Recombinant Human SR-BI Fc Chimera

Catalog Number: 8114-SRB

DESCRIPTION

Source: Human embryonic kidney cell, HEK293-derived

Human SR-BI (Pro33-Tyr443) Accession # CAA80277	IEGRMD	Human IgG_1 (Pro100-Lys330)

N-terminus C-terminus

N-terminal Sequence Analysis	Pro33
Structure / Form	Disulfide-linked homodimer
Predicted Molecular Mass	73 kDa

SPECIFICATIONS

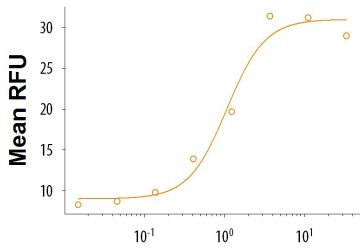
SDS-PAGE	84-115 kDa, reducing conditions
Activity	Measured by its ability to bind fluorescein-conjugated *S. aureus* Bioparticles. Jiang, Y. *et al.* (2006) J. Biol. Chem. **281**:11834. The ED_{50} for this effect is 0.4-2 µg/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>90%, 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 500 µg/mL in 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.

DATA

Bioactivity



Recombinant Human SR-BI Fc Chimera (Catalog # 8114-SRB) binds to fluorescein-conjugated *S. aureus* Bioparticles with an ED_{50} of 0.4-2 µg/mL.

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BACKGROUND

Scavenger Receptor, class B, member 1 (SR-BI), gene name SCARB1, is also known as CD36L1 (CD36-like 1) or CLA-1 (CD36 and LIMPII analogous 1) (1-5). SR-BI is a transmembrane glycoprotein found on macrophages, liver cells and other steroidogenic cells as a lipoprotein receptor. The 552 amino acid (aa) human SR-BI contains a central extracellular domain (ECD), flanked by N- and C-terminal transmembrane domains. Human splice variants differ at the N-terminal cytoplasmic and transmembrane domains (SR-BIII, 474 aa), the N-terminal end of the ECD (SR-BII, 409 aa), or the C-terminal cytoplasmic domain (isoform 3, 552 aa) (2). The human SR-BI ECD shares 80%, 80%, 89%, 86% and 84% aa sequence identity with mouse, rat, porcine, rabbit, and bovine SR-BI, respectively. SR-BI functions in reverse cholesterol transport (RCT), which is thought to be anti-atherogenic by facilitating transport of cholesteryl esters from macrophages back to the liver for degradation (3). In rodent hepatocytes, SR-BI is the main receptor mediating RCT, while human hepatocytes also express a second mediator, CETP (cholesteryl ester transfer protein) (3-5). The importance of SR-BI in humans is shown by human SR-BI genetic variants that alter lipid metabolism (3-7). For example, the P297S polymorphism lowers uptake of high-density lipoprotein (HDL) cholesterol in the liver and increases plasma HDL cholesterol (3-5). On endothelial cells, signaling through SR-BI activates nitric oxide production, which attenuates monocyte adhesion (6). On adrenocortical cells, SR-BI mediates uptake of cholesteryl esters from HDL for the synthesis of glucocorticoid hormones such as cortisol (3-5). On platelets, HDL binding to surface SR-BI inhibits aggregation and increases platelet survival time (3-5). On human ovarian granulosa cells, deficiency of SR-BI correlates with low fertility (3). SR-BI and its SR-BII isoform also bind bacterial lipopolysaccharides, facilitating uptake of various bacteria by cells such as peritoneal macrophages (8, 9). This uptake enhances inflammatory responses which, unless properly controlled, can result in sepsis (9-11).

References:

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