

MYCOPLASMA ARTHRITIS ANTIBODIES ISRAEL JOINTS INFLAMMATION CYTOKINES

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Description: Evidence implicates common Mycoplasma bacteria in the triggering or exacerbating of rheumatoid arthritis. Journal of Rheumatology 27: 2747 (Dec 2000)

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COMMON BACTERIUM IMPLICATED IN THE TRIGGERING OF RHEUMATOID ARTHRITIS Beer-Sheva, March 19, 2001 - Researchers at Ben-Gurion University of the Negev have shown that a well-known bacterium of the Mycoplasma family - commonly found in the human throat - may be involved in the triggering or exacerbation of rheumatoid arthritis (RA).

The team found that fluids from the inflamed, arthritic joints of many patients contained the specific DNA characteristic of Mycoplasma fermentans, as well as antibodies against this organism. Their studies also indicate that mycoplasmic membrane proteins capable of triggering inflammation may also be present. Collaborating in this investigation are Prof. Shulamith Horowitz and research assistant Bela Evinson at BGU's Department of Microbiology and Immunology, and Prof. Jacob Horowitz and Dr. Abraham Borer of the Department of Medicine at the Joyce and Irving Goldman Medical School. Prof. Jacob Horowitz also serves as head of Department of Medicine A at Soroka University Medical Center. A report on their work appears in a recent issue of the Canadian publication Journal of Rheumatology.

Rheumatoid arthritis, notes husband-wife team Jacob and Shulamith Horowitz, is an autoimmune disease, in which the body's immune system is triggered to attack normal body tissues. Determining the ultimate cause of RA therefore requires the identification of an agent in arthritic joints that interacts with the immune system.

Because Mycoplasmas definitely cause arthritis in animals, doctors have suspected since the early '50s that a Mycoplasma found in humans might be involved in the disease in man. While a number of researchers over the decades have claimed to isolate live Mycoplasma bacteria from the joint fluid of RA patients, others who attempted to repeat their findings failed to do so.

However with the development of advanced DNA analysis techniques.

Identification of traces of bacterial genomes has become easier to ascertain. Thus, British and French scientists have recently shown that M. fermentans DNA is present in the joint (synovial) fluid of many RA patients, findings confirmed by the studies at BGU. In their initial test group of three-dozen RA patients, the BGU scientists found that M.fermentans DNA was present in some 20 percent of the arthritic joints examined. None of 57 patients with other forms of arthritis had this DNA in their joints.

Of critical significance was the additional discovery that half of the RA patients studied, even those with no detectable DNA, had abnormally large quantities of antibodies against M. fermentans in their arthritic joints. Because these patients had the same low quantities of anti-M. fermentans antibodies in their blood

serum as do healthy individuals; the BGU team believes that the antibodies they found in the synovial fluid were produced there in response to Mycoplasma that had entered the joint. In 57 patients with other varieties of arthritis, the anti-M. fermentans antibody level in their joints was negligible, even lower than that in their serum.

The BGU scientists also identified the mycoplasmic proteins recognized by the antibodies. These are specific membrane components known to activate the production of immune system factors, such as TNF-alpha, which are inducers of inflammation. This finding indicates a further mechanism that may contribute to the appearance of RA because of M. fermentans entering the joint.

"Our studies suggest," says Jacob Horowitz, a Rheumatology specialist "That Mycoplasmas in the joint may stimulate the immune system to produce antibodies and protein factors known as cytokines, several of which produce local inflammation and tissue damage. There are clearly different agents leading to RA. Among them, M. fermentans may play an important role. This finding adds to the growing list of organisms that have long been considered benign residents of the human body but that modern research indicates may be involved in disease."

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