

## Plexin Isoform Antibody Sampler Kit



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Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Plexin A1 Antibody	3813	40 µl	211 kDa	Rabbit
Plexin A2 (D42B5) Rabbit mAb	5658	40 µl	212 kDa	Rabbit IgG
Plexin A3 (D2G12) Rabbit mAb	5512	40 µl	207 kDa	Rabbit IgG
Plexin A4 (C5D1) Rabbit mAb	3816	40 µl	212 kDa	Rabbit IgG
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat

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### Description

The Plexin Isoform Antibody Sampler Kit provides an economical means of evaluating Plexin A1, A2, A3 and A4 protein expression. The kit contains enough primary antibody to perform four western miniblots experiments per primary antibody.

### Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at -20°C. Do not aliquot the antibody.

### Background

Class 3 secreted semaphorin (Sema3A) is a chemorepellent that acts upon a wide variety of axons. As such, it induces a dramatic redistribution and depolymerization of actin filaments that results in growth cone collapse. Plexins are single membrane-spanning signaling proteins encompassing Plexin A1, A2, A3, and A4. Plexins form a complex with neuropilin-1 and -2 and the cell adhesion protein L1 to form a functional semaphorin receptor (1,2). The GTPase Rnd1 binds to the cytoplasmic domain of Plexin A1 to trigger cytoskeletal collapse. In contrast, the GTPase RhoD blocks Rnd1-mediated Plexin A1 activation and repulsion of sympathetic axons by Sema3A (3). Sema6A is a ligand for Plexin A2. Both Sema6A and Plexin A2 knock-out mice have a granule cell migration defect, where cells remain in the molecular layer. Furthermore, Plexin A2 also controls nucleus-centrosome coupling that modulates cell migration (4). Both Plexin A3 and A4 mediate the responses to class 3 semaphorins in sensory and sympathetic neurons. In particular, Plexin A4 is responsible for signaling of Sema 3A via neuropilin-1, while Plexin A3 is responsible for signaling of Sema 3F via neuropilin-2 (5).

### Background References

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2. Takahashi, T. and Strittmatter, S.M. (2001) *Neuron* 29, 429-39.
3. Zanata, S.M. et al. (2002) *J Neurosci* 22, 471-7.
4. Renaud, J. et al. (2008) *Nat Neurosci* 11, 440-9.
5. Yaron, A. et al. (2005) *Neuron* 45, 513-23.

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