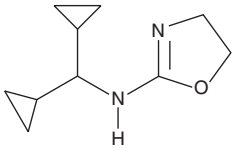
PRODUCT INFORMATION

Rilmenidine

Item No. 32879

CAS Registry No.:	54187-04-1
Formal Name:	N-(dicyclopropylmethyl)-4,5-dihydro-2-oxazolamine
Synonym:	NSC 664312, Oxaminozoline
MF:	$C_{10}H_{16}N_2O$
FW:	180.2
Purity:	≥95%
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

Rilmenidine is supplied as a crystalline solid. A stock solution may be made by dissolving the rilmenidine in the solvent of choice, which should be purged with an inert gas. Rilmenidine is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide. The solubility of rilmenidine in these solvents is approximately 30 mg/ml.

Rilmenidine is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, rilmenidine should first be dissolved in ethanol and then diluted with the aqueous buffer of choice. Rilmenidine has a solubility of approximately 0.14 mg/ml in a 1:6 solution of ethanol:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Rilmenidine is an antihypertensive agent.[1] It binds to imidazole receptors in bovine rostral ventrolateral medulla homogenates (K_i = 6.1 nM), as well as α_2-adrenergic receptors in bovine prefrontal cortex homogenates (K_i = 87 nM). Rilmenidine induces hypotension and bradycardia in anesthetized rats (ED_{50}s = 0.25 and 0.35 mg/kg, respectively). It also reduces mean arterial pressure and renal sympathetic nerve activity in a rabbit model of renal hypertension induced by a renal artery clip when administered at a dose of 2.5 mg/kg.[2] Rilmenidine (1 µM) increases levels of LC3-II, a marker of autophagy, in PC12 cells.[3] Formulations containing rilmenidine have been used in the treatment of hypertension.

References

1. Gomez, R.E., Ernsbarger, P., Feinland, G., *et al*. Rilmenidine lowers arterial pressure via imidazole receptors in brainstem C1 area. Eur. *J. Pharmacol.* **195(2)**, 181-191 (1991).
2. Burke, S. L., Evans, R.G., and Head, G. A. Effects of chronic sympatho-inhibition on renal excretory function in renovascular hypertension. *J. Hypertens.* **29(5)**, 945-952 (2011).
3. Williams, A., Sarkar, S., Cuddon, P., *et al*. Novel targets for Huntington's disease in an mTOR-independent autophagy pathway. *Nat. Chem. Biol.* **4(5)**, 295-305 (2008).

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