Correspondence

Faecal alpha-1-antitrypsin in chronic inflammatory bowel disease

SIR,—Fischbach et al.¹ found that patients with chronic inflammatory bowel disease (CIBD) had significantly raised faecal alpha-1-antitrypsin (FA1AT) concentrations compared with controls and other patients with non-inflammatory bowel disorders. Patients with clinically active disease had higher FA1AT's than those with quiescent disease, but the difference was not significant. Clinical disease activity correlated better with ESR, albumin, orosomucoid, serum A1AT, and ¹¹¹In labelled granulocyte excretion. Their CIBD patients had lower FA1AT's compared with those we reported² and by Meyers.³ Fischbach et al suggested that this discrepancy could have been because of ‘patient selection’ rather than a ‘technical problem’. They stated that most of their patients were not very ill; the range of disease activity scores was not presented in their paper.

Fischbach and colleagues concluded that assessment of inflammatory activity cannot be solely based upon FA1AT excretion because factors other than mucus inflammation could result in excessive enteric protein loss. While this might be true, I disagree with some of the author’s interpretations of their results. First, in our experience, patients with moderate to severe CIBD have higher FA1AT's.⁴ One would expect lower values for patients, such as described by Fischbach et al, with only mild clinical disease.

Second, it is possible that the authors’ findings were the result of a technical problem with the assay. The FA1AT concentrations reported in our study⁵ were generated on now unavailable Calbiochem Behring M-Partigen radial immunodiffusion (RID) plates. Since then our laboratory has had extensive experience in performing this assay on different RID systems.⁶ Other investigators have also reported variation in A1AT values done on different commercially available RID plates.⁷ The manufacturer suggests that 20 μl supernatant be placed into the wells of the newer LC Partigen RID plate. We have found that 15 μl is optimal; Fischbach et al used 5 μl in their study. By doing so, they may have used an insufficient volume to allow adequate diffusion of the sample from the wells into the surrounding gel. Thus, many of their values could have been spuriously low. They also did not specify their source of A1AT standards. Standard heterogeneity has been a problem we have encountered with commercial RID systems. One cannot extrapolate standard concentrations determined by nephelometry directly to RID because values determined by the former method are consistently lower. Details of assay technique are extremely important when measuring FA1AT with LC Partigen RID plates because of its narrow quantitative range and the low concentrations of A1AT in stool compared to serum.

Interestingly, Fischbach et al found lower A1AT intestinal clearance for patients with active CIBD. This probably can be attributed to their patients’ elevated serum A1AT concentrations and the use of this value as the denominator in the equation for calculating clearance.

I recommend that differences in techniques be taken into account for comparing data from other studies involving this assay. Because the determination of FA1AT is relatively inexpensive and easy to perform, when done correctly, I feel that its use in the assessment of disease activity for CIBD remains valuable.

DAN W THOMAS

Division of Gastroenterology,
Department of Pediatrics,
Children’s Hospital of Los Angeles, and the
University of Southern California School of Medicine,
Los Angeles, CA 90027, USA

References


Reply

SIR.—Thomas comments on some important aspects concerning the usefulness of faecal alpha-1-antitrypsin (A-1-AT) in assessing disease activity in chronic inflammatory bowel disease (CIBD). We appreciate to answer to some of his remarks considering our results as well as general aspects of faecal A-1-AT.

Faecal A-1-AT concentrations were lower in our patients with CIBD compared with those of Thomas¹
Faecal alpha-1-antitrypsin in chronic inflammatory bowel disease.

D W Thomas

*Gut* 1988 29: 262-263

doi: 10.1136/gut.29.2.262

Updated information and services can be found at:
[http://gut.bmj.com/content/29/2/262.1.citation](http://gut.bmj.com/content/29/2/262.1.citation)

**Email alerting service**

*These include:*

Receive free email alerts when new articles cite this article.
Sign up in the box at the top right corner of the online article.

**Notes**

To request permissions go to:
[http://group.bmj.com/group/rights-licensing/permissions](http://group.bmj.com/group/rights-licensing/permissions)

To order reprints go to:
[http://journals.bmj.com/cgi/reprintform](http://journals.bmj.com/cgi/reprintform)

To subscribe to BMJ go to:
[http://group.bmj.com/subscribe/](http://group.bmj.com/subscribe/)