has been produced north of the fracture zone. This could be achieved either by asymmetric spreading or by a ridge jump, both of them processes that are known to occur. The effect of asymmetric spreading on the magnetic anomalies in our example between points *a* and *b* would be to extend the wavelength of the magnetic anomaly, so that the troughs of the (say) negative anomaly between *a* and *b* should still lie on average mid-way between the lineations *a* and *b*. It is therefore quite valid to do as Schouten and Klitgord have done and map these peaks and troughs. However, in the area between anomalies 20 and 21 mapped in the North Atlantic at 45°N (ref. 4) identical geometry to that of anomalies *a* and *b* is found and south of the fracture zone an additional 'wiggle' has been mapped in the magnetic anomalies. This indicates additional crust belonging to another magnetic polarity event which might have been juxtaposed here by a ridge-jump, that is, when the location of the ridge crest itself changed, thus destroying the expected pattern of magnetic lineations. If variable offsets over fracture zones are due to ridge jumps, it seems even more difficult to visualize a memory effect remaining constant relative to the ridge crest.

The Schouten and Klitgord model proposes a high degree of order in the structure of the sea floor which leads to the question of the scale of order in nature — or at least the scale of order in the Earth's crust. On the one hand plate tectonic theory predicts constancy on a regional scale but on the local scale many a well surveyed area has turned out to be anomalous. Our perpetual hunt for 'normal' sea floor has often proved elusive.

How real are the kinks in magnetic lineations? Far from giving a greater understanding of ocean crust magnetization, the Deep Sea Drilling Project has shown us how complicated and chaotic crustal magnetization is. We also know that the elongate spreading cell is a simplified generalization since the random discrete volcano-injection pattern was observed in the FAMOUS area[5]. On the scale of macro lineations, however, order exists and the magnetic dataset south-west of Bermuda and its interpretation is quite convincing.

The point in their paper about which I remain most sceptical is the stability of the spreading cell system. Intuitively I feel that once a fracture zone forms and its immediate crust cools, even if it later becomes a zero-offset fracture zone there will be some time delay before its crust can re-heat and form part of a spreading cell. It may well be that the time scale for occurrences of changes in stress is shorter than the 40–50 Myr time scale required for re-heating the crust. Thus should a change in stress occur within this period, the former fracture zone location is still a zone of weakness and is likely to be the location of a new-generation fracture zone, but if no changes in stress occur the fracture zone location ought to heat up and become part of the spreading cell.

Schouten and Klitgord see the picture having more constancy than this and view the spreading cells in the ocean floor as stationary features separated by fracture zones along which anomalous crust is formed and that once formed these will remain anomalous and will always be the sites of offset or non-offset fracture zones. They see a high degree of order in the structure of the sea floor. Such optimism deserves encouragement, I hope they are right! It would certainly make cruise planning easier. □

C.A. Williams is at the Bullard Laboratories, University of Cambridge, Madingley Road, Cambridge CB3 0EZ.

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Chromosome translocations

Still more about oncogenes

from Peter Newmark

1982, the year of the oncogene, went out with a flurry of papers on c-*myc*, the cellular oncogene whose activation in virally induced chicken lymphomas (*Nature* **290**, 475; 1981) fired much of the current research.

Five papers document evidence (some of it already publicized through meeting reports — see *Nature* **300**, 403; 1982) that links c-*myc* to both Burkitt's lymphoma and mouse plasmacytomas.

The link to Burkitt's lymphoma is made three times over in the last issue of the *Proceedings of the National Academy of Sciences* for 1982 (volume 79). Dalla-Favera *et al.* (p.7824), Taub *et al.* (p.7837) and Neel *et al.* (p.7842) independently locate the c-*myc* gene at band q24 of human chromosome 8, within the segment that is translocated from chromosome 8 to 14 or, less frequently, to chromosome 2 or 22 in Burkitt's lymphoma.

Taub *et al.* go on to show that in eight out of twelve Burkitt's lymphoma cell lines, one of the two allelic copies of c-*myc* has been rearranged and that in two of the cell lines the rearrangement is clearly the result of the translocation since it has placed c-*myc* in proximity with the immunoglobulin heavy-chain region on chromosome 14. Dalla-Favera *et al.* mention similar evidence in a note added in proof; the details are soon to be published.

The link between c-*myc* and mouse plasmacytomas is also made three times over. Both Taub *et al.* (op.cit) and Crews *et al.* (*Science* **218**, 1319; 1982) show there to be c-*myc* sequences in a non-immunoglobulin-related sequence attached to the immunoglobulin heavy-chain gene region of several mouse plasmacytomas. The heavy-chain genes are on chromosome 12 and evidence already exists (see *Nature* **300**, 477; 1982) to show that the non-immunoglobulin-related sequence attached to it comes from chromosome 15 by translocation. Obviously, the translocation takes c-*myc* from chromosome 15 and places it on chromosome 12 at the heavy-chain locus.

To be more precise c-*myc* finishes up in the 'switch' region of the heavy chain locus according to both Taub *et al.* and Shen-Ong *et al.* (*Cell* **31**, 443; 1982). The latter provides several details of the fate of the translocated c-*myc* gene. First, the c-*myc* and the heavy-chain genes are found in opposite orientations, a conclusion confirmed by Crews *et al.* Second, in each of four plasmacytomas with a translocated c-*myc*, Shen-Ong *et al.* found a species of *myc* RNA transcript about 400 bases shorter than normal, the suspicion being that one exon had been displaced by translocation. Finally, the level of *myc* RNA is no greater than normal.

The question that is left open is whether the shift of c-*myc* from its normal chromosome to the immunoglobulin heavy-chain locus in both man and mouse is important for the induction of the lymphoma/plasmacytoma or is simply an irrelevant consequence of the translocation. The evidence of Shen-Ong *et al.* counters any suggestion that tumour induction is the result of increased transcription of c-*myc* in its new locus, but raises the possibility that an altered c-*myc* product is the problem. On the other hand, as several of the papers emphasize, in a sizeable minority of Burkitt's lymphoma lines and plasmacytoma mouse there is no evidence of any c-*myc* rearrangement.

Another note of caution is sounded by Crews *et al.* They have transformed NIH 3T3 cells with mouse plasmacytoma DNA and have shown that it is not the translocated c-*myc* sequence that is responsible for transformation, a conclusion that fits with Cooper and Neiman's observation that it was not the activated c-*myc* of virally induced chicken lymphomas that transformed NIH 3T3 cells (*Nature* **292**, 857; 1981) but another presumptive oncogene. Does this mean the NIH 3T3 assay is dead? Or that c-*myc* is irrelevant to the lymphoma? Or should one accept the somewhat glib explanation that a multistep process of oncogenesis has been observed. The answers should emerge in 1983. □

Peter Newmark is Deputy Editor of Nature.