From the study of our cases we think that the process does not seem to be a coincidence of both diseases but rather of the action of the X factor or factors of RA upon the domain of AS which would result in a variant of associate AS. This would be the true 'rheumatoid spondyloarthrits'. Its rate of frequency, since the predominance of AS marked by HL-A 27 is 6% in our country, would be somewhat higher than that reported by Fallet, if our hypothesis proves to be correct. Evidently it would not reach the ratio of 1/1600 which is the ratio to be expected if every factor of RA, by incidence of the HL-A 27, would cause this mixed picture.

Ankylosing spondylitis and epidemic Reiter's syndrome: genetics and environment. A. Calin and J. F. Fries

The greatly increased frequency of HL-A 27 in both ankylosing spondylitis (AS) and Reiter's syndrome (RS) has been well recognized. Both environmental and genetic factors appear to contribute. To elucidate this interplay, two parallel studies were performed.

(1) Evaluation of 78 presumed 'healthy' HL-A 27 positive blood donors (30 males, 48 females) yielded 14 cases (20% and 16.7% respectively) of definite AS, the New York criteria being used as a minimum standard. 122 race, sex, and age-matched HL-A 27 negative controls failed to yield a single case. Using published disease frequencies, the expected prevalence of AS among HL-A 27 positive individuals should be 2% and 0.2% for males and females, respectively. In contrast, this study strongly suggests that at least 20% of subjects with HL-A 27 are likely to develop AS, and that the frequency is comparable in both sexes. (2) An attempt was made to locate and HL-A type the 9 men who developed RS after the 1963 epidemic of proven shigellosis in 602 individuals on a U.S. naval ship. To date, 5 have been traced and 4 of these are HL-A 27. Given the racial make-up of the U.S. navy, about 36 of those who developed dysentery would have been HL-A 27 positive, suggesting that between 12% and 25% of those at genetic risk developed RS after this single environmental insult.

It thus appears that in both AS and epidemic RS some 12% to 25% of those with HL-A 27 may develop the disease. In the case of RS this may follow shigellosis; in AS some unknown environmental insult may be implicated; in both there may be interplay with other unidentified genetic factors. The presence of typical clinical findings in the occasional HL-A 27 negative individual suggests either such additional operative genetic factors or 'environmental over-ride'. Preliminary data showing that there is an increased proportion of HL-A 27 negative spondylitic patients with shigella dysentery or inflammatory bowel disease compared with idiopathic AS subjects, indicates that a sufficient environmental insult can result in the expression of disease even in a genetically 'nonsusceptible' individual.

Histocompatibility antigens in polyarthritis in the hand. J. Muñoz Gómez, M. A. Brancos Cunilla, and G. Ercilla Gonzáles

In 1958 Kellgren and Lawrence described a pattern of arthrosis in the hand, appearing mostly in women. It has been shown that when there are more than five arthritic joints in the hand, there is a significant correlation (P < 0.01) with the existence of a pseudopondyloysisis secondary to arthrosis in the posterior interapophyseal joints. This arthritic pattern seems to be genetically determined. For this reason we attempted to study the histocompatibility antigens in these cases. There seems to be no significant statistical differences when comparing the frequency rates of the diverse antigens in the arthritic group studied and in the controls.


HL-A frequencies in less common arthropathies. A. Robitaille, C. Cockburn, D. C. O. James, and B. M. Ansell (MRC Rheumatism Research Unit, Taplow, and Tissue Typing Laboratories, Westminster Hospital)

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Study of diagnostic importance of a group of immunological parameters in 212 rheumatic patients, with special reference to rheumatoid arthritis. E. Noguera Hernando, A. Larrea Gayarre, E. Fernández Cruz, M. Kreisler, and A. Bootello

(1) The most frequent associations, referring to immunological alterations, are rheumatoid factor, ANA, and polyclonal increase of immunoglobulins, all of which are basically visible in RA. (2) The increase in immunoglobulin levels was observed in a great number of rheumatic processes, and does not seem to be specific, constituting an index of the disease activity. (3) With the exception of RA, rheumatoid factor appeared in a low proportion in other processes, 1 out of 5 in DE, 1 out of 2 in scleroderma, and in 4 out of 14 in nonspecific polyarthritis, in our series. (4) ANA was present in 39.5% of seropositive RA, among other processes. Titration and staining pattern are of main importance for the diagnosis, as it allows differentiation of two entities which at certain evolutive stages may display great clinical similarity, as with DE and RA. (5) Low values of serum complement were observed in RA (13.9%) and in other processes of the so-called autoimmune diseases such as SLE and scleroderma. (6) The presence of AAML is of specific diagnostic importance as in the case of some chronic hepatopathies, its incidence being low in RA (10%). AAML showed more specificity for primary biliary cirrhosis. We could only find it in one case of seropositive RA (1.6%). Evidence of both in RS would reflect the existence of one more autoimmunity phenomenon.


Thirty-six patients with ankylosing spondylitis have been treated with a daily dose of 500 mg naproxen for 1–30 months. Diagnosis was based on radiographic evidence of sacroiliitis and characteristic symptoms started before the age of 30. Patients with sacroiliitis associated with psoriasis, ulcerative colitis, regional ileitis, Reiter's and Behçet's disease were excluded. At the end of the first month of treatment 35 of 36 patients assessed naproxen as being equal to or better than previous therapy. At 12 months, 3 had gone into remission and
Study of diagnostic importance of a group of immunological parameters in 212 rheumatic patients, with special reference to rheumatoid arthritis [Proceedings].

E Noguera Hernando, A Larrea Gayarre, E Fernández Cruz, M Kreisler and A Bootello

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