



## BoNT/E (BE3): sc-51782

### BACKGROUND

Botulism is a rare but serious paralytic illness caused by a nerve toxin, which is produced by the anaerobic bacillus *Clostridium botulinum*. This neuromuscular disorder occurs through a complex series of molecular events, ultimately ending with the arrest of acetylcholine (ACh) release and flaccid paralysis. Botulinum neurotoxin type E, also referred to as BoNT/E, cleaves synaptosomal-associated protein (SNAP-25) at the C-terminal domain releasing a 26-mer peptide. This peptide product may act as an excitation-secretion uncoupling peptide (ESUP) to inhibit vesicle fusion which causes a long (at least 3 weeks) halt of ACh release after the cleavage of SNAP-25. BoNT/E also inhibits glutamate release and blocks the spike activity of pyramidal neurons. BoNT/E treatment reduces both focal and generalized kainic acid-induced seizures and also prevents the neuronal loss and long-term cognitive deficits that are associated with these seizures.

### REFERENCES

1. Barron, A.L., et al. 1954. *Clostridium botulinum* type E toxin and toxoid. *Can. J. Microbiol.* 1: 108-117.
2. Lawrence, G.W., et al. 1996. Distinct exocytotic responses of intact and permeabilised chromaffin cells after cleavage of the 25 kDa synaptosomal-associated protein (SNAP-25) or synaptobrevin by botulinum toxin A or B. *Eur. J. Biochem.* 236: 877-886.
3. Ferrer-Montiel, A.V., et al. 1998. The 26-mer peptide released from SNAP-25 cleavage by botulinum neurotoxin E inhibits vesicle docking. *FEBS Lett.* 435: 84-88.
4. Sadoul, K., et al. 1998. SNAP-23 is not cleaved by botulinum neurotoxin E and can replace SNAP-25 in the process of insulin secretion. *J. Biol. Chem.* 272: 33023-33027.
5. Vaidyanathan, V.V., et al. 1999. Proteolysis of SNAP-25 isoforms by botulinum neurotoxin types A, C, and E: domains and amino acid residues controlling the formation of enzyme-substrate complexes and cleavage. *J. Neurochem.* 72: 327-337.
6. Washbourne, P., et al. 1999. Botulinum neurotoxin E-insensitive mutants of SNAP-25 fail to bind VAMP but support exocytosis. *J. Neurochem.* 73: 2424-2433.
7. Blanes-Mira, C., et al. 2001. Thermal stabilization of the catalytic phosphorylation of a single tyrosine residue. *Biochemistry* 40: 2234-2242.
8. Agarwal, R., et al. 2005. Analysis of active site residues and structural studies: Glu335Gln is an apoenzyme. *Biochemistry* 44: 8291-8302.
9. Costantin, L., et al. 2005. Antiepileptic effects of botulinum neurotoxin E. *J. Neurosci.* 25: 1943-1951.

### SOURCE

BoNT/E (BE3) is a mouse monoclonal antibody raised against BoNT/E of *Clostridium botulinum* origin.

### RESEARCH USE

For research use only, not for use in diagnostic procedures.

### PRODUCT

Each vial contains 100 µg IgG<sub>1</sub> in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

### APPLICATIONS

BoNT/E (BE3) is recommended for detection of BoNT/E of *Clostridium botulinum* origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000).

Molecular Weight of BoNT/E: 156 kDa.

### STORAGE

Store at 4° C, **\*\*DO NOT FREEZE\*\***. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

### PROTOCOLS

See our web site at [www.scbt.com](http://www.scbt.com) for detailed protocols and support products.