News
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**Immunology of arthritis will be focus of research at new Simmons Arthritis Center.

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DALLAS--The body's own white blood cells, usually the defense against disease, suddenly collect to inflame and swell membranes that lubricate the joints. The result: arthritis.

It has been estimated that 20,000,000 Americans suffer from arthritis and related diseases. And even though the common types of arthritis are not often fatal, 27,000,000 work days are lost each year to this group of diseases, second only to heart disease as a cause of chronic work loss.

The cause of inflammatory arthritis is not known. But it is linked to immunology, the focal point of research in the newly established Harold Simmons Arthritis Center at The University of Texas Health Science Center at Dallas. The study of immunology is the most important aspect of arthritis research today, says Dr. Morris Ziff, director of the new center. "Almost all the immunological phenomena that we study and teach to medical students occur within rheumatoid joints."

Both rheumatoid arthritis and ankylosing spondylitis (arthritis of the spine) are diseases of chronic inflammation produced by the body's own immune system.

The inflammation occurs in the synovial membrane of the joint. Since the purpose of the synovial membrane is to secrete a fluid to keep the joint moist and lubricated, the membrane is well supplied with small blood vessels, says Ziff. In patients with rheumatoid arthritis and ankylosing spondylitis, the lymphocytes pass through the capillary wall and collect in the joint tissue for reasons not as yet understood. These white blood cells multiply and, in addition, secrete proteins that stimulate other cells to proliferate. This proliferation of cells--lymphocytes, endothelial cells (in the capillary walls) and fibroblasts (in the joint tissues)--causes the joint to swell and become chronically inflamed.

The lymphocytes are also the villains in the development of more acute inflammation, characterized by warmth and sometimes redness. The lymphocytes produce antibodies that collect in the joint fluid and form immune complexes. Another type of white blood cell, the polymorphonuclear leukocyte, is attracted to the area.

(over)

"All these things attack the cartilage and destroy it," says Ziff. "A joint with injured cartilage becomes stiff and very painful because when it moves, particularly, bone rubs on bone."

Much of the stiffness is also due to the collection of fluid in the joint. Finally, as a result of inflammation, the ligaments which surround the joint become scarred and shrink, sometimes causing the joint to become permanently bent. Movement becomes very restricted and painful.

In ankylosing spondylitis, the joints of the spine between vertebrae and the supporting ligaments of the spine are affected. The inflammation causes the ligaments to become calcified. Patients will become very stiff in the back, and some are permanently hunched over. In rheumatoid arthritis usually the hands, shoulders, knees and feet are affected.

"All this results from the stimulation of lymphocytes," says Ziff. "This is all an immune process -- one event after the other in a series of immunological interactions. What causes the lymphocytes to collect? What causes them to become activated and produce proteins to spread inflammation? We don't know.

"Rheumatoid arthritis has an autoimmune feature -- autoantibodies are produced -- as in lupus erythematosus. Ankylosing spondylitis is probably not autoimmune disease.

"We haven't been able to prove an infectious agent in either rheumatoid arthritis or ankylosing spondylitis. However, the spinal inflammation in ankylosing spondylitis may be triggered by diarrhea or infection, particularly diarrheal infection. This is also true in Reiter's syndrome, which is related to ankylosing spondylitis."

The Simmons gift, from a man who himself suffers from ankylosing spondylitis, will enable the arthritis center to expand the previous work of the unit on the inflammatory reactions leading to arthritis.

Research projects to be undertaken or expanded include:

* suppression of chronic joint inflammation by blockade of emigration of lymphocytes from the small blood vessels -- Dr. Morris Ziff

* electron microscopic studies of the rheumatoid synovial membrane -- Dr. Morris Ziff

* mechanisms of cartilage, tendon and ligament injury in rheumatoid arthritis and ankylosing spondylitis -- Dr. Hugo Jasin

* regulatory role of vascular endothelial cells in chronic inflammation -- Dr. Peter Lipsky

* mechanism of action of remission-inducing drugs in rheumatoid arthritis -- Dr. Peter Lipsky

* control of antibody formation in man -- Dr. Peter Lipsky

* control of adjuvant arthritis through induction of tolerance to bacterial products - Dr. Eliot A. Goldings

* investigation of the development of ankylosing spondylitis in young boys -- Dr. Chester Fink

Also engaged in the program will be Dr. Joseph LoSpalluto, professor of Biochemistry, and Dr. Stanley B. Cohen, clinical assistant professor of Internal Medicine. Additional faculty will be recruited for other planned research projects.

Ziff has previously directed the Rheumatic Diseases Unit of the Department of Internal Medicine. He is the health science center's first Ashbel Smith Professor, the highest honor The University of Texas can bestow on a faculty member. He is the Morris Ziff Professor of Rheumatology, occupying a professorship established in his honor; director of the Arthritis Clinic at Parkland Memorial Hospital and the recipient of a Research Career Award of the U.S. Public Health Service. He is a world authority on rheumatic diseases.

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