
The therapeutic potential of the HLA-G molecule

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The HLA-G gene has several peculiarities that distinguish it from the class I HLA genes. Its singular molecular structure provides a limited antigen presentation and allows the modulation of immune system cells (NK and lymphomononuclear cells), and the result is that HLA-G acts like a tolerogenic and immunosuppressive molecule.¹ Its major physiological function lies in participating in the tolerance between maternal and fetal cells in the placental interface.² HLA-G is implicated in the etiopathogenesis of several human diseases, such as chronic viral infections (HIV, cytomegalovirus, hepatitis C and hepatitis B), rejection to the transplantation of solid organs (kidney, heart), neoplasias, and autoimmune diseases (rheumatoid arthritis, systemic lupus erythematosus, systemic sclerosis, multiple sclerosis, and type I diabetes mellitus).³ In this issue, Brenol CV et al.⁴ reviewed the role of HLA-G in several autoimmune rheumatic

diseases (rheumatoid arthritis, systemic lupus erythematosus, systemic sclerosis, Behçet's disease, juvenile idiopathic arthritis, Kawasaki disease, inflammatory myopathies, and sarcoidosis). The authors provide a general panel of the molecular structure of HLA-G, its major functions, and how the study of the polymorphism of its alleles associates with the occurrence of autoimmune rheumatic diseases. Because HLA-G is an immunosuppressive and tolerance-inducing molecule, the possibility of its future use in the treatment of autoimmune diseases, including the rheumatic diseases, has been considered.⁵

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