Product Data Sheet

Product Name: HNMPA
Cat. No.: GC13988

Chemical Properties

Cas. No.	132541-52-7		
Chemical Name	P-(hydroxy-2-naphthalenylmethyl)-phosphonic acid		
SMILES	OC(P(O)(O)=O)C1=CC2=CC=CC=C2C=C1		
Formula	$C_{11}H_{11}O_4P$	M.Wt	238.2
Solubility	≤20mg/ml in ethanol;1mg/ml in DMSO;15mg/ml in dimethyl formamide	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure	HNMPA		

Background

HNMPA is a tyrosine kinase inhibitor that inhibited both the receptor serine and tyrosine phosphorylation, including insulin receptor tyrosine kinase activity [1].

Receptor tyrosine kinases (RTKs) are the high-affinity cell surface receptors for growth factors, cytokines, and hormones. The insulin receptor is one of a number of growth factor receptors with intrinsic tyrosine kinase activity that can be activated upon ligands binding [1].

HNMPA (Hydroxy-2-naphthalenylmethyl Phosphonic Acid) is a tyrosine kinase inhibitor that blocks receptor serine and tyrosine phosphorylation. HNMPA does not affect protein kinase C or cyclic AMP-dependent protein kinase activities. HNMPA inhibited tyrosine kinase activity of autophosphorylated insulin receptor towards poly (Glu4, Tyr) or insulin receptor-(1155-1165) peptide by 82% and 81%, respectively. HNMPA also inhibited autophosphorylation of insulin receptors by 13% + 4.6% in the presence of insulin.

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HNMPA not only inhibited insulin receptor tyrosine phosphorylation but also effectively decreased insulin receptor serine phosphorylation [1]. In β-cells exposed to high glucose, HNMPA was able to further increase the exe-4-induced insulin secretion [2].

References:

[1]. Baltensperger K, Lewis RE, Woon CW, et al. Catalysis of serine and tyrosine autophosphorylation by the human insulin receptor. Proc Natl Acad Sci U S A. 1992 Sep 1;89(17):7885-9.

[2]. Moon MJ, Kim HY, Park S, et al. Insulin contributes to fine-tuning of the pancreatic beta-cell response to glucagon-like peptide-1. Mol Cells. 2011 Oct;32(4):389-95.

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