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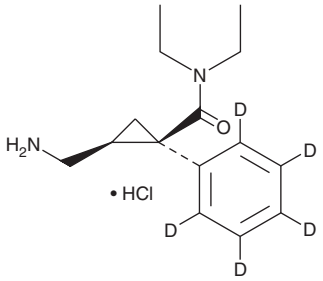
PRODUCT INFORMATION

Milnacipran-d_5 (hydrochloride)

Item No. 31981

Formal Name:	(1R,2S)-2-(aminomethyl)-N,N-diethyl-1-(phenyl-d_5)cyclopropane-1-carboxamide, monohydrochloride
MF:	$C_{15}H_{17}D_5N_2O \bullet HCl$
FW:	287.8
Chemical Purity:	≥98% (Milnacipran)
Deuterium Incorporation:	≥99% deuterated forms (d_1-d_5); ≤1% d_0
Supplied as:	A solid
Storage:	-20°C
Stability:	≥2 years



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

Laboratory Procedures

Milnacipran-d_5 (hydrochloride) is intended for use as an internal standard for the quantification of milnacipran (Item No. 23837) by GC- or LC-MS. The accuracy of the sample weight in this vial is between 5% over and 2% under the amount shown on the vial. If better precision is required, the deuterated standard should be quantitated against a more precisely weighed unlabeled standard by constructing a standard curve of peak intensity ratios (deuterated *versus* unlabeled).

Milnacipran-d_5 (hydrochloride) is supplied as a solid. A stock solution may be made by dissolving the milnacipran-d_5 (hydrochloride) in the solvent of choice, which should be purged with an inert gas. Milnacipran-d_5 (hydrochloride) is soluble in DMSO and methanol.

Description

Milnacipran is an orally bioavailable serotonin and norepinephrine reuptake inhibitor (SNRI).[1] It selectively inhibits the human serotonin transporter and norepinephrine transporter over the dopamine transporter (IC_{50}s = 420, 77, and 6,100 nM, respectively). It also selectively inhibits sodium-dependent serotonin (Item No. 14332) and norepinephrine (Item No. 16673) uptake over dopamine (Item No. 21992) uptake in rat cerebral cortical synaptosomes (IC_{50}s = 28.0, 29.6, and >10,000 nM, respectively).[2] Milnacipran is an antagonist of the serotonin (5-HT) receptor subtype 5-HT_{3A} as well as nicotinic acetylcholine receptors (nAChRs; IC_{50}s = 63.5 and 14.3 µM, respectively) but does not inhibit other 5-HT, adrenergic, dopamine, muscarinic acetylcholine, histamine, NMDA, sigma, opioid, or GABA receptors (K_is = >10,000 nM).[2,3] *In vivo*, milnacipran (30 mg/kg) increases withdrawal threshold and latency in response to tactile and heat stimulation, respectively, in nerve-ligated mice.[4] Formulations containing milnacipran have been used in the treatment of fibromyalgia pain.

References

1. Chen, C., Dyck, B., Fleck, B.A., *et al.* Studies on the SAR and pharmacophore of milnacipran derivatives as monoamine transporter inhibitors. *Bioorg. Med. Chem.* **18(4)**, 1346-1349 (2008).
2. Mochizuki, D., Tsujita, R., Yamada, S., *et al.* Neurochemical and behavioural characterization of milnacipran, a serotonin and noradrenaline reuptake inhibitor in rats. *Psychopharmacology (Berl)* **162(3)**, 323-332 (2002).
3. Ueta, K., Suzuki, T., Uchida, I., *et al. In vitro* inhibition of recombinant ligand-gated ion channels by high concentrations of milnacipran. *Psychopharmacology (Berl)* **175(2)**, 241-246 (2004).
4. Suzuki, T., Ueta, K., Tamagaki, S., *et al.* Antiallodynic and antihyperalgesic effect of milnacipran in mice with spinal nerve ligation. *Anesth. Analg.* **106(4)**, 1309-1315 (2008).

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