

Product Data Sheet

Product Name: Conivaptan-d4

Cat. No.: GC47120

Chemical Properties

Cas. No. 1129433-63-1

SMILES O=C(NC1=C([2H])C([2H])=C(C(N2CCC(N=C(C)N3)=C3C4=C2C=CC=C4)=O)C([2H])=C1[2H])C(C=CC=C5)=C5C6=CC=CC=C6

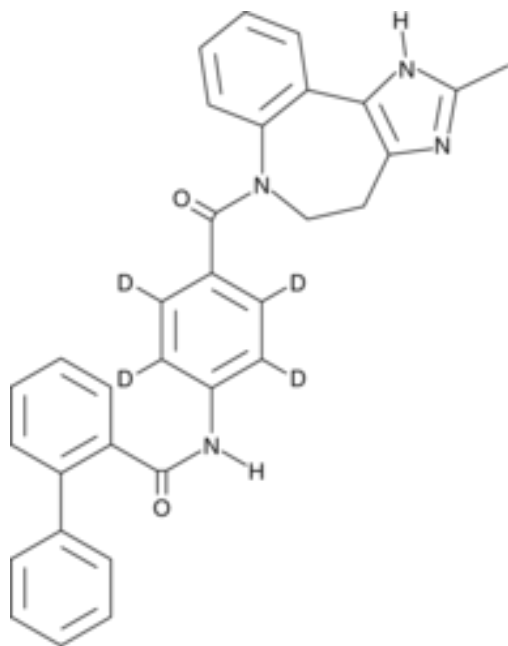
Formula C32H22D4N4O2 M.Wt 502.6

Solubility DMSO: soluble Storage Store at -20°C

General tips For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.

Structure



Background

Conivaptan-d₄ is intended for use as an internal standard for the quantification of conivaptan by GC- or LC-MS. Conivaptan is an antagonist of the arginine vasopressin (AVP) receptors V_{1A} and V₂ (K_is

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

Address: 10292 Central Ave. #205, Montclair, CA, USA

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= 0.48 and 3.04 nM for rat liver V_{1A} and kidney V₂, respectively).¹ It also competitively inhibits oxytocin binding to rat uterine oxytocin receptors (K_i = 44 nM) but has no effect on AVP binding to anterior pituitary V_{1B} receptors at concentrations up to 100 μ M in a radioligand binding assay. Conivaptan suppresses AVP-induced increases in intracellular calcium in vascular smooth muscle cells (VSMCs) *in vitro* and the pressor response in pithed rats. Conivaptan (0.01-0.3 mg/kg, i.v.) increases urine output and decreases urine osmolality in dehydrated conscious rats in a dose-dependent manner. It also reduces brain edema and blood-brain barrier disruption in a mouse experimental stroke model.²

1. Tahara, A., Tomura, Y., Wada, K.-I., et al. Pharmacological profile of YM087, a novel potent nonpeptide vasopressin V_{1A} and V₂ receptor antagonist, *in vitro* and *in vivo*. *J. Pharmacol. Exp. Ther.* 282(1)301-308(1997)
2. Zeynalov, E., Jones, S.M., Seo, J.W., et al. Arginine-vasopressin receptor blocker conivaptan reduces brain edema and blood-brain barrier disruption after experimental stroke in mice. *PLoS One* 10(8)e0136121(2015)

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