DPN

Cat. No.: HY-12452

CAS No.: 1428-67-7

Molecular Formula: $C_{15}H_{13}NO_2$

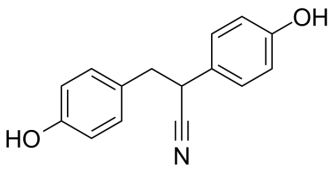
Molecular Weight: 239.27

Target: Estrogen Receptor/ERR; Apoptosis; Autophagy

Pathway: Others; Apoptosis; Autophagy

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (417.94 mM; Need ultrasonic)

H_2O : 0.67 mg/mL (2.80 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.1794 mL	20.8969 mL	41.7938 mL
5 mM	0.8359 mL	4.1794 mL	8.3588 mL
10 mM	0.4179 mL	2.0897 mL	4.1794 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (10.45 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (10.45 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (10.45 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

DPN (Diarylpropionitrile) is a non-steroidal estrogen receptor β (ERβ) selective ligand, with an EC_{50} of 0.85 nM. DPN has neuroprotective effects in a number of neurological diseases[1][2].

IC_{50} & Target

ERβ

0.85 nM (EC50)

In Vitro	DPN has a 70-fold ERα relative binding affinity selectivity, and it is a full ERα agonist with a 78-fold ERα potency selectivity (EC_{50}=0.85 nM for ERβ; EC_{50}=66 nM for ERα)[1]. DPN (10 nM) prevents morphological alterations from $Aβ_{1-42}$ (10 μM)-induced toxicity in cultured cortical neurons[2]. DPN (0.1-100 nM) decreases ROS levels in a non-dose response manner[2]. DPN (0.1-100 nM) significantly reduces $Aβ_{1-42}$-stimulated expression of Bax in a non-dose dependent manner[2]. DPN (0.1-100 nM) reduces activated IL-1 levels induced by $Aβ_{1-42}$ treatment on cultured cortical neurons[2]. DPN (0.1-100 nM) suppresses the $Aβ_{1-42}$-upregulated phosphorylation of JNK and p38[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	DPN (10 μg; s.c.; daily; for 11 days) increases swimming and decreases immobility in the FST, and increases TPH protein expression in the dorsal raphe nucleus (DR) in rat model[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Adult Sprague-Dawley female rats (220-250 g), ovariectomized animal models[3]
Dosage:	10 μg/rat
Administration:	Subcutaneous injections, daily, for 11 days
Result:	Increased swimming and decreased immobility in the FST.

CUSTOMER VALIDATION

- Cell Death Dis. 2021 Oct 5;12(10):907.
- Cell Death Dis. 2019 Jul 22;10(8):565.
- J Clin Endocrinol Metab. 2021 Feb 1;dgab020.
- Front Immunol. 2022 May 19;13:818173.
- Molecules. 2020 Apr 23;25(8):1960.

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REFERENCES

[1]. Suwanna N, et al. Neuroprotective effects of diarylpropionitrile against β-amyloid peptide-induced neurotoxicity in rat cultured cortical neurons. Neurosci Lett. 2014 Aug 22;578:44-9.

[2]. Meyers, M. J., et al. Estrogen Receptor-β Potency-Selective Ligands: Structure−Activity Relationship Studies of Diarylpropionitriles and Their Acetylene and Polar Analogues. Journal of Medicinal Chemistry, 2001. 44(24), 4230–4251.

[3]. Fuzhong Yang, et al. Physiological dosages of estradiol and diarylpropionitrile decrease depressive behavior and increase tryptophan hydroxylase expression in the dorsal raphe nucleus of rats subjected to the forced swim test. Neuroreport. 2019 Jan 16;30(

[4]. Sherry A. Said, et al. Effects of long-term dietary administration of estrogen receptor-beta agonist diarylpropionitrile on ovariectomized female ICR (CD-1) mice. GeroScience. 2018 Aug; 40(4): 393–403.

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

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