Irritable bowel syndrome (IBS)

Disorder of gut–brain interaction characterised by recurrent abdominal pain associated with altered bowel habits, without an identifiable structural cause on routine assessment.

Dr Paul Tervit

Domain	Clinical summary
Rome IV criteria	Recurrent abdominal pain, on average **≥1 day/week during the previous 3 months**, associated with at least two of: relation to defecation, change in stool frequency, or change in stool form. Symptom onset should be ≥6 months before diagnosis.
Subtypes	IBS-C: constipation predominant; IBS-D: diarrhoea predominant; IBS-M: mixed; IBS-U: unclassified.
Key history	Pain pattern and relation to defecation; Bristol stool type; frequency, urgency and nocturnal symptoms; bloating; dietary triggers; medication use; psychosocial stressors; previous infection; family history of IBD, coeliac disease or colorectal cancer.
Examination	General appearance, weight/BMI, pallor, lymphadenopathy, abdominal masses or focal tenderness; consider rectal examination where clinically indicated.
Alarm features	Rectal bleeding, iron-deficiency anaemia, unintentional weight loss, nocturnal diarrhoea, fever, palpable mass, persistent vomiting, late/new onset, or relevant family history. These should prompt investigation for organic disease rather than presumptive IBS.
Initial investigations	Usually FBC, CRP or ESR, and coeliac serology. In diarrhoeal presentations, consider faecal calprotectin and targeted stool testing. Further testing should be guided by age, alarm features and clinical phenotype. NICE supports a positive, symptom-based diagnosis after appropriate limited exclusion testing.
Important differentials	Coeliac disease, IBD, colorectal malignancy, microscopic colitis, bile-acid diarrhoea, lactose or other carbohydrate intolerance, medication effects, thyroid disease, endometriosis and chronic infection.
Management principles	Clear explanation and therapeutic relationship; regular meals, hydration and exercise; soluble fibre such as ispaghula rather than insoluble bran; dietitian-supervised low-FODMAP trial where appropriate; symptom-directed antispasmodics, laxatives or antidiarrhoeals; gut–brain neuromodulators and psychological therapies for persistent symptoms.

IBS has a demonstrable pathophysiology, but not a single unifying lesion or biomarker. It is best understood as a **heterogeneous disorder of gut–brain interaction**, in which several mechanisms converge to produce pain and altered bowel habit.

Mechanism	Current interpretation
Visceral hypersensitivity	Probably the most consistent physiological feature. Normal distension, gas or contractions may be perceived as painful because of sensitisation at enteric, spinal and central levels. It is present in a substantial subgroup, not universally.
Altered gut–brain processing	Abnormal bidirectional signalling can affect autonomic tone, attention to visceral signals, stress responses and descending pain modulation. This does **not** imply that IBS is purely psychological; central processing interacts with genuine peripheral gut abnormalities.
Motility and secretion	Transit and contractile patterns may be accelerated in IBS-D or delayed in IBS-C, but abnormalities are variable and are not sufficiently specific for diagnosis.
Mucosal immune activation	Some patients show subtle mast-cell activation, cytokine changes, increased epithelial permeability and sensitisation of nearby afferent nerves, especially following infection. These changes are far below the overt inflammation of IBD.
Microbiome and metabolites	Group-level differences in microbial composition and fermentation are reported, but there is no reproducible diagnostic “IBS microbiome”. Microbial metabolites, bile acids and gas production may modify permeability, motility and nociception.
Food–luminal interactions	FODMAPs, bile acids and other luminal stimuli can increase water content or distension. Symptoms depend partly on the patient’s sensory threshold rather than simply the amount of gas produced.

Serotonergic dysregulation - Serotonin is one component rather than the sole cause.

Point	Clinical meaning
Most gastrointestinal serotonin is produced by enterochromaffin cells	It regulates peristaltic reflexes, secretion, urgency and signalling from the gut to sensory nerves.
5-HT_3 signalling	Promotes secretion, transit and nociceptive signalling. Excessive or prolonged signalling may contribute to IBS-D phenotypes; this provides the rationale for 5-HT_3 antagonists in selected IBS-D patients.
5-HT_4 signalling	Facilitates motility and secretion; 5-HT_4 agonism can be useful in constipation-related disorders.
Observed abnormalities	Studies report altered serotonin release, receptor function or serotonin-transporter activity, but findings vary between subtypes and studies. There is no clinically useful serum or mucosal serotonin test for IBS.

Post-infectious IBS - This is one of the strongest causal models for IBS.

Feature	Interpretation
Definition	New-onset IBS after a documented episode of acute infectious gastroenteritis in someone without preceding IBS.
Relationship to severity	Risk rises with more severe or prolonged gastroenteritis, particularly where there is fever, bleeding, significant mucosal injury or prolonged diarrhoea.
Proposed sequence	Infection → epithelial injury and altered permeability → persistent low-grade immune activation/mast-cell activity → microbial alteration → enteric nerve sensitisation → visceral hypersensitivity and altered motility.
Additional risk modifiers	Female sex, psychological distress around the infection and possibly antibiotic exposure are repeatedly associated, though none is deterministic.
Typical phenotype	Often IBS-D or IBS-M initially, although bowel phenotype may evolve.

IBS is **partly heritable but strongly polygenic and environmentally modified**.

- A large genome-wide analysis identified several susceptibility loci, but individual effects were very small.
- Common-variant SNP heritability was estimated at approximately **6%** in that study, much lower than for classic inflammatory bowel diseases.
- Some genetic signals overlap with anxiety, depression and neuronal pathways. This likely reflects shared neurobiological susceptibility rather than IBS being caused by psychiatric disease.
- Family clustering also includes shared diet, microbiome, infection exposure, learned illness behaviour and early-life environment.

There is therefore no useful routine genetic test for IBS.

Integrative model

A useful formulation is:

Predisposition
genetic/early-life/stress-related susceptibility

+ precipitant
gastroenteritis, antibiotics, dietary or psychosocial stress

→ **peripheral gut change**
microbiome, permeability, immune and enteroendocrine signalling

→ **neural sensitisation**
enteric, spinal and cerebral

→ **clinical phenotype**
pain, bloating, diarrhoea/constipation and sometimes bladder or other somatic overlap.

The relative contribution of each element differs considerably between patients, which explains why no single investigation or treatment works universally.

FODMAP

Letter	Meaning	Main examples
F	Fermentable	Rapidly fermented by colonic bacteria
O	Oligosaccharides	Fructans in wheat, onion and garlic; galacto-oligosaccharides in beans and pulses
D	Disaccharides	Lactose in milk, yoghurt and soft cheese
M	Monosaccharides	Excess fructose in honey, mango and some fruit juices
A	And	—
P	Polyols	Sorbitol and mannitol in some fruits and vegetables; sweeteners such as xylitol and maltitol

FODMAPs are **short-chain carbohydrates that are incompletely absorbed in the small intestine**. They can:

- draw water into the bowel through an osmotic effect;
- undergo rapid bacterial fermentation;
- increase intestinal gas and luminal distension;
- provoke pain, bloating, urgency, diarrhoea or altered bowel habit in patients with visceral hypersensitivity.

They do not necessarily cause IBS; rather, they can trigger symptoms in susceptible patients.

Typical food sources

FODMAP group	Higher-FODMAP examples	Common lower-FODMAP alternatives
Fructans	Onion, garlic, leek, wheat-based bread and pasta	Garlic-infused oil, chives, spring-onion green tops, rice, oats, quinoa
Galacto-oligosaccharides	Chickpeas, lentils, kidney beans, cashews	Firm tofu, tempeh; small portions of well-rinsed canned legumes
Lactose	Ordinary milk, ice cream, custard, soft cheeses	Lactose-free milk, hard cheeses, suitable plant milks
Excess fructose	Honey, mango, apples, pears, high-fructose syrups	Oranges, kiwifruit, grapes, strawberries
Polyols	Stone fruit, cauliflower, mushrooms; sorbitol-containing sweets and chewing gum	Carrots, courgettes, aubergine, potatoes, blueberries

FODMAP content is **portion-dependent**. A food may be tolerated in a small serving but become high-FODMAP in a larger serving. Food lists therefore need serving-size information rather than a simple permitted/forbidden classification.

The diet has three phases

Phase	Practical approach
1. Restriction	Reduce major high-FODMAP foods for a limited period, commonly around **2–6 weeks**, while keeping the diet nutritionally balanced.
2. Reintroduction	Once symptoms are reasonably controlled, challenge one FODMAP subgroup at a time, generally using increasing portions over several days. The rest of the diet remains relatively low-FODMAP so that the response is interpretable.
3. Personalisation	Resume all tolerated foods and restrict only the specific groups and quantities that reproducibly cause symptoms.

The objective is **not lifelong universal FODMAP avoidance**. It is to identify individual thresholds and achieve the least restrictive effective diet. NICE recommends that exclusion diets such as low-FODMAP be delivered by a professional with expertise in dietary management, and both BDA and Monash guidance emphasise structured reintroduction.

Important clinical nuances

- **Wheat sensitivity is often due to fructans, not gluten.** A low-FODMAP diet is therefore not intrinsically gluten-free.
- **Lactose restriction is not required** when lactose is tolerated.
- **Symptoms are dose-dependent and cumulative.** Several moderate-FODMAP foods eaten together may create a larger total load.
- It is primarily an **IBS symptom-management intervention**, not a test for food allergy or intolerance.
- Prolonged restriction may reduce dietary variety, fibre, calcium and some prebiotic substrates; it may also reinforce food anxiety.
- Coeliac disease should be investigated **before** substantial wheat or gluten restriction, because restriction can make serological and histological testing less reliable.

A dietitian-guided food-and-symptom diary, including portion size, stool form, pain, bloating and urgency, usually makes reintroduction more clinically useful.

Role of peppermint oil in IBS

Aspect	Clinical relevance
Mechanism	Menthol relaxes intestinal smooth muscle, largely through calcium-channel blockade, producing an **antispasmodic effect**. This may reduce visceral pain and postprandial cramping.
Best evidence	Enteric-coated peppermint oil can improve **global IBS symptoms and abdominal pain** over the short term. Guideline recommendations are generally supportive but recognise low-to-moderate certainty and heterogeneity among formulations.
Symptoms most likely to improve	Abdominal pain, cramping and possibly bloating. It has no consistent direct effect on stool frequency or consistency, so it is not a primary treatment for diarrhoea or constipation.
Formulation	Use an **enteric-coated capsule**, which releases oil in the small bowel rather than the stomach. Peppermint tea and non-enteric preparations have not been studied equivalently and are more likely to cause reflux.
Administration	Product-specific dosing should be followed; commonly studied regimens use approximately **180–225 mg two or three times daily**, usually before meals, for a short therapeutic trial of about 4–8 weeks.
Adverse effects	Heartburn, reflux, peppermint taste or belching, nausea and perianal burning—particularly with diarrhoea.
Avoid or use cautiously	Significant gastro-oesophageal reflux or hiatus hernia, gallstones or biliary obstruction, severe liver disease, and pregnancy or breastfeeding where product-specific safety information is limited. Capsules should generally be swallowed whole.
Clinical position	A reasonable first-line, symptom-directed option for IBS with prominent pain or cramps, alongside dietary and lifestyle measures. Review benefit after several weeks and discontinue if ineffective or poorly tolerated.

Peppermint oil treats **symptoms**, not the underlying cause, and should not delay evaluation where bleeding, weight loss, anaemia, nocturnal symptoms, fever or other alarm features are present.