Product Data Sheet

Product Name: SB 202474
Cat. No.: GC16019

Chemical Properties

Cas. No.	172747-50-1		
Chemical Name	4-[4-ethyl-2-(4-methoxyphenyl)-1H-imidazol-5-yl]-pyridine		
SMILES	CCC1=C(C2=CC=NC=C2)N=C(C3=CC=C(OC)C=C3)N1		
Formula	$C_{17}H_{17}N_3O$	M.Wt	279.3
Solubility	Soluble in methanol;Soluble 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	SB 202474		

Background

SB 202474 is a structural analog of SB 202190 and SB 203580 that is used as a negative control in researches of p38 inhibition. SB 202190 and SB 203580 are potent and selective inhibitors of the MAP kinases p38α and p38β [1][2][3].

Mitogen-activated protein kinases (MAPKs) are ser/thr-specific protein kinases that regulate gene expression, proliferation, differentiation, cell survival and apoptosis. Three most widely characterized MAPK subfamilies are ERK1/2, JNK and p38MAPK, of which JNK and p38MAPK are identified as a stress-activated protein kinase (SAPK) that primarily mediates inflammatory response and promotes cell death [3].

SB 202474 is a structural analog of SB 202190 and SB 203580 that is used as a negative control in researches of p38 inhibition. In 3T3-L1 adipocytes and L6 myotubes, SB203580 but not SB202474 prevented insulin-stimulated glucose transport [2].

Pretreatment with microinjection into the bilateral rostral ventrolateral medulla (RVLM)

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA

of SB203580 (2 nmol) significantly exacerbated the depressor effect and blunted the augmented power density of the LF component of SAP signals during the pro-life phase. SB203580 also significantly shortened the pro-life phase to 60 min. SB202474 (2 nmol) was ineffective against the phasic cardiovascular responses in the aCSF-control group or Mev-experimental group [3].

References:

[1]. Davies SP, Reddy H, Caivano M, et al. Specificity and mechanism of action of some commonly used protein kinase inhibitors. Biochem J. 2000 Oct 1;351(Pt 1):95-105.
[2]. Sweeney G, Somwar R, Ramlal T, et al. An inhibitor of p38 mitogen-activated protein kinase prevents insulin-stimulated glucose transport but not glucose transporter translocation in 3T3-L1 adipocytes and L6 myotubes. J Biol Chem. 1999 Apr 9;274(15):10071-8.
[3]. Chang AY. Pro-life role for c-Jun N-terminal kinase and p38 mitogen-activated protein kinase at rostral ventrolateral medulla in experimental brain stem death. J Biomed Sci. 2012 Nov 17;19:96.