computer models cannot yet account for present climatic features, never mind their changes, the only practical approach is to define as closely as possible the nature and wavelength of climatic cycles and to compare them with any cyclical phenomenon that may alter climate. Lamb, for example, demonstrated that there was no strong correlation between climatic change and volcanic activity (*Phil. Trans. R. Soc.*, **A266**, 425; 1970). If correlations can be established, causal relationships can be investigated bearing in mind that the two factors could be indirectly linked through some other variable.

But climatic records are of only limited range and difficult to quantify—Tacitus's description of the British climate in 75 BC as "objectionable, with frequent rains and mists, but no extremes of temperature" bears distinct resemblances to the British summer of 1974 AD, but cannot be taken to mean that we are at the same point of a climatic cycle! The only quantitative climatic measure for periods prior to meteorological measurements is that of temperature determined from the ratio of ^{18}O to ^{16}O now trapped in ice on Greenland and Antarctica and in skeletal remains preserved in sediments. This ratio can be measured with a high precision, but both situations raise difficulties about how the ratio should be interpreted as temperature. The snowfall over the Antarctic comprises a large amount of redeposited snow and the oxygen ratio in marine skeletal organisms is strongly affected by changes in salinity as well as by temperature. The oxygen ratios in ice sheets can be dated by short-lived isotopes or simply counting summer/winter layers—usually starting from the commencement of nuclear weapon testing which now provides an easily detectable marker horizon. But increasing compaction with recrystallisation tends to destroy the accuracy of counting and this method can only go back reliably for a few thousand years. Fossil foraminifera and other organisms can however be used to extend temperature determinations well into geological time and can be dated by standard geological methods, thereby allowing the detection of long-term cycles.

What about the factors that may alter climate? As Chappell says on page 199, there has been general acceptance that cyclical changes in the degree of solar radiation received at the Earth's surface occur because of long-term changes in the Earth's orbit. The periodicity of these changes was largely established by Milankovitch in 1938 and, assuming no changes in the solar radiation 'constant', it is possible to determine precisely the changes in the amount of insolation received at the Earth's surface. Calder, on page 216, re-evaluates Milankovitch's hypothesis in the light of revised insolation tables and concludes simply that these orbital perturbations are the first-order control on glaciation. But as Chappell points out, many critics have claimed that the radiation changes associated with these astronomical factors are very small and the direct effect of insolation may be insignificant compared with indirect factors. Chappell has previously shown many earlier correlations to be spurious, but now provides new and revised evidence for a degree of correlation between orbital perturbations, sea-level changes and palaeotemperatures for the last 250,000 years. His conclusion is also that

More putative human tumour viruses

IN this issue of *Nature* (page 247), McGrath, Grant, Soule, Glancy and Rich describe the release from a human breast carcinoma cell line (MCF-7) of virus particles that they believe may be human mammary tumour viruses. The particles have all the standard biophysical and biochemical properties of RNA tumour viruses, and virus-producing cells react specifically in indirect immunofluorescence tests with antiserum against murine mammary tumour virus. These findings support previous reports by Schlom, Axel, Spiegelman and their colleagues and by Moore's laboratory that virus particles related to murine mammary tumour viruses can be detected in human milk and in breast carcinomas. Unfortunately virus production from the MCF-7 cell line is rather poor and fickle, and this has hampered a more thorough analysis of the particles.

The sceptical reader will wonder whether the MCF-7 cells are really human cells and if so whether they are really derived from a breast carcinoma rather than a common laboratory cell, such as HeLa. On these points, McGrath *et al.* stand on reasonably firm ground, for in a previous publication (*J. natn. Cancer Inst.*, **51**, 1409; 1973) Soule, Fazquez, Long, Albert and Brennan characterised the MCF-7 cell line in some detail. MCF-7 cells are derived from cultures of a pleural effusion of a patient with metastatic mammary carcinoma. The subtetraploid cells seem to have a human chromosome pattern, although banding patterns have not been examined; they have a ribosomal RNA profile resembling that of human cells, and they carry human cell surface antigens and human glucose-6-phosphatase isozyme. MCF-7 cells show three features characteristic of mammary gland cells: cytoplasmic receptors for 17β-oestradiol, synthesis of lactalbumin, and the formation in monolayer culture of epithelial 'domes' similar in morphology to those previously described by McGrath in murine mammary tumour cultures. Not all subclones of the MCF-7 cell line release virus particles. There remains the possibility, of course, that although the cells seem to be genuine human mammary cancer cells, the virus may be a laboratory contaminant. But McGrath *et al.* feel that they can distinguish the MCF-7 particles from murine mammary tumour viruses, and there is no relationship at all with murine leukaemia virus.

In a paper published in the November 1 issue of *Nature* (**252**, 78), Lewis, Tannenberg, Smith and Schwartz suggest that an antigen detectable on lymphocyte membranes of systemic lupus erythematosis (SLE) patients is related to a C-type viral antigen. Antiserum prepared against the C-type virus produced by murine plasmacytoma SP 104 reacted in immunofluorescence tests to SLE lymphocyte membranes, but not to lymphocytes of normal subjects. Moreover, the serum of an SLE patient also reacted to SLE lymphocytes and this reactivity was specifically removed by absorption with gradient-purified SP 104 virus. Thus there seems to be a common antigen between SP 104 virus and human SLE cells.

SP 104 cells have a curious history and interesting properties. The plasmacytoma arose following inoculation of cell-free filtrates of canine SLE cells into CAF_1 mice (Lewis *et al.*, *J. clin. Invest.*, **52**, 1893; 1973). Yet the SP 104 virus stock includes a leukaemia virus containing murine gs-1 antigen. The SP 104 cells produce antibody against double-stranded DNA, as is found in SLE patients, and inoculation of SP 104 virus into normal mice or dogs induces the appearance of anti-nuclear antibodies. Thus the canine cell-free extracts and the SP 104 virus induce symptoms resembling SLE in recipient hosts. It is not known whether SP 104 virus contains canine as well as murine components; neither is it known whether the relation of SLE cell membrane antigens to viral antigens is specific to SP 104 virus alone. Nevertheless these studies do implicate a virus in the aeteiology of SLE, and they suggest that the human, canine and experimental murine forms are closely related.

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