

## DESCRIPTION

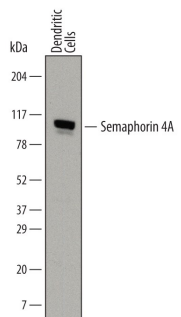
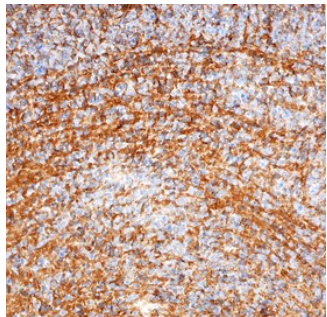
<b>Species Reactivity</b>	Human
<b>Specificity</b>	Detects human Semaphorin 4A in direct ELISAs and Western blots. In direct ELISA, no cross-reactivity with recombinant human Semaphorin 4B, 4C, or 4D is observed and approximately 5% cross-reactivity with recombinant mouse Semaphorin 4A is observed.
<b>Source</b>	Monoclonal Mouse IgG <sub>1</sub> Clone # 741509
<b>Purification</b>	Protein A or G purified from hybridoma culture supernatant
<b>Immunogen</b>	Mouse myeloma cell line NS0-derived recombinant human Semaphorin 4A Gly32-His683 Accession # Q9H3S1
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied 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
<b>Western Blot</b>	2 µg/mL	See Below
<b>Immunohistochemistry</b>	8-25 µg/mL	See Below

## DATA

<p><b>Western Blot</b></p>  <p><b>Detection of Human Semaphorin 4A by Western Blot.</b> Western blot shows lysates of human dendritic cells. PVDF membrane was probed with 2 µg/mL of Mouse Anti-Human Semaphorin 4A Monoclonal Antibody (Catalog # MAB46941) followed by HRP-conjugated Anti-Mouse IgG Secondary Antibody (Catalog # HAF007). A specific band was detected for Semaphorin 4A at approximately 90-95 kDa (as indicated). This experiment was conducted under reducing conditions and using Immunoblot Buffer Group 1.</p>	<p><b>Immunohistochemistry</b></p>  <p><b>Semaphorin 4A in Mouse Embryo.</b> Semaphorin 4A was detected in immersion fixed frozen sections of mouse embryo (13 d.p.c.) using Mouse Anti-Human Semaphorin 4A Monoclonal Antibody (Catalog # MAB46941) at 25 µg/mL overnight at 4 °C. Tissue was stained using the Anti-Mouse HRP-DAB Cell &amp; Tissue Staining Kit (brown; Catalog # CTS002) and counterstained with hematoxylin (blue). Specific staining was localized to neuronal processes. View our protocol for <a href="#">Chromogenic IHC Staining of Frozen Tissue Sections</a>.</p>
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## PREPARATION AND STORAGE

<b>Reconstitution</b>	Sterile PBS to a final concentration of 0.5 mg/mL.
<b>Shipping</b>	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
<b>Stability &amp; Storage</b>	<b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b> <ul style="list-style-type: none"> <li>● 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>● 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>● 6 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

**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<sup>-/-</sup> mice shows severe degeneration, and mutations of Sema4A are associated with retinitis pigmentosa and cone rod dystrophy in humans (8, 9).

**References:**

1. Kumanogoh, A. *et al.* (2003) *J. Cell Sci.* **116**:3463.
2. Swissprot Accession # Q9H3S1.
3. Entrez Accession # CAI15528, CAI15529, CAI15531, CAI15532, CAI15533 and EAW52993.
4. Kumanogoh, A. *et al.* (2002) *Nature* **419**:629.
5. Kumanogoh, A. *et al.* (2005) *Immunity* **22**:305.
6. Yukawa, K. *et al.* (2005) *Int. J. Mol. Med.* **16**:115.
7. Toyofuku, T. *et al.* (2007) *EMBO J.* **26**:1373.
8. Rice, D.S. *et al.* (2004) *Invest. Ophthalmol. Vis. Sci.* **45**:2767.
9. Abid, A. *et al.* (2007) *J. Med. Genet.* **43**:378.