

PCB's in cow's milk

Some dairy farms have had their products removed from the market because of polychlorinated biphenyls (PCB's) in the milk of their cows. Aroclor 1254, a PCB with 54 percent chlorine, was used as an ingredient of coatings for the insides of silos, and the cows ingested the substance in feed that had been stored in the silos. The amounts in milk exceeded Food and Drug Administration maximums.

Three U.S. Department of Agriculture scientists report that the PCB remains in cow's milk for a considerable period after the feeding with contaminated materials ceases. The rate of removal of PCB from the cow's milk was only about 1 percent a day. "When the farmers are removed from the market it is a long and costly process before they can return."

The researchers—G. F. Fries, G. S. Marrow Jr. and C. H. Gordon—note that the fat-soluble PCB was concentrated in milk fat and that its behavior generally "was quite similar to the most persistent chlorinated hydrocarbon residues."

Pesticides in ambient air

The evidence grows that the atmosphere is a major route for transmission of DDT worldwide (SN: 1/8/72, p. 30). It would follow that DDT, and perhaps other pesticides, may be ambient air pollutants.

A group of researchers from the Environmental Protection Agency and the Oklahoma State Department of Health sampled air at 45 locations in the United States and the Bahamas in 1970 and 1971 for pesticide residues. They found various pesticides in the air at all locations. Levels do not approach unacceptable threshold limits but "respiration of air containing some of the higher levels reported would contribute to the individual's total pesticide exposure." Both organochlorine and organophosphate pesticides were discovered, with the highest concentrations during times of field spraying.

In 1970, 30 sites collectively produced evidence of 15 organochlorine and 4 organophosphate pesticides. In 1971, analysis at 45 sites revealed 21 organochlorine and 11 organophosphate residues. Some pesticides reported at half or more of the 1971 sites include: pp-DDT, pp-DDE, Dieldrin, op-DDT, Diazinon, Heptachlor, Malathion, esters of 2-4-D and Endrin.

The researchers are A. R. Yobs, B. L. Stevenson, J. J. Boland and H. F. Enos of EPA and J. A. Hanan of the Oklahoma state agency.

Wayward pesticide aerosols

A University of California at Davis researcher reports that aerosols from pesticide spraying of crops can contaminate an entire airshed and be found 50 miles from point of discharge under certain conditions.

Norman B. Akesson of the Agricultural Engineering Department says farm operators would probably have to increase the use of coarse dry granules and of large-droplet applications to prevent pollution of surrounding areas. Engineers are now working on techniques and procedures to control atomization. He adds that even with the use of large droplet nozzles, 5 to 50 percent of emissions are in aerosol form.

A further complication for farmers is that microbial insecticides require very small droplets. This makes eventual conversion more difficult.

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T and B cells' teamwork in immunity

There are two kinds of immune responses in the body—antibody protection and lymphocyte protection. How the two interact is, as one immunologist puts it, subject to "witchcraft." Researchers have been pushing their techniques to the limits to come up with some scientific answers.

"T-cell" lymphocytes derive from the thymus; "B-cell" lymphocytes are made in the bone marrow, spleen or other areas of the body. Since T cells do not secrete antibodies and B cells do, it seems plausible that T cells might cause B cells to produce antibodies. Now Marc Feldmann and Antony Basten of the Hall Institute of Medical Research in Melbourne report strong evidence that T and B cells do collaborate in providing antibody response. They have found, *in vitro*, that T cells interact with antigen, then give off a chemical of some sort. This chemical in turn triggers immunity in the B cells.

It is possible, the authors write in the May 3 *NATURE NEW BIOLOGY*, that the T-cell chemical is a special kind of antibody that concentrates antigen either into the B cell or onto a third-party cell, such as a macrophage. Or the chemical exuded by T cells may be induced by the antigen.

"Reverse transcriptase" and cancer

In 1970, Howard Temin of the University of Wisconsin rocked the biological world by discovering an enzyme that makes DNA from RNA rather than the usual RNA from DNA. Since then, cancer researchers have been trying to prove that the enzyme, "reverse transcriptase," causes cancer.

To date the enzyme has been found not just in tumor viruses, but also in animal and human cancer cells. There is some controversy whether it might also exist in normal cells. So a better understanding of how the enzyme works is imperative before it can be proved guilty, or exonerated, of turning a normal cell into a cancer cell.

In the May 19 *SCIENCE*, Marjorie S. Robert, R. G. Smith and R. C. Gallo of the National Cancer Institute report that they have made a wide comparison of a number of enzymes that make DNA. They have found that reverse transcriptase can be distinguished from the other DNA polymerases by several methods. The most blatant criterion is that reverse transcriptase can make DNA from its own genetic RNA material; the other enzymes cannot. Also, if the DNA that is made is put back with the virus RNA, it will complement the latter in its genetic makeup.

Diabetic cells and resistance to insulin

Diabetes is essentially a deficiency in the hormone insulin, which in turn decreases the body's use of carbohydrates. But researchers in pharmacology at Johns Hopkins University School of Medicine have found that the problem is compounded by an inefficient response of target cells to the insulin that is made available to them. G. Vann Bennett and Pedro Cuatrecasas have found that fat cells taken from diabetic animals respond less efficiently to insulin than normal cells do.

The reason for the diminished response, they report in the May 19 *SCIENCE*, is not that insulin does not bind to the fat cells properly. It does. They conclude that the decreased response to insulin must take place after insulin binds to the cells. Enzymes on the cells' membranes, for example, might abolish the effect of insulin on the cells.