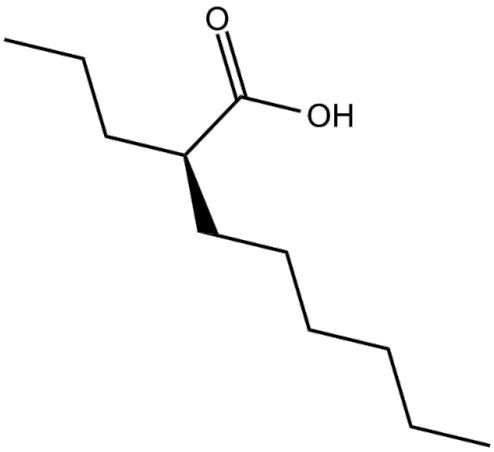
Product Data Sheet

Product Name: ONO 2506 (Arundic Acid)
Cat. No.: GC10652

Chemical Properties

Cas. No.	185517-21-9		
Chemical Name	(R)-2-propyloctanoic acid		
SMILES	OC([C@H](CCC)CCCCCC)=O		
Formula	$C_{11}H_{22}O_2$	M.Wt	186.29
Solubility	<18.63mg/ml in DMSO; <18.63mg/ml in ethanol	Storage	Store 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.		

Structure



Protocol

Cell experiment [1]:

Cell lines 1321N1 astrocyte cells

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Preparation Method	1321N1 astrocyte cells were maintained in DMEM/F12 medium supplemented with fetal bovine serum (FBS) and penicillin-streptomycin at 37°C in 95% humidified atmosphere/5% CO_2. The cells were pretreated with ONO 2506 50μM for 24 hours, then stimulated with Aβ25-35 (200μM) for 24 hours.
Reaction Conditions	50μM; 24 hours pretreatment
Applications	ONO 2506 ameliorated the gliotoxic effects of Aβ, increased cell viability, and decreased both intracellular and extracellular S100B levels.

Animal experiment [2]:

Animal models	Male C57BL/6 mice, 8 weeks of age
Preparation Method	Mice received four intraperitoneal injections of 1-Methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP; 20mg/kg) at 2-hour intervals. ONO 2506 at a dose of 30mg/kg was administered intraperitoneally 3, 4, 5, and 6 days after the last MPTP injection. The mice were killed 7 days after MPTP treatment for analysis.
Dosage form	30mg/kg; i.p.; administered on days 3, 4, 5, and 6 after the last MPTP injection

Caution: Product has not been fully validated for medical applications. For research use only.
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Applications	Delayed treatment with ONO 2506 ameliorated the severe reductions in densities of tyrosine hydroxylase (TH) immunoreactivity in the striatum and partly ameliorated the severe reductions in the number of neuronal nuclei (NeuN) immunoreactive neurons in the substantia nigra 7 days after MPTP treatment. ONO 2506 ameliorated the increases in number of glial fibrillary acidic protein (GFAP) immunoreactive astrocytes, S100β immunoreactive astrocytes, isolectin reactive microglia in both the striatum and substantia nigra, and nestin immunoreactive astrocytes in the striatum.

References:
[1] Keshavarz M, Farrokhi MR, Amiri A, et al. The contribution of S100B to the glioprotective effects of valproic and arundic acids. Iran J Basic Med Sci. 2019 May;22(5):557-562.
[2] Oki C, Watanabe Y, Yokoyama H, et al. Delayed treatment with arundic acid reduces the MPTP-induced neurotoxicity in mice. Cell Mol Neurobiol. 2008 May;28(3):417-30.

Background

ONO 2506 (Arundic Acid) is an orally active astrocyte modulator. ONO 2506 regulates astrocyte activation by inhibiting S-100β synthesis and upregulates EAAT1 expression by activating the Akt, ERK, and NF-κB pathways to reduce excitatory neurotoxicity. ONO 2506 can be used in research related to stroke and Alzheimer's disease[1-4].

In vitro, 1321N1 astrocyte cells were pretreated with ONO 2506 (50μM) for 24 hours, followed by stimulation with Aβ25-35 (200μM) for 24 hours. ONO 2506 significantly ameliorated the gliotoxic effects of Aβ, increased cell viability, and decreased both intracellular and extracellular S100B levels[5]. Mouse Müller cells were treated with ONO 2506 (0-100μM) for 14 days, or H4 human glioblastoma cells were treated with ONO 2506 (0-100μM) for 9 days. ONO 2506 significantly increased glutamate uptake activity, GLAST/EAAT1 mRNA and protein expression, and activated the EAAT1 promoter in H4 cells[6].

In vivo, ONO 2506 (2μg/μl; at a dose of 0.5% of body weight) was administered via intracerebroventricular injection in a rat model of intracerebral hemorrhage. ONO 2506 improved

motor dysfunction, reduced S100B levels, astrogliosis, and microglial activation, thereby decreasing the release of pro-inflammatory cytokines IL-1β and TNF-α and ROS production[7]. ONO 2506 (30mg/kg) was administered intraperitoneally on days 3, 4, 5, and 6 after the last injection of 1-Methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP; 20mg/kg; at 2-hour intervals; four times total) in C57BL/6 mice treated with MPTP. ONO 2506 ameliorated MPTP-induced neuronal damage in the striatum and substantia nigra and reduced astrogliosis and microglial activation[8].

References:

[1] Karki P, Hong P, Johnson J Jr, et al. Arundic Acid Increases Expression and Function of Astrocytic Glutamate Transporter EAAT1 Via the ERK, Akt, and NF-κB Pathways. Mol Neurobiol. 2018 Jun;55(6):5031-5046.
[2] Kato H, Kurosaki R, Oki C, et al. Arundic acid, an astrocyte-modulating agent, protects dopaminergic neurons against MPTP neurotoxicity in mice. Brain Res. 2004 Dec 24;1030(1):66-73.
[3] Fernandes RA, Ingle AB. Arundic acid a potential neuroprotective agent: biological development and syntheses. Curr Med Chem. 2013;20(18):2315-29.
[4] Asano T, Mori T, Shimoda T, et al. Arundic acid (ONO-2506) ameliorates delayed ischemic brain damage by preventing astrocytic overproduction of S100B. Curr Drug Targets CNS Neurol Disord. 2005 Apr;4(2):127-42.
[5] Keshavarz M, Farrokhi MR, Amiri A, et al. The contribution of S100B to the glioprotective effects of valproic and arundic acids. Iran J Basic Med Sci. 2019 May;22(5):557-562.
[6] Yanagisawa M, Aida T, Takeda T, et al. Arundic acid attenuates retinal ganglion cell death by increasing glutamate/aspartate transporter expression in a model of normal tension glaucoma. Cell Death Dis. 2015 Mar 19;6(3):e1693.
[7] Cordeiro JL, Neves JD, Nicola F, et al. Arundic Acid (ONO-2506) Attenuates Neuroinflammation and Prevents Motor Impairment in Rats with Intracerebral Hemorrhage. Cell Mol Neurobiol. 2022 Apr;42(3):739-751.
[8] Oki C, Watanabe Y, Yokoyama H, et al. Delayed treatment with arundic acid reduces the MPTP-induced neurotoxicity in mice. Cell Mol Neurobiol. 2008 May;28(3):417-30.