

MHC class II (IBL-5/22): sc-59322

BACKGROUND

Major histocompatibility complex (MHC) molecules, also designated human leukocyte antigen (HLA) molecules, are cell-surface receptors that bind foreign peptides and present them to T lymphocytes. MHC class I molecules consist of two polypeptide chains, an α or heavy chain and β -2-Microglobulin, a non-covalently associated protein. Cytotoxic T lymphocytes bind antigenic peptides presented by MHC class I molecules. Antigens that bind to MHC class I molecules are typically eight to ten residues in length and are stabilized in a peptide binding groove. MHC class II molecules are encoded by polymorphic MHC genes and consist of a non-covalent complex of an α and β chain. Helper T lymphocytes bind antigenic peptides presented by MHC class II molecules. MHC class II molecules bind 13-18 amino acid antigenic peptides. Accumulating in endosomal/lysosomal compartments and on the surface of B cells, HLA-DM and -DO molecules regulate binding of exogenous peptides to class II molecules (HLA-DR) by sustaining a conformation that favors peptide exchange. The differential structural properties of MHC class I and class II molecules account for their respective roles in activating different populations of T lymphocytes.

REFERENCES

1. Murphy, D.B., et al. 1989. A novel MHC class II epitope expressed in thymic medulla but not cortex. *Nature* 338: 765-768.
2. AYu., R., et al. 1991. On the complexity of self. *Nature* 353: 660-662.

CHROMOSOMAL LOCATION

Genetic locus: H2-Ea-ps (mouse) mapping to 17 B1.

SOURCE

MHC class II (IBL-5/22) is a rat monoclonal antibody raised against spleen cells of mouse origin.

PRODUCT

Each vial contains 100 μ g IgG₁ in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

MHC class II (IBL-5/22) is recommended for detection of MHC class II of mouse origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and flow cytometry (1 μ g per 1 x 10⁶ cells).

Molecular Weight of MHC class II α : 34 kDa.

Molecular Weight of MHC class II β : 29 kDa.

Positive Controls: I-11.15 whole cell lysate: sc-364370, mouse spleen extract: sc-2391 or mouse PBL whole cell lysate.

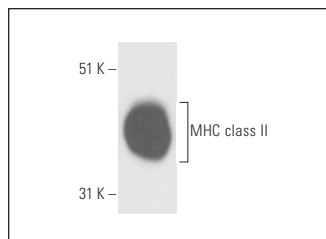
STORAGE

Store at 4° C, ****DO NOT FREEZE****. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

DATA



MHC class II (IBL-5/22): sc-59322. Western blot analysis of MHC class II expression in mouse PBL whole cell lysate.

SELECT PRODUCT CITATIONS

1. Butovsky, O., et al. 2006. Glatiramer acetate fights against Alzheimer's disease by inducing dendritic-like microglia expressing Insulin-like growth factor 1. *Proc. Natl. Acad. Sci. USA* 103: 11784-11789.
2. Gennari, F., et al. 2009. Single-chain antibodies that target lentiviral vectors to MHC class II on antigen-presenting cells. *Hum. Gene Ther.* 20: 554-562.
3. Hinterberger, M., et al. 2010. Autonomous role of medullary thymic epithelial cells in central CD4⁺ T cell tolerance. *Nat. Immunol.* 11: 512-519.
4. Choi, S.H., et al. 2015. SYK regulates macrophage MHC-II expression via activation of autophagy in response to oxidized LDL. *Autophagy* 11: 785-795.
5. Benque, I.J., et al. 2018. The neuropeptides of ocular immune privilege, α -MSH and NPY, suppress phagosome maturation in macrophages. *Immunohorizons* 2: 314-323.
6. Hernández-Pérez, S., et al. 2019. B cells rapidly target antigen and surface-derived MHCII into peripheral degradative compartments. *J. Cell Sci.* 133: jcs235192.
7. Pinto, B., et al. 2020. Rescuing over-activated microglia restores cognitive performance in juvenile animals of the Dp(16) mouse model of Down syndrome. *Neuron* 108: 887-904.e12.
8. Lee, J., et al. 2021. Overexpression of cathepsin S exacerbates lupus pathogenesis through upregulation TLR7 and IFN- α in transgenic mice. *Sci. Rep.* 11: 16348.
9. Aguilar-Pineda, J.A., et al. 2021. Vascular smooth muscle cell dysfunction contribute to neuroinflammation and Tau hyperphosphorylation in Alzheimer disease. *iScience* 24: 102993.
10. Alvarez, K.L.F., et al. 2022. Protocol to assess the effects of dysfunctional human vascular smooth muscle cells on other brain cells using *in vitro* models of Alzheimer's disease. *STAR Protoc.* 3: 101149.

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.