Human SR-BI Antibody

Monoclonal Mouse IgG_1 Clone # 947007

Catalog Number: MAB8114

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human SR-BI in direct ELISAs and Western blots.
Source	Monoclonal Mouse IgG_1 Clone # 947007
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Human embryonic kidney cell line HEK293-derived human SR-BI
Formulation	Lyophilized from a 0.2 μm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 μm filtered solution in PBS.

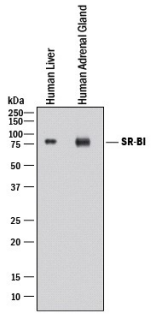
APPLICATIONS

Please Note: *Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.*

	Recommended Concentration	Sample
Western Blot	2 µg/mL	See Below
Immunohistochemistry	5-25 µg/mL	See Below

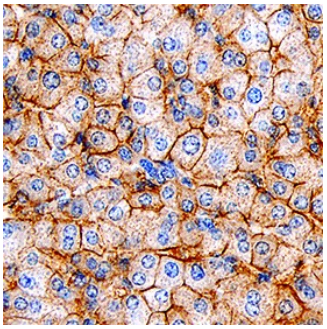
DATA

Western Blot



Detection of Human SR-BI by Western Blot. Western blot shows lysates of human liver tissue and human adrenal gland tissue. PVDF membrane was probed with 2 µg/mL of Mouse Anti-Human SR-BI Monoclonal Antibody (Catalog # MAB8114) followed by HRP-conjugated Anti-Mouse IgG Secondary Antibody (Catalog # HAF018). A specific band was detected for SR-BI at approximately 80 kDa (as indicated). This experiment was conducted under reducing conditions and using Immunoblot Buffer Group 1.

Immunohistochemistry



SR-BI in Human Liver. SR-BI was detected in immersion fixed paraffin-embedded sections of human liver using Mouse Anti-Human SR-BI Monoclonal Antibody (Catalog # MAB8114) at 15 µg/mL overnight at 4 °C. Tissue was stained using the Anti-Mouse HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS002) and counterstained with hematoxylin (blue). Specific staining was localized to cell membranes in hepatocytes. View our protocol for Chromogenic IHC Staining of Paraffin-embedded Tissue Sections.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
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. ● 6 months, -20 to -70 °C under sterile conditions after reconstitution.

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BACKGROUND

Scavenger Receptor, class B, member 1 (SR-B1), gene name SCARB1, is also known as CD36L1 (CD36-like 1) or CLA-1 (CD36 and LIMPII analogous 1) (1-5). SR-B1 is a transmembrane glycoprotein found on macrophages, liver cells and other steroidogenic cells as a lipoprotein receptor. The 552 amino acid (aa) human SR-B1 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-B1 ECD shares 80%, 80%, 89%, 86% and 84% aa sequence identity with mouse, rat, porcine, rabbit, and bovine SR-B1, respectively. SR-B1 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-B1 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-B1 in humans is shown by human SR-B1 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-B1 activates nitric oxide production, which attenuates monocyte adhesion (6). On adrenocortical cells, SR-B1 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-B1 inhibits aggregation and increases platelet survival time (3-5). On human ovarian granulosa cells, deficiency of SR-B1 correlates with low fertility (3). SR-B1 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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