adequate means of estimating genetic variability within the species. If the western side of the Andes constituted the only refugial area, then modern-day populations would be expected to have overall genetic similarity. One would also expect lower diversity in populations found in the east, because these would have been established by a limited number of individuals that managed to disperse across the ice-laden divide of the mountain chain. But if there were separate eastern and western refugia, distinct genetic differences would be expected between today's eastern and western populations, because they will have been isolated from each other since at least the start of the last glaciation.

The authors found that the eastern and western populations are genetically distinct, so it seems that the single-refugium hypothesis must be abandoned. On the face of it, it is surprising that warmth-demanding species survived in the interior of the South American continent, which one might expect to be colder than the coasts. Perhaps areas east of the Andes had relatively little snowfall (because of the precipitation shadow effect), limiting ice development and leaving some sites ice-free.

The genetic patterns of the eastern populations are also diverse, suggesting that there were many refugia on the Argentinian side of the Andes. The more southerly of the eastern populations are the most varied, implying that those in the northeast were derived from those in the south as a result of postglacial spread. This is also surprising: one would expect refugia for temperate trees to be found at warmer latitudes further north. Premoli *et al.* question whether the extensive latitudinal movements seen widely in the Northern Hemisphere are to be expected in the Southern Hemisphere, where very different climatic patterns and less extreme variations were experienced in the Pleistocene. It seems that the biotic impact of the glacial cycles, in terms of both the latitudinal extent of vegetation shifts and the frequency of extinctions, was greater in the Northern Hemisphere. For Northern Hemisphere biogeographers, the south is clearly a foreign country and plants do things differently there. ■

Peter D. Moore is in the Division of Life Sciences, King's College London, Franklin–Wilkins Building, 150 Stamford Street, London SE1 8WA, UK.
e-mail: peter.moore@kcl.ac.uk

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Earthquake science

Shaking faults loose

Chris Marone

Earthquakes often induce aftershocks on other faults, but the mechanisms remain elusive. An innovative analysis tells us more about the effects of dynamic stresses caused by the passage of seismic waves.

How does an earthquake on one fault affect the likelihood of failure on nearby faults? In tackling this question, Kilb *et al.*[1] (page 570 of this issue) distinguish between two different triggering effects and document the possible influence of one of them — seismic waves.

For anyone who has ever sat down with a child to play *Don't Spill The Beans* — a game in which players take turns adding beans to a shallow pot that pivots like a see-saw — it will be no surprise that earthquake triggering depends on some details of the initial earthquake rupture. In *Don't Spill The Beans*, dropping a bean in the pot is more likely to cause a spill than carefully adding one over the pot's pivot, even though the static load is the same in both cases. Likewise, if fault slip occurs slowly and without dynamic shaking (add that bean cautiously) the question is whether the static stress transferred to the surroundings is enough to cause failure on neighbouring faults. But earthquakes also produce seismic waves which cause intense shaking in the immediate region of the fault concerned. These dynamic stresses might also trigger aftershocks —rather like bumping the pot.

A key problem in earthquake triggering is that of understanding the effects of static stress transfer compared with those of dynamic stresses and shaking-induced fault weakening. Until now, distinguishing between these effects has been possible only when the secondary earthquake is triggered far from the original[2]. In this situation, changes in static stress are negligible; only dynamic stresses are significant. But 'directivity', which can amplify shaking in the direction of earthquake rupture, provides a way to distinguish between dynamic and static stresses close to an earthquake. Kilb and co-workers[1] apply this approach to the magnitude 7.3 Landers earthquake of 1992. They find that aftershocks are more likely in areas of high dynamic shaking — as long as the change in static stress does not have the opposite effect and inhibit fault failure.

Research on earthquake triggering and fault interaction has been driven in part by the need to understand changes in seismic hazard on human timescales. The situation is particularly clear in the San Francisco Bay area (Fig. 1, overleaf): a large earthquake on either the San Andreas or the Hayward fault could increase or decrease seismic risk across the entire area. But how do we codify fault interaction, and what causes one earthquake to trigger another?

One factor is surely the transfer of static (ambient) stresses[3,4]. Earthquakes release the stresses supported by faults and, because earthquakes are shear failures — slip parallel to the fault dominates over any net perpendicular (normal) motion — they primarily alter the shear stresses parallel to the fault. But on some faults both the normal and shear stresses are affected. Fault strength is a friction problem, so understanding earthquake triggering by stress transfer involves evaluating whether the change in so-called Coulomb failure stress (shear stress plus normal stress multiplied by the coefficient of friction) is enough to cause fault failure or inhibit it. Close to a rupture, where the potential for damaging aftershocks is greatest, the change in static stress may be of the same order as the amplitude of dynamic shaking (see Fig. 1 of the paper on page 570). So in this circumstance, aftershocks could be the result of changes in static stresses, dynamic stresses or both.

Kilb and co-workers[1] show how the question can be addressed in the immediate vicinity of large earthquakes. They also introduce a new wrinkle by explicitly acknowledging that the frictional properties of faults are likely to be altered by dynamic shaking. Their thinking is sound — we have all seen stubborn jar lids come off after a good whack against the kitchen counter — but the principle has been difficult to test with previous methods and data.

In theory, the effects of dynamic stresses carried by seismic waves can be evaluated using the Coulomb failure stress and the approach developed for static stresses. But two difficulties arise.

First, the most sensitive way to evaluate fault interactions is to measure changes in the rate of seismicity. Ideally, one would evaluate variations in seismicity rate as a function of time, in the form of Fig. 1 of Kilb and co-workers, and compare them directly with the dynamic (time varying) and static Coulomb failure stress. But in most cases it is not possible to measure seismicity rates with sufficient temporal resolution. Second, we do not expect instantaneous failure of another fault, even when the stresses exceed a nominal threshold. Modern friction laws[5] show that brittle frictional strength depends on strain-rate and slip history in a way that produces delayed failure in many cases. The effects are small, but

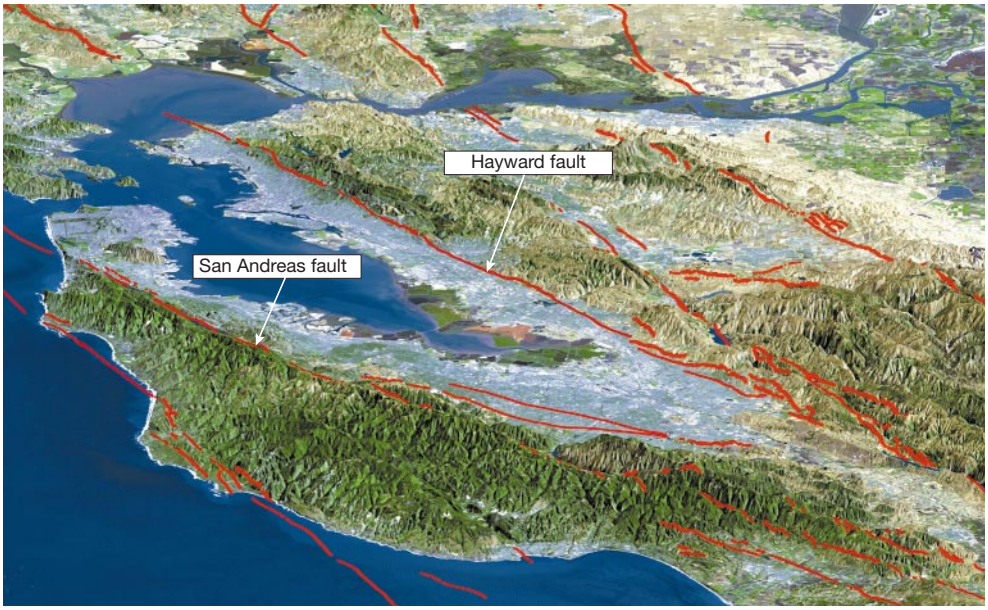

Figure 1 **The San Francisco Bay area is cut by several large faults, including the San Andreas, which runs through the city of San Francisco, and the Hayward fault east of the bay. A large earthquake on one fault could trigger earthquakes on other nearby faults, but the mechanisms of such interactions are poorly understood. Possible triggering agents include transfer of static stresses, dynamic stressing by seismic waves, or shaking-induced fault weakening; the latter two in particular are addressed by Kilb *et al.*[1]. Distinguishing between these mechanisms is important for understanding earthquake-induced changes in seismic hazard. (Figure modified from ref. 3.)**

critical to the initiation of earthquake-like instabilities[6].

Kilb *et al.* have taken advantage of strong rupture directivity in the Landers earthquake to distinguish between dynamic and static stresses. Directivity effects become significant when the speed of rupture propagation approaches that of radiated seismic waves. Dynamic stress amplitudes are increased in the propagation direction, but static stresses are unaffected. The Landers mainshock propagated from southeast to northwest, producing much greater shaking to the northwest. Kilb *et al.* show that this region of high dynamic stressing extended at least 100 km from the end of the mainshock, producing large differences in the spatial pattern of static and dynamic stresses, and identifiable aftershocks to the northwest.

In the same way that increases in Coulomb stress promote fault failure, stress decreases should inhibit failure, and one factor that Kilb and co-workers do not address is such 'stress shadowing'[3]. Proponents of static-stress-transfer hypotheses for fault interaction point out that neither dynamic stress transfer nor fault-weakening by dynamic shaking can explain stress shadows. This could seem a criticism of Kilb *et al.*'s approach. But to my mind it is unlikely that dynamic stressing would produce stress shadows in any case: dynamic shaking is likely to strengthen faults only under unusual conditions. One way to prove the role of dynamic weakening would be to document a shaking-induced increase in seismicity within a static stress shadow, but this would be difficult given present data limitations.

Distinguishing between the influence of static and dynamic stresses in earthquake triggering is intriguing, and may help in building a more general understanding of fault interaction. In the future, initiatives such as EarthScope[7] should allow detailed study of surface deformation and earthquake-induced strain changes in the Earth's crust. Those data, and more work of the sort reported by Kilb *et al.*, will shed further light on earthquake triggering and the associated changes in seismic hazard. ■

Chris Marone is in the Department of Earth, Atmospheric and Planetary Sciences, Massachusetts Institute of Technology, Cambridge, Massachusetts 02139-4307, USA.
e-mail: cjm@westerly.mit.edu

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Osteoimmunology

Bone versus immune system

Joseph R. Arron and Yongwon Choi

A molecule on activated T cells triggers bone loss by switching on bone-resorbing cells. Fortunately, it seems that this mechanism is kept in check by another molecule, secreted by the T cells.

The importance of bone to the immune system is well known: immune cells form in the bone marrow. But the importance of immune cells to bone is less clear. Little is known beyond the fact that normal bone growth and restructuring are disrupted in disorders such as autoimmune diseases. Several years ago, however, a particular molecule on the surface of activated T cells was found to activate bone-resorbing cells[1]. This raised a problem: T cells are working constantly to fight off the universe of foreign particles in which we live, so, at any point in time, some T cells are activated. What prevents these T cells from causing extensive bone loss? On page 600 of this issue, Takayanagi and colleagues[2] provide an answer: T cells also secrete a molecule that inhibits the development and activation of the bone-resorbing cells. This finding underscores the dynamic relationship between bone and immune system.

Often thought of as a rigid, unchanging entity, skeletal bone is actually the result of a dynamic process involving the secretion and resorption of the bone matrix. These opposing actions are carried out by two cell types — osteoblasts and osteoclasts, respectively — and must be kept in balance to maintain skeletal integrity and calcium metabolism, as bone is the main source and repository of the body's calcium. The most common problem with this balancing act occurs when the rate of resorption exceeds the rate of mineral deposition. This results in a loss of bone mass, as seen in osteoporosis, many inflammatory diseases, such as rheumatoid arthritis, and many cancers[3]. One way of maintaining the balance is to keep in check the resorption of bone by osteoclasts.

Over the past few years, the molecular mechanisms underlying the maturation and activation of osteoclasts have been worked out from studies of genetic alterations resulting in skeletal abnormalities in mice, rats and humans. Osteoclasts are