Severe Arthritis Under Attack

Arthritis, which strikes one in seven people in the United States, costs $14 billion each year, including $5 billion in health care and another $6 billion in lost income due to illness, estimates the Arlington, Va.-based Arthritis Foundation. Even more painful is the physical and emotional price paid by the patients — particularly by the 7 million who suffer from rheumatoid arthritis, a potentially life-threatening form that can destroy joints and attack body organs. But medical researchers are searching for better treatments, and some recent results give hope for a cure.

Traditional treatments for rheumatoid arthritis include anti-inflammatory drugs and injection of gold salts. Because the disease is considered an autoimmune disorder, with a patient's own cells triggering tissue destruction, newer treatments like irradiation are aimed at the body's immune system. But these can be toxic to the patient, causing harmful side effects. Searching for drugs that may be effective in nontoxic doses, U.S. researchers earlier this year developed treatment regimens using the anticancer agent methotrexate and an ingestible form of gold.

Now, unpublished data suggest two more methods to stop the disease and help identify its mysterious etiology.

By manipulating the immune system, scientists at the National Institute of Arthritis, Diabetes and Digestive and Kidney Diseases (NIADDK) in Bethesda, Md., hope to halt the erosion of joint tissue. After an NIADDK seminar last week, senior investigator Ronald L. Wilder said he is reluctant to label the immunosuppressive drug cyclosporin A (CSA) anything more than a "potentially promising approach" to treating rheumatoid arthritis, but he admitted his group is excited about research results with the drug at NIADDK's Arthritis and Rheumatism Branch.

In this study, CSA was injected into laboratory rats previously treated with bacterial cell walls that somehow induce arthritis. The drug inhibited the later development of chronic arthritis in many of the animals, Wilder says. Although CSA already has federal approval for use in organ transplant patients to reduce transplant rejection (SN: 10/24/81, p. 263), its use for rheumatoid arthritis would have to wind through a maze of research and regulatory red tape before being clinically tested in humans.

Closer to a place on the pharmacy shelf is a by-product of the genetic engineering industry. Last week officials from Biogen Inc. in Cambridge, Mass., confirmed that results from early human trials using a type of interferon injected into rheumatoid arthritis patients "appear promising." Gamma interferon, produced by Biogen using recombinant DNA techniques (SN: 1/26/80, p. 52), apparently ameliorates patient symptoms by stimulating some component of the immune system.

According to Biogen spokesperson Robert Gottlieb, in two studies recently completed, two-thirds of the 80 patients reported having less pain, swelling and joint tenderness after injection with the protein. Just as significant was the absence of serious side effects from the low doses of interferon given patients. Another study is beginning in Europe, but plans for large-scale testing of the interferon are awaiting consultation with the U.S. Food and Drug Administration. —*D.D. Edwards*

Cystic fibrosis marker

A first step toward identifying the gene responsible for cystic fibrosis was reported last week by two biotechnology companies. The work is expected to lead to a screening test for carriers of the disease and eventually to new treatments.

Cystic fibrosis is the most common lethal genetic disease in the United States. Approximately one in every 2,000 U.S. Caucasian babies is born with the disease; most do not survive beyond their early twenties.

Two genetic markers for cystic fibrosis were described in Salt Lake City by independent research groups at the meeting of the American Society for Human Genetics. A genetic marker is a segment of DNA identified by scientists that is located near an unidentified gene of interest. By following inheritance of the marker within a family, scientists can determine who is likely to inherit the gene. The marker also indicates where within the chromosomes a gene is located.

One genetic marker for cystic fibrosis is located on chromosome 21, reports Integrated Genetics of Framingham, Mass. In collaboration with scientists at the University of Rochester and Yale University, company researchers determined there is a 94 percent probability that the marker and cystic fibrosis gene will be inherited together.

Another genetic marker for cystic fibrosis was reported by scientists at Collaborative Research, Inc., of Lexington, Mass., and at the Hospital for Sick Children in Toronto. The chromosomal location of this genetic marker is still being determined, but it appears to be inherited with the cystic fibrosis gene 85 percent of the time.

The two new cystic fibrosis markers appear to be linked to different genes, says Thomas O. Oesterling of Collaborative Research. He suggests that there are two genes that cause the disease in different families, and that the marker discovered by Collaborative Research is linked to the major gene. The companies are planning an exchange of markers to test on different families. —*J.A. Miller*

FDA okays heart savers

The Food and Drug Administration approved two ways of preventing heart attacks last week, and in turn had its regulations for dealing with new medical devices streamlined by the Secretary of Health and Human Services.

Aspirin and an implantable defibrillator were both okayed as heart attack preventors. The aspirin ruling permits manufacturers to inform physicians that aspirin can reduce the chance of a second heart attack occurring in a person who has already suffered one, and can lessen the chance of heart attack in people who have bouts of heart pain. The FDA based its decision on seven large studies that indicate aspirin's positive effects.

The device that received the FDA nod is a bit more dramatic. Called an implantable defibrillator (SN: 8/9/80, p. 87), it is an internalized rendition of the big metal paddles medical personnel sometimes use to shock a quivering heart into a normal pattern of beats. Developed by Michel Mirowski of Sinai Hospital and Johns Hopkins University in Baltimore, the device has proven itself in more than 700 patients.

These people had hearts that occasionally beat too rapidly (tachycardia) or quiver instead of beating (fibrillation), placing them in danger of sudden death. Each year, 400,000 to 450,000 people in the United States who go into tachycardia or fibrillation cannot be helped by drugs; mortality estimates for this group range from 27 to 66 percent in the first year. About 10,000 to 20,000 of those people are candidates for the device, estimates a spokesperson for its manufacturer, Cardiac Pacemakers, Inc., of St. Paul, Minn.

The implantable defibrillator was evaluated by the FDA because it is a medical device. Secretary of Health and Human Services Margaret M. Heckler at the same time announced streamlined regulations for drug companies submitting devices for approval. The new rules, she said, should expedite approval of other devices. —*J. Silberner*