

## DESCRIPTION

<b>Species Reactivity</b>	Mouse
<b>Specificity</b>	Detects mouse Semaphorin 3F in direct ELISAs. In direct ELISAs, no cross-reactivity with recombinant mouse Semaphorin 3A, 3B, 3C, 3E, 4C, 4D, 4G, recombinant human Semaphorin 3D or 3F is observed.
<b>Source</b>	Monoclonal Rat IgG <sub>2A</sub> Clone # 741307
<b>Purification</b>	Protein A or G purified from ascites
<b>Immunogen</b>	Mouse myeloma cell line NS0-derived recombinant mouse Semaphorin 3F Ala19 Pro775 (Arg583Ala and Arg586Ala) Accession # O88632
<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.

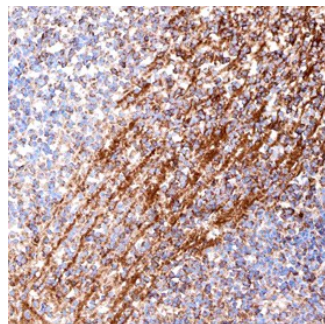
## APPLICATIONS

**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.

	<b>Recommended Concentration</b>	<b>Sample</b>
<b>Immunohistochemistry</b>	8-25 µg/mL	See Below

## DATA

### Immunohistochemistry



#### Semaphorin 3F in Mouse Brain.

Semaphorin 3F was detected in immersion fixed frozen sections of embryonic mouse brain (13 d.p.c.) using Rat Anti-Mouse Semaphorin 3F Monoclonal Antibody (Catalog # MAB3237) at 25 µg/mL overnight at 4 °C. Tissue was stained using the Anti-Rat HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS017) and counterstained with hematoxylin (blue). Specific staining was localized to neuronal processes. View our protocol for [Chromogenic IHC Staining of Frozen Tissue Sections](#).

## 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 3F (Sema 3F; previously Sema IV) is one of six Class 3 (secreted) semaphorins which in the mouse share 40-50% amino acid (aa) identity. Class 3 semaphorins are potent chemorepellents that function in axon guidance and/or vascular tip cell guidance during development (1). Sema 3F is expressed in the developing nervous system, especially in the dorsal spinal cord (2, 3). In adults, Sema 3F is expressed in the lung and most other tissues (2). Crystal structures of semaphorins reveal that the 500 aa N-terminal Sema domain forms a seven-blade β-propeller similar to that found in integrin molecules. Fourteen conserved cysteine residues and one or more N-glycosylation sites are thought to be critical for forming the secondary structure (4). Isoform A is missing aa 153-183 within the Sema domain relative to the long form (isoform B) but appears to have similar activity. C-terminal to the Sema domain, Sema 3F has a basic domain, a cysteine-knot plexin/semaphorin/integrin (PSI) domain, an Ig-like domain, a cysteine for dimerization and another basic domain at the C-terminus. Dimerization and cleavage at the C-terminus are required for repulsing activity of class 3 semaphorins (5). Mouse Sema 3F shares 96%, 99%, 92%, 97% and 82% aa identity with human, rat, bovine, canine and chick Sema 3F, respectively. Type 3 semaphorins transduce signals through transmembrane plexins, either directly or by binding associated neuropilin receptors. Sema 3F signaling is transduced by type-A plexins, especially Plexin-A3, via interaction with neuropilin-2 (3, 6). Genetic disruption of either Sema 3F or neuropilin-2 alters motor axon trajectory to the ventral forelimb (3). Sema 3F is deleted or downregulated in many metastatic tumors. Restoration of Sema 3F decreases tumorigenicity, vascularization and adhesiveness, most likely through repulsive interactions, VEGF antagonism and downstream integrin regulation (7).

### References:

1. Kruger, R.P. *et al.* (2005) *Nature Rev. Mol. Cell Biol.* **6**:789.
2. Eckhardt, F. and A. Meyerhans (1998) *Neuroreport* **9**:3975.
3. Huber, A.B. *et al.* (2005) *Neuron* **48**:949.
4. Gherardi, E. *et al.* (2004) *Curr. Opin. Struct. Biol.* **14**:669.
5. Adams, R.H. *et al.* (1997) *EMBO J.* **16**:6077.
6. Yaron, A. *et al.* (2005) *Neuron* **45**:513.
7. Chedotal, A. *et al.* (2005) *Cell Death Differ.* **12**:1044.