the data are not clear-cut. There is no evidence that double-stranded RNA is an intermediate in the case of transcription and replication of negative-strand viruses (D. Kolakofsky, personal communication); rather, RNA is complexed to nucleocapsid protein, which probably prevents formation of double-stranded RNA.

Double-stranded RNA is nevertheless formed as a dead-end side product of Qβ RNA replication, presumably when there is a failure of the mechanism that separates or keeps apart plus and minus strands during synthesis[2]. In the case of eukaryotic minus-strand viruses it may be a similar rare event that gives rise to a double-stranded RNA that can be taken apart by the modifying/unwinding enzyme of Bass and Weintraub[4] and give rise to hypermutated RNA[5].

Were double-stranded RNA an obligatory intermediate of eukaryotic viral RNA replication, one would expect hypermutational events to be frequent, rather than very rare[5]. Maybe it is the avoidance of double-stranded RNA intermediates that enables RNA viruses to circumvent lethal hypermutation in eukaryotic cells.

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Human T-cell receptor expression

SIR—Janeway, in his recent News and Views article[1], stated that intestinal epithelial lymphocytes "in both chickens and man are predominantly CD8⁺ T cells with γ/δ receptors". In our immunohistochemical studies of human intraepithelial lymphocytes, we also find that most intraepithelial lymphocytes express CD8, although approximately 20% of intraepithelial lymphocytes express neither CD4 nor CD8.

Using an antibody against the δ-chain of the T-cell receptor (δTCS1, T Cell Sciences Inc., Cambridge, Massachusetts) however, we have failed to demonstrate a predominance of δ-chain-positive lymphocytes in normal gut epithelium. We are unable to detect any δ-chain-positive cells in human fetal ileum at 20 weeks' gestation, when intraepithelial lymphocytes expressing CD3 are present[2]. We find that only 2 per cent of intraepithelial lymphocytes in normal adult jejunum, increasing to approximately 33 per cent in jejunum from children with newly diagnosed coeliac disease, express this form of the T-cell receptor. These findings suggest that accumulation of intraepithelial lymphocytes expressing the δ-chain of the T-cell receptor is antigen driven, but that in normal, immunologically stable intestine, the proportion expressing the γ/δ heterodimer is small.

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JANEWAY REPLIES—Spencer and Isaacson's statement that human intestinal epithelial lymphocytes may not be enriched in γ/δ T cells is further evidence of the need to correlate anatomy to structure and function. My statement regarding the presence of such cells in man[1] was based on results using the same antibody carried out by Bucy in Cooper's laboratory, and reported at the last FASEB conference in Las Vegas, Nevada.

I have since learned that another antibody of similar specificity also fails to reveal dominance of such T cells in gut epithelium in man (Groh *et al.*, personal communication). However, we now have evidence that intestinal epithelial lymphocytes of mice are using a V_γ:V_δ different from that found in any other tissue, including skin. Thus, one must be sure that the reagent used will react with all T cells bearing γ/δ receptors before such conclusions are validated. It is also possible that the results in the mouse reflect the early age at which such cells are collected. The γ/δ receptors in the gut and skin of the mouse are of a form found only very early in ontogeny in the thymus, and seem to rapidly seed these unique peripheral sites. The decline in such cells cited by Spencer and Isaacson may reflect this ontogenetic history.

However, the data of Spencer and Isaacson, and of Groh *et al.*, could reflect a major interspecies difference. If this last hypothesis is correct, would it be too far-fetched to speculate that the reason humans have spontaneous carcinomas, whereas mice seem to have sarcomas and lymphomas as their main tumour types, is that humans have a defect in the disposition of γ/δ T cells in their epithelia, thus allowing transformed epithelial cells to arise and proliferate sufficiently to invade and cause disease?

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Taxonomic stability

SIR—Crisp and Fogg[1] correctly point out that changes in names for taxonomic reasons, which arise from differing opinions on classifications or new data, are tiresome. Nevertheless, when changes arise from a more thorough understanding of evolutionary relationships they must be accepted as part of the development of science, as stressed by Newman[2].

The current International Union of Biological Sciences *Lists of Names in* Current Use[3] initiative aims to limit changes arising from the application of International Codes[4,5], rather than as a result of new scientific research. The most effective tool for restricting irresponsible taxonomic changes is peer pressure. Progress in that direction is starting to be made in the Ascomycotina, the largest class of fungi (6,100 generic names; 29,000 species). An interactive eclectic approach to the production of a generally accepted system for ranks from genus to order was initiated in 1982, based on a twice-yearly publication of this institute and the University of Umeå, *Systema Ascomycetum*, which reports changes in classification and reactions to them. Depending on worldwide peer reaction, changes are incorporated into an annually revised "Outline of the ascomycetes"[6], providing recommended dispositions of generic and higher ranks. Users have an annually updated listing to which they can refer. Parallel publications could clearly be initiated for other groups of organisms, were the necessary resources made available.

At the species level, the International Commission on the Taxonomy of Fungi (ICTF) produces a series "Name changes in fungi of microbiological, industrial and medical importance"[7]. The reasons for changes proposed in the naming of these fungi are reviewed by an international panel of 11 mycologists, and their views are embodied in this series. Again, this could be adopted on a wider front.

International peer reactions are the most likely prospect for restricting unwelcome changes of the type Crisp and Fogg abhore. In the longer term, improvements in the standards of taxonomic practice, such as the ICTF "Code of Practice for Systematic Mycologists"[8,9], are required.

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