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. Its major physiological function lies in participating in the tolerance between maternal and fetal cells in the placental interface. 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, 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.

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