

of the University of Arizona, the large, hot infant will begin to cool and contract for 1 million to 2 million years, until its density causes nuclear fusion to replace gravitational pull as the main source of luminous energy. By then a juvenile, the star can look forward to an adult life of about 10 billion years.

— T. Kleist

Plugging up leukemia cells

As scientists learn more about the immune system, they are developing sharper tools for manipulating it. At the Sixth International Congress of Immunology in Toronto earlier this month, Thomas A. Waldmann of the National Cancer Institute (NCI) in Bethesda, Md., described the use of an antibody that specifically targets only active forms of T cells, one of the two types of lymphocytes that make up the immune system. In experimental use against adult T cell leukemia, the agent caused temporary remission; Waldmann hopes it will eventually be useful in other T-cell-related conditions as well.

Each T cell is sensitive to a specific foreign antigen. To generate enough T cells to mount a defense, a T cell that has "seen" its target antigen initiates the cloning process in two ways—by producing interleukin-2, which stimulates T cell division, and by manufacturing specific cell-surface receptors for interleukin-2, making the T cell more sensitive to its presence.

Interleukin-2 can be helpful therapeutically. Steven A. Rosenberg and his colleagues at NCI have had initial success in using it to stimulate immune cells to fight cancer (SN:12/7/85,p.359). But in adult T cell leukemia, naturally produced interleukin-2 promotes an eventually fatal proliferation of T cells.

Waldmann and his colleagues injected five patients with an antibody to the interleukin-2 receptor in order to block its cell-stimulating effect. Since the interleukin-2 receptor appears only on activated T cells, including the leukemic cells, the antibody leaves the rest of the immune system alone. The treatment initially worked in two patients but, for undetermined reasons, eventually wore off. None of the patients had any side effects. As a next step, the researchers have just begun animal trials with a toxin-armed antibody aimed at killing the leukemic cells rather than just blocking their division.

"I think [the antibody] treatment has much broader ramifications," Waldmann says. T cells are also at fault in other conditions, such as transplant rejection and diseases marked by the immune system mistaking normal tissue as foreign; the antibody, he suggests, might prove useful in these cases.

— J. Silberner

Signs of how lead toxicity begins

Researchers at the University of California at Los Angeles believe they've uncovered evidence of how lead, at least initially, damages the brain. Their tissue culture studies, involving mouse cells, suggest that this heavy metal not only inhibits the growth of cells making up the blood-brain barrier but also may damage the barrier's membrane so that it no longer prevents entry of substances that could disrupt normal brain function. Lead's apparent ability to make this membrane leaky could explain the brain hemorrhages, tissue swelling and nerve dysfunction seen in people suffering from acute lead toxicity, according to an account of the research in the most recent (June 30) *TOXICOLOGY AND APPLIED PHARMACOLOGY*.

The researchers studied the endothelial cells that make up those capillaries in the brain that serve as the blood-brain barrier. Ordinarily, explains Karen Maxwell, one of the researchers, these cells prevent transfer of all but a few essential nutrients—like glucose—into the brain from the blood.

In their studies, the researchers incubated endothelial cells in a normal cell-growth medium to which they had added

fetal-calf blood serum containing inorganic lead. At those lead concentrations normally regarded as acutely toxic—for example, 60 to 80 micrograms of lead per deciliter ($\mu\text{g}/\text{dl}$) of serum—the researchers witnessed a number of adverse cell-function changes, all of which grew more pronounced as lead concentrations increased. Among these changes were an inhibition of cell growth; a reduction in the transport of blood glucose, the brain's energy source; and a reduction in "cell drinking"—the capture of substances from the fluid outside the cell by the cell's membrane. At lower concentrations of lead, such as 40 $\mu\text{g}/\text{dl}$, they saw no cell changes. Taken together, the researchers say, these findings suggest that the membrane of these cells may be lead's initial target.

Lead toxicologist Bruce Fowler of the National Institute of Environmental Health Sciences in Research Triangle Park, N.C., characterized the work as "potentially a very significant finding." However, adds Fowler, still to be resolved is whether this membrane effect is the sole early mechanism of lead toxicity or one of several.

— J. Raloff

Concern over Chernobyl-tainted birds

After the meltdown and the fallout, the effects of the Chernobyl nuclear accident continue to ripple outward. A meeting between European and U.S. scientists at an ornithological convention last month has raised concerns that migratory birds may be carrying radiation throughout Europe. And the National Wildlife Federation (NWF) has requested that the United States become "more actively involved" in determining Chernobyl's effects on birds.

According to I. Lehr Brisbin Jr. of the University of Georgia at Athens, who led the roundtable discussion at the Nineteenth International Ornithological Congress in Ottawa, Chernobyl sits in a major migratory flyway. "The birds funnel north from Africa... and come up through the Ukraine," he says. The accident occurred during the northward spring migration.

Monitoring of radiation levels since the April 28 disaster has concentrated on immediate dangers to human health, such as those posed by livestock due to be slaughtered for food. But wildlife may be more heavily contaminated than livestock, which have been given relatively "clean" feed, says Brisbin—and because many Europeans hunt, and eat what they catch, the contamination may threaten more than long-term ecological stability. In addition, he says, "many Third World countries depend on fish and wild game

for food."

Scientists at the meeting pointed out that radiation has been measured in wild reindeer, according to Douglas Inkley of the Washington, D.C.-based NWF, and the government of Sweden has reportedly recommended that citizens limit their consumption of wild game. But migratory birds would be a more efficient vector for radioactive isotopes, Inkley says. Italy, "downstream" from the Ukraine during the fall migration, is considering a ban on bird hunting this year; more than 20 million birds are hunted and eaten each year in that country.

Prompted by concerns raised at the ornithological convention, the NWF in June asked the Environmental Protection Agency (EPA) to study the problem, citing a 1978 U.S./USSR convention to protect migratory birds and their habitats. The letter was forwarded to the Department of Interior, an EPA spokesperson told *SCIENCE NEWS*, adding, "We agree that something ought to be done."

At this point, nobody knows whether there is a serious problem, according to Brisbin. "What we need are good [bird] population studies," he says. "It may be smoke and no fire. [But] it would be a shame to spend all the time and energy we're spending on [monitoring] livestock and vegetables, and ignore an important source of [potential] contamination."

— L. Davis