Recombinant Mouse CD31/PECAM-1

Catalog Number: 3628-PC

DESCRIPTION

Source	Mouse myeloma cell line, NS0-derived mouse CD31/PECAM-1 protein Glu18-Lys590, with a C-terminal 6-His tag Accession # Q08481
N-terminal Sequence Analysis	Glu18
Predicted Molecular Mass	65.5 kDa (monomer)

SPECIFICATIONS

SDS-PAGE	91-100 kDa, reducing conditions
Activity	Measured by its binding ability in a functional ELISA. Recombinant Mouse CD31/PECAM-1 (Catalog # 3628-PC) binds to Recombinant Human Integrin α8β1 with an ED_{50} of 0.060-0.720 µg/mL.
Endotoxin Level	<0.01 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 200 µg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** • 12 months from date of receipt, -20 to -70 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 3 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

PECAM-1 (platelet-endothelial cell adhesion molecule-1; also known as CD31) is a 130 kDa type I transmembrane glycoprotein adhesion molecule in the immunoglobulin superfamily (1, 2). Expression is restricted to cells involved in circulation, especially endothelial cells, platelets, monocytes, neutrophils and lymphocyte subsets. PECAM-1 is concentrated at cell-cell junctions and is required for transendothelial migration (TEM) (1 - 3). The extracellular domain (ECD) of PECAM-1 has ten potential N-linked glycosylation sites and six C2-type Ig-like domains, the first of which is critical for adhesion and extravasation (3, 4). The cytoplasmic domain contains immunoregulatory tyrosine-based inhibitory and switch motifs (ITIM, ITSM) that mediate both inhibition and activation via phosphotyrosine-mediated engagement of SH2-containing signaling molecules (1, 5). Metalloproteinase-mediated ectodomain shedding occurs during apoptosis (6) but increased serum PECAM-1 ectodomain in HIV and active multiple sclerosis occurs independent of apoptosis (7, 8). In humans, expression of six isoforms with exon deletions in the cytoplasmic domain is tissue- and stage-specific, but full-length PECAM-1 is predominant. A form lacking the ITSM predominates in mouse (9). Mouse PECAM-1 ECD shows 77%, 63%, 63%, 63% and 61% amino acid (aa) identity with rat, human, canine, porcine and bovine PECAM-1, respectively. PECAM-1 participates with other adhesion molecules in some functions, but is the critical molecule for TEM. Homotypic PECAM-1 adhesion in trans, combined with cycling of PECAM-1 to and from surface-connected endothelial cell vesicles, leads leukocytes across endothelial tight junctions (3, 10). Homotypic adhesion and signaling functions also strongly suppress mitochondria-dependent apoptosis (11). In platelets, PECAM-1 is necessary for limiting thrombus formation (12) and promoting integrin-mediated clot retraction and platelet spreading (13), but mechanisms for these phenomena are unclear. $PECAM^{-/-}$ mice are deficient in chemokine-mediated chemotaxis (14).

References:

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