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Welcome to the HiPP Symposium

Disorders of the gut-brain-interaction: News in diagnostic understanding and nutritional therapy approaches

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Finally, the Rome V criteria are out; What is new?

The Rome IV criteria played a major role in advancing the understanding and research of pediatric disorders of gut-brain interaction (DGBI). However, as more studies emerged, it became clear that classifying these disorders by age did not accurately reflect how they present and evolve across childhood. In response, the Rome V pediatric committees shifted to an anatomy-based classification system, dividing DGBI into upper and lower gastrointestinal (GI) disorders while still considering developmental differences within each diagnosis.

The history of this framework dates back to 1999 with the Rome II criteria, which defined pediatric "functional GI disorders" and sparked research into conditions like recurrent abdominal pain. Over time, Rome III (2006) and Rome IV (2016) refined these definitions, leading to wider adoption in both research and clinical practice. This growing body of work demonstrated that DGBI are common in children and can significantly affect quality of life, with notable prevalence rates for conditions such as constipation and abdominal pain—related disorders.

Rome V introduced several important updates. For upper GI disorders, Rome V introduced largely new diagnostic categories. These include esophageal pain disorders (such as reflux hypersensitivity), functional feeding disorders, and disorders of air transit like supragastric belching. Some conditions, like infant regurgitation, were removed after being reclassified as normal physiological processes rather than disorders.

For lower GI disorders, conditions are now grouped into three categories: abdominal pain disorders (such as irritable bowel syndrome, abdominal migraine, and centrally mediated abdominal pain syndrome), defecation and anorectal disorders (like functional constipation and fecal incontinence), and other conditions including functional abdominal bloating and infant distress syndrome (formerly infant colic). The classification of abdominal pain disorders was further refined based on symptom patterns—intermittent, episodic, or continuous—to better reflect clinical reality.

The committees also addressed how pain is assessed in children, emphasizing that pain is subjective and best measured by self-report. Because young children cannot reliably describe pain intensity, Rome V recommends that chronic abdominal pain—related DGBI should not be formally diagnosed before age four.

Overall, Rome V significantly expands and clarifies the classification of pediatric DGBI. By moving away from age-based categories and adopting a more anatomically and clinically driven system, it provides a clearer framework for diagnosis, research, and treatment, ultimately aiming to improve care and outcomes for affected children.

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Psychogastroenterology — the biopsychosocial model and how we can implement it in clinical practice

Psychogastroenterology is a field that sits at the intersection of gastroenterology, psychology, and neuroscience and is grounded in the recognition that disorders of gut—brain interaction (DGBIs) emerge from dynamic interactions among biological, psychological, and social/environmental processes.[1] Although the biopsychosocial framework has long been acknowledged within the field, its translation into day-to-day clinical practice remains inconsistent. In this presentation, I will introduce our recently published psychobiological model of DGBIs,[2] a novel, integrated, and testable framework designed to bridge the longstanding translational gap between basic mechanistic science and clinical research and care.

The psychobiological model expands upon the traditional biopsychosocial model1 by 1) explicitly identifying key cognitive, affective, and behavioral mechanisms relevant to DGBIs and 2) proposing hypothesized pathways for how they interact with each other and with peripheral physiology to influence symptom onset, persistence, and recovery. The model integrates conceptual advances from decades of research in psychogastroenterology with insights from related fields, including chronic pain, panic disorder, and anxiety. The model proposes two interacting pathways: a cyclical risk pathway, in which threat appraisal, fear, symptom-focused attention, avoidance, and emotional distress amplify gastrointestinal symptoms and functional impairment; and a recovery pathway, in which adaptive cognitive and behavioral responses facilitate symptom coping, resilience, and return to functioning and valued living. Importantly, these pathways are embedded within the bidirectional gut—brain axis and interact continuously with peripheral biological factors such as motility disturbances, immune activation, and microbiome-related signaling. These bidirectional interactions occur, in part, through activation of the stress response and immune systems, which provide important biological pathways through which psychological processes can influence gastrointestinal function and symptom perception.

A key objective of this session is to demonstrate how this model can move beyond theory and directly inform clinical assessment and intervention. Rather than conceptualizing psychological factors as secondary or merely comorbid features, the psychobiological model positions them as mechanistic processes that can serve as clinically actionable treatment targets. Specific examples include threat appraisal, fear, interoceptive threat monitoring, avoidance behaviors, and maladaptive beliefs about gastrointestinal sensations. These processes can perpetuate symptom burden even in the context of minimal or resolved structural abnormalities and may help explain discrepancies between physiologic findings and symptom severity frequently encountered in gastroenterology practice.

The presentation will focus on practical implementation strategies for clinicians working with patients with DGBIs and related symptom-based gastrointestinal conditions. First, I will discuss how the model can be used to guide qualitative and quantitative assessment, including the use of brief screening questions and validated patient-reported outcome measures to identify psychological processes, such as fear, avoidance, and symptom-related distress, as mechanistic targets during routine visits. Second, I will highlight how this framework facilitates communication with patients by providing a non-stigmatizing, biologically grounded explanation of how psychological and physiological processes interact to shape symptom experience. Third, I will review how these mechanisms can inform personalized treatment planning, including when to integrate brain-gut behavioral therapies such as gastrointestinal-focused cognitive behavioral therapy, gut-directed hypnotherapy, mindfulness-based stress reduction, acceptance-based approaches, and multidisciplinary care models.[3]
Special attention will be given to strategies for discussing these concepts in a way that reduces stigma and improves patient engagement. By normalizing processes such as increased attention to bodily sensations as understandable protective responses that can become self-reinforcing over time, clinicians can foster therapeutic alliance and enhance receptivity to behavioral treatment recommendations. The model also offers a common language for interdisciplinary collaboration among gastroenterologists, GI psychologists, dietitians, and other specialists.

Finally, this session will emphasize how the psychobiological model provides a roadmap for the future of psychogastroenterology by linking mechanistic research with precision clinical care. Through a clearer understanding of *which* psychological mechanisms are most relevant for *which* patients, the field can move toward more targeted, mechanism-informed interventions and improved patient outcomes. This framework has the potential to improve both how we conceptualize DGBIs and how we implement the biopsychosocial model in everyday gastroenterology practice.

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Update on promoting gut health and gastrointestinal tolerance in infant nutrition

Gut health plays an important role in the healthy development of infants and is influenced by various factors, including mode of birth and feeding mode. Vaginal birth and breastfeeding are two factors promoting optimal gut health development in infants. Different parameters such as the composition and the age appropriate maturation of the microbiota, the synthesis of metabolites like short chain fatty acids (SCFA) and the development of the gut associated immune system, can provide insights into the gut health development of infants. However, impaired development of these parameters such as dysbiosis of the microbiota may contribute to the development of disorders of gut brain interactions (DGBIs) previously referred to as functional gastrointestinal disorders (FGIDs).[1] DGBIs include constipation and infant colic, the latter mainly characterized by excessive and inconsolable crying, which represents a considerable burden for the families.

Human milk contains numerous bioactive components such as prebiotics and probiotics that beneficially act on gut health development in infants. In non-breastfed infants or when exclusive breastfeeding is not possible, supplementing infant formula (IF) with prebiotics and probiotics can help to promote gut health. A randomized, controlled intervention study with the administration of a synbiotic IF (*Limosilactobacillus fermentum* CECT5716 (*L. fermentum*) and galactooligosaccharides (GOS)) was conducted. Infants were fed up to 12 months of age and stool samples were collected at 4 and 12 months of age. Stool analysis revealed that the synbiotic IF group showed a lower stool pH and higher acetate concentrations at 4 months of age and a significantly higher concentration of secretory Immunoglobulin A at 12 months of age compared to infants fed a standard formula.[2]

The type of protein source may play an additional role in modulating gut health. Goats milk casein protein composition is closer to human milk compared to cow's milk protein. In an ex vivo gastrointestinal simulation model, donor microbiota from six term infants born by caesarean section was selected. Caesarean delivery is described to be more often associated with microbiota dysbiosis, which is one of the intensively discussed underlying causes of DGBIs.[3] A formula based on goat's milk protein in combination with GOS led to increased formation of total SCFA and reduced gas formation compared to goats' milk-based formula without GOS (internal data, publication in preparation).

To address colic symptoms in non-breastfed infants in clinical practice, changes can be made to the formula formulation, either individually or in combination. The adjustments can include the addition of synbiotics (e.g. GOS and *L. fermentum*), a reduced lactose content, hydrolyzed protein and structurally adapted fat, namely β-palmitate. The feeding of such a Comfort formula, including the just mentioned adaptations, was investigated in a randomized controlled study for 28 days in infants with colic as defined by modified Wessel criteria. Feeding the Comfort formula resulted in 26 minutes less crying time in infants with colic after 28 days compared to feeding a standard formula without the described adaptations. While stool frequency remained unaffected, significantly softer stools were observed in the Comfort formula group.[4] Due to the high prevalence of constipation in infancy and early childhood, softer stools may be beneficial as constipation is less likely to occur.[5]

In summary, these data show that various specific formulation adjustments of IF could have a positive impact on gut health and gastrointestinal tolerance in non-breastfed infants. However, further research and clinical trials for innovative IF are needed to investigate the optimal development of healthy gut function in non-breastfed infants.

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