10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

Anti-Synaptobrevin (VAMP) Antibody

Our Anti-Synaptobrevin (VAMP) primary antibody from PhosphoSolutions is mouse monoclonal. It detects
Catalog # AN1566

Product Information

Application	WB, IHC
Primary Accession	P23763
Reactivity	Rat, Pig
Host	Mouse
Clonality	Monoclonal
Isotype	IgM
Clone Names	SP10
Calculated MW	12902

Additional Information

Gene ID	6843
Other Names	SYB antibody, SYB1 antibody, Synaptobrevin 1 antibody, VAMP antibody, VAMP1 antibody, Vesicle associated membrane protein 1 antibody
Target/Specificity	Synaptobrevin (aka VAMP) is an integral membrane protein of synaptic vesicles that plays a major role in the formation of larger SNARE complexes, along with SNAP-25 and syntaxin. Synaptobrevin has been shown to be essential for two fast synapse-specific membrane trafficking processes: fast exocytosis for neurotransmitter release and fast endocytosis that mediates rapid recycling of synaptic vesicles (Deak et al., 2004). Decreased levels of synaptobrevin in human hippocampus and cortex have been correlated with cognitive deficits in Alzheimer's disease (Sze et al., 2000).
Dilution	WB~~1:1000 IHC~~1:100~500
Format	IgM Purified culture supernatant
Storage	Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	Anti-Synaptobrevin (VAMP) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.
Shipping	Blue Ice

Background

Synaptobrevin (aka VAMP) is an integral membrane protein of synaptic vesicles that plays a major role in the formation of larger SNARE complexes, along with SNAP-25 and syntaxin. Synaptobrevin has been shown to be essential for two fast synapse-specific membrane trafficking processes: fast exocytosis for

neurotransmitter release and fast endocytosis that mediates rapid recycling of synaptic vesicles (Deak et al., 2004). Decreased levels of synaptobrevin in human hippocampus and cortex have been correlated with cognitive deficits in Alzheimer's disease (Sze et al., 2000).