

**DESCRIPTION**

<b>Source</b>	Chinese Hamster Ovary cell line, CHO-derived human L-Selectin/CD62L protein Trp52-Leu332, with a C-terminal 6-His tag Accession # EAW90856.1
<b>N-terminal Sequence Analysis</b>	Trp52
<b>Predicted Molecular Mass</b>	32 kDa

**SPECIFICATIONS**

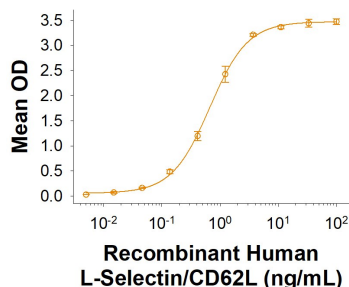
<b>SDS-PAGE</b>	65-75 kDa, under reducing conditions.
<b>Activity</b>	Measured by its binding ability in a functional ELISA. When Human L-Selectin/CD62L Antibody (Catalog # AF728) is immobilized at 0.1 µg/mL (100 µL/well), Recombinant Human L-Selectin/CD62L His-tag (Catalog # 11374-LS) binds with an ED <sub>50</sub> of 0.180-1.80 ng/mL.
<b>Endotoxin Level</b>	<0.10 EU per 1 µg of the protein by the LAL method.
<b>Purity</b>	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

<b>Reconstitution</b>	Reconstitute at 500 µg/mL in PBS.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
<b>Stability &amp; Storage</b>	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> <li>• 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>• 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>• 3 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

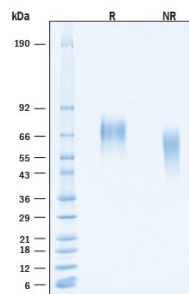
**DATA**

**Binding Activity**



**Recombinant Human L-Selectin/CD62L His-tag Protein Binding Activity.** When Human L-Selectin/CD62L Antibody (Catalog # AF728) is immobilized at 0.1 µg/mL (100 µL/well), Recombinant Human L-Selectin/CD62L His-tag (Catalog # 11374-LS) binds with an ED<sub>50</sub> of 0.180-1.80 ng/mL.

**SDS-PAGE**



**Recombinant Human L-Selectin/CD62L His-tag Protein SDS-PAGE.** 2 µg/lane of Recombinant Human L-Selectin/CD62L His-tag Protein (Catalog # 11374-LS) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 65-75 kDa.

**BACKGROUND**

L-Selectin, also known as Leukocyte adhesion molecule 1 (LAM-1) and CD62L, is a type-1 cell surface glycoprotein and cell adhesion molecule of the Selectin family (1). In humans, there are 3 Selectins, P, E, and L, and they are Ca<sup>2+</sup> dependent lectins that help mediate the initial adhesive step during inflammation and immune surveillance (2). Mature L-Selectin consists of an extracellular domain (ECD) with a C-type lectin domain and an epidermal growth factor (EGF)-like domain, a transmembrane domain, and a short cytoplasmic domain. Within the ECD, human L-selectin shares 76% and 78% amino acid sequence identity with mouse and rat L-selectin, respectively. Several isoforms arising from alternative splicing have been reported, some with potential therapeutic implications (2,3). L-selectin is constitutively expressed on a wide variety of leukocytes and plays a role in the migration of lymphocytes into peripheral lymph nodes and sites of chronic inflammation, and of neutrophils into acute inflammatory sites (1-4). Acting in cooperation with P-Selectin and E-Selectin, L-Selectin mediates the initial interaction of circulating leukocytes with endothelial cells that produces a characteristic "rolling" of the leukocytes on the endothelium (5). This initial interaction, also involving ICAM-1 and VCAM-1, leads eventually to extravasation of the white blood cell through the blood vessel wall into the extracellular matrix tissue (6). L-selectin function is required for normal Treg cell migration and over expression might be result in reduced tumor growth (7). Several studies have reported that levels of L-Selectin may be elevated or lowered in subjects with a variety of conditions, such as in Alzheimer's disease or rheumatoid arthritis (3, 5).

**References:**

1. Ivetic, A. *et al.* (2019) *Front. Immunol.* **10**:1068.
2. Grailer, J.J. *et al.* (2009) *J. Dermatol sci.* **56**:141.
3. Hirata, T. *et al.* (2015) *Biochem Biophys Res. Commun.* **462**:371.
4. Wedepohl, S. *et al.* (2012) *Euro. J. Cell Biol.* **91**:257.
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6. Granger, D.N. and Senchenkova, E. (2010) *Inflammation and the Microcirculation*. San Rafael (CA): Morgan & Claypool Life Sciences Chapter 7.
7. Watson, H.A. *et al.* (2019) *Frontiers in immunology* **10**:1321.