

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse Semaphorin 3C in direct ELISAs and Western blots. In Western blots, less than 5% cross-reactivity with recombinant human (rh) Semaphorin 3A, rhSemaphorin 3B, rhSemaphorin 6A, recombinant mouse (rm) Semaphorin 3E, rmSemaphorin 3F, rmSemaphorin 6A, and rmSemaphorin 7A is observed.
Source	Polyclonal Sheep IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse Semaphorin 3C Gln24-Ser751 (Arg48Ala, Arg52Ala) Accession # Q62181
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

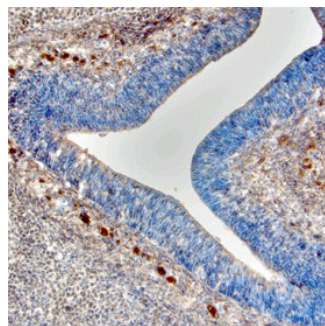
APPLICATIONS

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	0.1 µg/mL	Recombinant Mouse Semaphorin 3C Fc Chimera, Truncated
Immunocytochemistry	5-15 µg/mL	See Below
Immunohistochemistry	5-15 µg/mL	See Below

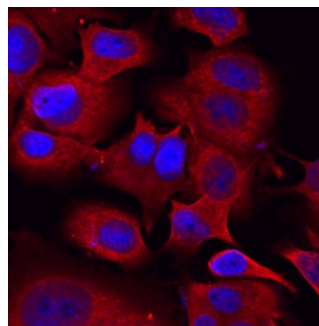
DATA

Immunohistochemistry



Semaphorin 3C in Mouse Embryo. Semaphorin 3C was detected in immersion fixed frozen sections of mouse embryo (15 d.p.c.) using Sheep Anti-Mouse Semaphorin 3C Antigen Affinity-purified Polyclonal Antibody (Catalog # AF1728) at 1.7 µg/mL overnight at 4 °C. Tissue was stained using the Anti-Sheep HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS019) and counterstained with hematoxylin (blue). Specific labeling was localized to the plasma membrane of mesenchymal cells. View our protocol for [Chromogenic IHC Staining of Frozen Tissue Sections](#).

Immunocytochemistry



Semaphorin 3C in MCF-7 Human Cell Line. Semaphorin 3C was detected in immersion fixed MCF-7 human breast cancer cell line using Sheep Anti-Mouse Semaphorin 3C Antigen Affinity-purified Polyclonal Antibody (Catalog # AF1728) at 10 µg/mL for 3 hours at room temperature. Cells were stained using the NorthernLights™ 557-conjugated Anti-Sheep IgG Secondary Antibody (red; Catalog # NL010) and counterstained with DAPI (blue). Specific staining was localized to cytoplasm. View our protocol for [Fluorescent ICC Staining of Cells on Coverslips](#).

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	<p>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</p> <ul style="list-style-type: none"> ● 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.

BACKGROUND

Semaphorin 3C (Sema 3C; previously semaE) is one of six Class 3 secreted semaphorins which share 40-50% amino acid (aa) identity. Class 3 semaphorins are potent chemorepellents that function in axon and/or vascular guidance during development, and may be upregulated in tumor progression (1, 2). The 751 amino acid (aa) mouse Sema3C is highly modular. It contains a 20 aa signal sequence, an ~500 aa N-terminal Sema domain that forms a β -propeller structure similar to that found in integrin molecules, a cysteine knot, a furin-type cleavage site, an Ig-like domain, and a C-terminal basic domain (1-3). Covalent dimerization plus cleavage at the C-terminus are required for activity of class 3 semaphorins (4). Mouse Sema 3C shares at least 95% aa identity with human, rat, cow and dog Sema 3C, and 89% and 75% aa sequence identity with chick and zebrafish Sema 3C, respectively. Type 3 semaphorins transduce signals through transmembrane plexins, either directly or by binding associated neuropilin receptors (1, 2). Sema 3C signaling is transduced by Plexin-D1 indirectly via neuropilin-1 or neuropilin-2 receptors (5). Sema 3C is expressed in all somitic motor neurons, in lung buds and in cardiac neural crest cells during development (1, 5-8). Sema 3C activates integrins in certain cells so, in addition to its repulsive activities, it sometimes acts as a chemoattractant (6, 9). In the developing nervous system, this chemoattraction appears to complement Sema 3A repulsion in adjacent cell layers (1, 6, 7). Sema 3C also provides an attractive force opposing Sema 6A and Sema 6B to guide migration of neural crest endothelial cells to the cardiac outflow tract (10). Consequently, defects in aortic arch formation occur when Sema 3C or Plexin-D1 genes or Sema 3C-neuropilin interactions are disrupted (5, 11, 12).

References:

1. Hinck, L. (2004) *Dev. Cell* **7**:783.
2. Neufeld, G. *et al.* (2005) *Front. Biosci.* **10**:751.
3. Gherardi, E. *et al.* (2004) *Curr. Opin. Struct. Biol.* **14**:669.
4. Adams, R.H. *et al.* (1997) *EMBO J.* **16**:6077.
5. Gitler, A.D. *et al.* (2004) *Dev. Cell* **7**:107.
6. Bagnard, D. *et al.* (1998) *Development* **125**:5043.
7. Cohen, S. *et al.* (2005) *Eur. J. Neurosci.* **21**:1767.
8. Puschel, A. W. *et al.* (1995) *Neuron* **14**:941.
9. Herman, J.G. and G.G. Meadows (2007) *Int. J. Oncol.* **30**:1231.
10. Toyofuku, T. *et al.* (2008) *Dev. Biol.* **321**:251.
11. Feiner, L. *et al.* (2001) *Development* **128**:3061.
12. Gu, C. *et al.* (2003) *Dev. Cell* **5**:45.