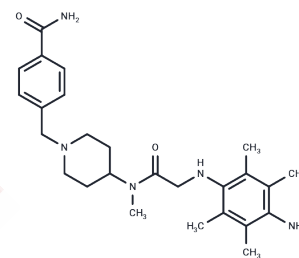


SUN11602

## Chemical Properties

CAS No. : 704869-38-5  
 Formula: C<sub>26</sub>H<sub>37</sub>N<sub>5</sub>O<sub>2</sub>  
 Molecular Weight: 451.6  
 Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year  
 Actual storage temperature shall be subject to the COA.



## Biological Description

Description	SUN11602, an aniline compound, mimics the neuroprotective mechanisms of basic fibroblast growth factor. It mimics the neuroprotective effects of bFGF and prevents glutamate-induced neuronal death. It also increases levels of CALB1 gene expression in cerebrocortical neurons and triggers phosphorylation of FGFR and ERK1/2.
Targets(IC50)	ERK,FGFR
In vitro	Physiological actions of SUN11602 mimic several phenomena of the neuroprotection that is induced by bFGF. SUN11602 plays a pivotal role in allowing primary cultured neurons to survive in adverse environments of glutamate toxicity and activating intracellular key molecules that are involved in the neuroprotective mechanisms. These actions are quite similar to those of bFGF. Such neuroprotective mechanisms are specific and distinctive to SUN11602 and bFGF and differs clearly from those of the other growth factors that are investigated. But unlike bFGF, SUN11602 can either directly or indirectly trigger the phosphorylation of the cytosolic domain of the FGFR without binding to the extracellular domain of the FGFR-1[1]. SUN11602 demonstrates no cell proliferative activity of somatic cells, unlike bFGF. SUN11602 significantly affects neuronal survival in adverse conditions through a FGFR1-mitogen-activated protein kinase/extracellular signal-regulated kinase-1/2 kinase (FGFR-1-MEK/ERK) signaling pathway[2].
In vivo	In WT mice, SUN11602 and bFGF increase the levels of newly synthesized Calb in cerebrocortical neurons and suppress the glutamate-induced rise in intracellular Ca <sup>2+</sup> . This Ca <sup>2+</sup> -capturing ability of Calb allows the neurons to survive severe toxic conditions of glutamate. In contrast, Calb levels remain unchanged in Calb <sup>-/-</sup> mice after exposure to SUN11602 or bFGF, and due to a loss of function of the gene, these neurons are no longer resistant to toxic conditions of glutamate[1]. Neuroprotective activities of SUN11602 and FGF-2 are due to exogenously induced hyperexpression of CalB in hippocampal neurons. The pharmacokinetic properties of SUN11602 appear to hold promise in terms of bioavailability (>65%) after oral administration in rodents (rats and mice) and dogs (beagles)[2].
Cell Research	Actinomycin D or cycloheximide is first added to the cultures, and, 2 h later, SUN11602 or bFGF is added. After a 24 h incubation, neurons in the cultures are exposed to 150 μM glutamate for another 24 h, and cell viability is determined by a (3-(4,5-dimethyl-2-thiazolyl)-2,5-diphenyl-2H tetrazolium bromide (MTT) assay.(Only for Reference)

## Solubility Information

Solubility	Ethanol: 9 mg/mL (19.93 mM),Sonication is recommended. DMSO: 40 mg/mL (88.57 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 2 mg/mL (4.43 mM),Sonication is recommended. <i>Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.</i>

## Preparing Stock Solutions

	1mg	5mg	10mg
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
50 mM	0.0443 mL	0.2214 mL	0.4429 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

## Reference

Murayama N, et al. ACS Chem Neurosci. 2013, 4(2):266-76.

Murayama N, et al. Brain Res. 2015, 1594:71-81.

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