biological sciences

From our reporter at the meeting of the American Society for Microbiology in Philadelphia

DNA repair and cancer therapy

Tsugio Satoh and Nobuto Yamamoto of the Temple University School of Medicine in Philadelphia report that human and animal tumor cells damaged by ultraviolet radiation can repair themselves better than can normal cells. This appears to be due to an increased concentration of DNA polymerase, one of the enzymes that is known to be needed for repairs.

Such news is not particularly encouraging to chemotherapists, who radiate cancer patients to selectively knock out actively dividing cancer cells. Obviously not only are cancer cells able to repair themselves better than normal cells; normal cells indiscriminately hit by radiation may be destroyed because their DNA repair mechanisms are not as resilient as those of tumor cells. Whether laboratory experiments with ultraviolet radiation can be extrapolated to the clinical situation, though, is not certain. The X-rays used in chemotherapy are vastly more powerful than ultraviolet radiation applied to tissue cultures in the laboratory. And whether X-rays would favor tumor cells' DNA repair, or normal cells' DNA repair, is not known.

DNA repair by photoreactivating enzyme

R. J. Centanni and N. Nakamura of Laredo Junior College in Texas report that when *Trypanosome cruzi*, a single-celled animal, has its DNA damaged by UV light, DNA can be subsequently repaired with exposure to certain, carefully controlled levels of visible light. The enzyme thought to assist in this repair is called photoreactivation enzyme. Scientists are mystified as to why this animal, which lives in the insides of other animals, should have use for such a light-dependent repair enzyme system under normal conditions.

When viruses compete for a host cell

Polio virus turns off the mosquito virus sindbis when both are put in a human cell under experimental conditions, T. Sreevalsan and Hortensia Rosemond of the Georgetown University School of Medicine have found. This work underscores some other scientific evidence that there is a hierarchy of competing viruses for a host cell.

Like about half of all known viruses, polio virus is believed to take over the processes by which a cell replicates. It may be able to do so by incorporating its genetic information—six to ten genes—into the genetic material of the host cell. Thus, if one virus can compete with another virus for replication in a cell, it is possible that one day harmless viruses might be used to usurp disease-causing viruses in people.

Antibodies and cancer antigen

Antibodies that react with a protein component, or antigen, of viruses that cause cancer in mice and cats have been found in the blood of human cancer patients. This work was reported by David S. Yohn and Richard G. Olsen of the Ohio State University College of Veterinary Medicine.

The antibodies appear to be directed to an antigen shared by all known RNA cancer viruses in animals. The antigen is also present in two viruses currently among the human cancer virus contenders. This finding means that the patient has come into contact with the antigen, has made an immune response to it and has produced antibodies. It does not prove that some human cancers are RNA-virus induced, but it is added evidence in that direction. The antibodies were found in the blood of patients with several kinds of cancers—acute lymphatic leukemia, breast cancer, osteosarcoma, and lymphosarcoma. All these diseases have been suspected of being virus-induced.

The findings also suggest that immune responses to RNA tumor viruses occur in humans.

Cancer virus and immune response

For the past several years there has been strong scientific interest about whether and how the body's immunological defenses might respond to cancer viruses. Robert Campbell, Walter Ceglowski and Herman Friedman of Pennsylvania State University present evidence on the immune responses of mice to leukemia virus.

They first examined mice that developed leukemia and died. They detected an immune response, but an inadequate one. Then, looking at mice that resisted the virus and did not get cancer, they witnessed an immune response as well. But it came at a cost. After the animals were exposed to the virus they became particularly susceptible to other kinds of infection. In other words, it looked as if the mice that were managing to fight off the cancer virus had their immunological defenses weakened as a result.

The immune responses detected in the resistant and nonresistant mice appear to be both humoral and cell-mediated. When immunity is humoral, protection is provided by antibodies. When immunity is cell-mediated, lymphocytes fight off the foreign invader.

Cell membrane erosion by an invading virus

Most microbiologists believe that the cell membrane is particularly crucial in the cell's resistance against disease. Yet little is known about the makeup of a membrane, and even less about how the cell defends itself at the membrane against a virus invader. However, Toby Hecht and D. F. Summers of the Albert Einstein College of Medicine in New York City have found that vesicular stomatatis virus, a cow virus, causes a decrease in a protein on the host-cell membrane called Transplantation Antigen. This antigen is probably only one of several minute structures on the membrane that create hostile hypersensitivity reactions if the cell is threatened with invasion by a foreign substance.

The finding has several implications. One is that certain viruses, such as cow virus, may make their way into and out of the host cell by causing changes in preexisting antigens on the membrane. And clinically, it may one day become possible to use such viruses to lessen cell hostility to foreign tissue, as in organ transplants. Any viruses used, of course, must not be those that cause human disease.