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Bridging the Educational Gap: Integrating Gut–brain Axis Science Into Health Professional Curricula to Improve Clinical Care

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Abstract

The microbiota-gut-brain axis (GBA) constitutes a two-way regulatory system that incorporates microbial metabolites, epithelial barrier health, immune signaling, autonomic routes, and neuroendocrine responses. Mechanistic progress evidences that the cytokine cascades, short-chain fatty acids, vagal modulation and hypothalamic-pituitary-adrenal axis interactions play a role in multisystem conditions that cut across gastrointestinal, neuropsychiatric, and metabolic disorders. In spite of the fact that this scientific framework has grown up, health professions education is still structured largely in terms of compartmentalized organ-based models, constraining learners' ability to use systems-informed solutions to problematic clinical presentations. This study is a narrative review that synthesizes and evaluates recent mechanistic and translational literature (2020-2025) and explores ways in which the curriculum can be improved. The research suggests a competency-based framework of combining GBA literacy longitudinally in foundational sciences, clinical education, and evaluation frameworks. The framework puts emphasis on mechanistic coherence, structured curriculum mapping, case-based and simulation-based reinforcement, as well as alignment with competency-driven evaluation. Integration is viewed as restructuring of the curriculum as opposed to expansion, thus alleviating overloading of content. The heterogeneity of evidence of microbiome, the risk of premature clinical extrapolation, and faculty development and educational outcomes research should be critically taken into consideration. The integration of curricula with current systems biology can enhance diagnostic reasoning, cross-disciplinary cooperation, and evidence-based patient care. Critical and proportionate incorporation of gut-brain science provides an example of how translational gains can be offered even-handedly in coherent clinical training in a mature and continuously intricate chronic disease.

Keywords*:* gut–brain axis; microbiome education; health professions curriculum; systems-based learning; clinical reasoning; integrative biomedical training

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1. Introduction

The gut–brain axis (GBA), also known as the microbiota–gut–brain axis, is a multiscale multidirectional communication system that connects the gut and its associated microbes with the host's central nervous system (CNS) by integrating neural, immune, endocrine, and metabolic signals. Modern molecular studies have redefined the GBA from a predominantly digestive system network to a moving neurochemical interface that controls cognition, affective processing, and responsiveness to stress as well as systemic inflammatory tone. At the cellular level, crosstalk between the gut and brain is mediated by microbial metabolites, receptor-dependent signal transduction cascades, immune modulators (including cytokines), and neutrally derived signaling pathways that collectively regulate synaptic plasticity, neurogenesis, and barrier function. Pivotal work revealed that short-chain fatty acids (SCFAs), cytokine signaling networks, and vagal afferent activity serve as biochemical messengers connecting microbial metabolism to host neural and immune regulation, while central nervous system outputs reciprocally govern gastrointestinal motility, permeability, and microbial ecology (Glinert et al., 2022; Montagnani et al., 2023; Qi et al., 2021). This systems-based concept recasts human physiology as a host–microbe signaling network, whereas it questions the current reductionist organ-defined paradigms of disease by emphasizing molecular cross-talk across different biological realms.

Rapid development of gut–brain biology has brought insights into molecular dysfunction in this communication axis underlying these and related neuropsychiatric, gastrointestinal, metabolic, and neurodegenerative diseases. Experimental and translational data suggest that dysbiosis impinges on cytokine signaling, glial activation pathways, and neurotransmitter production via metabolite-triggered immune and neuroendocrine cascades. Such molecular changes contribute to synaptic reorganization, regulation of the hypothalamic–pituitary–adrenal (HPA) axis, and inflammatory signaling pathways, supporting mechanistic models for this bidirectional association between physiological and behavioral outcomes. (Rusch et al., 2023; Yang et al., 2021). Clinical co-associations evidencing a disequilibrated gut microbiota with disease outcomes and therapeutic responses, including those of irritable bowel syndrome, anxiety disorders/depressive symptoms, autism spectrum disorders, and Parkinson's disease, among others, underscore the microbiome's roles as a biologically active mediator of susceptibility to and responses to therapy for disease. (Nakhal et al., 2024; Naufel et al., 2023). Critical are the microbiome-mediated molecular pathways being identified as intervention targets, such as diet-modulated metabolite signaling, psychobiotic therapies, and precision medicine approaches. Together, these advances highlight the translational impact of gut–brain communication for current biomedical research.

Mechanistically, gut–brain crosstalk at the cellular level results from a coordinated interplay of cellular processes such as receptor-mediated metabolite signaling, epigenetic modification, immune activation and neuroendocrine regulation (Sun et al., 2024; Yang et al., 2023). Effects of microbial metabolites, including SCFAs, on host physiology mediated through G-protein–coupled receptor signaling, histone deacetylase regulation, and activation of inflammatory

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transcription pathways that connect dietary substrates with microbial ecology and the neural and systemic effects (Zhang et al., 2024). At the same time, peripheral signals are conveyed to central neural circuits via immune-derived mediators and barrier-altering mechanisms to influence glial activity and neuroinflammatory tone. Together, these integrated pathways define the microbiota as an active biochemical regulator of host homeostasis rather than a passive commensal niche. A clear understanding of this molecular circuitry is fundamental to transition the expanding discovery in microbiome science into clinically actionable paradigms that embrace systems-level cross-talk and therapies. (Glinert et al., 2022).

Despite the accelerating throughput of molecular discovery, most training models for health professionals are organized around fragmented organ-system curricula that do not convey integrative biological signal transduction systems. This disconnect limits clinicians from interpreting evidence associated with the microbiome, performing a critical appraisal of mechanistic information, and providing systems-level decision-making in the light of patient care. This kind of educational atomization may therefore hinder the potential for gut–brain science to influence diagnostic reasoning, therapeutic considerations, and prevention policy. As such, the current health professions education literature is beginning to understand that modern clinical reasoning requires a literacy in interaction among biological systems, rather than one among organ models as standalone phenomena (Cadet, 2024; Matinho et al., 2022; Morales, 2025). Gut–brain biology is a paradigm example of this, embodying the potential of molecular cross-fertilization to break down historical siloes between disciplines, while also demanding mechanistic insight as a basis for translational competency in this milieu.

This would make it a translational and professional imperative to shift the field from molecular gut–brain science (for an academic audience) to clinical education (for a professional audience). Integrating mechanistic literacy into preclinical and clinical education fosters systems-based thinking, interdisciplinary collaboration, and evidence-based translational medicine. Matching curricula with innovations in microbiome biology will empower clinicians to place new research into context, thoughtfully assess interventions that target the microbiome, and communicate biologically informed plans of care with their patients. This review synthesizes recent gut–brain molecular mechanisms, discusses their translational applications, and addresses how mechanistic literacy may influence health professional training. By placing microbiome–gut–brain biology within an integrative molecular model, the field advances toward clinical reasoning models that more accurately represent human physiology and modern biomedical science. Figure 1 depicts the multilevel molecular and cellular networks, which regulate gut–brain signaling, through metabolite-induced immune activation, neural communication circuits, and barrier-regulatory processes that underlie this axis.

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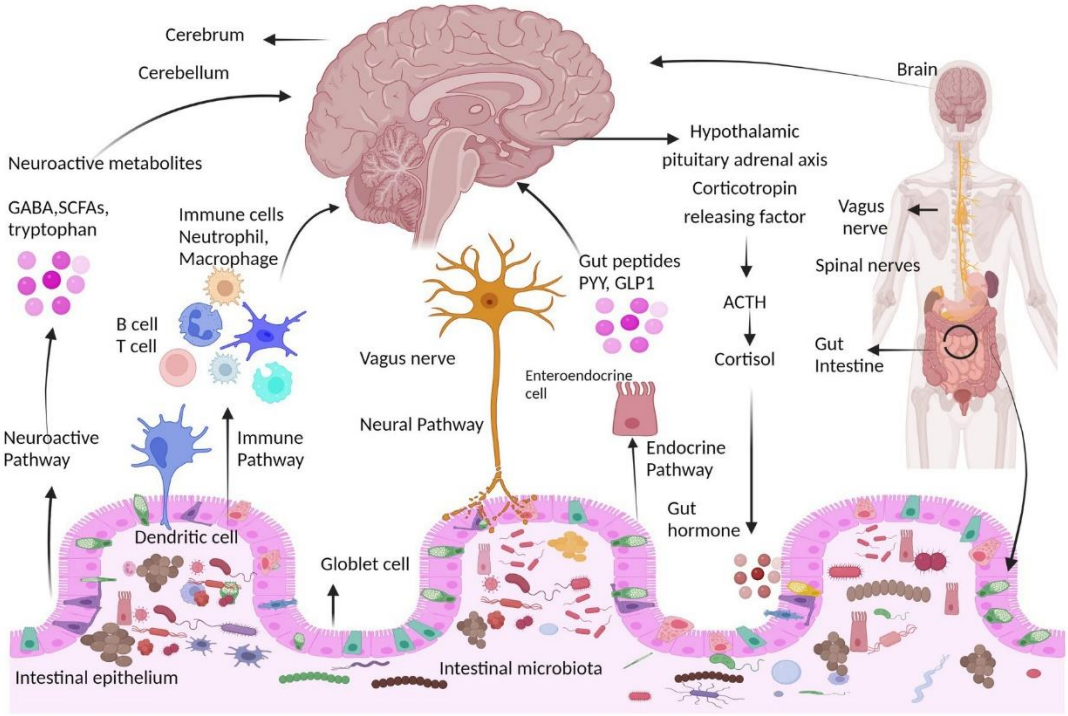

Figure 1: Communication pathways
Source: (Ullah et al., 2023)

2. Methodology

The paper is a narrative review, and the goal is to summarize the existing mechanistic knowledge of the microbiota-gut-brain axis and discuss the implications of this research on health professions education. The search was performed methodically in PubMed, Scopus and Google Scholar, 2020-2025, based on the following keywords: gut-brain axis, microbiome education, systems-based curriculum, health professions training, neuroimmune signalling, and translational medicine. The preference was given to peer-reviewed articles (especially systematic reviews, mechanistic, and educational scholarship linked to curriculum design and competency-based training). Sources were selected to achieve a conceptual representation of mechanistic and educational themes. This methodology shows the purpose of the manuscript of the integrative synthesis instead of a systematic review.

3. Scientific Foundation: The Gut–Brain Axis

The GBA, is defined as the microbiota–gut–brain axis and is considered to be a bidirectional and multilevel two-way signaling pathway between commensal, symbiotic, or pathogenic organisms inhabiting the gastrointestinal tract and the brain. Current syntheses highlight that this is not a single pathway. This means that it is rather a system in which microbial metabolites, neural circuits, especially the primary vagal and enteric signaling, immune mediators, and endocrine

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outputs converge to inform brain function, behavior, and systemic physiology (Guo et al., 2025; Lu et al., 2024). Molecularly, this convergence is achieved through receptor-mediated signaling cascades, metabolite-mediated transcriptional regulation, and immune cell activation machineries that link microbial metabolism to host neural function. These pathways include G-protein–coupled receptor signaling, cytokine-based inflammatory networks, and barrier-protecting processes controlling central and peripheral physiological responses. Furthermore, a systems perspective is emerging to become one of the fundamental building blocks in neurogastroenterology. It has gained prominence as it is a target of primary focus in neurology, psychiatry, endocrinology, and primary care, as it can mechanistically explain the interrelationship of GI symptoms, mood/cognition, and metabolic states. These all co-exist and all respond to similar interventions such as dietary manipulation, stress reduction techniques, and targeted pharmacotherapies. (Guo et al., 2025; Loh et al., 2024).

3.1. Molecular Architecture of the Gut–Brain Axis

A general architectural framework of microbial signaling in the gut–brain axis. The gut–brain axis is underpinned by a structured molecular design that allows microbial signals to sense, interpret, and propagate. As far from a mere abstract signaling system, the gut–brain communication via archetypes of permeability, receptor accessibility, immune sensing, and neural connectivity organization (Kasarello et al., 2023). It regulates the encoding of luminal microbial signals into host physiological responses and is regarded as the cellular machinery needed for stabilizing host–microbe interactions in a particular environment (Macpherson et al., 2023). Alternatively, thinking of the gut–brain axis as a system of architectures highlights the importance of physical and molecular interfaces that tune signaling prior to its systemic effects.
Fundamental to this infrastructure is the intestinal epithelial barrier, a permeability switch and signaling surface. Tight junctions (containing claudins, occludin, and scaffold proteins, e.g., zonula occludens) are essential for controlling paracellular permeability and supporting intracellular signaling networks for cytoskeletal organization and gene transcription (Gwak & Chang, 2021). Comprehensively, the junctional structures sense metabolic and immune signals in the gut lumen and permit epithelial modulation of their barrier function, and complex structural organization of the epithelial barrier that interdigitates with a variety of pattern-recognition receptors that monitor microbial-associated molecular patterns on the luminal side and transduce them to host cellular inputs which control immune tone and systemic homeostasis (Macpherson et al., 2023). To this end, the epithelium interface is not merely a barrier but acts rather as an active mucosal gate regulating environmental stimuli-driven immune sensing in concert with host homeostatic programs.

Underneath this epithelial boundary, a robust system of immune surveillance and tolerance permeates and dynamically reacts to the actions of microbes and allergens. It is divided into spatial niches, where dendritic cells, mucosal macrophages, innate lymphoid cells, and numerous regulatory lymphocytes cooperate to interrogate the integrity of the epithelial barrier and microbial detection (Macpherson et al., 2023). Rather than simply responding to antigenic perturbation, these immune cells offer continuous regulatory signaling that sustains tissue

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homeostasis. Local cytokine gradients are created within this network and are characterized by diverse effects on epithelial renewal, vascular leak, and cellular circulation, aiding in the maintenance of an intact barrier and also affording adaptation to and sensing of local risk factors. Where there is the architecture of a calibrated “checkpoint”, a microbe-sensing network: microbes communicate downwards, so that at the checkpoint, all systemic phenomena come together. This system is intended to prevent excessive inflammatory hyperactivation and allow local signaling to distal organs. Importantly, immune surveillance is embedded into tissue structure as opposed to an independent boundary to ensure host–microbe dynamic homeostasis under tight cellular confines.

The conjoinedness among neuroepithelia provides a second architectural facet that interlinks luminal sensing with neural integration, in the presence of tightly coordinated cellular configurations. A network of enteric neurons interacts with enteroendocrine cells and enteric glia to form local circuits in the gut wall. Enteroendocrine cells are chemosensory pathways that can sense a combination of chemical and mechanical stimuli in the lumen and rapidly convert this information into cellular responses with the ability to alter adjacent neural resources (Foster & Clarke, 2024). The spatial close-proximity in a location enhances signal conversion without system-wide diffusion and preserves fidelity and timing. Complementaries from enteric glial populations contribute to localised structural support, metabolic buffering, and local control, enhancing the resilience and stability of epithelial cells. These clustered assemblies develop microdomains that cohere in response to sensory perception, neuronal activation, and tissue regulation. Anatomical organization enables localized gut signaling modulation of environmental signals to provide isolated pathways (i.e., compartmentalization) while still integrated communication with the central nervous system.

In addition to this, hormone responsiveness by gut cell-level constructs is embedded in further organoid structure, with further localization of the structural organization through neuroendocrine coordination of local activity with whole-organism regulation. Targeted cells transcriptionally program the behavior of epithelial and immune cells in response to signaling from the hypothalamic–pituitary–adrenal axis, thereby adapting barrier stability and stress responses (Lee & Kim, 2021). Such endocrine embedding guarantees transient systemic physiological conditions that recentralize intestinal activity without the need to preserve tissue structure. Gut-derived signals can thus be involved in homeostasis at large, integrating peripheral sensing and the central system by means of bidirectional communication. As a consequence, the system would favor coordinated, not isolated, hormonal effects, and context-independence would not be an adaptive response to stress that also calls for concomitant immune protection (Lee & Kim, 2021). This structural coupling enables environmental and endogenous stressors to drive calibrated cellular adaptations that, in turn, promote stability across biological resolutions.

This epithelial interface, immune surveillance networks, neuroepithelial microdomains, and endocrine regulatory constructs therefore provide a coherent molecular framework for gut–brain interactions together. These layers have different structural roles, selective permeability,

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immunotuning, neuronal connections, and systemic economy, and they work together as balance-maintaining mechanisms in physiology. The brilliance of it, however, is the hierarchical cellular checks that insulate local perturbations so that small changes do not ripple out of control throughout the system, and, simultaneously, it is able to be sensitive to specific change. Structural levels of disruption may interfere with the readout and translation of microbial information, indicating that good gut–brain communication functions not just genetically but also at the cellular level. This structural perspective emphasizes that gut–brain communication occurs within coordinated regulatory networks rather than isolated signaling pathways.

3.2. Mechanistic Pathways

Microbial metabolisms are one of the primary molecular "languages" used by gut microbiota to communicate with the CNS, as exemplified in Figure 1. One of the most heavily researched classes of such mediators is short-chain fatty acids (SCFAs) such as acetate, propionate, and butyrate, which are generated by bacterial fermentation of dietary fibers. Rather than inert metabolic waste, these metabolites are themselves bioactive signaling molecules. At the cellular level, SCFAs are known to activate G-protein–coupled receptors (GPR41 and GPR43), inhibit histone deacetylases, and contribute to repression of proinflammatory transcription pathways such as NF-κB signaling. In this way, SCFAs mediate epithelial integrity, immune cell differentiation, and neuro-immune crosstalk, linking microbial metabolites directly to host cellular regulation (Ullah et al., 2023). Consequences of these changes also include neuroactive signaling, synaptic plasticity, and endocrine modulation, with effects on early development. Specific disease consequences exhibit unique details, but they converge on a common mechanistic motif: microbial metabolites as regulatory intermediates between diet–microbiota interactions and peripheral neural activity and systemic physiology. (Guo et al., 2022; Silva et al., 2020).

Neural signaling is a quick two-way mechanism of interaction between gut activity and its central neural pathways. According to recent neurobiological models, metabolites and microbiome-related immune-related molecules that regulate vagal afferent firing through receptor-mediated neurochemical signaling dynamically affect autonomic CNS regulation (Foster & Clarke, 2024). GI motility, secretion and permeability, conversely, entail brain-derived autonomic outflow, and this, in its turn, affects gut microbial stability and composition (Gwak & Chang, 2021). This feedback mechanism resembles a structured cell signaling circuit where peripheral immune signaling and neurotransmitter regulation converge in a central autonomic control domain mediating an adaptive response to stressors. Specifically, the tendency to consider vagal pathways as a viable treatment mode is paramount. Neural signaling can also be targeted to influence gut-brain communication, and provide a tool to regulate inflammation, microbial ecology and neuropsychiatric outcomes (Faraji et al., 2025; Guo et al., 2025), which underscores the plasticity of this communication on a molecular level. As it is observable in Figure 2, the vagal pathways play an essential role in this network of integrated signals.

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Immune signaling represents a key inter-kingdom translational nexus between gut ecology and CNS function. Loss of epithelial barrier function secondary to dysbiosis also results in enhanced microbial-associated molecular pattern signaling (e.g., endotoxin ligands that trigger toll-like receptor pathways and the subsequent downstream inflammatory cascade). These cascades drive cytokine induction of NF-κB signaling, immune cell maturation, and peripheral inflammation amplification. (Macpherson et al., 2023). Blood-borne immune factors may affect CNS homeostasis via blood–brain barrier interactions and the activation of glial cells, including microglia and astrocytes. Prolonged alteration of these cellular signaling pathways leads to the neuroinflammatory states, which have been associated with neurodegenerative or mood-related diseases. Therefore, immune communication in the gut–brain axis is a molecular channel through which microbial dynamics influence central neural function. (Guo et al., 2025; Loh et al., 2024).

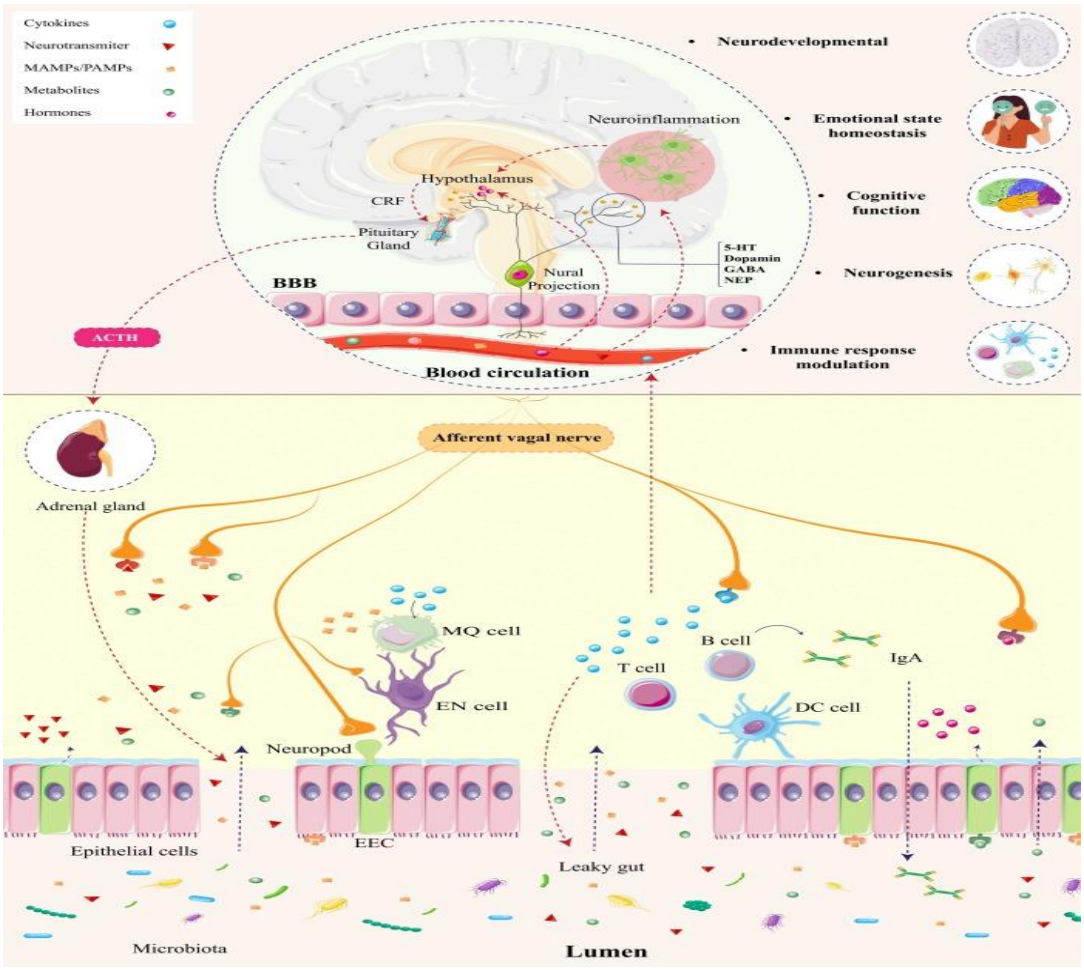

Figure 2: Role of the vagus nerve
Source*: (Faraji et al., 2025)*

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Additionally, the gut–brain axis interacts closely with neuroendocrine regulation, particularly through the hypothalamic–pituitary–adrenal (HPA) axis. Additionally, stress-induced glucocorticoid signaling negatively modulates gut permeability and immune response to microbial content through the transcriptional regulation of barrier and inflammatory genes. Conversely, microbial metabolites also affect the neuroendocrine feedback loops via immune and neurotransmitter signaling pathways, regulating sensitivity to stress and behavior (Lee & Kim, 2021). These connections give a mechanistic model for the relationship between psychological stress, GI dysregulation, and changes in mood. Stress-Related Gut Symptoms are not isolated Psychosomatic events, but molecularly integrated signaling across immune, endocrine, and neural systems (Bertollo et al., 2025).

3.3. Clinical Relevance

Emerging clinical data are supportive of the gut–brain axis as a common molecular platform for a variety of disease processes. 'Gut–brain interaction' in the development of IBS has been gaining popularity: visceral hypersensitivity, immune activation, and stress-related neuroendocrine signaling enhance symptom expression. Epidemiological studies reporting high rates of comorbidity between IBS and anxiety or depressive disorders are consistent with such models implicating cytokine signaling, neurotransmitter modulation, and metabolite-driven inflammatory processes that converge on both the gastrointestinal and affective pathways. (Hu et al., 2021). Such overlapping mechanisms of symptom clusters are consistent with dysregulation in communication between the gut and brain that leads to multisystem, rather than organ-specific, pathology.

In addition to functional GI disorders, gut-derived immune and metabolic signaling are involved in the progression of neurodegenerative disease. Disrupted metabolic activity of the microbiota and chronic inflammatory signaling affect glial biology and neuroinflammatory processes, which are gradually associated with diseases such as Parkinson's disease and other related neurodegenerative conditions. (Loh et al., 2024). A parallel could also be drawn with stress-related disorders that reflect a bidirectional pattern of molecular feedback where dysbiosis-induced inflammasome priming amplifies depressive signaling pathways, and reciprocal chronic HPA axis activation alters microbial community composition due to a series of pathological loops (Bertollo et al., 2025). Similar processes exist for inflammation-induced metabolic syndrome. In the setting of metabolic syndrome and type 2 diabetes, dysregulated microbial metabolite signaling and resulting immune activation perturb energy homeostasis, insulin sensitivity, and systemic inflammatory tone with downstream consequences for neurocognition. (Sasidharan Pillai et al., 2024). These diseases also provide an example of gut–brain signaling that may be integrating metabolic and neuroimmune within the cellular domain. Taken together, these findings further support the idea that gut–brain dysfunction stems from molecular dysregulation in shared physiological pathways. Understanding these pathways allows for a consistent interpretation of disease processes and provides the basis for novel, microbiome-directed therapeutic strategies.

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4. Educational Gap in Health Professional Training

Despite the explosion of gut–brain axis research, healthcare professional training remains predominantly designed around traditional organ-based models, which artificially divide physiological systems into discrete curricular silos. The historical value of this model could strengthen siloed clinical reasoning and ignore the complex biology that underlies many multisystem disorders, and even if organ-system curricula are adopted, the degree of integration may also vary and will need to be intentional instructional strategies rather than simply regrouping content (Xia et al., 2025; Zhang et al., 2025). These constraints are especially pertinent to the science of gut–brain, as an important integrative biological context that interconnects gastroenterological, neuroimmune, and neuroendocrine regulation. If microbiology, gastrointestinal physiology, and behavioral health are taught independently without an explicit blueprint for integration, learners might gain knowledge that is not integrated in a manner that works at the level of multisystem irritability patterns. The complexity science challenge to health professions education is that modern healthcare is informed by interconnected biological, behavioral, and environmental determinants and that training must mirror these connections in order to allow for the reasoning under uncertainty (Ogden et al., 2023). In the absence of such an integrated lens, gut–brain disorders may be construed narrowly through specialty-driven perspectives rather than viewed as biologically interlinked physiopathological phenomena that necessitate coordinated clinical reasoning.

4.1. Insufficient Microbiome Literacy

A similar challenge is one of uneven depth of microbiome education at different training levels. Although there has been a growth of knowledge on the relation between microbiota and health, teaching is frequently introductory or fragmented when compared to its clinical relevance. This is due to the possibility of learners having a variation in their ability to interpret microbiome research findings, apply mechanisms in clinical scenarios, or alternatively communicate emerging evidence to patients (Alamri & AlKhater, 2022; Davarci & Davarci, 2025). This illiteracy is not merely a problem with vocabulary; They prove that the trainees can evaluate the evidence critically, are aware of the limitations of translation, or can integrate the considerations of microbiomes into the larger context of diagnostic thinking. Public health education is beginning to react by proposing models that propose to place the upstream influences (e.g., the microbiome, diet, antibiotic exposures, and early life) and the downstream health side by side, in which case the frame of mind of a system is reinforced. (Melby et al., 2025). Nevertheless, due to the fact that most clinical training programs offer little time in practicing the ideas of microbiome in repeated scenarios involving real-life experience, involving patients facing a similar type of case, this form of education is missing or incomplete. Without introducing a form of reinforcement, there is a danger of the microbiome concepts being the conceptual level, and not the operationally defined concept within clinical practice, as opposed to just conceptual. Microbiome literacy must therefore be built through not only expanding exposure but also incorporating applied learning experiences with a bridge in both the biological process and clinical relevance.

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4.2. Clinical Consequences

There are also applied implications of the deficit of integrative and microbiome-aware education in clinical practice. For clinicians trained largely within siloed paradigms, the presentation of gastrointestinal symptoms with mood and sleep disruption, fatigue, or risk profiles of metabolic derangement has ceased to register as surprising; rather, it has become biologically integrated (Sahu et al., 2026). Disjoint training may encourage symptoms to become compartmentalized, delaying the recognition of shared regulatory roots and the opportunity for coordinated care/hierarchical approaches. In addition, lack of familiarity with microbiome-linked evidence may restrict the clinician in his/her capacity to frame lifestyle measures, pharmacologic effects, or stress-linked influences within more integrated physiological models. Crucially, the goal is not a broad mastery of microbiome science but rather basic literacy that allows clinicians to interpret new evidence judiciously and incorporate it into patient care. Educational literature in systems thinking suggests that those who develop an awareness of the relational nature of biological systems are more capable when dealing with complex, multi-factorial situations (Davarci & Davarci, 2025; Ogden et al., 2023). From this perspective, restricted curricular incorporation of gut–brain phenomena is a translational divide between emergent scientific knowledge and clinical reasoning in everyday practice

5. Proposed Curriculum Framework for Gut–Brain Axis Integration

Gut-brain axis (GBA) science should not be taught side by side but needs to be structurally integrated into health professions curricula to be taught effectively. Current medical education is shifting towards more competence-based and vertically integrated courses of medical education, which bridge the basics of science with clinical reasoning at various levels of training in the health care industry (Gruppen et al., 2012; Goodwin et al., 2024). GBA literacy, in this paradigm, must be integrated longitudinally into physiology, pathology, pharmacology, and clinical training, and integrated into assessment schemes that strengthen the interpretation of the mechanism. Figure 3 visually operationalizes the proposed framework by demonstrating how mechanistic science is translated into competency domains, longitudinal mapping, and assessment reinforcement, supported by institutional enablers. The bidirectional structure underscores that educational integration is iterative rather than linear.

5.1. Competency-Based Integration

Competency-based learning determines the learning outcomes as the visible skills that are based on the whole body of knowledge and professional judgment (Gruppen et al., 2012; Katoue & Schwinghammer, 2020). To prevent surface knowledge, the concept of microbiome literacy must be modeled by competencies that are measurable.

The skill of describing mechanistic pathways between microbial metabolites is also an element of knowledge competencies that ought to be applied to short-chain fatty acids (SCFAs) to epithelial barrier regulation, immune signaling, and neuroendocrine modulation (Guo et al., 2022; Macpherson et al., 2023). Dysbiosis can also contribute to inflammatory cascades, vagal

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signaling, and hypothalamic-pituitary-adrenal (HPA) axis interactions in the context of multisystem disease (Lee & Kim, 2021; Loh et al., 2024).

There should be the inclusion of the application of gut-brain mechanisms in the process of clinical reasoning during the demonstration of gastrointestinal, neuropsychiatric, and metabolic symptoms by a patient. Using an example of comorbid irritable bowel syndrome and anxiety, it is possible to analyze them in terms of relying on common inflammatory and neuroendocrine mechanisms (Hu et al., 2021). The skill of critical evaluation of microbiome research (understanding the difference between mechanistic plausibility and premature clinical generalization) should also be demonstrated by learners (Davarci & Davarci, 2025).

There is also a professional communication competency. Clinicians need to be capable of talking about microbiome-informed lifestyle or therapeutic interventions and clearly recognize levels of evidence and uncertainty. This prevents overinterpretation of the fast-growing research area. GBA integration will be organized and quantifiable, not the conceptual advocacy through expression of competencies in educational models that are definite.

5.2. Longitudinal and Horizontal Curriculum Mapping

Inclusivity in an enlightening curriculum has proved to lead to better knowledge acquisition and transfer because basic sciences are re-examined in more difficult clinical situations (Cook et al., 2013; Goodwin et al., 2024). Based on this, GBA science must be spread across subjects instead of being clustered in the microbiology courses. Microbial metabolite signaling (e.g., SCFA-GPR) may be added to autonomic and neuroimmune instruction in physiology and neuroscience (Guo et al., 2022; Yang et al., 2023). Dysbiosis can be contextualized in pathology as a cause of dysfunction of the barricades and the amplification of the inflammatory processes (Macpherson et al., 2023). In contrast to pharmacology, the new information about drug-microbiota interactions can be applied to the concept of therapeutic variability and precision medicine (Verdegaal & Goodman, 2024; Zhao et al., 2023).

The biological interconnections that the GBA research points to can be seen in horizontal mapping across disciplines, i.e., between gastroenterology, psychiatry, endocrinology, and primary care. The vertical mapping will guarantee reinforcement at both preclinical and clinical phases with adaptive proficiency in the multisystem thinking (Ogden et al., 2023). Notably, integration must be done by reorganization as opposed to increasing the curriculum to curb overload. The strategies of curriculum mapping can enable teachers to establish natural nodes among the current objectives (Matinho et al., 2022).

5.3. Applied Learning and Assessment Alignment

The assessment leaves a strong impact on learning behavior (Katoue & Schwinghammer, 2020). Thus, the integration of GBA needs to go further than content delivery to the evaluative frameworks that strengthen the mechanistic mode of reasoning. Case-based learning is a feasible

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method. For example, a patient who comes to the clinic exhibiting gastrointestinal conditions, mood swings, and metabolic risk factors can be examined regarding the paths of barrier regulation, inflammatory signals, and neuroendocrine adjustments (Lee & Kim, 2021). Simulation-based/problem-based education has been demonstrated to improve the system-level thinking and the use of fundamental science in complex clinical situations (Sistermans, 2020; Park et al., 2025). The assessment practices can also be structured in the form of short-answer questions that have to be explained, case tests that will test the integrated diagnostic reasoning, and OSCE questions that will test the communication of microbiome-informed intervention. These types of methods encourage mechanistic modeling inside as opposed to pattern identification, which fits into educational systems that recognize complexity (Ogden et al., 2023).

5.4. Faculty Development and Institutional Support

Successful implementation depends on interdisciplinary collaboration and faculty preparedness. Educational reform literature emphasizes that integration requires institutional structures supporting cross-disciplinary dialogue and shared curricular ownership (Gruppen et al., 2012; Morales, 2025). Educational programs about systems biology and translational interpretation of microbiome research have the potential to enhance the coherence of teaching and avoid falsification of new findings.

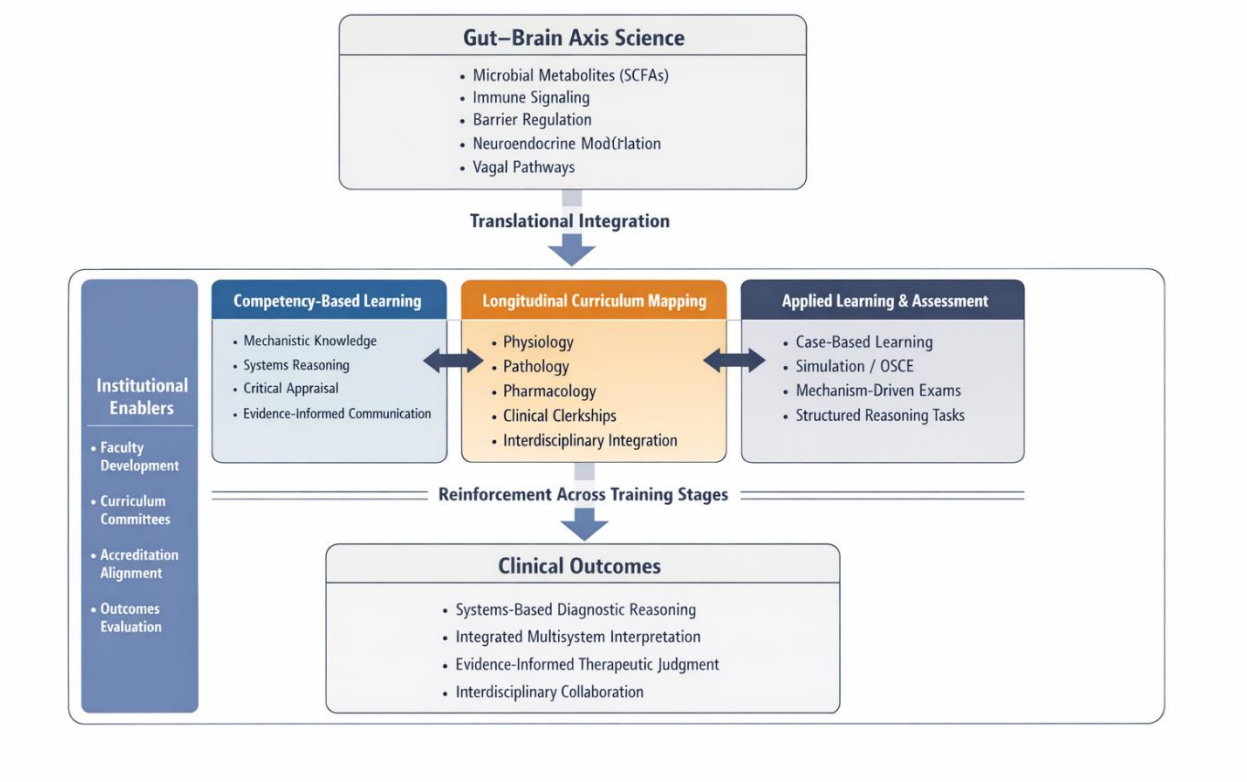

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Figure 3: Conceptual Framework for Integrating Gut-Brain Axis Science into Health Professions Education
Source: (*Constructed by Author*)

6. Curricular Integration Strategies

While Section 4 outlines the conceptual framework for integration, this section operationalizes that framework through specific curricular design strategies. Whether or not GBA science becomes integrated into clinical practice will depend upon the development of a curriculum that reflects knowledge about microbiome-host interactions as core medical knowledge rather than as an add-on to the practice of medicine. The highest level of health professions education today focuses on a model of deep integration, such that applied and clinical excellence, foundational science, and assessment are longitudinally aligned (i.e., a model in which the learner has opportunities to revisit basic principles in an ever more complex context) (Goodwin et al., 2024; Gruppen et al., 2012). The content of GBA should therefore be integrated into physiology/pathophysiology and pharmacology rather than segregated from microbiology in this context. Backed by educational literature that indicates that integration of basic science instruction improves conceptual transfer and clinical reasoning, this approach creates opportunities for learners to make thematic links between mechanistic biology and patient presentations repeatedly (Cook & Steinert, 2013; Goodwin et al, 2024). Integrating microbial metabolites, barrier function regulation, and neuroimmune signaling, along with neuroendocrine physiology, allows students to view gut–brain communication as a more routine regulatory mechanism rather than a curious evolutionary accident. In contrast, pathophysiology education can conceptually connect dysbiosis to inflammatory cascades, stress physiology, and multisystem disease expression, and facilitates a systems-based understanding of health care that underlies modern frameworks of biomedicine (Katrakazas et al., 2020). A further enhancement of microbiome integration into pharmacology instruction is a review of how it may affect metabolism and variability of drug therapies, a nascent field that is garnering more recognition as having clinical relevance (Verdegaal & Goodman, 2024; Zhao et al., 2023).

6.1. Case-Based Learning Modules

Case-based learning is a method of providing a structured space in which mechanistic biology can be connected to patient presentations. As an illustration, a learning module about a patient with irritable bowel syndrome, anxiety, and metabolic risk factors enables learners to investigate shared regulatory pathways that consist of epithelial barrier malfunction, inflammatory cytokine signalling, vagal modulation and hypothalamic-pituitary-adrenal (HPA) axis activation (Lee & Kim, 2021; Hu et al., 2021).

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Instructional design can encompass:

- A cursory mechanistic overview of microbial metabolite signaling (e.g., SCFA-GPR pathways) (Guo et al., 2022).
- Immune-neuroendocrine interaction mapping in small groups.
- Systematic review of a microbiome-targeted intervention study.

The strategy fosters the integration of gastroenterology, psychiatry, and endocrinology, as well as strengthening evidence-based interpretation.

6.2. Simulation and OSCE Integration

There is a chance to evaluate applied reasoning and communication through simulation-based education and Objective Structured Clinical Examinations (OSCEs) (Katoue & Schwinghammer, 2020). A scenario on GBA in a scenario of the OSCE might ask learners to describe biologically plausible connections between gut barrier integrity, inflammation, and mood symptoms with an appropriate qualification of evidentiary constraints. Such assessment emphasizes mechanistic coherence and clinical prudence to prevent overgeneralization during the counseling of patients. The integration of GBA scenarios in the current assessment frameworks supports the idea that microbiome literacy is not a marginal concept to the multisystem clinical reasoning framework.

6.3. Reinforcement Through Longitudinal Exposure

Training at different stages enhances retention and adaptive expertise as a result of repeated exposure (Goodwin et al., 2024; Ogden et al., 2023). The introduction of microbial signaling in early preclinical units can be reintroduced into the process of clerkship, utilizing either structured case reflection, interdisciplinary rounds, or integrative morbidity discussions. This vertical reinforcement helps in the transfer of the basic knowledge to the actual diagnostic decision-making. Such ground-level inclusion needs linking, however, to structured problem-solving if molecular tenets are to impact actual clinical practice. This problem-based learning makes it possible to translate cellular and regulatory systems into real-life diagnostic reasoning of multiple patients having a complicated clinical presentation, like presented by the brain-gut interactions (Sistermans, 2020). Clinical observations of gut disturbances, metabolic dysregulation, or stress-related symptom exacerbations (all similarly described in some patients) also indicate that there is reason for precisely defining skin corticosterone formation beyond the rheumatology-immunology-endocrinology self-subsided silos. The association of an increase in cognitive flexibility and a complex pattern of multisystem disorders (Rezaei & Saghazadeh, 2022) indicates a collaborative thinking type that is necessary for managing interactions between the fields like gastroenterology, psychiatry, nutrition science, and pharmacology. Simulation-based education advances this translation further by placing the learner within the systems where physiology interfaces with psychosocial motivators and subsequent decision support. Experiential engagement has repeatedly been shown to enhance knowledge retention and application of fundamental science as well as help students identify the ways in which principles learned at the molecular level affect clinical decision-making (Park et al., 2025).

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Sustainable curricular improvement hinges on assessment that is tied to learning goals and values, molecular interpretation of biology, and integration across areas. Competency-based medical education frameworks demonstrate that examinations drive how learners interact with basic science; therefore, examinations should assess the capacity to understand mechanistic relationships among microbial activity, barrier regulation, immune signals, and neuroendocrine control, leading to a clinical profile. (Chappell et al., 1999; Katoue & Schwinghammer, 2020). OSCE-style assessments can be constructed to determine whether learners apply molecular information in a clinically sound manner to diagnosis, such as recognition of how microbial-derived inflammatory signaling or microbiome-related variation manifests systemically.

Case reports and structured reasoning exercises allow students to be assessed on the degree to which they apply cellular and regulatory principles to the analysis of complex, multisystem applications. This evaluation will be critical to converting emerging microbiome evidence into actionable guidance for health and practice by ensuring that focus remains on biological plausibility and mechanistic coherence rather than simple pattern recognition in complex multiomic datasets. Reinforcement of molecular reasoning leads students to build internal models of an analytic framework, which correlate positively with modern systems biology. Operationalizing microbiome literacy as a measurable clinical competency that promotes accommodating thinking through strengthening gut–brain concepts in an assessment framework. In this light, assessment is not simply an assessment of recall but an impetus to establish deep molecular reasoning that informs patient-specific decision-making. (Katoue & Schwinghammer, 2020).

7. Implications For Clinical Practice

The gut-brain axis literacy transmutation into clinical learning is particularly significant regarding patient care, as it displays a modification of the previously altered paradigm through which clinicians had previously been interpreting multisystem disease through the lens of a molecular paradigm. This leads to elevating an etiological diagnostic model based on biologic integration away from a purely organ-segregative model when one accepts that the gut, CNS, and immune system, and their metabolic dysfunctions, could emanate from common cellular signaling and regulatory channels. For professionals, if patterns in symptoms can be viewed in the perspectives of barrier regulation, immune modulation and neuroendocrine feedback, differential diagnosis seems a lot more rational and physiologic. This mechanistic approach then adds complexity to the clinician–patient interaction by treating diet, stress physiology, sleep regulation and lifestyle habits as agents in the modulation of cellular pathways and not as an ‘add-on’ type of wellness recommendation. And knowing how these elements impact the host-microbe encounter and the modulation of inflammation may aid in patient compliance by allowing behavior modification to be linked to measurable biological changes. Likewise, molecular awareness informs treatment decisions about treatment, prompting the clinician to assess novel microbiome-targeting therapies for efficacy, balancing mechanistic plausibility with net biological effects. The ideas also lend themselves to preventive approaches, where early metabolic or lifestyle modifications can be considered as interventions that reset control circuits,

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not just treat symptoms. When the gut–brain connection comes to life within standard clinical reasoning, then systems of care should be framed in a way that is based on recent molecular biology and in a manner that allows for more coherent and integrative models of care based on the integrated nature of human physiology.

8. Challenges and Considerations in Curricular Integration

Although the concept of the translational relevance of the gut-brain axis (GBA) gains more and more prominence among researchers, there are a number of aspects that responsible curricular integration should be based on. To begin with, the basis of evidence is heterogeneous. A major part of the mechanistic literature is based on preclinical models, associative human research, or small-scale trials, which make it difficult to infer causal inferences and extrapolate to large populations (Guo et al., 2025; Loh et al., 2024). Even though microbial metabolites like short-chain fatty acids have shown biologically plausible mechanisms between immunity and neural regulation (Guo et al., 2022; Yang et al., 2023), clinical effects are varied in magnitude and reproducibility. Translating curriculum should then be focused on critical evaluation instead of prescriptive use.

Second, premature clinical extrapolation may develop. The implications of the rapid expansion of microbiome-related findings, especially in fields like psychobiotics or individualized nutrition, can cause a situation where the recommendations go beyond what the evidence has shown (Davarci & Davarci, 2025; Verdegaal & Goodman, 2024). Builds of mechanistic plausibility and established therapeutic efficacy must be well differentiated in educational frames.

Third, there is a structural obstacle in terms of curriculum density. The health professions programs are already overloaded in content, and additive methods could contribute to the overload. This should be integrated via curricular reorganization and mapping and not expansion, integrating the GBA material with the already established goals in the physiology, pathology, pharmacology, and behavioral sciences (Matinho et al., 2022; Xia et al., 2025).

Fourth, there must be research relating to educational outcomes. Even though the integration of models and the emphasis on capabilities imply enhanced knowledge transfer (Gruppen et al., 2012; Goodwin et al., 2024), the particular assessment of microbiome-informed curricula is scanty. These should then be implemented by systematic evaluation of learner reason, systems thinking, and evidence-based communication.

Lastly, there should be faculty development. Integrative reform implies interdisciplinary cooperation and mutual conceptualizations (Morales, 2025). In the absence of the fluency of the faculty in systems biology and translational interpretation, the coherence of the curriculum can be undermined. By treating these issues, GBA integration becomes more robust in supporting clinical reasoning and retains the scientific viability and pedagogical capability of education.

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9. Conclusion

Advances in gut–brain axis research increasingly demonstrate that microbial metabolites, immune signaling, epithelial barrier regulation, autonomic pathways, and neuroendocrine feedback operate within an integrated physiological network. These mechanisms provide biologically coherent explanations for multisystem presentations spanning gastrointestinal, neuropsychiatric, and metabolic disorders. However, current health professions curricula remain largely compartmentalized, limiting learners' ability to apply systems-based reasoning to complex clinical cases. This review suggests that the incorporation of gut-brain literacy should be done in structured curricular mapping, competency-based learning goals, and an assessment framework whose artifacts and strategies strengthen mechanistic interpretation as opposed to the acquisition of individual facts. The provision of mechanisms related to microbiomes should not be some additive information, but must be aligned with existing physiology, pathology, pharmacology, and behavioral medicine modules by curriculum committees. Systems-based and translational reasoning must be considered educational standards that can be measured by accreditation bodies. Critical evaluation of microbiome evidence needs to be promoted among educators to avoid premature clinical extrapolation, but also interdisciplinary clinical reasoning.

As applied in proportional degrees to the evidencing maturity in perspective and incorporated in established competence considerations, gut-brain integration is an evolution of curricular organization, not expansion. An integration of education to correspond to modern systems biology enhances the diagnostic coherence, interchange of abilities, and application of evidence-based patient care in a time of more sophisticated chronic disease. Curriculum committees should formally map these competencies within existing modules, accreditation bodies should recognize systems-based translational reasoning as measurable standards, and faculty development programs should support interdisciplinary implementation.

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