The Nature of the Landers-Mojave Earthquake Line

Amos Nur, Hagai Ron,* Gregory C. Beroza

The Landers, California, earthquake of 28 June 1992 (magnitude = 7.3) is the latest of six significant earthquakes in the past 60 years whose epicenters and slip directions define a 100-kilometer alignment running approximately N15°W across the central Mojave region. This pattern may indicate a geologically young throughgoing fault that replaces numerous older strike-slip faults by obliquely cutting across them. These older faults, and perhaps also the bend in the San Andreas fault, may be losing their ability to accommodate upper crustal deformation because they have become unfavorably oriented with respect to the regional stress field.

The Landers earthquake provided several surprises. The southern segment of the Landers and the Joshua Tree [April 1992, magnitude (M) = 6.1] earthquake ruptures fell together on an ~30-km line that had not before been recognized as a potential throughgoing, capable, and continuous seismogenic fault (*1*). This line was most clearly defined by the aftershocks, which crossed the left-lateral strike-slip Pinto Mountain fault, which runs east-west. The Joshua Tree–Landers fault system may therefore be a young fault that postdates slip on the Pinto Mountain fault (*2*).

Furthermore, the rupture pattern was kinked 30°, which is unusual (Fig. 1). The southern part of the Landers rupture was oriented about N15°W, but the northern part was oriented N45°W. Also, rupture formed along the five well-documented faults, including the Camp Rock, Emerson, and Johnson Valley faults; some earthquake models assume that seismic rupture should stop at kinks or bends, not propagate through them (*3*).

We suggest that the southern Landers and the Joshua Tree ruptures fell on a line that has had at least four other earlier earthquakes with similarly unexpected directions: the Galway Lake (1975, M = 5.4); Homestead Valley (1979, M = 5.7), Manix (1987, M = 6.5), and Calico (September 1965, M = 5.2) earthquakes (Fig. 2A). We call this line, which is over 100 km long, the Landers-Mojave line. The Landers-Mojave line occurs in a broad region of distributed faulting that has been documented by geological (*4*) and geodetic studies (*5*) and is often referred to as the eastern California shear zone (*4–7*). It is widely thought that distributed faulting is the main mechanism for deformation in this zone, but the mechanics of this distributed deformation is uncertain.

In 1989, Nur *et al.* (*7*) proposed, on the basis of an earlier mechanical model of distributed faulting (*8, 9*), that a new set of faults trending roughly north-south may be in the process of formation in the central Mojave region. The documented and geologically well developed strike-slip faults in the central Mojave region (*10*) have rotated counterclockwise relative to the eastern Mojave region (*11*), or, equivalently, the stress has rotated clockwise (*10*) over the past few million years. Consequently, most of these faults are today in a mechanically unfavorable orientation relative to the direction of maximum tectonic compression (Fig. 3), N10°E to N30°E (*7, 8*), which makes an angle of 55° to 75° to the northwest-trending faults. As a result, faults must develop in a new faulting direction (Fig. 4) to accommodate crustal deformation in the future. The two right-lateral strike-slip earthquakes in the Mojave region, the Galway Lake and Homestead Valley earthquakes, ruptured previously unmapped faults oriented roughly N10°W, not the well-developed northwest-trending old faults. These two ruptures were colinear and together define a possibly extended fault 30 km long (Fig. 2A). Although segments of this fault were recognized in the field before 1992 (*10*), it was not recognized as a throughgoing, coherent, and seismogenic fault or a potential fault.

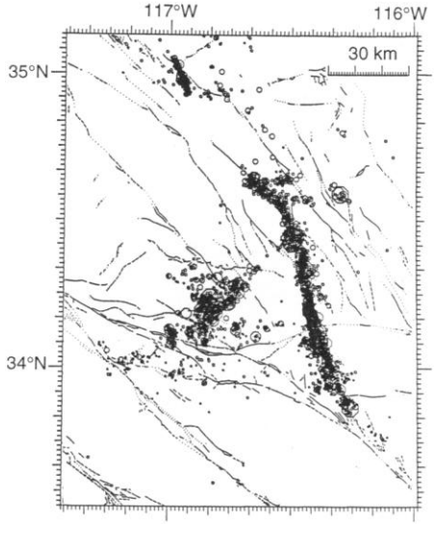

Fig. 1. Aftershocks of the Joshua Tree, Landers, and Big Bear earthquakes through 18 August 1992. The kink of about 30° is well defined by these events, as is the unexpected N15°W to N20°W direction of faulting [adapted from (*1*)].

The azimuth of rupture and the sense of slip of the Joshua Tree earthquake of April 1992 were similar to the smaller 1975 and 1979 events (Fig. 2A), and its epicenter fell close to the extension of their line to the south. Because the Joshua Tree rupture cuts across the east-west trending Pinto Mountain fault, we reconsidered also the Manix earthquake of 1947 (M = 6.2), over 100 km north of the Joshua Tree epicenter. The focal mechanism for this earthquake, though not well constrained (*12*), is probably also consistent with right-lateral strike slip on an unmapped ~N20°W-trending fault, not on the mapped east-west–trending one. Furthermore, this unmapped Manix rupture also falls on the continuation of the line of the 1975, 1979, and the 1992 events (Fig. 2A). And finally, the epicenter of another earthquake, the Calico event, south of Manix, also fell on this line

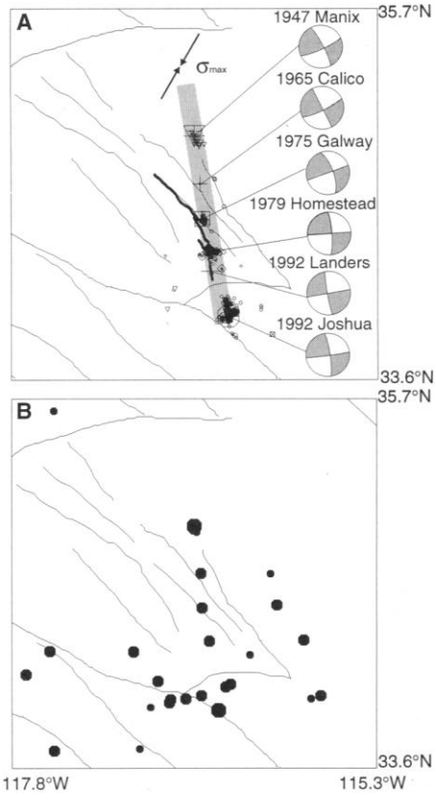

Fig. 2. (**A**) The fault plane solutions of the Manix, Calico, Galway Lake, Homestead Valley, Joshua Tree, and Landers events together define the Landers-Mojave line, which is probably a new emerging fault. (**B**) All $M > 5$ events in the Mojave between 1932 and 1991.

Department of Geophysics, Stanford University, Stanford, CA 94305–2215.

*Also at the Institute of Petroleum Research and Geophysics, 58852 Holon, Israel.

and, like the other events, had a rupture azimuth of N15°W or so. Together, these five events suggest an emerging, throughgoing seismogenic fault ~120 km long.

Some of the surprises of the June 1992 Landers earthquake can be explained by our block rotation and faulting model. First, the southern part of its rupture is coincident with both the location and sense of slip of the previous N15°W ruptures and thus provides further evidence for the emergence of a throughgoing fault. Second, the northern part of the rupture occurred on the old N45°W-trending Camp Rock fault, giving rise to a kink in the earthquake rupture. Kinks and bends in seismogenic faults are thought to act as barriers to earthquake rupture, and the sense of this particular kink would inhibit rupture (*3*). However, the rupture propagation through the Landers kink is consistent with our model, which suggests that slip can be partitioned during the transition from old, poorly oriented, weak faults to new, optimally oriented ones (Fig. 4).

The likelihood that the alignment of both the epicenters and the fault planes of the set of the six events that define the Landers-Mojave line is only a coincidence is quite low in consideration of the small number of $M \geq 5$ events in the Mojave during the past 60 years (Fig. 2B). Also, the six events on the Landers-Mojave line exhibit a systematic migration of epicenters from north to south, suggesting that they may be interrelated, perhaps as in an episodically propagating crack, a pattern that has been observed for example along the north Anatolian fault in Turkey and the Bocono fault in Venezuela (*13*). An alternative proposal (*14*) is that the Landers-Mojave line is a reactivated fault in the central Mojave that is older than the northwest-trending ones. This notion is inconsistent, however, with the documented right-lateral offsets on the northwest faults. These offsets would have by now segmented such an older fault, rendering it kinematically and mechanically incoherent. Finally, direct observations reveal new rock fractures on many of the north-south segments of the Landers rupture (*15*).

On the basis of our model, we originally proposed (*7*) that the emerging central Mojave fault system was needed to accommodate crustal deformation only on a relatively local scale, doing so by replacing the older faults only in the central Mojave region. However, the large magnitude of the Landers earthquake, geodetic data (*5, 6, 15*), and the length of the Landers-Mojave earthquake line raise the possibility that this fault system is important on a large scale (*16*). Perhaps not only are the old and local faults in the Mojave region being replaced by newer, better oriented ones, but the emerging Landers-Mojave fault line may be competing to some extent with the San Andreas fault itself. Because the San Andreas significantly deviates in the big bend region from the direction of slip between the North American and Pacific plates, it presumably can accommodate slip here only because it is weak, as are many mature, major faults (*17*). And like the rotation of the smaller faults in the Mojave, the bend in the San Andreas has probably also rotated (and is probably still rotating) counterclockwise because of the north-south shortening of the transverse ranges and may eventually lock. The new Mojave seismic alignment, in contrast with the San Andreas bend, is favorably oriented but, because it is new, may be stronger and require higher shear stress to slip. Perhaps it is just becoming equally easy for a system such as the Landers-Mojave fault to form as it is for slip on the unfavorably oriented segment of the San Andreas.

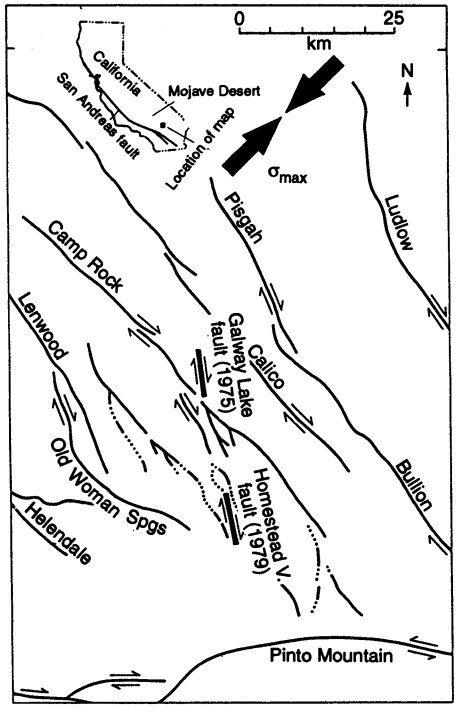

Fig. 3. The nearly fault-normal orientation of the Mojave compression to the older faults and its optimal orientation to the Homestead Valley and Galway Lake ruptures, which suggests the emergence of a new fault line caused by the gradual locking of the older faults. [Reprinted from (*7*)]

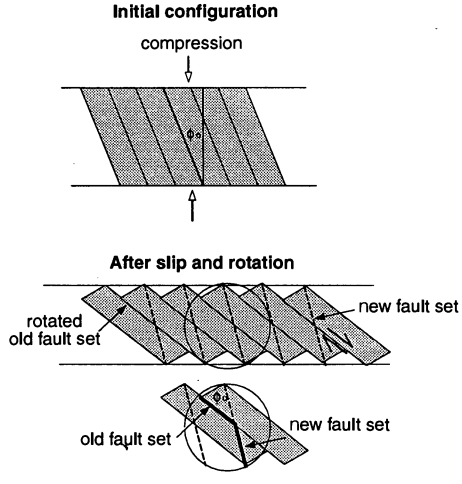

Fig. 4. The origin of kinks. When blocks and their bounding faults rotate away from the direction of compression or shortening, they eventually lock up. Further deformation requires that a new set of faults develop. When the new faults begin to develop and the old ones begin to lock, slip can occur simultaneously on both, leading to kinked slip, as illustrated by the heavy line.

REFERENCES AND NOTES

1. Working group report on "Future seismic hazards in Southern California. Phase I: Implications of the 1992 Landers earthquake sequence" (sect. IV, November 1992).
2. *ibid.*, p. 12.
3. See, for example, K. Aki, *J. Geophys. Res.* **89**, 5867 (1984); J. Lomnitz-Adler, *Geophys. J. Int.* **83**, 435 (1985); C. A. Aviles, C. H. Scholz, J. Boatwright, *J. Geophys. Res.* **92**, 331 (1987); P. Okubo and K. Aki, *ibid.*, p. 345.
4. R. K. Dokka, *Geology* **11**, 305 (1983); ——— and C. J. Travis, *Tectonics* **9**, 311 (1990).
5. M. Lisowski, J. C. Savage, W. H. Prescott, *J. Geophys. Res.* **96**, 8369 (1991).
6. A. Nur, *ibid.* **89**, 6256 (1984); ———, H. Ron, O. Scotti, *Geology* **14**, 746, (1986).
7. A. Nur, H. Ron, O. Scotti, *Int. Union Geod. Geophys. Monogr.* **4**, 31 (1989).
8. R. Freund, *Tectonophysics* **21**, 93 (1974); B. P. Luyendyk, J. J. Kamerling, R. Terres, *Geol. Soc. Am. Bull.* **91**, 211 (1980); H. Ron, R. Freund, Z. Garfunkel, A. Nur, *J. Geophys. Res.* **89**, 6256 (1984).
9. There is substantial paleomagnetic evidence for counterclockwise rotations in the central Mojave [D. B. Burke *et al.*, *U.S. Geol. Surv. Bull. 11524-E* (1982); J. Morton and J. W. Hillhouse, unpublished manuscript; G. D. Acton, thesis, University of Arizona (1986); B. J. McFadden, N. D. Opdyke, M. O. Woodburne, *Eos* **68**, 291 (1987); S. Potts, L. Brown, M. Valentine, M. Golombek, *ibid.* **70**, 1068 (1989); M. J. Valentine, thesis, University of Massachusetts, Amherst (1990)]. However, questions have been raised regarding the reliability of these results because of possible resetting, secular variations, poor age constraints, and insufficient sampling. Furthermore, two studies [R. W. Wells and J. W. Hillhouse, *U.S. Geol. Surv. Bull. 1529-E* (1989); M. McFadden *et al.*, *J. Geophys. Res.* **95**, 4597 (1990)] show no relation when the block tectonics model anticipates it, and one study shows clockwise, not counterclockwise, rotation [M. Golombek and L. Brown, *Geology* **16**, 126 (1988)]. We believe that additional data analyzed together with detailed structural information are needed to resolve this issue.
10. M. Rymer, personal communication.
11. See discussion in R. S. Stein, G. C. P. King, J. Lin, *Science* **258**, 1328 (1992), especially references and notes 11 through 14. The evidence for counterclockwise rotations in the central Mojave (CM) has been dismissed by some [for example, R. K. Dokka, *Eos*, Fall Meeting, **73**, 381 (1992)]. However, it is clear that a relative block rotation of 55° to 75°, between the eastern transverse ranges and eastern Mojave (EM) region and the CM, has taken place during the past few million years and that this rotation must be related to the relative direction of faults and the direction of stress within

these domains. One can imagine for example that the initial angle between the faults in the EM and the CM was close to the optimal failure direction for N5°E compression three million years ago; the fault azimuths at that time would have been N35°E and N25°W, respectively. For the assumption that the maximum stress bisects the angle between these sets also today, with the EM faults oriented N85°E and CM ones at N45°W, the present day compression direction should be N20°E. This direction would then imply a 15° clockwise rotation of the stress, a 55° clockwise rotation of the EM blocks, and a 20° counterclockwise rotation of the CM blocks.

12. The discrepancy between the surface-mapped east-west Manix and the ~N20°W direction of the 1947 Manix rupture was first pointed out by C. F. Richter and J. M. Nordquist [*Seismol. Soc. Am. Bull.* **41**, 347 (1951)] and detailed by C. F. Richter [*Elementary Seismology* (Freeman, San Francisco, CA, 1968)]. D. Doser has carefully reanalyzed this event and its aftershocks [*Seismol. Soc. Am. Bull.* **80**, 267 (1990)].
13. J. W. Dewey, *Seismol. Soc. Am. Bull.* **66**, 843 (1976); A. Aydin *et al.*, *Eos*, Fall Meeting, **73**, 381 (1992).
14. G. Davis, personal communication.
15. K. Lajoie, personal communication; J. Sauber, W. Thatcher, S. C. Solomon *J. Geophys. Res.* **91**, 12683 (1986).
16. R. E. Wallace, *J. Geophys. Res.* **89**, 5763 (1984).
17. M. D. Zoback *et al.*, *Science* **238**, 1105 (1987).
18. Supported in the past by the National Aeronautics and Space Administration's geodynamic program, by past and current grants from the Israel-U.S. Binational Science Foundation, and by a National Science Foundation Presidential Young Investigator Award (G.C.B.). We thank G. Mavko and J. Dvorkin for their helpful discussions and W. Thatcher and P. Bird for insightful comments.

12 February 1993; accepted 26 April 1993

Interaction Between Transcription Regulatory Regions of Prolactin Chromatin

Katherine E. Cullen, Michael P. Kladde,* Mark A. Seyfred†

The regulation of transcription requires complex interactions between proteins bound to DNA sequences that are often separated by hundreds of base pairs. As demonstrated by a nuclear ligation assay, the distal enhancer and the proximal promoter regions of the rat *prolactin* gene were found to be juxtaposed. By acting through its receptor bound to the distal enhancer, estrogen stimulated the interaction between the distal and proximal regulatory regions two- to threefold compared to control values. Thus, the chromatin structure of the *prolactin* gene may facilitate the occurrence of protein-protein interactions between transcription factors bound to widely separated regulatory elements.

Three models have been proposed to explain the mechanism by which transacting factors act at a distance: the scanning model, the structural transmission model, and the DNA looping model (*1*). Data have been obtained in support of the DNA looping model, in which the intervening DNA sequence between the DNA-bound transacting factor and the transcription initiation complex is looped out, but it is unclear what drives the formation of the loops (*2*). Interacting proteins may extend and contact one another, bridging the distance between the proteins and forcing the intervening DNA to loop out (*3*). Alternatively, the DNA may be intrinsically bent, thereby allowing widely separated regions of DNA to be juxtaposed. Over short distances [200 to 300 base pairs (bp)], supercoiling may provide enough bending of the DNA to allow association between DNA binding proteins (*4*). Over large distances, the packaging of the DNA into chromatin may orient two widely separated protein binding sites to permit the association of the DNA binding proteins (*5*).

Expression of the rat *prolactin* (*PRL*) gene is regulated by a number of different polypeptide and steroid hormones that act through two distinct regulatory regions separated by approximately 1500 bp (Fig. 1) (*6*). The steroid hormone estrogen (E2) induces the transcription of the *PRL* gene by binding to the estrogen receptor (ER), which in turn binds to the estrogen response element (ERE) (*7*). This element is located at the 3′ end of the distal enhancer region between −1550 and −1578 bp (*8*). How the ER complex influences the activity of RNA polymerase II located 1500 bp downstream at the promoter is unknown. Data from studies with a cell-free transcription system containing purified ER and templates with an ERE a short distance from the promoter led Elliston *et al.* (*9*) to suggest that the ER enhances transcription by facilitating the formation of a stable preinitiation complex. For the ER complex to perform a similar function in the *PRL* gene in vivo, the spatial distance between the ER complex and the promoter must be reduced. We have previously shown that the chromatin surrounding the ERE and the promoter becomes hypersensitive to nucleases after treatment of cells with E2, although the region between the ERE and the promoter remains insensitive to nucleases (*10*). This suggests that the ER complex does not scan along the DNA to the promoter nor does the activated ER complex initiate a change in the DNA structure that is propagated from the ERE to the promoter.

To determine if DNA looping may facilitate the interaction between the ER and the transcription initiation complex, we examined the chromatin looping potential of the 5′ upstream regulatory elements of *PRL* (Fig. 2A) (*11*) with a modified DNA looping assay of Mukherjee *et al.* (*12*). We used *PRL*-Tn*5*–bovine papillomavirus (BPV) minichromosomes as our source of *PRL* chromatin. These minichromosomes are packaged into nucleosomal arrays (*10*) and replicate extrachromosomally at a level of 40 to 60 copies per cell in a stable, clonal cell line (G1I) obtained by the transfection of rat pituitary GH_3 cells with the *PRL*-Tn*5*-BPV vector. The transcription of the Tn*5* gene, which acts both as a selectable marker (G418 resistance) and as a reporter, is under the control of the *PRL* regulatory elements (Fig. 1) and can be induced by E2 (*13*). When nuclei isolated from G1I cells were partially digested with Pst I, a number of different *PRL*-Tn*5* chromatin fragments were produced that could theoretically form Pst I ligation products (Fig. 2B). Polymerase chain reaction (PCR)–mediated analysis principally detected only the formation of Pst I ligation products that correspond to the P_3-P_4 and P_3-P_5 *PRL* chromatin loops (Fig. 2C, lane 5).

The PCR products were analyzed by

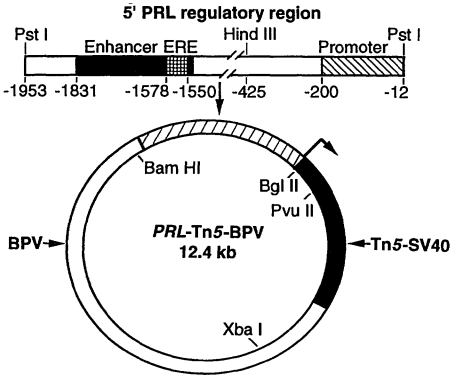

Fig. 1. The *PRL*-Tn*5*-BPV minichromosome. The minichromosome contains the transcription regulatory elements (hatched) of the rat *PRL* gene (−1953 to −12), which controls the expression of the Tn*5*-SV40 reporter gene (solid). The entire BPV genome is present in the minichromosome (unshaded). Further details on the characterization of the cell lines containing the *PRL*-Tn*5*-BPV minichromosome have been described (*10, 13*).

K. E. Cullen and M. A. Seyfred, Department of Molecular Biology, Vanderbilt University, Nashville, TN 37235.
M. P. Kladde, Department of Biochemistry, University of Wisconsin, Madison, WI 53706.

*Present address: Laboratory of Cellular and Developmental Biology, National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, Bethesda, MD 20892.
†To whom correspondence should be addressed.