changing coherently throughout the world. The face-value interpretation of these results is that, during the Miocene, seafloor spreading did indeed proceed discontinuously.

There is another possible explanation, however. The basis of comparison used by Blakely in his analysis was the time scale of Heirtzler *et al.*, a scale which assumes that seafloor spreading was constant at the mid-Atlantic ridge during the Cainozoic and which used the single magnetic profile from Vema cruise 20 in the South Atlantic. But this interpretation may not be valid. If, instead, it is assumed that the Pacific, Indian and Antarctic plates were each moving uniformly during the Miocene, then the Heirtzler scale must be adjusted. For various reasons (for example, that accelerations of entire plates relative to the asthenosphere would have had to have been improbably high at 20 km Myr^{-2} or even 40 km Myr^{-2}) Blakely concludes that such uniformity is likely, or at least the most likely situation given current data. As Blakely points out, although many examples of variable spreading rates have been described by other workers, most are from localised areas and do not include evidence for variations synchronised on several plates. The assumption of zero acceleration for plates is thus the simplest, but still "tenuous at best".

Nevertheless, using this assumption Blakely goes on to construct a new Miocene time scale which adds 18 reversals to the Heirtzler time scale for the period 22.7–7.6 Myr. Sixteen of these reversals are derived from the eight short-wavelength anomalies, and the remaining two are obtained by dividing the positive interval at 13 Myr on the Heirtzler scale into two smaller positive events. The revised scale now contains 14 short events lasting less than 0.1 Myr. It also has an average reversal frequency of 3.7 Myr^{-1}, which is a significant increase on the previous 2.5 Myr^{-1}. At the same time, the new figure is higher than the 3.0 Myr^{-1} rate derived from known reversals over the past 45 Myr, which suggests that there may also be some undetected events outside the more limited Miocene period considered by Blakely.

Finally, by comparing magnetic profiles for crustal magnetisation models with real profiles, Blakely is able to estimate the width of the transition zones from one polarity to the other. In each of the three models concerned the magnetised crust is regarded as a 0.5 km thick horizontal slab with its upper boundary at a depth of 3.5 km, but the models differ in the width of the band about the spreading centre over which new crust becomes magnetised. The best fit to the data from the north-east Pacific is obtained for a model in which the transition between polarities takes place over a horizontal distance of 6 km. This figure is, however, highly dependent on the thickness of the magnetic layer; for a layer thicker than 0.5 km, 6 km would be an overestimate.

Anti-actin

from Dennis Bray

THE rumour that antibodies cannot be produced against actin lasted about five years. Born of one or two unsuccessful attempts to raise antisera to this major protein of muscle, the rumour was nurtured by a familiar process of *post hoc* rationalisation. Actin is non-antigenic it was said, because it has such a strongly conserved structure that the small differences between species are not able to provoke an immune response. Actin is non-antigenic because it is present in almost every cell of the body so that all animals will have innately acquired tolerance. Anybody thus dissuaded from an attempt to make this most useful antiserum, however, has cause for regret. For there are now three independent reports of its production.

The starting point for two of the reports was the observation that patients with certain aggressive forms of hepatitis have autoimmune antibodies in their circulation. Although it was known from work with fluorescent antibodies that these were directed against smooth muscle, the precise nature of the antigens was not known. Gabbiani and colleagues (*Am. J. Path.*, **72**, 473–488; 1973) have now examined this by absorption studies and have found that this serum cross reacts with the actomyosin of blood platelets and with the purified component known in platelet circles as thrombosthenin A, but elsewhere simply as actin. Concluding from their tests that the serum is essentially anti-actin, the authors used it in immunofluorescent tests to show the distribution of this protein. Many non-muscular tissues, including liver cells, brush borders and intestinal epithelial cells, were found to be specifically stained.

Stimulated by the same medical findings, and in the laboratory in which they were originally made, Trenchev and colleagues (*Clin. exp. Immun.*, **16**, 125–136; 1974), sought to compare the autoimmune sera with those against various protein components of human smooth muscle. These were prepared by conventional methods against muscle myosin, tropomyosin and actin, and used in immunofluorescent tests. Once again a constellation of tissues were stained and a correlation between staining and the presence of microfilament arrays was noted; as in the networks of stained filaments seen in HeLa cells. The different types of artificially induced antibodies gave slightly different staining patterns but none seemed to resemble the hepatic sera more closely than the others.

Quite a different method and a highly original one was used by Lazarides and Weber (*Proc. natn. Acad. Sci. U.S.A.*, **71**, 2268–2272; 1974). Rather than prepare antisera to conventionally prepared actin, with its persistent complement of other proteins such as tropomyosin, these authors used the bold and very direct approach of injecting material recovered from SDS (sodium dodecyl sulphate) gels. The source of their material, mouse fibroblasts, is rich in actin and even a crude fractionation is adequate to give a band on acrylamide gels which is fairly pure. The denatured protein extracted from the gels is apparently a potent antigen and able to induce antibodies which cross react with both fibroblast and muscle actin. When used to stain the fibroblasts by the indirect fluorescent method this serum gave striking results. Brightly stained filaments in a wide variety of patterns are seen, sometimes aligned in parallel with the long axis of the cell or close to the outer surface, at other times arranged radially around central focal points.

Taken together these three reports provide good evidence that antibodies to actin can be produced, although the legally minded will not be without reservations. In not every case was the purity of the actin adequately monitored—a boring point but important in the light of previous experience—nor were all of the immunological controls carried out. Surprisingly, not one of the antisera was tested against adult skeletal myofibrils which surely would give a direct test of their specificity. And the use of antisera to denatured proteins is an uncertain business which, again to invoke rumours, may not have such a high degree of specificity as one is used to with native proteins.

But these are quibbles which will no doubt be resolved in time. In general the results are most encouraging, both from a medical point of view as well as that of cell biology. The technique of Lazarides and Weber, using the combination of immunofluorescence and SDS gels would seem to be of particular potency. One of its strengths is its applicability to structural proteins normally classed as insoluble and almost the first question about such proteins is: where are they in the cell? Electron microscopy can give some indication, but is limited in its ability to identify different chemical types and in the area of the cell which it can cover. Methods involving antisera and the light microscope are potentially free of both of these restrictions.