Memo may undermine lead phasedown plan

"It just seemed to me that anytime you make scientific decisions, you'd want to have supporting information." That was the reasoning, explains Eric Goldstein of the Natural Resources Defense Council in New York, behind his suspicion that the Environmental Protection Agency might have outdoor-monitoring data on lead concentrations in ambient air. After all, the agency was proposing a relaxation of the levels of lead permitted in gasoline (SN: 2/27/82, p. 132). Goldstein suspected that to justify such a relaxation the agency must have monitored lead in the air and been satisfied that existing regulations were proving more conservative than necessary. But that's not what he found.

What he did find was evidence of consistent misplacement of lead monitors in the United States and a resulting underestimation of airborne lead levels.

Goldstein made a Freedom of Information Act request to the agency, asking EPA to hand over whatever information it had on lead monitoring and the accuracy of existing monitors to reliably gauge lead pollution. Among documents he received was a 15-page memorandum dated Jan. 27, 1982, by Robert Kenney, chief of the State and Local Programs Section within EPA's office of Air, Noise and Radiation. In it, Kenney noted that, "as part of the agency's review of lead-in-gasoline phasedown regulations, I was asked to review the impact that changes to this regulation would have on ambient concentrations of lead." To do that, Kenney wrote, "it is crucial" that an adequate data base exist recording past concentrations. However, he reported that "such a data base is lacking."

There are data. And Kenney reviewed three EPA studies completed in the fall of 1981 that attempt to describe trends in ambient-lead levels based on those data. The problem, as he points out in graphic detail, is not only that the data are very spotty, but also that they are unrepresentative of the maximum levels to which individuals (particularly children and urban dwellers) might be exposed.

Kenney noted that most of the monitors offering lead data were primarily designed to measure total suspended particulates, and thus were situated in places other than where one would expect to find high lead levels. For example, EPA criteria establish 7 meters as the maximum height for measuring lead from auto emissions along roadways. Yet some monitors were 25 to 32m above the street. And though the agency made 15m the maximum allowable setback from a roadway for taking lead readings, one Boston monitor was 600m back from traffic. At least one Washington, D.C., monitor appeared to be on the upwind side of the road, something an EPA study indicates could seriously affect the accuracy of readings attributed to downwind exposures by as much as 0.6 microgram per cubic meter of air. (That's important when the lead level permitted by EPA is only 1.5 $\mu g/m^3$.) Nonetheless, two of the four most reliable roadway monitors showed readings that exceeded EPA's lead standard. Another indication of the importance of placing roadway monitors properly is a warning contained in one of the 1981 EPA studies: that automotive emissions are believed to be the "predominant source" of airborne lead in urban settings.

The Kenney memo, released publicly for the first time earlier this month, illustrates all too well, Goldstein says, something characteristic of EPA's posture in a number of public-health controversies: "Namely, that if you don't have sufficient research or you don't do sufficient monitoring, it's easier to say there is no problem." But Goldstein hopes that publicizing the memo will stifle any potential assertion of "no problem" in this case. —*J. Raloff*

Now brain proteins fight disease

The endorphins and enkephalins — the "natural opiate" brain proteins that influence the mammalian mind and behavior — were some of the most exciting medical discoveries of the 1970s (SN: 11/25/78, p. 374). Now they have been found to influence the body's immune system and its ability to fight disease, three new research reports reveal.

The first report comes from N.P. Plotnikoff, C.G. Miller and A.J. Murgo of Oral Roberts University School of Medicine in Tulsa, Okla. They inoculated mice with leukemia cells, then gave some of the mice leucine enkephalin, others methionine enkephalin and still others no treatment at all. Whereas the control mice died two weeks later, the enkephalin-treated mice survived four weeks. The researchers repeated the experiment, but gave the enkephalin-treated mice the enkephalin antagonist naloxone as well. The treated mice lived a little longer than the control mice, but not much.

The results of both experiments, the researchers reported at the Second International Conference on Immunopharmacology held in Washington recently, "suggest that the enkephalins are endogenous immunomodulators" — that is, that they help the immune system fight disease. The results, Plotnikoff said in an interview, also suggest that the enkephalins might benefit cancer patients. In fact, he and his coworkers have already found in a test-tube experiment that the enkephalins enhance the responses of T cells taken from cancer patients. T cells are a major category of cells of the body's immune system known to play a signficant role in the fight against tumors. The researchers are now seeking approval from the Food and Drug Administration to inject enkephalins into cancer patients.

The second report comes from J. Edwin Blalock and colleagues at the University of Texas Medical Branch at Galveston. They attempted to see whether in *in vitro* experiments, various forms of endorphin and enkephalin influenced B cells' production of antibodies. B cells, like T cells, help make up the immune system. The researchers report in the July PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES that all brain proteins inhibited antibody production, but to varying degrees. What's more, the proteins' ability to prevent antibody production appeared to derive from their binding to specific receptors on B cells.

B cells, in fact, even seem capable of producing their own endorphins, Blalock and his colleagues have found in recent months. T cells, too, seem capable of the same feat. But perhaps most important, the endorphins produced by immune cells seem to affect mammalian behavior as do endorphins made by the pituitary gland below the brain. Thus, as endorphins manufactured by the pituitary influence the immune system, endorphins made by the immune system influence the brain. Blalock and his co-workers are now studying the structures of the endorphins made by B and T cells.

The third report comes from Steven C. Gilman of Scripps Clinic in LaJolla, Calif., and colleagues. While alpha-endorphin and methionine enkephalin do not enhance the ability of T cells to proliferate, beta-endorphin does, they report in the July PNAS. This is one more indication that beta-endorphin influences the immune system's ability to fight disease.

Because beta-endorphin is known to be released from the pituitary in times of stress, it may particularly affect the disease process at such times, Gilman and his team suspect. Whether the protein enhances T cell proliferation, inhibits antibody production or affects disease by some other means during stress has not yet been determined. —*J.A. Treichel*

Landsat 4 launched

Amid uncertainty and controversy about the future role of the U.S. in monitoring the earth's resources from space, the Landsat 4 satellite (SN: 7/3/82, p. 4) was successfully launched on July 16. Its multispectral scanner, similar to sensors flown on the three previous Landsats, began returning data three days later for processing into images that combine four different spectral bands. New on Landsat 4 is its "thematic mapper," using seven high-resolution bands extending from the visible to the far (thermal) infrared. Its visible-light channels were working by July 21, and early reports indicated the satellite's overall health to be good. □