Earthquakes: slow down for safety

Paul Segall

In December 1992, part of the San Andreas fault ruptured. No damage was caused, because instead of lasting for a few seconds, this earthquake sequence took a week. So why are other earthquakes fast?

Is the term 'slow earthquake' a contradiction? It might seem so, as earthquakes typically propagate at velocities limited only by the speed of sound in rock — several kilometres per second. As the rupture propagates, the fault surfaces slip by up to several metres (depending on the size of the quake) in the course of one or two seconds, causing seismic waves to be radiated into the Earth. The more slowly the fault slips, the less efficiently it generates seismic waves; if fault slip is slow enough, no waves are radiated and the earthquake is silent. Indeed, if all earthquakes were slow they would not be very hazardous, because ground shaking causes nearly all the damage in earthquakes. Nevertheless, slow fault slip, or creep, does occur, notably on the central San Andreas fault in California. Most creep events, however, are small and shallow. Because they generate little, if any, seismic energy, slow earthquakes are best studied using highly sensitive strain meters. Earthquakes lasting as long as a few hours have been detected in the past. Now, on page 65 of this issue[1], Linde *et al.* report a week-long sequence of slow earthquakes, which would have been equivalent to a magnitude 4.8 earthquake had it occurred rapidly.

Why do faults sometimes slip slowly and harmlessly, and at other times catastrophically? To answer this question, it is useful to consider the simple spring–slider earthquake model described by G. K. Gilbert in 1884. It was Gilbert who first explained how earthquakes were caused by the accumulation of strain in the Earth's crust: "... this strain increases until it is sufficient to overcome the starting friction on the fractured surface. Suddenly, and almost instantaneously, there is an amount of motion sufficient to relieve the strain, and this is followed by a long period of quiet, during which the strain is gradually reimposed"[2].

Gilbert, who was decades ahead of his time in understanding the mechanics of earthquakes, devised the following analogy[2]:

"Attach a rope to a heavy box and drag it slowly, by means of a windlass, across a floor. As the crank is turned, the tension in the rope gradually increases until it suffices to overcome the starting friction, as it is called.

"Once started, the box moves easily, because sliding friction is less than starting friction. The rope shortens or sags until its tension is only sufficient for the sliding friction, and it would continue in that state but that the box, having acquired momentum, is carried a little too far. This slacks the rope more, and the box stops, to be started only when the tension again equals the starting friction. In this way the box receives an uneven, jerky motion. Something of this sort happens with the mountain."

David Parker/Science Photo Library

Danger area: the San Andreas fault crosses the Carrizo plain, about 450 km south of San Francisco. But some sections of the fault can release their strain slowly and safely.

Gilbert's earthquake analogy includes the three key ingredients of any earthquake machine: a driving mechanism (the windlass), which we now understand to be the motion of the Earth's tectonic plates; an elastic element (the rope), which represents the Earth's crust adjacent to the fault and its ability to store elastic energy; and the frictional fault surface (the box–floor interface).

We now know that Gilbert's concept of friction was too simple; the transition from the 'starting' or static friction to sliding friction is not instantaneous, but occurs gradually with increasing slip displacement[3]. In fact, if the transition were instantaneous, slow earthquakes, such as those found by Linde *et al.*, could not exist. Modern laboratory experiments show that the coefficient of friction depends on slip speed and past slip history. If sliding continues at a constant rate, the friction coefficient approaches a unique steady-state value[4].

Whether or not the slider exhibits jerky, stick–slip motion or continuous, stable sliding depends on the interaction of the frictional properties and the elastic loading system. Unstable, accelerating slip occurs only when the frictional strength decreases more rapidly than the elastic element unloads. So stiff systems favour stable slip whereas compliant systems favour unstable stick–slip — try replacing Gilbert's rope with a steel cable or a bungee cord. Stability also depends on the normal stress acting on the fault surface (that is, the weight of Gilbert's box). High normal stress (a heavy box) favours unstable, stick–slip motion, whereas low normal stress favours stable sliding. Of course, the frictional properties enter into the stability equation. Slip can be linearly unstable when the steady-state friction decreases as a function of slip speed, but is generally stable when it increases[4].

Faults in the Earth's crust contain fluid-saturated 'fault gouge', finely fragmented rock material whose properties can greatly influence fault stability. If the fluids within the gouge are maintained at high pressure, the effective normal stress acting across the fault is diminished, promoting stable slip. These arguments point to two competing hypotheses for the stable creeping section of the San An-

dreas: that low-permeability rocks next to the fault trap pore fluids at high pressure, or that these same rocks have intrinsically stable frictional properties.

The dynamics of fluid-saturated fault zones have other complexities. As they shear, granular materials dilate, that is, they increase pore volume, because the grains ride over one another. If exchange of fault-zone pore fluids with the surrounding rocks is slow, then dilation transiently decreases pore-fluid pressure and is thus stabilizing[5]. On the other hand, fault slip generates frictional heat, which, depending on fluid transport properties, can lead to thermal pressurization of pore fluids and so to instability[6].

Other non-fluid effects may also influence fault stability. High-frequency seismic waves trapped in the fault zone can briefly reduce fault strength, promoting instability[7]. And it has recently been suggested that differences in elastic properties on either side of the fault may lead to unstable slip pulses, ripple-like disturbances accompanied by transient decreases in normal stress[8].

These processes can all be studied by numerical simulation, and some have been verified in laboratory experiments. Their relationship to unstable slip on natural faults, however, remains clouded because of our poor knowledge of conditions and material properties at depths at which earthquakes nucleate (typically 10 to 15 km on the San Andreas). The slow earthquakes recorded by Linde *et al.* were near the transition between the creeping segment of the San Andreas and the northern, locked segment, which in some respects may be analogous to the transition from stick–slip to stable sliding that occurs at a depth of about 15 km elsewhere on the fault. Only by comparing theoretical predictions with field and laboratory observations will it be possible to isolate those effects central to earthquake instabilities. In this way, observations of slow and silent earthquakes may lead to a better understanding of how their faster, more damaging cousins begin. □

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1. Linde, A. T., Gladwin, M. T., Johnston, M. J. S., Gwyther, R. L. & Bilham, R. G. *Nature* **383,** 65–68 (1996).
2. Gilbert, G. K. *Am. J. Sci.* **27,** 49–53 (1994); reprinted from the *Salt Lake Tribune* of 30 September 1883.
3. Linker, M. F. & Dieterich, J. H. *J. Geophys. Res.* **97,** 4923–4940 (1992).
4. Ruina, A. L. *J. Geophys. Res.* **88,** 10359–10370 (1983).
5. Rudnicki, J. W. & Chen, C.-H. *J. Geophys. Res.* **93,** 4745–4757 (1988).
6. Lachenbruch, A. H. *J. Geophys. Res.* **85,** 6097–6112 (1980).
7. Melosh, H. J. *Nature* **379,** 601–606 (1996).
8. Andrews, D. J. & Ben-Zion, Y. *Trans. Am. Geophys. Union* 414 (EOS Suppl. **76,** No. 46, 1995).

TRANSCRIPTION

A growing coactivator network

Ralf Janknecht and Tony Hunter

STEROIDS, thyroids, retinoids and vitamin D all bind to nuclear hormone receptors, which interact with promoter elements to regulate gene transcription. Three reports[1–3], including one on page 99 of this issue[2], show that nuclear hormone receptors depend on the coactivator CREB-binding protein, CBP, or the related P300 protein, to trigger RNA polymerase II and thus transcription. CBP and P300 are huge nuclear molecules, consisting of more than 2,400 amino acids, and can interact with a variety of different DNA-binding factors and also with components of the basal transcription machinery[4]:

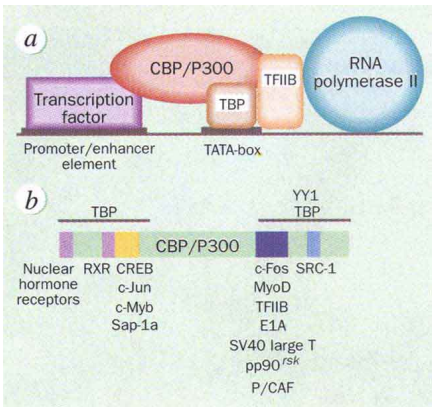

FIG. 1 *a*, Model of CBP/P300 bridging between a sequence-specific transcription factor and components of the basal transcription machinery. TBP, TATA-box-binding protein; TFIIB, transcription factor IIB. *b*, Binding sites of the various partners that interact with CBP/P300. RXR, retinoid X receptor. CREB, c-Jun, c-Myb, Sap-1a, c-Fos, MyoD and YY1 are all DNA-binding factors. E1A and SV40 large T, viral proteins that suppress activation of transcription. pp90^{rsk} and P/CAF, enzymes recruited by CBP/P300. SRC-1, coactivator which interacts with both CBP/P300 and nuclear hormone receptors.

CBP/P300 thereby links these two classes of proteins (Fig. 1). Indeed, the ability of adenoviral E1A oncoprotein and SV40 large T antigen to interfere with these interactions explains how they suppress transcriptional activation[5].

The new reports[1–3] show that nuclear hormone receptors interact directly with CBP/P300 through their ligand-binding domains, and that hormone binding greatly stimulates this interaction. Two receptor-interaction domains have been mapped within CBP: the amino-terminal 101 amino acids, which bind to all tested nuclear receptors, and amino acids 356–495, which bind weakly to the retinoid X receptor but not to the retinoic acid or thyroid hormone receptors[1,2]. Nuclear-receptor-mediated transactivation is enhanced by CBP/P300 in a hormone-dependent manner *in vivo* and *in vitro*, and is blocked by injection of anti-CBP antibodies into cells. So the interaction between CBP/P300 and nuclear hormone receptors is essential for their transactivation function[1,2].

In addition to the coactivators CBP/P300, nuclear hormone receptors interact with a variety of other coactivating or silencing mediators. One of them (SRC-1) interacts with a carboxy-terminal region of CBP/P300 (refs 1, 6). Nuclear hormone receptors can bind simultaneously to CBP/P300 and SRC-1, in principle forming a ternary complex bound to DNA. This suggests that multiple coactivators may jointly modulate transactivation mediated by a single DNA-binding transcription factor. A corollary of this may be that multiple components of the basal transcription machinery are contacted, but how this finally triggers initiation of transcription remains a mystery.

A further element to the story has come from the discovery of a P300/CBP-associated factor (P/CAF)[7], which competes with viral E1A protein for binding to CBP/P300: this competition is responsible for the counteraction of E1A-induced mitogenicity by P/CAF. Functionally, P/CAF is an enzyme that, *in vitro*, acetylates histones H4 and H3 but not histones H2A or H2B (ref. 7), and may therefore destabilize nucleosomes and promote transcription. Another enzyme recruited by CBP/P300 is the protein kinase pp90^{rsk} (ref. 8), which after Ras-induced activation and nuclear translocation can bind to the same region as E1A and thereby suppress CREB-mediated transcription. In contrast, association of pp90^{rsk} with CBP is required for induction of Ras-responsive genes, possibly because pp90^{rsk} phosphorylation of DNA-bound factors, components of the basal transcription machinery or CBP/P300 itself can enhance transcription of such genes.

Loss of one CBP allele apparently causes Rubinstein–Taybi syndrome, which is an autosomal dominant inheritable disease associated with mental retardation, broad big toes and thumbs, and facial abnormalities[9]. This suggests that CBP can be a limiting component within the cell, and that competition for CBP may allow cross-talk between different signalling pathways. Consistent with this, nuclear hormone receptors can antagonize the AP-1 transcription factor, which is composed of Fos and Jun proteins, and overexpression of CBP or P300 abrogates the inhibition of AP-1 function by hormone-bound nuclear receptors. In addition, a synthetic ligand abolishes interaction of the retinoic acid receptor with CBP/P300,