MOPIPP

Cat. No.: HY-148114

CAS No.: 1485521-76-3

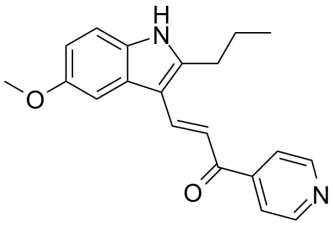
Molecular Formula: $C_{20}H_{20}N_2O_2$

Molecular Weight: 320.39

Target: Others

Pathway: Others

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

MOPIPP is a novel indolebased chalcone, and vacuolin-1, is a non-lethal vacuoleinducing 2-propyl analog of MOMIPP (HY-148114). MOPIPP induces cellular vacuolization and increases autophagosomes numbers. MOPIPP also triggers methuosis, and interrupts glucose uptake and glycolytic metabolism. MOPIPP can cross the blood-brain barrier and shows efficacy in suppressing tumor progression agaisnt glioblastoma cells[1][2][3].

In Vitro

MOPIPP (10 μM; 48 h) induces cellular vacuolization but does not cause cell death in U251 glioblastoma cells exhibiting characteristics of late endosomes[1][2].

MOPIPP (10 μM; 24 h) results an increasing LC3 fluorescence associated with the number of autophagosomes and inhibits fusion of autophagosomes with lysosomes[1].

MOPIPP (10 μM; 24 h) increases the amounts of exosomal marker proteins in vesicle fractions recovered from 293T cells[2].

MOMIPP (10 μM; 24 h and 5 h, respectively) causes early disruptions of glucose uptake and glycolytic metabolism, induces methuosis, a form of non-apoptotic cell death, in glioblastoma and other cancer cell lines[3].

MOMIPP (10 μM; 4 h and 24 h) selectively activates the JNK1/2 stress kinase pathway, resulting in phosphorylation of c-Jun, Bcl-2 and Bcl-xL[3].

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

Immunofluorescence[1]

Cell Line:	U251 glioblastoma cells
Concentration:	10 μM
Incubation Time:	24 hours; incubated with 2.5 μg/ml Acridine Orange (HY-101879) for 45 min
Result:	Caused accumulation of autophagosome markers. Increased punctate LC3 fluorescence and suggested an increase in the number of autophagosomes in cells.

Western Blot Analysis[3]

Cell Line:	U251 glioblastoma cells
Concentration:	10 μM
Incubation Time:	4 and 24 hours

Result:	Triggered increaseing phosphorylation of Bcl-2 and Bcl-xL, accompanied the activation of JNK.

In Vivo

MOMIPP (80 mg/kg; i.p.; single dose) readily penetrates the bloodbrain barrier in female Swiss Webster Mice and (80 mg/kg; i.p.; every 24 h; 15 d) is effective in suppressing progression of intracerebral glioblastoma xenografts in female NCR-Foxn1 mice[3].

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Animal Model:	Intracerebral xenograft model in NCR-Foxn1 mice (female, 7-8 weeks, injected with U251-LUC cells)[3]
Dosage:	80 mg/kg
Administration:	Intraperitoneal injection; every 24 hours for 15 days; monitored tumor progression by BLI on the days 7, 11, 15
Result:	Significantly inhibited tumor progression.

REFERENCES

[1]. Mbah NE, et al. Disruption of endolysosomal trafficking pathways in glioma cells by methuosis-inducing indole-based chalcones. Cell Biol Toxicol. 2017 Jun;33(3):263-282.

[2]. Li Z, et al. Vacuole-inducing compounds that disrupt endolysosomal trafficking stimulate production of exosomes by glioblastoma cells. Mol Cell Biochem. 2018 Feb;439(1-2):1-9.

[3]. Li Z, et al. The JNK signaling pathway plays a key role in methuosis (non-apoptotic cell death) induced by MOMIPP in glioblastoma. BMC Cancer. 2019 Jan 16;19(1):77.

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