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Produktinformation

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- Laborgeräte & Service

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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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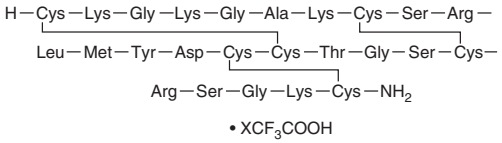
PRODUCT INFORMATION

Ziconotide (trifluoroacetate salt)

Item No. 23913

CAS Registry No.: 1660960-77-9
Formal Name: L-cysteinyl-L-lysylglycyl-L-lysylglycyl-L-alanyl-L-lysyl-L-cysteinyl-L-seryl-L-arginyl-L-leucyl-L-methionyl-L-tyrosyl-L-α-aspartyl-L-cysteinyl-L-cysteinyl-L-threonylglycyl-L-seryl-L-cysteinyl-L-arginyl-L-serylglycyl-L-lysyl-L-cysteinamide, cyclic (1→16),(8→20),(15→25)-*tris*(disulfide), trifluoroacetate salt
Synonyms: ω-Conotoxin MVIIA, SNX-111
MF: $C_{102}H_{172}N_{36}O_{32}S_7 \bullet XCF_3COOH$
FW: 2,639.1
Purity: ≥95%
Supplied as: A lyophilized powder
Storage: -20°C
Stability: ≥2 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

Ziconotide (trifluoroacetate salt) is supplied as a lyophilized powder. A stock solution may be made by dissolving the ziconotide (trifluoroacetate salt) in water. The solubility of ziconotide (trifluoroacetate salt) in water is approximately 1 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

Ziconotide is a synthetic version of ω-conopeptide MVIIA, a peptide toxin originally found in the venom of the fish-eating marine snail *C. magus*, that blocks N-type calcium channels in rat brain membranes (K_ds = 1.1-18 pM; IC_{50}s = 2-55 pM).[1,2] It blocks high-voltage-activated calcium currents in rat superior cervical ganglion neurons (IC_{50} = 32 nM) as well as depolarization-induced norepinephrine release by rat peripheral sympathetic efferent neurons and in rat hippocampus (IC_{50}s = 1.2 and 5.5 nM, respectively).[1] Intrathecal administration of ziconotide inhibits formalin-induced flinch responses and increases the paw withdrawal threshold in the paw pressure test in rats (ED_{50}s = 0.11 and 0.60 µg, respectively), indicating antinociceptive effects.[3] It decreases latency to tail withdrawal in a hot plate test in a rat model of sciatic chronic constriction injury.[1] Ziconotide also reduces infarct volume post ischemia in a rat model of transient focal ischemia.[4]

References

1. McGivern, J.G. Ziconotide: A review of its pharmacology and use in the treatment of pain. *Neurophyschiatr. Dis. Treat.* **3(1)**, 69-85 (2007).
2. Olivera, B.M., Cruz, L.J., de Santos, V., *et al.* Neuronal calcium channel antagonists. Discrimination between calcium channel subtypes using ω-conotoxin from *Conus magus* venom. *Biochemistry* **26(8)**, 2086-2090 (1987).
3. Wang, Y.X., Gao, D., Pettus, M., *et al.* Interactions of intrathecally administered ziconotide, a selective blocker of neuronal N-type voltage-sensitive calcium channels, with morphine on nociception in rats. *Pain* **84(2-3)**, 271-281 (2000).
4. Zhao, Q., Smith, M.L., and Siesjö, B.K. The omega-conopeptide SNX-111, an N-type calcium channel blocker, dramatically ameliorates brain damage due to transient focal ischaemia. *Acta. Physiol. Scand.* **150(4)**, 459-461 (1994).

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