

Mouse E-Selectin / CD62e / SELE Protein (His Tag)

Catalog Number: 50736-M08H



Sino Biological
Biological Solution Specialist

General Information

Gene Name Synonym:

CD62E; E-selectin; Elam

Protein Construction:

A DNA sequence encoding the mouse SELE (NP_035475.1) (Met1-Pro564) was expressed with a C-terminal polyhistidine tag.

Source: Mouse

Expression Host: HEK293 Cells

QC Testing

Purity: > 95 % as determined by SDS-PAGE

Bio Activity:

Measured by the ability of the immobilized protein to support the adhesion of U937 human histiocytic lymphoma cells. When 5×10^4 cells/well are added to mouse E-Selectin coated plates (2 $\mu\text{g/mL}$ with 100 $\mu\text{L/well}$), approximately 30%-60% cells will adhere for 1 hour incubation at 37°C.

Endotoxin:

< 1.0 EU per μg of the protein as determined by the LAL method

Stability:

Samples are stable for up to twelve months from date of receipt at -70 °C

Predicted N terminal: Trp 29

Molecular Mass:

The recombinant mouse SELE comprises 547 amino acids and has a predicted molecular mass of 60 kDa. The apparent molecular mass of the protein is approximately 70-80 kDa in SDS-PAGE under reducing conditions due to glycosylation.

Formulation:

Lyophilized from sterile PBS, pH 7.4

Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

Usage Guide

Storage:

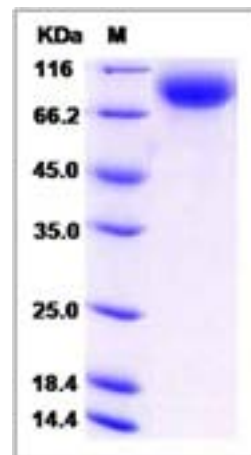
Store it under sterile conditions at -20°C to -80°C upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

E-selectin, also known as endothelial leukocyte adhesion molecule-1 (ELAM-1) and CD62E, is an inducible adhesion molecule that is expressed on the surfaces of stimulated vascular endothelial cells and is sometimes involved in cancer cell metastasis. E-selectin exhibits a complex mosaic structure consisting of a large extracellular region comprised of a lectin domain, an EGF-like domain, and a short consensus repeat (SCR) domain, followed by a transmembrane region and a relatively short (32 aa) cytoplasmic tail. As a member of the LEC-CAM or selectin family, E-selectin recognizes and binds to sialylated carbohydrates including members of the Lewis X and Lewis A families found on monocytes, granulocytes, and T-lymphocytes. E-selectin supports rolling and stable arrest of leukocytes on activated vascular endothelium, and furthermore, it was indicated that it can also transduce an activating stimulus via the MAPK cascade into the endothelial cell during leukocyte adhesion. E-selectin regulates adhesive interactions between certain blood cells and endothelium. The soluble form of E selectin (sE-selectin) is a marker of endothelial activation, and has a potential role in the pathogenesis of cardiovascular disease as raised levels have been found in hypertension, diabetes and hyperlipidemia, although its association in established atherosclerosis disease and its value as a prognostic factor is more controversial. Soluble E-selectin is inversely associated with the muscular component of the left ventricle, thereby suggesting that the lack of such a reparative factor may be associated with cardiac remodeling in end-stage renal disease (ESRD) patients. In addition, this adhesion molecule appears to be involved in the pathogenesis of atherosclerosis.

References

1. Roldn V, *et al.* (2003) Soluble E-selectin in cardiovascular disease and its risk factors. A review of the literature. *Thromb Haemost.* 90(6): 1007-20.
2. Kawase J, *et al.* (2009) Increase in E-selectin expression in umbilical vein endothelial cells by anticancer drugs and inhibition by cimetidine. *Oncol Rep.* 22(6): 1293-7.
3. Matsumoto K, *et al.* (2010) Soluble adhesion molecule E-selectin predicts cardiovascular events in Japanese patients with type 2 diabetes mellitus. *Metabolism.* 59(3): 320-4.

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