

Product Data Sheet

Siglec-E Protein, Mouse (HEK293, Fc)

Cat. No.:	HY-P71314
Synonyms:	SiglecE; Siglec-E; Sialic Acid Binding Ig-like Lectin E
Species:	Mouse
Source:	HEK293
Accession:	Q6PJ50 (Q20-F355)
Gene ID:	83382
Molecular Weight:	Approximately 95 kDa

PROPERTIES

An Jequence	QNPQEGFTLN VERKVVVQEG LCVLVPCNFS YLKKRLTDWT	
	DSDPVHGFWY REGTDRRKDS IVATNNPIRK AVKETRNRFF	
	LLGEPWRNDC SLNIREIRKK DAGLYFFRLE RGKTKYNYMW	
	DKMTLVVTAL TNTPQILLPE TLEAGHPSNL TCSVPWDCGW	
	TAPPIFSWTG TSVSFLSTNT TGSSVLTITP QPQDHGTNLT	
	CQVTLPGTDV STRMTIRLNV SYAPKNLTVT IYQGADSVST	
	ILKNGSSLPI SEGQSLRLIC STDSYPPANL SWSWDNLTLC	
	PSKLSKPGLL ELFPVHLKHG GVYTCQAQHA LGSQHISLSL	
	SPQSSATLSE MMMGTF	
Appearance	Lyophilized powder.	
Formulation	Lyophilized from a 0.2 μm filtered solution of 20 mM Tris-HCl, 150 mM NaCl, pH 8.5.	
Endotoxin Level	<1 EU/µg, determined by LAL method.	
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 ug/mL in ddH ₂ O. For long term storage it is	
	recommended to add a carrier protein (0.1% BSA, 5% HSA, 10% FBS or 5% Trehalose).	
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein).	lt is
otoruge a otashity	recommended to freeze aliquots at -20°C or -80°C for extended storage.	15
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Shipping	Room temperature in continental US;may vary elsewhere.	

DESCRIPTION

Background

Sialic acid binding Ig-like lectin E (Siglec-E) is a mouse orthologue of human Siglec-9 and functions as a key immunosuppressive checkpoint molecule. Siglec-E is a sialic acid binding lectin predominantly expressed on the surface of myeloid cells to transduce inhibitory signal via recruitment of SH2-domain containing protein tyrosine phosphatase SHP- 1/2 upon binding to its sialoglycan ligands. Siglec-E interacts with CD36 to inhibits downstream VAV signaling involved in modified LDL uptake, thereby delaying atherosclerosis. The endogenous inducible Siglec-E plays crucial anti-inflammatory and neuroprotective roles following ischemic stroke, and thus might underlie an intrinsic mechanism of resolution of inflammation and self-repair in the brain. Siglec-E is an important negative regulator of neutrophil recruitment to the lung and β_2 integrin-dependent signaling and suppresses CD11b β_2 -integrin-dependent signaling^{[1][2][3]}.

Caution: Product has not been fully validated for medical applications. For research use only.

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