too high to allow sufficient flow. Munk and MacDonald (*J. Geophys. Res.*, **65**, 2169; 1960), for example, who sought to explain the Earth's excess equatorial bulge in terms of a "fossil" bulge remaining from an Earth previously rotating more rapidly, required a viscosity of 10^{25} poises in the lower mantle.

Then, some years later, Gordon (*J. Geophys. Res.*, **70**, 2413; 1965) suggested that if the physical phenomenon which makes convection possible is diffusion creep, mantle viscosity must increase rapidly with depth. This led naturally to the idea that if mantle viscosity was going to be low enough to support convection anywhere, it would be in the upper regions. The existence of a comparatively fluid, low velocity layer in the upper mantle tied in nicely with this view; and the asthenosphere rapidly became all things to all men— most importantly, a source of magma, a natural place to look for the source of isostatic compensation, and, of course, a convenient zone within which thermal convection could be confined. Moreover, as Knopoff (*Rev. Geophys.*, **2**, 89; 1964) pointed out, if there are, as there seem to be, well defined phase changes at several depths within the upper mantle, their existence would seem to be inconsistent with penetration by large-scale convection cells.

Hitherto, any serious deviation from this general view has been restricted largely to conversations at conferences, though lately the literature has been giving the impression of leaving the options open. Thus Dicke (*J. Geophys. Res.*, **74**, 5895; 1969) has concluded, from a study of the relaxation time for a second-order harmonic distortion of the Earth, that the viscosity of the lower mantle is about 10^{22} poises; and Goldreich and Toomre (*J. Geophys. Res.*, **74**, 2555; 1969), in disagreeing with Munk and MacDonald's explanation of the excess equatorial bulge, come up with 10^{22}–10^{24} poises for the same quantity. Moreover, Schubert *et al.* (*Science*, **169**, 1075; 1970) and Schubert and Turcotte (*J. Geophys. Res.*, **76**, 1424; 1971) have suggested that the phase change at a depth of 400 km, far from ruling out large-scale convection, may actually accentuate it. And, finally, Weertman (*Rev. Geophys.*, **8**, 145; 1970) takes the view that the physics of mantle convection is based on dislocation motion rather than diffusion creep, which will mean a smaller increase in the supposed viscosity with depth.

Now, however, Schubert and Turcotte (*J. Geophys. Res.*, **77**, 945; 1972) have made explicit the opposition to asthenospheric convection—that is, to the convection model in which the motion of the lithosphere in one direction is balanced by a return flow within the asthenosphere. The basis of

the argument is a mathematical model in which the variables depend only on depth, a simplification which is quite valid away from ridges and trenches where flow in both the lithosphere and asthenosphere must be about horizontal. The inputs to the model are the velocity of the lithosphere and the viscosity function with temperature and pressure.

This last function is unknown, although Schubert and Turcotte use a class of functions which assume the deformation mechanism to be diffusion creep (the mechanism which favours upper mantle flow) and a wide range of numerical parameters representing a range of several orders of magnitude in the viscosity. Heating by viscous dissipation is also taken into account because by increasing the temperature it decreases the viscosity, thus providing a self-lubricating mechanism. The final outputs from the model are then the heat flow due to viscous dissipation, the horizontal pressure gradient and the sheer stress on the lithosphere.

What emerges in numerical terms is that, for the wide range of functions and parameters chosen, the horizontal pressure gradients lie between 0.1 and 1.0 bar km^{-1} and shear stresses in the crustal plate lie in the range 0.1 to 0.4

kb. By contrast, the surface heat flux is restricted to the very small range 0.2–0.3 μcal cm^{-2} s^{-1}. But in the context of the scale of mantle convection the numerical results are less important than the qualitative consequences. Whether the force on the lithospheric plate is tension caused by a gravitational body force on the descending limb of the plate, or compression caused by gravitational sliding on the ridge flank, the return flow in the asthenosphere which balances the opposite motion of the lithosphere is driven by a hydrostatic pressure which increases with distance from the ridge. The necessary consequence of this pressure gradient is that the elevation of the ocean floor must also increase away from the ridge. Moreover, there should also be a positive gravity anomaly in the same direction.

Quite simply, neither the increase in elevation nor the positive gravity anomaly is observed. Appropriate figures suggest, for example, that the increase in elevation on the ocean floor across the Pacific from the East Pacific Rise to the Japanese trench should be about 5 km and the corresponding gravity anomaly would be 2,500 mgal. Yet observation suggests that the Pacific becomes shallower in the opposite direction; and certainly

Antigen in Human Breast Cancer Sera

THE possibility that an RNA virus analogous to the mouse mammary tumour viruses may have a role in the aetiology of breast cancer in women merits very serious consideration. In the past two years Moore and Spiegelman and their respective associates have shown not only that some human milks contain particles which in their structure and in their biochemical and biophysical parameters closely resemble the mouse mammary tumour viruses but also that polysomal RNAs present in human breast cancer cells, but not present in healthy breast cells, benign breast tumour cells or cancer cells from other organs, hybridize to a significant extent to mouse mammary tumour virus DNA.

These observations suggest that the human milk virus may be a close relative of the mouse mammary tumour viruses, that it may specifically replicate in human breast cancer cells and that it may have a role in the oncogenic transformation of breast cells. What Müller and Grossmann have to report in next Wednesday's *Nature New Biology* (May 24) certainly supports these notions.

Using a micro-double-diffusion technique, Müller and Grossmann surveyed sera taken from healthy women and from women with breast cancer for antigens which will cross-react with highly absorbed rabbit anti-mouse mammary tumour virus sera. Thirty-five

sera taken from healthy women failed completely, as expected, to cross-react with anti-mouse mammary tumour virus sera, but ten of thirty-six sera from women with breast cancer cross-reacted with three preparations of anti-mouse virus sera and the women from whom these cross-reacting sera were obtained had not had surgical treatment.

The precipitation lines with these ten human sera were completely confluent with one of the precipitation lines obtained with ether-disrupted mouse mammary tumour virus particles, and according to Müller and Grossmann there can be no doubt that the cross-reaction involving the human sera is an immunological reaction. Either, therefore, these human sera have an antigen(s) which is very similar but not identical to one of the antigens of mouse mammary tumour virus particles or the human sera have an antigen very similar to an antigen which cosediments and copurifies with the mouse virus particles.

The first of these alternatives seems by far the most probable and lends support to Charney and Moore's claim (*Nature*, **229**, 627; 1971) that sera from some women with breast cancer reduce the biological activity of mouse mammary tumour virus as well as to the idea that human breast cancer may be, at least in part, a viral disease.