HLA-G (Major Histocompatibility Complex, class I, G) Antibody

Mouse Monoclonal Antibody [Clone HLAG/7856]

Catalog No	Format	Size
3135-MSM13-P0	Purified Ab with BSA and Azide at 200ug/ml	20 ug
3135-MSM13-P1	Purified Ab with BSA and Azide at 200ug/ml	100 ug
3135-MSM13-P1ABX	Purified Ab WITHOUT BSA or Azide at 1.0mg/ml	100 ug

Applications	Tested Dillution	Note
Immunohistochemistry (IHC)	1-2ug/ml	30 min at RT. Staining of formalin-fixed tissues requires heating tissue sections in 10mM Tris with 1mM EDTA, pH 9.0, for 45 min at 95°C followed by cooling at RT for 20 minutes

Product Details

Clone	HLAG/7856
Immunogen	Recombinant human HLA-G protein
Host	Mouse
Clonality	Monoclonal
Isotype / Light Chain	IgG1 / Kappa
Mol. Weight of Antigen	38.22kDa
Cellular Localization	Cell membrane, Cell projection, Early endosome, Early endosome membrane, Endoplasmic reticulum membrane, Filopodium membrane, Secreted
Species Reactivity	Human
Positive Control	Expressed in adult eye (PubMed:1570318)

**Optimal dilution for a specific application should be determined.*

Product Images for HLA-G (Major Histocompatibility Complex, class I, G) Antibody

Specificity & Comments

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)., Likely does not bind B2M and presents peptides. Negatively regulates NK cell- and CD8+ T cell-mediated cytotoxicity (PubMed:11290782)., 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: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). Reprograms B cells toward an immune suppressive phenotype via LILRB1 (PubMed:24453251)., Likely does not bind B2M and presents peptides.

Supplied As

200ug/ml of Ab purified from Bioreactor Concentrate by Protein G. Prepared in 10mM PBS with 0.05% BSA & 0.05% azide. Also available WITHOUT BSA & azide at 1.0mg/ml.

Storage and Stability

Antibody with azide - store at 2 to 8 °C. Antibody without azide - store at -20 to -80 °C. Antibody is stable for 24 months. Non-hazardous. No MSDS required.

Limitations and Warranty