

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Semaphorin 4A in direct ELISAs and Western blots. In direct ELISA, no cross-reactivity with recombinant human Semaphorin 4B, 4C, or 4D is observed and approximately 5% cross-reactivity with recombinant mouse Semaphorin 4A is observed.
Source	Monoclonal Mouse IgG ₁ Clone # 741509
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human Semaphorin 4A Gly32-His683 Accession # Q9H3S1
Conjugate	Alexa Fluor 405 Excitation Wavelength: 405 nm Emission Wavelength: 421 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

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.

Western Blot	Optimal dilution of this antibody should be experimentally determined.
Immunohistochemistry	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

Semaphorin 4A (Sema4A, previously semB) is a Class 4 transmembrane Semaphorin with activity in the immune and nervous systems (1). The 761 amino acid (aa) human Sema4A precursor contains a 32 aa signal sequence, a 651 aa extracellular domain (ECD) containing sema, PSI and C2-type immunoglobulin domains, a 21 aa transmembrane domain, and a 57 aa cytoplasmic domain with two Ser/Thr phosphorylation sites (2). Human Sema4A ECD shares 87%, 87%, 86% and 85% aa identity with mouse, rat, bovine and canine Sema4A, respectively, and shares 32-37% aa identity with other human Sema4 family members. Of six reported splice variants with 723, 629, 370, 321, 236 and 220 aa, five lack the N-terminus and/or portions of the sema domain, and three lack the transmembrane and cytoplasmic domains in the C-terminus (3). Sema4A was first described as a molecule that enhances T cell activation and interacts with TIM-2 (T cell immunoglobulin and mucin domain-2) (4). Mice with targeted disruption of Sema4A show defects in dendritic cell-mediated T cell priming and Th1 responses (5). Roles for Sema4A have also been identified in the brain, the endothelium and the eye. It mediates hippocampal neuron growth cone collapse *in vitro* through interaction of the sema domain with plexin-B1 (6). Interaction of Sema4A with endothelial cell plexin-D1 causes opposition to the angiogenic, proliferative, chemotactic and integrin-mediated adhesive actions of VEGF (7). The retina of Sema4A^{-/-} mice shows severe degeneration, and mutations of Sema4A are associated with retinitis pigmentosa and cone rod dystrophy in humans (8, 9).

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