Product Data Sheet

Product Name: AG957
Cat. No.: GC11737

Chemical Properties

Cas. No.	140674-76-6		
Chemical Name	4-[[(2,5-dihydroxyphenyl)methyl]amino]-benzoic acid, methyl ester		
SMILES	OC1=C(CNC2=CC=C(C(OC)=O)C=C2)C=C(O)C=C1		
Formula	$C_{15}H_{15}NO_4$	M.Wt	273.3
Solubility	≤5mg/ml in DMSO	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	AG957		

Background

AG957 is a tyrosine phosphorylation inhibitor that targets transforming Bcr-Abl fusion proteins (p185Bcr-Abl, p210Bcr-Abl), as well as normal c-Abl [1].

The abl proto-oncogene is expressed in all cell types. The protein p140c-abl is localized both to the nucleus and the cytoplasm. The abl transforming proteins p160gag-abl, p210bcr-abl, and p185bcr-abl are exclusively cytoplasmic proteins. The intrinsic protein tyrosine kinase activity of the transforming abl proteins is higher than that of the normal p140c-abl [1].

AG957 is a tyrosine phosphorylation inhibitor. AG957 inhibited human p185Bcr-Abl, p210Bcr-Abl and normal c-Abl with IC50 values of 4.3, 1, and 7.1 μM, respectively. AG957 inhibited mouse normal c-Abl with IC50 value of 6 μM. AG957 also inhibited epidermal growth factor receptor with IC50 value of 0.25 μM. In chronic myelogenous leukemia (CML) K562 cells, AG957 inhibited p210bcr-abl kinase activity and cell growth. AG957 at 20 μM also inhibited DNA synthesis as early as 2 h by 60% [2]. In K562 cells,

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AG957, a selective Bcr-Abl inhibitor, blocked taxol-induced PKCi activation and sensitized these cells to taxol-induced apoptosis [3].

In mice, AG957, the c-Abl inhibitor, attenuated LPS-induced pulmonary permeability [4].

References:
[1]. Anafi M, Gazit A, Gilon C, et al. Selective interactions of transforming and normal abl proteins with ATP, tyrosine-copolymer substrates, and tyrphostins. J Biol Chem. 1992 Mar 5;267(7):4518-23.
[2]. Kaur G, Gazit A, Levitzki A, et al. Tyrphostin induced growth inhibition: correlation with effect on p210bcr-abl autokinase activity in K562 chronic myelogenous leukemia. Anticancer Drugs. 1994 Apr;5(2):213-22.
[3]. Jamieson L, Carpenter L, Biden TJ, et al. Protein kinase Ciota activity is necessary for Bcr-Abl-mediated resistance to drug-induced apoptosis. J Biol Chem. 1999 Feb 12;274(7):3927-30.
[4]. Fu P, Usatyuk PV, Lele A, et al. c-Abl mediated tyrosine phosphorylation of paxillin regulates LPS-induced endothelial dysfunction and lung injury. Am J Physiol Lung Cell Mol Physiol. 2015 May 15;308(10):L1025-38.

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