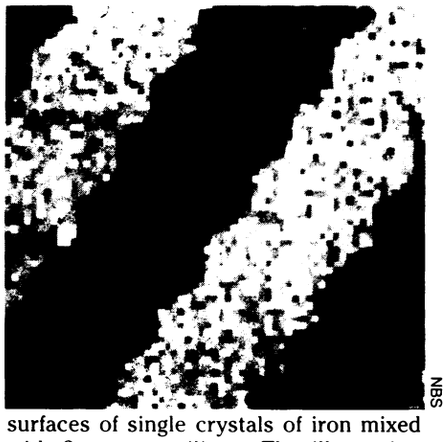

NBS

surfaces of single crystals of iron mixed with 3 percent silicon. The illustration above shows magnetic domains in a section of the crystal surface 50 by 50 microns. The difference in polarization between light and dark domains is approximately 60 percent. The best resolution of detail so far achieved with the device is 50 nanometers, a limit imposed not by electron characteristics but by vibration of the stage on which the sample rested.

— D.E. Thomsen

Ruling on nuclear waste

Pushed by a court order, the Environmental Protection Agency (EPA) last week issued rules strictly limiting the radiation that may be allowed to leak into the human environment as the result of the burial of high-level radioactive waste. The new rules apply both to military nuclear waste and to depleted nuclear fuel from commercial reactors.

The EPA rules, more than nine years in the making (SN: 1/8/83, p. 24), establish the general environmental standards for radioactivity that any storage or disposal facility must meet. This means that the Department of Energy (DOE), supervised by the Nuclear Regulatory Commission, must design and construct underground repositories that isolate nuclear waste for at least 10,000 years. If these containment requirements are met, EPA expects that no more than one cancer death per decade should result from any radiation leaks over a repository's lifetime.

"The small residual risks allowed by the disposal standards are comparable to those faced by future generations," says EPA Administrator Lee M. Thomas, "if the uranium ore used to produce the waste had not been mined to begin with."

EPA missed its deadline for issuing environmental standards, as specified by the Nuclear Waste Policy Act, by more than a year. Delays also plague DOE's search for a repository site. Late last year, DOE narrowed its search to three locations (SN: 1/5/85, p. 6), but lawsuits disputing the choices now blanket all three. □

HTLV-III virus: Themes and variations

HTLV-III, the virus that causes AIDS, consists of a whole spectrum of closely related but genetically distinct viruses, reports a team of researchers from the National Cancer Institute in Bethesda, Md., Litton Bionetics Inc. in Kensington, Md., and the Walter Reed Army Institute of Research in Washington, D.C., in the Aug. 23 SCIENCE.

The researchers isolated the AIDS-related virus from the blood of one healthy homosexual and nine patients who had either AIDS or the AIDS-related complex (ARC) — which has some but not all AIDS characteristics — and from lymph and brain tissue from eight deceased AIDS or ARC patients. Although all of the virus isolates had the same basic structure, no two were identical, and some varied considerably from the others. "The way we see the virus now is that there aren't strains — A, B and C — but rather a continuum of virus isolates," National Cancer Institute researcher Robert Gallo told SCIENCE NEWS.

None of the different types of virus could be associated with whether the patient had AIDS or ARC or was healthy. However, Gallo suggests that genetic differences in the virus may explain why different AIDS patients have such different sets of symptoms.

Two of the 18 patients were infected with more than one form of the virus, leading researchers to wonder whether the virus had infected the two patients more than once, or whether the virus changes while in the body.

Because none of the other patients in the group had such multiple infections, in spite of presumed ample exposure to other forms of the virus, the researchers suggest that one form of the virus tends to become dominant and somehow interferes with infections by other forms. However, the rarity of multiple infections might be only an artifact of *in vitro* culturing, the researchers say.

In culture, the HTLV-III virus doesn't change much, so many of the genetic changes in the virus probably occur when the viral DNA is transcribed into DNA in the body, says Gallo.

Whether the virus's genetic diversity will affect the difficulty of developing an AIDS vaccine is unknown.

Meanwhile, the virus has been discovered in the tears of an AIDS patient. "I don't think that tears are a major mode of transmission," says Gallo. "But this tells us that the virus is in places where we didn't know it could exist." The virus, which has been found in blood, lymph nodes, semen, saliva and now tears, is generally thought to replicate almost exclusively in the T4 white blood cells. Now Gallo says he thinks the virus is replicating somewhere else — exactly where, he says, will be revealed in a research report to be published in LANCET. *—J. Dusheck*

'Off switch' for cell division found

Certain cells in the body carry an "on switch" that enables them to begin dividing after a period of lying quiet. Researchers from the University of Connecticut in Farmington have now identified a corresponding "off switch." While their work is preliminary and does not at the moment present a way to turn off cancer cells, it does offer insight into the basic biology of cell reproduction.

Some cells constantly divide; others don't have the capability to divide at all. A third class, which includes liver cells, neurons and lymphocytes, can remain dormant for months or years, but when needed switch into a dividing state. Previous experiments have shown that dividing cells contain a "wake-up" factor, or activator, that promotes cell division by inducing DNA synthesis. The presence of an inhibitor has been suggested by other experiments showing that when resting and dividing cells are fused together, DNA production is somehow halted.

Janice K. Gutowski, Ann West and Stanley Cohen of UConn were able to extract a protein from resting white blood cells that inhibits DNA replication in cell nuclei stimulated by the activator. They describe the action of the protein in the August PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES (No. 15). The researchers have since found that the inhibitor can not only prevent activation but also turn off already activated nuclei.

"The picture that's emerging is that DNA synthesis in dividing cells represents a balance between positive and negative factors," Gutowski says. "It may be that the balance controls a normal cell's growth."

What needs to be determined, she says, is just how the inhibition works—or, in the case of tumor cells, doesn't work. "The [tumor] nuclei may not be responsive to the inhibitor or the cells don't make it," she says. "Either way you'd get a loss of growth control." If the problem is in the manufacture, there may be ways to manipulate the system. "If we have a way of getting an inhibitor into the cell," she says, "it may be able to slow tumor growth."

Manjusri Das of the University of Pennsylvania in Philadelphia, who did early work on cell activation, comments, "The research attacks an important issue." The question now, she says, is whether the protein is the key inhibitor, or whether its effect is a by-product of the experimental setup and does not play a role in the *in vivo* cell. *—J. Silberner*