

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse SEMA 3A
Source	Recombinant Monoclonal Rabbit IgG Clone # 2701F
Purification	Protein A or G purified from cell culture supernatant
Immunogen	Chinese Hamster Ovary cell line, CHO-derived mouse SEMA 3A Asn21-Lys747 Accession # O08665
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.

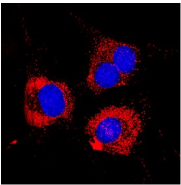
APPLICATIONS

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.

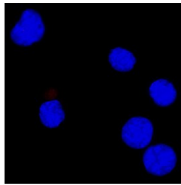
	Recommended Concentration	Sample
Immunocytochemistry	3-25 µg/mL	Immersion fixed C2C12 mouse myoblast cell line

DATA

Immunocytochemistry



Positive (C2C12 cells)



Negative (K562 cells)

Semaphorin 3A in C2C12 Mouse Cell Line. Semaphorin 3A was detected in immersion fixed C2C12 mouse myoblast cell line (positive staining) and K562 human chronic myelogenous leukemia cell line (negative staining) using Rabbit Anti-Mouse Semaphorin 3A Monoclonal Antibody (Catalog # MAB10903) at 3 µg/mL for 3 hours at room temperature. Cells were stained using the NorthernLights™ 557-conjugated Anti-Rabbit IgG Secondary Antibody (red; Catalog # NL004) and counterstained with DAPI (blue). Specific staining was localized to cytoplasm. Staining was performed using our protocol for Fluorescent ICC Staining of Non-adherent Cells.

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	<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 3A (Sema3A; previously sem D, sema III or collapsin) is one of six Class 3 secreted semaphorins which share ~40-50% amino acid (aa) identity (1-3). Class 3 semaphorins are potent chemorepellents that function in axon and/or vascular guidance during development (2, 3). The 772 aa mouse Sema3C 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 PSI domain, a furin-type cleavage site, an Ig-like domain, and a C-terminal basic domain (3, 4). Covalent dimerization plus cleavage at the C-terminus are required for activity of class 3 semaphorins (5, 6). The 95 kDa mature mouse Sema3A shares at least 95% aa identity with human, rat, equine and canine Sema3A, and 90% and 86% aa identity with chick and zebrafish Sema3A, respectively. Type 3 semaphorins transduce signals through transmembrane plexins, either directly or by binding associated neuropilin receptors (3). Sema3A signaling is transduced by plexin A1-4, indirectly via neuropilin-1 (3). Sema3A activity is mediated by small GTPases that influence actin rearrangement and integrin activity (7-9). It is important in developmental organization of central and peripheral nerves, including those in heart, lung, kidneys, bones, teeth, and visual and olfactory systems (1, 2, 10, 11). Gradients of Sema3A repel axons, but attract dendrites (11, 12). Sema3A affect vasculogenesis by inhibiting integrin function and, with Sema3F, promoting apoptosis of endothelial cells (3, 9, 12). It is thought to suppress cancer-related angiogenesis (3). In the immune system, Sema3A influences T cell proliferation, migration, response to activation, and interactions with dendritic cells (7, 13). It negatively regulates platelet activation (14). Expression of Sema3A in relevant parts of the nervous system may be increased in Alzheimer's disease, multiple sclerosis, ischemia and schizophrenia (2).

References:

1. Puschel, A.W. *et al.* (1995) *Neuron* **14**:941.
2. Roth, L. *et al.* (2009) *Cell. Mol. Life Sci.* **66**:649.
3. Neufeld, G and O. Kessler (2008) *Nat. Rev. Cancer* **8**:632.
4. Gherardi, E. *et al.* (2004) *Curr. Opin. Struct. Biol.* **14**:669.
5. Adams, R. H. *et al.* (1997) *EMBO J.* **16**:6077.
6. Klosterman, A. *et al.* (1998) *J. Biol. Sci.* **273**:7326.
7. Lepelletier, Y. *et al.* (2006) *Eur. J. Immunol.* **36**:1782.
8. Schlomann, U. *et al.* (2009) *J. Cell Sci.* **122**:2034.
9. Serini, G. *et al.* (2003) *Nature* **424**:391.
10. Ieda, M. *et al.* (2007) *Nat. Med.* **13**:604.
11. Chen, G. *et al.* (2008) *Nat. Neurosci.* **11**:36.
12. Guttmann-Raviv, N. *et al.* (2007) *J. Biol. Chem.* **282**:26294.
13. Lepelletier, Y. *et al.* (2007) *Proc. Natl. Acad. Sci. USA* **104**:5545.
14. Kashiwagi, H. *et al.* (2005) *Blood* **106**:913.