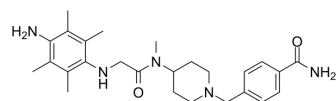


## SUN11602

Cat. No.:	HY-101493		
CAS No.:	704869-38-5		
Molecular Formula:	C <sub>26</sub> H <sub>37</sub> N <sub>5</sub> O <sub>2</sub>		
Molecular Weight:	451.6		
Target:	FGFR		
Pathway:	Protein Tyrosine Kinase/RTK		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	2 years
		-20°C	1 year



### SOLVENT & SOLUBILITY

#### In Vitro

DMSO : ≥ 37 mg/mL (81.93 mM)  
 \* "≥" means soluble, but saturation unknown.

	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
Preparing Stock Solutions	1 mM	2.2143 mL	11.0717 mL	22.1435 mL
	5 mM	0.4429 mL	2.2143 mL	4.4287 mL
	10 mM	0.2214 mL	1.1072 mL	2.2143 mL

Please refer to the solubility information to select the appropriate solvent.

#### In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline  
 Solubility: ≥ 2.5 mg/mL (5.54 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)  
 Solubility: ≥ 2.5 mg/mL (5.54 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil  
 Solubility: ≥ 2.5 mg/mL (5.54 mM); Clear solution

### BIOLOGICAL ACTIVITY

#### Description

SUN11602 is a novel aniline compound with basic fibroblast growth factor-like activity.

#### In Vitro

SUN11602 prevents glutamate-induced neuronal death in primary cultures of rat cerebrocortical neurons. SUN11602 increases the levels of CALB1 gene expression in cerebrocortical neurons<sup>[1]</sup>. SUN11602 exerts protective effects on hippocampal neurons through activation of FGFR1 and increases CalB expression<sup>[2]</sup>. SUN11602 promotes neurite outgrowth of primarily cultured rat hippocampal neurons<sup>[3]</sup>.  
 MCE has not independently confirmed the accuracy of these methods. They are for reference only.

### In Vivo

In WT mice, SUN11602 increases the levels of newly synthesized Calb in cerebrocortical neurons and suppresses the glutamate-induced rise in intracellular  $\text{Ca}^{2+}$ . This  $\text{Ca}^{2+}$ -capturing ability of Calb allows the neurons to survive severe toxic conditions of glutamate<sup>[1]</sup>. Oral administration of SUN11602 at the midpoint of A $\beta$ 1-40 and ibotenate injections attenuate short-term memory impairment in the Y-maze test, as well as spatial learning deficits in the water maze task. In addition, the SUN11602 treatment inhibits the increase of peripheral-type benzodiazepine-binding sites (PTBBS), which are a marker for gliosis<sup>[3]</sup>.

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## PROTOCOL

### Cell Assay <sup>[1]</sup>

Cerebrocortical neurons are pretreated with vehicle (Hanks' Balanced Salt Solution), SUN11602, bFGF, or the other growth factors for 24 h prior to the onset of glutamate toxicity. Subsequently, 10  $\mu\text{L}$  of the MTT solution (5 mg/mL) is added to each well (200  $\mu\text{L}$  of culture medium) of the microplates. Neurons in each well are then dried for 24 h, and 200  $\mu\text{L}$  of DMSO is poured into all of the wells in order to dissolve the reaction products thoroughly for the MTT assay<sup>[1]</sup>.

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

### Animal Administration <sup>[3]</sup>

Rats: SUN11602 (0.1, 1, and 10 mg/kg) is administered orally to the rat hippocampal-lesion model, once at 24 h after the A $\beta$ 1-40 injection. In the vehicle-treated groups, saline is administered instead of SUN11602<sup>[3]</sup>.

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## REFERENCES

[1]. Murayama N, et al. SUN11602, a novel aniline compound, mimics the neuroprotective mechanisms of basic fibroblast growth factor. ACS Chem Neurosci. 2013 Feb 20;4(2):266-76.

[2]. Murayama N, et al. SUN11602-induced hyperexpression of calbindin D-28k is pivotal for the survival of hippocampal neurons under neurotoxic conditions. Brain Res. 2015 Jan 12;1594:71-81.

[3]. Ogino R, et al. SUN11602 has basic fibroblast growth factor-like activity and attenuates neuronal damage and cognitive deficits in a rat model of Alzheimer's disease induced by amyloid  $\beta$  and excitatory amino acids. Brain Res. 2014 Oct 17;1585:159-66.

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

Tel: 609-228-6898

Fax: 609-228-5909

E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA