

anti-human CD11a (no azide)

monoclonal antibody MEM-25 to human CD11a

Cat-No: **21270110**

100 µg in 100 µl

Clone: MEM-25

Specificity: The antibody MEM-25 reacts with CD11a (α-subunit of human LFA-1), a 170-180 kDa type I transmembrane glycoprotein expressed on B and T lymphocytes, monocytes, macrophages, neutrophils, basophils and eosinophils. HLDA IV; WS Code NL 209

Isotype subclass: Mouse IgG1

Form: Purified from ascites by protein-A affinity chromatography.

Purity: > 98% (by SDS-PAGE)

Physical state: Liquid

Buffer/Additives/Preservative: PBS (sterile), (pH 7.4)

Expiration date: The reagent is stable until the expiry date stated on the vial label.

Storage conditions:

Store at -20°C. Avoid freeze/thaw cycles. Should be handled under aseptic conditions.

Application:

Flow Cytometry

Immunoprecipitation: excellent antibody for immunoaffinity purification of LFA-1 complex

Functional Application: The antibody blocks binding of LFA-1 complex to ICAM-1.

References:

Leucocyte Typing IV. Knapp W et al. (Eds.), Oxford University Press (1989).

Bazil V. et al., Folia Biol. (Praha) 36, 41 (1990).

Background: CD11a (LFA-1α) together with CD18 constitute leukocyte function-associated antigen 1 (LFA-1), the αLβ2 integrin. CD11a is implicated in activation of LFA-1 complex. LFA-1 is expressed on the plasma membrane of leukocytes in a low-affinity conformation. Cell stimulation by chemokines or other signals leads to induction of the high-affinity conformation, which supports tight binding of LFA-1 to its ligands, the intercellular adhesion molecules ICAM-1, -2, -3. LFA-1 is thus involved in interaction of various immune cells and in their tissue-specific settlement, but participates also in control of cell differentiation and proliferation and of T-cell effector functions. Blocking of LFA-1 function by specific antibodies or small molecules has become an important therapeutic approach in treatment of multiple inflammatory diseases. For example, humanized anti-LFA-1 antibody Efalizumab (Raptiva) is being used to interfere with T cell migration to sites of inflammation; binding of cholesterol-lowering drug simvastatin to CD11a allosteric site leads to immunomodulation and increase in lymphocytic cholinergic activity

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