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.1-3* Studies with gastric juice performed by others, however, showed lower viscosity values for H pylori positive patients than that of H pylori negative patients. Also hydrophobicity measurement data 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 most important for the preparation for the measurements. As such, the ‘mucus gel’ samples obtained after the centrifugation in both cases represent only the degraded 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 degraded insoluble mucus gel. No reference of any sort was made in 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 degraded mucin, 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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Re:—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 as an instance of idiopathic hepatic copper toxicity. A glance at the 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 α1 antitrypsin deficiency. This diagnosis had been recognised and published in the Hungarian literature; I was not aware of this when I asked for the 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 she of course may have had both α1 antitrypsin deficiency and another defect involving copper handling, but this is speculation.

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Reply—We are extremely grateful to Dr Knisely for this further information on the case reported by Bartók et al, 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 α1 antitrypsin deficiency, was between 2947 and 3564 μg/dry weight (normal <50 μg/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 has...