haemopoietic cells as anchorage-independent by differentiation, but geared to require constant signalling for continued growth, as seen in this viral lymphoma example.

Using the DNA 'transfection' technique, B. Shilo (Massachusetts Institute of Technology) showed that DNA from several cell lines transformed with carcinogens could cause 3T3 fibroblasts to acquire the fully transformed phenotype of anchorage independence and tumorigenicity. The frequency of 'transfection' in these experiments is consistent with the idea that a single dominant gene is responsible for the transformed phenotype; this had been shown previously in the majority of cell hybridisation experiments. Furthermore, transformation of normal recipient cells with the transferred DNA was associated with a unique restriction fragment of DNA. Thus one, or a cluster of tightly linked genes is capable of dominantly causing the expression of the transformed phenotype. The analysis of this 'transforming' fragment and the characterisation of the protein product (if any) will be awaited with interest.

It is well established that the *src* gene of avian sarcoma viruses such as Rous sarcoma virus (RSV) is necessary for expression of the transformed phenotype. As yet it is not quite proven that the *src* gene product alone is sufficient, but the available evidence strongly points in this direction. The 60,000 molecular weight (60K) protein now thought to be coded by the *src* gene is a protein kinase and is very similar to a host cell protein kinase. T. Hunter (Salk Institute) showed that an *in vitro* rabbit reticulocyte protein synthesising system primed with RSV RNA produced a 60K protein that is indistinguishable from the protein identified in RSV-transformed cells. It was also shown that the 60K Src protein is a tyrosine kinase, and that in normal cell extracts phosphotyrosine is a minor component of the phosphorylated amino acids, but that after RSV transformation, the amount of phosphotyrosine, relative to phosphoserine, was increased 5–10 fold. A. Levinson (University of California, San Francisco) reported that the 60K Src protein is tightly bound to, and therefore probably integrated into, the plasma membrane. After mild proteolysis a 56K protein is found free from the membrane, and retains the kinase activity. Attempts to label the 60K Src protein on the outside of the cell have been unsuccessful up to now, and indicate that it is attached to the inside of the plasma membrane. As the Src protein kinase is very similar to the normal cell kinase but is distinct both structurally and in its kinase activity, the suspicion is that the viral Src product initiates transformation by radically upsetting the regulatory functions of the normal cell 'Src' kinase. How this upset of kinase activity leads to transformation is not clear, except that Src protein is required continuously for the expression of the transformed phenotype, as evidenced by temperature-sensitive mutants of *src*.

Although the Abelson leukaemia virus (ALV) lacks an identifiable *src* gene, O. Witte (Massachusetts Institute of Technology) showed that a 120K protein with tyrosine kinase activity is produced from the ALV genome. This kinase differs from the RSV kinase in that it requires Mn^{2+} instead of Mg^{2+} cations, but is similar inasmuch as there is in murine bone marrow cells (the preferred viral target *in*

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Palmdale bulge: fact or fiction?

from Peter J. Smith

SOME curious things have been happening to southern California recently; so much is certain. The only problem is that the curious phenomena that everyone thought were taking place may not have been taking place at all, whereas some quite different curious behaviour may have been going on. Or, what appeared to be certain facts with uncertain explanations now seem to have been uncertain facts with even more uncertain explanations.

Bits of the story have been coming out at intervals for several months now, although the full extent of the problem only become clear at last month's meeting of the American Geophysical Union in San Francisco. The most startling piece of experimental evidence was presented by A.E. Niell of NASA's Jet Propulsion Laboratory, who reported that the distance between his Pasadena laboratory and NASA's Goldstone Deep Space Station some 193 km to the northeast had increased by 20.3 cm in just 7 months. This remarkable measurement was made by repeatedly recording radio noise from quasars simultaneously at the Pasadena and Goldstone radio telescopes, a procedure that can give distance changes between the telescopes to within ± 5 cm; but the expansion has been confirmed in general terms by laser ranging experiments carried out independently by the US Geological Survey. According to J.C. Savage and W.H. Prescott, laser measurements show that between 1974 and 1978 southern California was undergoing a nearly uniform contraction in the north–south direction but that since 1978 there has been east–west expansion with little or no north–south compression. So the laser data for the post-1978 period are consistent with the 1979 radio interferometry.

Unfortunately, however, the laser data as a whole seem not to be in agreement with the behaviour of the so-called Palmdale bulge, the 84,000 km^2 area of southern California which covers the southwestern section of Niell's interferometry baseline and which has undergone well-publicised ups and downs over the past 20 years. The rise of this zone apparently began in 1959, although this was not discovered until 1976 when it immediately set off speculation about an impending earthquake. By 1974 the maximum uplift had reached 45 cm; but since then there has been a remarkably rapid deflation accompanied by a tilt of the whole region to the north, an increase in the horizontal strain and an increase in radon emission — a group of phenomena which has again sparked off seismic speculations. The strange thing is, however, that the behaviour of the bulge is inconsistent with much of the laser-derived information about the larger area. When southern California was undergoing north–south contraction from 1974 to 1978, a simultaneous east–west or vertical expansion was to be expected. But during this period the bulge was actually contracting, which it continued to do even when the north–south contraction stopped. As Barry Raleigh of the US Geological Survey recently noted, "no physical model has yet been proposed which incorporates these apparently inconsistent results."

And maybe no model is necessary, for what if the observations are wrong or perhaps, let us say, not quite what they appear to be? After pouring more than $1 million into the study of a supposedly deflating bulge, this may seem an odd point to make; but D.D. Jackson and W.B. Lee made it at San Francisco in all seriousness. From an examination of the data going right back to 1959, especially those along the profile San Pedro — Los Angeles — Saugus — Palmdale, Jackson and Lee conclude that most, if not all, of the rise centred on Palmdale never in fact took place at all. They find that the altitudes of larger features such as mountains, as indicated by the raw observations, have changed by the same amounts and at the same times as those of the smaller features such as ridges; and although a tectonic cause for such behaviour cannot be ruled out completely, the simplest explanation is the presence of a systematic error, probably in the calibration of the levelling rods.

Many people are simply not going to believe that, at least without much more evidence, if only because of the embarrassment it would cause. Yet there is clearly something going on in southern California. Monitoring continues.

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0028-0836/80/03247-01$01.00