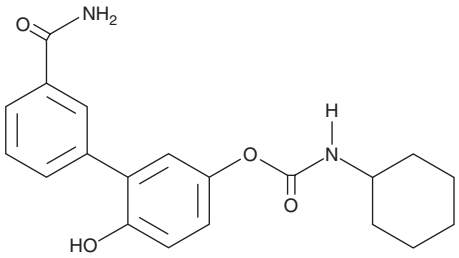
PRODUCT INFORMATION

URB937

Item No. 10674

CAS Registry No.:	1357160-72-5
Formal Name:	N-cyclohexyl-carbamic acid, 3'-(aminocarbonyl)-6-hydroxy[1,1'-biphenyl]-3-yl ester
MF:	$C_{20}H_{22}N_2O_4$
FW:	354.4
Purity:	≥95%
UV/Vis.:	λ_{max}: 215, 298 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥4 years



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

Laboratory Procedures

URB937 is supplied as a crystalline solid. A stock solution may be made by dissolving the URB937 in the solvent of choice. URB937 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide, which should be purged with an inert gas. The solubility of URB937 in these solvents is approximately 10, 15, and 10 mg/ml, respectively.

URB937 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, URB937 should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. URB937 has a solubility of approximately 0.1 mg/ml in a 1:10 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

URB937 is a potent fatty acid amide hydrolase (FAAH) inhibitor (IC_{50} = 26.8 nM, *in vitro*) that does not penetrate the blood-brain barrier, thus preventing arachidonoyl ethanolamide (AEA; Item No. 90050) deactivation only in peripheral tissues.[1] Its ED_{50} value for FAAH inhibition in brain is 200-fold higher than the ED_{50} value for FAAH inhibition in liver when administered systemically in mice (40 mg/kg *versus* 0.2 mg/kg, respectively). Subcutaneous administration of URB937 reduces acetic acid-induced writhing in mice with an ED_{50} value of 0.1 mg/kg. A single 1 mg/kg injection of URB937 sufficiently attenuates behavioral responses elicited in mouse models of neuropathic and inflammatory pain. As a peripherally-specific FAAH inhibitor, URB937 may offer an alternative approach to pain therapy devoid of unwanted central effects.

Reference

1. Clapper, J.R., Moreno-Sanz, G., Russo, R., *et al.* Anandamide suppresses pain initiation through a peripheral endocannabinoid mechanism. *Nat. Neurosci.* **13(10)**, 1265-1270 (2010).

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