

**HLA-E Antibody (Center) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP8517c****Specification**

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**HLA-E Antibody (Center) Blocking Peptide - Product Information**Primary Accession [P13747](#)**HLA-E Antibody (Center) Blocking Peptide - Additional Information****Gene ID** 3133**Other Names**

HLA class I histocompatibility antigen, alpha chain E, MHC class I antigen E, HLA-E, HLA-62, HLA-E

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP8517c](/products/AP8517c) was selected from the Center region of human HLA-E. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

**Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

**Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

**HLA-E Antibody (Center) Blocking Peptide - Protein Information****Name** HLA-E {ECO:0000303|PubMed:9486650, ECO:0000312|HGNC:HGNC:4962}**Function**

Non-classical major histocompatibility class Ib molecule involved in immune self-nonself discrimination. In complex with B2M/beta-2-microglobulin binds nonamer self-peptides derived from the signal sequence of classical MHC class Ia molecules (VL9 peptides - VMAPRT[V/L][L/V/I/F]L) (PubMed: [18083576](http://www.uniprot.org/citations/18083576) target="\_blank">18083576</a>, PubMed: [18339401](http://www.uniprot.org/citations/18339401) target="\_blank">18339401</a>, PubMed: [35705051](http://www.uniprot.org/citations/35705051) target="\_blank">35705051</a>, PubMed: [37264229](http://www.uniprot.org/citations/37264229) target="\_blank">37264229</a>, PubMed: [9754572](http://www.uniprot.org/citations/9754572) target="\_blank">9754572</a>). Peptide-bound HLA-E- B2M heterotrimeric complex primarily functions as a ligand for natural killer (NK) cell inhibitory receptor KLRD1-KLRC1, enabling NK cells to monitor the expression of other MHC class I molecules in healthy cells and to tolerate self (PubMed: [17179229](http://www.uniprot.org/citations/17179229) target="\_blank">17179229</a>),

PubMed:<a href="http://www.uniprot.org/citations/18083576" target="\_blank">18083576</a>, PubMed:<a href="http://www.uniprot.org/citations/37264229" target="\_blank">37264229</a>, PubMed:<a href="http://www.uniprot.org/citations/9486650" target="\_blank">9486650</a>, PubMed:<a href="http://www.uniprot.org/citations/9754572" target="\_blank">9754572</a>). Upon cellular stress, preferentially binds signal sequence-derived peptides from stress- induced chaperones and is no longer recognized by NK cell inhibitory receptor KLRD1-KLRC1, resulting in impaired protection from NK cells (PubMed:<a href="http://www.uniprot.org/citations/12461076" target="\_blank">12461076</a>). Binds signal sequence-derived peptides from non- classical MHC class Ib HLA-G molecules and acts as a ligand for NK cell activating receptor KLRD1-KLRC2, likely playing a role in the generation and effector functions of adaptive NK cells and in maternal-fetal tolerance during pregnancy (PubMed:<a href="http://www.uniprot.org/citations/30134159" target="\_blank">30134159</a>, PubMed:<a href="http://www.uniprot.org/citations/37264229" target="\_blank">37264229</a>, PubMed:<a href="http://www.uniprot.org/citations/9754572" target="\_blank">9754572</a>). Besides self-peptides, can also bind and present pathogen-underived peptides conformationally similar to VL9 peptides to alpha-beta T cell receptor (TCR) on unconventional CD8-positive cytotoxic T cells, ultimately triggering antimicrobial immune response (PubMed:<a href="http://www.uniprot.org/citations/16474394" target="\_blank">16474394</a>, PubMed:<a href="http://www.uniprot.org/citations/20195504" target="\_blank">20195504</a>, PubMed:<a href="http://www.uniprot.org/citations/30087334" target="\_blank">30087334</a>, PubMed:<a href="http://www.uniprot.org/citations/34228645" target="\_blank">34228645</a>). Presents HIV gag peptides (immunodominant KAFSPEVIPMF and subdominant KALGPAATL epitopes) predominantly to CD8-positive T cell clones expressing a TRAV17-containing TCR, triggering HLA-E-restricted T cell responses (PubMed:<a href="http://www.uniprot.org/citations/34228645" target="\_blank">34228645</a>). Presents mycobacterial peptides to HLA-E- restricted CD8-positive T cells eliciting both cytotoxic and immunoregulatory functions (PubMed:<a href="http://www.uniprot.org/citations/20195504" target="\_blank">20195504</a>, PubMed:<a href="http://www.uniprot.org/citations/35705051" target="\_blank">35705051</a>).

### Cellular Location

Cell membrane; Single-pass type I membrane protein. Golgi apparatus membrane

### Tissue Location

Expressed in secretory endometrial cells during pregnancy (at protein level). The expression in nonlymphoid tissues is restricted to endothelial cells from all types of vessels, including arteries, veins, capillaries, and lymphatics (at protein level). In lymphoid organs, it is mainly expressed in endothelial venules, B and T cells, monocytes, macrophages, NK cells and megakaryocytes (at protein level).

## HLA-E Antibody (Center) Blocking Peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Blocking Peptides](#)

## HLA-E Antibody (Center) Blocking Peptide - Images

## HLA-E Antibody (Center) Blocking Peptide - Background

HLA-E preferably binds to a peptide derived from the signal sequence of most HLA-A, -B, -C and -G molecules.