and hadn't yet succeeded in expanding beyond its homeland in the Kinai region.

I then visited Easter Island, famous for its giant stone statues. There I learned that later statues are bigger than early ones, and that the tallest ever erected (the one named Paro, 9.8 m tall) was the last — in apparent disagreement with Marcus. But Easter Island, unlike Egypt or the Valley of Mexico or Peru, never became tightly unified and remained divided into rival clans that continued to compete visibly with each other. Hence Easter Island might violate the letter but supports the spirit of Marcus's hypothesis.

Finally, the Maya city-states in Central America are famous for their own pyramids and temples. As on Easter Island, later Maya rulers built bigger temples, but again the Maya states were never unified but stayed locked in fierce competition and warfare. In addition, Marcus herself points out that some big late Maya buildings, such as Pacal's tomb at Palenque and Hasaw Chan K'awil's tomb at Tikal, were erected by usurpers or else by kings weaker than their predecessors, and thus with a special need to indulge in flashy displays of power.

Archaeologists studying other ancient monuments will find it challenging to test or expand Marcus's arguments. As she concludes, "We should be as skeptical of ancient propaganda as we are when dealing with modern politicians". ■

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Earth science

Tiny triggers deep down

Harry W. Green II

The documentation and characterization of remotely triggered earthquakes deep within the Earth is an achievement that provides insight into the mechanisms that initiate such events.

Earthquakes occur widely in the planet's crust and to depths approaching 700 km in subduction zones, where oceanic crust and the associated 50–100 km of mantle dive back into Earth as the return flow of plate tectonics. But we know little about the physics of earthquake initiation (nucleation), especially at great depth, because the mechanisms known to operate close to the surface — brittle failure of virgin rock, or frictional sliding on a pre-existing fault — cannot occur at the high pressures at depth[1].

A new window on the problem may have been opened by Tibi *et al.*[2] (page 921 of this issue). They provide the first analysis of two large (magnitudes 7.6 and 7.7), very deep earthquakes that occurred on 19 August 2002 in the Tonga subduction zone beneath the southwestern Pacific Ocean. Although these earthquakes were separated by about 300 km on the map and by 65 km in depth, they occurred within 7 minutes of each other. Tibi *et al.* argue that the second large earthquake, and a magnitude-5.9 precursor of it, were triggered by passage of the seismic waves generated by the first earthquake.

Although remote triggering is known for earthquakes near Earth's surface[3], Tibi *et al.* provide the first such demonstration for a deep earthquake. Moreover, the authors discuss an earthquake series that occurred beneath Tonga in 1986 (Fig. 1) that now can be seen as probably another remotely triggered sequence. It is clear that regions in which earthquakes are triggered by the small disturbances generated by earthquake waves far from the source must be primed for failure, but for some reason nucleation does not occur readily. It is also clear that the delay between arrival of the triggering seismic waves and the time of the ensuing deep earthquakes varies from minutes to tens of minutes (see Table 1, page 922). Thus, the timescale of this 'incubation' period is likely to be a characteristic of the triggering mechanism.

Three mechanisms have been proposed as potentially responsible for deep earthquakes: (1) dehydration embrittlement[4]; (2) faulting induced by a phase transformation between one mineral form (olivine) and another, denser form (spinel)[5]; and (3) adiabatic shear instability[6]. All three have an experimental basis (although for crystalline materials, the last has been demonstrated only in metals). In each case, shear failure is the end result — rapid slip across a narrow zone such as a fault.

Mechanism 1 basically extends brittle fracture to high pressures by the generation of a pore fluid that assists opening of tensile microcracks, which then self-organize and lead to shear failure. Mechanism 2 is similar in outcome, but the underlying physics is fundamentally different. It involves the generation of another type of defect — microanticracks — which are small, crack-shaped lenses filled with a low-viscosity nanocrystalline aggregate of the stable phase; the microanticracks then self-organize and lead to shear failure[7]. Mechanism 3 involves the localization of deformation into a shear zone as a result of strain-softening: the rock becomes weaker as it flows. Runaway shear heating follows, leading to failure.

All three processes have specific requirements for generating an earthquake. The first requires a hydrous phase at or slightly beyond

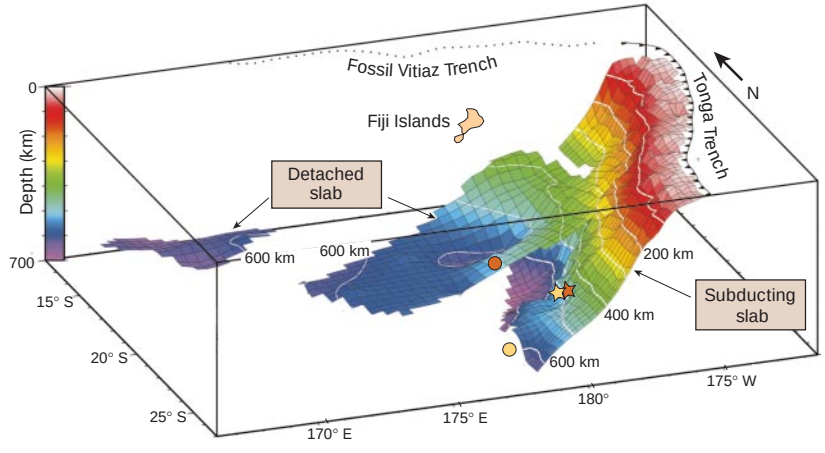

Figure 1 **Earthquakes and subducted slabs beneath the Tonga–Fiji area. The subducting slab and detached slab are defined by the historic earthquakes in this region: the steeply dipping surface descending from the Tonga Trench marks the currently active subduction zone, and the surface lying mostly between 500 and 680 km, but rising to 300 km in the east, is a relict from an old subduction zone that descended from the fossil Vitiaz Trench. The locations of the mainshocks of the two Tongan earthquake sequences discussed by Tibi *et al.*[2] are marked in yellow (2002 sequence) and orange (1986 series). Triggering mainshocks are denoted by stars; triggered mainshocks by circles. The 2002 sequence lies wholly in the currently subducting slab (and slightly extends the earthquake distribution in it), whereas the 1986 mainshock is in that slab but the triggered series is located in the detached slab, which apparently contains significant amounts of metastable olivine[8,9]. (Modified from ref. 13 with permission of the American Geophysical Union.)**

its limit of stability that can break down to produce the fluid necessary to produce the instability. For the second, because of low temperatures in the core of the subducting slab, olivine must have failed to react to the spinel phase that is stable at depths of 400–700 km, and must be slowly transforming and causing earthquakes as it warms up[1]. The third requires specific conditions for slow, continuing flow to be concentrated into a narrow zone in which the strain rate can accelerate as the heat generated by the straining accumulates, leading to an explosive increase in temperature, melting and shear failure. Each of these mechanisms has different implications for the temperature of subducting slabs and for the possible recycling of water back into the deep mantle from the surface.

Can the incubation times of the Tongan sequences help to discriminate between these possibilities? I think that they can. One cannot be certain whether the required phases are present for mechanisms 1 or 2, but we know from experimental work in the laboratory that a few minutes to tens of minutes is sufficient time in which to generate the primary microcracks or microanticracks, and for them to self-organize and lead to failure. In contrast, an adiabatic shear instability, in which strain rates are initially low, must inherently have a slow lead-up period as strain-induced heat accumulates to drive the rapid stage of the process. This time has been estimated as 10–10,000 years[8]. So unless the regions in which the earthquakes beneath Tonga were triggered contained shear zones that were close to thermal runaway, it is difficult to see how mechanism 3 could be responsible. This is particularly true for the events of August 2002, because the triggered earthquakes lie in a region in which an earthquake had never been detected previously (Fig. 1).

The 1986 Tongan sequence might tell us even more about deep-earthquake nucleation. As Tibi *et al*. point out[2], the triggering mainshock lay in the steeply dipping, currently active subduction zone, but the triggered earthquakes were in a remnant slab lying above it (Fig. 1). Various data[9,10] are consistent with the presence of a significant amount of metastable olivine in this slab but are less consistent with other possibilities. Thermal models of subduction zones show that the currently active Tonga slab is the coldest on Earth and therefore has the highest probability that metastable olivine is preserved within it[11]. Thus, if there is metastable olivine in the remnant slab, its presence in the active slab is virtually assured, which could be responsible for the initiation of all of the earthquakes in these sequences — although, after initiation, it is possible that adiabatic shear heating could contribute to the total size and magnitude of the earthquakes[12]. In contrast, hydrous phases are difficult to reconcile with the properties of the detached slab beneath Fiji[8,9,13], and more generally it is not clear that they can trigger earthquakes at depths of more than 400 km (ref. 1).

Tibi and colleagues' observations are a major advance in understanding deep earthquakes, and they might provide a new constraint on the mechanism by which these earthquakes begin. This long-standing problem in geophysics is far from solved, however. Further searches for other triggered sequences of deep earthquakes, and for the possible existence of metastable olivine and/or hydrous phases, will be necessary for us to take the next steps in understanding. ■

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Developmental biology

Hotspots for evolution

Michael K. Richardson and Paul M. Brakefield

Two studies of fruitflies suggest that although development relies on a diverse toolkit of genes, the evolution of physical characteristics might be powered by variation in just a few of these tools.

The physical characteristics of animals evolve because their genes change over successive generations. It is not always clear, though, which genes are involved[1,2]. The genes that regulate embryonic or larval development are likely candidates, because they control how the animal develops its characteristic form and features. It is possible that natural selection might produce evolutionary change after the adjustment of just a few such switches on the genetic control panel of development. Writing in this issue, Sucena *et al.*[3] and Gompel and Carroll[4] provide evidence that this can indeed happen. They show that modifications at just a few developmental hotspots underlie 'parallel' evolutionary changes that occurred independently in different species.

Tinkering with developmental genes is not necessarily an easy route to evolutionary change. For example, mutations in the *antennapedia* gene — an important regulator of development in the fruitfly *Drosophila* — can produce a fly with legs growing on its head[5]. This may be fascinating to developmental geneticists, but from the fly's point of view it is not helpful.

So, how are developmental genes altered during the normal course of evolution in natural populations? To answer this question, researchers need to marry a knowledge of evolutionary changes with developmental genetics — as Sucena *et al.*[3] and Gompel and Carroll[4] have now done. Both groups had already made preliminary studies with a single species of the fruitfly *Drosophila melanogaster* (one of the key model species in developmental genetics). But model species alone cannot tell us everything about evolution, and so each team broadened their investigations to include other fruitflies with distinctive physical characteristics (morphology). They then searched for changes in genetic pathways that might account for the differences.

Sucena and colleagues[3] examined the development of hairs — called trichomes — in the larvae of different *Drosophila* species. They found that hairless patches on the young larvae had evolved independently in three of the lineages included in the study (Fig. 1). If something evolves once, it can be difficult to find out why, but if it evolves three times independently within a species group, we can look for correlations by mapping developmental changes and trait evolution onto a 'phylogenetic' tree (a sort of family tree)[6]. And Sucena and colleagues found such a correlation: the activity of a gene called *shavenbaby* (*svb*) was absent from the naked areas of all three lineages that showed hair loss.

These findings could mean that a loss of *svb* expression was directly responsible for the trichome loss. Alternatively, the loss of *svb* could simply be a consequence of another, more important, change that occurred earlier in development. But, as Sucena and colleagues note, previous studies have shown that several 'upstream' genes involved in trichome patterning, including *wingless* and *engrailed*, are not altered in certain species, related to *Drosophila virilis*, that show trichome loss[7,8]. To determine exactly how the *svb* gene affected trichome development, the authors used another powerful tool — they crossed certain species