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- Expressversand

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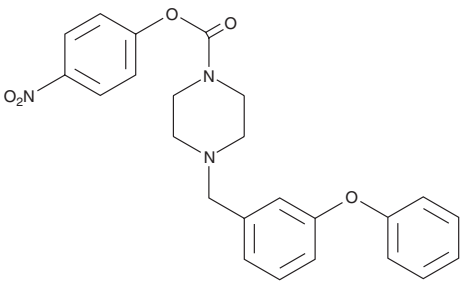
PRODUCT INFORMATION

JZL 195

Item No. 13668

Sold under license from The Scripps Research Institute

CAS Registry No.: 1210004-12-8
Formal Name: 4-nitrophenyl-4-(3-phenoxybenzyl)piperazine-1-carboxylate
MF: $C_{24}H_{23}N_3O_5$
FW: 433.5
Purity: ≥98%
UV/Vis.: λ_{max}: 273 nm
Supplied as: A crystalline 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

JZL 195 is supplied as a crystalline solid. A stock solution may be made by dissolving the JZL 195 in the solvent of choice. JZL 195 is soluble in organic solvents such as DMSO and dimethyl formamide, which should be purged with an inert gas. The solubility of JZL 195 in these solvents is approximately 1.25 and 5 mg/ml, respectively.

If aqueous stock solutions are required for biological experiments, they can best be prepared by diluting the organic solvent into aqueous buffers or isotonic saline. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. We do not recommend storing the aqueous solution for more than one day.

Description

Fatty acid amide hydrolase (FAAH) and monoacylglycerol lipase (MAGL) mediate the hydrolysis of the endocannabinoids arachidonoyl ethanolamide (AEA) and 2-arachidonoyl glycerol (2-AG), respectively. JZL 195 is a potent inhibitor of both FAAH and MAGL (IC_{50}s = 2 and 4 nM, respectively).[1] It poorly inhibits neuropathy target esterase and ABHD6 and does not inhibit other brain serine hydrolases. JZL 195 displays time-dependent inhibition of FAAH and MAGL *in vivo*, consistent with a covalent mechanism of activation.[1] The *in vivo* inhibitory actions of JZL 195 against FAAH and MAGL are comparable to those of the selective inhibitors PF-3845 (Item No. 13279) and JZL 184 (Item No. 13158), respectively.[1] Through its inhibitory actions, JZL 195 simultaneously augments brain levels of AEA and 2-AG, producing antinociceptive, cataleptic, and hypomotility effects like those produced by direct CB_1 agonists.[1]

Reference

1. Long, J.Z., Nomura, D.K., Vann, R.E., *et al.* Dual blockade of FAAH and MAGL identifies behavioral processes regulated by endocannabinoid crosstalk *in vivo*. *PNAS* **106(48)** 20270-20275 (2009).

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