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Zuschläge

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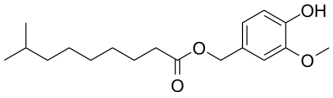
Dihydrocapsiate

Cat. No.:	HY-124073
CAS No.:	205687-03-2
Molecular Formula:	$C_{18}H_{28}O_4$
Molecular Weight:	308.41
Target:	TRP Channel
Pathway:	Membrane Transporter/Ion Channel; Neuronal Signaling
Storage:	-20°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



BIOLOGICAL ACTIVITY

Description	Dihydrocapsiate, as a compound of capsinoid family, is an orally active TRPV1 agonist. Dihydrocapsiate can be used for the research of metabolism disease[1].
IC_{50} & Target	TRPV1

In Vitro

Dihydrocapsiate (10, 25 and 50 μM; 48 hours; human preadipocytes) does not affect cell viability[1].
Dihydrocapsiate (10 and 20 μM; 8 days; mature adipocytes) markedly decreases the expression levels of other adipogenic markers (such as SREBP1, FABP4, PLIN1, ADIPOQ and LEPTIN) and inflammatory markers (MCP1 and TNFα), whereas it enhances the expression levels of PGC1α (master regulator of mitochondrial biogenesis) and TBX1 (marker of “brite” cell) [1].
Dihydrocapsiate (25~200 μM; RAW 264.7 cells) prevents NO release and intracellular ROS generation[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	Human preadipocytes
Concentration:	10, 25 and 50 μM
Incubation Time:	48 hours
Result:	Did not affect cell viability.

RT-PCR[1]

Cell Line:	Mature adipocytes
Concentration:	10 and 20 μM
Incubation Time:	8 days
Result:	Markedly decreased the expression levels of other adipogenic markers (such as SREBP1, FABP4, PLIN1, ADIPOQ and LEPTIN) and inflammatory markers (MCP1 and TNFα), whereas it enhanced the expression levels of PGC1α (master regulator of mitochondrial biogenesis) and TBX1 (marker of “brite” cell).

In Vivo

Dihydrocapsiate (2 and 10 mg/kg; p.o.) improves morphometric parameters and insulin levels, prevents high fat diet (HFD)-

induced adipocyte size and enhances energy expenditure-related genes in WAT, alleviates HFD-induced hepatic steatosis, prevents HFD-induced fat deposition and enhances mitochondrial biogenesis genes in BAT and improves intestinal morphology and modulates SCFA availability.

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

Animal Model:	HFD-fed mice[1]
Dosage:	2 and 10 mg/kg
Administration:	P.o.
Result:	Improved morphometric parameters and insulin levels, prevented HFD-induced adipocyte size and enhanced energy expenditure-related genes in WAT, alleviated HFD-induced hepatic steatosis, prevented HFD-induced fat deposition and enhanced mitochondrial biogenesis genes in BAT and improved intestinal morphology and modulates SCFA availability.

REFERENCES

[1]. Baboota RK, et al. Dihydrocapsiate supplementation prevented high-fat diet-induced adiposity, hepatic steatosis, glucose intolerance, and gut morphological alterations in mice. Nutr Res. 2018;51:40-56.

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