

Product Data Sheet

Product Name: Desmethylglycitein

Cat. No.: GC38482

Chemical Properties

Cas. No. 17817-31-1

SMILES O=C1C(C2=CC=C(O)C=C2)=COC3=CC(O)=C(O)C=C13

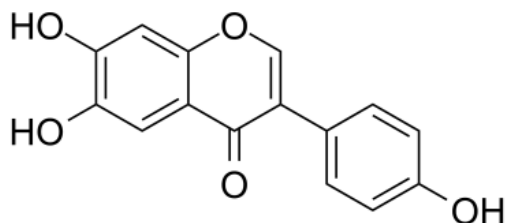
Formula $C_{15}H_{10}O_5$ M.Wt 270.24

Solubility DMSO: 125 mg/mL (462.55 mM) Storage Store at 2-8°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 MCF10CA1a cells

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

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Preparation Method MCF10CA1a cells were grown in DMEM/F12 medium supplemented with 1% penicillin/streptomycin, 5% horse serum, and HEPES buffer at 37°C in an incubator with 5% CO₂. Cells were cultured in 96-well plates in culture medium at 1000 cells/well for 24h. Different concentrations of Desmethylglycitein (0, 1, 10, 20, 50 and 100µM) were added and incubated at 37°C with 5% CO₂ for 72h. Cell viability was measured.

Reaction Conditions 0, 1, 10, 20, 50 and 100µM; 72h

Applications Desmethylglycitein treatment significantly reduced cell viability of MCF10CA1a cells in a dose-dependent manner.

Animal experiment [2]:

Animal models Female athymic nude mice

Preparation Method Female athymic nude mice (5 weeks old; 20g) were housed in an environmentally controlled room (12h light–dark cycle, 50%±10% relative humidity) at 23±2°C in a facility with free access to food and water. Each mouse was subcutaneously injected with HCT116 cells (2×10⁶ cells/200µl) in the left flank and the cells were allowed to form tumors. When tumors reached a size of 100mm³, the mice were randomly assigned to groups (six mice/group) and treated with Desmethylglycitein (25mg/kg) in 50% dimethyl sulfoxide/phosphate-buffered saline buffer, given intraperitoneally every other day for 20 days. Tumor size was measured every other day using calipers and tumor volume.

Dosage form 25mg/kg/day; every other day for 20 days; i.p.

Applications Desmethylglycitein treatment inhibited tumor growth in mice with HCT-116 xenografts.

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References:

- [1] Lee J H, Lee H J. A daidzein metabolite, 6, 7, 4'-trihydroxyisoflavone inhibits cellular proliferation through cell cycle arrest and apoptosis induction in MCF10CA1a human breast cancer cells[J]. Journal of the Korean Society for Applied Biological Chemistry, 2013, 56(6): 695-700.
- [2] Lee D E, Lee K W, Jung S K, et al. 6, 7, 4'-trihydroxyisoflavone inhibits HCT-116 human colon cancer cell proliferation by targeting CDK1 and CDK2[J]. Carcinogenesis, 2011, 32(4): 629-635.

Background

Desmethyleglicitein, a metabolite of daidzein, is a potent tyrosinase inhibitor with an IC₅₀ value of 9.2μM for mushroom tyrosinase [1]. Desmethyleglicitein inhibits the development of *Aedes aegypti* larvae by strongly suppressing the activity of glutathione S-transferase Noppera-bo, with an IC₅₀ value of 0.287μM[2]. Desmethyleglicitein simultaneously reduces Pin1 isomerase activity and the downstream activation targets of Pin1 to suppress esophageal cancer growth [3]. Desmethyleglicitein has been widely used to inhibit neuronal cell death and alleviate oxidative stress[4].

In vitro, Desmethyleglicitein treatment for 72 hours significantly inhibited the proliferation of MCF10CA1a cells, with an IC₅₀ value of 12.4μM [5]. Treatment with 80μM Desmethyleglicitein for 8 days significantly inhibited adipogenesis in 3T3-L1 preadipocytes and reduced the expression of PPAR-γ and C/EBP-α[6].

In vivo, Desmethyleglicitein treatment via a single oral dose of 5mg/kg improved the cognitive impairment caused by scopolamine in mice within one hour, and enhanced the learning and memory abilities in the Y-maze test[7]. Intraperitoneal injection of 25mg/kg dose of Desmethyleglicitein was administered every other day for 20 days, which significantly inhibited the growth of HCT-116 xenograft tumors in mice and reduced the activities of CDK1 and CDK2 within the tumors[8].

References:

- [1] Chang T S, Ding H Y, Lin H C. Identifying 6, 7, 4'-trihydroxyisoflavone as a potent tyrosinase inhibitor[J]. Bioscience, biotechnology, and biochemistry, 2005, 69(10): 1999-2001.
- [2] Inaba K, Ebihara K, Senda M, et al. Molecular action of larvicidal flavonoids on ecdysteroidogenic glutathione S-transferase Noppera-bo in *Aedes aegypti*[J]. BMC biology, 2022, 20(1): 43.

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- [3] Lim T G, Lee S Y, Duan Z, et al. The prolyl isomerase Pin1 is a novel target of 6, 7, 4'-trihydroxyisoflavone for suppressing esophageal cancer growth[J]. Cancer Prevention Research, 2017, 10(5): 308-318.
- [4] Ko Y H, Kwon S H, Kim S K, et al. Protective effects of 6, 7, 4'-trihydroxyisoflavone, a major metabolite of daidzein, on 6-hydroxydopamine-induced neuronal cell death in SH-SY5Y human neuroblastoma cells[J]. Archives of Pharmacal Research, 2019, 42(12): 1081.
- [5] Lee J H, Lee H J. A daidzein metabolite, 6, 7, 4'-trihydroxyisoflavone inhibits cellular proliferation through cell cycle arrest and apoptosis induction in MCF10CA1a human breast cancer cells[J]. Journal of the Korean Society for Applied Biological Chemistry, 2013, 56(6): 695-700.
- [6] Seo S G, Yang H, Shin S H, et al. A metabolite of daidzein, 6, 7, 4'-trihydroxyisoflavone, suppresses adipogenesis in 3 T 3-L 1 preadipocytes via ATP-competitive inhibition of PI 3 K[J]. Molecular nutrition & food research, 2013, 57(8): 1446-1455.
- [7] Ko Y H, Kim S Y, Lee S Y, et al. 6, 7, 4'-Trihydroxyisoflavone, a major metabolite of daidzein, improves learning and memory via the cholinergic system and the p-CREB/BDNF signaling pathway in mice[J]. European Journal of Pharmacology, 2018, 826: 140-147.
- [8] Lee D E, Lee K W, Jung S K, et al. 6, 7, 4'-trihydroxyisoflavone inhibits HCT-116 human colon cancer cell proliferation by targeting CDK1 and CDK2[J]. Carcinogenesis, 2011, 32(4): 629-635.

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