

Product Data Sheet

Product Name: UBCS039

Cat. No.: GC34849

Chemical Properties

Cas. No. 358721-70-7

SMILES N12C(C(C3=CC=CN=C3)NC4=C2C=CC=C4)=CC=C1

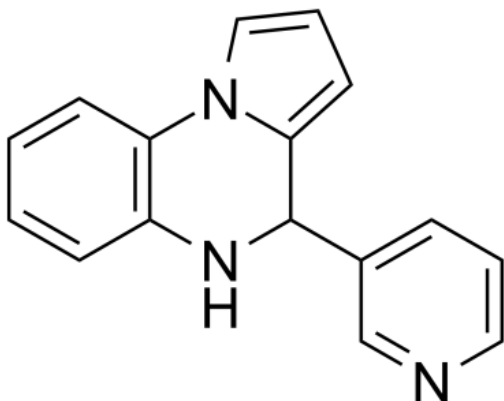
Formula $C_{16}H_{13}N_3$ M.Wt 247.29

Solubility DMSO : 125 mg/mL (505.48 mM) Storage Store at -20°C

General tips For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines RAW264.7 cells

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

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Preparation Method The cells were cultured in 96-well plates at a density of 1×10^4 cells/well, and then the cells were exposed to different concentrations of UBCS039 for 24h. After treatment, the culture medium was replaced with 10μl CCK-8 solution and 90μl medium for 1h at 37°C.

Reaction Conditions 0-200μM; 24h

Applications High doses of UBCS039 (50μM, 100μM, and 200μM) were cytotoxic to macrophages, with a cell viability lower than 90%.

Animal experiment [2]:

Animal models Sixty five-week-old healthy female BALB/c nude mice (BALB/c FOXN1^{-/-})

Preparation Method Mice were randomly assigned to groups. Every three days, each group was given intraperitoneal UBCS039, PBS, or DMSO injections at a 10mg/kg concentration, for a total of five injections. Each group included twenty mice. Cultured HeLa, LM3, Hcc827 or MDA-MB-231 cells were collected and resuspended in PBS 1 day after the final UBCS039 injection into nude mice. Mice pretreated with UBCS039 received a subcutaneous injection of 1×10^7 cells/ml into the right axillary area.

Dosage form 10mg/kg; i.p.; administered once every 3 days for 5 times

Applications UBCS039 promoted the growth of various tumor xenografts, and elevated levels of GM-CSF, IL-10, and ADO, decreased IFN-γ, as well as increased indicators related to PD-L1, PD-1, and M2 macrophages/TAM.

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References:

- [1]. Jiao, Fangzhou, et al. "SIRT6 activator UBCS039 inhibits thioacetamide-induced hepatic injury in vitro and in vivo." *Frontiers in Pharmacology* 13 (2022): 837544.
- [2]. Wang, Yan, et al. "Sirt6 promotes tumor growth and suppresses immune surveillance." *Cancer Cell International* (2026).

Background

UBCS039 is a synthetic specific activator of SIRT6, with EC₅₀ value of 38μM^[1]. UBCS039 is often used in studies of SIRT6-mediated autophagy, anti-inflammatory effects, antioxidant stress, lipid metabolism, macrophage polarization, renal and hepatic injury, and thrombosis^[1].

In vitro, UBCS039 (75-100μM; 24-72h) treatment induced H3K9 deacetylation, promoted the increase of LC3B-II, the formation of GFP-LC3 puncta. UBCS039 (100μM; 72h) treatment induced ROS-AMPK/ULK1/mTOR-related autophagy and autophagy-related cell death and apoptosis in H1299 and HeLa cells^[2]. UBCS039 (40μM; 2h) pretreatment inhibited lipopolysaccharide (10μg/ml; 24h)-induced inflammatory responses in RAW264.7 macrophages^[3]. UBCS039 (40μM; 24h) inhibited the oxidative stress in thioacetamide(TAA)-induced hepatocyte injury in LO2 cells^[3]. UBCS039 (100μM; 24h) improved the senescence phenotype induced by D-gal, increased cell viability and TERT expression, and reduced cell apoptosis, ROS, HK2 and SA-β-gal levels in H9c2 cardiac muscle cells^[4].

In vivo, UBCS039 (10mg/kg; i.p.; once every 3 days for a total of five injections) promoted the growth of various tumor xenografts, and elevated levels of GM-CSF, IL-10, and ADO, decreased IFN-γ, as well as increased indicators related to PD-L1, PD-1, and M2 macrophages/TAM^[5]. UBCS039 (30mg/kg; i.p.; once) reduced lipid accumulation in the renal tissues and apoptosis of renal tubular epithelial cells in LPS-induced acute kidney injury (AKI) mice^[6].

References:

- [1]. You, Weijie, et al. "Structural basis of sirtuin 6 activation by synthetic small molecules." *Angewandte Chemie International Edition* 56.4 (2017): 1007-1011.
- [2]. Iachettini, Sara, et al. "Pharmacological activation of SIRT6 triggers lethal autophagy in human

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cancer cells." Cell death & disease 9.10 (2018): 996.

[3]. Jiao, Fangzhou, et al. "SIRT6 activator UBCS039 inhibits thioacetamide-induced hepatic injury in vitro and in vivo." Frontiers in Pharmacology 13 (2022): 837544.

[4]. Zhou, Hongji, et al. "Downregulation of Sirt6 by CD38 promotes cell senescence and aging." Aging (albany NY) 14.23 (2022): 9730.

[5]. Wang, Yan, et al. "Sirt6 promotes tumor growth and suppresses immune surveillance." Cancer Cell International (2026).

[6]. Wang, Dan, et al. "SIRT6 mitigates acute kidney injury by enhancing lipid metabolism and reducing tubular epithelial cell apoptosis via suppression of the ACMSD signaling pathway." Cellular Signalling 131 (2025): 111757.

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