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for the Science of Tomorrow™

Purified Mouse anti-Human CD11a **Monoclonal Antibody**

CLX16AP

Lot:

Size: $0.1 \, \text{mg}$

Clone: **MEM-83**

Mouse IgG1 **Isotype:**

Specificity: The antibody MEM-83 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 211

Immunogen: Human peripheral blood lymphocytes.

Species Reactivity: Human

Application: Functional Application

> The antibody MEM-83 directly induces the binding of T cells to purified ICAM-1. Using an in vitro-translated CDlla cDNA deletion series, the MEM-83 activation epitope was mapped to the "I" domain of the LFA-1 α-subunit. The studies have therefore identified a novel LFA-1 activation epitope mapping to the I domain of LFA-1, which could play a role in the regulation of

LFA-1 binding to ICAM-1.

Flow Cytometry **Immunoprecipitation**

Purity: > 95% (by SDS-PAGE)

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

Continued...

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Concentration: 1 mg/ml

Storage Buffer: Phosphate buffered saline (PBS) with 15 mM sodium azide,

approx. pH 7.4

Storage / Stability: Store at 2-8°C. Do not use after expiration date stamped on vial

label. For long-term storage aliquot and store at -20°C. Avoid

freeze/thaw cycles.

Background: CD11a (LFA-1a) together with CD18 constitute leukocyte function-

associated antigen 1(LFA-1), the aLb2 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 the high-affinity conformation, which supports tight binding of LFA-1 to its ligands, the intercellular adhesion

molecules

activity.

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 For example, humanized anti-LFA-1 Efalizumab (Raptiva) is being used to interfere with T cell migration to sites of inflammation; binding of cholesterol-lowering simvastatin to CD11a allosteric site leads immunomodulation and increase in lymphocytic cholinergic

References:

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