Uncharacteristic Earthquakes on the San Andreas Fault

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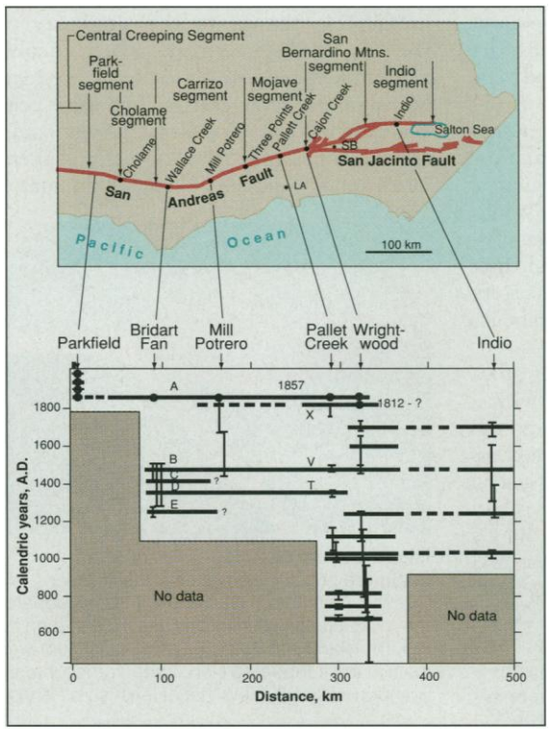
In the mid-1980s, earth scientists were excited about the possibility of predicting the general occurrence and frequency of earthquakes on the San Andreas and other faults. This enthusiasm was partly caused by the introduction of a model that accounted for the recurrence of large earthquakes. In this model (1), large earthquakes are thought to recur with regular or "characteristic" rupture patterns on distinct segments of faults. When coupled with a simple elastic strain-accumulation model, this behavior implies that characteristic earthquakes occur at fairly regular intervals. Data from the Carrizo Plain and other study sites along the San Andreas fault, however, are calling the characteristic earthquake model into question.

The characteristic earthquake model is appealing because it suggests that forecasting the general frequency of future earthquakes can be reduced to a series of steps: divide a fault into segments that behave characteristically, study previous earthquakes on those segments, and use that information to evaluate future earthquake hazards (2, 3). In the last decade, scientists collected data on past earthquakes to try to understand the seismic history of segments of many active faults. Findings of irregular earthquake recurrence, clusters of earthquakes, and variations in surface rupture are showing that the dynamics of faulting is complex and presents difficulties for the characteristic earthquake model (4).

The San Andreas is one of the best-studied faults in the world. Therefore, models of the behavior of the San Andreas fault have been applied to many less studied faults worldwide. The characteristic earthquake model was developed largely from data on the south central San Andreas fault in California. Since the model was proposed, the San Andreas has been the subject of numerous additional studies and the Parkfield Earthquake Prediction Experiment. Recent data indicate that there is significant variability in earthquake recurrence times and rupture patterns on the San Andreas fault rather than predictable repetition (see figure).

The most recent large earthquake on the southern half of the San Andreas fault occurred in 1857. Nearly 400 km of the fault ruptured in an earthquake of magnitude 8. The south central San Andreas has been divided into characteristic fault segments based on the pattern of the 1857 rupture (3). Each segment was thought to have a characteristic recurrence interval and rupture pattern. Repetition of large ruptures similar to that in 1857 was tied to failure of the Carrizo segment in combination with adjacent segments every 240 to 450 years (5).

One of the first indications of uncharacteristic complexity in recurrence times emerged from analysis of tree rings and records of the Spanish missions in southern California (6). The San Juan Capistrano Mission was devastated in 1812 by a large earthquake originally attributed to the nearby Newport-Inglewood fault. Tree-ring studies demonstrated that the 1812 earthquake actually occurred on the more distant San Andreas fault, only a few decades before the larger 1857 rupture. Subsequent detailed studies of the geologic record of prehistoric earthquakes at Pallett Creek, on the Mojave segment of the San Andreas fault, also revealed irregular recurrence times for surface ruptures (7). However, the Mojave segment is the southernmost segment that ruptured in the great 1857 earthquake, and the characteristic earthquake model does allow for some irregularity in ruptures near the boundaries of characteristic segments. Perhaps the Mojave segment was rupturing irregularly because it is the end of the characteristic 1857 master segment. Additional studies at Wrightwood seem to confirm this hypothesis (8). The San Andreas fault ruptured at both Pallett Creek and Wrightwood in 1857, implying that both sites are on the same segment of the fault. However, comparison of the prehistoric record of earthquakes at both sites shows that they have not always ruptured together (8). Again, this could be explained by invoking the segment-boundary hypothesis: Ruptures of the San Bernardino segment of the San Andreas fault, or the nearby San Jacinto fault, could have influenced the occurrence of earthquakes at Wrightwood (9).



A characteristic pattern. Segmentation and inferred temporal and spatial distribution of surface-rupturing earthquakes along the San Andreas fault in central and southern California. Segments [adapted from (*3, 7*)] and the 1857 rupture (bold) are shown above. A compilation of the approximate dates (vertical bars) and estimated locations (horizontal lines) of prehistoric earthquakes recorded at several study sites are shown below. The rupture pattern is more complicated than the characteristic earthquake model. [Adapted from (*7, 10*)] The stippled bars show areas where no data is available or work is not completed.

Despite these findings of complex rupture patterns near segment boundaries, the characteristic earthquake model appeared to apply to the Carrizo and Parkfield segments. The Parkfield segment was thought likely to generate a characteristic earthquake by 1992, on the basis of its historic record of repeated similar earthquakes and its location between actively creeping and locked sections of the fault. The Parkfield Earthquake Prediction Experiment was designed to capture and monitor the occurrence of this expected earthquake (2). Although Parkfield is still a favored site for the next San Andreas earthquake, the expected event has so far not occurred.

When the Parkfield segment inevitably does rupture, it could behave less characteristically than in the past. This possibility was recently considered by the California Earthquake Prediction Evaluation Council after review of new data on the past behavior of the nearby Carrizo segment. The characteristic earthquake model was based in large part on interpretations of the earthquake record in the Carrizo Plain. Reported regular recurrence of large earthquakes ac-

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companied by 9 to 12 m of surface displacement along the Carrizo segment have formed the cornerstone of the characteristic earthquake model for the San Andreas fault (*1*, *3*, *5*, *7*). The Carrizo segment was hypothesized to rupture only in large-magnitude events similar to the characteristic 1857 earthquake. Recent studies reveal a more complex history of earthquakes in the Carrizo Plain. A cluster model has been proposed to describe irregular recurrence times for Carrizo earthquakes, and recent studies show that the amount of displacement per earthquake has varied substantially (*10*). In addition, it appears that some Carrizo earthquakes have been smaller in magnitude or had a significantly different rupture pattern than the characteristic 1857 earthquake (*10*). The more complex rupture patterns revealed by recent research on this section of the San Andreas are difficult to explain with a simple characteristic earthquake model.

More than a decade of research results are allowing scientists to piece together parts of the San Andreas earthquake puzzle. In my view, the incomplete picture that is emerging is inconsistent with repeated, predictable characteristic earthquakes. The model may indeed be useful as a convenient way to try to understand nature, but its usefulness in routine methods of seismic hazard assessment should be reevaluated. We need physical models that better explain the observed irregularities in fault rupture. In any case, there is cause for enthusiasm among earthquake scientists because the acquisition of sufficient data to test the characteristic earthquake model is, in itself, a major step toward the larger goal of understanding earthquakes. As additional pieces are added to the earthquake puzzle, a clearer picture will emerge.

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Transcription Factor IIA: A Structure with Multiple Functions

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Millions of years of evolutionary pressure ensured that the readout of the genome—transcription and gene expression—is tightly regulated. In metazoans, the core transcriptional machinery responds to multiple signals, which trigger cascades of gene expression that ultimately lead to the proper formation of an embryo. How does the transcription machinery transduce and integrate the vast repertoire of converging signals to correctly increase or decrease messenger RNA production from a particular gene? An important way station on the route to answering this central question is reported in this issue of *Science*. Geiger and co-workers have solved the crystal structure of a core component of the transcription machinery (*1*)—a complex of DNA, TATA-binding protein (TBP), and transcription factor (TF) IIA.

In eukaryotic cells, RNA polymerase II and its associated factors (general initiation factors TFIIA, -B, -D, -E, -F, and -H) form a large structure, containing some 40 to 50 proteins, that initiates accurate transcription. Despite this enormous complexity, a remarkably detailed understanding of transcription by RNA polymerase II has been revealed by two decades of biochemical fractionation and in vitro–reconstituted transcription reactions. Crystal structures of TBP complexed with the TATA DNA element gave us a first glimpse of the architecture of the complex as it exists before initiation of transcription (the preinitiation complex) (*2*, *3*). TBP impressively deforms the promoter DNA by introducing a sharp bend and a dramatic widening of the minor groove. More recently, the triple complex of TBP and the general initiation factor TFIIB bound to DNA was solved—information that started to define the rules by which additional initiation factors enter into the preinitiation complex (*4*). Now the new crystal structure of a ternary complex containing general initiation factor TFIIA, TBP, and DNA (*1*) adds the next piece of the puzzle in our developing picture of the preinitiation complex.

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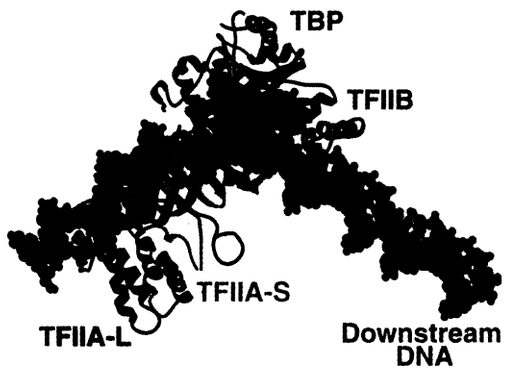
TFIIA appears to be a "coactivator," important for mediating activated transcription (*5*). Although TFIIA's exact role in transcriptional regulation is still somewhat unclear, this factor enhances the DNA-binding affinity of TBP and mediates efficient activation of transcription by various enhancer-binding proteins (*5*, *6*). The structure of the TFIIA/TBP/DNA complex reassuringly confirms TFIIA's functional assignments made on the basis of biochemical experiments. For example, it can easily be seen how TFIIA enhances the DNA-binding properties of TBP: The TBP/TFIIA complex has extended contacts to DNA (see figure). The ability of TFIIA to stabilize TBP/DNA interactions may also in part explain the derepression of basal transcription by TFIIA, although at least for in vitro transcription, TFIIA is not required.



The complex structure of the core of the transcription machinery: TBP/DNA/TFIIA/TFIIB. TFIIA has a large (L) and a small (S) subunit.

As in TFIIB/TBP/DNA, recognition of the TBP/promoter complex by TFIIA does not require further deformation of either TBP or the DNA. Only the β-barrel domain makes contacts that stabilize the protein/DNA complex, leaving the majority of the helical domain free to interact with other as yet unidentified factors. Thus, the apparent coactivator properties of TFIIA in directing activated transcription may result from direct contact between enhancer-bound activators and the exposed surfaces of TFIIA.

TFIIA activity requires expression of two genes that form a large and a small subunit. In *Drosophila* and in humans, the large subunit of TFIIA is proteolytically processed, clipped into two pieces somewhere