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## TITLE PAGE

**Title:** Aetiopathogenesis of Functional Dyspepsia.

**Authors:** Alexander C Ford<sup>1,2</sup>.

<sup>1</sup>Leeds Gastroenterology Institute, St. James's University Hospital, Leeds, UK.

<sup>2</sup>Leeds Institute of Biomedical and Clinical Sciences, University of Leeds, Leeds, UK.

**Correspondence:** Dr. Alex Ford  
Leeds Gastroenterology Institute  
Room 125  
4<sup>th</sup> Floor  
Bexley Wing  
St. James's University Hospital  
Beckett Street  
Leeds  
United Kingdom  
LS9 7TF

Email: [alex12399@yahoo.com](mailto:alex12399@yahoo.com)

Telephone: +441132684963

Facsimile: +441132429722

**Abbreviations:** EPS epigastric pain syndrome

FD functional dyspepsia

IBS irritable bowel syndrome

PDS postprandial distress syndrome

**Keywords:**

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Editor;

I read the paper by Fang et al. with interest. [1] The authors reported risk factors for functional dyspepsia (FD), defined using the Rome III criteria, in a large referral population from Taiwan. They reported that these criteria performed poorly in distinguishing between organic pathology and FD, that those with FD were more likely to exhibit concomitant irritable bowel syndrome (IBS), and that there were distinct differences in risk factors between epigastric pain syndrome (EPS) and postprandial distress syndrome (PDS). The latter observation led them to conclude that these two FD subgroups have a distinct aetiopathogenesis.

The Rome III criteria have been shown to be no better than previous criteria in predicting FD by other investigators, [2] and the prevalence of organic disease in patients with uninvestigated dyspepsia following upper gastrointestinal endoscopy in the present study was broadly similar, although it is perhaps surprising that there were no cases of gastric cancer detected among the 771 patients with uninvestigated dyspepsia, given the South East Asian population. The fact that the authors demonstrated that 29% of patients with FD had coexistent IBS again is confirmatory of the findings of others, [3] and the odds ratio of 6.89 corroborates the findings of a previous meta-analysis of cross-sectional surveys examining this issue. [4]

A recent meta-analysis of population-based studies suggested that gender, smoking, non-steroidal use, H. pylori infection and other proposed risk factors for dyspepsia were only modestly associated with the likelihood of reporting symptoms. [5] The stronger associations reported by Fang et al. may well relate to the fact that their study was conducted in a referral population. However, their conclusion that EPS and PDS have distinct aetiologies is perhaps an over-simplistic interpretation of their data, given the fact that they conducted multiple

analyses, and therefore some of the significant results they observed could have occurred by chance alone. This is reinforced by the fact that one in three of those with FD met criteria for both EPS and PDS, suggesting that these two subgroups are not as easily separated as the authors suggest. A more convincing case could perhaps have been made if they had shown that any of the associations they observed were unique to EPS or PDS, and not found among those with organic causes of dyspepsia, when compared with healthy controls. Unfortunately, these analyses were not conducted, although given that many of the proportions reported in Table 2 for each of the risk factors they examined were similar for the 491 patients with FD, and the entire group of 771 patients with uninvestigated dyspepsia, I suspect this is unlikely.

In conclusion, while the results of Fang et al. are interesting, they do not provide convincing mechanistic evidence that EPS and PDS are distinctly different conditions.

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**Authors:** Alexander C Ford<sup>1,2</sup>.

<sup>1</sup>Leeds Gastroenterology Institute, St. James’s University Hospital, Leeds, UK.

<sup>2</sup>Leeds Institute of Biomedical and Clinical Sciences, University of Leeds, Leeds, UK.

## **Competing Interests:**

ACF: none to declare.

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