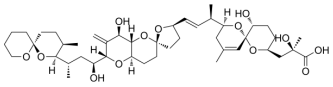
Okadaic acid

Cat. No.:	HY-N6785
CAS No.:	78111-17-8
Molecular Formula:	$C_{44}H_{68}O_{13}$
Molecular Weight:	805.00
Target:	Phosphatase; Apoptosis
Pathway:	Metabolic Enzyme/Protease; Apoptosis
Storage:	Solution, -20°C, 2 years



BIOLOGICAL ACTIVITY

Description

Okadaic acid, a marine toxin, is an inhibitor of protein phosphatases (PP). Okadaic acid has a significantly higher affinity for PP2A (IC_{50}=0.1-0.3 nM), and inhibits PP1 (IC_{50}=15-50 nM), PP3 (IC_{50}=3.7-4 nM), PP4 (IC_{50}=0.1 nM), PP5 (IC_{50}=3.5 nM), but does not inhibit PP2C. Okadaic acid increases of phosphorylation of a number of proteins by inhibiting PP, and acts a tumor promoter. Okadaic acid induces tau phosphorylation[1][2].

IC_{50} & Target

PP1 15-50 nM (IC_{50})	PP2A 0.1-0.3 nM (IC_{50})	PP3 3.7-4 nM (IC_{50})	PP4 0.1 nM (IC_{50})
PP5 3.5 nM (IC_{50})	PP2B ~4000 nM (IC_{50})	PP7 ≥1000 nM (IC_{50})	

In Vitro

Okadaic acid (0-100 nM; 24 h or 48 h) inhibits the proliferation of AGS, MNK-45, Caco 2 cells[3].
Okadaic acid (10 nM; 8 hours) increases Drp1 phosphorylation and mitochondrial fission in rat cortical neurons[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[3]

Cell Line:	AGS, MNK-45 and Caco 2 cell lines
Concentration:	0-100 nM
Incubation Time:	24 h or 48 h
Result:	Inhibited the proliferation of AGS, MNK-45, Caco 2 cells.

In Vivo

Okadaic acid can be used to induce Alzheimer's disease models[6].

Induction of Alzheimer's disease model[6]

- Background

  Okadaic acid is a Ser/Thr phosphatase enzyme inhibitor that causes Tau hyperphosphorylation and induces the

intracellular aggregation of neurofibrillary tangles.

- Specific Modeling Methods

  Mice: Wistar • male •-250-300 g

  Administration: 100μg • icv • single dose

- Modeling Indicators

  Molecular changes: Induced increases in $pTau^{ser396}$ in the hippocampus.

- Correlated Product(s): /
- Opposite Product(s): /

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Biomaterials. 2024 Aug 6:312:122749.
- Bone Res. 2025 Jan 2;13(1):3.
- Cancer Lett. 2021 Mar 3;S0304-3835(21)00101-4.
- Cell Commun Signal. 2024 Aug 7;22(1):391.
- Int J Biol Macromol. 2023 Jun 2;125171.

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REFERENCES

[1]. Natalia Dos Santos Tramontin, et al. Gold Nanoparticles Treatment Reverses Brain Damage in Alzheimer's Disease Model . Mol Neurobiol. 2020, 57, 2.

[2]. Kleppe R, et al. Cell Death Inducing Microbial Protein Phosphatase Inhibitors--Mechanisms of Action. Mar Drugs. 2015 Oct 22;13(10):6505-20.

[3]. Valdiglesias V, et al. Okadaic acid: more than a diarrheic toxin. Mar Drugs. 2013 Oct 31;11(11):4328-49.

[4]. del Campo M, et al. Okadaic acid toxin at sublethal dose produced cell proliferation in gastric and colon epithelial cell lines. Mar Drugs. 2013;11(12):4751-4760.

[5]. Cho MH, et al. Increased phosphorylation of dynamin-related protein 1 and mitochondrial fission in okadaic acid-treated neurons. Brain Res. 2012 May 15;1454:100-10.

[6]. Baker S, et al. A local insult of okadaic acid in wild-type mice induces tau phosphorylation and protein aggregation in anatomically distinct brain regions. Acta Neuropathol Commun. 2016;4:32.

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

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