

Tideglusib

Catalog No: tcsc0613



Available Sizes

Size: 10mg

Size: 50mg

Size: 100mg



Specifications

CAS No:

865854-05-3

Formula:

$C_{19}H_{14}N_2O_2S$

Pathway:

Stem Cell/Wnt;PI3K/Akt/mTOR

Target:

GSK-3;GSK-3

Purity / Grade:

>98%

Solubility:

DMSO : 12.5 mg/mL (37.38 mM; Need ultrasonic and warming)

Alternative Names:

NP-12;NP031112

Observed Molecular Weight:

334.39

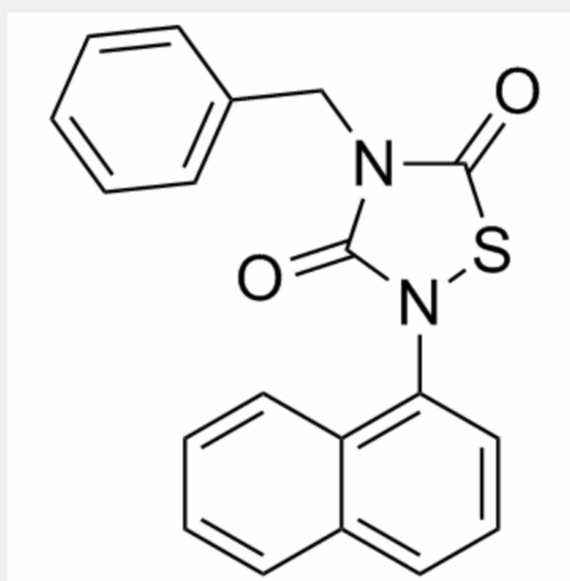
Product Description

Tideglusib is an irreversible **GSK-3** inhibitor with **IC₅₀** of 5 nM and 60 nM for **GSK-3β^{WT}** (1 h preincubation) and **GSK-3β^{C199A}** (1 h preincubation), respectively.

IC50 & Target: IC50: 5 nM (GSK-3β^{WT}), 60 nM (GSK-3β^{C199A})^[1]

In Vitro: Tideglusib (NP12) is a small heterocyclic thiadiazolidinone (TDZD) derivative, which is an ATP-non competitive inhibitor of GSK-3β with an IC₅₀ value in the micromolar range^[2]. Incubation of both astrocyte and microglial cultures with Tideglusib (NP031112) completely abrogates the induction of TNF-α and COX-2 expression after glutamate treatment. These effects of NP031112 are not caused by a loss of cell viability, because the 24 h exposure of astrocyte and microglial cells to this TDZD does not modify cell viability^[3].

In Vivo: Tideglusib (NP12) treatment correlates with an increase of 46% as an average in the inhibitory phosphorylation of GSK-3β at Ser-9 in the brains of APP^{SW}-tau^{VLW} mice, and the levels of the inactive form of the enzyme in NP12 treated mice are comparable to those found in wild-type littermate controls (p=0.893) (n=6-8 for each treatment). NP12 treatment results in significantly decreased phosphorylation at the putative GSK-3β-directed sites Ser-202 (CP13) and Ser-396/404 (PHF-1) in 15-month-old mice by more than 60% (p=0.023 and p=0.024, respectively)^[2]. Injection of Tideglusib (NP031112) (50 mg/kg) into the rat hippocampus dramatically reduces kainic acid-induced inflammation, as measured by edema formation using T2-weighted magnetic resonance imaging and glial activation and has a neuroprotective effect in the damaged areas of the hippocampus^[3].



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