Letters

to accept that the rather even distribution of ICAM-1 below the follicle associated epithelium, as reported by Fujimura and Kihara, should have anything to do with the numerically and phenotypically heterogeneous distribution of lymphocytes in this epithelium.

The authors furthermore discuss extensively the nature of membrane cells; without reservation it is claimed that these cells express MHC class II molecules and therefore may be present luminal antigens to ICAM-1 cells. This area is quite controversial, however, and the first study on class II expression in rat Peyer's patches reported that the complete follicle associated epithelium is negative.\(^{13}\) This has been contradicted subsequently but we found human membrane cells to be negative for HLA-DR compared with the strongly positive remaining follicle associated epithelium.\(^{14}\) \(^{15}\) The antigen presenting capacity of membrane cells is therefore questionable although they are probably to some extent able to degrade foreign material as suggested by their lysosome like structures\(^{16}\) and cathepsin E expression.\(^{17}\) We have recently proposed that membrane cells might provide an opportunity for juxtaposed B cells to present partially processed luminal antigens to CD4\(^+\) memory T cells, thereby promoting diversification of mucosal immune responses.

In view of this immunobiological complexity of gut associated lymphoid tissue we feel that it is too speculative when Fujimura and Kihara on the basis of their findings in rat Peyer's patches, claiming high ICAM-1 as potential treatment for inflammatory bowel disease in the future.

P BRANDTZÆG
I N FARSTAD
Laboratory for Immunohistochemistry,
Institute of Pathology (LIIPAT),
The National Hospital, Rikshospitalet,
N-0027 Oslo, Norway

6 Kvale D, Brandtzæg P. Constitutive and cytokine-induced expression of HLA molecules, secretory component (SC), and ICAM-1 are modulated by butyrate in the colonic epithelial cell line HT-29. Gut 1995; 36: 837–42.

Ménétreïer's disease

EDITOR.—We read with interest the case report by Bayerdorffer et al showing that Helicobacter pylori is a potential cause of Ménétreïer's disease (Gut 1994; 35: 701–4).

We urge authors and editors not to use the term Ménétreïer's disease as a generic designation for conditions associated with the gut associated lymphoid tissue express HLA-DR. Further investigations are also required to find the mechanism regulating lymphocyte migration into the follicle associated epithelium of human gut associated lymphoid tissue.

Y FUJIMURA

T KIHARA

Division of Gastroenterology,
Department of Medicine,
Kansai University Medical School,
577 Matsushima, Koyashi 701-01, Japan

1 Farstad IN, Halstensen TS, Fausa O, Brandtzæg P. Heterogeneity of M cell associated B and T cells in human Peyer's patches. Immunology 1994; 83: 457–64.

Gut first published as 10.1136/gut.36.6.945-a on 1 June 1995. Downloaded from http://gut.bmj.com/ on May 12, 2022 by guest. Protected by copyright.
Reply

EditoR,—We agree with Hendrix and Yardley that Helicobacter pylori infection with hypertrophic gastric folds and a protein loss syndrome should in future be termed merely 'Helicobacter pylori induced hypertrophic gastri-

tis' and not Ménetrier's disease; and further, that on detection of hypertrophic gastric folds a search should be carried out for other possible infectious causes or underlying diseases with hypertrophic folds, or both. In the past, this has not been done, either in case reports or reviews of large numbers of cases of Ménetrier's disease. In particular, most publications failed to evaluate inflammatory infiltration as an exclusion criterion for Ménetrier's disease. Thus, for example, Schindler1 described Ménetrier's disease in his article on 'hypertrophic glandular gastritis', and Scharschmidt2 in his review, expressly emphasised that 'round cell infiltration of the lamina propria was also frequently noted and was very prominent in some cases' of Ménetrier's disease.

The suggestion that only massive foveal hypertrophy and absolute absence of gastritis could be termed Ménetrier's disease, was made comparatively late by Appelman,3 but was often ignored. The cases of Ménetrier's disease reported from the Mayo Clinic were, for example, accepted as being Ménetrier's disease when 'a hypertrophic gastropathy with hypoproteinaemia' presented. Indeed, in the first synopsis of 43 patients, a 'systematic histologic study of the gastric mucosa' was even deliberately not undertaken.4 In the latest review of the Mayo Clinic cases, it was then shown that a comparatively large percentage of these patients had a 'lymphocytic gastritis', as well as loss of protein,5 and recall that the former 'gastritis en nappe'6 has more recently been shown to be a special form of lymphocytic gastritis,7 and that this gastri-

tis can also include appreciable foveal hypertrophy. This also takes into account the fact that Ménetrier made reference to 'polyanéologies gastriques', which he further subdivided into 'polyanéologies polypeux' and 'polyanéologies en nappes', there are justifi-

able doubts as to whether Ménetrier's disease as such ever was an individual entity in the first place.

The description 'polyanéodies polypeux' given by Ménetrier himself, did not include macroscopically evident hypertrophic gastric folds, and his histological description fits that of present day 'hyperplastic' polyps, which the drawing of the macroscopic findings in Ménetrier's publication also seems to confirm, as it shows multiple polyps in the antrum and corpus, but no hypertrophic folds.

Also, in these polyps — in contrast with the interpretation of Hendrix and Yardley — Ménetrier described an inflammatory infiltrate ('infiltre de cellules migratrices'). And also in the case of the 'polyanéodies en nappes', Ménetrier noted 'des phénomènes de gastritis chronique'.

When it is further considered that the histological technology of 1888 was, of necessity, inferior to that of the present day, that the descriptions were based on necropsy find-

ing, and that we now know that 'polyanéodies en nappes' changes in the corpus and fundus are typical of lymphocytic gastritis, doubts that Ménetrier had ever really described an individual entity or that the cases of Ménetrier's disease published over the past decades really represented an individual entity, are considerably strengthened.

Our case history was intended merely to draw attention to the fact that when the clinical and endoscopic pictures suggest Ménetrier's disease, we should in future give more consideration to the possibility that the patient has a Helicobacter pylori induced gastric gastritis, which can then be cured by eradication of the H pylori.

M STOLTE

Institut des Pathologie,
Bayreuth, Germany

E BAYERDORFER

Medical Department II,
Maximilian Gastroheiders,
University of Munich,
Germany

1 Schindler O. On hypertrophic glandular gastritis, hypertrophic gastritis, and parietal cell mass. Gastroenterology 1963; 45: 77-82.

2 Scharschmidt BF. The natural history of hyper-


3 Appelman HD. Localized and extensive exten-


Polyethylene glycol (PEG) as a marker of small intestinal permeability

EditoR,—We were very interested to read the paper by Peeters et al who, in an attempt to standardise intestinal permeability test conditions, have studied the effects of probe solution composition and osmolality on the absorption of radio-labelled PEG and PEG 400 (MW 280-634) across the small intestine (Gut 1994; 35: 1404-8). We endorse the need for standardisation in the conduct of small intestinal permeability tests and in a recent investigation of the effects of hydration status on urinary probe recovery in normal subjects we found that fluid hydration had a considerable effect on the recovery of PEG (MW range 280 to 1100). In contrast, mannitol and lactulose recovery were not affected.1 Peeters et al expressed their results as 1CrEDTA/PEG 400 ratios and found that the permeation of both markers was significantly reduced in the presence of lactulose and mannitol. We wonder whether differences in the hydration status of their subjects might explain their results.

The dependence of PEG permeation on the state of subjects' hydration at the time of the test is just one problem facing those determined to use PEG as a marker of small intestinal permeability. PEG 400 is absorbed in vivo and the rate of absorption is greater than other probes of similar molecular weight such as lactulose (MW 342)2 and the absorption of PEG is reduced in conditions associated with flat mucosa whereas that of lactulose is increased.3 These anomalies have never been fully explained by the proponents of PEG as a passive permeability marker. In their recent paper, Peeters et al attempt to explain the unusually avid permeation of PEG 400 in comparison with other probes of similar size on the basis of theoretical con-

considerations regarding the long, thin shape of PEG. It has been proposed that PEG has access to absorptive areas inaccessible to other molecules of comparable size because of its shape.3 However, this theory has not stood up to experimental analysis.4 Furthermore the concept of PEG as a long, thin molecule does not take into account that the effective state of PEG may in fact be much larger because of hydrogen bonding by the available oxygen atoms along the backbone of the molecule. Another possible explanation for the avid permeation of PEG across cell walls, because PEG is lipid soluble, it interacts with the phospholipid bilayer of the cell wall.5 In a recent in vitro study comparing PEG with lactulose and mannitol we were able to show that PEG 400 (in common with other molecules) could traverse lipid barriers with comparative ease.6 Another problem with the use of PEG as a marker of intestinal permea-

bility is the wide variability in the recovery of PEG 500.7 This variability suggests that the smallest polymers may have a larger volume of distribution in the body, a suggestion that was supported by our finding that the recovery of the smallest molecules in a range of PEGs is comparatively delayed in normal subjects, suggesting a slow wash out of these molecules from the extravascular space into the circulation.8

Given all these problems with regard to the permeation of PEG across the intestine and its subsequent urinary recovery we suggest that the time has probably come for this molecule to finally be abandoned as a marker of passive small intestinal permeability in vivo.

T H IQBAL

M A COX

K M WILLS

B T COOPER
Gastroenterology Unit,
City Hospital NHS Trust,
Dudley Road,
Birmingham B18 7QH


2 Uekaham SO, Cooper BT. Small intestinal permea-


Gut first published as 10.1136/gut.36.6.945-a on 1 June 1995. Downloaded from http://gut.bmj.com/ on May 12, 2022 by guest. Protected by copyright.