

DESCRIPTION

Source	Mouse myeloma cell line, NS0-derived human LAR protein Ala27-Glu1251 with a C-terminal 6-His tag, and its proteolyzed forms Ala27-Arg1169 and Gln1170-Glu1251 with a C-terminal 6-His tag Accession # P10586-2
N-terminal Sequence Analysis	Ala27 and Gln1170
Predicted Molecular Mass	136 kDa

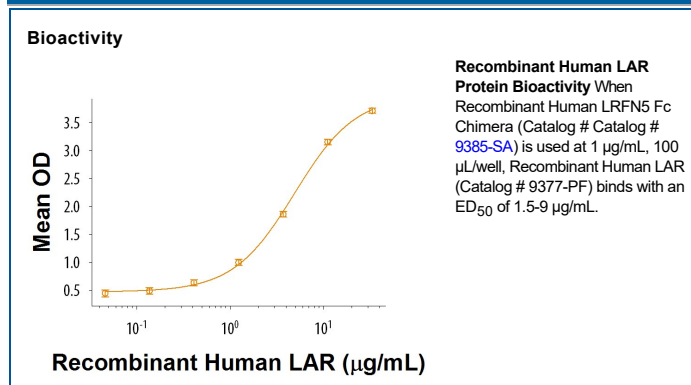
SPECIFICATIONS

SDS-PAGE	114-146 kDa and 14 kDa, reducing conditions
Activity	Measured by its binding ability in a functional ELISA. When Recombinant Human LRFN5 Fc Chimera (Catalog # 9385-SA) is used at 1 µg/mL, the concentration of Recombinant Human LAR that produces 50% optimal binding response is 1.5-9 µg/mL.
Endotoxin Level	<0.10 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	Supplied as a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Shipping	The product is shipped with dry ice or equivalent. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> • 12 months from date of receipt, -70 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after opening. • 3 months, -20 to -70 °C under sterile conditions after opening.

DATA



BACKGROUND

PTPRF (Receptor-type tyrosine-protein phosphatase F), also called LAR (Leukocyte common antigen related), is a 212 kDa single-pass type I membrane protein that is a member of the protein tyrosine phosphatase (PTP) family and the receptor class 2A subfamily (1). Human LAR cDNA encodes 1,907 amino acids (aa) including a 29 aa signal sequence, a 1234 aa extracellular region with six potential N-glycosylation sites, a 21 aa transmembrane sequence, and a 623 aa cytoplasmic domain (1). An 1,898 aa isoform appears from alternative splicing; it is missing aa 772 to 780 (1). Human LAR shares a 95% aa sequence identity with mouse and rat LAR. LARs have been shown to function in the regulation of epithelial cell-cell contacts at adherens junctions, as well as in the control of beta-catenin signaling (2). An increased expression level of LARs have been found in the insulin-responsive tissue of obese, insulin-resistant individuals, and may contribute to the pathogenesis of insulin resistance (3). LARs have been identified as novel ligands of SALM5/LRFN-5 that mediates SALM5/LRFN-5 dependent presynaptic differentiation in a splicing-dependent manner. SALM5/LRFN-5 interacts directly with the Ig domain of LAR family receptor protein tyrosine phosphatases. The postsynaptic SALM5/LRFN-5 promotes synapse development by trans-synaptically interacting with presynaptic LARs which is important for the regulation of excitatory synaptic strength (4). Clinically, LAR has been shown down-regulated in cancers and its up-regulation is associated with better clinical outcomes (5).

References:

1. SwissProt Accession # P10586.
2. Um J.W. *et al.* (2013) Trends Cell Biol. **23**:465.
3. Mander A. *et al.* (2005) FEBS Lett. **579**:3024.
4. Choi, Y. *et al.* (2016) Sci Rep. **6**:26676.
5. Bera, R. *et al.* (2014) Hepatology. **59**:2238.