

Product Information

Monoclonal Anti-Vascular Cell Adhesion Molecule 1 Clone 1.4C3

produced in mouse, purified immunoglobulin

Catalog Number **V9263**

Synonym: Monoclonal Anti-VCAM-1

Product Description

Monoclonal Anti-Vascular Cell Adhesion Molecule 1 (mouse IgG1 isotype) is derived from the hybridoma produced by the fusion of mouse myeloma NS1 cells with splenocytes from BALB/c mice immunized with stimulated HUVEC cells and mouse myeloma NS1 cells. The antibody is purified by protein G chromatography.

Monoclonal Anti-VCAM-1 recognizes human vascular cell adhesion molecule-1 (also known as CD106 or INCAM-110). The antibody has been used in immunohistochemistry on frozen or formalin-fixed, paraffin-embedded tissue sections and immunoblotting.

Vascular Cell Adhesion Molecule-1 is a 110 kDa cell surface integral membrane glycoprotein expressed by cytokine-activated endothelium. VCAM-1 is a member of the immunoglobulin-related gene superfamily of adhesion molecules. The members of this family share the immunoglobulin domain of 90 and 100 amino acids arranged in a sandwich of two sheets of β -strands, usually linked by a disulfide bond in the center. Unlike the immunoglobulins and the TCR/CD3 complex, these molecules do not possess variable regions that undergo somatic diversification. All members participate in T cell adhesive interactions and include antigen-specific receptors of T and B cells.

VCAM-1, whose expression is induced by inflammatory cytokines such as IL-1, mediates the adhesion of mononuclear cells to the endothelium by binding to the VLA-4/ β 1 integrin complex on monocytes and lymphocytes. VCAM-1 plays an important role in various immunological and inflammatory responses. The mature VCAM-1 protein has six sites for N-glycosylation. VCAM-1, rapidly induced by IL-1 and TNF- α on endothelium, is expressed at high levels on

stimulated vascular endothelial cells and at minimal levels on unstimulated endothelial cells. It is also present on follicular and interfollicular dendritic cells of lymph nodes, myoblasts, and some macrophages. In inflammatory conditions and in cardiac allografts undergoing rejection, VCAM-1 is upregulated in the endothelium of postcapillary venules. Arterial expression of VCAM-1 is also found in experimental models of atherosclerosis in the rabbit.¹⁻⁴

The deficiencies of the cell adhesion molecules VCAM and α 4 integrin result in epicardial dissolution and subsequent myocardial thinning. Studies have shown that NF- κ B activation is the underlying molecular mechanism for constitutive expression of E-selectin, VCAM-1 and ICAM-1 on human B lymphocytes and plasma cells. Clinical studies have shown that elevated serum concentrations of cell adhesion molecules such as inter-cellular adhesion molecule-1 (ICAM-1), VCAM-1, E-selectin (ESEL) and P-selectin (PSEL) may be independent risk factors for atherosclerosis and cardiovascular disease (CVD). The concentration of soluble CAMs, particularly sICAM-1 and sESEL, reflect the level of established CVD risk factors in apparently healthy men and women, adding to the evidence that these factors contribute to CVD through their inflammatory effects on the vascular endothelium. VCAM-1 expression is reportedly correlated with adhesion of melanoma, Burkitt's lymphoma, osteosarcoma, and kidney carcinoma but not colon carcinoma cells to endothelial cells.⁵⁻⁷

Reagent

Supplied as a solution in phosphate buffered saline and 0.08% sodium azide as a preservative.

Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

Store at -20°C . Upon initial thawing freeze the solution in working aliquots for extended storage. Avoid repeated freezing and thawing to prevent denaturing the antibody. Do not store in frost-free freezers. The antibody is stable for at least 12 months when stored appropriately. Working dilutions should be discarded if not used within 12 hours.

Product Profile

Immunohistochemistry: a working concentration of 2-4 $\mu\text{g}/\text{mL}$ is recommended using formalin-fixed, paraffin-embedded human tonsil tissue sections.

Immunohistochemistry: a working concentration of 2-4 $\mu\text{g}/\text{mL}$ is recommended using frozen sections of human tonsil tissue.

Note: In order to obtain the best results using various techniques and preparations, we recommend determining optimal working concentration by titration.

References

1. Kumar, A.G., et al., Murine VCAM-1 molecular cloning, mapping, and analysis of a truncated form. *J. Immunol.*, **153**, 4088-4098 (1994).
2. Rice, G.E., et al., Vascular and nonvascular expression of INCAM-110. A target for mononuclear leukocyte adhesion in normal and inflamed human tissues. *Am. J. Pathol.*, **138**, 385-393 (1991).
3. Bevilacqua, M.P., Endothelial-leukocyte adhesion molecules. *Ann. Rev. Immunol.*, **11**, 767-804 (1993).
4. Foster, C.A., VCAM-1/ $\alpha 4$ -integrin adhesion pathway: therapeutic target for allergic inflammatory disorders. *J. Allergy Clin. Immunol.*, **98**, S270-277 (1996).
5. Cunningham, A.C., et al., Constitutive expression of MHC and adhesion molecules by alveolar epithelial cells (type II pneumocytes) isolated from human lung and comparison with immunocyto-chemical findings., *J. Cell Sci.*, **107**, 443-449 (1994).
6. Belmont, H.M., et al., Up-regulation of endothelial cell adhesion molecules characterizes disease activity in systemic lupus erythematosus. The Schwartzman phenomenon revisited. *Arthritis Rheumatism*, **37**, 376-383 (1994).
7. Xia, Y.F., et al., NF- κB activation for constitutive expression of VCAM-1 and ICAM-1 on B lymphocytes and plasma cells. *Biochem. Biophys. Res. Comm.*, **289**, 851-856 (2001).

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