REVIEW

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Cognitive Impairment in IBS: A Narrative Overview

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ABSTRACT

Irritable Bowel Syndrome (IBS) is a prevalent functional gastrointestinal disorder that significantly impacts the quality of life and the healthcare system. Beyond well-established symptoms such as recurrent abdominal pain and altered bowel habits, emerging evidence highlights a crucial yet underexplored aspect of cognitive dysfunction in patients suffering from IBS. This review aims to investigate the potential mechanisms linking IBS to cognitive dysfunction, emphasizing the role of the gut-brain axis and its biological and psychosocial determinants. A literature review was conducted using major medical databases, including Pubmed and Scopus, to identify relevant studies published in the last decade, focusing on clinical and experimental research assessing cognitive impairment in IBS patients. The review underscores the need for increased clinical recognition of cognitive dysfunction in IBS by highlighting the cognitive dysfunction in IBS patients with a focus on pathogenesis from disturbances in gut microbiota, inflammation, altered neurotransmitter levels, and psychological stress with various treatment modalities targeting these pathways, including probiotics, cognitive-behavioural therapy, and pharmacological interventions, showing promising results in mitigating cognitive symptoms.

Key words: irritable bowel syndrome – mind-gut axis – gut microbiota – intestinal barrier integrity – cognitive impairment.

Abbreviations: ACTH: adrenocorticotropic hormone; ANS: autonomic nervous system; CRF: corticotropin releasing factor; FD: functional dyspepsia; fMRI: functional magnetic resonance imaging; HPA: hypothalamic-pituitary-adrenal; IBS: irritable bowel syndrome; IBS-C: IBS with predominant constipation; IBS-D: IBS with predominant diarrhea; IBS-M: IBS with mixed bowel habits.

INTRODUCTION

Irritable bowel syndrome (IBS) is a common gastrointestinal disorder characterized by altered bowel habits, chronic abdominal pain, and visceral hypersensitivity without any discernible organic cause, affecting millions of individuals worldwide. The current Rome IV Global Study data shows that the prevalence of IBS is approximately 4.1% globally, 4.7% to 5.0% in the Western world and 1.5% to 2.0% in the Asian countries. There are also varied age and gender-related prevalence rates, with a sharp decline after age 50 and females being 1.5 times more likely to suffer from IBS. This disorder is often classified as a functional illness and poses a significant burden on healthcare and other resources. Direct costs are estimated at 1.6 billion, with an additional $19.2 billion in indirect costs, primarily from employee absenteeism and reduced quality of life [1, 2]. The Rome IV criteria defines IBS as a functional bowel disorder characterized by recurrent abdominal pain occurring at least one day per week in the last three months, with symptom onset at least six months before the diagnosis. There are four described subtypes: IBS with predominant constipation (IBS-C), IBS with predominant diarrhea (IBS-D), IBS with mixed bowel habits (IBS-M), or IBS un-subtyped [3].

While the gastrointestinal symptoms of IBS are well-recognized, evolving evidence suggests that individuals with IBS may also experience cognitive dysfunction, traditionally considered a hallmark of neurological disorders [4]. Many patients with IBS report cognitive difficulties such as impaired concentration, short-term memory issues, slower

information processing, and a general sense of reduced mental clarity. These cognitive complaints are not just by-products of chronic discomfort or pain but are believed to be directly linked to the complex interactions between the gastrointestinal tract and the central nervous system—a concept known as the "gut-brain axis" [5, 6]. Various biological and psychosocial factors including disruptions in gut microbiota composition, intestinal barrier integrity, immune activation, alterations in the hypothalamic-pituitary-adrenal (HPA) axis, and psychosocial factors like anxiety and depression, have been implicated in both IBS and cognitive dysfunction [7-9]. In addition, maladaptive coping strategies and illness perceptions further amplify the impact of IBS on cognitive function, creating a vicious cycle of symptom exacerbation and psychological distress [10]. Emerging neuroimaging studies such as functional magnetic resonance imaging (fMRI) studies have demonstrated aberrant processing of visceral stimuli and emotional regulation in regions implicated in cognitive processing, including the prefrontal cortex, anterior cingulate cortex, and insula which may underlie the cognitive symptoms observed in IBS [11]. Cognitive dysfunction in IBS still remains poorly understood and often overlooked in clinical practice. This article reviews the pathophysiology and treatment modalities for patients with IBS who also suffer from cognitive dysfunction.

PATHOPHYSIOLOGY

Functional dyspepsia (FD) and IBS are now grouped under the term "gut-brain axis disorders." Central to these conditions is a disruption in the gut-brain axis, driven by multiple pathological mechanisms such as altered gut motility, intestinal barrier dysfunction, immune dysregulation in the gut, visceral hypersensitivity, changes in gastrointestinal secretion, the presence and extent of bile acid malabsorption, and gut microbiota dysbiosis [12, 13]. These mechanisms are strongly impacted by patient psychiatric factors through a bidirectional relationship, making it essential to understand the complex interplay of psychological factors that can contribute to both the causation and the effects of these disorders. The gut microbiota plays a vital role in regulating communication between the gut and the nervous system, as well as maintaining gastrointestinal homeostasis and influencing higher cognitive functions. The brain-gut-microbiome system includes the central and enteric nervous systems, along with various neural, metabolic, endocrine, and immune mediators. The afferent vagus nerve transmits information from visceral organs to specific brain regions like the hypothalamus, amygdala, and insular cortex, as well as brainstem nuclei. These centres are crucial in relaying descending modulatory output from affective and cognitive cortical circuitry, as well as regulating autonomic and HPA axis output. The multiple pathways of communication work together, allowing reciprocal influence between the brain and gut and manifestations of symptoms of IBS [14]. Gut microbiome is the most essential component that alters these signalling pathways between the enteric and central nervous system. While numerous studies have indicated a connection between the gut microbiome and psychiatric disorders, the precise mechanisms driving this association remain incompletely understood. It is worth considering that the microbiome is influenced to some extent by host genetics. Therefore, the correlation between mental disorders and the gut microbiome could potentially stem from shared genetic factors influencing both traits [15]. Changes in the gut microbiota in experimental animals lead to gastrointestinal dysmotility, visceral hypersensitivity, alterations in intestinal permeability, and changes in behaviour [16]. In the pathogenesis of IBS, alterations in intestinal bacteria play a significant role by disrupting epithelial permeability, allowing the translocation of bacteria, which can trigger inflammation and provoke both local and systemic immune responses. This causes an imbalance in cytokines which perpetuates intestinal inflammation, contributing to the clinical manifestations of IBS. Additionally, cytokines such as interleukin (IL)-6, tumour necrosis factor-alpha, and IL-1b have been associated with anxiety and depression and ultimately impact the overall quality of life [17]. Microbial degradation products also play a significant role in disease pathogenesis. Microbial metabolites, particularly short-chain fatty acids, affect neurochemistry by boosting neurotransmitter production related to cognitive processes. Short-chain fatty acids (SCFAs) appear to be the most evident communication channel between the gut microbiota and the brain, with various hypothesized mechanisms for their action. SCFAs help to maintain intestinal barrier integrity and regulate gastrointestinal immune cells, which positively affects the peripheral immune system and, ultimately, protects against neuroinflammation. Furthermore, SCFAs can regulate neurotransmitter concentrations [e.g., serotonin, glutamate, γ-aminobutyric acid (GABA), and neurotrophic factors], affecting neuronal development, synaptic excitability, and cognitive processes including learning and memory. Finally, SCFAs influence the HPA axis, the primary neuroendocrine regulator of stress reactions. A recent human trial emphasized this point, showing that SCFAs decreased the cortisol response to psychological stress [18].

The biopsychosocial model of IBS pathogenesis explores the influence of the brain-gut microbiome axis on cognition and other brain functions. This model highlights the interaction between genetic and environmental factors, such as early life experiences, trauma, and social learning, affecting the brain and the gut. These interactions occur bidirectionally via the autonomic nervous system and the HPA axis. Corticotropin releasing factor (CRF) released in the hypothalamus is the initial step in HPA activation during the stress response. This indicates the primary endocrine response system to stress. The pituitary gland responds to CRF by releasing adrenocorticotropic hormone (ACTH), which stimulates the adrenal glands to secrete the stress hormone cortisol. Stress alters neurotransmitter and proinflammatory cytokine levels, which may directly or indirectly affect the microbiome. This change in gut flora may affect pain perception and intestinal permeability [19]. Visceral hypersensitivity and gut barrier disruption have also been shown to be directly mediated *via* CRF-Toll-like receptor 4 (TLR4)-proinflammatory cytokine signalling in animal experiments [20]. The combined effects of altered physiology and the individual's psychosocial status determine the experience of illness and ultimately impact clinical outcomes. Moreover, these outcomes, in turn, can

influence the severity of the disorder. Various psychological factors, such as health anxiety, symptom-specific anxiety, attentional bias, symptom hypervigilance, and catastrophizing, have been associated with functional gastrointestinal disorders independent of psychiatric comorbidity [21]. Symptoms and healthcare-seeking behaviours associated with IBS arise from a complex interplay of factors, as outlined in the biopsychosocial model. These factors include early life events, coping styles, learned behaviours, alterations in GI physiology, and psychological morbidity [22]. While psychological disorders can coincide with IBS, they often precede its onset, suggesting that they should not be viewed solely as a response to IBS. Early life risk factors for developing IBS encompass various forms of childhood adversity, such as increased socioeconomic stress, physical and sexual abuse during childhood, low birth weight, and strained parental relationships resulting from parental loss. Exposure to early life stress may lead to the persistence of childhood psychological distress into adulthood, potentially reducing the relative impact of current IBS symptoms on the individual's current psychological state [23]. Fig. 1 provides a detailed illustration integrating all the pathophysiological processes.

CLINICAL EVIDENCE

The literature on the association between IBS and cognitive impairment is conflicting (Table I). Studies assessed various parameters and domains of cognitive functioning with mixed results and various possible pathophysiological mechanisms. Several studies identified varying levels of cognitive impairment in patients with IBS [24-26], whereas others studies did not find definitive evidence of cognitive impairment within the IBS group [27-29]. Cognitive assessments typically encompass multiple cognitive domains, such as memory, attention, language, and executive functioning. Attree et al. [24] noted a statistically significant difference in verbal deficits between IBS and IBD groups compared to controls, with the IBD group scoring notably lower on object recognition but higher on spatial recognition. No such differences were observed in the IBS group [24].

Another study investigating the link between IBS and verbal deficits found no conclusive evidence supporting association between verbal IQ and IBS. However, IBS patients demonstrated a tendency to selectively process gastrointestinal symptom-related words when presented

Table I. Breakdown of literature review including author, study title, design, sample, and primary findings

Author	Title	Design	Sample	Findings
Attree EA, 2010 [24]	Cognitive function in people with chronic illness: inflammatory bowel disease and irritable bowel syndrome	Quasi-experimental design	70 participants (27 with IBS, 16 with IBD, 27 with control) performing cognitive function assessment tests	Illness group showed deficits in verbal IQ relative to own performance IQ and control but no difference in memory, recognition scores
Farup PG, 2015 [27]	Cognitive functions and depression in patients with irritable bowel syndrome	Cross-sectional study	66 participants (18 Neurological patients, 48 Depression patients) assessed on IBS severity scoring system, depression scale and cognitive function test	Cognitive functions did not differ significantly between the patients with and without IBS in any of the two groups. IBS was independent predictor of depression but not associated with differences in cognitive functions
Aizawa E, 2012 [25]	Altered cognitive function of prefrontal cortex during error feedback in patients with irritable bowel syndrome, based on FMRI and dynamic causal modelling.	Cross-sectional imaging study	60 participants (30 with IBS, 30 controls) underwent fMRI of brain to evaluate cognitive flexibility which was measured using Wisconsin Card Sorting Test	Participants with IBS have impaired cognitive flexibility due to reduced activity and connectivity between different brain regions
Kennedy PJ, 2014 [26]	Cognitive performance in irritable bowel syndrome: evidence of a stress-related impairment in visuospatial memory	Cross-sectional study	97 participants (39 with IBS, 18 disease controls with Crohn's disease in remission, 40 healthy controls) performed cognitive tests assessing several parameters	IBS patients revealed reduced visuospatial memory performance even after controlling for presence of psychiatric co-morbidity. But no major deficit in working memory, selective attention or executive function noted in IBS patients
Berrill, 2013 [28]	An observational study of cognitive function in patients with irritable bowel syndrome and inflammatory bowel disease	Cross-sectional study	231 participants (40 with IBS, 150 with IBD and 41 controls) completed Cardiff Cognitive Battery, series of neuropsychological performance tests	IBS or IBD patients don't possess any intrinsic disease pathology that was linked to cognitive impairment. Cognitive performance in IBD patients may be affected my concurrent mood disorders
Lam NC, 2019 [29]	Cognitive impairment in irritable bowel syndrome (IBS): A systematic review	Systematic review	Twelve studies were analysed based on inclusion criteria	Inconclusive or insufficient evidence to demonstrate that IBS patients have cognitive deficits in intelligence, executive function or memory. Study does note presence of attentional bias in IBS patients towards negative emotional words and GI sensation words

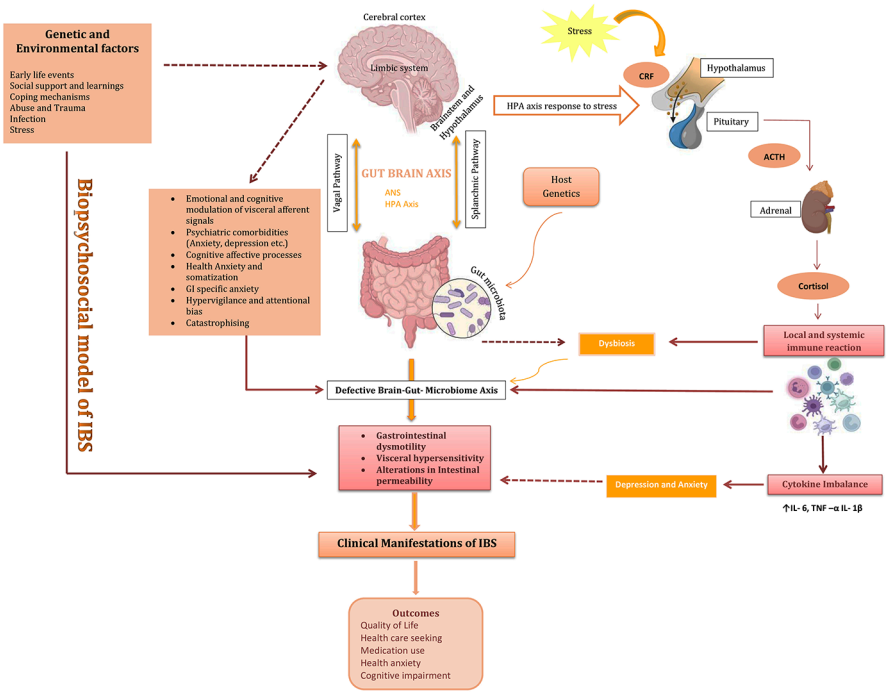

Fig. 1. Pathophysiology of the defective brain-gut-microbiome axis and its role in the development of IBS and associated cognitive impairments.

subliminally, suggesting a potential association between IBS and attentional bias [29]. Aizawa et al. [25] using MRI and the Wisconsin Card Sorting Test showed that individuals with IBS have subtle impairments in cognitive flexibility, likely due to altered activity in the dorsolateral prefrontal cortex, insula, and hippocampus, along with impaired connectivity between the dorsolateral prefrontal cortex and the pre-supplementary motor area [25].

A cross-sectional study assessing visuospatial memory performance reported deficits in this area among IBS patients, linked to low morning cortisol levels seen in the IBS group [26]. Another study evaluating attention, cognitive processing, and verbal fluency found no significant statistical difference between IBS and non-IBS groups, though depression and abdominal symptoms were more prominent in IBS patients [27]. Lastly, an observational study by Berrill et al. [28] reported no significant differences in fluid intelligence, crystallized intelligence, psychomotor speed, or attention between IBS, IBD patients, and healthy volunteers after accounting for mood disorders.

The initial thought paradigm explored the strong correlation between depression and cognitive loss, and with IBS patients frequently experiencing depression, this explored the possibility that they have some sort of cognitive impairment [30].

Attree et al. [24] reported that when comparing the verbal IQ of IBS patients to that of their own performance IQ and to healthy controls, there is a decrease in verbal IQ, but not a significant decline in performance on incidental memory assessment, troop color word test, or Wechsler Abbreviated Scale of Intelligence [24].

Based on the Mini-Mental State Examination, Trail-making tests, Grooved Pegboard test, Hopkins verbal learning test, brief visual memory test, Wechsler Adult Intelligence Scale 3rd Edition, Stroop test, or Controlled oral word association test, other authors did not discovered a significant correlation between cognitive function and IBS [27].

Overall, cognitive dysfunction in IBS has been a largely overlooked and under investigated manifestation of the brain-gut axis dysregulation in IBS. The initial cognitive assessments performed in IBS populations were based on the cognitive behavioural model which shows that patients have attentional biases toward stimuli associated with gastrointestinal symptoms or words that have negative connotations [31] According to this paradigm, those who are susceptible to IBS often exhibit symptoms of anxiety or depression in addition to having high standards for themselves that are frequently unreal (high levels of perfectionism). These people push through and stay busy when they have an infection like gastroenteritis, but this exacerbates their symptoms and forces them to rest. They attempt to resume their previous level of activity as soon as possible because resting exacerbates emotions of tension and anxiety. It is described as “all-or-nothing behaviour” and is typically associated with pushing too hard and then being forced to take a break. Such repetitive behaviour could lead to a perception or belief of having a chronic, incurable illness and becoming more and more upset about their symptoms. This model can account for enduring symptoms linked to distress and unfavourable perceptions about their condition [32].

Alternatively, according to a proposed cognitive neurobiological model, some of the main pathophysiological characteristics of IBS, such as immune activation, stress, and chronic pain, may influence important domains of executive function, working memory, attention, and episodic memory in addition to emotion- or symptom-related cognition [33]. An fMRI study that found reduced cognitive flexibility in the Wisconsin Card Sorting Test and differences in regional activity

in frontal and temporal brain regions in individuals with IBS has supported this paradigm [25].

As previously reported, individuals with IBS have elevated levels of anxiety and depression. Depressive and anxiety disorders are well known to be associated with cognitive abnormalities and a prior study has described the association between the cortisol awakening response, which assesses HPA-axis functioning, and hippocampal-mediated cognition in individuals who are not on medication for depression [34]. However, interestingly, another study found that there was a strong correlation between IBS and deficiencies in visuospatial memory performance, even when individuals with psychiatric co-morbidities were excluded from the analysis. This demonstrates that these cognitive deficiencies can exist independently of coexisting psychiatric medical conditions [33].

Structurally, differences are noted between IBS patients and healthy controls when evaluating brain imaging data, particularly in the areas of the brain linked to stress, visceral stimulation, sensory integration, affective processing, cognitive/executive functions, and somatic pain. Because pain has an emotional component, as do other associated symptoms of IBS like anxiety and depression, most of these findings point to a greater engagement of regions linked to emotional processing, such as the hypothalamus, amygdala, pregenual anterior cingulate cortex, and anterior insula [30].

Understanding the connection between cognition impairment and IBS is essential for effectively managing the comorbid conditions and improving patient outcomes. To achieve this, it is crucial to employ techniques such as screening, pharmacological therapy, and lifestyle modifications. Adopting integrated clinical management approaches can reduce the burden of these interconnected conditions. Recognizing the link between cognition impairment and IBS is fundamental in developing strategies for optimal patient care.

CONCLUSIONS

Cognitive dysfunction in IBS is yet an under-researched topic, with current studies showing mixed results regarding cognitive impairment and the specific neurocognitive domains affected, as well as the mechanisms behind this connection. The relationship between IBS and cognitive dysfunction involves the gut-brain axis. Biological and psychosocial factors, including gut microbiota changes, intestinal barrier integrity, immune activation, and factors like anxiety and depression, influence it. Future research should employ specialized assessment tools and extensive multicenter studies to address existing literature limitations and improve clinical care.

Conflicts of interest: None to declare.

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