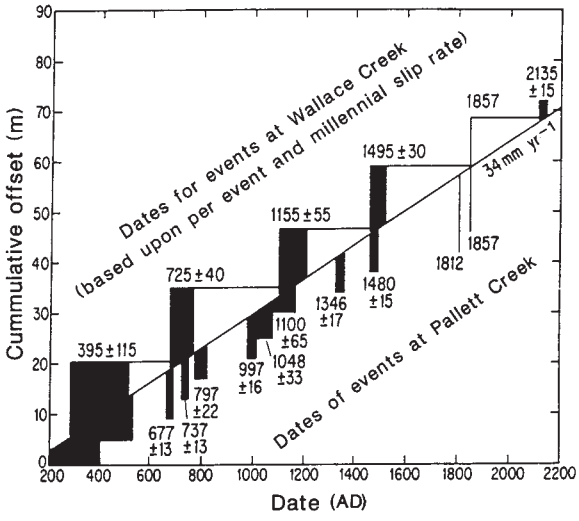
GEOPHYSICS

Earthquakes as chaos

Christopher H. Scholz

EARTHQUAKE hazard analysis has for some time been based on the supposition that faults are broken into discrete segments that independently fail in 'characteristic' earthquakes. Long-range predictions can then be made based on the identification of these segments and estimating their recurrence times. It is becoming increasingly obvious that this description is far too simple. One problem is that the segments are not entirely independent: two or more adjacent segments sometimes rupture together to produce a much larger earthquake than would otherwise have been expected. On page 234 of this issue[1], Huang and Turcotte provide a crisp explanation of this phenomenon. They have constructed a model in which two sliders with different friction are coupled together by one spring and driven at the same rate by another. They find that this system evolves into chaos, in which the two sliders exhibit independent stick–slip oscillations for some periods but sometimes couple together to slip in a larger event.



The history of slip in earthquakes at two sites about 200 km apart on the San Andreas fault (from ref. 2).

An example of this behaviour can be seen in the history of slip in earthquakes at two sites, Wallace Creek and Pallett Creek, some 200 km apart on the San Andreas fault (see figure) which has been deduced for the past 2,000 years using techniques akin to those used in archaeology[2]. The last great earthquake to have ruptured this section of the fault in 1857 ruptured through both of these sites, slipping 9 m at Wallace Creek and 2 m at Pallett Creek, values of slip that appear characteristic of these sites for the prehistoric earthquakes as well. Earthquakes at Pallett Creek occur in clusters of two or three events, each of which corresponds in time to one event at Wallace Creek. Although impossible to prove with radiocarbon dates, it is reasonable to suppose that the Wallace Creek events are coupled events like that of 1857, in which both sites were ruptured. If the coupled events were the last in each Pallett Creek sequence, then in detail the behaviour is astonishingly like Huang and Turcotte's model, in which Wallace Creek corresponds to the stronger block and Pallett Creek the weaker. After each coupled event, there is a long quiescence at Pallett Creek followed by a series of more frequent events, culminating in another coupled event, just as this model predicts (see their Fig. 3, on page 235 of this issue).

Other interesting cases have yet to be evaluated. The Loma Prieta earthquake has refocussed attention on seismic hazard in the San Francisco Bay Area. One intriguing question is the possibility of coupling between the Hayward and San Andreas faults on either side of San Francisco Bay. In 1836 there was a magnitude-7 event on the northern half of the Hayward fault, followed two years later by a similar San Andreas earthquake on the San Francisco Peninsula. In 1865 a magnitude-6.5 event in the vicinity of Loma Prieta was followed three years later by a magnitude-7 event on the southern Hayward fault. An analysis of this problem with Huang and Turcotte's model should be revealing.

The general problem appears to be considerably deeper than these examples indicate. Thatcher[3] has shown that the rupture segments are not stationary in time: their shape and spatial distribution on the fault change from earthquake cycle to cycle (see Max Wyss's recent News and Views article[4]). Furthermore, the recurrence of rupture on any fault sector is now known not to be periodic but lognormally distributed[5]. These factors indicate behaviour that is far too complex to be described by the low-dimensional chaos of the type studied by Huang and Turcotte, but progress is being made on this front as well.

The model of Bak *et al.*[6] of a spatially extended dissipative system which evolves to a self-organized critical state is a very attractive general model of earthquakes, as they have pointed out[7]. It predicts that earthquakes obey a fractal size distribution — a distribution known historically in seismology as the Gutenberg–Richter

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therefore compressed) in any way. An alternative explanation is that some factor other than the dimensions of the class I binding site controls the length of peptide isolated by acid extraction. One possibility is that the mechanism which transports peptides from the cytosol to the endoplasmic reticulum, and which provides peptides for binding to class I, is selective for sequences of 8 or 9 amino acids.

Peptide transport is independent of hydrophobic signal sequences, and is therefore distinct from the classical vectorial transport of secreted or membrane proteins (for reviews, see refs 6 and 7). Although there is no clue as to the molecular basis of peptide transport, there is a precedent for transport of peptides across bacterial membranes in the form of oligopeptide permease, which may be selective for sequences of 3–5 amino acids. This molecule is a member of the 'ABC' (ATP-binding cassette) transporters, which have several counterparts in eukaryotes, including the product of the yeast *STE6* gene that transports a defined peptide sequence (for reviews, see refs 18 and 19). So it is conceivable[6] that there is a peptide transporter which selects peptides of particular lengths for translocation across the membrane of the endoplasmic reticulum, and which is defective in the mutants RMA-S and .174/T2. Finally, it may be that controlled proteolysis in the cytosol gives rise to peptides with an average length of 8 or 9 amino acids. By isolating a large number of naturally presented peptides and looking for common features of their N and C termini, it may be possible to identify elements associated with specific proteolysis or transport. □

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relation. But this is not the whole story. For good scientific reasons, Bak *et al.* were careful to evaluate the behaviour of their model far from its boundaries, to avoid any spurious effects arising therefrom. It now seems that this missed an essential feature of earthquake behaviour.

Faults slip unstably in earthquakes only in a narrow depth range between the surface and a depth, commonly about 15 km, at which the rocks become ductile. Thus the problem contains a characteristic dimension not included by Bak *et al.* in their idealization. It has been known for some time that earthquakes of size smaller than this dimension are fundamentally different from those larger, whose rupture is then constrained to be channelled horizontally. 'Small' earthquakes, so defined, obey different scaling laws from 'large' earthquakes[8,9]. Furthermore, although both small and large earthquakes have fractal size distributions, they belong to disjoint sets so that for a given section of fault many more large earthquakes will occur than predicted by extrapolating from small events[10].

Carlson and Langer[11] studied a model with spring slider elements as in Huang and Turcotte's model but which was spatially extended like the Bak *et al.* model. They found that this model also exhibits self-organized criticality and the Gutenberg–Richter law, but owing to what was first thought to be a weakness of their model — its finite size — they observed the 'artefact' that many more large events were produced than expected from the distribution of small events. Following up this serendipitous clue, my colleagues and I studied a model in the form of a narrow strip that deliberately emphasizes the effect of the lateral boundaries[12,13]. This model produces two populations, of small and large events. The large events (running from boundary to boundary) are much more frequent than predicted from the distribution of small events. Concentrating on the large events, we found that the model also exhibits two other primary features of natural seismicity: the large events greatly dominate the moment release rate of the fault, and the recurrence time of large events is lognormally distributed.

In the rather short time since this approach to earthquakes was first discussed at a seminal meeting in 1989 (see my report in News and Views[14]), progress has been very rapid. Whereas the earthquake process is now being viewed as far more complex than before, there is clearly a breakthrough in the making in understanding the underlying physics. It is too early to say what effect this will have on practical matters such as earthquake prediction. □

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PROTEIN CONFORMATION

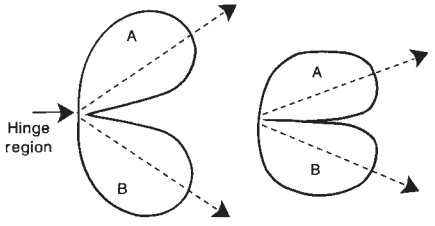
Hinge-bending and folding

Christopher M. Dobson

THE idea that conformational transitions in proteins can involve the relative movement of essentially rigid structural elements is an attractive one, not least because of its simplicity and ready visual impact. Particularly well-known examples are the transitions of allosteric proteins, such as haemoglobin[1] or phosphorylase[2], where individual subunits move relative to each other in a cooperative manner. Within single subunit proteins, a related and intriguing concept has been that of 'hinge-bending', whereby the relative flexibility of short regions of the polypeptide chain allows significant movement of structural regions, or domains, within the cooperative folding unit. X-ray diffraction studies by Faber and Matthews, reported on page 263 of this issue[3], provide a striking illustration of this idea, as well as demonstrating the effects of interactions within crystals on the selection of particular conformational states of proteins.

Faber and Matthews describe experiments with a mutant form of lysozyme from the T4 bacteriophage. T4 lysozyme is an enzyme of 164 amino-acid residues, whose structure consists of two lobes (or domains) of approximately equal size. Although having no apparent sequence similarity, its structure is related to that of the avian and mammalian c-type lysozymes of which that from hen egg white is the best known. It has long been advocated that hinge-bending occurs in egg-white lysozyme[4], but experimental verification has proved elusive[5].

The mutant T4 lysozyme (Met 6 → Ile) crystallizes in a trigonal space group which is isomorphous with the wild-type protein; the structure in this form is very like that of the wild type. Faber and Matthews found, however, that the mutant lysozyme also crystallizes in an orthorhombic space group, containing four molecules in the asymmetric unit. Solving this structure by isomorphous replacement proved to be more complicated than expected, because the relative orientation of the domains of the lysozyme molecules in the orthorhombic crystals differs significantly from that found in the structures of the wild-type and mutant protein in the trigonal crystals. Moreover, it differs substantially between the four molecules within the asymmetric unit. The domains themselves are closely similar in each case, but the hinge-bending angle varies by up to 32°. Despite the large 'global' changes in structure giving rise to a relative movement of certain residues of close to 10 Å, the conformational changes at the hinge-bending site turn out to be small.



A schematic example of one concept of hinge-bending. A protein structure may consist of distinct structural 'domains' (A and B in the diagram above), linked together by a segment, or segments, of the polypeptide chain, denoted the hinge region, in such a way that the relative orientation of the domains can vary without significant structural changes in the domains themselves. The domains may be composed of continuous regions of the polypeptide chain, as in T4 lysozyme where the N- and C-terminal regions each fold to distinct domains. They may also arise from the association of discontinuous regions of the sequence; examples are the c-type lysozymes which, although topologically related to T4 lysoyme, have one domain consisting of the N- and C-terminal region together, with the region in the centre of the sequence forming the other domain. The relative movement of the domains can cause large global conformational changes which may permit access of substrates and generate an appropriate environment for catalysis.

The conformational flexibility offered by hinge-bending is of particular note in that it may be important in allowing access of substrates to the active site, which is formed at the interface of the two domains. Indeed, there are several well-documented cases of such structural transitions resulting from the binding of ligands to proteins (hexokinase and phosphoglycerate kinase being particularly clear examples[6]). What distinguishes the Faber and Matthews result is that it seems