PERSPECTIVES: GEOPHYSICS

A Strained Earth, Past and Present

John G. Ramsay

Some geological processes, such as earthquakes and volcanic eruptions, are catastrophically fast, but most movements of Earth's crust are very slow by human standards. These so-called tectonic processes lead to the uplift of mountains, the formation of oceanic rift systems, and the horizontal sliding of plates. Oceanic plates separate by between 1 and 20 centimeters per year (*1*); this extensional displacement is generally believed to be counterbalanced by the subduction of surface crustal material along narrow subduction zones.

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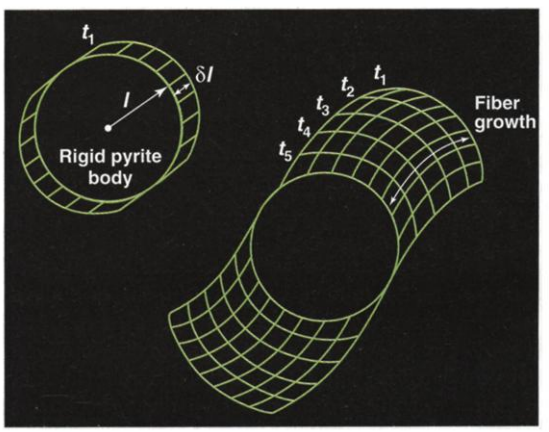
The subduction of one crustal plate beneath another deforms other plates adjacent to the subduction zone, as does shear between sideways moving plates. Some of this deformation is brittle and is released by local earthquakes, but ductile rock creep also takes place in both the upper (subducting) and the lower plate. In the upper plate, such ductile flow often leads to crustal thickening, resulting in mountain building at certain plate contacts. The internal deformation rates within converging or transforming plates are difficult to evaluate. On page 2195 of this issue, Müller *et al.* (*2*) report a new technique for overcoming this problem in past and present orogenic (mountain-building) regions, many of which are known to have particularly complex deformation patterns.

To solve the problem of measuring strain rates, geologists have previously looked to the methods used by glaciologists to derive displacement and strain rates of glacial flow. Glaciologists "peg" the upper glacier surface and observe the displacement rates of these markers (*3*); from the displacement rates, they then compute the strain rates. Similar techniques have been applied by geologists in regions of present tectonic activity. These geodetic methods have, until recently, only enabled a few measurements of sufficient accuracy and proximity that could bear on the systematic measurement of regional surface displacements (*4*). However, the recent development of the global positioning system (GPS), which uses satellite data point fixing on well-established observational stations, has led to a revolution in data accuracy, providing a basis for establishing present surface displacements (*5*) and displacement rates from which average surface strain rates may be computed.

Geological interest in strains and strain rates also extends to past orogenic (mountain-building) activity. Unfortunately, we cannot identify fixed "pegs" in orogenic zones. The structural forms of regions that are no longer tectonically active suggest that strain accumulation was complex; many orogenic belts of the past were built up by a complex interaction of differently directed displacement fields of differing strain intensity. Folds are superposed on preexisting folds; shear zone and fracture patterns suggest substantial differences in the orientations of the axes of the principal stress systems that led to their formation (*6*). To decipher these patterns and determine the systematic regional changes of strain and stress that have occurred in the crustal deformation zones formed in the past are thus formidable tasks.

Faced with this problem, geologists have determined the finite strain characteristics seen in individual outcrops and put these together to make a comprehensive regional assessment of the regional strain field. The forms of deformed objects of known initial shape embedded in a rock can be used to compute the state of finite strain (*6*, *7*), which results from the accumulation of small strain increments over a period of time. To interpret these increments and define the strain rates, a geometrical method for determining the incremental system for strain accumulation is required. If geological constraints on the progressive buildup of finite strain (*8*) are accepted and steady-state flow is assumed, then average strain rates between 10^{-13} and 10^{-15} s^{-1} are deduced from the finite strain data.

The geometry of changing incremental strains can be determined from certain structural features seen in naturally deformed rocks (*9*). Certain rocks contain deformation-resistant markers; for example, pyrite crystals are well known to be deformation resistant. The rock around such deformation-resistant objects flows away from the marker. The space that evolves between matrix and marker becomes progressively filled with crystalline material derived from fluids in the deforming material. The crystals of this material are often fibrous, and the fiber axes are controlled by the separation directions of the matrix and the deformation-resistant object in the "pressure fringe" around the object. The matrix pulls away from the marker in the direction of the maximum incremental stretch length, δl (see the figure). By standardizing this value with respect to the radius of the undeformable object, the value of the principal axis of incremental extension, $\delta l/l$, can be evaluated. As the deforming system changes its principal stretching axis, so will the fiber orientations. The crystal fibers change the direction of their long axis (but keep their optic orientation through fiber growth sense), allowing evaluation of the changing direction and values of incremental strain.



Strain increment measurement with fibrous crystals in pressure fringes. A spherical rigid pyrite body of radius l shows pressure fringe growth of length δl in the direction of incremental maximum strain **(left)**. Successive stretch increments over time periods t_1, t_2, ..., t_n **(right)** enable the geometric increments to be calculated.

The very important advance of Müller *et al.* (*2*) has been to determine the Rb-Sr isotopic ratios on very small crystals of chlorite within the fiber structures and so determine the differing formation ages along the crystal fibers. By combining these age data with measurements of incremental strain, it becomes possible to put figures on the rates of incremental strain growth. Their strain rates of 1 to 8×10^{-15} s^{-1} fit rather well within the average strain rate fields previously suggested for geological deformational processes (*8*). Refined techniques for analyzing small sample volumes with increased isotopic precision now open a very exciting possibility for evaluating the geo-

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logical strain rates of past processes. By isotopic dating of the growing crystal fibers, it should be possible to constrain the geometric data in a time frame that enables the strain rates at differing times within the evolution of rock deformation to be calculated. Field geologists, in collaboration with isotope chemists, now have to seek rocks appropriate to undertake the type of analysis set out in the report.

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PERSPECTIVES: MOLECULAR BIOLOGY

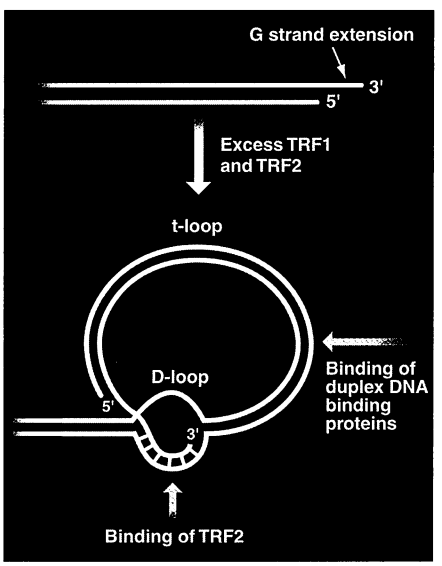
Telomeres Keep On Rappin'

Victoria Lundblad

Many fundamental cellular processes have been conserved throughout evolution, and the biology of telomeres (the ends of chromosomes) is no exception. In most eukaryotes, chromosome ends consist of tandem reiterations of a closely related G-rich repeat sequence, with the G-strand protruding as a single-strand extension. The enzyme telomerase, which synthesizes these telomeric repeats, is similarly highly conserved from unicellular eukaryotes to human cells. Studies in both yeast and human cells also point to a central role for proteins that bind the duplex regions of telomeres in maintaining telomere length homeostasis. But, a missing piece of the telomeric puzzle has been the apparent lack of conservation between human duplex DNA binding proteins and those of budding yeast. Now, in a recent issue of *Cell*, de Lange and colleagues (*1*) describe a human counterpart of the well-characterized yeast duplex DNA binding protein, Rap1, illustrating an additional point of convergence between telomere biology in unicellular and vertebrate organisms.

In budding yeast, Rap1 is a major regulator of telomere length. It binds with high affinity to tandem GGTGT sites present within the irregular $G_{1\text{-}3}T$ telomeric repeat tracts found in yeast telomeres (*2*). The carboxyl terminus of Rap1 interacts with two different sets of proteins, thereby exerting effects on transcriptional repression of subtelomeric genes (through binding of the Sir proteins) and telomere length (through binding of the Rif proteins). Targeting additional copies of this protein interaction domain to the telomere is sufficient to reset telomere length. This has led to the proposal that a protein-counting mechanism can discriminate the number of Rap1 proteins bound to the telomere (*3*). This establishes a negative-feedback loop that is thought to modulate telomere length by inhibiting access of telomerase to the chromosome terminus. As Rap1 is capable of bending DNA, one mechanism proposed for this length-sensing process is a number-dependent switch that flips the telomere between a telomerase-accessible "open" structure and a "closed" conformation that is refractory to telomerase action.

Duplex binding factors also modulate telomere length in human cells, but the picture is more complex. Not one, but two related proteins, TRF1 and TRF2, bind the TTAGGG repeats found at human chromosome termini (*4*). The amount of TRF1 and TRF2 bound to chromosome ends inversely correlates with the length of the telomere (*5*). Increased expression of either TRF1 or TRF2 in a telomerase-positive cell line results in a gradual decline in telomere length (*5*, *6*). Conversely, removal of endogenous TRF1 from telomeres leads to telomere elongation (*6*). These observations indicate that a longer telomere recruits more TRF1 and TRF2 protein, thereby blocking the ability of telomerase to act on that terminus. Thus, results in both yeast and human cells suggest that the telomere itself inhibits further elongation by telomerase when telomere length exceeds a certain threshold.



Loop the loop. A t-loop structure forms when the 3′ G-strand extension at the end of a chromosome (telomere) invades duplex telomeric repeats, thereby forming a displacement loop (D-loop). Embedding the 3′ end of the chromosome not only has the ability to sequester the telomere from telomerase action, but also can protect the terminus from exposure to DNA damage checkpoints and repair activities. Consistent with a role for t-loops in chromosome end protection, loss of TRF2 from telomeres induces rapid telomere dysfunction and subsequent chromosome end-to-end fusions (*11*).

Although the properties of duplex telomere binding proteins in yeast and humans mirror each other, it has been perplexing that the vertebrate TRF proteins do not bear any notable sequence similarity to the budding yeast Rap1 protein, nor have orthologs of the vertebrate TRF proteins been identified in the sequenced yeast genome. The current report from de Lange's group potentially supplies a missing link by demonstrating that a human ortholog of Rap1 is indeed present at human telomeres, although in somewhat unfamiliar territory (*1*). Like its yeast counterpart, hRap1 overexpression results in telomere elongation, showing that hRap1 contributes to the length-sensing mechanism that sets human telomere length. However, one intriguing difference distinguishes the budding yeast and mammalian Rap1 proteins: hRap1 associates with telomeric chromatin through an interaction with TRF2, rather than by binding directly to the DNA.

The identification of hRap1 strengthens the argument for a common mechanism for negative length regulation, but it raises a new question: Why does budding yeast lack TRF proteins? De Lange and co-workers suggest an evolutionary model. They propose that the ancestral telomeric complex, still represented in human cells, consisted of a TRF-like protein that bound telomeric DNA and tethered a Rap1-type protein to the telomere. During the evolution of budding yeast, loss of TRF function and concomitant acquisition of Rap1's ability to bind DNA—perhaps arising as the result of a divergence in telomeric sequence (*7*)—may have changed the specific players involved, but preserved the key components that regulate telomere extension.

Does the proposed protein counting mechanism for telomere length proceed by the same mechanism in yeast and human cells? A framework for modeling telomere length regulation comes from the recent

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