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GUEST EDITORIAL:

The Gut–Brain Axis in Consultation–Liaison Psychiatry: A Biopsychosocial Framework for Assessment, Formulation and Management.



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ABSTRACT

The gut-brain axis is an important conceptual framework for understanding the bidirectional relationship between physical and mental health. Consultation-Liaison Psychiatry is particularly well-positioned to integrate emerging evidence into routine clinical practice. This narrative synthesises evidence from liaison psychiatry, psychosomatic medicine, and the literature on disorders of gut–brain interaction (DGBI). A practical biopsychosocial (BPS) framework for assessment, formulation, management and training is proposed. Psychological distress is widespread across medical specialties, while disorders of gut-brain interaction have important implications for healthcare services. Using a structured biopsychosocial assessment and identifying relevant gut-brain mechanisms strengthens clinical formulation, improves patient understanding and promotes effective multidisciplinary care.

KEYWORDS

Brain-Gut Axis; Gastrointestinal Microbiome; Mental Health; Models, Biopsychosocial; Psychosomatic Medicine; Psychological Distress; Referral and Consultation.

INTRODUCTION

The principle that there is no physical health without mental health underpins assessment in the general hospital.[1] Physical illness frequently coexists with psychological distress, yet recognition and treatment remain suboptimal. Low detection rates of depression, anxiety and somatisation in medical settings contribute to poorer outcomes and increased healthcare utilisation with significant economic costs.[2] Long-term conditions (LTCs) and multiple chronic conditions (MCCs) affect approximately 37% of people, globally.[3] They represent a significant burden, with individuals living with LTCs and MCCs experiencing greater impairment, poorer quality of life, and higher rates of hospitalisation and mortality.[3] A systematic review reported substantial variation across studies, but the pooled prevalence of multi-morbidity in adults was 42.4% (95% CI 38.9%–46.0%). No meaningful differences were observed between high-income and low- and middle-income countries (LMICs), although regional patterns varied. African studies reported the lowest prevalence, while North American studies reported the highest.[4]

Engel postulated an aetiology grounded in the interplay of biological, psychological, and social elements[5]. However, the biopsychosocial model has at times been reduced to a simple checklist, with clinicians listing factors without explaining how they interact. This occurred partly because biological

systems were easier to study, leading to limited understanding of psychosocial relationships and encouraging reductionist approaches. Consequently, some have dismissed the model as ambiguous or relevant only to psychiatry, failing to appreciate that Engel aimed to revolutionise medicine through a unified causal framework. Its real strength lies in demonstrating how these domains influence one another[6], with spirituality recognised as an integral component.[7] The next step is to develop mechanistic research that transforms this interface into a testable causal model of disease.

The gut–brain axis provides a biological foundation for understanding how psychological processes influence gastrointestinal symptoms and vice versa. Both bacterial and non-bacterial components of the gut microbiome contribute to suicide risk and major mental disorders through inflammatory pathways, although important mechanisms remain unclear.[8] Preclinical and emerging evidence suggests that disruptions in microbial balance may shift the central nervous system from a non-inflammatory to an inflammatory state by increasing the influx of pro-inflammatory cytokines into the brain.[9]

PSYCHIATRIC MORBIDITY IN PHYSICAL ILLNESS

Clinicians frequently identify distress through behavioural cues such as tearfulness, suicidal ideation, poor adherence, or idiosyncratic symptom presentations.[9] Endicott's criteria for depression in medically ill patients remain clinically relevant.[10] It captures symptoms such as fearful or depressed mood, social withdrawal, psychomotor retardation, and thoughts of self-harm or death. It is also useful to consider screening tools recommended by the National Institute for Health and Care Excellence (NICE),[11] including PHQ-9 and HADS, whilst DSM-5[12] or ICD-11[13] criteria remain the gold standard.

People with severe symptoms of common mental disorder (CMD) are significantly more likely to have a chronic physical condition than those with minimal symptoms. This pattern was consistent across specific illnesses, with severe CMD linked to double the rate of asthma. Chronic physical conditions were also associated with lower overall mental wellbeing.[14,15]

Somatic symptom and related disorders (SSRDs) often present as complex, intertwined experiences where physical symptoms and emotional distress blend together.[12] People may present with illness anxiety, persistent somatic symptoms, functional neurological disorder (FND), or other patterns where their symptoms feel very real but cannot be fully explained by medical tests. These conditions can lead to repeated investigations. A biopsychosocial approach helps clinicians make sense of this complexity, supporting clearer assessment, better communication, and more effective care.

APPLICATION OF THE BIOPSYCHOSOCIAL MODEL

The biopsychosocial (BPS) model encourages a holistic understanding of illness that considers the patient as a whole person, including their personality, emotional resources and social circumstances. Engel's work on conditions such as ulcerative colitis, depression and psychogenic pain challenged the traditional biomedical model by showing that illness cannot be reduced to pathology within isolated body systems. Instead, the experience of illness emerges from the complex interaction of biological, psychological and social factors.[16] Routine BPS assessment focusing on symptom-specific anxiety, cognitions, behaviours, stress, sleep, and psychiatric comorbidity enhances formulation and outcomes.

Embedding this formulation into everyday clinical encounters supports more accurate communication. The aetiology based on the BPS model (Table 1) can directly inform a stratified management plan (Table 2). This may be structured collaboratively into short-, medium- and long-term goals. This approach can guide referral to multidisciplinary care and may improve both patient outcomes and satisfaction.

THE GUT–BRAIN AXIS: BIOLOGICAL, PSYCHOLOGICAL AND SOCIAL PATHWAYS

The biological pathways that support the gut–brain axis illustrate the continuous, bidirectional interaction between gut physiology and central neural processes. Signals affecting mood, digestion, and inflammation travel directly via the vagus nerve. The hypothalamic–pituitary–adrenal (HPA) axis connects stress responses to gut motility through hormonal regulation, while the neuro-immune system communicates via cytokine signalling, clarifying how inflammatory activity can shape emotional states. In addition, microbiome-derived metabolites act as chemical messengers that influence cognition and emotions.[17]

Symptom perception and reporting in DGBI are shaped by psychosocial influences acting through central executive, emotional arousal, and autonomic networks that modulate visceral signalling. These processes interact with biological and psychosocial risk factors, including trauma, family responses, and psychiatric or physical comorbidity, all of which affect quality of life and treatment outcomes.

Irritable Bowel Syndrome (IBS) arises from dysregulation of the central and enteric nervous systems, with symptoms reflecting dysmotility and visceral hypersensitivity. Psychosocial processes modify symptom expression and illness behaviour. IBS exemplifies a non-linear, multilevel model of illness.[18] People with IBS experience repeated episodes of abdominal pain, changes in bowel habits, while medical tests might show no structural or inflammatory evidence. Although IBS has also been described as a “functional” disorder, it is now known to involve changes in several body systems, including the nerves, immune system, hormones, and the gut microbiome. Even when routine tests may look normal, IBS can significantly impact daily life, comfort, confidence, and wellbeing. A personalised BPS formulation using the 4-P model of predisposing, precipitating, perpetuating, protective and iatrogenic assessment is indicated.

GUT-MIND AXIS AND CHILDREN

Three features distinguish the paediatric picture of DGBI from the adult one:

- **Early life is a sensitive window:** Mode of delivery, feeding, antibiotic exposure and early nutrition shape microbial colonisation during the same period in which the brain’s stress-response and immune systems are maturing.
- **Adversity acts through the same channels:** Early-life stress alters microbiota composition and HPA-axis reactivity in preclinical models, and trauma-focused reviews describe convergence of dysbiosis, permeability and neuroinflammation in trauma-related presentations. A child with recurrent abdominal pain, poor school attendance and a history of adversity should prompt a trauma-informed assessment rather than another round of investigation.
- **The family is part of the mechanism:** Outcomes in paediatric functional abdominal pain are strongly influenced by how the child and the family respond to pain; parental threat appraisal and child pain catastrophising predict treatment response, in some analyses more than anxiety itself. Formulation must therefore include the family’s model of the illness.

In childhood, gut-brain axis composition undergoes rapid maturation, reaching an adult-like profile by 12-36 months, decisively shaped by delivery mode, feeding practices, antibiotic exposure, diet and environment. Vaginal delivery promotes vertical transmission of maternal Bifidobacterium species, while caesarean section enriches pro-inflammatory Klebsiella and Enterococcus and is associated with immune dysregulation. Human milk oligosaccharides foster a Bifidobacteria-dominant microbiome, whereas formula-fed infants remain microbiologically distinct even with probiotic supplementation. This early window is fragile: children are uniquely vulnerable to antibiotic-induced dysbiosis, which can produce a persistent reduction in diversity and a pro-inflammatory state linked to allergies, obesity and neurodevelopmental disorders.[19]

Evidence increasingly links early dysbiosis to paediatric neuropsychiatric vulnerability. A 20-year cohort of 16,440 Swedish children found that disturbed gut flora in the first years of life, influenced by antibiotic treatment, parental smoking and lack of breastfeeding, was associated with later diagnoses of autism spectrum disorder (ASD), Attention-deficit/hyperactivity disorder (ADHD) and speech disorders.[20] Recent meta-analyses confirm that childhood antibiotic exposure increases risk for ASD (OR 1.13) and ADHD (OR 1.18), while umbrella reviews document reduced alpha-diversity and altered abundance of specific taxa affecting cognitive functioning in young children.[21]

In adolescence, this axis takes on new importance. The transition to complementary feeding, urbanisation, and diet diversity already modulate maturation, but adolescence brings HPA axis stress sensitisation, diet autonomy, and a second wave of myelination and pruning where microbial metabolites continue to shape neurotransmission (serotonin, dopamine, GABA). The paediatric microbiota-gut-brain axis therefore represents both a critical window of vulnerability and an actionable window for precision prevention. Moving from association to longitudinal cohorts and targeted interventions should be the next priority.

PRIMARY CARE APPROACH TO GUT-BRAIN AXIS

Most people with DGBI and with common mental disorder are seen in primary care, and most never reach a liaison service. Given that more than 40% of adults internationally meet criteria for at least one DGBI,[22] primary care is where the gut–brain axis is actually managed, and where Consultation Liaison (CL) Psychiatry can have most leverage through consultation, education and shared pathways rather than direct assessment alone.

British Society of Gastroenterology guidance supports a positive diagnosis of IBS based on symptom pattern with limited investigation, rather than a diagnosis of exclusion reached after extensive negative testing.[23] This matters psychologically as well as economically: repeated normal investigations reinforce the belief that something has been missed, and each cycle increases both anxiety and iatrogenic risk. The general practitioner is well placed to combine a positive diagnosis with a brief enquiry into mood, anxiety, sleep, alcohol and adversity. Referral to liaison psychiatry should be considered when there is diagnostic uncertainty, comorbid severe mental illness, complex trauma or non-response to first- and second-line treatments.

First-line management includes dietary advice, soluble fibre, antispasmodics and, where appropriate, dietitian-led dietary modification.[23] For persistent symptoms, the ATLANTIS trial showed that titrated low-dose amitriptyline significantly improved IBS Symptom Severity Score at six months compared with placebo.[24] Gut–brain neuromodulators at low dose are not being prescribed here as antidepressants, and explaining that distinction carefully is often the difference between a patient accepting or declining treatment.

Brain–gut behavioural therapies, including cognitive behavioural therapy and gut-directed hypnotherapy, are recommended for patients whose symptoms do not respond to first-line measures.[23] Access remains the principal constraint, and this is an area where liaison and primary care mental health services can reasonably be asked to collaborate.

CASE FORMULATION AND MANAGEMENT

Figure 1 shows how the mechanisms described above enter the formulation itself. Biological, psychological and social domains act through identifiable gut–brain mechanisms to produce clinical expression. The resulting formulation drives a stratified, multidisciplinary plan. It is important to identify symptoms, investigations and health-care experience.

Figure 1

Biopsychosocial Formulation Incorporating Gut–Brain Mechanisms.

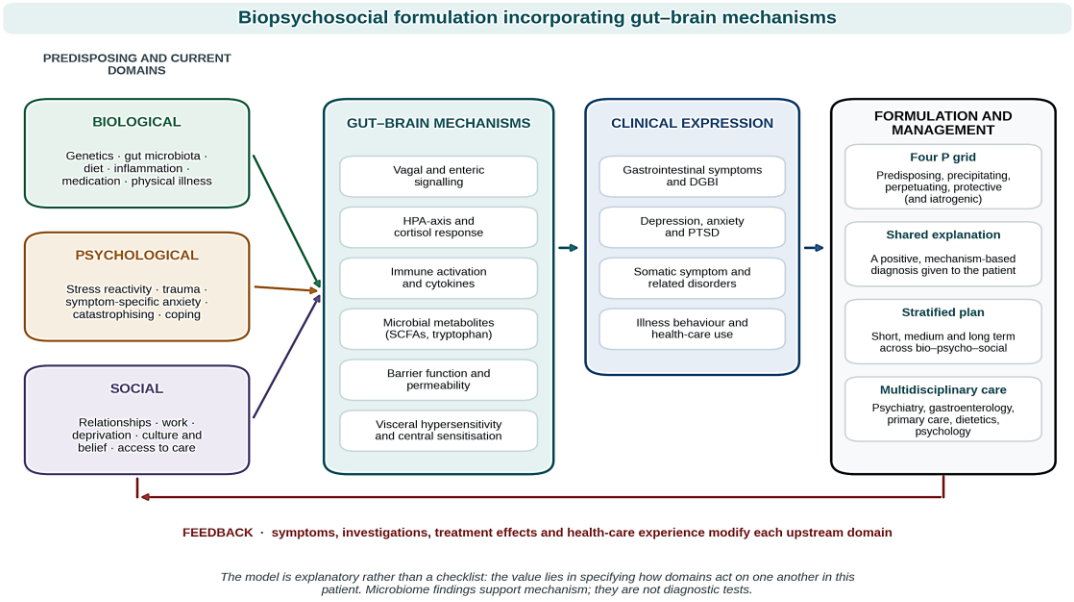

Table 1 describes an example of a biopsychosocial aetiological hypothetical formulation for a 40-year-old man referred from gastroenterology with low mood, in whom thorough assessment confirmed post-traumatic stress disorder (PTSD) missed during years of gastroenterological follow-up.

Table 1

Example of a biopsychosocial aetiological grid: a 40-year-old man with ulcerative colitis, referred with

low mood, in whom PTSD was diagnosed.

Phase	Biological	Psychological	Social
Predisposing	Ulcerative colitis diagnosed at age 16	Stress and inability to express emotions; previous self-harm; nightmares; fluctuating mood	Emotional and physical abuse in childhood
Precipitating	Response to treatment and adverse effects of medication	Poor concentration; ongoing thoughts of self-harm	Lack of support; conflicting spiritual advice
Perpetuating	Ongoing inflammatory burden and symptom unpredictability	Dissociative episodes; suicidal ideation	Conflicting spiritual advice; impact on relationships
Protective	Identifiable improvement with medication; periods of improved physical health	Learning from therapeutic intervention; ability to identify improved mood and relapse signatures	Job flexibility; employer understanding with occupational health support; improved family relationships; financial stability
Iatrogenic	Steroids; awaiting surgical intervention	Secondary demoralisation; lack of consistent care	Disruption through repeated hospital admissions

Table 2 shows how this formulation informs a management plan structured into short-, medium- and long-term goals, developed collaboratively with the patient and their network. The formulation distinguishes a coherent aetiological understanding from a mere checklist. For example, childhood abuse, early-onset inflammatory bowel disease, and alexithymia form a single interconnected pattern. Adversity shapes HPA-axis reactivity**,** inflammation influences mood**,** and alexithymia alters how symptoms are communicated.

Table 2
Illustrative Biopsychosocial Management Grid, Agreed Collaboratively with the Patient and Their Wider Network.

Time horizon	Biological	Psychological	Social & Spiritual
Short term	Medication optimisation; review of steroid burden; sleep and symptom relief	Establishing trust; safety planning; psychoeducation about the gut–brain axis	Engaging the social network; occupational health contact. Seeking advice from relevant hospital Chaplain.

Medium term	Medication review; dietetic input; consider gut–brain neuromodulator	Trauma-focused CBT or cognitive analytic therapy	Partner involvement; workplace adjustments Attending a place of worship
Long term	Joint MDT working with gastroenterology; agreed re-investigation thresholds	Acceptance-based therapy; relapse-signature work	Addressing work and family issues; sustaining financial stability

FUTURE DIRECTIONS

Liaison psychiatrists work at the interface of physical and mental health, with patients whose presentations reflect interactions between gastrointestinal, immunological, psychological and social processes. Despite DGBI high prevalence and clinical burden, medical education at both undergraduate and postgraduate levels gives it limited attention.[25]

An important objective is to help trainees move beyond traditional mind-body dualism. DGBI such as IBS and persistent physical gastrointestinal symptoms challenge any clear distinction between purely physical and mental illnesses.[26] Training should emphasise that symptoms are real experiences produced through biological, psychological and social interactions.

Communication and psychosocial assessment skills deserve specific attention within training programmes. DGBI frequently carry stigma, and this can be associated with treatment non-adherence, more clinical symptoms, reduced self-esteem and self-efficacy.[27] Therefore, breaking stigma is vital to the doctor-patient relationship and the outcomes of care. Communication skills teaching should emphasise positive, mechanism-based explanations that validate the patient's experience while comprehensively explaining how gut-brain processes may contribute to their symptoms. Translating complex concepts into simple explanations can be in itself a therapeutic skill and may reduce repeated investigations. The biopsychosocial model should be integrated into history taking, including symptom-specific anxiety, health beliefs, illness behaviours and noting their social, occupational, cultural and spiritual factors. A trauma-informed approach is critical, as patients may have had adverse childhood experiences and/or trauma histories.[28] Trainees should formulate links between trauma and symptom presentation, and explore these sensitively and compassionately.

Experiential learning opportunities are valuable. Where possible, trainees should be encouraged to collaborate in multidisciplinary teams such as gastroenterology, psychology, dietetics, pain services and primary care. These opportunities may arise as joint clinics, multidisciplinary meetings and shared inpatient discussions. Exposure to patients with DGBI and medically complex presentations helps trainees appreciate the clinical relevance of these concepts, and to practise applying this approach when assessing, formulating and managing patients.

Curriculum development should reflect the growing importance of the gut-brain axis across healthcare settings. Incorporating DGBI competencies, communication of gut-brain mechanisms and multidisciplinary management would better prepare psychiatrists for integrated models of care.

Research opportunities should also be encouraged, enabling trainees to develop critical appraisal skills and contributing to the emerging evidence base.
Clinicians must communicate the limits of current evidence, avoiding biomarker-like interpretations of microbiome associations and framing microbiome-targeted treatments as emerging adjuncts rather than substitutes for established care.[29] Research priorities include longitudinal cohorts with repeated microbiome, dietary and psychiatric measures to clarify temporal relationships, and intervention trials that specify strain, dose and duration while reporting clinically meaningful outcomes in populations with diagnosed psychiatric conditions. Mechanistic studies should examine whether mediators such as short-chain fatty acids, kynurenine-pathway metabolites and inflammatory markers change alongside clinical improvement.[30] Finally, implementation research is needed to test whether formulation-led multidisciplinary pathways reduce unnecessary investigations and improve real-world outcomes.

CONCLUSION

Integrating the gut–brain axis into liaison psychiatry strengthens holistic care for patients with complex medical and psychological needs. A biopsychosocial approach improves recognition, assessment, and management of distress in medical settings. Future training must embed these principles to support high-quality, compassionate, and evidence-based practice.

Future training should aim for a holistic approach with a focus on the gut-brain axis as an integral and dynamic feedback system where the influence of psychological and biological factors on gut function and symptom perception should be assessed. Effective management should target an integrated treatment pathway that could address both mind and body, improve quality of life and reduce healthcare burden.

Key Points for Clinical Practice

1. Gut–brain communication is biologically well established; causal claims linking a specific microbiome profile to a psychiatric diagnosis in humans are not.
2. Microbiome testing has no current diagnostic role in psychiatry. Commercial profiling should not be ordered as part of psychiatric assessment.
3. The biopsychosocial model earns its place only when it explains *how* domains act on one another in a particular patient — not as a list of factors.
4. A positive, mechanism-based explanation of symptoms is itself an intervention and reduces the drive to repeat normal investigations.
5. Microbiome-directed and dietary interventions are plausible adjuncts, not replacements for established psychiatric and gastroenterological treatment.

CONFLICT OF INTEREST

None to be reported

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Patient consent

The case described in Tables 1 and 2 is a hypothetical case used as an example and did not require consent from an individual.

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