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Produktinformation

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- Diagnostik & molekulare Diagnostik
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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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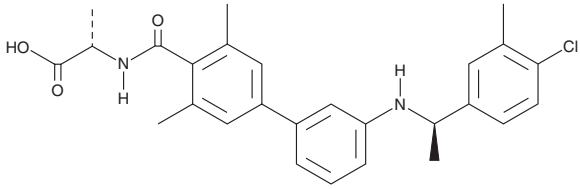
PRODUCT INFORMATION

NIBR0213

Item No. 21513

CAS Registry No.:	1233332-14-3
Formal Name:	N-[[3'-[[(1R)-1-(4-chloro-3-methylphenyl) ethyl]amino]-3,5-dimethyl[1,1'-biphenyl]-4-yl]carbonyl]-L-alanine
MF:	$C_{27}H_{29}ClN_2O_3$
FW:	465.0
Purity:	≥98%
UV/Vis.:	λ_{max}: 212, 249, 316 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

NIBR0213 is supplied as a crystalline solid. A stock solution may be made by dissolving the NIBR0213 in the solvent of choice. NIBR0213 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide, which should be purged with an inert gas. The solubility of NIBR0213 in these solvents is approximately 25 mg/ml.

NIBR0213 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, NIBR0213 should first be dissolved in ethanol and then diluted with the aqueous buffer of choice. NIBR0213 has a solubility of approximately 0.15 mg/ml in a 1:5 solution of ethanol:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

NIBR0213 is an orally bioavailable, potent, and selective antagonist of sphingosine-1-phosphate receptor-1 ($S1P_1$) with IC_{50} values of 2.5 and 2.0 nM for the $hS1P_1$ in a Ca^{2+} mobilization assay and GTPγS assay, respectively.[1] It is inactive at $S1P_2$, $S1P_3$, and $S1P_4$ receptors (IC_{50}s = >20, >10, and >10 µM, respectively in a Ca^{2+} mobilization assay). $S1P_1$ plays a key role in the immune system, regulating lymphocyte egress from lymphoid tissues into the circulation.[2] In rats, NIBR0213 (30 mg/kg) reduces peripheral blood lymphocytes by 75-85% for up to 24 hours.[1] In a mouse model of experimental autoimmune encephalitis (EAE), it significantly reduces disease severity up to 26 days, when administered at 30 mg/kg twice per day for three days, then 60 mg/kg twice per day. However, in a rat model of adjuvant-induced arthritis, chronic administration of 30 mg/kg induces pulmonary damage and chronic lung inflammation.[3]

References

1. Quancard, J., Bollbuck, B., Janser, P., *et al.* A potent and selective $S1P_1$ antagonist with efficacy in experimental autoimmune encephalomyelitis. *Chem. Biol.* **19(9)**, 1142-1151 (2012).
2. Huwiler, A., Kolter, T., Pfeilschifter, J., *et al.* Physiology and pathophysiology of sphingolipid metabolism and signaling. *Biochim. Biophys. Acta.* **1485(2-3)**, 63-99 (2000).
3. Bigaud, M., Dincer, Z., Bollbuck, B., *et al.* Pathophysiological consequences of a break in S1P1-dependent homeostasis of vascular permeability revealed by S1P1 competitive antagonism. *PLoS One* **11(12)**, e0168252 (2016).

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