mented double-stranded RNA genome which consists of ten different segments. The terminal nucleotides of the double stranded RNA have recently been reported (*J. molec. Biol.*, **85**, 31–48; 1974) and the 5′ nucleotide sequence of one strand of each of the segments is A*GU, where A* is a methylated adenosine. SCPV contains a virion-associated transcriptase which transcribes all ten double stranded RNA fragments *in vitro* to give mRNA molecules each of which begins with a 5′ terminal sequence ppAG (*J. molec. Biol.*, **85**, 21–30; 1974; see also *Nature*, **250**, 13–14; 1974).

Since the activity of the SCPV transcriptase is low compared with that of the enzyme from reovirus, and because the genomic RNA is methylated, Furuichi tried adding a donor of methyl groups to the *in vitro* reaction mixture, and observed a huge stimulation of mRNA synthesis on addition of S-adenosyl-methionine (SAM). The product formed *in vitro* was found to be virus-specified and consisted of complete copies of one strand of each of the genome segments containing, on average, one methyl group per RNA molecule. The methyl group incorporated into the viral mRNA was detected in 2-0-methyl adenylic acid which is also present at the 5′ terminus of the genomic double stranded RNA.

Furuichi examined the RNA made during very short incubations and found that methylation occurs early in the transcription process when the nascent chains are less than ten residues long. Since mRNA synthesis is almost totally dependent on addition of the methyl donor, and methylation is such an early event, Furuichi suggests that methylation may be involved in the initiation of mRNA synthesis and that SCPV transcriptase utilises a methylation-coupled transcription system, which is so far unique.

It will be of interest to establish whether other viral transcription systems are similarly coupled. It is already known, for example, that the RNA tumour viruses contain virion-associated methylase enzymes, as well as reverse transcriptase. So far the effect of SAM *in vitro* has not been reported in this system.

Of even greater interest will be an understanding of the initiation of hnRNA synthesis in normal cells. Bajszar, Samarina and Georgiev (*Molecular Biology Reports* **1**, 305–310; 1974) have isolated the 5′ terminal nucleotides of mouse cell hnRNA and identified them as pppAp and pppGp. So far there is no evidence for methylation in these positions. It is possible, however, that methylation occurs at internal positions in hnRNA and that such modifications act as markers for subsequent cleavage reactions. It may be significant that the chromatographic mobility of some of the methylated nucleotides detected by Perry and Kelly in mouse mRNA suggested that more than one phosphate group is present. Perhaps some of the methyl groups are attached to nucleotides (pX*p) which orgininate from the 5′ end of mature cellular mRNA.

# Models of magnetic anomalies

*from Peter J. Smith*

A MAGNETOMETER towed at or near the ocean surface will record the well known linear magnetic anomalies which arise from alternating normal and reversed magnetic material within the oceanic crust. A magnetometer towed along very close to (say, within 100–200 m) of the sea floor, on the other hand, records anomalies which are much narrower and yet much greater in amplitude. But to what is this fine structure due? Do these small scale variations within magnetic polarity epochs and events reflect real physical variations of internal crustal properties or are they merely the result of topography or some other equally uninteresting phenomenon?

Conclusions on this point seem to differ. Atwater and Mudie (*J. geophys. Res.*, **78**, 8665; 1973) measured near-bottom anomalies on the flank of the Gorda Rise and found that almost all of them could be accounted for by a uniformly magnetised basement with topography. But some years ago, Luyendyk (*J. geophys. Res.*, **74**, 4869; 1969) found such an explanation unsatisfactory in connection with the fine structure in an area on anomaly 10 west of southern California. Instead, he invoked variations in magnetisation which arise either from ancient magnetic field variations or changes in petrology with distance from the ridge crest. Similarly, Larson and Spiess (*Science*, **163**, 68; 1969) concluded from a profile across the East Pacific Rise crest that small scale anomalies within the Brunhes epoch are unrelated to basement topography, and again appealed to fluctuation in palaeointensity.

Insofar as these conclusions refer to difficult areas they are not necessarily inconsistent, for it is quite conceivable that apparently similar phenomena in different regions could be explained in different ways. On the other hand, it is clearly important to know whether a common observation may be dismissed as an effect of little significance or whether it is necessary to look for deep-seated physical causes. So to investigate this matter further, Larson *et al.* (*J. geophys. Res.*, **79**, 2686; 1974) have constructed and analysed magnetic block models based on the volcanic basement profile actually obtained across the East Pacific Rise by Larson and Spiess—a profile carefully obtained by continuously and simultaneously measuring the depth of the magnetometer with an up-looking sonar, the height of the instrument above the sea floor with a down-looking sonar, and the thickness of sediment with another down-looking sonar.

The first model was given a constant magnetisation of 0.011 e.m.u. $cm^{-3}$ to see if such uniformity could reproduce the observed magnetic anomalies. As far as general widths and amplitudes were concerned, agreement between model and observation was quite good; and some specific anomalies were particularly well matched. In such cases it follows that the relevant near-bottom anomalies may be accounted for solely in terms of the shape of the basement profile and variations in the magnetometer depth. But in many cases specific anomalies were reproduced poorly by the uniform model, notwithstanding its topography. For such anomalies good agreement between model and observation could only be obtained by allowing magnetisation to vary along the profile between extremes of 0.0048 and 0.0257 e.m.u. $cm^{-3}$.

In summary, then, it would seem that however valid were the conflicting conclusions of Luyendyk and Atwater and Mudie in their respective geographic contexts, neither party is completely correct in general. Moreover, it is now clear that Larson and Spiess were not entirely correct in the specific case of the East Pacific Rise in attributing small scale magnetic anomalies solely to lateral variations of magnetisation. Near-bottom anomalies apparently have no unique cause; in any given instance it may be necessary to invoke both topographic and magnetic effects.

# Antitumour immunity

*from A. J. S. Davies*

THE antagonists of the notion of an immunological antitumour defence mechanism very properly ask why the process involved seems so often to fail. The explanations are increasingly ingenious. Initially, antibody was thought to block the response of hostile lymphocytes to the antigenic tumour; then antigen–antibody complexes and finally antigen have become popular as the blocking factor. It is not too difficult to envisage that antibody would mask those sites on the tumour cell which would otherwise act as recognition foci for lymphocytes. The antibody component of an antigen–antibody complex could do the same as long as it has some free binding sites (that is, the