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.\(^1\) Studies with gastric juice performed by others,\(^1\) 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 mode of 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 was made as in populations whose \(H\) pylori status is defined. Time will tell whether God has been kind to them or to Slomiany and coworkers.

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.\(^1\)

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. This is because God has been kind to them or to Slomiany and coworkers.

**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 as an instance of idiopathic hepatic copper toxicosis. 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 \(\alpha\)-antitrypsin deficiency. This diagnosis had been recognised and published in the Hungarian literature;\(^3\) 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,\(^3\) she of course may have had both \(\alpha\)-antitrypsin deficiency and another defect involving copper handling, but this is speculation.

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