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Product Information

Monoclonal Anti-I-CAM-1 (CD54) antibody produced in mouse
clone 8.4A6, purified immunoglobulin

Product Number **C2969**

Product Description

Monoclonal Anti-Human ICAM-1 (CD54) (mouse IgG1 isotype) is derived from the 8.4A6 hybridoma produced by the fusion of mouse myeloma cells and splenocytes from BALB/c mice immunized with a TNF-α activated human endothelial cells.[1] The 8.4A6 producing hybridoma was developed by D.O. Haskard and coworkers[1] at the Rheumatology Unit, the Division of Medicine, Guy's Hospital, London.

The isotype is determined by a double diffusion immunoassay using Mouse Monoclonal Antibody Isotyping Reagents, Product Number ISO2. The antibody is purified from ascites fluid using protein A.

Monoclonal Anti-Human ICAM-1 (CD54) reacts specifically with ICAM-1 (CD54) expressed on the surface of activated endothelial cells, lymphocytes, monocytes and granulocytes. The epitope recognized by the antibody is localized in domain D2 of the ICAM-1 (CD54) molecule.[2] The antibody may be used in flow cytometry, ELISA,[3] immunocytochemistry,[2] and immunohistochemistry (frozen tissues). It inhibits leukocytes adhesion to activated endothelial cells,[2] but not adhesion *of Plasmodium falciparum*-infected erythrocytes.[4] The antibody stimulates homotypic aggregation of SKW3 cells.[2] It is also useful for studying soluble CD54 (ICAM-1) using capture ELISA.[3]

Human CD54, also known as intercellular adhesion molecule-1 (ICAM-1), is a 85-110 kDa single-chain type 1 integral membrane glycoprotein with an extra-cellular domain of five immunoglobulin superfamily repeats, a transmembrane region and a cytoplasmic domain. It shares considerable amino acid sequence homology with CD50 (ICAM-3) and with CD102 (ICAM-2).[5,6] ICAM-1 (CD54) is expressed by activated endothelial cells. It is detected on cells of many other lineages (e.g. epithelial cells, fibroblasts, chondrocytes, B lymphocytes, T lymphocytes (low), monocytes, macrophages, dendritic cells and neutrophils, with lower levels that increase in inflammation.[1,2,5] Also, ICAM-1 (CD54) is detected in some carcinoma and melanoma cells.[2] Soluble ICAM-1 (CD54) is detectable in the plasma and is elevated in patients with various inflammatory syndromes.[3] ICAM-1 (CD54) mediates myeloid cells adhesion to activated vascular endo-thelium at the vessel wall and to other leucocytes.[1,2] ICAM-1 (CD54) mediates T cell interactions with antigen presenting cells or target cells and also other T-T or T-B cell interactions. It seems to serve as an adhesion receptor for *Plasmodium falciparum*, thereby enabling binding of infected RBC to the vascular endothelia in selected organs, causing the typical sequestration of severe malaria. ICAM-1 (CD54) is the ligand for the integrin LFA-1 (CD11a/CD18).[5] In addition, ICAM-1 has binding sites for Rhinovirus,[6] Mac-1(CD11b/CD18)[7] and *Plasmodium falciparum*-infected erythrocytes.[4] *In vitro* upregulation of CD54 (ICAM-1) expression of cultured endothelial cells occurs upon activation by various inflammatory mediators, such as IL-1β, TNF-α or IFN-γ. It is induced by phorbol esters, retinoic acid, and lipopolysaccharides. Peak levels of ICAM-1 usually occur within 10-24 hours.[1]

Reagents

Supplied in 0.01 M phosphate buffered saline, pH 7.4, 0.2 μm filtered.

Antibody concentration is approximately 2 mg/ml as determined by E_{280}.

Storage/Stability

For continuous use, store sterile at 2–8 °C. For extended storage freeze in sterile working aliquots. Repeated freezing and thawing, or storage in "frost-free" freezers, is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use.

Product Profile
Indirect immunofluorescence: a minimum working concentration of 10-20 μg/ml is determined by staining of acetone-fixed, frozen human tonsil sections.

Note: In order to obtain best results in different techniques and preparations we recommend determining optimal working dilutions by titration test.

References

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3. Mason, J.C., et al., *Arthritis Rheum*., **36**, 519 (1993).
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5. Springer, T.A., et al., *Nature*, **346**, 425 (1990).
6. Staunton, D.E., et al., *Cell*, **61**, 243 (1990).
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