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# Anti-HLA-G Reference Antibody (Roche patent anti-HLA-G)

Catalog # APR11365

## Product Information

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|---|---|
| **Application** | Functional assay, Kinetics (SPR), Kinetics (BLI), FACS, E, FTA |
| **Primary Accession** | P17693 |
| **Reactivity** | Human |
| **Clonality** | Monoclonal |
| **Isotype** | IgG1 |
| **Calculated MW** | 38224 |

## Additional Information

| | |
|---|---|
| **Target/Specificity** | HLA-G |
| **Expression system** | CHO |

## Protein Information

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|---|---|
| **Name** | HLA-G {ECO:0000303\|PubMed:1570318, ECO:0000312\|HGNC:HGNC:4964} |
| **Function** | [Isoform 1]: Non-classical major histocompatibility class Ib molecule involved in immune regulatory processes at the maternal-fetal interface (PubMed:19304799, PubMed:23184984, PubMed:29262349). In complex with B2M/beta-2 microglobulin binds a limited repertoire of nonamer self-peptides derived from intracellular proteins including histones and ribosomal proteins (PubMed:7584149, PubMed:8805247). Peptide-bound HLA-G-B2M complex acts as a ligand for inhibitory/activating KIR2DL4, LILRB1 and LILRB2 receptors on uterine immune cells to promote fetal development while maintaining maternal- fetal tolerance (PubMed:16366734, PubMed:19304799, PubMed:20448110, PubMed:23184984, PubMed:27859042, PubMed:29262349). Upon interaction with KIR2DL4 and LILRB1 receptors on decidual NK cells, it triggers NK cell senescence-associated secretory phenotype as a molecular switch to promote vascular remodeling and fetal growth in early pregnancy (PubMed:16366734, PubMed:19304799, PubMed:23184984, PubMed:29262349). Through interaction with KIR2DL4 receptor on decidual macrophages induces pro-inflammatory cytokine production mainly associated with tissue remodeling (PubMed:19304799). Through interaction with LILRB2 receptor triggers differentiation of type 1 regulatory T cells and myeloid-derived suppressor cells, both of which actively maintain maternal-fetal tolerance (PubMed:20448110, PubMed:27859042). May play a role in balancing tolerance and antiviral-immunity at maternal-fetal interface by keeping in check the effector functions of NK, CD8+ T cells and B cells (PubMed:10190900, PubMed:11290782, PubMed:24453251). Reprograms B cells toward an immune suppressive |

phenotype via LILRB1 (PubMed:24453251). May induce immune activation/suppression via intercellular membrane transfer (trogocytosis), likely enabling interaction with KIR2DL4, which resides mostly in endosomes (PubMed:20179272, PubMed:26460007). Through interaction with the inhibitory receptor CD160 on endothelial cells may control angiogenesis in immune privileged sites (PubMed:16809620).

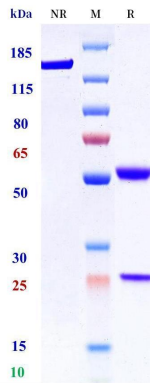
**Cellular Location**

[Isoform 1]: Cell membrane; Single-pass type I membrane protein. Endoplasmic reticulum membrane. Early endosome membrane [Isoform 2]: Cell membrane; Single-pass type I membrane protein [Isoform 4]: Cell membrane; Single-pass type I membrane protein [Isoform 6]: Secreted Cell projection, filopodium membrane. Note=HLA-G trogocytosis from extravillous trophoblast's filopodia occurs in the majority of decidual NK cells.

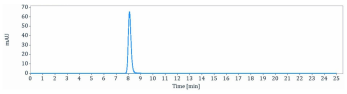
**Tissue Location**

Expressed in adult eye (PubMed:1570318). Expressed in immune cell subsets including monocytes, myeloid and plasmacytoid dendritic cells and regulatory T cells (Tr1)(at protein level) (PubMed:20448110). Secreted by follicular dendritic cell and follicular helper T cells (PubMed:24453251) [Isoform 7]: Expressed in placenta, amniotic membrane, skin, cord blood and peripheral blood mononuclear cells

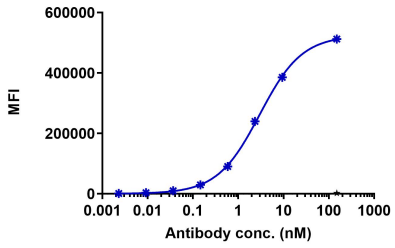
## Images




Anti-HLA-G Reference Antibody (Roche patent anti-HLA-G) on SDS-PAGE under reducing (R) condition. The purity of the protein is greater than 95%.



The purity of Anti-HLA-G Reference Antibody (Roche patent anti-HLA-G) is 100%, determined by SEC-HPLC.




HLA-G/A375 cell line were stained with Roche patent anti-HLA-G and negative control protein respectively, washed and then followed by PE and analyzed with FACS, with the EC50 is 2.971 nM.

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