

DESCRIPTION

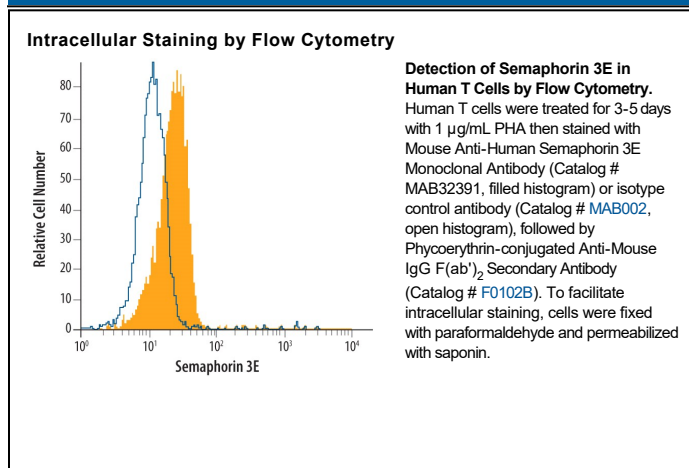
Species Reactivity	Human
Specificity	Detects human Semaphorin 3E in direct ELISAs. In direct ELISAs, approximately 15% cross-reactivity with recombinant human (rh) Semaphorin 3B is observed and no cross-reactivity with rhSemaphorin 6A is observed.
Source	Monoclonal Mouse IgG ₁ Clone # 400513
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human Semaphorin 3E Thr25-Ser775 (Arg557Ala and Arg560Ala) Accession # O15041
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
Intracellular Staining by Flow Cytometry	0.25 µg/10 ⁶ cells	See Below
CyTOF-ready	Ready to be labeled using established conjugation methods. No BSA or other carrier proteins that could interfere with conjugation.	

DATA



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	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <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 3E (Sema3E; previously SemaH) is a 90-95 kDa member of the Class 3 (secreted) semaphorins which, in human, share 40-50% amino acid (aa) sequence identity. Class 3 semaphorins are potent chemorepellents that function in axon guidance and/or vascular tip cell guidance during development (1). Sema3E is highly expressed in developing somites, where it acts as a repulsive cue for PlexinD1-expressing endothelial cells of adjacent intersomitic vessels (2, 3). 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. This is accompanied by 14 conserved cysteine residues and one or more N-glycosylation sites are thought critical for forming the secondary structure (4). C-terminal to the Sema domain, Sema3E has a consensus sequence for furin cleavage which, when used, creates a 61 kDa form that does not dimerize, and is highly expressed in tumor cell lines with metastatic potential (5, 6). Further C-terminal are a cysteine-knot plexin/semaphorin/integrin (PSI) domain, an Ig-like domain, a cysteine for dimerization and a basic domain containing another furin cleavage site. Dimerization and cleavage at the C-terminal site are required for repulsing activity of class 3 semaphorins (7). Human Sema3E shares 90%, 85% and 57% aa sequence identity with mouse, bovine and canine Sema3E, respectively. Like other semaphorins, Sema3E signaling is transduced by a transmembrane Plexin dimer, which also has a Sema domain and is coupled to kinase pathways. Unlike other Class 3 semaphorins, Sema3E binds directly to its plexin and does not require interaction with a neuropilin for activity (7). Genetic disruption of either Sema3E or PlexinD1 creates mouse mutants with excessive and disorganized vascular growth and branching, indicating the importance of this ligand-receptor pair for vascular guidance (3, 8).

References:

1. Eichmann, A. *et al.* (2005) *Genes Dev.* **19**:1013.
2. Cohen, S. *et al.* (2005) *Eur. J. Neurosci.* **21**:1767.
3. Gu, C. *et al.* (2005) *Science* **307**:265.
4. Gherardi, E. *et al.* (2004) *Curr. Opin. Struct. Biol.* **14**:669.
5. Christensen, C. *et al.* (1998) *Cancer Res.* **58**:1238.
6. Christensen, C. *et al.* (2005) *Cancer Res.* **65**:6167.
7. Adams, R. H. *et al.* (1997) *EMBO J.* **16**:6077.
8. Gitler, A. D. *et al.* (2004) *Dev. Cell* **7**:107.