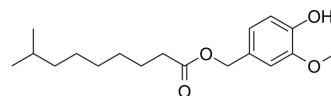


Dihydrocapsiate

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



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] .																
IC₅₀ & Target	TRPV1																
In Vitro	<p>Dihydrocapsiate (10, 25 and 50 μM; 48 hours; human preadipocytes) does not affect cell viability^[1].</p> <p>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].</p> <p>Dihydrocapsiate (25~200 μM; RAW 264.7 cells) prevents NO release and intracellular ROS generation^[1].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <p>Cell Viability Assay^[1]</p> <table border="1"> <tr> <td>Cell Line:</td> <td>Human preadipocytes</td> </tr> <tr> <td>Concentration:</td> <td>10, 25 and 50 μM</td> </tr> <tr> <td>Incubation Time:</td> <td>48 hours</td> </tr> <tr> <td>Result:</td> <td>Did not affect cell viability.</td> </tr> </table> <p>RT-PCR^[1]</p> <table border="1"> <tr> <td>Cell Line:</td> <td>Mature adipocytes</td> </tr> <tr> <td>Concentration:</td> <td>10 and 20 μM</td> </tr> <tr> <td>Incubation Time:</td> <td>8 days</td> </tr> <tr> <td>Result:</td> <td>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).</td> </tr> </table>	Cell Line:	Human preadipocytes	Concentration:	10, 25 and 50 μM	Incubation Time:	48 hours	Result:	Did not affect cell viability.	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).
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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.

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

Tel: 609-228-6898

Fax: 609-228-5909

E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA