correlation between intensity of magnetisation and climatic indicators means that the latter correlate with geomagnetic field strength.

Amerigian then goes on to show that the reported correlations between palaeomagnetic inclination and climatic indicators can result from variable inclination errors introduced by bottom-current velocity changes which are also climatically controlled. He does recognise, however, that his various mechanisms are only valid if the magnetisations of sediments are depositional rather than post-depositional, for climatically controlled depositional processes can obviously only be relevant to the observed correlations as long as sediment magnetisations arise at the time the sediment is formed. There are known to be several ways in which post-depositional magnetisations can be, and probably are, produced, although it is by no means clear that such processes are universal. In any case, Amerigian's aim seems to be less to push particular explanations or combinations of explanations, for the observed correlations than simply to demonstrate that the correlations need not be direct.

Where Amerigian may be on less certain ground is in his treatment of correlations between directly observed geomagnetic field changes and climatic variations. Since their work on sedimentary cores, for example, Wollin *et al.* (*Nature*, **242**, 34; 1973) have reported just such correlations, but Amerigian dismisses them because of what he takes to be "unsatisfactory" data selection. Whether or not he is justified in this particular instance, the fact is that other workers are also finding relationships between magnetism and climate. It is by no means clear that these will be so easily refuted.

PETER J. SMITH

Lymphocytes at war

IT is a precept of many immunologists that the primary role of the vertebrate immune response is defensive, enabling the responding organism to resist attack by such foreign agencies as bacteria and viruses. Although there are objections to this notion—for example that invertebrates do not have such specific immune responses and yet they seem to defend themselves well—attack and defence have become so firmly rooted in the thinking of immunologists that many of their present-day analytical methods could be described as war games. One of the games with the most immediate impact is that which is fought *in vitro* between two genetically and thus antigenically distinct cell populations. It is supposed that the outcome of this battle in some way reflects the manner in which foreign mammalian cells are dealt with in the living animal after tissue or organ transplantation. There is also the idea that tumours are antigenically distinctive and thus the various kinds of stimulation which can be shown *in vitro* are a manifestation of an anti-tumour surveillance mechanism. Like many war games the moves of those played by immunologists are complex and irritatingly abstruse to the uninitiated. Three recent communications in *Nature* illustrate the genre.

Lafferty and his colleagues Misko and Cooley (*Nature*, **249**, 275–276; 1974) lay down the basic rule of their game in their first sentence. In translation it says that the response to a particular set of transplantation antigens *in vitro* is sometimes enhanced if there is a second and different set of antigens available to respond: two adversaries are better than one. Lafferty *et al.* cultured the cells from the lymph nodes of C57BL/6J mice with γ-irradiated BALB/c spleen cells. This is said to be a one-way mixed lymphocyte reaction in which the outcome is simplified by inactivating the capacity of one of cell populations to respond but leaving its stimulatory property apparently intact. The severity of the reaction can be measured either by incorporation of tritiated thymidine into the cultures at an appropriate time or by subculture of the responder cells onto a ^{51}Cr-labelled tumour cell population which has some of the same surface antigens as the BALB/c stimulator cells (H-2^d).

Lafferty *et al.* go on to show that although γ-irradiated cells are adequate stimulators, ultraviolet-irradiated cells are not. It seems that ultraviolet-irradiated cells still have the relevant antigens as they are effective absorbers of the appropriate anti-H-2 antisera. If, however, to cultures containing responder C57BL/6J lymph node cells (H-2) and (poor) stimulator ultraviolet-irradiated BALB/c spleen cells (H-2^k) is added a γ-irradiated CBA spleen cell population (H-2^k), then the C57BL cells respond vigorously and generate anti-H-2^d cytotoxic cells. Lafferty and his colleagues think that their experiments raise the possibility that "the antigenic differences between donor and host may not constitute a major barrier to all transplantation" but that if the graft contains more effectively antigenic donor lymphocytes then a more vigorous response against the antigens of the major portion of the graft will ensue.

Dennert's experiments (*Nature*, **249**, 358–360; 1974) are like those of Lafferty *et al.* in that the DBA/2J mastocytoma (H-2^d) is again a target cell for attacking lymphocytes. Dennert injected the tumour into C57BL/6J mice and then assayed for cytotoxic cells in their spleens 9 days later. He found that if he used fresh tumour cells to immunise, then cytotoxicity developed but if he used cells fixed with glutaraldehyde or formaldehyde little cytotoxicity was discovered. Dennert then went on to estimate the immunological capacity of the spleens of the variously immunised C57BL mice for their 'helper' capacity by incubating them *in vitro* with trinitrophenyl (TNP)-coupled tumour cells and subsequently measuring the anti-TNP response by a plaque assay. If the spleen cells are immune anti the tumour cells then, because the tumour cells are acting as the carrier for the TNP hapten in the culture, an anti-hapten response should follow. Paradoxically the spleen from mice immunised with fresh tumour cells, which had in the other tests shown such a vigorous cytotoxic response, had little helper activity whereas those from mice immunised with fixed cells had helper activity but little cytotoxicity.

Dennert goes on to show that spleen cells with high cytotoxic capacity are also able to suppress the expression of helper function. These experiments serve to exemplify the intricacy of contemporary cellular immunology and Dennert feels that they suggest strongly that cytotoxic effector cells and helper cells are different 'subsets' of T (thymus-derived) cells in contradiction to the earlier suggestion of Kreth and Williamson (*Nature*, **234**, 454; 1971) that they were the same cells. Categorisation of heterogeneity of T cells is presently popular but it will remain to be seen whether the various putative classes are related to some basic diversity or represent alternative physiological conditions of initially the same cell populations. Thus Dennert's article raises the larger question of whether lymphocytes are heterogeneous before antigenic stimulation or even as a result of it.

The third of the trilogy by Plata and Levy (*Nature*, **249**, 271–274; 1974) draws attention to the fact that although all the effector cells in chromium release assays of cell-mediated cytotoxicity against murine sarcoma virus, induced tumours in C57BL/6 mice seem to be T cells, both T and non-T cells can be detected in the microcytotoxicity assay (Takasugi and Klein, *Transplantation*, **9**, 219; 1970). Furthermore, it proved possible to show that the capacity of T cells to kill as detected by chromium release was not blocked by serum from tumour-bearing animals whereas in the microcytotoxicity assay blocking occurred. Like Dennert, Plata and Levy wish to conclude that they are dealing with two populations of T cells distinguished by their functional capacities *in vitro*.

A. J. S. DAVIES