

record of the course of molecular dynamics in a single measurement would be invaluable. In this context, the article by Poulin and Nelson represents a valuable advance.

The authors show how one can obtain—in a single shot—time-sequenced transient absorption spectra of irreversible chemical reactions that are initiated with a laser in a solid sample. To do so they use echelons, which are windows cut in steps to provide stripes of different thickness through which the laser traverses. By crossing two echelons, pixels of square profile are created. Each pixel generates a different time delay, allowing events happening at different times to be probed in a single shot.

Poulin and Nelson use this method to record the caging process of the triiodide ion upon its photodissociation in organic crystals. Caging results in the reformation of the broken bond. The bond reformation proceeds

coherently in the tight cages, while there is significant dispersion in the timing of recombination in the looser solids. Although the observed caging process is reversible, discoloration of the irradiated spot indicates that nonreversible structural change also occurs, precluding the repetition of measurements on the same spot. The work also highlights that in condensed-phase systems, linear spectroscopies are not uniquely interpretable: Simulations are essential to connect the one-dimensional spectra to the underlying multidimensional dynamics (4).

Poulin and Nelson have successfully put into practice the concept of the pixellated window in time. The use of echelons, however, limits the observation window to the picosecond time range. Clever optics will be required to follow processes on femtosecond to millisecond time scales and thus unravel the multiscale dynamics peculiar to solids; for exam-

ple, to follow not only the bond breaking, but also the process of permanent discoloration seen in the crystals used in their experiments.

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GEOPHYSICS

Do Earthquakes Rupture Piece by Piece or All Together?

Chris Marone and Eliza Richardson

Violent shaking and destruction caused by earthquakes are the result of rupture and frictional slip on tectonic faults, and bigger earthquakes break bigger fault segments. But how do brittle ruptures of Earth's crust grow? Seismologic evidence shows that quakes begin in a small nucleation region and propagate at speeds up to 16,000 kilometers per hour. Two competing models of rupture growth describe this expansion (see the figure). In the crack model, the nucleation region slips throughout the quake and the slipping region expands until the rupture stops, a process akin to stretching a penny into the size of a half-dollar. In the pulse model, only a small portion of the total fault area slips at any one time, so as to cover the fault surface the way an inchworm crawls. Distinguishing between the two models is important for hazard assessment because they predict different degrees of strong shaking and ground acceleration with distance from the nucleation site. Recent seismological observations favor the pulse model, but efforts to connect these data with theoretical models of

earthquake physics have been stymied because rupture pulses have never been reliably observed in the laboratory. However, as reported on page 1765 by Lykotrafitis *et al.* (1), new laboratory experimental evidence on brittle fracture, showing the existence of pulse-like ruptures and the conditions under which they exist, may help resolve the debate.

Lykotrafitis and co-workers sheared photoelastic material in frictional contact in a dynamic impact apparatus and monitored rupture propagation with high-speed photography. They show that the rupture propagation mode varies systematically with the strength of initial forcing (as produced by impact speed). Pulse-like ruptures are favored by slower impact speeds relative to those for crack-like ruptures. Also, the frictional slip velocity during rupture is lower for pulse-like ruptures than for crack-like ruptures. Thus, pulse-mode ruptures are the slow cousins of breaks that propagate as classical cracks. The data of Lykotrafitis *et al.* show a clear relationship between stress level and rupture propagation mode, with larger shear stress levels resulting in crack-like propagation.

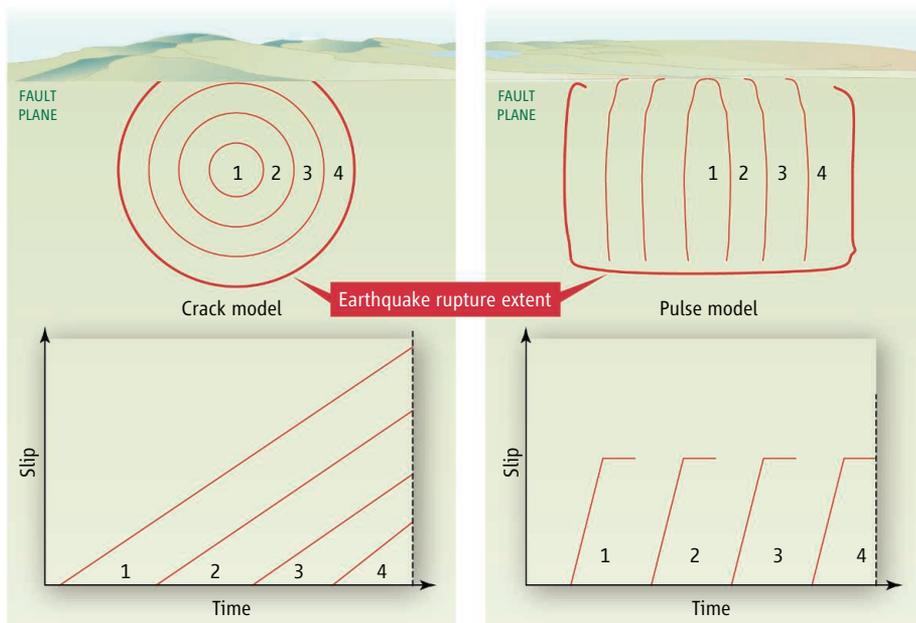
The experiments of Lykotrafitis *et al.* address perhaps the most important question in earthquake physics: What controls seismic slip

Laboratory measurements are being used to resolve which of two models is better at explaining how Earth's crust ruptures to create earthquakes.

at a point on a fault? Virtually every quantifiable aspect of earthquakes depends on slip, but local fault slip cannot be measured directly from seismograms. If the initial tectonic shear stress determines slip, it would imply that dynamic frictional strength is zero and that stress on the fault drops to zero during an earthquake. In this scenario, seismic slip ceases because the local energy budget is depleted, but this runs counter to laboratory data on frictional stick-slip and seismic estimates of radiated energy, which indicate that seismic stress drop is a mere 10% of the tectonic stress level. Or, if the boundary conditions of fault strength determine seismic slip, then earthquake rupture stops when it encounters a strong barrier. Alternatively, frictional behavior during rupture—possibly abetted by dynamic variations in normal stress—could determine slip. The self-healing pulse model belongs to this last class of models. In order for rupture to propagate as a slip pulse, the fault must strengthen rapidly after slip so that local slippage ceases.

The crack model of earthquake rupture emerged in the 1970s as an extension of the mechanics of dislocations in solids, and much progress has been made in connecting seismic phenomena with the mechanics of

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dynamically propagating cracks. In the past decade, this model has been challenged by seismic observations and theoretical work that support a slip-pulse model. However, modern earthquake science (2) is based on the recognition that seismic records contain a spectral signature of rupture, with larger quakes releasing longer period waves such that the maximum period scales with rupture area. This observation dovetails nicely with crack models, for which the whole rupture area slips simultaneously, but presents a problem for self-healing pulses because the slipping region (see the figure), which represents the largest coherent spatial dimension, does not scale with overall rupture dimension. Moreover, earthquakes that propagate as pulses must accumulate the slip appropriate for the rupture size in a time window that itself does not scale with rupture duration.

There are several aspects of the scaling relationships among earthquake source parameters that are problematic for the pulse model. The simplest observable source parameters of earthquakes include rupture length and width, average slip, and the seismically induced reduction in local tectonic stress. A large catalog of data and their scaling relations are consistent with the crack model for earthquake rupture but require special (and in many cases physically implausible) circumstances to support the pulse model. For example, do ruptures know how big they will be when they begin to grow? Scaling relations indicate that small and large earthquakes begin the same way, but the pulse model does not easily fit within this framework. It is perhaps worth noting that these two end members are not the only possible rupture modes. In

fact, the experiments discussed by Lykotrakis *et al.* show both modes of rupture in the cases where pulses were generated. Extending these results by applying them to geologic materials and field observations will be the next challenge.

Rupture mechanisms. The diagram shows two different ways that ruptures might occur in a section of Earth (the fault plane faces front). In the crack model (**top left**), the nucleation region 1 slips throughout the quake, the slipping region expands until rupture stops, and the entire fault plane slips simultaneously. The lines (**bottom left**) represent slip history for four regions on the fault plane. In the pulse model (**top right**), only a small portion of the total fault area slips at any one time. All points on a pulse-mode rupture plane exhibit identical slip histories (**bottom right**).

As permanent seismic detection networks increase in density and we accumulate high-quality seismic recordings close to large earthquakes, the mode of rupture propagation may be known routinely. But to understand the physical processes that govern dynamic rupture nucleation, growth, and arrest, we will need additional information provided by careful laboratory studies such as those described by Lykotrakis *et al.*

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MOLECULAR BIOLOGY

Little Molecules with Big Goals

Bert W. O'Malley

Sets of master genes may control the expression of the relevant proteins involved in complex cellular processes such as growth and metabolism.

The field of nuclear receptor coregulators is approaching its 11th birthday (1), and the number of known constituents—coactivators and corepressors—has grown to more than 200 (2). Their characterization began modestly, with a simple concept that a limited number of coactivators existed that functioned merely as “adaptors” for stabilizing the cellular machinery that transcribes genes. But coregulator actions have expanded to chromatin modification and remodeling, initiation of transcription, RNA elongation and splicing, and protein degradation (3). In fact, we now know that coregulators comprise multiple (5 to 10) proteins of large regulatory machines, conveying the enzymatic activi-

ties needed to achieve diverse functions (3, 4).

A central question emanates from the plethora of recent information on this class of regulatory molecules: Why have genes that code for so many coactivators and corepressors evolved? Are there big-picture goals behind their evolution, or do they exist simply to provide a series of disconnected catalytic reactions to activate or repress gene expression? Given the recent evidence for their expansive roles in biology, the latter seems clearly not to be the case. Rather, coregulators appear to constitute “little molecules with big goals” and likely represent the elusive “master genes” that were first proposed nearly 50 years ago (5), albeit different in form and substance.

The body controls, in a temporally and spatially coordinated manner, hundreds of genes needed to affect any single major complex process such as metabolism,

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