This is an author produced version of *Screening for bile acid diarrhoea in suspected irritable bowel syndrome*.

White Rose Research Online URL for this paper:
http://eprints.whiterose.ac.uk/97312/

**Other:**

http://dx.doi.org/10.1136/gutjnl-2014-307579
Title: Screening for Bile Acid Diarrhoea in Suspected Irritable Bowel Syndrome.

Authors: Imran Aziz¹, Matthew Kurien¹, David S Sanders¹, Alexander C Ford²-³.
¹Department of Gastroenterology, Royal Hallamshire Hospital, Sheffield Teaching Hospitals, Sheffield, UK.
²Leeds Gastroenterology Institute, St. James’s University Hospital, Leeds, UK.
³Leeds Institute of Biomedical and Clinical Sciences, University of Leeds, Leeds, UK.

Correspondence: Dr. Alex Ford
Leeds Gastroenterology Institute
Room 125
4th Floor
Bexley Wing
St. James’s University Hospital
Beckett Street
Leeds
United Kingdom
LS9 7TF
Email: alexf12399@yahoo.com
Telephone: +441132684963
Facsimile: +441132429722
Editor;

We read the paper by Bajor et al. with interest. (1) The authors demonstrated that 18% of patients who meet criteria for irritable bowel syndrome (IBS) may have underlying bile acid diarrhoea (BAD), using 23-seleno-25-homo-tauro-cholic acid (SeHCAT) scanning. This issue has been the subject of a previous systematic review and meta-analysis, (2) which reported that up to 30% of individuals with IBS had evidence of idiopathic BAD. However, many of the included studies were retrospective, and few used accepted symptom-based criteria to define the presence of IBS, underlining the importance of the data from Bajor et al., who recruited a well-characterised and rigorously defined cohort of patients meeting the Rome III criteria for IBS.

We therefore congratulate the authors on conducting this study. The concept that underlying organic gastrointestinal (GI) disease may explain symptoms compatible with IBS, is not novel. The current gold-standard for diagnosing IBS, the Rome III criteria, perform only modestly in diagnosing IBS, (3) and several studies have reported that GI symptoms in organic conditions such as coeliac disease, (4;5) exocrine pancreatic insufficiency, (6) inflammatory bowel disease, (7;8) and small intestinal bacterial overgrowth (9) may overlap with those of IBS. However, with Bajor and colleagues demonstrating that up to one in five patients with suspected IBS have underlying BAD, the magnitude of this issue has several implications for both clinical practice and future research.

Firstly, the prevalence of BAD in patients with suspected IBS appears to be far higher than that of coeliac disease, estimated at 4% in a recent meta-analysis. (4) Current guidelines for the management of IBS in both the UK and USA recommend screening for coeliac disease in patients with suspected IBS routinely. (10;11) However, as yet there has been no consensus recommendation on the merits of screening for BAD in suspected IBS. A recent
study demonstrated that uptake of SeHCAT scans in patients with suspected IBS is low, with only 2% of patients undergoing this as part of their diagnostic work-up, (12) and a median time of 30 weeks between initial consultation and a diagnosis of BAD, during which other less useful investigations were requested. The results of Bajor et al. suggest the yield of SeHCAT testing in IBS is likely to be substantial. (1)

Secondly, the strategy of making a positive diagnosis of IBS, without recourse to investigations, which has always been advocated by experts, may no longer be appropriate or desirable. An alternative approach to the management of patients with suspected IBS could be akin to that for uninvestigated dyspepsia, and perhaps head-to-head trials of various management strategies, for example empirical “usual treatment” for suspected IBS versus “test and treat” for BAD, with SeHCAT scanning followed by bile acid sequestrants for those who test positive, are now warranted.

Finally, and perhaps most importantly of all, with almost 20% of people with suspected IBS demonstrating evidence of BAD, this suggests that treatment trials of novel therapies for the disorder are being “diluted” by patients who do not actually have IBS, and that this may be contributing to the lack of observed efficacy for some of these agents, or underestimating their true effectiveness.

While the results of Bajor et al. need to be replicated by others, they are exciting, and could lead to a paradigm shift in the way that we manage and treat suspected IBS.

“The Corresponding Author has the right to grant on behalf of all authors and does grant on behalf of all authors, an exclusive licence (or non-exclusive for government employees) on a worldwide basis to the BMJ Publishing Group Ltd and its Licensees to permit this article (if accepted) to be published in Gut and any other BMJPGL products to exploit all subsidiary
Authors: Imran Aziz\textsuperscript{1}, Matthew Kurien\textsuperscript{1}, David S Sanders\textsuperscript{1}, Alexander C Ford\textsuperscript{2,3}.

\textsuperscript{1}Department of Gastroenterology, Royal Hallamshire Hospital, Sheffield Teaching Hospitals, Sheffield, UK.

\textsuperscript{2}Leeds Gastroenterology Institute, St. James’s University Hospital, Leeds, UK.

\textsuperscript{3}Leeds Institute of Biomedical and Clinical Sciences, University of Leeds, Leeds, UK.

Competing Interests:

DSS and ACF have received grant support and speakers fees from GE Healthcare limited.

REFERENCES


