they looked at ". . . represent the most ancient unisexual lineage known". But they also argue that the nuclear genes in the lineage are continually being replaced by genes from the four sexual species. The only component of the genome that has existed, with little or no recombination, for 4 million years, is the mtDNA. But if, as seems likely (and as is consistent with the data from these three papers), mtDNA is uniparentally inherited, it has existed with little recombination for 10^9 years.

Hedges *et al.* do not argue that their data have any special significance for the evolution of recombination: their emphasis is on the independent histories of nuclear and mitochondrial DNA. Spolsky *et al.* say that "such long-term persistence raises a question about the necessity of sexual reproduction . . . to lineage survival", but they conclude (reasonably in my view) that rare replacement of nuclear genomes may have contributed to the long-term survival of clonal salamander lineages.

Quattro *et al.*, however, were specifically trying to estimate the age of a clone, because of its relevance to theories of sex. They regard their data as evidence against the view that clones are short-lived, and conclude that "the rarity of unisexual taxa might have as much to do with low origination rates as with high extinction probabilities". But, in evolutionary terms, 100,000 years is but an evening gone: it is important to know that a female genome of *Poeciliopsis* has existed without recombination for that long, but it does not contradict the conventional wisdom. The idea that low origination rates explain the rarity of parthenogenesis can at best be only a partial explanation. After all, sex probably originated just once, whereas parthenogenetic strains have arisen from sexual ones many times. If origination rates determined the relative frequencies of the two types of reproduction, we would all be parthenogens.

There are, however, some unisexual populations which seem to be genuinely old in evolutionary terms. The most dramatic are the bdelloid rotifers, in which no one has ever seen a male. Have these rotifers invented an alternative to males as a means of exchanging genetic material? If not, how do they manage without recombination? □

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SEISMOLOGY

A mantle thermometer

Peter Shearer

DENSE regional networks of seismographs in the western United States, originally deployed to study local earthquakes, are now being used to reveal details in the structure of subduction zones thousands of kilometres away. Using array processing techniques common in exploration geophysics, Vidale and Benz on page 678 of this issue[1] combine records of distant deep earthquakes recorded on hundreds of individual seismometers to image faint near-source reflections from seismic discontinuities in the upper mantle near subducting slabs. The depth of these discontinuities can be used as a thermometer for rocks hundreds of kilometres below the surface, to show how they are affected by the arrival of cold oceanic lithosphere sinking into the hot mantle.

For many years, the US Geological Survey and other organizations have operated regional networks of hundreds of seismographs in California, Nevada, Washington and Utah. Data from these networks are used to locate and study local earthquakes in these active seismic areas. However, data from more distant events around the world (teleseisms) have been pretty much overlooked. Vidale and Benz have collected and combined data from five different seismic networks to form a single array of 881 stations spanning over 1,000 km. This was a formidable organizational task, as the networks store their data separately in a variety of formats.

Vidale and Benz set out to use the arrays to obtain a high-resolution picture of the internal structure of the Earth's slowly circulating mantle. The great advantage of using such a large number of stations is that data 'stacking' techniques can be used to suppress the effects of incoherent noise and increase the visibility of weak seismic phases such as those resulting from reflections off upper-mantle discontinuities. The seismic waves, both fast compressional (P) and slower shear (S) waves, were from a number of deep earthquakes from subduction zones around the Pacific basin, where cold oceanic lithosphere sinks into the mantle beneath the surrounding plates. For each earthquake, Vidale and Benz consider waves that arrive after the first, direct P-wave arrivals and before the later, surface-reflected pP-wave arrivals (Fig. 1). By combining the results from hundreds of individual recordings, they identify waves converted from the S to the P phase on transmission through a discontinuity at a depth of 685 km near the source. They also recognize P-wave reflections from the underside of discontinuities at depths of 210 and 400 km (Fig. 2). The closeness of two of these to the depths of known global discontinuities[2,3] at 660 km and 410 km (more accurately, 415 km) suggests that the presence of the subducting slab depresses the former by about 25 km and elevates the latter by about 15 km.

S-to-P converted arrivals have been identified in previous studies of subduction zone events[4–6], but the underside P reflections had not previously been observed. Our global maps[7] of the 660-km discontinuity obtained from long-period shear-waves indicate regional depressions of about 20-km deep near subduction zones, but these maps have limited horizontal resolution (about 1,000 km). Vidale and Benz's data have much better resolution (100 km) and suggest that the 660-km discontinuity deepens slightly more in the immediate vicinity of the subducting slabs. The interesting question, of course, is what changes across these discontinuities, and the measured deflections are much less than predicted for a purely compositional change[8]. So it seems likely that the discontinuity at 660 km depth is due largely to a change of mineral phase.

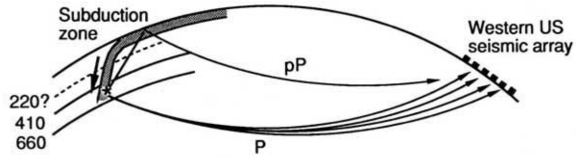

FIG. 1 Vidale and Benz study seismic waves from deep earthquakes generated as cold, dense oceanic lithosphere (shaded) descends into the mantle at Pacific subduction zones. They analyse waves that arrive after the direct P waves and before the surface-reflected pP arrivals. 220, 410 and 660, discontinuities at those respective depths.

High-pressure experiments in mineral physics also suggest that both the 410- and 660-km seismic discontinuities are primarily caused by phase changes in olivine resulting from the increased pressure in the upper mantle. The depth at which these phase changes occur depends upon the temperature, so, in principle, observations of variations in the depth can be used to determine *in situ* mantle temperatures. The elevation of the 410-km discontinuity and depression of the 660-km discontinuity are consistent with the effect of a cold subducting slab on the appropriate phase boundaries. However, laboratory values obtained for the scaling factor between pressure and temperature for these reactions vary by up to a factor of two, limiting the accuracy with which mantle temperature differences can be inferred.

The character of the 660-km discontinuity bears on the issue of whether the mantle convects as a whole unit or whether the boundary divides the mantle into two separate convection cells. The fate of the subducting oceanic slabs when they reach the discontinuity is one of the clues used to address this question. Thermal models which show subducting slabs passing cleanly through the 660-km discontinuity into the lower mantle predict greatly reduced mantle temperatures in a narrow zone within the subducting slab itself[9,10]. These models do not predict the depression seen by Vidale and Benz 100 km to the side of the slab, nor the 1,500-km-wide regional depression seen in our long-period data[7]. The most likely explanation for these anomalies is that the slab broadens or turns near 660 km, thus spreading the cold slab temperatures into the adjacent mantle. This model is supported by recent seismic velocity inversions[11,12] for northwest Pacific subduction zones which image horizontal extensions of the slabs just above 660 km. The apparent deflection of the slabs could result from resistance to slab penetration through the 660-km phase change, as some models of mantle convection have indicated[13,14]. Although these results argue against unhindered slab penetration into the lower mantle, they do not settle the long-standing controversy regarding whether convection is stratified near 660 km or not, as the slab material might eventually make its way into the lower mantle. (For more on this, see the recent News and Views article by Lay[15].)

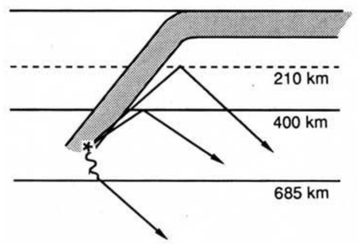

FIG. 2 S waves (wavy line) are converted into P waves on transmission through the (depressed) 660-km boundary. Vidale and Benz also identify P waves reflected from the undersides of less deep interfaces.

Among the most intriguing results of Vidale and Benz's analysis are the apparent reflections from a discontinuity near 210 km, which the authors suggest is the base of the asthenosphere, a plastic zone below the lithosphere. For years, seismologists have found sporadic evidence for a 220-km discontinuity in particular areas, but recent global studies indicate that this is restricted to certain continental regions and is not a significant global feature[3,16]. Vidale and Benz present a puzzle: if 220-km discontinuities are seen in locations as diverse as subduction zones and continental shields, why are they not observed elsewhere? One possible explanation, suggested by Vidale and Benz, is that the interface is laminated, making it more sensitive to the short-period data used in their analyses than to the long-period data used in the global analyses. □

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RAS REGULATION

NF is enough of GAP

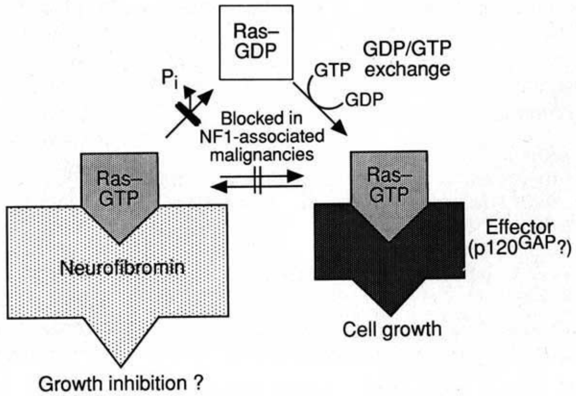
Gideon Bollag and Frank McCormick

NEUROFIBROMATOSIS type 1 (NF1) is a genetic disease characterized by benign and sometimes malignant tumours that originate in the neural crest. Two years ago, identification of the gene which is defective in NF1 patients revealed a telling pointer to its normal cellular function and its role in the disease: the gene product, now known as neurofibromin, is a GTPase-activating protein (GAP) for the $p21^{ras}$ proto-oncogene product that converts active GTP-bound $p21^{ras}$ to its inactive GDP-bound state[1].

Following up this pointer, Basu *et al.* (on page 713 of this issue[2]) and DeClue *et al.* (in the latest issue of *Cell*[3]) now show that cells derived from malignant NF1 tumours express very low levels of neurofibromin. As a result, $p21^{ras}$ accumulates in its GTP-bound state and thus contributes to the transformed state of the cells through interaction with certain effectors (as yet unknown). This is reminiscent of the role of $p21^{ras}$ in other tumour types; oncogenic mutations that render $p21^{ras}$ insensitive to GAP activity result in an accumulation of $p21^{ras}$–GTP and hence uncontrolled growth. From these new results we can infer that neurofibromin is a tumour suppressor, as its loss of function contributes to tumour growth.

Calculations based on the intrinsic kinetic parameters of purified $p21^{ras}$ proteins suggest that, in the absence of other factors, about 50 per cent of $p21^{ras}$ should be in the GTP state. In normal Schwann cells (as in other normal cells), GTPase activation reduces this level to about 5 per cent. In the NF1-derived malignant Schwannomas that lack neurofibromin but express normal levels of $p120^{GAP}$, the $p21^{ras}$–GTP pool approaches the 50 per cent level indicative of null GAP activity[2,3]. So it seems that, in Schwann cells, $p120^{GAP}$ has little or no part in $p21^{ras}$–GTPase activation, and that neurofibromin is the major negative regulator. Conversely, in mouse fibroblasts $p120^{GAP}$ activity is enough to maintain $p21^{ras}$ in its inactive state despite the presence of neurofibromin[4]. These observations imply that although neurofibromin and $p120^{GAP}$ are both ubiquitous, one or the other predominates to determine the fraction of $p21^{ras}$ that is in the active GTP state.

What then is the function of the alternative $p21^{ras}$ regulator ($p120^{GAP}$ in Schwann cells, neurofibromin in fibroblasts)? One possibility is that $p120^{GAP}$ and neurofibromin are involved in signal emission from $p21^{ras}$–GTP as well as downregulation (see figure)[5]. In this context, it is informative to note that $p21^{ras}$ can send both positive and negative growth signals in cells derived from the neural crest. In PC12 pheochromocytoma cells, for example, activated $p21^{ras}$



Model for $p21^{ras}$ function in neural crest cells.