



Irritable Bowel Syndrome Risk Factors and Quality of Life: An Evidence-Based Review with Implications for Family Medicine and Community Health

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Abstract

Background: Irritable bowel syndrome (IBS) is a common disorder of gut–brain interaction characterized by recurrent abdominal pain associated with altered bowel habits. Its clinical expression reflects a complex interaction among biological susceptibility, diet, intestinal microbiota, previous enteric infection, psychological stress, lifestyle, and social context. Beyond gastrointestinal symptoms, IBS can markedly impair daily functioning, emotional well-being, social participation, work productivity, and health-related quality of life (HRQoL). **Aim:** This review summarizes current evidence on major risk factors for IBS, explains how these factors interact with contemporary concepts of pathophysiology, and examines the determinants and clinical significance of impaired quality of life. **Methods:** Evidence was synthesized from international guidelines, systematic reviews, meta-analyses, population studies, and key mechanistic and clinical investigations, with emphasis on contemporary Rome IV concepts and clinically relevant findings for primary care. **Results:** Female sex, younger age, previous infectious enteritis, anxiety, depression, adverse psychosocial exposures, disturbed sleep, and selected dietary and environmental factors have been associated with IBS, although the strength and causal certainty of these associations vary. Genetic susceptibility, altered gut–brain signaling, visceral hypersensitivity, intestinal dysmotility, low-grade immune activation, bile acid abnormalities, and microbiome changes may connect these exposures to symptoms. HRQoL impairment is driven more consistently by overall symptom severity, gastrointestinal-specific anxiety, psychological comorbidity, extraintestinal symptoms, and functional interference than by bowel-habit subtype alone. **Conclusion:** IBS should be approached as a multidimensional disorder rather than a purely intestinal condition. Family physicians can improve care by combining a positive diagnosis with targeted assessment of modifiable risk factors, psychological burden, diet, lifestyle, and HRQoL, followed by individualized treatment and appropriate follow-up.

Keywords: Irritable bowel syndrome, Risk factors, Quality of life, Primary care



Introduction

Irritable bowel syndrome (IBS) is now classified among disorders of gut–brain interaction, a term that better reflects the dynamic relationship between intestinal function, neural processing, behavior, and psychosocial context. Rome IV defines IBS by recurrent abdominal pain, on average at least one day per week during the previous three months, associated with defecation or a change in stool frequency or form, with symptom onset at least six months before diagnosis. This symptom-based framework allows a positive clinical diagnosis rather than defining IBS only after exhaustive exclusion of structural disease. [1]

IBS is common across regions, but estimates vary substantially according to the diagnostic criteria, survey method, and population studied. A large global meta-analysis based on Rome III and Rome IV criteria demonstrated major differences in prevalence between diagnostic systems, while the Rome Foundation Global Study confirmed that disorders of gut–brain interaction are frequent worldwide and impose a considerable symptom and health-care burden. More recent pooled evidence continues to show marked geographic heterogeneity, reinforcing that prevalence figures should always be interpreted in relation to the diagnostic method used. [2,3]

The clinical importance of IBS extends beyond abdominal pain and altered bowel habits. Patients commonly report bloating, urgency, incomplete evacuation, fatigue, sleep disturbance, anxiety, food-related concerns, and limitations in social and occupational activities. The condition may persist for years with fluctuating symptom intensity, and its burden is often underestimated when clinical assessment focuses only on stool pattern. A contemporary view therefore needs to consider both the factors that increase susceptibility to IBS and the factors that determine how severely the condition affects everyday life. [4]

The present review examines the main demographic, biological, dietary, infectious, psychological, and lifestyle factors associated with IBS, links them with current concepts of pathophysiology, and evaluates their relationship with health-related quality of life. Particular attention is given to evidence that is useful in family medicine, where early recognition, reassurance, selective investigation, lifestyle assessment, and longitudinal management can reduce unnecessary testing while addressing the broader functional burden of the disorder. [5]

Epidemiology and Diagnostic Context

Population estimates illustrate why IBS cannot be represented by a single universal prevalence figure. Earlier pooled studies reported a worldwide prevalence close to 11%, with higher rates in women and lower rates in people older than 50 years, whereas analyses using more recent Rome criteria have produced different estimates. A 2025 meta-analysis including studies from more than 50 countries reported substantial variation by Rome version and IBS subtype, reflecting differences in case definition as well as population characteristics. [6]

Rome IV also tends to identify a clinically more symptomatic group than previous definitions. Studies comparing Rome III with Rome IV populations found that individuals meeting the newer criteria often had greater abdominal pain, higher gastrointestinal symptom severity, more psychological symptoms, and poorer quality of life. This distinction is important when comparing older and newer risk-factor studies, because apparent differences across studies may partly reflect changes in the population classified as having IBS rather than a true change in disease biology. [7]

Risk Factors for Irritable Bowel Syndrome

Risk in IBS is multifactorial, and no single exposure explains most cases. A recent umbrella review grouped reported determinants into dietary, genetic, environmental, psychological, microbiome-related, socioeconomic, physiological, and pathological domains. Female sex and anxiety were among the most repeatedly reported associations, while evidence for depression and previous gastroenteritis was comparatively stronger. The review also highlighted a major limitation of the literature: many reported risk factors are supported mainly by observational evidence, so association should not automatically be interpreted as causation. [8]

Sex is one of the most reproducible demographic associations. Women are more frequently diagnosed with IBS in many community studies and are particularly represented among patients with constipation-predominant disease. Biological explanations include effects of sex hormones on motility, visceral sensation, immune activity, and pain processing, while social factors, health-care seeking, and cultural differences may modify the observed sex distribution. Sex-related differences may also extend to symptom reporting and quality of life, which can influence who reaches primary or specialist care. [6,9]

Age is also relevant, although its effect is less uniform across settings. IBS frequently begins in adolescence or early adulthood, and pooled epidemiologic work has generally found lower prevalence in older populations. Younger adults may be exposed to combinations of irregular eating, academic or occupational stress, sleep disruption, and changing dietary patterns, but these contextual factors should be separated from age itself. Clinically, new-onset bowel symptoms at an older age require greater attention to alternative diagnoses and alarm features rather than being attributed automatically to IBS. [4]



Socioeconomic findings are complex. Earlier international analyses did not show a simple relationship between national development level and IBS prevalence, suggesting that broad country-level economic indicators are poor substitutes for individual social exposures. More recent cohort evidence indicates that lower individual socioeconomic position may be associated with greater incident IBS risk, with part of the association mediated by lifestyle factors. Financial strain, reduced access to health information, occupational stress, dietary constraints, and barriers to continuity of care may therefore shape both disease risk and subsequent illness burden. [10,11]

Genetic susceptibility contributes to IBS, but the effect of individual variants is modest and interacts with environmental and psychological exposures. A large genome-wide association study involving more than 53,000 IBS cases identified several susceptibility loci and demonstrated substantial shared genetic architecture with anxiety, neuroticism, and depression. These data do not mean that IBS is primarily a psychiatric disorder; rather, they support biological overlap in neural and gut–brain pathways that may influence visceral perception, stress responsiveness, and symptom expression. [12]

Early-life exposures may also influence later vulnerability. A systematic review identified associations between adult IBS and childhood gastrointestinal symptoms, adverse experiences, illness behavior learned within families, and selected socioeconomic conditions. The evidence is heterogeneous and largely observational, but it supports a life-course model in which early biological stressors and behavioral responses can modify gut–brain regulation long before IBS becomes clinically established. This perspective may be especially relevant for family physicians who follow patients across different stages of life. [13]

Diet is one of the most frequently reported symptom-related domains in IBS. Patients commonly attribute abdominal pain, bloating, diarrhea, or urgency to meals, particularly large meals, fatty foods, caffeine, alcohol, spicy foods, and fermentable carbohydrates. Dietary exposure can alter luminal water content, fermentation, gas production, bile acid handling, motility, and visceral signaling. However, a food that triggers symptoms after IBS is established is not necessarily a factor that originally caused the disorder, and this distinction is important when counseling patients who have begun progressively restrictive diets. [14]

Fermentable oligosaccharides, disaccharides, monosaccharides, and polyols (FODMAPs) are a well-studied example. These short-chain carbohydrates can be incompletely absorbed in the small intestine, increase luminal water, and undergo rapid colonic fermentation. Controlled trials and network meta-analysis support a low-FODMAP approach for short-term improvement of global symptoms in selected patients, but restriction should normally be followed by structured reintroduction and personalization to reduce unnecessary dietary limitation and potential adverse effects on nutritional adequacy and the microbiome. [15]

Fiber illustrates the importance of distinguishing fiber type rather than simply advising patients to increase total intake. Soluble fiber, particularly ispaghula or psyllium, has evidence for improving global IBS symptoms, whereas coarse insoluble fiber may worsen bloating or discomfort in some patients. Meal regularity, adequate fluid intake, and gradual dietary modification are often more practical in primary care than abrupt extensive exclusions, especially when constipation, irregular meals, or low habitual fiber intake coexist. [16]

The intestinal microbiome has become a major research focus because some patients with IBS show differences in microbial composition and metabolic function compared with healthy individuals. A systematic review reported recurring alterations involving several bacterial groups, but no single microbial signature has sufficient consistency to diagnose IBS. Differences between studies are influenced by diet, geography, medication exposure, sequencing methods, IBS subtype, and disease severity, so dysbiosis is better viewed as one component of a heterogeneous pathophysiologic network rather than a universal abnormality. [17]

Experimental work provides biologic plausibility for a microbiome contribution. Transfer of fecal microbiota from patients with IBS into germ-free mice produced changes in gastrointestinal transit, intestinal barrier function, immune activity, and anxiety-like behavior in recipient animals. Such studies cannot establish that microbiota changes are the initiating cause of human IBS, but they support the possibility that microbial communities can transmit functional characteristics relevant to gut–brain signaling and symptom generation. [18]

Previous infectious gastroenteritis is among the best documented acquired risk factors. Post-infectious IBS can follow bacterial, viral, or protozoal enteritis, with increased risk reported after more severe acute illness and among individuals with psychological distress around the time of infection. A systematic review and meta-analysis found a persistent excess risk after infectious enteritis, supporting a model in which mucosal immune activation, altered permeability, microbiome disruption, and neural sensitization may remain after the acute infection has resolved. [19]

Bile acid metabolism may be particularly relevant in patients with diarrhea-predominant IBS. Research has identified a subgroup with excessive fecal bile acid excretion, altered serum markers of bile acid synthesis and feedback regulation, and distinct microbiome features. One mechanistic study linked a *Clostridia*-rich microbial pattern with increased bile acid synthesis and excretion through changes involving fibroblast growth factor 19 signaling. These findings help explain why apparently similar diarrhea symptoms can arise through different biological mechanisms. [20]



Psychological factors have one of the strongest and most clinically relevant associations with IBS. Anxiety and depression occur more often in people with IBS than in healthy controls, and the relationship is bidirectional: emotional distress can amplify gastrointestinal symptoms, while recurrent unpredictable bowel symptoms can worsen anxiety, low mood, and anticipatory fear. A meta-analysis confirmed substantially increased anxiety and depressive symptoms across IBS populations, supporting routine assessment of psychological burden without implying that symptoms are imagined or purely psychogenic. [21]

Stress influences IBS through several interconnected pathways, including autonomic nervous system activity, hypothalamic-pituitary-adrenal signaling, central pain modulation, immune responses, and changes in motility and permeability. Repeated stress may lower the threshold at which intestinal sensations become painful and increase attention to gastrointestinal cues. The clinical effect varies widely between individuals, which is why a careful history of stressors, symptom timing, coping responses, and gastrointestinal-specific fears often yields more useful information than simply asking whether a patient is “stressed.” [22]

Sleep disturbance is another frequently overlooked factor. Systematic reviews show that poor sleep is common in IBS and is associated with greater gastrointestinal symptom burden, although the direction of causality remains uncertain. Inadequate or fragmented sleep may increase pain sensitivity and stress reactivity, while nocturnal worry, abdominal discomfort, and comorbid mood symptoms may themselves impair sleep. Asking about sleep quality therefore adds useful information to the broader assessment of symptom amplifiers and potentially modifiable contributors. [23]

Physical inactivity is not established as a major independent cause of IBS, but activity level can influence symptom experience and general well-being. In a randomized trial, increased physical activity improved IBS symptom scores compared with usual activity. Regular movement may also benefit sleep, mood, constipation, cardiometabolic health, and self-efficacy, making graded physical activity a reasonable component of first-line care when tailored to the patient’s baseline fitness and symptom pattern. [24]

How Risk Factors Converge in IBS Pathophysiology

The modern pathophysiologic model of IBS is based on altered communication within the gut–brain axis. This system includes the central nervous system, autonomic pathways, hypothalamic-pituitary-adrenal axis, enteric nervous system, immune signaling, enteroendocrine mechanisms, and gut microbiota. Signals move in both directions: intestinal events can alter mood and cognition, while psychological and central neural processes can change motility, secretion, permeability, and visceral sensation. This framework explains why biological and psychosocial risk factors often interact rather than operate independently. [25]

Visceral hypersensitivity and altered central processing help explain why normal levels of intestinal distension or contraction may be experienced as painful. Dysmotility contributes to diarrhea or constipation, while low-grade immune activation and barrier dysfunction may amplify sensory signaling in susceptible patients. In a detailed physiologic study, psychological distress, visceral hypersensitivity, and abnormal transit had cumulative associations with patient-reported symptom severity, showing that clinical burden often emerges from several abnormalities acting together rather than from a single dominant mechanism. [26]

This multidimensional model also explains why patients with the same bowel-habit subtype can have very different illness experiences. One patient may be driven mainly by post-infectious changes and diarrhea, another by constipation and pelvic or motility factors, and another by severe gut-specific anxiety with relatively modest changes in transit. Treatment therefore works best when the clinician identifies the most relevant modifiable mechanisms for the individual instead of applying a uniform sequence of therapies to all patients with the IBS label. [5]

Health-Related Quality of Life in IBS

Health-related quality of life describes the patient’s perception of how illness affects physical, psychological, and social functioning. IBS can disrupt daily planning because symptoms are intermittent and often unpredictable. Patients may limit travel, avoid meals outside the home, seek constant access to toilets, reduce exercise, withdraw from social events, or experience concern about odor, urgency, or visible bloating. These restrictions can become more disabling than the bowel symptoms themselves and may persist even when symptom intensity fluctuates. [27]

Disease-specific measurement is useful because generic quality-of-life scales may miss concerns that are particularly important in IBS. The 34-item IBS Quality of Life questionnaire was developed to assess domains including dysphoria, interference with activity, body image, health worry, food avoidance, social reaction, sexual concerns, and relationships. It demonstrated strong internal consistency and reproducibility, allowing clinicians and researchers to capture aspects of burden that are not represented by stool frequency or pain intensity alone. [28]

The IBS-QOL is also responsive to clinical change. Work examining health-related quality of life in functional bowel disorders showed that improvement after treatment can be quantified and that clinically meaningful changes in disease-specific scores can be distinguished from statistical fluctuation. This supports the use of quality-of-life assessment as an outcome rather than treating it as an optional descriptive measure, particularly in patients whose main concern is restricted function rather than bowel frequency. [29]



Overall symptom severity is one of the most consistent determinants of poor quality of life, but bowel-habit subtype alone has a weaker and less consistent relationship. Some studies have found differences between IBS-C, IBS-D, and mixed disease, whereas others have shown that the intensity of pain, bloating, urgency, and global symptom burden is more important than subtype classification. This matters clinically because a patient with “mild” stool abnormality but severe pain, fear, or activity restriction may have greater overall disease burden than a patient with more frequent bowel changes but little functional impairment. [26]

Gastrointestinal-specific anxiety is especially important. It refers to fear, worry, and hypervigilance focused on gastrointestinal sensations and their anticipated consequences. Patients may constantly scan for abdominal changes, avoid situations where a toilet is not immediately accessible, or interpret normal sensations as warning signs of an impending episode. Research has shown that gastrointestinal-specific anxiety is strongly associated with symptom severity and mental quality of life, independently of general anxiety and depression in some analyses. [30]

More recent work confirms that disease-specific quality of life reflects a combination of gastrointestinal symptoms, psychological factors, and extraintestinal symptom burden. In a large clinical analysis, gastrointestinal-specific anxiety, fatigue, somatic symptoms, and psychological distress were important correlates of IBS-QOL. This suggests that simply normalizing stool form may not restore quality of life if fear, fatigue, hypervigilance, and broad somatic burden remain untreated. [31]

Sex and social context can also influence quality of life. Studies have reported sex-related differences in clinical symptoms and IBS-QOL, while socioeconomic disadvantage may add barriers related to health literacy, access to dietetic or psychological care, employment flexibility, and continuity of follow-up. The direction and magnitude of these associations vary across populations, so they are best assessed individually rather than assumed from demographic characteristics alone. [9]

Comorbidities broaden the burden further. IBS frequently overlaps with functional dyspepsia, chronic pelvic pain conditions, migraine, fibromyalgia, fatigue, and mood disorders. Overlapping disorders can increase symptom count, diagnostic uncertainty, health-care use, and functional impairment. When several conditions coexist, treating each symptom in isolation can lead to fragmented care; a coordinated biopsychosocial approach is more likely to address the cumulative effect on quality of life. [32]

The socioeconomic burden of IBS is also substantial. Direct costs arise from consultations, investigations, medications, and repeated health-care contact, while indirect costs include absenteeism, reduced productivity while at work, and impairment in unpaid activities. A 2023 analysis demonstrated that indirect productivity losses represented a large component of total costs and that poorer disease-specific quality of life and depressive symptoms were associated with greater economic burden. Thus, improving quality of life has relevance not only to patient well-being but also to health systems and workplaces. [33]

Clinical Implications for Family Medicine

Primary care is well positioned to make a positive diagnosis of IBS when symptoms are typical and alarm features are absent. International guidance recommends a focused history, physical examination, and limited targeted testing rather than indiscriminate investigation. Depending on the clinical presentation, evaluation may include blood tests, celiac serology, and inflammatory markers or fecal calprotectin in patients with diarrhea or suspected inflammatory disease. Colonoscopy is reserved for patients with age-appropriate screening indications, alarm features, or findings that suggest an alternative diagnosis. [34]

The first consultation should also identify what matters most to the patient. Two people who meet the same Rome criteria may have very different priorities: pain control, reliable bowel function, confidence to travel, relief of food-related anxiety, better sleep, or the ability to work without interruption. Explaining the gut–brain model in clear non-stigmatizing language can validate symptoms while opening discussion of diet, stress, sleep, and psychological treatment as legitimate components of gastrointestinal care. A strong therapeutic relationship is itself an important part of long-term management. [35]

Initial management usually combines education with practical lifestyle and dietary advice. Regular meals, adequate hydration, individualized fiber modification, moderation of obvious dietary triggers, and graded physical activity are reasonable starting points. More restrictive approaches such as a low-FODMAP diet are best used for selected patients and, when possible, with dietetic guidance so that restriction is followed by reintroduction and personalization. The objective is symptom control with the least restrictive sustainable diet, not permanent avoidance of a growing list of foods. [15]

Psychological therapies should be considered when stress, anxiety, gastrointestinal-specific fear, or persistent symptom-related disability is prominent. Meta-analyses support cognitive behavioral therapy, gut-directed hypnotherapy, and other gut–brain behavioral interventions for global IBS symptoms, with benefits extending beyond mood alone. These treatments are particularly relevant when repeated investigations have not reassured the patient or when avoidance and hypervigilance continue to restrict daily life despite reasonable control of bowel habit. [36]



Pharmacologic treatment remains symptom directed and should be matched to the dominant clinical problem. Soluble fiber and selected antispasmodic therapies can help global symptoms in some patients, peppermint oil can reduce pain and global symptoms, and subtype-specific agents may be appropriate when constipation or diarrhea remains persistent. Pharmacologic therapy is most effective when it is integrated with education, diet, and attention to psychological and functional burden rather than used as the only component of care. [16,37]

Manipulation of the microbiome remains an evolving area. Trials of probiotics show possible benefit for global IBS symptoms, but effects vary by strain, combination, dose, and study design, making broad claims about “probiotics” inappropriate. Rifaximin has a defined role in selected patients with IBS-D in major guidelines, while routine microbiome testing and commercial stool profiling do not currently have an established role in standard IBS diagnosis. Family physicians should distinguish therapies supported by controlled evidence from microbiome claims that have not reached clinical validation. [38]

Quality of life should be reassessed during follow-up, particularly when symptom counts improve but the patient remains functionally restricted. Persistent avoidance, work impairment, sexual concerns, social withdrawal, or health anxiety may require a different intervention from persistent constipation or diarrhea. Using a short structured discussion or a validated disease-specific instrument can help determine whether treatment is changing the outcomes that matter to the patient and can reveal needs that routine symptom questions miss. [28]

Research Gaps and Future Directions

Several evidence gaps remain. Risk-factor studies are often cross-sectional, use different diagnostic criteria, and vary in measurement of diet, stress, socioeconomic status, and microbiome features. The 2025 umbrella review found that many systematic reviews were of low methodological quality, which limits causal interpretation even when associations are repeatedly reported. Future prospective studies should define exposures before IBS onset, use standardized Rome-based case definitions, and include diverse populations from regions that remain underrepresented in current research. [8]

A second priority is movement from broad IBS categories toward clinically meaningful phenotypes. Integration of symptom profiles with psychological measures, bile acid metabolism, immune markers, microbiome function, diet, and objective motility may identify subgroups that respond to different treatments. New dietary network meta-analyses already suggest that several structured dietary approaches can improve symptoms, but long-term comparative effectiveness, nutritional safety, cost, patient preference, and quality-of-life outcomes require clearer evaluation before one strategy can be generalized to all patients. [39]

Research should also give greater weight to patient-centered endpoints. Symptom severity remains important, but return to normal activity, food flexibility, work participation, reduction of gastrointestinal-specific fear, and sustained improvement in HRQoL may better represent meaningful recovery. Studies that combine symptom and quality-of-life outcomes are more likely to clarify whether an intervention changes the lived burden of IBS rather than only producing statistically significant differences in isolated gastrointestinal measures. [40]

Conclusion

In conclusion, IBS is a heterogeneous, multifactorial, and potentially disabling disorder that requires a comprehensive biopsychosocial approach. Family physicians play a central role in early recognition, reassurance, selective investigation, lifestyle assessment, and longitudinal management. By addressing both the factors that increase susceptibility to IBS and the factors that determine how severely the condition affects everyday life, clinicians can reduce unnecessary testing, alleviate symptom burden, and improve the quality of life of patients living with this chronic and often underestimated condition.

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