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## Autoimmune Diseases Caused by Mycoplasmas

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From the book Why Arthritis?

Searching for the Cause and the Cure of Rheumatoid Disease Submitted by The Arthritis Trust of America, 7376 Walker Road, Fairview, TN 37062-8141; http://www.arthritistrust.org. The primary function of the immune system is to distinguish foreign invaders, such as microbes, from the components of host tissues and facilitate in their elimination. Even though there are experimental animal models of autoimmunity, reacting against their own tissues, there is no general unifying theory to explain how the autoimmune processes like rheumatoid arthritis (RA) and Lupus (SLE) get started in humans and progresses in chronic and cyclic patterns. It would seem that autoimmunity and autoimmune diseases have multiple origins including environmental factors such as microbial infections and genetics. Current investigations of autoimmune diseases have focused on the concept of molecular mimicry by viral, bacterial, mycoplasma or other microbial antigens having antigenic epitopes or structure similar to and reactive with both the host proteins and the foreign proteins. In the laboratory mycoplasmas have been found to mimic their culture media contributing to their variable physical and chemical properties. They also can detach membrane components from erythrocytes and leukocytes resulting in autoantibody production and disease.

Now for the first time a natural mechanism that could apply to multiple Autoimmune (AI) Diseases has been demonstrated in mycoplasma infected rabbits. Mycoplasma are ubiquitous with unique properties, making them widely suspected as a potential cause of hypersensitivity in the multifaceted AI diseases. Being the

smallest free-living microorganisms limits their metabolic action. Mycoplasmas are highly pleomorphic with a lipoprotein membrane controlling their permeability and adherence to vascular and neural tissue membranes. Mycoplasma's fastidious growth requirements are provided by their saprophytic activity and available pre-formed macro molecules in a cell-free tissue digest broth. These include basic peptides, cholesterol and fatty acids (lipoglobulins), nucleotides (basic). Mycoplasma affinity for mucoproteins is indicated by their frequent colonization of the nasopharyngeal (NP) and the urogenital (UG) tracts, Finding mycoplasma and ureaplasma strains in the central nervous system would indicate their potential role in the neurologic disorders. Of special consideration is finding the greatest mycoplasma infectivity in females (4:1) reflecting their prevalence in most autoimmune (AI) diseases and thus the basis for the Gender Gap. Also the organ and tissue preference of the several human mycoplasma strains could contribute to a variety of AI diseases.

Mycoplasmas cultured in serum enriched broth specifically incorporate basic proteins such as IgG gamma globulin from the serum. The molecular attachment alters the basic protein structure making them foreign and autoantigenic to the host. When attached to the mycoplasma lipoprotein membrane the cells act as both carrier and adjuvant for the altered basic tissue proteins now autoantigenic to the host. The altered IgG causes the production of autoantibodies characteristic of the so-called rheumatoid factor (RF) and other autoantibodies. Rheumatoid arthritis developing after *M.pneumoniae* infection is host dependent producing immune complex (IC) with IgG autoantibodies.

To test this autoirnmune mechanism, in the absence of human tissue, *M. pneumoniae* was cultured in a rabbit digest broth enriched with rabbit serum. The cultured and washed mycoplasma cells were used to immunize rabbits. The resulting rabbit antisera was positive to both *M. pneumoniae* and IgG a rabbit autoantibody to its altered self. Although not required for growth the mycoplasma incorporated various amounts of the basic IgG protein from the serum enriched culture. Injections of rabbits with their own native serum does not elicit autoantibodies to the native IgG unless conformed and/or given with an adjuvant carrier such as with the available mycoplasma lipoprotein membrane. The production of an experimental autoimmune disease requires the host antigen, such as basic myelin protein, to be given with some adjuvant.

Rheumatoid Arthritis (RA), one of the more prevalent autoimmune diseases, has long-been suspected of being caused by some microbial agent and immune complex. Investigators have directed their research towards typical microbial infections and not the host's hypersensitivity response mechanism of immune complex (IC) diseases as currently suspected. The microbial antigens in the immune complex should be identified to determine and eliminate their al-

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lergic cause. The function of immune complex (Antigen+Antibody) diseases as currently suspected are those in which the deposition of circulating IC's initiate injury and inflammation in multiple tissues from the activation of the proteolytic complement system. Many of the AI diseases are associated with an immune complex and diffuse connective tissue disease symptoms.

Answers to autoimmunity will come from conclusive tests when mycoplasmas are cultured in human tissues: erythrocytes, leucocytes, myelin, pancreas, brain and other tissues associated with specific AI diseases. The AI diseases result when the host's immune system, meant to defend against bacteria, viruses, and other foreign substances, produces autoantibodies against specific basic proteins in normal host tissues, cells and organs. Mycoplasmas are now recognized as a cause of rheumatoid arthritis. Not all people with mycoplasma infections develop rheumatoid arthritis indicating genetics and other cofactors are involved.

As with most autoimmune disorders and allergic hypersensitivity dietary changes and detoxification are usually helpful especially under experienced medical supervision. Mycoplasma hypersensitivity as a cause of arthritis has been demonstrated in rabbits previously immunized against a strain of mycoplasma and then challenged with the autologous strain. The safe and effective antibiotic treatment of RA, one of the more prevalent autoimmune diseases, should also be tested in the other AI diseases. The tetracyclines multiple action; antioxidant, antiinflammatory, immunosuppressant, chelation in suitable dosage could provide less toxic and effective therapy for the complex IC diseases.