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CONCISE REPORT

Identification of the *PTPN22* functional variant R620W as susceptibility genetic factor for giant cell arteritis

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ABSTRACT

Objective To analyse the role of the *PTPN22* and *CSK* genes, previously associated with autoimmunity, in the predisposition and clinical phenotypes of giant cell arteritis (GCA).

Methods Our study population was composed of 911 patients diagnosed with biopsy-proven GCA and 8136 unaffected controls from a Spanish discovery cohort and three additional independent replication cohorts from Germany, Norway and the UK. Two functional *PTPN22*

Although the aetiology of GCA remains unclear, it is well known that innate and adaptive immune responses are involved in its pathogenesis. Several lines of evidence indicate that this vasculitis is a T cell-mediated disease with both Th17 and Th1 cells contributing to inflammation. While Th1 response is associated with chronically persistent vascular lesions, Th17 immunity appears to be more important for acute manifestations, both systemically and in the blood vessels.[3 4]