The results from drilling beneath South Inyo Crater may have startled researchers, but having two types of rock in hand from essentially the same eruption pleases them to no end because it is a splendid example of a much discussed but poorly documented means of triggering eruptions. As Eichelberger and his colleagues see it, one portion of the mantle many tens of kilometers below the surface partially melted and sent the same basaltic magma rising beneath the entire length of the Inyo chain. To the south, the magma simply rose until it struck ground water whose steam blew bits of magma and rock onto the surface.

To the north, the magma encountered a barrier and never reached the surface. It must have oozed into the bottom of a shallow silicic magma chamber, perhaps the one whose top is thought to reach within 5 kilometers of the surface in the northwest corner of the caldera. Being heavier, the basaltic magma could not rise through the chamber. It could, however, encourage the silicic magma to push toward the surface. The added volume of magma would pressurize the chamber, the added heat would make the silicic magma more fluid and drive out its dissolved gases, and the rapid chilling of the basaltic magma would extract additional gas. All this would make for conditions favoring an eruption. Evidence that the two magmas actually came in contact with each other has been found in the form of globules of basalt in the silicic rock of the most southerly dome by Robert Varga and Gene Suemnicht of Unocal Geothermal Division and Roy Bailey of the U.S. Geological Survey in Menlo Park.

Investigations into eruption triggering and eruption style in the Inyo chain are hardly academic. The Inyo chain and the contiguous Mono Craters to the north form a volcanic trend that became active 40,000 years ago and for all practical purposes must be considered still active. The trend crosses into a caldera underlain by a magma chamber that is showing all too clear signs of life. During the past decade new magma has entered the chamber, triggering major earthquakes and bulging the caldera by as much as 50 centimeters. A cluster of earthquake activity on the southern edge of the caldera, just a few kilometers off the Mono-Inyo trend, has behaved as if a dike of magma was forcing its way toward the surface there.

A bit disconcertingly, the magma influx to the chamber seems to be continuing at a reduced rate of about 20 million cubic meters per year. At that rate, only a few years would be needed to accumulate the volume of magma produced by one of the eruptions 600 years ago. ■ **RICHARD A. KERR**

Gene Identity Confirmed

Last spring, Yueh-hsiu Chien, Mark Davis, and their colleagues at Stanford University School of Medicine found a new gene that appeared to encode a previously missing T cell receptor protein (*Science*, p. 1187, 5 June). Recent work by the Stanford workers and several additional groups has now confirmed the identity of the gene.

The gene in question encodes the δ chain of the T cell receptor, which is the cell surface molecule used by T cells to recognize and bind foreign antigens, thereby triggering the immune activities of the cells. A T cell receptor consists of two nonidentical, variant proteins that are associated with a third invariant protein, called T3. In most of the T cells of mature animals the nonidentical chains are of the α and β type, but in a small minority—up to 5%—they are of the γ and δ type.

By the beginning of 1987, all the genes had been cloned, except the δ gene. Immunologists wanted to track down this gene because knowing the δ chain structure would help them to clarify the function of the γδ receptor. They want to know whether it works the same way that the αβ receptor does in triggering T cell activities. In addition, there are indications that the γδ receptor may help to regulate T cell development in the thymus gland.

The finding of a candidate δ chain gene by the Chien-Davis group therefore attracted a great deal of attention, especially in view of the gene's unusual location. It is nestled among the coding sequences of the α chain gene.

Since then, Michael Brenner, Michael Krangel, and their colleagues at Harvard Medical School also identified a possible δ chain gene and showed that it is the human equivalent of the gene originally found by the Stanford researchers, which is of mouse origin.

Both the Stanford and Harvard groups, and that of James Allison at the University of California, Berkeley, have recently produced immunological evidence indicating that the gene encodes the δ protein, as proposed. They have found that antibodies that recognize a peptide specified by a portion of the δ chain gene also bind to the δ chain itself, and conversely, an antibody that is specific for the δ chain binds to a synthetic peptide made to correspond to the δ gene sequence.

In addition, Philippa Marrack and John Kappler's group at the Howard Hughes Medical Institute at the National Jewish Center for Immunology and Respiratory Medicine in Denver has shown that a partial amino acid sequence of an isolated mouse δ chain matches that of the predicted sequence of the protein encoded by the Stanford gene. "The immunological evidence is pretty convincing, but the most definitive evidence is the sequencing," Davis says.

With genes for all four T cell receptor proteins in hand, the way is open to exploring the relation between the structures of the two types of receptors and their functions in T cell activation and development. An unusual structural feature has already turned up in the δ gene.

The genes for the α, β, and γ chains, as well as the genes for other, related immunological proteins are assembled from either three or four separate segments of DNA. But Chien and Davis have found that some genes for δ chains are assembled from five DNA segments. These genes contain two D (for diversity) segments, instead of the usual one. The extra D should help to generate the structural variation that the protein products of the genes need to recognize a wide range of antigens. ■ **JEAN L. MARX**

ADDITIONAL READING

S. Hata, M. B. Brenner, M. S. Krangel, "Identification of putative human T cell receptor δ complementary DNA clones," *Science* **238**, 678 (1987).

H. Band *et al.*, "Immunochemical proof that a novel rearranging gene encodes the T cell receptor δ subunit," *ibid.*, p. 682.

E. Y. Loh *et al.*, "Identification and sequence of a fourth human T cell antigen receptor chain," *Nature (London)* **330**, 569 (1987).

W. Born *et al.*, "Peptide sequences of T-cell receptor δ and γ chains are identical to predicted *X* and γ proteins," *ibid.*, p. 572.

M. Bonyhadi, A. Weiss, D. W. Tucher, R. E. Tigelaar, J. P. Allison, "Delta is the C_x-gene product in the γ/δ antigen receptor of dendritic epidermal cells," *ibid.*, p. 574.

Y.-h. Chien *et al.*, "T-cell receptor δ gene rearrangements in early thymocytes," *ibid.*, p. 722.