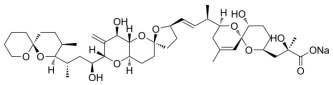
Okadaic acid sodium

Cat. No.:	HY-N6785A
CAS No.:	209266-80-8
Molecular Formula:	$C_{44}H_{67}NaO_{13}$
Molecular Weight:	826.98
Target:	Phosphatase; Apoptosis
Pathway:	Metabolic Enzyme/Protease; Apoptosis
Storage:	-20°C, sealed storage, away from moisture * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



BIOLOGICAL ACTIVITY

Description

Okadaic acid sodium, a marine toxin, is an inhibitor of protein phosphatases (PP). Okadaic acid (sodium) 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 sodium increases of phosphorylation of a number of proteins by inhibiting PP, and acts a tumor promoter. Okadaic acid sodium 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 sodium (0-100 nM; 24 h or 48 h) inhibits the proliferation of AGS, MNK-45, Caco 2 cells[3].
Okadaic acid sodium (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 sodium (100 μM; injected unilaterally to the lateral amygdala) induces Tau phosphorylation and protein aggregation in anatomically distinct brain regions 24 h post-injection[5].

Induction of alzheimer's disease model[6]

- Background

Okadaic acid sodium is a Ser/Thr phosphatase enzyme inhibitor that causes Tau hyperphosphorylation and induces the intracellular aggregation of neurofibrillary tangles.

- Specific Mmodeling Methods

  Mice: Wistar • male •? 250–300 g

  Administration: 100μg • icv • single dose

- Modeling Indicators

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

- Opposite Product(s):

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

CUSTOMER VALIDATION

- Cancer Lett. 2021 Mar 3;S0304-3835(21)00101-4.
- Int J Biol Macromol. 2023 Jun 2;125171.
- Int J Biochem Cell Biol. 2021, 106036.
- Biochem Biophys Res Commun. 2023 Nov 5, 680, Pages 127-134.
- bioRxiv. 2023 Jun 6.

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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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