(lower Eocene/middle Eocene, middle Eocene/upper Miocene, upper Miocene/Pliocene, Pliocene/Pleistocene). The latter three are unconformable contacts with equivalents at Site 612 and on the shelf, but only the last two were recovered. The fourth sequence boundary comprises a disturbed 'zone' of intense slumping, separating lower and middle Eocene strata.

One of the most interesting new developments regarding unconformities along this segment of the margin is our documentation of a long hiatus (25–37 Myr) at both sites between the late Miocene and the early Oligocene or middle Eocene. Extrapolation of the core data along the grid of seismic lines suggests that upper Eocene, lower and upper Oligocene and lower and middle Miocene strata are virtually absent from the lower slope and upper rise. We cannot, however, rule out the possibility that very thin layers of these units might exist undetected by the limited resolution of our seismic lines.

Seismic lines which follow the depositional strike near Site 613 reveal a series of stacked buried channels, whose ages and origins have challenged seismostratigraphers since they were discovered. Under the influence of the Vail model of sea level change, the general consensus has been that the channeling resulted from a mid-Oligocene sea-level fall. Drilling at Site 613 revealed that one series of channels was formed between the early and middle Eocene. The lower Eocene surface is eroded from the shoreline across the shelf through Sites 612 and 613, and basinward.

Some channels completely penetrate the lower Eocene strata, cutting deeply in the underlying Paleocene section. At Site 613, however, which appears to have been near the slope-rise transition during the early and middle Eocene, the presence of frequent slumps indicates a depositional regime in which displaced sediments were accumulating. These base-of-the-slope accumulations increased during the middle Eocene and eventually filled the channels on the lower Eocene-Paleocene surface.

A second series of channels is stacked upon the middle Eocene surface. The middle Eocene unit has had a complex history of erosion, as revealed by a wide outcrop belt now present along the lower slope, and by truncated reflections in its upper part. It has presumably undergone several periods of erosion since the late Eocene, and is still being worn away along its outcrop. Middle Eocene clasts were recorded in the Miocene and Pleistocene strata at Site 613 and in surficial piston cores along the lower slope and rise.

Filling of these middle Eocene channels took place during the late Miocene, as revealed at Sites 604 and 613, perhaps during the low stand of sea level associated with the Messinian salinity crisis. The coarse sands, gravels, and lithoclastic conglomerates at Sites 604 and 613 indicate that the channel fill came from the shelf and was dumped on the lower slope and upper rise in rather chaotic fashion.

These findings demonstrate that with continously cored, shallow-penetration sections, carefully placed on seismic transect lines, we can easily obtain the fundamental geologic data necessary to unravel the complex Cenozoic stratigraphy and depositional history of sediment-rich passive margins. The concept of multi-site transects has developed late in the DSDP program but the immense value of their systematic approach to margin evolution has been amply demonstrated by the results of such legs as 78,79, 80, 93 and 95. Moreover, we would hope that the sections now drilled constitute only the initial steps toward a more comprehensive appraisal of margin development. The New Jersey Transect, in particular, should stimulate new proposals for additional sites along Line 25 and its joining seismic grid, especially those aimed at deeper targets within the Mesozoic sequences. □

The scientific party aboard *Glomar Challenger* for Leg 95 of the Deep Sea Drilling Project, International Phase of Ocean Drilling, consisted of: C. Wylie Poag (U.S. Geological Survey, Woods Hole), A.B. Watts (Lamont-Doherty Geological Observatory, Columbia University, Palisades, New York), Michel Cousin (Universite Pierre et Marie Curie, Departement de Geotectonique, Paris), David Goldbert (Lamont-Doherty Geological Observatory, Columbia University, Palisades, New York), Malcolm B. Hart (Plymouth Polytechnic, Department of Environmental Sciences, Plymouth), Kenneth G. Miller (Lamont-Doherty Geological Observatory, Columbia University, Palisades, New York), Gregory Mountain (Lamont-Doherty Geological Observatory, Columbia University, Palisades, New York), Yuji Nakamura (University of Tokyo, Laboratory for Earthquake Chemistry, Nakano, Tokyo), Amanda Palmer (Princeton University, Department of Geological and Geophysical Sciences, Princeton), Paul A Schiffelbein (Scripps Institution of Oceanography, La Jolla), B. Charlotte Schreiber (Queens College (SUNY), Department of Earth and Environmental Science, Flushing, New York), Martha Tarafa (Woods Hole Oceanographic Institution, Chemistry Department, Woods Hole), Jean E. Thein (Geologisches Institut der Universitat Bonn, Bonn), Page C. Valentine (U.S. Geological Survey, Woods Hole), Roy H. Wilkens (Department of Earth & Planetary Sciences, Massachusetts Institute of Technology).

Oncogenic intelligence

More *ras*matazz

from Peter Newmark

DESPITE the wealth of information that has emerged in the last 18 months about normal human *ras* genes and the oncogenes that arise from them by single point mutations, several vital questions remain unanswered. First, what causes the point mutation? And second, what are the functions of the proteins encoded by the *ras* genes and oncogenes? Papers in this issue suggest one answer to the former question and a new way to ask the latter.

In the article beginning on page 658, M. Barbacid and his colleagues show that a point mutation distinguishes an H-*ras*-1 oncogene of carcinogen-induced rat mammary carcinomas from the normal rat *ras* gene and suggest that the mutation is a direct consequence of the reaction with DNA of the carcinogen. Nine carcinomas which appeared some months after a single injection of 50 day old female rats with nitroso-methylurea, were examined. Each had a similar H-*ras*-1 oncogene. One oncogene that was sequenced, differed from the normal rat H-*ras*-1 gene simply by having deoxyadenosine (A) instead of deoxyguanosine (G) as base number 34. The predicted result is the substitution of glycine by glutamic acid as the twelfth amino acid of the p21 protein encoded by the gene (just as human *ras* oncogenes often have a mutation that leads to the replacement of their glycine at the equivalent position in p21).

Barbacid *et al.* point out that nitroso-methylurea is an alkylating agent that has a preference for methylating Gs. Once methylated, a G might base pair with thymidine instead of the normal deoxycytosine; subsequently the methylated G would be replaced by A. Hence G would have been substituted by A. As the authors point out, they have only proved the substitution of G by A in one carcinoma but if that is invariably the case it will strongly support their suggestion for the generation of the point mutation. In any case their animal model promises to be very useful in the study of *ras* genes and oncogenes.

So, too, does yeast now that two laboratories have shown it to contain genes closely related to mammalian *ras* genes. Gallwitz *et al.* (p.704) came across their *ras* gene by chance, at first not recognizing the identity of the sequence close to a yeast actin gene. By contrast, DeFeo-Jones *et al.* (p.707) deliberately set out to look for *ras*-like genes in yeast. They found two and sequenced one. Comparing its predicted protein with that from the human *ras* gene, the same amino acids are found in about 60 per cent of the positions; the equivalent figure from Gallwitz *et al.* is about 40 per cent. (Incidentally,the Gallwitz *et al.* protein would have glycine as aminoacid 12, as do normal human and rat *ras* genes, whereas the DeFeo-Jones *et al.* protein would not, as in many *ras* oncogenes.)

Given the variety of clever tricks that can be played with yeast genes (see K. Struhl *Nature* **305**, 391), it may be easier to learn something of the function of *ras* genes in yeast than in mammals. One obvious experiment would be to replace the normal yeast *ras* gene with a disrupted version and observe the resulting phenotypic effects on yeast — in particular, to check if they are similar to those induced by disruption of cell cycle genes in yeast. A more subtle variation on this theme would be to find a temperature sensitive mutant of the yeast *ras* gene. Experiments of that kind are already underway in 'yeast' laboratories and the results are eagerly awaited. □

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