

## Anti-human HLA-G Antibody

<b>Catalog Number:</b>	112601, 112602
<b>Size:</b>	25 ug, 100 ug
<b>Target Name:</b>	HLA-G, Human Leukocyte Antigen-G
<b>Regulatory Status:</b>	RUO

### PRODUCT DETAILS

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<b>Clone:</b>	HLAGAM1
<b>Application:</b>	Flow Cytometry
<b>Reactivity:</b>	Human
<b>Format:</b>	Purified
<b>Isotype:</b>	Mouse IgG1
<b>Antibody Type:</b>	Monoclonal
<b>Formulation:</b>	Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide
<b>Protein Concentration:</b>	0.5 mg/mL
<b>Storage&amp;Handling:</b>	The antibody solution should be stored between 2°C and 8°C
<b>Recommended Usage:</b>	For flow cytometric staining, it is recommended to use less than 0.2 ug of this reagent per 0.5-1.0 million cells in a 100 µL volume. Optimal reagent performance should be determined by titration for each specific application
<b>Isotype Control:</b>	301401

### BACKGROUND INFORMATION

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HLA-G is a non-classical major histocompatibility complex (MHC) class I molecule encoded within the human leukocyte antigen (HLA) region. Unlike classical MHC class I proteins, HLA-G has limited polymorphism and a restricted tissue distribution, primarily expressed at the maternal-fetal interface, where it plays a critical role in immune tolerance during pregnancy.

Structurally, HLA-G consists of a heavy chain associated with  $\beta$ 2-microglobulin and presents peptides similarly to other MHC class I molecules. However, alternative splicing generates multiple isoforms, including both membrane-bound (e.g., HLA-G1) and soluble forms (e.g., HLA-G5). These structural variants contribute to its diverse immunomodulatory functions.

HLA-G interacts with inhibitory receptors such as ILT2 (LILRB1), ILT4 (LILRB2), and KIR2DL4 expressed on immune cells including natural killer (NK) cells, T cells, and antigen-presenting cells. Through these ligand-receptor interactions, HLA-G suppresses immune responses by inhibiting cytotoxic activity, reducing cytokine production, and promoting regulatory cell phenotypes.

In disease contexts, aberrant expression of HLA-G has been associated with cancer, viral infections, and autoimmune disorders. Many tumors exploit HLA-G expression to evade immune surveillance, leading to poorer clinical outcomes. Conversely, reduced HLA-G expression may contribute to pregnancy complications such as preeclampsia or recurrent miscarriage.

Therapeutically, HLA-G represents a promising target in both immunosuppression and immuno-oncology. Enhancing HLA-G activity could be beneficial in transplantation and autoimmune diseases by promoting immune tolerance. In contrast, blocking HLA-G or its receptors is being explored as a strategy to restore anti-tumor immunity. Ongoing research aims to better understand its mechanisms and develop targeted therapies that modulate HLA-G pathways.

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