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- Mindermengenzuschlag
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3BDO

Cat. No.: HY-U00434

CAS No.: 890405-51-3

Molecular Formula: $C_{18}H_{17}NO_5$

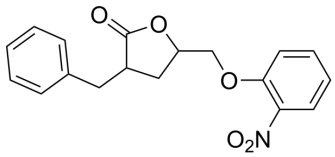
Molecular Weight: 327.33

Target: mTOR; Autophagy; Autophagy; Apoptosis

Pathway: PI3K/Akt/mTOR; Autophagy; Apoptosis

Storage:

Pure form	-20°C	3 years
	4°C	2 years
In solvent	-80°C	1 year
	-20°C	6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (305.50 mM)

Ethanol : 20 mg/mL (61.10 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.0550 mL	15.2751 mL	30.5502 mL
5 mM	0.6110 mL	3.0550 mL	6.1100 mL
10 mM	0.3055 mL	1.5275 mL	3.0550 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 (7.64 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (7.64 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.64 mM); Clear solution

BIOLOGICAL ACTIVITY

Description	3BDO is a new mTOR activator which can also inhibit autophagy.
IC_{50} & Target	mTOR
In Vitro	Phosphorylation of RPS6KB1 and EIF4EBP1 is significantly increased by 3BDO with vector alone but suppressed with FKBP1A

	overexpression. Rapamycin fails to decrease the phosphorylation of MTOR and RPS6KB1 in the presence of 3BDO. 3BDO suppresses the increase in MAP1LC3B puncta induced with rapamycin. 3BDO also inhibits the effect of rapamycin in HUVECs. The phosphorylation of Ser residues is decreased in HUVECs treated with 10 μM rapamycin, and 60 μM 3BDO reverses the phosphorylation. The results show that 3BDO suppresses the increased MAP1LC3B puncta number, MAP1LC3B-II level and decreased SQSTM1 protein level induced by rapamycin. 3BDO could dose- and time-dependently decrease FLJ11812 level in HUVECs. Overexpression of FLJ11812 reverses the inhibition of autophagy induced by 3BDO[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Immunofluorescence assay reveals that 3BDO treatment increases the level of p-p70S6K and decreases the protein level of ATG13 in plaque endothelium of mice. 3BDO does not affect the phosphorylation of mTOR direct downstream targets p70S6K and 4EBP1. As compare with controls, apoE$^{-/-}$ mice show inhibited endothelium autophagy and apoptosis with 3BDO treatment, so 3BDO protects against endothelium injury in atherosclerosis. 3BDO treatment stabilizes established atherosclerotic lesions in apoE$^{-/-}$ mice. In apoE$^{-/-}$mice, as compare with controls, with 3BDO treatment, the serum level of IL-6 and IL-8 is significantly decreased[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [1]	Total protein is obtained from HUVECs by using of IP lysis buffer after treatment with rapamycin (10 μM), 3BDO (60 μM) or both for 6 h. After centrifuging at 4°C, the supernatant is collected and incubated with protein A/G agarose beads and TIA1 antibody or normal mouse IgG as a control at 4°C overnight. The beads are washed 3 times with IP lysis buffer and then eluted with 4×SDS loading buffer. Ser phosphorylation is detected by western blot assay with Ser phosphorylation antibody [1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Cell Assay [1]	HUVECs are isolated from umbilical cords and cultured in M199 medium with 20% (v/v) fetal bovine serum and 10 IU/mL fibroblast growth factor 2 (FGF2) in a humidified incubator at 37°C with 5% CO_2. Cells up to passage 10 are used for experiments. When HUVECs are grown to 80% confluency, HUVECs are treated with DMSO or 60 μM 3BDO for 24 h, then total RNA is extracted[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Male apoE$^{-/-}$ mice (8 weeks old) are used in this study. ApoE$^{-/-}$ mice are fed an atherogenic diet (containing 21% fat and 0.15% cholesterol). To avoid the potential confounding effects of variation among batches of diet, a single batch is reserved and used throughout the experiment. Mice at 20 weeks older are divided into 3 groups for treatment (n=8 mice/group) for 8 weeks: control (DMSO), low-dose 3BDO (50 mg/kg/d; 3BDO-L) and high-dose 3BDO (100 mg/kg/d; 3BDO-H). The body weight of mice is measured every week during 3BDO injection. Blood samples are taken from the inferior vena cava, and animals are killed by exsanguination[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Death Differ. 2023 Jun 12.
- EBioMedicine. 2023 Jan 27;88:104444.
- Acta Biomater. 2018 Nov;81:278-292.
- Appl Mater Today. 2021, 101066.
- Biochem Pharmacol. 2020 Aug;178:114038.

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REFERENCES

[1]. Ge D, et al. Identification of a novel MTOR activator and discovery of a competing endogenous RNA regulating autophagy in vascular endothelial cells. Autophagy. 2014 Jun;10(6):957-71.

[2]. Peng N, et al. An activator of mTOR inhibits oxLDL-induced autophagy and apoptosis in vascular endothelial cells and restricts atherosclerosis in apolipoprotein $E^{-}/^{-}$ mice. Sci Rep. 2014 Jul 1;4:5519.

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