Product Data Sheet

Product Name: Retigabine
Cat. No.: GC14029

Chemical Properties

Cas. No.	150812-12-7		
Chemical Name	(E)-ethyl hydrogen (2-amino-4-((4-fluorobenzyl)amino)phenyl)carbonimidate		
SMILES	CCO/C(O)=N/C1=C(N)C=C(NCC2=CC=C(F)C=C2)C=C1		
Formula	$C_{16}H_{18}FN_3O_2$	M.Wt	303.33
Solubility	≥ 12.95mg/mL 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	Retigabine		

Protocol

Cell experiment [1]:

Cell lines	PC12 cells
Preparation Method	PC12 cells were maintained in Dulbecco's modified Eagle medium (DMEM) supplemented with 5% fetal calf serum, 10% horse serum, and 50μg/ml penicillin/streptomycin in a humidified atmosphere containing 5% CO_2. Cells were seeded in 96-well plates at a density of 1×10^5 cells and cultured overnight. After 48h of treatment with l-glutamate (10mM) and 10μM of Retigabine, the cell viability was measured.

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Reaction Conditions	10μM; 48h
Applications	Retigabine treatment significantly enhanced the viability of PC12 cells exposed to l-glutamate.

Animal experiment [2]:

Animal models	Sprague-Dawley rat pups
Preparation Method	Sprague-Dawley rat pups (7 days old) were maintained in a temperature-controlled (21°C) room with a 12h light cycle. The mice in the treatment group were given an intraperitoneal injection of a 15mg/kg dose of Retigabine every four hours for a total of 12 hours. Control groups received equivalent volumes of saline vehicle (0.01ml/g body weight). Pups were perfused transcardially with ice-cold phosphate buffer, followed by 4% paraformaldehyde and brains were collected for analysis.
Dosage form	15mg/kg; every 4 hours for 12 hours; i.p.
Applications	Retigabine treatment promoted the apoptosis of brain neurons in newborn rats.

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References:
[1] Seyfried J, Evert B O, Rundfeldt C, et al. Flupirtine and retigabine prevent L-glutamate toxicity in rat pheochromocytoma PC 12 cells[J]. European journal of pharmacology, 2000, 400(2-3): 155-166.
[2] Brown L, Gutherz S, Kulick C, et al. Profile of retigabine-induced neuronal apoptosis in the developing rat brain[J]. Epilepsia, 2016, 57(4): 660-670.

Background

Retigabine is an anticonvulsant compound that activates low-threshold voltage-gated potassium channels [1]. Retigabine enhances potassium currents via destabilization of a closed conformation or stabilization of the open conformation of the potassium Kv7.2-7.3 channels[2]. Retigabine has been widely used to stabilize the resting membrane potential, reduce brain excitability, and enhance the current mediated by gamma-aminobutyric acid (GABA)[3].

In vitro, Retigabine treatment (10μM) for 48 hours prevented L-glutamate toxicity in PC12 cells, restored cell viability, and reduced the generation of reactive oxygen intermediates [4].

In vivo, Retigabine treatment via intraperitoneal injection at a dose of 15mg/kg every four hours for 12 hours significantly promoted the apoptosis of brain neurons in newborn rats[5].

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References:
[1] Splinter M Y. Ezogabine (retigabine) and its role in the treatment of partial-onset seizures: a review[J]. Clinical therapeutics, 2012, 34(9): 1845-1856. e1.
[2] Czuczwar P, Wojtak A, Cioczek-Czuczwar A, et al. Retigabine: the newer potential antiepileptic drug[J]. Pharmacological Reports, 2010, 62(2): 211-219.
[3] Stafstrom C E, Grippon S, Kirkpatrick P. Ezogabine (retigabine)[J]. Nature Reviews Drug Discovery, 2011, 10(10): 729-730.
[4] Seyfried J, Evert B O, Rundfeldt C, et al. Flupirtine and retigabine prevent L-glutamate toxicity in rat pheochromocytoma PC 12 cells[J]. European journal of pharmacology, 2000, 400(2-3): 155-166.
[5] Brown L, Gutherz S, Kulick C, et al. Profile of retigabine-induced neuronal apoptosis in the developing rat brain[J]. Epilepsia, 2016, 57(4): 660-670.

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