eclipses survive, they refer of necessity to total elipses. This is not necessarily true, as was already pointed out by Newton[3]. Furthermore, I recently presented a solar eclipse record which can serve as a counter-example, namely the only account of a solar eclipse from the *Chronographia*, a fourteenth century historical work. It is the only record of a solar eclipse in that work, but nevertheless it is with certainty a record of a partial eclipse[4,5]. It is therefore possible that the Ugarit record also refers to a partial eclipse. If so, dating the record is not feasible in view of the larger number of possibilities. In conclusion, the record of a solar eclipse of the second millennium is still not dated with certainty and conclusions drawn from the dating should be met with some reservation.

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Epithelial homing of γδ T cells?

SIR — Only one or two per cent of T lymphocytes in systemic lymphoid organs or blood, of mice and men, express the γδ T-cell receptor (TCR) for antigen; this minor fraction mainly consists of cells that carry the CD3 antigen but not the CD4 or CD8 antigens and so are termed $CD3^+CD4^-CD8^-$ or 'double-negative' [1]. It was therefore remarkable when intra-epithelial lymphocytes (IEL) isolated from the murine gut were reported to be exclusively $CD3^+CD4^-CD8^+$ and, by immunoprecipitation, seemed to bear mainly γδTCR (refs 2 and 3). Similar results were obtained by immunohistochemistry for avian and apparently also for human IEL[4]. A tissue-specific distribution of TCR subtypes has now been proposed[5], but has received a sceptical response[6,7].

We tested this theory with BMA031, an αβ framework antibody[8], and 11F2 that reacts with all types of γδTCR (ref. 9). In immunocytochemical tests on a $CD3^+$ double negative γδ T-cell line, these monoclonal antibodies behaved correctly; but they performed unsatisfactorily on cryostat sections. Labelling of viable T cells was therefore performed by permeation of fresh jejunal biopsy specimens with these two antibodies and with antibodies to CD8 and CD4 (Dakopatts)[10]. Membrane expression of αβTCR was seen abundantly in the lamina propria and also on a variable number of IEL (Fig. 1*a*). The proportions of $CD8^+$ and αβTCR IEL obtained in the seven normal cases were well correlated (Pearson's $r=0.865$). To compensate for variable antibody diffusion, the results for αβTCR, γδTCR, and $CD4^+$ IEL were adjusted by a factor derived from the corresponding $CD8^+$ to $CD3^+$ ratio, taking 90% as the average 'true' value[11]. The median αβTCR fraction of $CD3^+$ IEL was thus estimated to be 92% (64–100%, $n=3$) with labelling at 37 °C for two hours and 70% (41–100%, $n=4$) at 4 °C for two to eight hours. Conversely, the proportion of γδTCR was usually below 8% and that of $CD4^+$ IEL below 13% (median, 9%).

The figures obtained for three coeliac disease specimens indicated that there might be a relative increase of γδTCR (1–16%) compared with the αβTCR (median, 48%) and $CD4^+$ (median, 9%) IEL. Such a disease-associated change (Fig. 1*b*) could reflect the increased intra-epithelial $CD3^+$ double-negative subset recently identified in this disease[12].

In conclusion, we have shown that most human intestinal IEL express the αβTCR. Moreover, almost half of the $CD3^+$ IEL were found to be positive for the UCHL1/CD45 molecule[10]; this implies that they are probably $CD3^+CD8^+\alpha\beta$ memory T cells.

We have recently used another set of monoclonal antibodies, namely βF1 and TCRδ1 from T Cell Sciences (Cambridge, Mass.), and have confirmed by immunohistochemistry on cryostat sections that αβTCR cells are strikingly predominant among human IEL, and that the γδ TCR fraction is indeed increased (from a normal median of 2% to 22% in treated or untreated coeliac disease). The CD8 expression of this fraction is decreased (from 25% to 10%) and CD4 expression is not seen. Also notable is the finding that the intestinal $\gamma\delta$ TCR^+ cells are mainly located in the epithelium, both normally and in coeliac disease (T.S. Halstensen *et al.*, *Scand. J. Immun.*; in the press).

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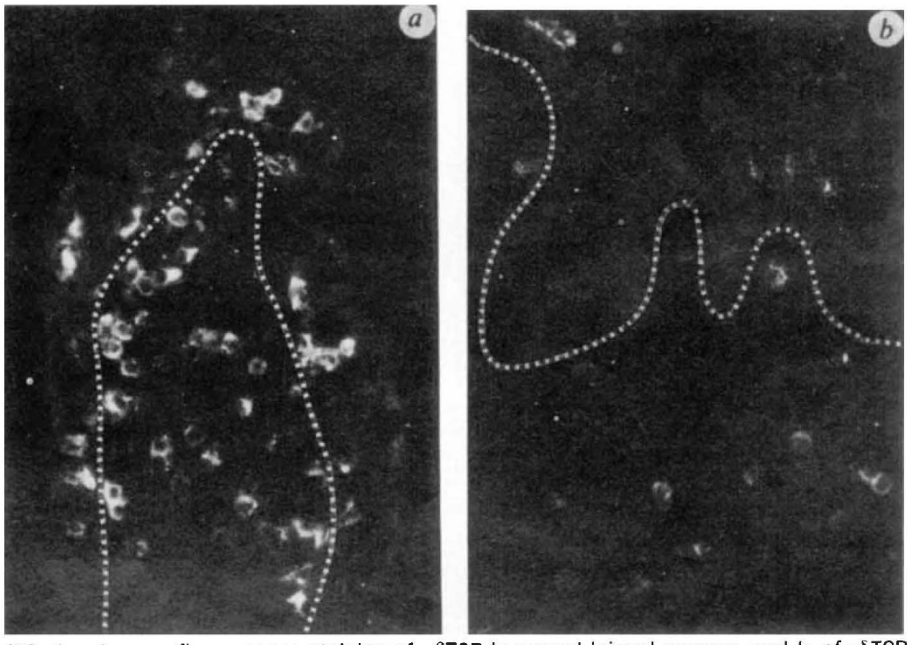

FIG. 1 *a*, Immunofluorescence staining of αβTCR in normal jejunal mucosa, and *b*, of γδTCR in coeliac disease mucosa with villous atrophy after permeation of the tissue specimens with monoclonal antibodies BMA031 (Behring, Marburg, FRG) and 11F2 (J. Borst), respectively. Mucosal surface at the top. Epithelial basement membrane indicated by broken line (x400).

SIR—Regarding the controversy that has already surfaced in *Scientific Correspondence* on the frequency of γδ T cells in human intestinal epithelium[7,13] my collaborators, Chen-lo Chen and Max Cooper, and I have investigated the tissue localization of cells expressing the δ-chain with the monoclonal antibody to TCRδ1 of Michael Brenner. This appears to recognize a constant region of the TCRδ chain[14], unlike δTCS1 used by Spencer and Isaacson[7], which recognizes a subset of the γδ T cells[14]. We found that 80% of γδ T cells in the intestinal mucosa are localized in the epithelium while the remaining 20% are in the lamina propria[15]. Nevertheless, these cells comprise a minor subset (about 7%) of the intraepithelial $CD3^+$ cells. In the chicken[4], we observed the same γδ T cells homing into the intestinal epithelium but it is noteworthy that only half of the epithelial $CD3^+$ cells express the γδTCR. A majority of these are $CD8^+$, and none express CD4.

The preferential tissue localization