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Faecal Microbiota Composition and Metabolomic Profiles as Diagnostic Markers in Patients with Irritable Bowel Syndrome: A Review

Tiina Virtanen[1*], Rui Carvalho[2] and Stefan Horvath[3]

[1]Department of Gastroenterology, University of Helsinki, Helsinki, Finland

[2]Department of Clinical Microbiology, University of Coimbra, Coimbra, Portugal

[3]Department of Metabolomics, Semmelweis University, Budapest, Hungary

***Correspondence to: Tiina Virtanen, Department of Gastroenterology, University of Helsinki, Helsinki, Finland**

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Abstract

Irritable bowel syndrome is diagnosed on symptoms alone, and a confirmatory test has been sought for two decades. Faecal microbiota and metabolite profiles differ reproducibly between patients and controls. Whether that difference is large enough to diagnose an individual is a separate question, and the answer so far is no.

Objectives: *To review the published evidence on faecal microbiota composition and metabolomic profiling as diagnostic markers in irritable bowel syndrome: which taxa and metabolites differ consistently, what accuracy classifiers based on them achieve, and how they compare with the one faecal test already in routine use.*

Material and Methods: *Narrative review of case-control cohorts, meta-analyses, metabolomic profiling studies and diagnostic guidelines indexed in PubMed and the major gastroenterology journals. Studies were eligible if they reported differences in faecal microbiota or metabolites between patients with irritable bowel syndrome and comparators, or the diagnostic accuracy of a marker or classifier built from them. No new patient data were generated; every estimate quoted is one published by the original investigators.*

Results: *Pooling 13 studies covering 360 patients and 268 controls, three taxa were consistently reduced: Bifidobacterium (standardised mean difference -1.17), Faecalibacterium prausnitzii (-1.05) and Lactobacillus (-0.85), with the reduction most marked in diarrhoea-predominant disease. Bacteroides-Prevotella and Escherichia coli did not differ. A faecal volatile metabolite model separated diarrhoea-predominant disease from Crohn's disease, ulcerative colitis and healthy controls with sensitivities of 94, 96 and 90 per cent and specificities of 82, 80 and 80 per cent. Combining microbial and metabolite profiling in a single Bayesian classifier achieved 84 per cent overall accuracy and 86 per cent for diarrhoea-predominant disease in cross-validation. By comparison, faecal calprotectin distinguishes inflammatory bowel disease from irritable bowel syndrome with pooled sensitivity of 85.8 per cent and specificity of 91.7 per cent across 17 studies and 1,956 patients.*

Conclusion: *Microbiota and metabolite differences in irritable bowel syndrome are real and reproducible at group level, and the reported classifier accuracies come from cross-validation within derivation cohorts rather than independent testing. The one faecal marker with established diagnostic utility remains calprotectin, which excludes an alternative diagnosis rather than confirming this one.*

Abbreviations: *IBS – Irritable Bowel Syndrome, IBS-D – Diarrhoea-Predominant IBS, IBS-C – Constipation-Predominant IBS, IBS-M – Mixed-Type IBS, IBD – Inflammatory Bowel Disease, SCFA – Short-Chain Fatty Acid, VOC – Volatile Organic Compound, SMD – Standardised Mean Difference, NMR – Nuclear Magnetic Resonance, PLS-DA – Partial Least Squares Discriminant Analysis, AUC – Area Under the Receiver Operating Characteristic Curve.*

Keywords: *Irritable Bowel Syndrome; Faecal Microbiota; Metabolomics; Short-Chain Fatty Acids; Diagnostic Biomarkers*

Introduction

Irritable bowel syndrome affects a substantial share of the adult population and is diagnosed entirely on symptom criteria [1,2,3]. The Rome criteria, revised most recently in 2016, define it by abdominal pain related to defecation or to a change in stool form or frequency, in the absence of an alternative explanation [4,5].

Symptom-based diagnosis has a specific weakness. In validation work the Rome IV criteria showed sensitivity of around 63 per cent against a specificity of 97 per cent [6], which means the criteria exclude well and capture poorly. Guidelines from both British and American societies therefore recommend limited investigation to exclude organic disease rather than any positive confirmatory test [7,8].

The search for such a test has concentrated on stool, for the obvious reason that the disorder is of the bowel and stool is

obtainable without endoscopy. Two lines have developed in parallel: characterising the faecal microbiota [9,10,11] and profiling the metabolites it produces [12,13,14]. Both have generated reproducible group-level differences.

This review sets out what those differences are, what accuracy has been achieved when they are turned into classifiers, and how that compares with faecal calprotectin, the one stool marker already established in this diagnostic pathway [15].

Material and Methods

Search strategy: PubMed and the archives of the major gastroenterology journals were searched for studies of faecal markers in irritable bowel syndrome. Search terms combined irritable bowel syndrome with faecal microbiota, gut microbiome, dysbiosis, metabolomics, short-chain fatty acids, volatile organic compounds, calprotectin and diagnostic accuracy. Reference lists of retrieved meta-analyses and guidelines were screened by hand.

Eligibility: Studies were included if they reported differences in faecal microbiota composition or metabolite profiles between patients with irritable bowel syndrome and healthy or disease controls, or the diagnostic performance of a marker or classifier derived from them. Studies in inflammatory bowel disease are cited where they define the comparator or establish the method, and are identified as such.

Data extraction and handling: Design, sample size, comparator group, sequencing or analytical platform, and reported effect or accuracy estimates were extracted as published. Whether an accuracy figure came from cross-validation within the derivation cohort or from an independent sample is stated in every case, because that distinction is decisive in this literature. No pooling or re-analysis was performed and no patient-level data were obtained. Figures 1 to 4 and Tables 1 to 3 reproduce published values only. This is a narrative synthesis and was not registered as a systematic review.

Results

Three taxa are consistently reduced: A meta-analysis pooling 13 studies with 360 patients and 268 healthy controls found significant reductions in Bifidobacterium (standardised mean difference -1.17, $p < 0.001$), Faecalibacterium prausnitzii (-1.05, $p < 0.001$) and Lactobacillus (-0.85, $p < 0.001$), but no difference in the Bacteroides-Prevotella group, Escherichia coli or other genera examined (Figure 1) [9]. Subgroup analysis showed the reductions were larger in diarrhoea-predominant disease, at -1.81 for Lactobacillus and -1.45 for Bifidobacterium [9].

Independent work supports the same direction. Case-control and cross-cohort analyses of publicly available datasets have confirmed compositional differences [10], a large meta-analysis identified 26 genera and twelve predicted pathways with classification potential [11], and earlier real-time PCR and sequencing studies established that the faecal community differs from that of healthy subjects [16,17]. A subtype defined by species-specific alterations has been described [18], and functional dysbiosis has been characterised specifically in constipation-predominant disease [19].

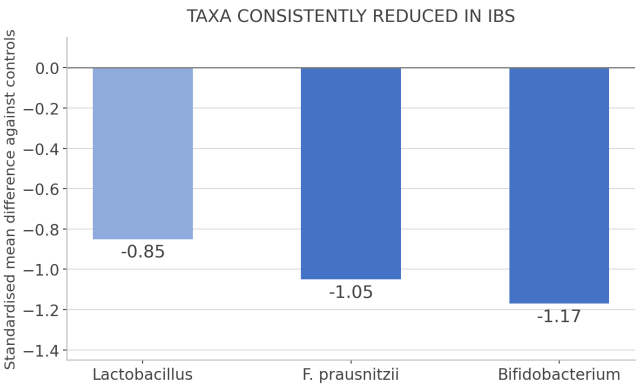

Figure 1: Taxa consistently reduced in irritable bowel syndrome across 13 pooled studies [9]

The heterogeneity that limits interpretation: The pooled estimate for Faecalibacterium prausnitzii carried no between-study heterogeneity, but that for Bifidobacterium was substantial at 79.3 per cent [9]. The reduction in Faecalibacterium prausnitzii is also not specific: it is reduced in inflammatory bowel disease, where pooled analysis put the standardised mean difference at -0.94, more markedly in Crohn's disease than in ulcerative colitis [20]. Loss of the same butyrate-producing species, alongside Roseburia hominis, has been proposed as defining dysbiosis in ulcerative colitis [21], and faecal dysbiosis is present in Crohn's disease and in unaffected relatives [22].

A marker reduced in both irritable bowel syndrome and inflammatory bowel disease cannot distinguish between them, which is the differential that matters clinically. That constraint applies to the taxa with the largest and most reproducible effects.

Study	n	Material	Principal reported finding
Malinen et al. 2005 [16]	—	Microbiota	Faecal community differs by real-time PCR
Jeffery et al. 2012 [18]	—	Microbiota	Subtype defined by species-specific alterations
Ahmed et al. 2013 [13]	—	Metabolites	Volatile model separates IBS-D from IBD and controls
Farup et al. 2016 [12]	50	Metabolites	Propionic minus butyric acid best discriminator
Meta-analysis 2017 [9]	628	Microbiota	Bifidobacterium, F. prausnitzii and Lactobacillus reduced
Wang et al. 2020 [10]	—	Microbiota	Dysbiosis confirmed across case-control studies

Table 1: Principal studies reviewed, with the material examined and the finding as reported by the original investigators

Metabolites perform better than taxa: Profiling what the community produces has yielded stronger discrimination than profiling the community itself. A model built from faecal volatile organic metabolites separated diarrhoea-predominant disease from active Crohn's disease, ulcerative colitis and healthy controls with sensitivities of 94, 96 and 90 per cent and specificities of 82, 80 and 80 per cent (Figure 2) [13]. Esters of short-chain fatty acids and cyclohexanecarboxylic acid derivatives were associated with the diarrhoea-predominant subtype, while aldehydes were more abundant in inflammatory bowel disease [13].

Short-chain fatty acids have been examined directly. In a case-control study of 25 patients and 25 controls, the difference between propionic and butyric acid concentrations discriminated better than any single acid [12], and total short-chain fatty acid concentration is reported lower in diarrhoea-predominant disease

while butanoic acid is higher [14]. Both butanoic and propanoic acid recur across studies as discriminating compounds [14]. Earlier work had established altered carbohydrate fermentation in the same subtype [23].

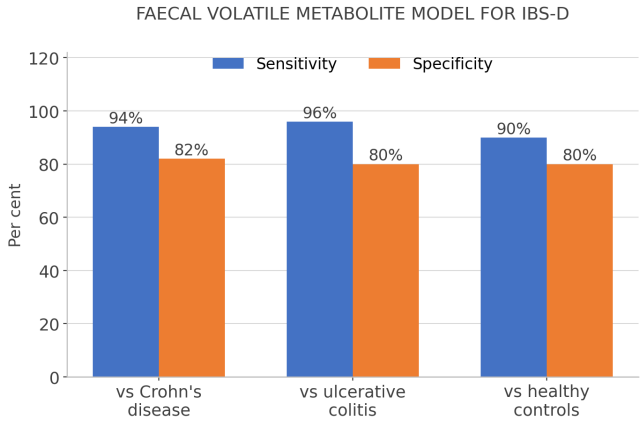

Figure 2: Faecal volatile metabolite model separating diarrhoea-predominant disease from three comparator groups [13]

Combining the two adds little: The obvious next step is to use microbial and metabolite data together. A study applying phylogenetic microarray profiling and proton nuclear magnetic resonance spectrometry to the same stool samples built separate partial least squares classifiers and integrated them by a Bayesian approach. The combined model achieved 84 per cent accuracy of group assignment and 86 per cent prediction for diarrhoea-predominant disease in cross-validation (Figure 3) [24].

Those figures are lower than the volatile metabolite model achieved alone [13], which suggests the two data types carry overlapping rather than complementary information. Both sets of estimates come from cross-validation within the cohorts in which the models were built, with only limited de novo testing on an independent sample [24].

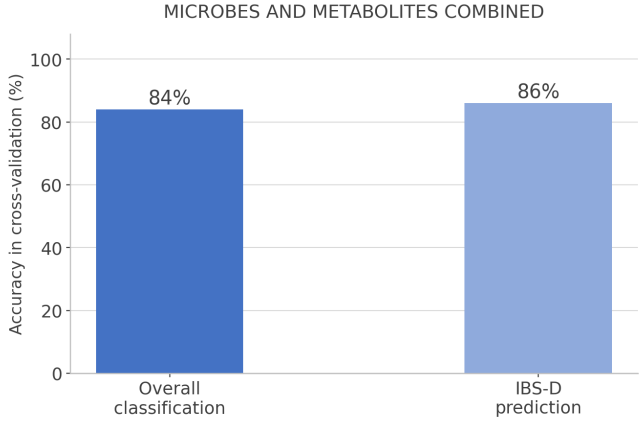

Figure 3: Accuracy of a combined microbial and metabolite classifier in cross-validation [24]

The comparator that already works: Faecal calprotectin is not a test for irritable bowel syndrome, and that is precisely why it is useful. Pooling 17 studies with 1,956 patients, it distinguished inflammatory bowel disease from irritable bowel syndrome with summary sensitivity of 85.8 per cent (95% CI 78.3-91.0) and specificity of 91.7 per cent (95% CI 84.5-95.7) (Figure 4) [15]. Performance was better in Western populations and at a threshold of 50 micrograms per gram [15].

The contrast is instructive. Calprotectin achieves clinically useful accuracy by answering a question with a mechanistic basis, whether neutrophils are present in the bowel, rather than by classifying a symptom-defined syndrome. It performs less well where inflammatory bowel disease is in remission and irritable bowel-type symptoms coexist, a group in which calprotectin is elevated in subsets of patients [25].

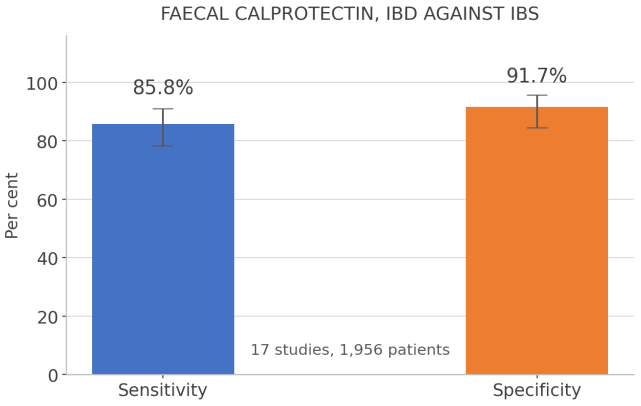

Figure 4: Pooled accuracy of faecal calprotectin for distinguishing inflammatory bowel disease from irritable bowel syndrome [15]

Marker	Best reported performance	Principal limitation
Individual taxa	SMD -0.85 to -1.17 against controls [9]	Group-level only; not specific to IBS [20]
Volatile metabolites	90-96% sensitivity, 80-82% specificity [13]	Single cohort; not independently validated [13]
Short-chain fatty acids	Propionic minus butyric acid discriminates [12]	50 subjects; direction inconsistent across studies [12,14]
Combined classifier	84% overall, 86% for IBS-D [24]	Cross-validation within derivation cohort [24]
Faecal calprotectin	85.8% sensitivity, 91.7% specificity [15]	Excludes IBD rather than confirming IBS [15,25]

Table 2: Faecal markers examined, their best reported performance and the limitation described for each

Discussion

The evidence supports a conclusion that is more sceptical than the volume of publication suggests. Faecal microbiota and metabolite profiles do differ between patients and controls, reproducibly and with effect sizes that are large in standardised terms [9,10,11]. But a standardised mean difference of around one, with overlapping distributions, describes two populations whose means differ, not two populations a clinician can tell apart in an individual patient.

The specificity problem compounds this. The taxon with the most consistent reduction, Faecalibacterium prausnitzii, is reduced in inflammatory bowel disease to a comparable degree [20,21], so it cannot serve the differential that matters most. A marker of generic gut disturbance is not a marker of irritable bowel syndrome.

Why the reported accuracies should be discounted

Classifier accuracies of 84 to 96 per cent look diagnostic until the design is examined. Both the volatile metabolite model and the combined microbial and metabolite classifier were developed and assessed largely within their own cohorts, with cross-validation rather than independent external testing [13,24]. Models fitted to high-dimensional data and tested by cross-validation reliably overstate performance, and the correction on external validation is typically substantial.

The comparison with calprotectin makes the point concretely. Its 85.8 per cent sensitivity and 91.7 per cent specificity come from 17 studies and nearly 2,000 patients across independent centres [15], and are therefore worth more than a nominally similar figure from a single derivation cohort.

Study limitations

The limitations belong to the underlying literature. Sample sizes in the metabolomic studies are small, with the short-chain fatty acid study including 50 subjects in total [12]. Between-study heterogeneity is substantial for several taxa, reaching 79.3 per cent for Bifidobacterium [9]. Sequencing platforms, analytical methods and stool handling differ across cohorts and are known to affect measured composition [11,17]. Subtype definitions differ between Rome revisions, so cohorts recruited under different criteria are not directly comparable [4,5,6]. Diet, which strongly influences both microbiota and metabolite profiles, is rarely controlled [26,27]. And most studies compare patients with healthy controls rather than with the symptomatic patients from whom they would need to be distinguished in practice [9,13].

Two gaps deserve naming. No classifier reviewed here has been tested prospectively in an unselected population presenting with lower gastrointestinal symptoms, which is the setting where a diagnostic test would be used [13,24]. And no study reports whether a positive result would change management, given that treatment is symptom-directed regardless [7,8].

Conclusion

Faecal microbiota composition in irritable bowel syndrome differs reproducibly from that of healthy controls, with Bifidobacterium, Faecalibacterium prausnitzii and Lactobacillus consistently reduced and the effect largest in diarrhoea-predominant disease. Metabolite profiling discriminates better than taxonomic profiling, and combining the two adds nothing. The accuracies reported for these classifiers, between 84 and 96 per cent, come from cross-validation within derivation cohorts and should be read as upper bounds. The most reduced taxon is reduced in inflammatory bowel disease as well, which disqualifies it from the differential that matters. Faecal calprotectin remains the only stool marker with established utility here, and it works by excluding an alternative diagnosis rather than confirming this one.

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Conflict of Interest

No competing interest is held by any author of this review.

Ethics Statement

Everything examined here comes from the published record. With no recruitment and no fresh patient data, the article did not require ethical clearance.

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