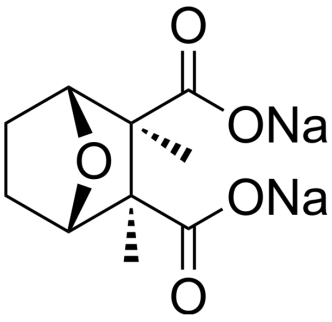
Cantharidic acid disodium

Cat. No.:	HY-137135A
CAS No.:	1465-77-6
Molecular Formula:	$C_{10}H_{12}Na_2O_5$
Molecular Weight:	258.18
Target:	Phosphatase; Apoptosis
Pathway:	Metabolic Enzyme/Protease; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Cantharidic acid disodium is the hydrolysis product of the acid anhydride Cantharidin that induces apoptosis in various human cancer cells. Cantharidic acid disodium is a selective protein phosphatase 2 (PP2A) and PP1 inhibitor withIC_{50} values of 50 nM and 600 nM, respectively[1][2].

In Vitro

Cantharidic acid (0-20 μM; 24 h) markedly reduces cell viability, which is revealed by the upregulation of caspase activation in extrinsic and intrinsic apoptosis pathways as well as the upregulation of ERK1/2, p38, and JNK1/2 pathways[1].

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

Cell Viability Assay[1]

Cell Line:	HONE-1, NPC-39, and NPC-BM
Concentration:	0, 2.5 μM, 5 μM, 10 μM, or 20 μM
Incubation Time:	24 h
Result:	Markedly reduced cell viability.

Apoptosis Analysis[1]

Cell Line:	HONE-1, NPC-39
Concentration:	0, 2.5 μM, 5 μM, 10 μM
Incubation Time:	24 h
Result:	Led to apoptosis in human nasopharyngeal carcinoma (NPC) cells through the upregulation of caspase activation.

Western Blot Analysis[1]

Cell Line:	HONE-1, NPC-39
Concentration:	0, 2.5 μM, 5 μM, 10 μM
Incubation Time:	24 h

Result:	Showed the upregulation of caspase activation in extrinsic and intrinsic apoptosis pathways as well as the upregulation of ERK1/2, p38, and JNK1/2 pathways.

In Vivo

Cantharidic acid (10 mg/kg; ip; once) causes extreme liver enlargement and congestion in mice. Hepatic glycogenolysis is increased as evidenced by elevations in blood glucose and hepatic glycogen phosphorylase levels and by corresponding reductions in hepatic glycogen content and glycogen synthase activity[3].

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

REFERENCES

[1]. Yi-Ching Chen, et al. Cantharidic acid induces apoptosis in human nasopharyngeal carcinoma cells through p38-mediated upregulation of caspase activation. Environ Toxicol. 2020 May;35(5):619-627.

[2]. Adam McCluskey, et al. Serine-threonine protein phosphatase inhibitors: development of potential therapeutic strategies. J Med Chem. 2002 Mar 14;45(6):1151-75.

[3]. M J Graziano, et al. Comparison of the acute toxicity of endothal and cantharidic acid on mouse liver in vivo. Toxicol Lett. 1987 Jul;37(2):143-8.

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