the effect of other degradative enzymes present in the samples. Hence, the results obtained by Markesich *et al* are not comparable to that of ours.[1 2 4–6] Studies with gastric juice performed by others,[3] however, showed lower viscosity values for *H pylori* positive patients than that of *H pylori* negative patients. Also hydrophobicity measurement data[7] totally argue against the conclusions reached by Markesich *et al.*

There may be several reasons for the different results obtained by Markesich *et al.* The most important are the lack of control for the peptic activity during the processing and the mode of mucus gel sample preparation for the measurements. As such, the 'mucus gel' samples obtained after the centrifugation in both cases represent only the undegraded mucus, as the degraded one would remain in the supernatant. Hence, the viscosity measurements on the materials prepared from patients with and without *H pylori* infection were carried out only on the undegraded 'insoluble mucus gel'. No reference of any sort is made as to the sample concentration (for example, mg protein/ml). The conditions used for 'mucus gel preparation' (for example, centrifugation 10 000 rpm for 15 min) would result in the insoluble fraction consisting of some undegraded mucus, cell debris, and other particulate matter. Consequently, this kind of material would give very high and erroneous viscosity values, as is the case in this study.

We would also like to point out that the study by Markesich *et al*, in contrast with their contention, represents typical in vitro experiments, and unless a method is devised to take the viscosity measurements directly in the stomach, the claim is not valid.

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Reply

EDITOR,—Slomiany and coworkers not surprisingly are unhappy with the results of our evaluation of the viscosity of gastric mucus gel. We believe that we studied the real stuff. I am surprised that the objectors did not comment on, or attempt to deal with, the fact that data were internally consistent and changed with treatment. We apologise because upon re-reading our manuscript it could be read that we implied that previous workers always purchased mucus in a bottle (*Gut* 1995; **36:** 327–9). We do contend that mucus obtained by instilling buffer into freshly dissected pig stomachs followed by filtering through a 0·45 μm filter, raising the pH to 9 for 30 minutes, dialysing it against distilled water, and then lyophilising it may not be comparable to the fresh mucus gel obtained from human beings.[1]

Our goal was to try to test reality. Our detractors believed that we failed in that objective. Their approach has been to identify reality through careful reconstructive experiments using sophisticated biochemical techniques. Slomiany and coworkers are internationally recognised authorities in mucus biochemistry; our study was of mucus biology. Since our paper was published we have even found an earlier reference with similar findings that also give references to even earlier work.[2] Thus, as is often the case, we were not the first but instead extend previous findings in populations whose *H pylori* status is defined. Time will tell whether God has been unkind to them or to Slomiany and coworker.

Finally, we would like to draw attention to the fact that the gastric mucus hydrophobicity story may be somewhat more complicated than it first seemed.[3 4]

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Copper associated childhood cirrhosis

EDITOR,—The report of copper associated childhood cirrhosis by Horslen *et al (Gut* 1994; **35:** 1497–500) is of considerable interest. But I take issue with one small point.

The authors cite a case reported by Bartók *et al* in 1971[1] as an instance of idiopathic hepatic copper toxicosis. A glance at the

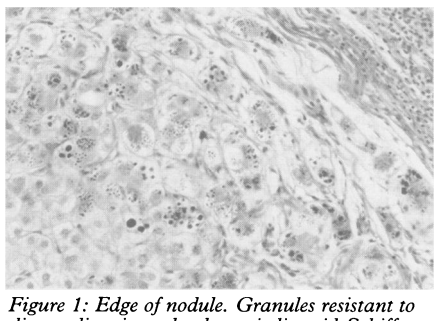

Figure 1: Edge of nodule. Granules resistant to distase digestion take the periodic acid-Schiff stain.

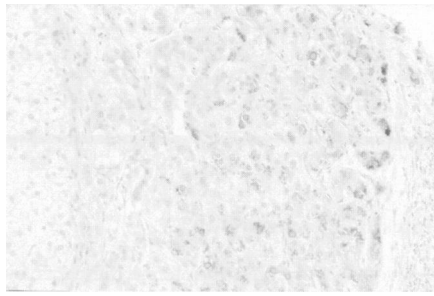

Figure 2: The granules mark for α_1 antitrypsin by immunostain.

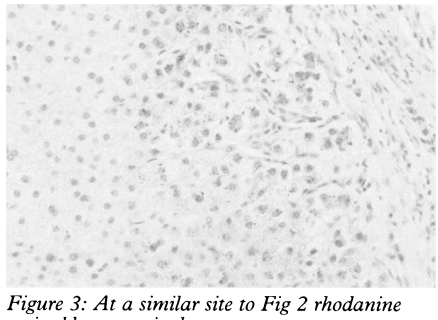

Figure 3: At a similar site to Fig 2 rhodanine stainable copper is shown.

original publication arouses doubts whether another diagnosis might be correct. Early last year, through the courtesy of Professor J Ormos of the University of Szeged, I was sent a block of liver tissue from that case (see Figures). Stainable copper in large amounts was present, but other findings – using both immunohistochemical and standard techniques – confirmed my suspicion that the underlying disease was $\alpha_1$antitrypsin deficiency. This diagnosis had been recognised and published in the Hungarian literature[2]; I was not aware of this when I asked for tissue.

In any event, it is time that this patient's case no longer be invoked as an instance of non-Indian copper associated childhood cirrhosis or idiopathic hepatic copper toxicosis. Given parental consanguinity,[1 2] she of course may have had both $\alpha_1$antitrypsin deficiency and another defect involving copper handling, but this is speculation.

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Reply

EDITOR,—We are extremely grateful to Dr Knisely for this further information on the case reported by Bartók *et al*,[1] and we agree that this case should not now be considered as a case of copper associated childhood cirrhosis (CACC). The postmortem liver copper content in this child, now known to have $\alpha_1$antitrypsin deficiency, was between 2947 and 3564 μg/g dry weight (normal <50 μg/g dry weight) thus emphasising the need to distinguish between secondary and primary causes of copper accumulation.

Evidence is now available to suggest that the remaining cases categorised as CACC type II do not all represent a single condition. The child described by Adamson *et al*[2] has