MOMIPP

MedChemExpress

Cat. No.:	HY-119624		
CAS No.:	1363421-46-8		
Molecular Formula:	C ₁₈ H ₁₆ N ₂ O ₂		
Molecular Weight:	292.33		
Target:	PIKfyve		
Pathway:	PI3K/Akt/mTOR		
Storage:	Powder	-20°C	3 years
	In solvent	-80°C	6 months
		-20°C	1 month

SOLVENT & SOLUBILITY

In Vitro

DMSO: 31.25 mg/mL (106.90 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent Mass Concentration	1 mg	5 mg	10 mg
	1 mM	3.4208 mL	17.1040 mL	34.2079 mL
	5 mM	0.6842 mL	3.4208 mL	6.8416 mL
	10 mM	0.3421 mL	1.7104 mL	3.4208 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY		
Description	MOMIPP, a macropinocyt	tosis inducer, is a PIKfyve inhibitor. MOMIPP penetrates the blood-brain barrier (BBB) $^{[1][2]}$.
In Vitro	MOMIPP can induce intense macropinocytosis, leading to methuosis in cultured glioblastoma cells at low micromolar concentrations ^[1] . In U373 and Hs683 cell lines, 3 μM for MOMIPP induces cell vacuolization ^[1] . MOMIPP (10 μM) causes early disruptions of glucose uptake and glycolytic metabolism. MOMIPP selectively activates the JNK1/2 stress kinase pathway, resulting in phosphorylation of c-Jun, Bcl-2 and Bcl-xL ^[2] . MCE has not independently confirmed the accuracy of these methods. They are for reference only. Western Blot Analysis ^[2]	
	Cell Line:	U251 cells
	Concentration:	10 μΜ
	Incubation Time:	4 h or 24 h
	Result:	Activated the JNK stress kinase pathway.

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С

In Vivo	MOMIPP (80 mg/kg; i.p.; once daily; for 15 consecutive days) shows moderately effective in suppressing progression of intracerebral glioblastoma xenografts ^[2] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.	
	Animal Model:	Athymic CrTac:NCR-Foxn1 mice (female, 7-8 weeks) ^[2]
	Dosage:	80 mg/kg
	Administration:	i.p.; once daily; for 15 consecutive days
	Result:	Suppressed progression of intracerebral glioblastoma xenografts.

REFERENCES

[1]. Margaux Colin, et al. Dysregulation of Macropinocytosis Processes in Glioblastomas May Be Exploited to Increase Intracellular Anti-Cancer Drug Levels: The Example of Temozolomide. Cancers (Basel). 2019 Mar 22;11(3):411.

[2]. Zehui Li, 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.

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

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