# Data Sheet (Cat.No.TMAH-00556)

TargetMol

## Anti-HLA-G Antibody (2G265)

### Product Details

| | |
|---|---|
| Ig Type: | Rabbit IgG |
| Reactivity: | Human |
| Conjugation: | Unconjugated |
| Clone: | 2G265 |
| Purification: | Affinity-chromatography |

### Applications

| | |
|---|---|
| Verified Activity: | Overlay Peak curve showing U937 cells stained with TMAH-00556 (red line) at 1:50. The cells were fixed in 4% formaldehyde and permeated by 0.2% TritonX-100. Then 10% normal goat serum to block non-specific protein-protein interactions followed by the antibody (1μg/1*10^6 cells) for 45min at 4°C. The secondary antibody used was FITC-conjugated Goat Anti-rabbit IgG (H+L) at 1:200 dilution for 35min at 4°C.Control antibody (green line) was rabbit IgG (1μg/1*10^6 cells) used under the same conditions. Acquisition of >10,000 events was performed. |
| Application: | ELISA, FCM |
| Recommended | FCM:1:50-1:200. |

### Properties

| | |
|---|---|
| Stability & Storage: | Store at -20°C or -80°C for 12 months. Avoid repeated freeze-thaw cycles. |
| Shipping: | Shipping with blue ice. |

### Antigen Details

| | |
|---|---|
| Immunogen: | A synthetic peptide: Human HLA-G |
| Antigen Species: | Human |
| Gene ID: | 3135 |
| Uniprot ID: | P17693 |
| Synonyms: | HLA G antigen;b2 microglobulin;MHC Class I Antigen G;MHC-G;HLA-G;HLA G;HLAG;MHC class Ib antigen;sHLA-G |
| Biology Area: | Immunology |

### Research Background

Non-classical major histocompatibility class Ib molecule involved in immune regulatory processes at the maternal-fetal interface. In complex with B2M/beta-2 microglobulin binds a limited repertoire of nonamer self-peptides derived from intracellular proteins including histones and ribosomal proteins. 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. 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. Through interaction with KIR2DL4 receptor on decidual macrophages induces proinflammatory cytokine production mainly associated with tissue remodeling.

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. 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. Reprograms B cells toward an immune suppressive phenotype via LILRB1. May induce immune activation/suppression via intercellular membrane transfer (trogocytosis), likely enabling interaction with KIR2DL4, which resides mostly in endosomes. Through interaction with the inhibitory receptor CD160 on endothelial cells may control angiogenesis in immune privileged sites. Likely does not bind B2M and presents peptides. Negatively regulates NK cell- and CD8+ T cell-mediated cytotoxicity. Likely does not bind B2M and presents peptides. Negatively regulates NK cell- and CD8+ T cell-mediated cytotoxicity. Likely does not bind B2M and presents peptides. Negatively regulates NK cell- and CD8+ T cell-mediated cytotoxicity. Non-classical major histocompatibility class Ib molecule involved in immune regulatory processes at the maternal-fetal interface. In complex with B2M/beta-2 microglobulin binds a limited repertoire of nonamer self-peptides derived from intracellular proteins including histones and ribosomal proteins. 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. 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. Through interaction with KIR2DL4 receptor on decidual macrophages induces proinflammatory cytokine production mainly associated with tissue remodeling. 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. Reprograms B cells toward an immune suppressive phenotype via LILRB1. Likely does not bind B2M and presents peptides. Likely does not bind B2M and presents peptides.



Tel:781-999-4286 E_mail:info@targetmol.com Address:34 Washington Street,Wellesley Hills,MA 02481