blood cells (leukocytes) when they are exposed to a virus is represented by genes in more than ten locations on human chromosomes. At least three of those genes are lined up together along one chromosome. The Genentech researchers have already transferred each of five interferon genes into bacteria and observed production of slightly different interferons. One other gene appears to be a "pseudogene" unable to code for a protein. In contrast, the scientists find only one gene (and perhaps a quite poorly related second gene) for the type of interferon made by fibroblasts, another class of human cells.

An impressive balancing act of the components of interferon molecules was reported by both Goeddel and Charles Weissmann, a University of Geneva biologist who is associated with the company Biogen. They have created new genes by combining portions of different human leukocyte interferon genes. For instance, by combining one end of an interferon more effective in bovine cells than in human cells with the other end from an interferon with the opposite specificity, the scientists created an interferon more active in mouse cells than either of the parent molecules. Goeddel reported preliminary evidence indicating that interferons also may be more active in some types of human cells than in others.

Learning how interferon production is controlled naturally in human cells is now a step nearer, according to Weissmann. In recent experiments he transferred an interferon gene and its surrounding DNA into mouse cells and found that the transplanted gene, like a naturally controlled interferon gene, was not expressed until the monkey cells were exposed to a virus. "Perhaps this is true induction of transformed cells," Weissmann says. □

Pirated genes key to viral cancer

The startling discovery that normal cells contain genes resembling those of cancer-causing viruses has been turned around. Now the viral genes are considered stolen versions of normal cellular genes, and scientists are beginning to see the cancer virus as a tool to learn about both cancer and the regulation of normal development. J. Michael Bishop told the meeting in San Francisco of the First Annual Congress for Recombinant DNA Research that certain viruses "may have excerpted vital regulatory elements from the vast pool of cellular genes; if so, these elements are readily accessible for study."

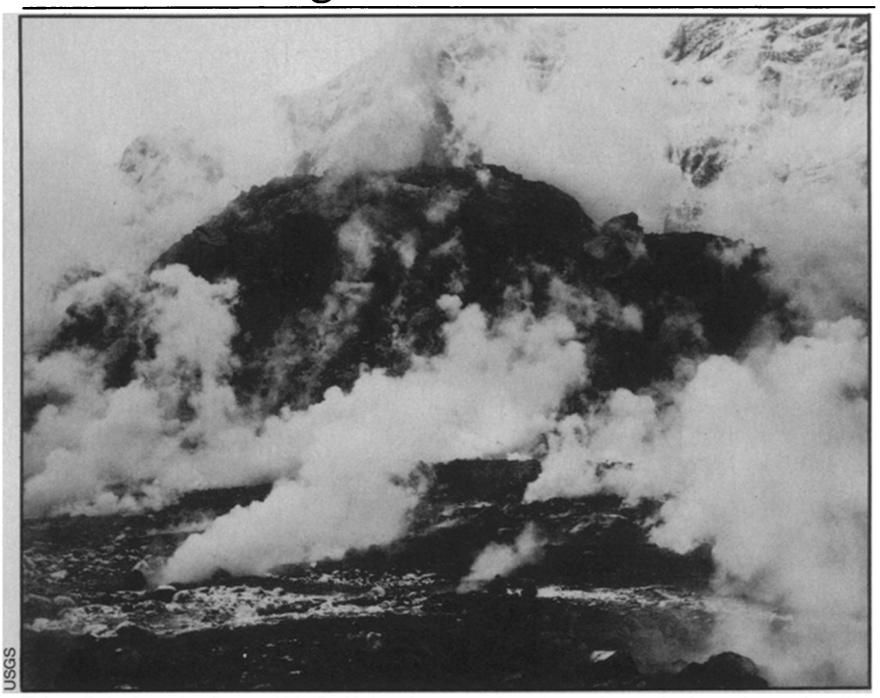
In each of about 15 animal viruses called retroviruses scientists have identified a single gene responsible for the virus's ability to transform a specific type of animal cell into a cancer cell. Although the genes that initiate cancer are different in these retroviruses, each has a corresponding normal vertebrate gene. In fact, in some experiments the normal cellular gene has been attached to a viral regulatory region and used to turn a normal cell into a tumor. The cellular genes that correspond to retrovirus genes appear to have a crucial function because they are highly conserved throughout the range of vertebrates. For example, viral genes that seem to have originated in chicken cells have homologues in creatures ranging from fish to humans.

A remarkable number of those genes responsible for cell transformation code for enzymes that attach phosphate groups to the amino acid tyrosine, Bishop says. The avian sarcoma virus was the first to be so identified (SN: 5/26/79, p. 345). Scientists have suggested that the gene inserted by the virus is overly enthusiastic, and an overabundance of the natural enzyme causes the cancer (SN: 8/4/79, p. 89).

Bishop now suggests that it is the timing of the activity, rather than simply the amount, that distinguishes cancerous from normal cells. At some points in an organism's development the natural cellular gene may be as active as the cancer-causing viral gene. The difference appears to be that the viral gene can't be turned off. Like developmental signals, the retroviruses are specific — each acts only on a limited set of cells. Bishop suggests each viral cancer gene aborts the developmental program of at least one line of normal cells. "We are pretty delighted to have our hands on one mechanism by which a neoplastic [cancerous] condition can be invoked," he says.

From the accumulated data Bishop speculates that the cellular genes identical to the genes that cause animal cancer via retroviruses are the same as the human "cancer genes," identified in pedigrees afflicted with high cancer risk, and also the same as the genes whose spontaneous or chemically induced mutation in body cells leads to cancer. Bishop, however, is skeptical that the recent results will have direct clinical applications. It will be difficult to meddle with a cancer gene that is also a normal gene essential for development. □

Mt. St. Helens grows a dome



Taken Feb. 6 on the floor of Mt. St. Helens's crater, this photo shows the lava dome that first developed in late December 1980. Between Dec. 27 and Jan. 4, scientists recorded an increased number of small earthquakes, accompanied by the oozing of molten rock through the small lava dome already in place. By mid-January, the dome was 300 feet high and 800 feet wide. Another flurry of small earthquakes occurred beneath the mountain Feb. 4 and 5, prompting scientists to issue an eruption alert. Plumes of steam rose about two miles above the volcano as more molten rock swelled the dome to about 500 feet high and 1,200 feet long by mid-February. Scientists monitoring the volcano, such as those in the lower center of the photo, have found little change in the shape of the volcano, but have detected a decline in the emission rates of carbon dioxide and sulfur dioxide since January. Based on the recent activity, scientists say the potential for explosive eruptions still exists, although the lack of precipitation and higher-than-normal temperatures in January have temporarily lessened the flood hazard from the sediment- and debris-laden rivers around the volcano.

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