

Letters to the Editor

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Psoriasis Linkage in the HLA Region

To the Editor:

We were pleased to see the article by Jenisch et al. (1998), providing additional evidence for linkage between (familial) psoriasis and human leukocyte antigen (HLA). This confirms, in partially new data, what we (Leder et al. 1998) and others (Nair et al. 1997; Trembath et al. 1997) have already demonstrated—that there is a psoriasis susceptibility locus (PSORS1) on chromosome 6, closely linked to HLA. Unlike genome scans (Nair et al. 1997; Trembath et al. 1997), the analyses of Jenisch et al. (1998) and our own analyses show that support for linkage is enhanced by use of haplotypes to account for HLA associations in linkage analysis.

However, we note that Jenisch et al. (1998) still hedge on the finding of linkage. Readers should be aware that our analyses of previously published data (Leder et al. 1998) already support tight linkage beyond any reasonable doubt (LOD score of 23.7 under the assumption of dominant inheritance).

The assertion by Jenisch et al. (1998) that “Previous studies based on limited numbers of families found only weak evidence . . . for linkage to the HLA region” is also belied by our analysis of the previously published data. For example, the 31 families reported by Civatte et al. 1977 (cited in Jenisch et al. 1998) actually yield a LOD score of 6.29 between psoriasis and HLA-B, and family data from Sun et al. 1987 (not cited by Jenisch et al. 1998) yield a LOD score of 4.29. When the Civatte and Sun data are combined with data from other families reported previously (including all of those cited in Jenisch et al. 1998), evidence for linkage to psoriasis in the HLA region is overwhelming (summarized in table 3 of Leder et al. 1998). Finally, we suggest that all investigators of HLA and psoriasis should use the official nomenclature for this psoriasis-susceptibility locus, PSORS1 (Leder et al. 1998; White et al. 1998; see also White et al. 1999). Now that HLA linkage in psoriasis is conclusively settled, it remains both to identify the HLA-linked psoriasis gene(s) and to solidify evidence for

the putative role(s) of non-HLA genes in the genetic etiology of psoriasis.

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