

Product Data Sheet

Product Name: Carvacrol

Cat. No.: GC35612

Chemical Properties

Cas. No. 499-75-2

SMILES OC1=CC(C(C)C)=CC=C1C

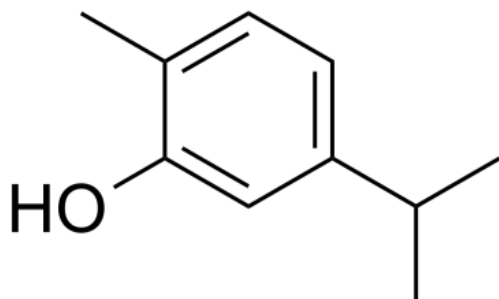
Formula $C_{10}H_{14}O$ M.Wt 150.22

Solubility Insoluble in Water; ≥ 28.1 mg/mL in EtOH; ≥ 28.8 mg/mL in DMSO Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°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 MCF-7 cells

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

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MCF-7 cells were cultured in T-25 flasks containing 10% FBS with DMEM, 1% 100U/mL penicillin and 100µg/mL streptomycin and were maintained at 37°C incubator with 5% CO₂ with 95% humidity. The culture medium was routinely replenished with complete media twice a week. After attaining confluence, the cells were trypsinized with Trypsin-EDTA solution. 1×10^4 MCF-7 cells per well Preparation Method were seeded in 96-well microplates for 24h. On achieving 70% confluence, cells were starved with an incomplete medium for 1h at 37°C. Post-starvation period, the cells were treated with different concentrations of carvacrol (0, 25, 50, 75, 100, 125, 150, 175, 200, 225, 250µM) for 24 and 48h. After treatment, MTT assays were used to detect cell viability. The cell cycle was analyzed by FACS. Annexin V-FITC/PI method was used for detection of cell apoptosis.

Reaction
Conditions 0-250µM; 24 or 48h

Applications Treatment of MCF-7 breast cancer cells with Carvacrol significantly reduced cell viability, induced cell cycle arrest at the G0/G1 phase, and promoted cell apoptosis.

Animal experiment [2]:

Animal models Female Sprague–Dawley rats

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Preparation Method

Forty-three adult female Sprague–Dawley rats weighting 200 to 300g were housed in polycarbonate cages in a room with controlled temperature ($25\pm 2^{\circ}\text{C}$), humidity ($50\pm 5\%$), and a 12h light/dark cycle. Animals had access to food and water ad libitum and were allowed to adapt to the local environment for 1 week before study. Feed and water were provided ad libitum. Rats were divided into six groups with regard to their body weights. Healthy Control Group (n=7): Rats in this group had access to food and water ad libitum during experimental period (7 days). Vehicle Control Group (n=7): 5% DMSO were administered by intragastric gavage in this group during 6 days. On the seventh day, 0.9% saline was injected to intraperitoneum (i.p.). Carvacrol Group I (n=6): 20mg/kg Carvacrol in 5% DMSO was administered to this group during 6 days, and on the seventh day, 1mg/kg LPS was injected i.p. Carvacrol Group II (n=8): 40mg/kg Carvacrol+1mg/kg LPS was administered to this group compatible with the same process of CVC group I. Carvacrol Group III (n=7): 80mg/kg Carvacrol+1mg/kg LPS was administered to this group compatible with same process of Carvacrol group I and II. Sepsis Group (LPS; n=8): Sublethal dose of E. coli LPS was given to rats intraperitoneal at 1mg/kg according to body weight and rats were killed at the indicated times following anesthetization. All tested solutions were administered orally (oral gavage) once a day at 9:00 a.m. for 6 days. Carvacrol and sepsis groups were treated with LPS on the seventh day. After 24h from LPS treatment, animals in all groups were sacrificed by exsanguination under anesthesia using a mixture of Ketamine/Xylazine. Serum samples were obtained by centrifuging the blood samples at $2,000\times g$ for 10min. All of the samples were stored at -80°C until use. Two enzyme-linked immunosorbent assay kits were used for the TNF- α and the IL-6 assays. Malondialdehyde (MDA) is detected in quantitative employing the thiobarbituric acid assay. NO was measured as nitrite using the Griess reagent.

Dosage form 20-80mg/kg/day; p.o.; 6 days

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Applications Carvacrol significantly reduced the levels of pro-inflammatory cytokines TNF- α and IL-6, malondialdehyde (MDA) and nitric oxide (NO) in a rat sepsis model induced by LPS in a dose-dependent manner.

References:

- [1] Mari A, Mani G, Nagabhishek SN, et al. Carvacrol Promotes Cell Cycle Arrest and Apoptosis through PI3K/AKT Signaling Pathway in MCF-7 Breast Cancer Cells. *Chin J Integr Med*. 2021;27(9):680-687.
- [2] Kara M, Uslu S, Demirci F, Temel HE, Baydemir C. Supplemental carvacrol can reduce the severity of inflammation by influencing the production of mediators of inflammation. *Inflammation*. 2015;38(3):1020-1027.

Background

Carvacrol is a monoterpene phenol with oral activity^[1]. Carvacrol has antioxidant, antibacterial, antifungal, anticancer, anti-inflammatory, hepatoprotective, antispasmodic, and vasodilatory properties^[2]. Carvacrol is usually used in research related to cancer and inflammation^[3]. Carvacrol is also used as a flavoring agent and preservative in food at low concentrations, as well as a fragrance component in cosmetic formulas^[4].

In vitro, treatment of MCF-7 breast cancer cells with Carvacrol (0-250 μ M; 24 or 48h) significantly reduced cell viability, induced cell cycle arrest at the G0/G1 phase, and promoted cell apoptosis^[5].

In vivo, Carvacrol (20-80mg/kg/day; p.o.; 6 days) significantly reduced the levels of pro-inflammatory cytokines TNF- α and IL-6, malondialdehyde (MDA), nitric oxide (NO), and arginase activity in a rat sepsis model induced by LPS in a dose-dependent manner^[6]. Carvacrol (30mg/kg/day; intraperitoneal injection from postoperative day 1 to day 9) significantly prolonged graft survival, reduced inflammatory cell infiltration in skin allografts, and decreased the expression of pro-inflammatory cytokines such as IL-6, TNF- α , and IL-17 in a mice skin allograft model^[7].

References:

- [1] Sharifi-Rad M, Varoni EM, Iriti M, et al. Carvacrol and human health: A comprehensive review.

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Phytother Res. 2018;32(9):1675-1687.

[2] Suntres ZE, Coccimiglio J, Alipour M. The bioactivity and toxicological actions of carvacrol. *Crit Rev Food Sci Nutr.* 2015;55(3):304-318.

[3] Singh J, Luqman S, Meena A. Carvacrol as a Prospective Regulator of Cancer Targets/Signalling Pathways. *Curr Mol Pharmacol.* 2023;16(5):542-558.

[4] Beninca T, Schmidt L, Thome Cardoso L, Rossini Augusti P, da Silva Malheiros P. Carvacrol as a food additive: Toxicological aspects and the role of nanotechnology in enhancing its antimicrobial and antioxidant properties. *Food Res Int.* 2024;197(Pt 1):115256.

[5] Mari A, Mani G, Nagabhishek SN, et al. Carvacrol Promotes Cell Cycle Arrest and Apoptosis through PI3K/AKT Signaling Pathway in MCF-7 Breast Cancer Cells. *Chin J Integr Med.* 2021;27(9):680-687.

[6] Kara M, Uslu S, Demirci F, Temel HE, Baydemir C. Supplemental carvacrol can reduce the severity of inflammation by influencing the production of mediators of inflammation. *Inflammation.* 2015;38(3):1020-1027.

[7] Zhao W, Tang H, Liang Z, et al. Carvacrol ameliorates skin allograft rejection through modulating macrophage polarization by activating the Wnt signalling pathway. *Phytother Res.* 2024;38(9):4675-4694.

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