Transfer factor can prevent disease

It has been exactly a quarter-century since a New York University scientist discovered transfer factor — the chemical in white blood cells that exerts immunity against a specific virus, bacterium, tumor antigen or some other chemical that is foreign to the body. It was hoped that when this factor was injected into another individual, it would impart immunity against the specific antigen. And it did — in some cases.

Because the discovery of transfer factor was the first demonstration that cellular immunity against specific antigens could be transferred from one human to another, it naturally raised hopes that injected transfer factor could cure a wide gamut of diseases (SN: 2/9/74, p. 86). Alas, these hopes haven't panned out. Only one double-blind, controlled trial has shown that transfer factor can successfully treat disease. Now, though, another promising use for transfer factor has been reported — disease prevention, a ploy that hasn't been tried in humans before. Russell W. Steele of the University of Arkansas for Medical Sciences in Little Rock and his colleagues report in the Aug. 14 NEW ENGLAND JOURNAL OF MEDICINE that transfer factor can prevent chickenpox in children who are seriously threatened by it.

Leukemia, once an invariably fatal disease in children, now is curable in one-third of newly diagnosed patients. However, young leukemia patients are still seriously threatened by infectious diseases, notably chickenpox, and there has been no way to safeguard them. Steele and his co-workers now find, however, that transfer factor can be effective against chickenpox in such patients.

They screened healthy adults for cellular immunity against the chickenpox virus until they found five adults with unusually strong responses to the virus. All five were convalescing from chickenpox. These five agreed to donate their chickenpox virus transfer factor. Steele and his team extracted the transfer factor from the donors and injected it into 31 young leukemia patients. Another 30 children with leukemia received a placebo injection. Then all 61 patients were observed during the next 12 to 30 months. During this period, 16 patients who had received transfer factor were exposed to chickenpox (at school or by other natural means); only one of the 16 got the disease. During the same time, 15 in the placebo group were exposed to chickenpox; 13 of the 15 got it. Thus it looks as if transfer factor is highly effective in safeguarding young leukemia patients against chickenpox, the researchers conclude.

But the finding has even larger implications, Charles H. Kirkpatrick, a physician with the National Jewish Hospital and Research Center in Denver, points out in an accompanying editorial. This finding, he says, suggests that transfer factor may be used to protect humans against not only chickenpox but against other diseases for which vaccines are not presently available. "This important contribution," he concludes, "should lead to additional controlled clinical trials." □

Melanoma finding: tumor-receptor link

Receptors for the hormone estrogen occur in nevi — skin tumors, moles and birthmarks — of persons with melanoma, but not in the similar-appearing nevi of persons free of the pigment cell cancer, report researchers from Chicago's Cook County Hospital and University of Illinois Medical Center in the Aug. 22 issue of the JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION.

Four years ago, estrogen receptors were found on malignant skin tumors by National Cancer Institute researchers who suspected steroid involvement in melanoma because the cancer mainly appears during times of dramatic hormonal change, such as at puberty and during a woman's child-bearing years.

The Chicago group, led by Prabir K. Chaudhuri, found that both benign and malignant tumors of persons with melanoma have the receptor. Of 27 malignant nevi, 37 percent had the receptor, and of 21 benign nevi, 41 percent had the receptor.

The finding may indicate a role for estrogen in the development of melanoma. It "suggests that steroid hormones such as estrogen may represent a molecular alteration associated with a potential pathophysiological transformation of a benign nevus to its malignant counterpart," the researchers say.

To measure the presence of estrogen receptors, the researchers removed the nevi, then froze, pulverized, and homogenized them in a chemical solution. They then centrifuged the homogenate and measured the amount of radioactive estrogen that bound to molecules in the "soup."

This finding suggests that the presence of the estrogen receptor may serve as a biological marker for melanoma, but, "It's too early to see if it will be a diagnostic tool," explains Chaudhuri. "If the data hold up, the marker might select a high risk group."

Steroid receptors also occur in some breast, kidney and uterine cancer, and hormones are sometimes used to treat tumors with steroid receptors. Chaudhuri and his associates are doing further study to determine whether melanoma marked by steroid receptors will respond to hormone treatments the way other estrogen-receptor cancers do. □

Natural radiation: Low on risk gauge?

In two geographical basins of China, fine particles of an earthy, metal-containing material — washed down from surrounding hills and mountains — raise the background radiation level about three times that of neighboring areas. The inhabitants of these "high background" regions were the focus of a health survey reported in the Aug. 22 SCIENCE.

Wei Luxin and colleagues of the Laboratory of Industrial Hygiene in Beijing compared the health status of high background and control area inhabitants. The Chinese investigators compared the frequency of hereditary disease, congenital deformities, malignancies and chromosomal aberrations, growth and development of children, rate of spontaneous abortion and daily intake of natural radionuclides. The results of the health survey conducted from 1972 to 1975 show no significant difference between the basin and control groups.

The Chinese researchers report that the average annual dose absorbed from external radiation was 196 millirads for basin inhabitants and 72 millirads for the control area person. In background radiation measurements, a millirad — a unit that measures how much energy comes from radiation — is nearly equivalent to a millirem (mrem) — the unit that scientists use to measure the damage that radiation causes to human cells. In the United States, average annual exposure ranges from 23 mrems in Atlantic and Gulf Coastal Plain states to 90 mrems on the Colorado Plateau. □

MS: A measles virus trigger?

What causes multiple sclerosis? Scientists aren't sure, but gradually mounting evidence, the latest of which is reported in the July PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES, suggests that the slow but devastating central nervous system disease might be triggered by a measles virus.

There already is evidence that one chronic, invariably fatal central nervous system disease — subacute sclerosing panencephalitis — is caused by a variant of a measles virus. Also a chronic, progressive encephalitis (inflammation of the brain) has been observed in children congenitally infected with a measles virus. In both these conditions the onset of neurological damage was delayed months or even years after the virus infection. Thus it is conceivable that still a third chronic central nervous system disease — multiple sclerosis — also might be caused

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by a measles virus, months or even years after virus exposure.

Previous studies have suggested that multiple sclerosis is associated with a viral infection early in life. Antibodies against measles virus have been found to be more prevalent in the spinal fluid of multiple sclerosis patients than in healthy subjects and measles virus-like particles have been been visualized in the brains of multiple sclerosis patients (SN: 12/2/72, p. 362).

Now Donald R. Carrigan and Kenneth P. Johnson of the University of California at San Francisco have inflicted a multiple sclerosis-like disease on hamsters by injecting them with a measles virus early in life. They used a strain of measles virus originally isolated from the brain of a child with subacute sclerosing panencephalitis, cultured the virus, then injected it into the brains of newborn hamsters. The virus produced an acute, lethal encephalitis in about one-fourth of the animals. The surviving hamsters, which showed no acute illness, as well as control hamsters, were placed in coded cages and observed for multiple sclerosis-like symptoms. The animals with symptoms were then sacrificed and examined for signs of disease.

As Carrigan and Johnson report in the PNAS, about one-fifth of the measles virus-infected animals developed multiple sclerosis-like symptoms and disease between 5 and 50 weeks after injection. What's more, the animals' symptoms — partial or complete paralysis, on-and-off muscle twitching, diminished movement and so forth — were similar to those of multiple sclerosis victims, and the animals' pathology was like that of multiple sclerosis patients as well. It consisted of an infiltration of mononuclear cells into the central nervous system, leading to removal of myelin from nerves, then a death of neurons and a replacement of them by glial cells. This pathology continued in a progressive, episodic manner until large areas of the central nervous system were involved. The major difference between this animal disease and multiple sclerosis in humans is that in the former, neuronal death is predominant, and in the latter, demyelination of neurons is predominant. But in both diseases the ultimate damage is replacement of nervous tissue with glial cell scars. Thus, these findings are further evidence that measles virus plays a role in the origin of multiple sclerosis, Carrigan and Johnson conclude.

Before a measles virus can be definitely implicated in mutliple sclerosis, though, scientists have to be able to identify it on a regular, reproducible basis in multiple sclerosis tissue, and even better, be able to isolate and culture it from multiple sclerosis tissue. So far this hasn't happened. In fact, Carrigan and Johnson were unable to detect measles virus in their hamsters' diseased central nervous system tissue. □