



alomone labs

Molecular Tools for the Neuroscience Community

DATA SHEETS

Certificate of Analysis

Headquarters: Alomone Labs Ltd. Har Hotzvim Hi-Tech Park P.O. Box 4287, Jerusalem 91042, Israel.

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PRODUCT # C-670

LOT # CN-07

CERTIFICATE OF ANALYSIS

ω -Conotoxin MVIIA

(Conus magus)

M.W.: 2639.20 daltons.¹

Sequence: CKGKG AKCSR LMYDC CTGSC RSGKC

Purity: > 99% by HPLC.

Solubility: Any aqueous buffer.

Reconstitution:

The peptide concentration and identification were determined by amino acid analysis. Each vial contains 50 μ g of unbuffered protein. Dissolving of 50 μ g in 19ml of any conventional buffer gives a stock solution of 1 μ M.

Before dissolving the toxin, the tube should first be centrifuged, to concentrate the lyophilized toxin in the bottom of the tube. After centrifuging, the toxin must be dissolved into a stock solution using distilled water, or an appropriate buffer (see below), to a concentration of 10⁻⁵-10⁻⁶M. After preparing the stock solution, it should be divided into aliquots and can be stored this way for up to three months at -20°C.

Storage and Stability:

Lyophilized form: 2-3 weeks at room temperature.

One year at -20° C.

Liquid form: Up to two weeks at 4° C.

Six months at -20° C.

Known action:

ω -Conotoxin MVIIA, specifically blocks Ca_v2.2 (α 1B, N-type) channels¹. The effect of the toxin is modulated by Ca_v β auxiliary subunit² and by voltage (i.e. it is more potent for inactivated channels)³.



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Bioassay:

This lot was tested to confirm its ability to inhibit K⁺-induced ³H-GABA release in hippocampus in vivo⁴. This effect was with high affinity (50% block, 200 nM). The toxin was used to inhibit synaptic transmission in several peripheral preparations^{5, 6, 7}. And it binds with high affinity to rat neocortical membranes⁸. It blocked cloned Cav2.2 channels transiently expressed in tsa-201⁹ and in *Xenopus* oocytes³.

References:

1. Olivera, B. M. *et al.* (1987) *Biochemistry* **26(8)**, 2086.
2. Luchian, T. (2001) *Biochim. Biophys. Acta.* **1512(2)**, 329.
3. Stocker, J. W. *et al.* (1997) *J. Neurosci* **17(9)**, 3002.
4. Newcomb, R. *et al.* (1994) *Brain. Res.* **638(1-2)**, 95.
5. Vega, T. *et al.* (1995) *Eur. J. Pharmacol.* **276(3)**, 231.
6. Hirata, H. *et al.* (1997) *Eur. J. Pharmacol.* **321(2)**, 217.
7. Sanger, G. J. *et al.* (2000) *Eur. J. Pharmacol.* **388(1)**, 89.
8. Stoehr, S. J. *et al.* (1993) *Neurosci. Lett.* **161(1)**, 113.
9. Feng, Z. P. *et al.* (2001) *J. Biol. Chem.* **276(19)**, 15278.