产品名称：MSDC 0160
产品别名：MSDC 0160

生物活性：

Description

MSDC 0160 act as an insulin sensitizer and a modulator of mitochondrial pyruvate carrier (MPC), a key controller of cellular metabolism that influences mTOR (mammalian target of rapamycin) activation. In Vitro: MSDC-0160 acts as insulin sensitizers without activating PPARγ. MSDC-0160 (10 μM) pretreatment (1 hour) prevents the MPP+ (10 μM)-induced loss of both tyrosine hydroxylase (TH)-immunoreactive differentiated Lund human mesencephalic (LUHMES) cells. MSDC-0160 protects only TH-immunoreactive neurons, which is consistent with the selected concentration of MPP+ primarily being toxic to dopamine neurons. In addition, MSDC-0160 counteracts both MPP+-induced shortening of neurite length and reduces branching in both LUHMES cells. MSDC-0160 (10 or 100 μM) prevents the loss of GFP-fluorescent dopaminergic neurons induced by MPP+ (0.75 mM) in nematodes (P =0.0001), whereas 1 μM MSDC-0160 does not. MSDC-0160 (10 μM) blocks LPS-induced increases in iNOS expression in BV2 cell lysates. MSDC-0160 is mainly to prevent the activation of mTOR produced by the metabolic changes rather than to directly inhibit mTOR kinase activity[1]. PPARγ sparing TZD, MSDC-0160, reduces resistance in the insulin/IGF-1 signaling pathway and restores IGF-1-induced akt phosphorylation. MSDC-0160 (10-20 μM) in conbination with IGF-1 prevents the loss of insulin content and maintains insulin secretion. Treatment of human islets with MSDC-0160 (1-50 μM) activates AMPK and downregulates mTOR. MSDC-0160 (1-50 μM) treatment maintains human β-cell phenotype[2]. The combined treatment with PPARγ ligands (MSDC 0160) and γ-radiation synergistically induces caspase-dependent apoptotic cell death, and PPARγ ligands significantly enhance the γ-radiation-induced DNA damage response in a PPARγ-independent manner[3]. In Vivo: MSDC-0160 (30 mg/kg per day, p.o.) can be observed in plasma and brain tissue of the mice, proving MSDC-0160 can effectively enter the brain. MSDC-0160 (30 mg/kg per day, p.o.) treatment 3 days after MPTP injection, improves motor behavior, protects nigrostriatal neurons, and suppresses disease progression in the MPTP mouse model of Parkinson's disease (PD), improves motor behavior in the open-field and rotarod tests in the En1+/- genetic mouse model of PD, and prevents dopaminergic neurodegeneration in the En1+/- genetic mouse model of PD. MSDC-0160 (30 mg/kg, p.o.) modulates mTOR signaling in C. elegans and the MPTP mouse model of PD. MSDC-0160 down-regulates mTOR signaling and restores autophagy in the En1+/- genetic mouse model of PD[1].

In Vitro:

DMSO : ≥ 30 mg/mL (80.99 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.6996 mL	13.4982 mL	26.9964 mL
5 mM	0.5399 mL	2.6996 mL	5.3993 mL
10 mM	0.2700 mL	1.3498 mL	2.6996 mL

*请根据产品在不同溶剂中的溶解度选择合适的溶剂配制储备液；一旦配成溶液，请分装保存，避免反复冻融造成的产品失效。

储备液的保存方式和期限：-80°C, 6 months; -20°C, 1 month。-80°C 储存时，请在 6 个月内使用，-20°C 储存时，请在 1 个月内使用。

In Vivo:

请根据您的实验动物和给药方式选择适当的溶解方案。以下溶解方案都请先按照 **In Vitro** 方式配制澄清的储

Solvent&Solubility	备液，再依次添加助溶剂： ——为保证实验结果的可靠性，澄清的储备液可以根据储存条件，适当保存；体内实验的工作液，建议您现用现配，当天使用； 以下溶剂前显示的百分比是指该溶剂在您配制终溶液中的体积占比；如在配制过程中出现沉淀、析出现象，可以通过加热和/或超声的方式助溶 1.请依序添加每种溶剂： 10% DMSO→40% PEG300 →5% Tween-80 → 45% saline Solubility: 2.5 mg/mL (6.75 mM); Suspended solution; Need ultrasonic 此方案可获得 2.5 mg/mL (6.75 mM)的均匀悬浊液，悬浊液可用于口服和腹腔注射。 以 1 mL 工作液为例，取 100 μL 25.0 mg/mL 的澄清 DMSO 储备液加到 400 μL PEG300 中，混合均匀向上述体系中加入 50 μL Tween-80，混合均匀；然后继续加入 450 μL 生理盐水定容至 1 mL。 2.请依序添加每种溶剂： 10% DMSO →90% corn oil Solubility: ≥ 2.5 mg/mL (6.75 mM); Clear solution 此方案可获得 ≥ 2.5 mg/mL (6.75 mM，饱和度未知) 的澄清溶液，此方案不适用于实验周期在半个月以上的实验。 以 1 mL 工作液为例，取 100 μL 25.0 mg/mL 的澄清 DMSO 储备液加到 900 μL 玉米油中，混合均匀。
References	[1]. Rohatgi N, et al. Novel insulin sensitizer modulates nutrient sensing pathways and maintains β-cell phenotype in human islets. PLoS One. 2013 May 1;8(5):e62012. [2]. Ghosh A, et al. Mitochondrial pyruvate carrier regulates autophagy, inflammation, and neurodegeneration inexperimental models of Parkinson's disease. Sci Transl Med. 2016 Dec 7;8(368):368ra174.

源叶生物