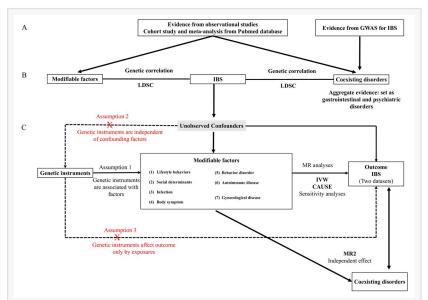


Chronic Pain and Mental Well-being Linked to IBS Risk: Genetic Study Identifies Modifiable Factors

Large Mendelian randomisation study links multisite chronic pain to IBS, urging combined gastrointestinal and mental health care approaches.

CHANGCHUN, CHINA, August 15, 2025 /EINPresswire.com/ -- Irritable bowel syndrome (IBS) is a prevalent and debilitating gastrointestinal disorder affecting approximately 5–10% of the global population. Characterized by abdominal pain, bloating, and altered bowel habits, IBS imposes a significant burden on quality of life and healthcare systems worldwide. Despite its prevalence, the exact pathogenesis of IBS remains elusive, and effective prevention strategies are lacking. Here, Liu et al. conducted a comprehensive Mendelian randomisation (MR)



Systematic review of modifiable factors/comorbidities; LDSC for genetic correlations with IBS; MR & MR2 for causal links. Solid arrows = causal effects; dashed/cross = prohibited by MR assumptions II/III. CAUSE, GWAS, IVW as defined.

study—an approach that uses genetic variants as instrumental variables to infer causality. The study integrates Mendelian randomization (MR) and multiresponse MR (MR2) analyses to distinguish genuine causal relationships from shared or spurious associations. The study was published in eGastroenterology.

The study reviewed observational evidence up to May 2024 and analysed GWAS data from over 50 studies, including 53,400 IBS cases and 433,201 controls, with validation in the FinnGen Biobank. Researchers assessed more than 50 modifiable factors across seven domains and 20 coexisting disorders. Using genetic correlation and MR methods, they identified causal and shared risk pathways between IBS and related gastrointestinal and psychiatric conditions, enhancing understanding of modifiable contributors to IBS.

The study identified numerous significant genetic correlations between IBS and modifiable

correlations were observed for:
 Lifestyle factors: lifetime smoking index, alcohol frequency, insomnia; Social determinants: education level, income, childhood maltreatment; Body symptoms: multisite chronic pain, migraine, frailty index; Gastrointestinal disorders, including gastro-oesophageal reflux disease, peptic ulcer disease, dyspepsia, non-alcoholic fatty liver disease; Psychiatric traits: neuroticism, depression, anxiety, bipolar disorder.
Among the evaluated factors, multisite chronic pain emerged as the strongest and most consistent causal factor. Notably, several dietary and behavioural exposures—including tea intake, coffee consumption, and physical activity—showed no consistent causal relationship with IBS, despite previously reported observational associations.
The MR analyses also highlighted several coexisting gastrointestinal and psychiatric disorders with convincing or suggestive causal links to IBS: Gastro-oesophageal reflux disease showed consistent associations across the main MR models. Diverticular disease of the intestine and schizophrenia had suggestive causal links.
Psychiatric traits such as depression, anxiety, bipolar disorder, and neuroticism were

factors, with correlation coefficients (rg) ranging from 0.0005 to 0.718 across datasets. Strong

The multiresponse MR (MR2) analysis suggested that factors such as lifetime smoking index, intelligence and childhood maltreatment were found to be linked to coexisting disorders rather than being independent risk factors for IBS.

significantly genetically correlated with IBS.

The study provides compelling evidence that certain psychological and somatic factors are not just correlated but causally linked to IBS. The most robust finding is the role of multisite chronic pain, which remains significant even after adjusting for psychiatric and gastrointestinal comorbidities. This supports the theory that central pain sensitization and brain-gut axis dysregulation are critical mechanisms underlying IBS. Psychological well-being also emerged as a strong determinant of IBS risk. Traits such as low positive affect, neuroticism, and depression not only co-occur with IBS but are also likely contributors to its pathogenesis. Meanwhile, associations with lifestyle factors such as smoking and alcohol use, though present, were weaker and possibly mediated through comorbid psychiatric disorders. These findings suggest that preventive strategies for IBS could benefit from targeting chronic pain syndromes and improving psychological well-being. Moreover, recognising and managing psychiatric comorbidities in IBS patients could improve outcomes and reduce the symptom burden.

This study significantly enhances the understanding of IBS by identifying modifiable and causal

risk factors through genetic approaches. Clinicians and public health practitioners should consider:
 Screening IBS patients for chronic pain conditions and addressing them as part of treatment; Recognising the psychological dimension of IBS and integrating mental health support into care models;
 Being cautious when interpreting associations from observational studies that don't adjust for psychiatric comorbidities;
Supporting multi-disciplinary prevention strategies targeting shared risk factors for IBS and its coexisting disorders.
These results also support shifting the paradigm from a symptom-based to a risk-based approach in IBS prevention and treatment.

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