dong and Gupta, 2019; Ponsero *et al.*, 2022). Conversely, Western dietary patterns high in saturated fat and refined carbohydrate are associated with reduced microbial diversity and enrichment of *Bilophila wadsworthia* and other pro-inflammatory taxa. The gut-brain axis, the bidirectional communication network linking gut microbial communities with central nervous system function via immune, endocrine, and vagal pathways, has emerged as a major mechanistic frontier with implications for mental health, neurodegenerative disease, and stress responsivity (Dickerson *et al.*, 2023).

4. The Microbiome and Mental Health Across the Lifespan

The gut produces over 90% of the body's serotonin, and microbial metabolites including SCFAs, tryptophan catabolites, and secondary bile acids modulate neurotransmitter biosynthesis, hypothalamic-pituitary-adrenal (HPA) axis reactivity, and neuroinflammatory tone (Dickerson *et al.*, 2023). Specifically, *Lactobacillus* and *Bifidobacterium* species produce gamma-aminobutyric acid (GABA) and serotonin, crucial neurotransmitters involved in mood regulation, and depletion of these beneficial bacteria can disrupt neurotransmitter signalling (Liu *et al.*, 2025).

Across the lifespan, the timing of microbiome-brain interactions appears critical. Preclinical evidence demonstrates that germ-free mice exhibit exaggerated stress responses that can be normalised by conventional colonisation only during a discrete early postnatal window, implying microbiome-dependent programming of the stress axis. In humans, maternal gut and vaginal dysbiosis during pregnancy, including *Gardnerella vaginalis*-predominant communities associated with bacterial vaginosis, have been linked to elevated offspring risk for autism spectrum disorder, anxiety, and cognitive delay, though causality has not been established (Dickerson *et al.*, 2023).

In adulthood and older age, gut dysbiosis has been associated with increased prevalence of depression and anxiety disorders. Meta-analyses of gut microbiome profiling in depressed individuals consistently identify reduced *Lactobacillus* and *Bifidobacterium* abundance alongside enrichment of pro-inflammatory taxa (Martín *et al.*, 2023). Psychobiotics, a term coined to describe live microorganisms that, when ingested in adequate amounts, confer mental health benefits via the gut-brain axis, have yielded notable results in clinical trials.

A systematic review of 51 randomised clinical trials involving 3,353 patients found notably high effectiveness of psychobiotic interventions, particularly *Lactobacillus* and *Bifidobacterium* strains administered over four to twenty-four weeks, in ameliorating depression symptoms (González-Leal *et al.*, 2024). These findings support a significant relationship between gut microbiome composition and psychological wellbeing.

5. The Ageing Microbiome: Immunosenescence, Inflammaging, and Disease Risk

The microbiome of older adults is characterised by patterns consistently distinguished from younger counterparts across multiple large-scale studies (Bradley and Haran, 2021; Aleman and Valdes, 2021). The dominant hallmarks of the ageing gut microbiome include reduced alpha diversity, contraction of *Bifidobacterium* and *Faecalibacterium* populations, increased inter-individual compositional variability, and enrichment of facultative anaerobes and potentially pathogenic taxa. A facultative anaerobe is a microorganism capable of surviving in both the presence and absence of oxygen, and their enrichment in the ageing gut reflects the breakdown of the oxygen-depleted mucosal environment that typically supports strictly anaerobic beneficial species. These shifts are closely intertwined with immunosenescence, the progressive age-related decline in immune function characterised by reduced capacity to mount adaptive immune responses, and inflammaging, the chronic, low-grade systemic inflammation that characterises biological ageing and underlies the pathophysiology of cardiometabolic disease, neurodegeneration, sarcopenia, and malignancy (Ponsero *et al.*, 2022; Bradley and Haran, 2021).

The gut microbiome contributes to inflammaging through multiple pathways. Reduced mucosal barrier integrity, partially attributable to declining *Akkermansia muciniphila* and SCFA-producing bacteria, facilitates translocation of lipopolysaccharide (LPS) and bacterial products into systemic circulation, triggering pattern recognition receptor-mediated inflammatory signalling. LPS, also called endotoxin, is a component of the outer membrane of gram-negative bacteria that, when translocated into the bloodstream through a compromised gut barrier, activates immune receptors and drives systemic inflammation (Xu *et al.*, 2021). Simultaneously, age-associated reductions in bile acid diversity, altered tryptophan metabolism, and declining production of immunomodulatory SCFAs compromise the capacity of the ageing gut to maintain immune tolerance (Bradley and Haran, 2021; Biagi *et al.*, 2010).

Comparative microbiome studies of elderly individuals across different health trajectories have provided important insights into what constitutes healthy ageing from a microbial perspective. Centenarians and individuals classified as healthy agers consistently harbour more diverse gut communities with higher proportions of Christensenellaceae, *Akkermansia muciniphila*, and secondary bile acid-producing bacteria than age-matched individuals with chronic disease burdens. *Christensenellaceae* are a family of gram-positive, strictly anaerobic bacteria associated with host genetics and consistently linked to leanness and reduced inflammation. An Italian centenarian microbiome study identified enrichment in taxa with anti-inflammatory and immunomodulatory properties, including *Bifidobacterium* and certain *Clostridiales*, suggesting that the microbiome may contribute not merely to lifespan but to healthspan, the period of life lived in good health (Xu *et al.*, 2021; Biagi *et al.*, 2010).

6. Mechanisms of Microbiome Change and the Role of Lifestyle

Multiple mechanisms drive microbiome evolution across the human lifespan. At the physiological level, host factors including intestinal transit time, gastric acid secretion, bile acid composition, and mucosal immune activity change substantially from infancy to old age, each exerting selective pressures on gut microbial communities. Declining oestrogen levels in post-menopausal women have been linked to reductions in *Lactobacillus*-dominant vaginal communities and increased susceptibility to bacterial vaginosis, urinary tract infection, and genitourinary microbiome dysbiosis (Ponsero *et al.*, 2022).

Beyond host physiology, exogenous modulators including polypharmacy contribute substantially to age-associated microbiome changes. Polypharmacy refers to the concurrent use of multiple medications, typically defined as five or more drugs, and is highly prevalent in older adults. Proton pump inhibitors, non-steroidal anti-inflammatory drugs, metformin, statins, and antidepressants have each been

shown to alter gut microbial composition, though the net health implications of these drug-microbiome interactions remain incompletely characterised (Bradley and Haran, 2021;Biagi *et al.*, 2010). The cumulative antibiotic burden accumulated over a lifetime is particularly consequential: studies demonstrate that each course of broad-spectrum antibiotics produces lasting microbiome perturbations, and that the resilience capacity of the gut microbiome to restore post-antibiotic diversity declines with advancing age (Dogra *et al.*, 2020).

Dietary patterns in older adults, often characterised by reduced food variety, lower fibre intake, and protein inadequacy, accelerate age-associated microbiome deterioration. Institutional care, which restricts dietary autonomy and increases exposure to repeated antibiotic courses, is associated with particularly pronounced microbiome impoverishment (Bradley and Haran, 2021). On the other hand, the NU-AGE trial found that older adults who maintain a Mediterranean diet can protect their gut health. The diet reduces age-related inflammation in the stomach by increasing good bacteria and lowering bad bacteria (Ponsero *et al.*, 2022).

7. Therapeutic and Preventive Implications Across Life Stages

In early life, promoting vaginal delivery where clinically safe, supporting breastfeeding, limiting unnecessary early antibiotic exposure, and ensuring diverse early-life dietary exposure represent the most evidence-grounded approaches to optimising microbiome development (Dogra *et al.*, 2020; Dong and Gupta, 2019). Probiotic supplementation of preterm neonates with *Bifidobacterium longum* and *Lactobacillus rhamnosus* GG-based formulations has demonstrated efficacy in reducing necrotising enterocolitis incidence, representing one of the most clinically validated applications of microbiome-targeted intervention (Dogra *et al.*, 2020). Necrotising enterocolitis is a serious gastrointestinal emergency occurring predominantly in preterm infants, characterised by intestinal tissue death and systemic inflammation with high associated morbidity and mortality.

In adulthood and old age, therapeutic strategies targeting the gut microbiome span probiotics, prebiotics, synbiotics, postbiotics (bioactive microbial fermentation products), and faecal microbiota transplantation (FMT). FMT, which involves transferring faecal material from a healthy donor to a recipient's gastrointestinal tract to restore microbial diversity, has demonstrated remarkable efficacy in recurrent *Clostridioides difficile* infection and is under active investigation for metabolic syndrome, inflammatory bowel disease, and age-associated cognitive decline (Bradley and Haran, 2021; Biagi *et al.*, 2010).

The concept of gerobiotics, which refers to probiotic formulations specifically designed to counteract age-associated microbiome shifts in older populations, represents an emerging frontier, with early clinical trials investigating the capacity of targeted *Lactobacillus* and *Bifidobacterium* supplementation to reduce inflammaging markers and improve immune function in older adults (González-Leal *et al.*, 2024; Liu *et al.*, 2025). The microbiome also intersects with immune maturation in ways that have therapeutic implications for vaccine responsiveness across the lifespan. Gut microbial composition has been identified as a significant determinant of vaccine immunogenicity, higher *Bifidobacterium* abundance has been associated with superior oral rotavirus vaccine responses in infants, and *Faecalibacterium prausnitzii* enrichment has been linked to improved influenza vaccine responses in older adults (Dogra *et al.*, 2020). These findings open the possibility of microbiome pre-conditioning as a strategy to enhance vaccine efficacy in populations where immune responses are suboptimal.

Conclusion

The human microbiome is not a static biological constant but a dynamic ecological system that evolves in concert with its host from the first moments of life through the final stages of ageing. Its early configuration is shaped by birth mode, feeding practice, antibiotic exposure, and environmental microbial richness; its adult stability by dietary diversity and lifestyle; and its age-associated decline by the intersection of physiological senescence, polypharmacy, and reduced dietary quality. Suboptimal microbiome programming during critical early-life windows may underlie elevated chronic disease risks associated with Caesarean delivery, formula feeding, and early antibiotic use. At the other end of life, the preservation of microbial diversity and anti-inflammatory community structure in older individuals suggests the microbiome contributes not only to lifespan, but also to health span. Longitudinal, life-course microbiome studies and scalable microbiome-targeted interventions across all developmental windows remain the most promising path forward.

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