

Product Data Sheet

Product Name: Capsaicin

Cat. No.: GC14065

Chemical Properties

Cas. No. 404-86-4

Chemical Name (E)-N-[(4-hydroxy-3-methoxyphenyl)methyl]-8-methylnon-6-enamide

SMILES CC(C)C=CCCCC(=O)NCC1=CC(=C(C=C1)O)OC

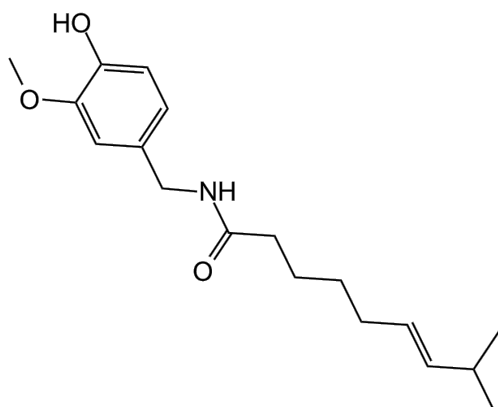
Formula $C_{18}H_{27}NO_3$ M.Wt 305.41

Solubility ≥ 15.27 mg/mL in DMSO, ≥ 52.3 mg/mL in EtOH with gentle warming Storage $4^{\circ}C$, protect from light

General tips For obtaining a higher solubility, please warm the tube at $37^{\circ}C$ and shake it in the ultrasonic bath for a while. Stock solution can be stored below $-20^{\circ}C$ for several months.

Shipping Condition Evaluation sample solution: ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines Human colorectal carcinoma cells (SW480, LoVo and HCT-116)

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Preparation Method The cells were grown at a concentration of 2×10^5 cells/ml and then treated with capsaicin at concentrations or time points indicated in figure legends.

Reaction Conditions 0, 50 and 100 μ M for 0, 1, 2, or 3 days

Applications Growth of all three human colorectal cancer cell lines was inhibited by capsaicin treatment in a dose- and time-dependent manner. Although 50- μ M capsaicin treatment decreased cell growth to some extent, significantly retarded cell growth was observed in cells treated with 100 μ M of capsaicin.

Animal experiment [2]:

Animal models Male Swiss albino mice

Preparation Method Group 1 (control) received olive oil throughout the course of the experiment. Group 2 were treated with BP (50 mg/kg-1 dissolved in olive oil) orally twice a week (Day 1 and Day 4) for four successive weeks. Group 3 received capsaicin (10 mg/kg-1 dissolved in olive oil) intraperitoneally once a week for 14 weeks to assess the cytotoxicity, if any, induced by capsaicin. Group 4 (BP + capsaicin) received BP (as for Group 2) along with capsaicin (10 mg/kg-1 dissolved in olive oil) intraperitoneally. Capsaicin treatment was started 1 week before the first dose of BP and continued for 14 weeks

Dosage form Intraperitoneal injection, 10 mg/kg-1 week-1 for 14 week

Applications Capsaicin pretreatment resulted in a free radical quenching effect, thereby significantly preventing the peroxidation of lipids in Group 4 animals.

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References:

- [1]: Lee S. H., Richardson R. L., Dashwood R. H., Baek S. J. (2011). Capsaicin represses transcriptional activity of β -catenin in human colorectal cancer cells. J Nutr Biochem.
- [2]: Anandakumar P., Jagan S., Kamaraj S., Ramakrishnan G., Titto A. A., Devaki T. (2008). Beneficial influence of capsaicin on lipid peroxidation, membrane-bound enzymes and glycoprotein profile during experimental lung carcinogenesis. J Pharm Pharmacol 60 803-8.

Background

Capsaicin is a highly selective agonist for the transient receptor potential cation channel, subfamily V, member 1 (TRPV1), a ligand-gated, nonselective cation channel, preferentially expressed on small-diameter sensory neurons [1]. capsaicin inhibits CYP1A2, CYP2C9, and CYP2C19, with IC50 values of 2.1, 2.0, and 3.2 μ M, respectively, and inhibits CYP2B6, CYP2D6, CYP3A4, and CYP3A5 with extrapolated IC50 values of 24, 18, 38, and 12 μ M, respectively [2].

Capsaicin inhibited Human cervical carcinoma HeLa growth (IC50 $\sim$ 30 μ M) and increased intracellular calcium, with effect blocked by capsazepine [3]. Capsaicin induced Human glioblastoma A172 apoptosis at $\geq$ 200 μ M, not inhibited by capsazepine or BAPTA/AM 250 μ M reduced basal generation of ROS and lipid peroxidation H2O2 reduced apoptosis, while NAC enhanced [4]. Capsaicin reduced Human urothelial cancer RT4 growth (IC50=80 μ M) via a TRPV1-dependent process, induced cell cycle arrest in G0/G1 phase Apoptosis occurred [5].

Capsaicin pretreated ICR mice CD-1 mice with 2.5 μ mol reduced VC-induced (5.8 mol) and TPA-promoted tumor incidence (by 62%) at 22 weeks (ICR) 1 μ mol topical capsaicin 24 and 1 hr before, also significantly inhibited tumors induced by topical BP (0.3 μ mol) then promoted with TPA (CD-1) [6]. Gavage with capsaicin significantly elevated phase II enzymes in liver and colon Oral capsaicin at 500 ppm for 4 weeks significantly inhibited ACF formation induced by AOM (20 mg/kg body weight, once a week for 2 weeks) In a 38-week study, 500 ppm capsaicin during the 4-week initiation phase significantly reduced (60%) incidence of colonic adenocarcinoma [7].

References:

- [1]. Bley, K.; Boorman, G.; Mohammad, B.; McKenzie, D.; Babbar, S. A comprehensive review of the

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- carcinogenic and anticarcinogenic potential of capsaicin. Toxicol. Pathol. 2012, 40, 847-873.
- [2]. Babbar S., Chanda S., Bley K. (2010). Inhibition and induction of human cytochrome P450 enzymes in vitro by capsaicin. Xenobiotica 40, 807-16.
- [3]. Takahata K., Chen X., Monobe K., Tada M. (1999). Growth inhibition of capsaicin on HeLa cells is not mediated by intracellular calcium mobilization. Life Sci 64, PL165-71.
- [4]. Lee Y. S., Nam D. H., Kim J. A. (2000). Induction of apoptosis by capsaicin in A172 human glioblastoma cells. Cancer Lett 161, 121-30.
- [5]. Amantini C., Ballarini P., Caprodossi S., Nabissi M., Morelli M. B., Lucciarini R., Cardarelli M. A., Mammana G., Santoni G. (2009). Triggering of transient receptor potential vanilloid type 1 (TRPV1) by capsaicin induces Fas/CD95-mediated apoptosis of urothelial cancer cells in an ATM-dependent manner. Carcinogenesis 30, 1320-29.
- [6]. Surh Y. J., Lee R. C., Park K. K., Mayne S. T., Liem A., Miller J. A. (1995). Chemoprotective effects of capsaicin and diallyl sulfide against mutagenesis or tumorigenesis by vinyl carbamate and N-nitrosodimethylamine. Carcinogenesis 16, 2467-71.
- [7]. Yoshitani S. I., Tanaka T., Kohno H., Takashima S. (2001). Chemoprevention of azoxymethane-induced rat colon carcinogenesis by dietary capsaicin and rotenone. Int J Oncol 19, 929-39.

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