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 above-mentioned comments raise concerns about the nature of membrane cells; without reservation it is claimed that these cells express MHC class II molecules and therefore may be able to present luminal antigens to T cells. This area is quite controversial, therefore, and the first study on class II expression in rat Peyer's patches reported that the complete follicle associated epithelium is negative.12 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.13,14 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 structures16 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 it is too speculative when Fujimura and Kihara on the basis of their findings in rat Peyer's patches and blocking of ICAM-1 as a potential treatment for inflammatory bowel disease in the future.


Reply

EDITOR,—We are grateful to Drs Brandtzæg and Farstad for drawing our attention to their paper. They pointed out that ICAM-1 could not be shown in human epithelium of the normal or diseased human gut (unpublished findings), but could in human adenocarcinoma cell line HT-29. Recently, we confirmed ICAM-1 expression on subepithelial fibroblasts, high endothelial venules, and migrating cells in rat Peyer's patches, but not in humans. We do not know how to explain this discrepancy and suppose that the difference in finding may be related to different species or the property of the monoclonal antibodies. We agree that it was too speculative when we, on the basis of our findings in rat Peyer's patches, suggested blocking of ICAM-1 as a potential treatment for inflammatory bowel disease in the future.

We also found very interesting their description of the heterogeneity of membrane cell associated B and T cells in human Peyer's patches—that is, that the B cells are strikingly heterogeneous with characteristics of both mantle (sIgD- sIgM+) and marginal (sIgD- sIgM+) zone lymphocytes, and that some lymphocytes in membrane cell pockets among follicle associated epithelium showed Ki-67 and CD45RO weakly.1 They, therefore, suggested that B lymphocytes can proliferate and concentrate typically in the membrane cell pockets. These findings and suggestions are very impressive and we agree with their conclusions.

We agree also that it is still controversial whether membrane cells are sites of 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.

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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.

Ménétrier's disease

EDITOR,—We read with interest the case report by Bayerdorffer et al showing Helicobacter pylori is a potential cause of Ménétrier's disease (Gut 1994; 35: 701–4). The findings in their patient showed clearly that H pylori gastritis can present as hypertrophic gastritis combined with protein loss and that eradication of the H pylori infection can lead to rapid disappearance of hypertrophic gastritis and restoration of normal gastric mucosa. We disagree, however, with the authors' presumption that Ménétrier's disease and hypertrophic gastropathy are synonymous. There are many reasons for believing that equating the two terms is ill advised. (1) A variety of conditions can cause enlarged gastric folds. In addition to the true ‘hyperplastic gastropathies’ Ménétrier's disease and Zollinger-Ellison syndrome, a number of other conditions of gastric folds are seen in hypertrophic gastritis associated with various infections, including H pylori, cytomegalovirus, histoplasmosis, and syphilis, and in miscellaneous other diseases such as lymphomas, sarcoidosis, allergic (eosinophilic) gastritis, and Cronkhite-Canada syndrome. (2) While increased gastric protein loss can be found occasionally in many disorders that are associated with enlarged gastric folds, protein loss is not a universal feature of any of these disorders. It is typically lacking in Zollinger-Ellison syndrome, and its reported occurrence in Ménétrier's disease is variable. (3) The cases described in 1888 by Ménétrier as ‘polype- nome en nappe’ had as their cardinal feature exuberant proliferation of gastric mucous cells. This is a distinct entity that has been appropriately termed ‘massive foveolar hyperplasia’.1,2 Furthermore, Ménétrier’s descriptions and illustrations do not suggest that chronic gastritis, the hallmark of H pylori gastritis, was present in Ménétrier's disease, is probably a manifestation of H pylori gastritis with large folds.4 We urge authors and editors not to use the term Ménétrier’s disease as a generic designation for any and all conditions associated with enlarged rugae. The eponym should be limited to those rare cases that fulfil Ménétrier’s original description of massive foveolar hyperplasia without gastritis. This approach is essential from a nosologic and patient treatment standpoint if the varied aetiologies and resulting treatment implications of hypertrophic gastropathy are to be meaningfully addressed.

Bayerdorffer et al we would have preferred to see it termed simply H pylori associated hypertrophic gastritis.