

Product Data Sheet

Product Name: SP 600125

Cat. No.: GC15344

Chemical Properties

Cas. No. 129-56-6

Chemical Name dibenzo[cd,g]indazol-6(2H)-one

SMILES O=C1C2=CC=CC3=C2C(C4=CC=CC=C41)=NN3

Formula $C_{14}H_8N_2O$ M.Wt 220.23

Solubility $\geq 11\text{mg/mL}$ in DMSO, $\geq 2.56\text{ mg/mL}$ in EtOH with gentle warming Storage Desiccate at -20°C

General tips For obtaining a higher solubility , please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.

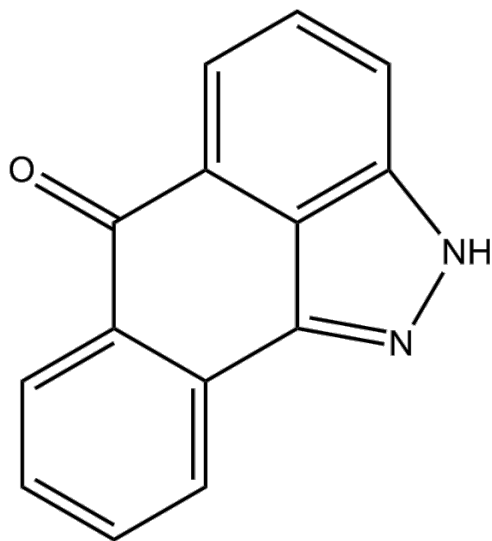
Caution: Product has not been fully validated for medical applications. For research use only.

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Structure



Protocol

Cell experiment [1]:

Cell lines OVCAR-3 cells

Preparation Method OVCAR3 cells were treated with SP600125(10-20) μ M for 24 hours.

Reaction SP600125(10-20) μ M ;24h
Conditions

Applications SP600125(10-20 μ M;24h) reduces PMA-dependent MMP-9 secretion and inhibits c-Jun phosphorylation.

Animal experiment [2]:

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Animal models MPTP-induced PD in mice

Preparation Method MPTP-induced PD in mice that they were given SP600125 (10/30/50 mg/kg;ip) once per day between days 1 and 6, respectively. Mice were sacrificed at day 10 after the final injection of MPTP.

Dosage form SP600125 (10/30/50 mg/kg)/ day; ip

Applications SP600125 inhibited MPTP-induced JNK activation and c-Jun phosphorylation in SNc;SP600125 prevented the reduction of dopamine concentrations in the striatum and attenuated behavioral impairment in MPTP-treated mice.

References:

- [1]. Shin M, Yan C, Boyd D. An inhibitor of c-jun aminoterminal kinase (SP600125) represses c-Jun activation, DNA-binding and PMA-inducible 92-kDa type IV collagenase expression. *Biochim Biophys Acta*. 2002 May 8;1589(3):311-6.
- [2]. Wang W, Shi L, Xie Y, Ma C, Li W, Su X, Huang S, Chen R, Zhu Z, Mao Z, Han Y, Li M. SP600125, a new JNK inhibitor, protects dopaminergic neurons in the MPTP model of Parkinson's disease. *Neurosci Res*. 2004 Feb;48(2):195-202.

Background

SP 600125 is an orally active, reversible, selective, ATP-competitive JNK inhibitor with IC₅₀ of 40, 40, and 90 nM for JNK1, JNK2, and JNK3, respectively. SP 600125 is commonly used in the research of ovarian cancer, tumors, Parkinson's disease (PD), breast cancer, and asthma^[1,2].

SP 600125 (10-20 µM; 24 h) reduces PMA-dependent MMP-9 secretion and inhibits c-Jun phosphorylation, and also inhibits PMA-stimulated MMP-9 promoter-driven luciferase reporter gene activity^[1]. SP 600125 inhibits c-Jun phosphorylation with an IC₅₀ of 5-10 µM. SP 600125 (5-10 µM, 24h) also inhibits LPS-induced increase COX-2 protein levels and PGE2 production^[3].

SP 600125 (10/30/50 mg/kg/day; ip) inhibits MPTP-induced JNK activation and c-Jun

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phosphorylation in the substantia nigra obturata (SNc) and prevents the decrease in dopamine concentration in the striatum and alleviates behavioral impairment in MPTP-treated mice^[2]. SP 600125(30mg/kg/day) can significantly reduce the expression of inflammatory cells in the bronchi and lungs under OVA stimulation^[4].

References:

- [1]. Shin M, Yan C, Boyd D. An inhibitor of c-jun aminoterminal kinase (SP600125) represses c-Jun activation, DNA-binding and PMA-inducible 92-kDa type IV collagenase expression. *Biochim Biophys Acta*. 2002 May 8;1589(3):311-6.
- [2]. Wang W, Shi L, Xie Y, Ma C, Li W, Su X, Huang S, Chen R, Zhu Z, Mao Z, Han Y, Li M. SP600125, a new JNK inhibitor, protects dopaminergic neurons in the MPTP model of Parkinson's disease. *Neurosci Res*. 2004 Feb;48(2):195-202.
- [3]. Nieminen R, Lahti A, Jalonen U, Kankaanranta H, Moilanen E. JNK inhibitor SP600125 reduces COX-2 expression by attenuating mRNA in activated murine J774 macrophages. *Int Immunopharmacol*. 2006 Jun;6(6):987-96.
- [4]. Wu HM, Fang L, Shen QY, Liu RY. SP600125 promotes resolution of allergic airway inflammation via TLR9 in an OVA-induced murine acute asthma model. *Mol Immunol*. 2015 Oct;67(2 Pt B):311-6.

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