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Sommario/riassunto

The non-classical HLA class I molecule HLA-G is different from classical HLA class I molecules because of the low polymorphism in the coding region, the fact that HLA-G primary transcript is alternatively spliced in seven isoforms, and the inhibitory action on immune cells. Although HLA-G is low polymorphic, variants in both promoter and 3' un-translated region (UTR) of HLA-G locus regulate its expression. In healthy conditions, a basal level of HLA-G gene transcription is observed in most cells and tissues; however, translation into HLA-G protein is restricted to trophoblasts in the placenta, where it participates in promoting tolerance at the fetal-maternal interface. HLA-G is also expressed by thymic epitelial, cornea, mesenchymal stem cells, nail matrix, pancreatic beta cells, erythroid, and endothelial precursors. HLA-G can be neo-expressed in adult tissues in pathological conditions, and its expression has been documented autoimmune disorders, viral infections, and cancer. In the latter setting de novo HLA-G expression is associated with the capability of tumor cells to evade the immune control. In the last decade it has become evident that HLA-G expression on T cells and antigenpresenting cells confers to these cells tolerogenic properties. This Research Topic focused on i) summarizing updated clinical and immunological evidences that HLA-G expression is associate with beneficial or detrimental tolerance, ii) gathering new insights into the mechanisms governing the expression of HLA-G in healthy and pathological

conditions, such as pre-eclampsia, and iii) examining the mechanisms underlying HLA-G mediated tolerance.