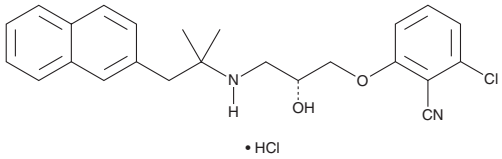
PRODUCT INFORMATION

NPS 2143 (hydrochloride)

Item No. 17903

CAS Registry No.: 324523-20-8
Formal Name: 2-chloro-6-[(2R)-3-[[1,1-dimethyl-2-(2-naphthalenyl) ethyl]amino]-2-hydroxypropoxy]-benzonitrile, monohydrochloride
Synonym: SB-262470A
MF: $C_{24}H_{25}ClN_2O_2 \bullet HCl$
FW: 445.4
Purity: ≥98%
UV/Vis.: λ_{max}: 225 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

NPS 2143 (hydrochloride) is supplied as a crystalline solid. A stock solution may be made by dissolving the NPS 2143 (hydrochloride) in the solvent of choice. NPS 2143 (hydrochloride) is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of NPS 2143 (hydrochloride) in ethanol is approximately 2 mg/ml and approximately 30 mg/ml in DMSO and DMF.

NPS 2143 (hydrochloride) is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, NPS 2143 (hydrochloride) should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. NPS 2143 (hydrochloride) has a solubility of approximately 0.25 mg/ml in a 1:3 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Calcium-sensing receptor (CaSR) is a G protein-coupled receptor that is involved in calcium homeostasis.[1,2] Activation of CaSR on parathyroid cells decreases the secretion of parathyroid hormone (PTH), which in turn reduces blood calcium concentration.[1,2] NPS 2143 is a potent and selective antagonist of CaSR (IC_{50} = 43 nM) that stimulates PTH secretion from parathyroid cells *in vitro* (EC_{50} = 39 nM).[3,4] It is effective *in vivo*, causing a sustained increase in plasma PTH in rats when given intravenously.[3,4] Daily oral administration of NPS 2143 to osteopenic ovariectomized rats results in a dramatic increase in bone turnover, as well as increased circulating PTH levels.[3]

References

1. Nemeth, E.F. *J. Mol. Endocrinol.* **29**, 15-21 (2002).
2. Filopanti, M., Corbetta, S., Barbieri, A.M., *et al. Clin. Cases Miner. Bone Metab.* **10(3)**, 162-165 (2013).
3. Gowen, M., Stroup, G.B., Dodds, R.A., *et al. J. Clin. Invest.* **105**, 1595-1604 (2000).
4. Nemeth, E.F., DelMar, E.G., Heaton, W.L., *et al. JPET* **299(1)**, 323-331 (2001).

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