suggested that this may be related to a non-linear response. Two different explanation were presented here. C. Nicolis (Institut d'Aeronomic Spatiale, Belgium) presented a very simple model which showed how internal feedbacks could amplify an otherwise weak 11- or 22-year sunspot-related signal. A very different model developed by J. Oerlemans and J. M. Bienfiet (Royal Netherlands Meterological Institute; see also *Nature* **287**, 430; 1980) also produces a 100,000-year periodicity in the climate record, but in this case resulting entirely from internal factors related to isostatic rebound. This interesting result may, paradoxically, *support* the Milankovitch theory. The strength of the 100,000-year periodicity is not constant and almost vanishes about 10^6 years BP. This would be hard to understand if the cause were purely internal, but less so if climatic changes were the result of orbital forcing. The lack of a 100,000-year periodicity in the early climate record may be related to the fact that perenniel sea-ice cover in the Arctic only began around 700,000 years ago (Margolis and Herman *Nature* **286**, 145; 1980). However, it may well be that around 10^6 years BP, the orbital parameters themselves, if more accurately known, have no 100,000-year signal. □

Peat — a resource reassessed

from J. A. Taylor and R. T. Smith

REPORTS at the Sixth International Peat Conference* suggest a need for a fundamental reassessment of the world's peat resources in industrial, agricultural and enviromental terms. The total area of the world's peatlands is now estimated to be about 500m ha, equivalent to an area over half that of the United States. Canada (150m ha, 36%) and the USSR (170m ha, 40%) together monopolize over three quarters of the world's peatlands but there are also extensive tropical peatlands in the lowlands of S. America, Central Africa and parts of SE Asia.

Peat is organic matter derived from the slow decay of plant remains by the action of bacteria and fungi. Decay is finally arrested in the absence of free oxygen and sunlight, normally under conditions of ground or soil waterlogging which may be aided by an accompanying wet and cool climate. Peat may thus accumulate in badly drained basins forming mires (up to 10-m deep in northern latitudes) or thinner blankets in persistent oceanic, montane or tundra conditions. In the tropics peat is normally located in badly drained, riverine and coastal flats but can also occur in wet, cool climates at high altitudes. Tropical peats are usually deeper (up to 20m or more have been recorded) and contain many more macro-remains of vegetation plus mineral matter than their temperate counterparts.

The value of peat as an alternative energy source has perhaps been under-assessed and a broad spectrum of work is now being undertaken to harness its potential more effectively. Canadian authorities estimate that there is more energy potential in their peat deposits than in their forests or their reserves of natural gas. The National Energy Board calculates Canadian peat reserves at 89 billion tons which is equivalent to 500 trillion cubic feet of natural gas. The peat reserves of the US are put at 13 billion tons and the US Department of Energy has recently allocated $2.2 billion for the development of alternative fuels, including peat as a major component. In the USSR peat fuels no less than 76 large electric generating plants. The Republic of Ireland generates one-third of its electricity and one-fifth of its total energy requirement from peat. Finland is in a stage of rapidly advancing technology in this field and it is estimated by 1990 that up to 8 per cent of the national energy requirement will be met from peat. Peat consumption in Europe has reached a point where regional producers cannot meet the demand. In the United Kingdom,

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Genes controlling cell proliferation

from Paul Nurse

AN attractive hypothesis for the control of eukaryotic cell proliferation is that it is regulated at a particular point during the G_1 period before the initiation of DNA replication. This G_1 control has been called the 'restriction point' in mammalian cells (Pardee, A. B. *Proc. natn. Acad. Sci. U.S.A.* **71**, 1286; 1974) and 'start' in the budding yeast *Saccaromyces cerevisiae* (Hartwell, L. H. *Bact. Revs.* **38**, 164; 1974; *J. Cell Biol*, **77**, 627; 1978). Cells made quiescent in a variety of ways become blocked at this G_1 control point, and cells shifted to conditions favouring proliferation have to pass this point before they become committed to the mitotic cell cycle. The control has been investigated genetically in *S. cerevisiae* by Hartwell and his colleagues, who have identified a gene *cdc* 28 whose function is required before 'start' can be traversed. These workers have suggested that before a cell traverses 'start', it monitors various conditions such as cell size, nutrient level, and the presence of conditions promoting conjugation and sporulation. If these conditions are satisfactory for cell proliferation, the cell begins a new mitotic cell cycle, but if they are not, the cell either becomes quiescent and enters stationary phase, or differentiates and undergoes conjugation or sporulation.

A recent study that has extended this genetic analysis in *S. cerevisiae* is described by Sudbery, Goodey and Carter in the current issue of *Nature* (p 401). They have isolated a novel series of mutants defining two *whi* genes, which undergo bud emergence and cell division at a reduced cell size compared with wild type. The *whi* 1 mutants show a reduced cell size both during exponential growth and in stationary phase, whilst the *whi* 2 mutation has a major effect only in stationary phase. When these mutants are made quiescent by deprivation of nutrients, they do not enter stationary phase by becoming blocked at 'start'. They stop increasing in mass but they continue to initiate new cell cycles and to undergo division. When they eventually do stop proliferating, they are of a reduced cell size and have a reduced cell viability compared with wild type, and are also blocked in later stages of the cell cycle than the normal block point 'start'. These mutants are clearly defective in the gene functions required for an orderly transition of cells from the proliferating state to the quiescent state.

It has been suggested that mammalian cells may become malignant if they lose their 'restriction point' control (Pardee op. cit.), and as a consequence cells fail to accumulate at the proper point in G_1 when shifted to conditions favouring quiescence. If this is the case, then the *whi* mutants may be analagous to some types of malignant cell lines. Therefore, the further study of the *whi* and *cdc* 'start' mutants may also have implications for the problem of malignancy in mammalian cells. □

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