Rheumatic diseases in blacks, whites and Asians—some comments

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The aetiology of rheumatic diseases is unknown but in many of them it appears that there are one or more trigger factors which induce inflammatory rheumatic disease against the genetic background of the patient. Both trigger factors and genetic background may differ in different parts of the world. Ethnic differences can therefore modulate rheumatic diseases in many ways which will appear as differences in disease prevalence as well as in disease expression. This appears to be true of all rheumatic diseases which have been examined in detail, from the common ones to the rarer forms of connective tissue disease.

Ankylosing spondylitis has the strongest genetic link of any disease in man. In Caucasians, virtually all cases show the HLA B27 antigen. The incidence of this HLA antigen is diminished in the population in both negroes and Japanese. Not surprisingly, the incidence of ankylosing spondylitis is lower in both of these populations. However, this is not necessarily simple cause and effect as the prevalence of B27 positive cases of ankylosing spondylitis is also lower in Japanese, and strikingly so in South African negroes. Thus, it appears that there are ethnic as well as genetic differences.

The prevalence of rheumatoid arthritis is greater in the United Kingdom and Northern Europe than it is in the Caribbean. This is not simply a north/south difference since in Caucasians in New Zealand, the prevalence of rheumatoid arthritis (RA) appears even higher and is markedly greater than it is in the resident Maori population. Differences between 2 populations are apparent in South Africa where rural negroes have been shown to have a much lower prevalence of RA than have urbanized blacks. The latter show a prevalence equivalent to that of the white urban population. This data has been interpreted as suggesting an infective trigger factor for RA. However, there is a genetic linkage as well. In the United Kingdom, RA shows a significant linkage with the HLA DR4 antigen. In Asians in the U.K.,

the linkage is different—apparently with the DR1 antigen. There are also racial differences in disease expression. Caucasians in the U.K. with RA show nodules in approximately 30%. Such subcutaneous nodules are extremely rare in both India and East Africa. A more severe complication of RA—Felty's syndrome—is virtually unknown in black patients with RA, in both the U.S.A. and South Africa.

Systemic lupus erythematosus (SLE) is another disease with marked geographical differences in prevalence. It appears to be considerably commoner in the U.S.A. than it is in the U.K. or Northern Europe. Within the U.S.A., the prevalence is greater in the black population than it is in the white. Similar increases in prevalence are seen in Chinese both on the main land and in Malaysia. However, SLE is rare in blacks in Southern Africa. Disease expression also differs in the black U.S.A. population which appears to have a more severe disease with a higher mortality rate than white patients with SLE.

In contrast, Behçet's disease is rare in the U.S.A. despite the considerable Japanese American population and a very high incidence of the disease in Japan. In the latter country, there are differences between the north and south. The pockets of very high prevalence are consistent with an infective focus.

Commoner diseases like osteoarthritis also show marked differences in prevalence in different ethnic groups and illustrate the influence of the sex as well. This is well documented, for example, in osteoarthritis of the hip in white and black populations in South Africa. The white population shows both a higher prevalence and a female preponderance which is reversed in the black population.

It is clear that ethnic differences in rheumatic diseases reflect genetic, infectious, social and other factors. This would be a rich field for further epidemiological study which would help unravel the aetiology of these common disabling conditions.