Reports

Regular Intervals Between Hawaiian Earthquakes: Implications for Predicting the Next Event

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During the years 1941 through 1983 five earthquake mainshocks of moderate magnitude occurred at regular intervals of 10.5 ± 1.5 years within a 6-kilometer radius in Hawaii. It is proposed that these Kaoiki earthquakes will continue to occur at regular intervals because the strain accumulation rate and the strained volume remain constant. With appropriate instrumentation, it may be possible to refine predictions of subsequent Kaoiki earthquakes.

ARGE EARTHQUAKES ALONG A GIVen fault segment do not occur at random intervals because it takes time to accumulate the strain energy for the rupture (1). The rates at which tectonic plates move and accumulate strain at their boundaries are approximately uniform. Therefore, in first approximation, one may expect that large ruptures of the same fault segment will occur at approximately constant time intervals. If subsequent mainshocks (2) have different amounts of slip across the fault, then the recurrence time (3)may vary, and the basic idea of periodic mainshocks must be modified (4). For great plate boundary ruptures the length and slip often vary by a factor of 2. Along the southern segment of the San Andreas fault the recurrence interval is 145 years with variations of several decades (5). The smaller the standard deviation of the average recurrence interval, the more specific could be the long-term prediction (6) of a future mainshock.

In the Kaoiki, Hawaii, area it appears that mainshocks happen at unusually constant intervals (Fig. 1). Earthquake locations for events since 1959 were recalculated in an extensive seismological study of the Kaoiki area (7) and were grouped in three time periods (7): (i) A mainshock in June 1962 of surface wave magnitude (M_S) of 6.1 was followed by events with local magnitudes $(M_{\rm L})$ of 4.9, 4.8, and 5.3 in the period between August and October 1963. (ii) In 1974 three earthquakes of M_1 4.8 (June), 5.5 (November), and 4.8 (December) formed another group. (iii) In November of 1983 a rupture ($M_{\rm S} = 6.6$) occurred. Before 1959 the earthquake record is less complete. The volcano letters state that the 1941 mainshock and three large aftershocks were "located 4 miles north of the Kapapala ranch house," and the 1951 shock and its aftershocks were directly attributed to the Kaoiki fault zone (8). The intensity of shaking (I_0 in Table 1) of these shocks was comparable to the more recent mainshocks with magnitudes ranging from 5.5 to 6.6. Therefore, it is concluded that in a volume of the earth's crust that has a radius of approximately 6 km (Fig. 2), mainshocks have occurred in 1941, 1951, 1962, 1974, and 1983 (Table 1). The recurrence interval $T_r(K) = 10.5 \pm 1.5$ years (9). A leastsquares regression of the dates as a function of event number estimates the arrival of the next event with 95% confidence between July 1993 and October 1996 (I0).

This observation is similar to the Parkfield, California, case where $T_r(P) = 21.9 \pm 3.1$ years (11, 12). The Kaoiki recurrence time and the standard deviation are about half those of the Parkfield ones. The magnitudes of the mainshocks in the two areas are similar; the last events measuring $M_s = 6.6$



Fig. 1. Event numbers of Kaoiki earthquakes as a function of dates show that a straight line fitted through the last five Kaoiki shocks suggests that a sixth shock would occur at the end of 1994, approximately. If the Kaoiki sequence is regular, one would expect to find historic evidence for mainshocks in or near the years of 1931 and 1920. A search of felt reports turned up a moderate mainshock that probably was located in the Kaoiki fault zone on 25 May 1930 (open circle). The inset shows the magnitudes of all Kaoiki earthquakes larger than 4.4 between 1959 and 1986 (7) as a function of time.

(Kaoiki) and $M_{\rm S} = 6.4$ (Parkfield). A dissimilarity may be found in the constancy of the mainshock size. Whereas the Parkfield earthquakes are nearly carbon-copy events (11), the magnitude and style of faulting varies at Kaoiki (7, table 1).

The Kaoiki area is not located at the boundary of a tectonic plate. Instead the strain is accumulated by crustal expansion which takes place in the volcanoes Mauna Loa and Kilauea (7, 13). Magma rises in narrow conduits under the volcano summits and then often intrudes along shallow paths into the volcanic rift zones, thus compressing the adjacent crust (13). Because the Kaoiki volume is located between the summits of Mauna Loa and Kilauea, the compressive stresses from the two volcanoes combine to create a stress tensor with the greatest principal stress in the direction connecting the two summits (Fig. 2). The resulting mainshock's focal mechanisms are therefore right-lateral strike-slip on a near vertical plane (7, 14) like the San Andreas style faulting at Parkfield (15). However, the Kaoiki faulting is more complicated. In addition to the strike-slip motion, a detachment along the near horizontal oceanic sediment layer (16) occurs in some, but not in all, of the mainshocks, such that a mixture of strike-slip and thrusting mechanisms is found among the aftershocks (7). Thrusting in the sediment layer occurred in the 1983 mainshock, whereas there was no evidence for this process in the 1974 event. This is probably the cause of the difference in magnitude between mainshocks in the Kaoiki sequence.

A refinement of the prediction of occurrence time might be possible if precursory anomalies can be observed. Before the 1983 Kaoiki mainshock a most pronounced period of seismic quiescence (17) existed. During 2.4 years the rate of occurrence of small earthquakes was reduced by 75% in the source volume, except for the immediate vicinity of the main-rupture initiation point, where the rate remained constant (Fig. 2) (18). This same pattern was observed for the 1975 Kalapana, Hawaii ($M_{\rm S} = 7.2$), earthquake (19). However, quiescence could not be found for the 1974 Kaoiki earthquake. Based on these observations it is concluded that a quiescence precursor may help to refine the prediction of the next Kaoiki mainshock. Because the 1983 precursor time lasted 2.4 years, one would expect the next quiescence to start in the first part of 1992. If quiescence appears again, then we can refine the estimate of the predicted time, provided that the variance of quiescence

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Table 1. Large Kaoiki mainshocks since 1940.

Date	$M_{ m S}$	I_0^{\star}	Epicenter		
			Latitude (N)	Longitude (W)	(km)
25 September 1941	6.0	VII	19°21′	155°27′	11
16 September 1951		V	19°20′	155°26′	n
27 June 1962	6.1	VI	19°24.14′	155°26.75′	8.9
30 November 1974	5.5		19°26.16′	155°25.03'	4.4
16 November 1983	6.6	VII	19°25.55′	155°27.19′	11.2

*The maximum intensity of shaking is approximately proportional to magnitude. VII, difficult to stand, damage to masonry; VI, walk unsteadily, windows broken; and V, unstable objects upset.

duration is less than that of recurrence time $(T_{\mathbf{r}}).$

The model for the Kaoiki mainshock sequence is not yet as well developed as that for the Parkfield case (11, 12). The following working hypothesis to explain the Kaoiki sequence is as yet supported by few facts only and may therefore need to be revised substantially as more data are analyzed. Magma rises at a steady rate under the volcanoes (20) and causes a strain accumulation that is constant if averaged over several years. The crustal volume caught between the two volcanoes' stress systems is constant in size; therefore, the extent of the source volume is approximately constant; hence the time between Kaoiki mainshocks is constant to a remarkable degree.

Tests can be done for parts of this hypothesis. For the years 1834 to 1939 there were 39 Hawaiian earthquakes reported as felt (8). Many of these were clearly not located near Kaoiki, but others are attributed to the nearby Mauna Loa or Kilauea areas. It may be possible to ascertain by searches in old documents and by interviews with senior residents which of these may have been Kaoiki mainshocks. Based on the above hypothesis one would expect that Kaoiki mainshocks took place in approximately early 1931 ± 1.5 years and around the year 1920 (21). Another test of the hypothesis will be provided by events between 1992 and 1996.

The Kaoiki earthquake sequence, and the model to explain it, have some advantages that recommend it for earthquake prediction research. (i) The area is sparsely populated. Therefore, announcements about future earthquakes may not be as sensitive an issue as in more populated areas, and the permission to place measuring devices into the area may be more readily available. (ii) A test of the hypothesis occurs every 10.5 years-that is, twice as frequently as at Parkfield, which means that progress in learning how to predict earthquakes can be made relatively rapidly. (iii) The type of faulting of a brittle crust under local stress concentrations may be similar in some aspects to intraplate

ruptures. Thus, a model derived from the Kaoiki events may be more pertinent to these areas than a Parkfield (San Andreas fault) prediction model, unless the strain rate plays an important role in the development of precursors (22).

The results presented here suggest that it may be possible to make long- and mediumterm predictions of some $M_{\rm S} = 6 \pm 0.5$ mainshocks with accuracies of approximately 1 ± 0.5 years. This capability might be improved by the detection of short-term precursors. For example, if foreshocks occur



Fig. 2. Cumulative number of microearthquakes $(M_{\rm L} \le 1.7)$ as a function of time for 6 years before the last Kaoiki mainshock ($M_{\rm L} = 6.6$, November 1983). The occurrence time of the mainshock is at the right edge of the figure; the volume in which the earthquakes were counted was located within the eastern part of the mainshock source volume and had dimensions of approximately 10×5 km (18). The arrow in mid-1981 points to the onset of quiescence, when the mean rate of microearthquakes decreased by 75% as shown by the clear change of slope of the cumulative number curve. The inset shows a map of Hawaii with the epicenters (7) of the 1962, 1974, and 1983 Kaoiki mainshocks marked by black dots. The open circles mark the 1941 and 1951 epicenters which are not as accurately known (8). All epicenters are located within a circle of radius 6 km.

and can be identified correctly, the time of the mainshock might be estimated to within a week (23), but the problem of how to recognize foreshocks without many false alarms is not yet solved. Randomly occurring triggering events might upset the schedule and cause possibly random deviations from the expected occurrence time (24). Although there is no doubt that we are slowly making progress in learning how to predict earthquakes, there is also no doubt that there will always be failures.

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- The expression "mainshock" is often restricted to the 2. largest event in a sequence. We will extend its use here to mean a relatively large earthquake with rupture length comparable to the fault segment or crustal volume considered. Such an earthquake will cause a major release of strain energy in the source volume. Energy will have to be built up anew by tectonic processes before another mainshock can occur. 3. The "recurrence time" is the time that elapses be-
- tween repeated mainshock ruptures of the same fault segment or source volume. The recurrence time would be exactly constant if the source volume, the strain release in each mainshock, the strain accumulation rate by tectonic forces, and the failure strengths would all be constant, but these parameters often vary by large amounts
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- 9 $T_r(K)$, the time of mainshocks to reoccur at Kaoiki, is calculated as the mean interval between the events in Table 1. The standard deviation of these, ± 1.5 years, is assumed to be the uncertainty.
- 10. The slope of the straight line in Fig. 1 is 10.74 ± 0.47 (95% confidence limit). The prediction proposed here is as follows: an earthquake of magnitude $5.5 \le M_S \le 6.6$ will occur at latitude $19^\circ 23.4' \pm 3'$ and longitude $155^\circ 26.4' \pm 3'$ with 95% confidence between July 1993 and October 1996.
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- The volcanic edifice of the Hawaiian Islands is deposited on top of the oceanic sea floor, which is created at the East Pacific Rise and travels by plate 16. motion toward Japan. On its way to Hawaii, about

half a kilometer of sediments accumulated on the sea floor. Due to the weight of Hawaii, the sea floor is depressed under the islands, and thus the oceanic sediment layer is a depth of 9 to 10 km from the earth's surface at Kaoiki. Geodetic and seismologic evidence shows that near horizontal slip occurs at the depth of these sediments, allowing the southeast flanks of the volcanoes Kilauea and Mauna Loa to move away from the volcanoes and the rifts toward move away from the volcances and the first toward the southeast (7, 13) [A. S. Furumoto and R. L. Kovach, *Phys. Earth Planet. Inter.* 18, 197 (1979);
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"Seismic quiescence" is a decrease of the rate of

- 17. earthquake occurrence within the volume in ques-tion. The detection of quiescence presumes that a nearly constant background rate can be defined in the same volume. In two mainshocks in Hawaii, where detailed data were available, only parts of the source volume showed quiescence, whereas major asperities produced microearthquakes at constant rates up to the mainshock (18, 19).
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- The Volcano Letters for seven earthquakes that might have been located in the Kaoiki area, according to the list given by Coffman and von Hake (8), showed that in all but one case, the writers called the earthquakes shallow (1 to 3 miles), saying they were part of ongoing eruptions and located on Kilauea or Mauna Loa. The single exception occurred on 25 May 1930. T. A. Jaggar [Volcano Letters, May 1930 (8)] did not attribute this event to either volcano. He even discussed and disproved the possibility that it might have been located at the west coast of Hawaii. He finally concluded, "All of these facts suggest a deep movement somewhere under Kilauea and Mauna Loa." In the terminology of the Volcano Letters of that time "deep" means about 10 km. Thus I conclude that the hypothesis of regular repeat times of the Kaoiki earthquake has passed its first test. The shock expected in 1931 ± 1.5 actually

occurred in 1930.4, 11.3 years before the earliest well located event (Table 1). Using this as a fifth known value, $T_r(K)$ is estimated to be 10.7 ± 1.2 years. With six data points the prediction yields a date of 1995.25 for the next event.

- The deformation characteristics and failure behavior of rocks in the laboratory are known to depend on strain rate. Therefore it is possible that precursors in Hawaii, where strain rates are high, may differ from those in areas of low strain rate.
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- specific triggering mechanism in Hawaii might be the intrusion of a large amount of magma into one of the volcanic rifts. Through the resulting stress
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T-Lymphocyte Priming and Protection Against Friend Leukemia by Vaccinia-Retrovirus env Gene Recombinant

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The current prevalence of the acquired immune deficiency syndrome in humans has provoked renewed interest in methods of protective immunization against retrovirusinduced diseases. In this study, a vaccinia-retrovirus recombinant vector was constructed to study mechanisms of immune protection against Friend virus leukemia in mice. The envelope (env) gene from Friend murine leukemia virus (F-MuLV) was inserted into the genome of a vaccinia virus expression vector. Infected cells synthesized gp85, the glycosylated primary product of the env gene. Processing to gp70 and p15E, and cell surface localization, were similar to that occurring in cells infected with F-MuLV. Mice inoculated with live recombinant vaccinia virus had an envelope-specific T-cell proliferative response and, after challenge with Friend virus complex, developed neutralizing antibody and cytotoxic T cells (CTL) and were protected against leukemia. In contrast, unimmunized and control groups developed a delayed neutralizing antibody response, but no detectable CTL, and succumbed to leukemia. Genes of the major histocompatibility complex influenced protection induced by the vaccinia recombinant but not that induced by attenuated N-tropic Friend virus.

NTEREST IN THE PRODUCTION OF VACcines against retroviruses has been sparked by the discovery that members of this family cause human leukemia and acquired immune deficiency syndrome (AIDS). Protective immunization against retroviruses has been achieved in some systems by using live or killed virus (1) or viral envelope gene (env)-encoded glycoprotein (2). In other systems, however, immunization with killed virus or viral envelope proteins induced immunosuppression and enhanced disease (3). Since antigen presentation by vaccinia virus recombinants mimics that of natural infections, we were interested in determining whether protection against retroviruses could be achieved by the use of a recombinant vaccinia virus carrying a retroviral env gene. Previously, protective immunity was demonstrated with vaccinia virus recombinants that expressed genes from

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members of other virus groups including influenza, herpes simplex, hepatitis B, rabies, vesicular stomatitis viruses, and respiratory syncytial virus (4).

The Friend virus (FV) complex system is a particularly good retrovirus model for studying the potential for immune protection since it causes erythroleukemia even after infection of immunocompetent adult animals. Furthermore, the genetics, cell biology, immunology, and molecular biology of FV have been extensively studied (5). The disease, characterized by hepatosplenomegaly and polycythemia or anemia, occurs 1 to 3 weeks after inoculation and usually results in death within 1 to 3 months. The virus complex consists of a replication-defective spleen focus-forming virus (SFFV) and a replication-competent helper virus, referred to as Friend murine leukemia virus (F-MuLV). By itself, F-MuLV can induce

leukemias with long latencies when inoculated into newborn mice; however, the rapid leukemia observed in adult mice inoculated with FV is believed to be induced by the defective SFFV component of the complex. The env gene of F-MuLV encodes a glycosylated protein, gp85, which is subsequently cleaved into the closely associated proteins gp70 and p15E (5). The SFFV is defective in its env gene, and so it utilizes the envelope protein of the helper F-MuLV. Thus, both F-MuLV and SFFV virions display the same envelope protein, and monoclonal antibodies directed to determinants on this polypeptide can neutralize infectious virus (6). gp70 is also expressed on the surface of infected cells where it can be recognized by cytotoxic antibodies or cytotoxic T lymphocytes (CTL) (7). Hunsmann et al. (2) showed that repeated immunization of mice with purified envelope protein resulted in protection against challenge with FV. Therefore, we chose to make a recombinant vaccinia virus capable of expressing the gp85 product of the F-MuLV env gene. The entire env gene, including a 60-nucleotide leader at the 5' end and approximately 150 nucleotides at the 3' end, was inserted into the Bam HI site of the vector pGS20 (8, 9). In the construct, the initiation codon of the env gene was the first ATG codon after the vaccinia virus P7.5 promoter. The resulting recombinant plasmid pPE2 was used as a vector to transfer the env gene into vaccinia virus by homologous recombination (8). Confirmation of the predicted location and structure of the env gene within the vaccinia virus genome was obtained by electropho-

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