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Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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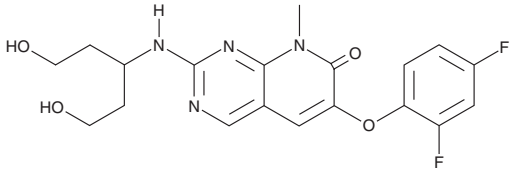
PRODUCT INFORMATION

Pamapimod

Item No. 20971

CAS Registry No.:	449811-01-2
Formal Name:	6-(2,4-difluorophenoxy)-2-[[3-hydroxy-1-(2-hydroxyethyl)propyl]amino]-8-methyl-pyrido[2,3-d]pyrimidin-7(8H)-one
Synonyms:	R 1503, Ro 4402257
MF:	$C_{19}H_{20}F_2N_4O_4$
FW:	406.4
Purity:	≥98%
UV/Vis.:	λ_{max}: 222, 299, 352 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

Pamapimod is supplied as a crystalline solid. A stock solution may be made by dissolving the pamapimod in the solvent of choice. Pamapimod is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of pamapimod in ethanol is approximately 0.33 mg/ml and approximately 30 mg/ml in DMSO and DMF.

Pamapimod is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, pamapimod should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. Pamapimod 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

Pamapimod is a potent inhibitor of p38α MAP kinase (IC_{50} = 14 nM).[1] It displays over 30-fold selectivity for p38α over p38β, has no activity against p38δ or p38γ, and has limited activity against a panel of 350 other kinases. Pamapimod blocks LPS-induced TNF-α production by monocytes and IL-1β generation in whole blood, and it inhibits spontaneous TNF-α release from synovial explants from patients with rheumatoid arthritis.[2,3] It is orally bioavailable and abrogates inflammation and bone loss in an animal model of arthritis and pain in a rat model of hyperalgesia.[2] Pamapimod suppresses the expression of inflammation-associated genes in primary, IL-1β-stimulated human chondrocytes.[4]

References

1. Goldstein, D.M., Soth, M., Gabriel, T., *et al. J. Med. Chem.* **54(7)**, 2255-2265 (2011).
2. Hill, R.J., Dabbagh, K., Phippard, D., *et al. J. Pharmacol. Exp. Ther.* **327(3)**, 610-619 (2008).
3. Fehr, S., Unger, A., Schaeffeler, E., *et al. Cell. Physiol. Biochem.* **36(6)**, 2237-2249 (2015).
4. Joos, H., Albrecht, W., Laufer, S., *et al. Br. J. Pharmacol.* **160(6)**, 1252-1262 (2010).

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