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* **Corresponding author.**
drushakini@gmail.com

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The Microbiome and the Thinking Gut

Usha Kini[1, 2, 3, 4*]

1 Professor of Pathology (Research), St. John's Medical College, Bangalore.
2 Emeritus Scientist, ICMR, India.
3 Professor of Eminence, SDUAHER, Kolar, Karnataka.
4 President, Indian Association of Pathologists & Microbiologists, 2025.

Virtually all humans live in close association with surrounding microbes, like all multicellular organisms are inhabited by a vast number of organisms, namely bacteria, archaea, viruses, and unicellular eukaryotes. Especially that group of microorganisms that live in peaceful coexistence with their hosts are specifically grouped as microbiota, microflora, or normal flora. Interestingly, the most heavily colonised group is the Gut microbiomes. When considering such a hidden group of intelligence, one is reminded of Hippocrates, who said “death sits in the bowels” and “bad digestion is the root of all evil” way back in 400 BC. This sector of the human gut microbiome has made tremendous progress, rapidly moving from scientific curiosity to the clinical frontier. However, amid the wave of research projects, their publications, use of probiotics and promises of precision, one truth demands emphasis: we are interpreting an ecosystem much more complex than we are prepared to control. Before we set the target, namely, the gut microbiome and fix it, it is a system to be understood.

Though historically, the roots of microbiome science date back to the first observations of gut flora in the late 19th and early 20th centuries, when the researchers first recognised the symbiotic relationship between microbes and human hosts, the concept gained momentum when there were advances in cultivation techniques, but remained limited by what could be grown in vitro. It was only with the advent of high-throughput sequencing and metagenomics in the 21st century that the true diversity and functional capacity of the gut microbiome were revealed, emphasising that the greatest discoveries were not the new organisms, but the new ways of identifying them.

Central to this ecosystem is the role of specific bacterial species, each contributing uniquely to the physiology of the host. As the group of commensal organisms, such as Bacteroides, Lactobacillus and Bifidobacterium, is found essential for maintaining intestinal homeostasis, they were found to participate in the fermentation of dietary fibre, the production of short-chain fatty acids, such as butyrate, thus reinforcing the integrity of the mucosal barrier of the gastrointestinal gut. These metabolic byproducts are not trivial, for they influence epithelial health, immune tolerance, and further influence systemic metabolism. They, thus, prove the point that not all microbes are the same; function defines meaning.

On the other hand, when there is a shift in the microbial composition with loss of beneficial species or excessive representation of the pathobionts, disturbing the delicate balance of the gut flora, permitting species such as certain strains of Escherichia coli or Clostridioides to result in mucosal inflammation, toxin production, and disease progression. Importantly, it is not just the presence of these organisms, but their relative abundance, genetic expression and

interaction with the host that determine pathogenicity to prove the point that pathogenicity is context and not identity.

The microbiome, therefore, must be understood not as a static inventory, but as a functional network, interacting further with the enteric nervous system with its dense network of between 500 and a million neurons within the gastrointestinal wall, creating a masterpiece of biological autonomy, nick named as the “second brain”. Interactions between species resulting in competition, cooperation and metabolic cross-feeding further shape the overall ecosystem. Critically, this is where pathology must reaffirm its central role.
The study of disease has always been rooted in morphology, but the microbiome requires an expansion of this lens – from structure to function and from cells to systems, and we must be in a unique position to couple histological findings with microbial signatures, correlating tissue response with underlying microbial dynamics to assess its impact on pathophysiology and hence treatment.

Emerging techniques such as in situ hybridisation, immunohistochemistry, and spatial transcriptomics permit localisation of microbial elements within the tissue architecture, permitting researchers not only to detect organisms but also to understand their spatial relationships with host cells – critical information for distinguishing colonisation from invasion and association from causality.

The integration of microbiome data into routine diagnostics has remained an unmet need. Probably, the histopathological patterns, namely, chronic inflammation, mucosal disruption, and dysplasia, may need to be interpreted alongside microbial profiles, thus emphasising the need to have integration of all the lab facilities which are under one roof for better correlation. This integrative approach has the potential to define and refine disease classification in various conditions, such as autism, metabolic syndrome, inflammatory bowel disease, cholelithiasis, minimal hepatic encephalopathy, colorectal carcinoma, etc., provided they are guided by validated frameworks rather than exploratory enthusiasm. Without standardisation, reproducibility, and clinical correlation, such integration risks add complexity without clarity, for dysbiosis remains a hypothesis and not a diagnosis.

The clinical applications – or putative uses – of microbiome science are expanding rapidly. Established use include faecal microbiota transplantation for recurrent Clostridioides difficile infection, where results have been transformative. Emerging areas include microbiota modulation in inflammatory bowel disease, metabolic disorders, neuropsychiatric conditions, and even oncology, where microbial signatures can influence the response to immunotherapy.

To note is that enthusiasm must be tempered with rigour. Probiotics and prebiotics are widely consumed across the globe, often with the assumption of universal benefit despite strain-specific effects and variable host responses. Personalised nutrition based on microbiome profiling is definitely an attractive concept, but it is still in its infancy. Equally concerning is the rise in direct-to-consumer microbiome testing. These reports, often presented with unwarranted certainty, offer dietary and therapeutic recommendations not supported by standardised clinical frameworks. They risk misleading patients and undermining trust in evidence-based medicine.

From a pathological perspective, it's interesting to note here that the microbiome challenges traditional disease classification paradigms. It forces us to reconsider boundaries, namely, between host and environment, health and disease, individuality and universality. No two microbiomes are identical, and therefore, there is no single “ideal” microbiome. This variability demands a shift toward precision medicine as a disciplined approach that integrates genomics,

metabolomics and clinical phenotyping. Thus, the future lies not in broadly altering microbial communities, but in understanding their functional outcomes and interactions with the host. The question, thus, is no longer 'What is there?' but 'What are you doing?'. Antibiotic overuse, dietary homogenization, and environmental disruption have not only altered microbial diversity but potentially reshaped disease epidemiology itself. In this light, stewardship becomes imperative – not only in relation to antibiotics, but also in relation to interventions that aim to "restore balance" without defining it.

The gut microbiome, thus, is not a therapeutic shortcut but a scientific challenge. Its promise is undeniable, but so is the responsibility it demands. As we as clinicians and researchers, we must resist the allure of premature application and instead commit to deeper, more integrated investigation.

Only through rigour and moderation can this field of gut microbiome mature from fascination to foundation!