

APC Anti-Human HLA-ABC Antibody

Catalog Number:	102815, 102816
Size:	25 tests, 100 tests
Target Name:	HLA-ABC, Major Histocompatibility Class I, MHC class I
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	W6/32
Application:	Flow Cytometry
Reactivity:	Human
Format:	APC
Isotype:	Mouse IgG2a
Antibody Type:	Monoclonal
Formulation:	Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide and 0.2% (w/v) BSA
Protein Concentration:	Supplied at a lot-specific concentration.
Storage and Handling:	The antibody solution should be stored undiluted between 2°C and 8°C, and protected from prolonged exposure to light. Do not freeze.
Recommended Usage:	For flow cytometric staining, it is recommended to use 5 µL 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. APC has an excitation max at 650 nm and an emission max at 660 nm.
Excitation Laser:	Red Laser (633 nm)
Isotype Control:	301503
RRID:	AB_3738661

BACKGROUND INFORMATION

HLA-ABC refers collectively to the classical human leukocyte antigen (HLA) class I molecules HLA-A, HLA-B, and HLA-C, which are essential for immune surveillance by cytotoxic lymphocytes. These molecules are expressed on nearly all nucleated cells and function to present intracellularly derived peptide antigens to CD8⁺ T cells, enabling the immune system to detect and eliminate virally infected or malignant cells.

Structurally, each HLA-A, -B, or -C molecule is a heterodimer composed of a polymorphic heavy α chain (~45 kDa) noncovalently associated with the invariant β 2-microglobulin (β 2m). The α chain consists of three extracellular domains (α 1, α 2, and α 3), a transmembrane region, and a short cytoplasmic tail. The α 1 and α 2 domains form a closed-ended peptide-binding groove that typically accommodates peptides 8-11 amino acids in length. Extensive polymorphism within these domains underlies differences in peptide-binding specificity among HLA alleles, shaping individual immune responses.

The ligands of HLA-ABC are peptides generated from endogenous proteins, including normal self proteins, viral antigens, and tumor-associated antigens. These peptides are produced by proteasomal degradation in the cytosol and transported into the endoplasmic reticulum by the transporter associated with antigen processing (TAP), where they are loaded onto HLA class I molecules. Once expressed on the cell surface, peptide-HLA-ABC complexes are recognized by the T cell receptor (TCR) on CD8⁺ T cells. In addition, HLA-ABC molecules interact with inhibitory and activating receptors on natural killer (NK) cells, such as killer cell immunoglobulin-like receptors (KIRs), allowing NK cells to sense “missing self” when HLA class I expression is reduced.

HLA-ABC molecules are strongly implicated in disease. Specific HLA-A, -B, and -C alleles are associated with susceptibility or resistance to infectious diseases, cancer outcomes, and autoimmune conditions such as ankylosing spondylitis (HLA-B27) and psoriasis (HLA-C*06:02). Many tumors downregulate HLA-ABC expression to evade CD8⁺ T cell recognition, contributing to immune escape. Conversely, loss of HLA class I can enhance susceptibility to NK cell-mediated killing.

Therapeutically, HLA-ABC is central to cancer immunotherapy, antiviral immunity, and transplantation. Effective CD8⁺ T cell-based therapies, including cancer vaccines, checkpoint inhibitors, and adoptive T cell therapies, rely on intact HLA class I antigen presentation. In transplantation, HLA-ABC matching is critical to reduce graft rejection. Additionally, modulation or restoration of HLA-ABC expression is an active area of research aimed at overcoming tumor immune evasion and improving immunotherapeutic outcomes.

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