less than optimum. When reliable depth measurements are made and initial crater shapes in icy bodies are better understood, these can be used directly in our procedure. (iii) Convective heat transport has been simplified by the parameterized approach, and the effects of non-Newtonian convection, pressure dependence of melting temperature, and phase boundaries have not been evaluated. We also followed only one evolutionary path, assuming inhomogeneous accretion (or rapid infall of a silicate core) with a primordial liquid mantle. Other paths, such as solid homogeneous accretion of ice and silicates [E. M. Parmentier and J. W. Head, *Proc. 10th Lunar Planet. Sci. Conf.* (1979), p. 2403], may lead to radically different outcomes. The possibility of silicates mixed with ice in the mantle must also be considered in terms of effects on thermal evolution. The effect of non-Newtonian creep on the relaxation of craters must also be considered [E. M. Parmentier and J. W. Head, *J. Geophys. Res.* **84**, 6263 (1979)], as well as the idea that the outer part of the lithosphere should be treated elastically. (iv) There is a similar set of problems associated with any flux model, chief among which is the form of that model. The parameters of the model should be treated as unknown quantities in any inversion process. In addition, the relationship between the velocity and size distribution of the impacting population, the effects of materials on crater geometry, and the production function of craters on the surface must be established, particularly for interplanetary comparisons.

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Hydrogen Release: New Indicator of Fault Activity

Abstract. *The hydrogen concentration in soil gas has been measured in the area around the Yamasaki Fault, one of the active faults in southwestern Japan. Degassing of a significant amount of hydrogen (up to more than 3 percent by volume) has been observed for sites along the fault zone. The hydrogen concentration in soil gas at sites away from the fault zone was about 0.5 part per million, almost the same as that found in the atmosphere. The spatial distribution of sites with high hydrogen concentrations is quite systematic. A hypothesis on the production of hydrogen by fault movements is postulated.*

The upward migration of various gases in the earth's crust is well known. The best known example is the degassing of helium (*1*). It has been suggested that He migrates from the upper mantle because of its anomalous isotopic ratio of ^3He to ^4He (*2*). The degassing rate of He at the surface is thought to be constant except in areas of high crustal activity. Volcanic fumaroles and the degassing observed in geothermal areas are the most extreme cases of this degassing phenomenon.

Degassing rates are assumed to be affected by local geotectonic features such as faulting. A significant amount of He with a high $^3He/^4He$ ratio has been observed along the fault zone formed by the 1966 Matsushiro earthquake swarms (*3*). From these observations, the He flux along the Matsushiro fault was calculated to be about 3×10^4 times (*3*) higher than that of the average crust (*4*).

Recently, the recognition of active faults has become a very important tool for earthquake prediction because many large earthquakes are known to occur along active faults. Several attempts have been made to evaluate the movements of active faults by topographic and geodetic studies and by the observation of microearthquakes. In order to obtain basic knowledge on fault movements by geochemical methods, we have carried out a series of field experiments in the area along the Yamasaki Fault.

This fault is one of the main strike-slip faults in the inner zone of southwestern Japan (*5*). The length of the fault system, trending in a northwest-southeast direction, is about 80 km. The basement rock of the area is primarily Paleozoic granite. The sedimentary layer is less than a few meters thick. Surface features are characterized by altered shales, and a vast amount of Cretaceous intrusives of acidic rocks is seen as rhyolitic dikes (*6*).

Shallow microearthquakes have occurred frequently along the Yamasaki Fault zone. The vertical distribution of the hypocenters of these shocks is concentrated in the region 10 to 15 km deep, the lower part of the granitic layer (*7*). An earthquake with a magnitude of over 4 occurs, on the average, once every several years. Comparatively large earthquakes which occurred recently had magnitudes of 5.1 (21 September 1973), 3.7 (30 September 1977), and 5.0 (28 December 1979). The mean velocity of the left-lateral slip motion of the major fault is calculated to be about 1 mm/year on the basis of neotectonic studies (*8*). Since this area was designated as a test field for earthquake prediction research, intensive studies have been carried out by scientists from several universities in Japan. These include high-sensitivity microseismometry, strainmetry based on continuous observations with an array of extensometers across the fault, leveling, geochemistry, and geomagnetism, among others (*9*).

Since November 1978, field measurements of soil gas have been made three times in the area along the Yamasaki Fault (*10*). A total of 21 sampling sites have been located in the region between Yamasaki and Fukuzaki, an approximate area of 20 km by 20 km (Fig. 1). Each sampling site consists of from 2 to 14 sampling points, so that we could obtain a representative value of the H_2 concentration of that site. A total of 70 sampling points were used in this study.

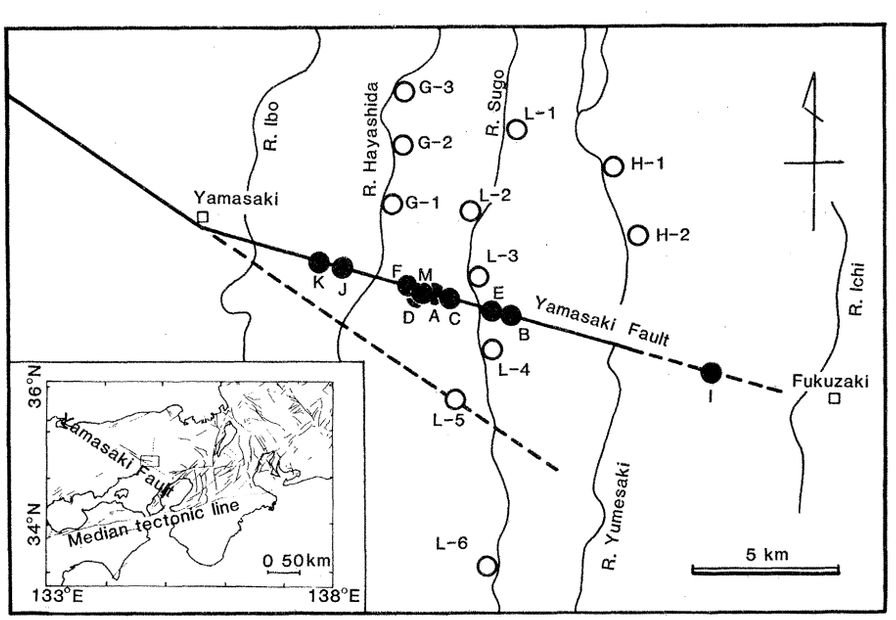

Fig. 1. Locations of the sampling sites of soil gas in the area around the Yamasaki Fault. Representative active faults and tectonic lines in southwestern Japan are shown (*5*). Closed circles indicate the sites where significant amounts of H_2 were measured. Open circles designate the sites where H^2 concentrations were almost the same as that of the atmosphere.

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The soil gas was collected in the following way. Holes 2.5 and 5.0 cm in diameter and 50 to 100 cm deep were drilled in the ground with an electric drill or a hand auger. Vinyl chloride pipes (10 cm long) were inserted into the holes. The top of each of the pipes was closed with a rubber stopper, and the outside of each pipe was sealed with bentonite. The soil gas was thus confined in the holes.

After a storage time of about 12 hours, a few milliliters of the gas sample were extracted with a hypodermic syringe and analyzed in the field. A pair of conventional gas chromatographs (Ohkura model 903) with a column filled with molecular sieve (5A, 80/100 mesh) were used. The concentrations of He, H_2, O_2, N_2, and CH_4 were determined simultaneously (with Ar used as the carrier gas). The Ar concentration was determined separately (O_2 was the carrier gas). About 100 ml of the soil gas was used in the determination of the CO_2 concentration with gas detection tubes (Gastec TC84-018).

The measured H_2 concentrations are listed in Table 1. An extraordinarily large amount of H_2 was observed in soil gas collected at ten sampling sites along the fault. In the extreme case, the H_2 concentration was more than 3 percent by volume. The H_2 concentration in soil gas at sites away from the fracture zone averaged about 0.5 part per million (ppm), almost the same as that found in the atmosphere. There is a clear correlation between the sites with the high H_2 concentrations and the fault system (Fig. 1); the spatial distribution of the sites with high H_2 concentrations is strictly systematic. This result suggests a strong correlation between H_2 degassing and fault movements.

No enrichment in He was observed at any sampling point. The concentrations of N_2 and Ar in the samples were almost the same as those in the atmosphere. The O_2 concentration of the soil gas was slightly lower than that in the atmosphere.

It has been suggested that H_2 in soil gas is produced by the biological activities of bacteria underground. In this case, great amounts of CO_2 and CH_4 are generally produced and the soil gas becomes greatly depleted in O_2 (*11*). No such enrichments of CO_2 and CH_4, however, were observed in the soil gas from the Yamasaki Fault. Concentrations of CO_2 for most samples were less than 0.9 percent, and, except for one sample, those of CH_4 were less than 150 ppm. The soil gas collected at site C contained about 800 ppm of CH_4.

We attempted to exclude from our sample set those sites where H_2 was suspected to be produced from biological sources. The bottoms of most holes drilled along the fault reached the bedrock. Sampling sites I, J, K, and M were selected in the area where shales or granitic rocks were directly exposed. Under these circumstances vegetation was sparse throughout the year and biological activity underground would be expected to be extremely low. Nonetheless, a large amount of H_2 was observed at these sites. Even though it is impossible to eliminate minor contributions of H_2 from biological sources, it is most unlikely that significant contributions from such sources are present in our samples.

Table 1. The H_2 concentration of soil gas obtained from the area around the Yamasaki Fault, Japan.

Sampling site	H_2 concentration (ppm)*		
	November 1978	March 1979	November 1979
A	3,060(4)	2(1)	3 (2)
B	87(5)		660 (2)
C	13,300(7)	30,600(14)	31,000 (4)
D		2(2)	
E		350(4)	~ 0.5(2)
F		70(4)	10 (2)
G-1			~ 0.5(3)
G-2			~ 0.5(2)
G-3			~ 0.5(2)
H-1			~ 0.5(2)
H-2			~ 0.5(2)
I			140 (2)
J			230 (2)
K			4,300 (2)
L-1			~ 0.5(3)
L-2			~ 0.5(2)
L-3			~ 0.5(2)
L-4			~ 0.5(2)
L-5			~ 0.5(2)
L-6			~ 0.5(2)
M			27,000 (6)

*The maximum value for the individual sampling site is listed. The numbers of sampling points measured is shown in parentheses.

We postulate that H_2 in the soil gas from the area along the Yamasaki Fault originated from chemical reactions between groundwater and the fresh surfaces of basement rock formed by fault movements. Fault movements imply both destruction of basement rock and pulverization of rock fragments. Fresh surfaces of solid substance are highly active chemically (*12*). The H_2 is probably produced by reactions between groundwater and fresh rock surfaces. Mylonitization of rock surfaces by the fault movement may also produce H_2. It is known that H_2 is produced by the pulverization of silica in the presence of water (*13*). Mylonite is a common constituent in the fracture zone of the Yamasaki Fault.

In the area of this fault, shallow earthquakes occur more frequently in the rainy season than in the dry season (*7*). Geoelectric studies indicate that the fracture zone along the Yamasaki Fault extends down to a depth of several kilometers (*14*). Goundwater can be expected to affect the deep layer through the fracture zone. The number of microearthquakes along the Yamasaki Fault has been noted to increase a few days after an isolated rainfall that follows a long dry period (*7*). We interpret this phenomenon as follows. The downward seepage of groundwater increases the pore pressure and decreases the effective stress. Destruction of rocks may result in the formation of fresh surfaces that have extremely high chemical reactivity. The reaction between these fresh surfaces and groundwater produces H_2.

In order to check the validity of this hypothesis, isotopic studies in the laboratory and continuous field measurements are needed. If the observed phenomenon can be accounted for by this hypothesis, the observations of H_2 degassing through the Yamasaki Fault made in this study will be the first evidence of the fault movement detected by a geochemical method.

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10. During our first series of observations in November 1978, sites A, B, and C were established inside the fault zone. In March 1979, observations were repeated at these sampling sites and were also made at sites D, E, and F. New ob-

servation sites I, J, K, and M were established in November 1979, extending the measurement line along the fault. Sites along the fault are distributed in the fracture zone (width, about 200 m). Measuring lines G, H, and L are perpendicular to the fault zone.
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Autoantibodies Against Axonal Neurofilaments in Patients with Kuru and Creutzfeldt-Jakob Disease

Abstract. *The serums of some patients with subacute spongiform encephalopathies contain an autoantibody in high titer against a normal fibrillar protein within the axon of mature central neurons in culture. The morphological features of this neurofilament, as demonstrated by immunofluorescence and immunoperoxidase staining, and the partial characterization of the antibody are described. The detection of this hetero-specific autoantibody is the first evidence of an immune reaction in the spongiform encephalopathies.*

Autoantibodies against normal filament protein of mature central neurons in culture from several species of laboratory rodents have been found in high titer in serum samples of 59 percent of patients with Creutzfeldt-Jakob disease (CJD), in 27 percent of patients with kuru, and, more rarely, in serums from patients with other chronic neurological diseases and from normal subjects. This antibody has been detected by indirect immunofluorescence and immunoperoxidase techniques, the substratum consisting of fixed cultures of large numbers of mature central neurons on coverslips prepared from fetal mice, hamsters, or rats as reported (*1*). Serum samples from a total of 22 patients with CJD, 28 patients with kuru, and 25 "normal" healthy control subjects have been studied for the presence and characterization of this autoantibody. We have also conducted a blind study of additional serum samples from patients with CJD, kuru, and other chronic neurological disorders.

All the serum samples and cerebrospinal fluids were stored at −70°C; they were tested at twofold dilutions in phosphate-buffered saline (PBS) starting at 1:16. The 22 CJD patients were verified both clinically and pathologically; from nine of them the disease has been experimentally transmitted to monkeys. The 28 kuru patients were all classical kuru victims who are now dead.

Neurons from the brains of rats, mice, or hamsters were grown in vitro as described (*1*) and tested after 20 days in culture when they were morphologically mature. The neurons and cell lines were tested by indirect immunofluorescence (*2*) and immunoperoxidase (*3*) techniques after they had been fixed for 10 seconds in cold acetone (−20°C) and washed in buffered saline. For demonstrating two different antigens in the same fixed culture we used rhodamine and fluorescein conjugates in a double fluorescence test (*4*). Indirect immunofluorescence staining was also done on sections cut from composite blocks of frozen tissues (rat liver, kidney, and stomach) (*5*), on frozen sections of brain from 7-week-old white Lewis rats, and on cultured fibroblasts from fetal rat lung. The serums were immunoabsorbed (*6*) with purified neurofilaments prepared as described by Schlaepfer (*7*) and with normal brain suspension made by homogenization of 0.5 g of brain tissue from a 7-week-old white Lewis rat in 100 ml of PBS.

Neuroblastoma (N_{2a}) and glioblastoma (C6) cell lines were from the American Type Culture Collection, Rockville, Maryland. Sheep brain (0A1B) and rabbit cornea (SIRC) cell cultures were purchased from Flow Laboratories, Rockville, Maryland. Rabbit antiserum to human globulins, goat antiserum to human globulins, rabbit antiserum to human IgG, IgM, or IgA (immunoglobulins G, M, or A), goat antiserum to human IgG specific for the Fab fragment, and goat antiserum to rabbit IgG rhodamine and fluorescein conjugates were from Cappel Laboratories, Cochranville, Pennsylvania. Rabbit antiserum to human globulins and rabbit antiserum to human IgG peroxidase conjugates were obtained from Miles Laboratory, Elkhart, Indiana. Rabbit antiserums to neurofilament and rabbit antiserums to intermediate (10 n*M*) filaments were obtained from B. H. Toh, Monash University, Australia; rabbit antiserum to tubulin was obtained from G. Rutter, H. Pette Institute, West Germany; rabbit antiserum to glial fibrillar acidic protein was obtained from A. Bignami, Boston, Massachusetts; and rabbit antiserum to actin was made in our laboratory as described by Owen (*8*). Cells were viewed with a Zeiss photomicroscope equipped with epifluorescence.

Serum samples from 13 of the 22 CJD patients (59 percent) and from 8 of the 28 kuru patients (27 percent), when examined by the immunofluorescence test, contained high titers of specific antibodies against the perikarya and the axonal neurofilaments of central neurons; these antibodies were observed along the full axonal length and thus revealed its tract throughout the cultures (Figs. 1 and 2). These same serum samples were also positive in the immunoperoxidase test (Fig. 3). None of the serum samples from 25 normal subjects showed similar staining. Antibody was not detected in the undiluted cerebrospinal fluid from the 13 CJD patients with positive serum.

We also examined, in a later fully blind study, serum samples from 38 patients with CJD, 63 patients with kuru, 71 patients with other chronic neurological and autoimmune diseases (eight with Parkinson's disease with dementia; seven with myasthenia gravis; six with neurosyphilis; eight with multiple sclerosis; nine with Alzheimer's; nine with Guamanian amyotropic lateral sclerosis and parkinsonism-dementia; and 24 with other miscellaneous diseases), and 50 normal subjects. The serum from 45 percent of the patients with CJD, 22 percent with kuru, 13 percent with the other neurological diseases (two with Parkinson's disease with dementia; two with Alzheimer's; one with Guamanian parkinsonism-dementia; two with Pick's disease; one with subacute sclerosing panencephalitis; and one with brain lymphoma), and 10 percent of the normal subjects, showed a similar autoantibody reaction (*9*). These positively reacting serums have not yet been characterized.

All of the positively reacting serum samples from the 13 patients with CJD and eight with kuru were titrated by means of the immunofluorescence test. Samples from four of the CJD patients were positive in dilutions up to 1:320; samples from one of the patients with kuru were positive in dilutions up to 1:1280 and, from another, up to 1:640. All of these samples with high titers of

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