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The role of HLA-B27 in spondyloarthritis.

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Source

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Abstract

This article summarizes the proceedings of a one-day international workshop held in July 2009 on the role of HLA-B27 in the pathogenesis of ankylosing spondylitis (AS) and related disorders. HLA-B27 is found in about 90% of patients with AS, with an odds ratio of about 100, but the mechanism underlying this association is not known. There are currently 3 major mechanistic hypotheses for this association: (1) T cell recognition of one or more B27 presented peptides; (2) B27 heavy-chain misfolding that induces an unfolded protein response; and (3) innate immune recognition of cell-surface expressed B27 heavy-chain dimers. None of these hypotheses accounts for the tissue specificity of the inflammation characteristic of AS. These hypotheses were discussed in the context of known epidemiologic, biochemical, structural, and immunologic differences among HLA-B27 subtypes; data from the HLA-B27 transgenic rat model of spondyloarthritis; the growing list of other genes that have been found to be associated with AS; and other data on the pathogenesis of spondyloarthritis. Proposed directions for future research include expanded efforts to define similarities and differences among the B27 subtypes; further development of animal models; identifying the interactions of B27 with the products of other genes associated with AS; and continued investigation into the pathogenesis of spondyloarthritis.

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