

MNS GENOTYPES IN ANKYLOSING SPONDYLITIS

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Sharon, Weinberg and Hussein (1985) reported two groups of Arab patients with ankylosing spondylitis (AS), both of which had a very high incidence of homozygous MM (92% and 100%). They were following up an investigation of the blood groups of white Americans with various rheumatic diseases carried out by Cohen et al. (1963), who found a slight negative correlation between these diseases and the presence of the N antigen in the MNSs system. Kornstad, Kornstad and Guldborg (1968) also tested this association in 200 Norwegian patients with ankylosing spondylitis, but found the same distribution of M, MN and N blood types as in healthy controls. We have tested the distribution of the MNSs-gene complex in 30 Danish patients known to suffer from AS.

Patients and results. All 26 men and 4 women had definite ankylosing spondylitis on the New York criteria.

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Their average age was 46.5 years, with a mean duration of disease of 15.7 years. The HLA-B27 antigen was present in 29 of the 30 patients. They were unrelated apart from one father and son. The incidence of the nine

Table I. Distribution of MNSs genotypes

MNSs genotypes	Reported in 1000 blood donors*	Observed in 30 patients with AS
MS/MS	57	1
MS/Ms	140	3
Ms/Ms	101	1
	298	5
MS/NS	39	1
MS/Ns or Ms/NS	224	8
Ms.Ns	226	9
	489	18
NS/NS	3	0
NS/Ns	54	2
Ns/Ns	156	5
	213	7

* Cleghorn 1960
 $0.80 < p < 0.90 (G^2 = 3.60; f = 8)$

possible combinations of the MN and Ss loci did not differ from that accepted as normal (Table I).

Discussion. Several investigators have suggested that ankylosing spondylitis is transmitted by an autosomal dominant gene, and it has been assumed that such a gene could be closely linked to HLA-B27. However, recent studies (van der Linden and Khan 1984) indicate that the prevalence of AS among B27-positive adults in the general population is only 1% to 10%.

Calin et al. (1983) suggest that there may be genetic differences between B27-positive patients with AS and B27-positive healthy control subjects. If there was a second "disease gene" close to the MNSs genes in AS, this would mean that there would have to be abnormal genes on two separate chromosomes (Numbers 2 and 6).

The patients reported by Sharon et al. (1985) were either highly consanguineous (20 patients from an Arab village) or from a small group (13 unrelated patients with AS). The high incidence of homozygous MM which they reported was not confirmed by our study. We found a normal distribution of the MN bloodtypes even in the

nine MNSs genotypes, as Kornstad et al. (1968) reported for the bloodtypes of 200 AS patients in Norway. We conclude that at least in Scandinavian patients with ankylosing spondylitis, the MNSs bloodtypes are distributed as in English blood donors (Cleghorn 1960), and that the determination of the MN bloodtype is not of use in screening for ankylosing spondylitis.

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