medical sciences

Success with leukemia treatment

Acute childhood lymphocytic leukemia, which constitutes 80 percent of all childhood leukemia, has in the past nearly always proven fatal. But reports from the St. Jude's Children's Research Hospital in Memphis show increasing success in treating the disease. Seventeen percent of lymphocytic leukemia patients entering St. Jude's for treatment from 1962 to 1965 have been free of leukemia for six years and have been off all treatment for four years or more. Sixty percent of patients receiving treatment between 1967 and 1968 have been free of leukemia for three years or more and are off treatment.

The therapeutic formula that Dr. Donald Pinkel, medical director of the hospital, and his colleagues use consists of a combination of all the drugs that have been established as effective against lymphocytic leukemia, and radiation treatment of leukemia in the central nervous system, which drugs do not reach in effective concentrations. Although the therapy may touch off some serious side effects, such as pneumonia, liver damage or depression of bone marrow, Dr. Pinkel believes the chances of cure outweigh possible toxic effects.

The St. Jude formula, with some modifications, is now being tried at some other hospitals in the United States as well.

Detection of colon cancer

Since 1965 Dr. Phil Gold of Montreal's McGill University, and co-workers, have shown that all human cancers of the colon and rectum contain an identical tumor-specific antigen (SN: 1/2/71, p. 12). They have also developed a test to measure the antigen in human blood samples. Because cancers of the colon and rectum are the second leading cause of cancer deaths in the United States, perfection of this early detection method might help save many lives.

The National Cancer Institute has now contracted with Tufts University School of Medicine in Boston to evaluate Dr. Gold's early detection test for colon and rectal cancers. If this test is indeed shown specific for cancers of the colon and rectum (other investigators have been unable to confirm the specificity of Dr. Gold's test), Tuft researchers will then determine the assay's usefulness for early cancer detection.

Gestation and immunity

To determine the role of an antigen in increased immunity against disease, Drs. Thomas Gill, Heinz Kunz and Colette Bernard of the Harvard Medical School immunized female rats with the antigen, then mated them. First and second generation litters indeed produced more antibodies. The antigen was transmitted from mothers to offspring, and became localized in bone marrow and sometimes in thymus and spleen as well.

The Harvard workers thus postulate that the offsprings' ability to make larger amounts of antibody after their mothers were immunized was due, at least in part, to stimulation of the lymphoid system of the developing fetuses by antigen transmitted from the immunized mothers.

The experiments, the researchers report in the June 25 SCIENCE, also suggest clinical immunological engineering possibilities. Giving a long-acting vaccine during pregnancy, say, might counter certain viruses' teratogenic effects on the fetus, as well as provide the fetus with greater immunity against infectious diseases in the postnatal period.

Juncture of nerve and muscle

The synapse operation has intrigued neurobiologists for some time. In simple terms, what has been known is that a nerve cell releases a transmitter substance called acetylcholine. Acetylcholine causes a muscle cell touching the nerve cell to contract. An enzyme called acetylcholinesterase is then released, destroying the transmitter and turning off muscle contraction. Work reported in the July 14 NATURE NEW BIOLOGY throws more light on these mechanics.

By using an enzyme called collagenase to detach acetylcholinesterase from rat muscle tissue, Drs. Zach Hall and Regis Kelly of the Harvard Medical School have shown that acetylcholinesterase found at the juncture of nerve fiber and muscle fiber can be removed without damaging either the enzyme or nerve-muscle cell membranes. (They then found that this enzyme material is indeed responsible for the switch-off of neurally released acetylcholine.) By contrast acetylcholinesterase in other portions of the muscle cell was not released by collagenase treatment, suggesting that the structure or site of attachment of this enzyme material must differ from that of the acetylcholinesterase at the juncture of nerve and muscle fiber.

"Although the work deals with the nerve-muscle juncture," Dr. Hall explains, "it may provide insight into the structure of other cell surfaces as well. Interaction between cell surfaces is thought to be crucial for tissue organization, regulation of cell growth and other physiological processes."

Genes and hemophilia

It has been known for some time that Negro women who are hemophilia carriers have lower concentrations of a blood protein called "Factor VIII." It is also known that the genetic locus for this deficiency is closely linked to the locus on the X chromosome that determines the kind of blood protein called glucose-6-phosphate dehydrogenase a woman inherits. Dr. Paul McCurdy of the Georgetown University School of Medicine in Washington used this knowledge, along with family pedigree studies and a technique called electrophoresis, to determine whether a pregnant Negro woman, who had a brother with hemophilia, might be a hemophilia carrier. Her chances of being one were quite low, Dr. McCurdy found.

Since readily detectable G-6-PD variants are also known in some women from Mediterranean backgrounds, the diagnosis might also be applied to them, Dr. McCurdy reports in the July 22 NEW ENGLAND JOURNAL OF MEDICINE. However, there must be evidence of hemophilia in the family to determine whether a woman might be a carrier, Dr. McCurdy points out. G-6-PD detection coupled with pedigree studies might also be used to diagnose hemophilia in fetuses of mothers from these racial or ethnic backgrounds, when hemophilia is evident in the family, Dr. McCurdy says.