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

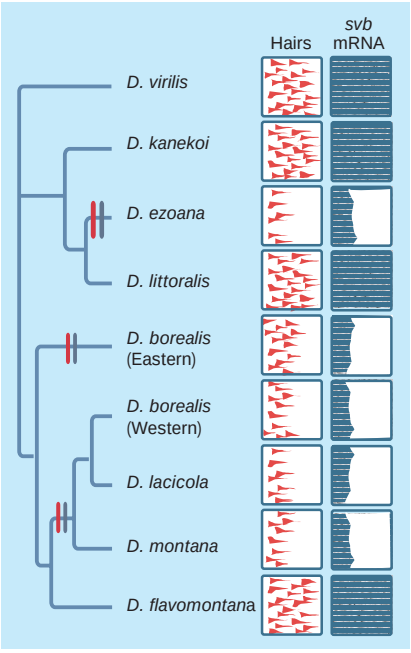

Figure 1 **Shavenbabies are hairy. As described by Sucena *et al.*[3], the correlation between the loss of trichomes (hairs) in different fruitfly species and the loss of the activity of the *shavenbaby* gene (*svb*) is revealed when the data are mapped onto a family tree (a phylogeny). The boxes represent trichome coverage and *svb* expression (assessed through measuring levels of messenger RNA, mRNA) in an anterior segment of the fruitfly's abdomen. In all three lineages in which trichome loss arose independently, *svb* expression is also lost (vertical red and grey bars, respectively, on the tree). Gompel and Carroll[4] offer a similar but more detailed picture of their results in their Fig. 2 (page 933).**

of *Drosophila* and looked at gene activity in hybrid animals. These additional data enabled them to infer that a loss of *svb* expression itself was probably directly responsible for the evolution of hair loss.

Meanwhile, Gompel and Carroll[4] surveyed variation across species in the abdominal pigmentation pattern of adult fruitflies. Each segment of the abdomen in *D. melanogaster* bears a dark band. The sexes also differ: the two terminal segments are darkly pigmented in males, but not in females. This patterning is controlled by the products of two *bric-a-brac* genes, *bab1* and *bab2*. The Bab proteins repress pigmentation and are produced in segment-specific and sex-specific patterns in the pupal abdomen during development. In *D. melanogaster*, Bab function is required not only for pigmentation but also for trichome development.

The authors analysed 13 species of *Drosophila* and found several different patterns of *bab2* expression. In most of the species, patterns of *bab2* activity were well correlated with the pigmentation of the adult flies, suggesting that, in these animals, Bab2 does indeed function as a regulator of pigmentation and is involved in the evolution of similar pigment changes in different lineages. But in some species, *bab2* activity was correlated not with pigmentation but instead with the pattern of trichomes on the abdomen. So although pigmentation and trichome patterning changes are often coupled, their evolution has become uncoupled in some groups. Gompel and Carroll suggest that the two traits become uncoupled when they are under different selective pressures. For example, when pigmentation changes, but trichomes do not, the biological function of the latter might somehow be important for survival.

Both studies suggest that although many genes are involved in the development of physical characteristics, some evolutionary changes — including examples of convergence, in which independent evolutionary events in different species result in similar physical characteristics — involve key regulatory points. The *bab2* and *svb* genes might each function as convenient switches for modifying, or turning on and off, a given trait during evolution. Each gene might represent a developmental 'hotspot' for evolution — or, as Sucena *et al.* put it, a regulator that preferentially accumulates evolutionary change. Another possible example of a developmental hotspot can be envisaged during vertebrate limb evolution: changes to genes that determine the duration of limb growth in the embryo might have been important in the evolution of specialized limb types, such as the dolphin flipper[6].

Although not all instances of trait convergence rely on the same genetic mechanism, these studies have uncovered several instances that do. And this creates a headache for biologists struggling with the concept of homology (in developmental genetics, homologous characteristics are defined as being 'identical by descent', which means they are derived from equivalent genetic networks in common ancestors rather than arising independently in separate lineages). Loss of trichomes in different lineages is traditionally considered an example of parallel evolution and not true homology, because the loss was not inherited — instead, it arose independently in the different species. But if changes in the same gene are involved in each lineage, we could perhaps say that trichome loss is homologous at the level of the underlying process.

The activity of *bab2* and *svb* has been modified repeatedly in evolution, probably largely through changes in the regulatory sequences that switch the genes on or off. Understanding how such modifications have occurred might reveal why some genes and not others are developmental hotspots for driving evolutionary change. Sucena *et al.* suggest that *svb* might be a hotspot because it integrates numerous genetic inputs to control the final output — the pattern of hairs[5].

The two new studies[3,4] have revealed some of the genetic mechanisms that might underlie the evolution of hair loss and pigmentation. But they also point to a future challenge — understanding the function of the physical characteristics, and the causes of the evolutionary change. The integration of evolution with development is beginning to encompass the broad tools of quantitative genetics and functional genomics. This approach will show us how physical changes are generated, but it will tell us nothing about why evolutionary change has taken place in natural environments[9]. The brightest future for evolutionary developmental biology might lie instead with the study of systems in which we can analyse the cause as well as the function of evolutionary change. ■

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Mathematics

The 24-dimensional greengrocer

Ian Stewart

The best way to stack oranges has been evident in markets around the world for centuries, but the mathematics of the problem is far from trivial. The solution for the 24-dimensional case is now within reach.

Put a handful of identical coins on the table, and push them around until they fit together as closely as possible. You will get a honeycomb pattern, or hexagonal lattice, in which each coin is tightly surrounded by six others (Fig. 1, overleaf). This experiment suggests two things: that the 'kissing number' in two dimensions is six, and that the hexagonal lattice is the most efficient way to pack circles. The Greeks could have proved the first statement with complete logical rigour, had they thought to do so. The second statement, though widely suspected, was not properly