

Product Data Sheet

Product Name: Coumestrol

Cat. No.: GC10002

Chemical Properties

Cas. No. 479-13-0

Chemical Name 3,9-dihydroxy-6H-benzofuro[3,2-c][1]benzopyran-6-one

SMILES O=C1OC2=C(C=CC(O)=C2)C3=C1C4=CC=C(O)C=C4O3

Formula $C_{15}H_8O_5$

M.Wt 268.2

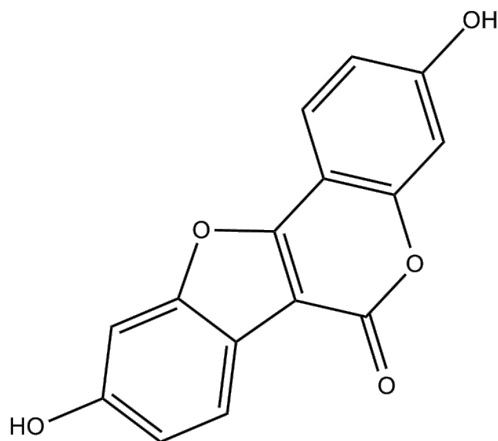
Solubility $\leq 25\text{mg/ml}$ in DMSO

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

143B, HOS cells (human osteosarcoma cell line)

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Preparation Method	143B and HOS cells were treated with Coumestrol at different concentrations for 24h; cell viability was evaluated by CCK-8, proliferation by EdU, apoptosis and cell cycle by flow cytometry, migration and invasion by Transwell, and DDX39A protein by Western blot; 143B and HOS cells were also treated with 50μM Coumestrol for 24h in cellular thermal shift assay.
Reaction Conditions	0-100μM; 24h
Applications	Coumestrol decreased 143B and HOS cell viability and EdU-positive cells, increased apoptosis, increased G1-phase cells and reduced S-phase cells, inhibited migration and invasion, and reduced DDX39A protein expression; cellular thermal shift assay showed DDX39A bound Coumestrol.
Animal experiment [2]:	
Animal models	7 week old female C57BL/6J mice
Preparation Method	Mice were subjected to bilateral ovariectomy or sham operation. Treatment with Coumestrol (5mg/kg) or 17β-estradiol (50μg/kg) was initiated the day after surgery and administered once daily for 10 weeks by subcutaneous injection or oral gavage. All mice were fed a phytoestrogen-free high-fat diet. At 6 weeks after surgery, mice were housed singly with running wheels for 3 days to determine voluntary running activity. At the end of the study, mice were sacrificed; blood was collected via cardiac puncture, and tissue samples (parametrial adipose tissue, liver, white adipose tissue, brown adipose tissue, skeletal muscle) were isolated for histological staining, Western blot, and TBARS assay.
Dosage form	5mg/kg; s.c. or p.o.; once daily for 10 weeks

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Applications	Coumestrol prevented body weight gain, visceral and subcutaneous fat accumulation, and adipocyte hypertrophy; normalized hepatic lipid accumulation and reduced fatty acid synthase protein expression; reduced hepatic TBARS level; increased voluntary running activity; increased FGF21 protein expression in liver and white adipose tissue; increased UCP1, PPAR α , and p-p38 in white adipose tissue; increased PPAR γ in brown adipose tissue; increased phosphorylation of PI3K and Akt in skeletal muscle and liver.
References:[1] Wang H, Jin Y, Zhang F. DExD-box helicase 39 A, targeted by coumestrol, facilitates the malignant behaviors of osteosarcoma cells. Hereditas. 2025 Oct 28;162:218.[2] Park S, Sim K, Heo W, et al. Protective Effects of Coumestrol on Metabolic Dysfunction and Its Estrogen Receptor-Mediated Action in Ovariectomized Mice. Nutrients. 2023 Feb;15(4):954.	

Background

Coumestrol is a natural, orally active coumestan phytoestrogen. Coumestrol competitively binds estrogen receptor ER α (IC₅₀=11nM) and ER β (IC₅₀=2nM) to regulate estrogen-responsive genes. Coumestrol inhibits aromatase and 17 β -hydroxysteroid dehydrogenase and weakly antagonizes pregnane X receptor to reduce xenobiotic-metabolizing enzyme expression. Coumestrol is used for breast cancer, endometrial cancer, osteoporosis, and postmenopausal metabolic disorder research^[1-4].

In vitro, 143B and HOS osteosarcoma cells were treated with 0-100 μ M Coumestrol for 24h. Coumestrol reduced cell viability and proliferation, promoted apoptosis, increased G1-phase cell proportion, decreased S-phase cell proportion, and inhibited migration and invasion^[5]. ES2 human epithelial ovarian cancer cells were treated with 0-100 μ M Coumestrol for 1-4 days. Coumestrol inhibited cell proliferation and invasion^[6]. HCT116, HT-29, and A549 cells were treated with 80 μ M Coumestrol for 3-14h. Coumestrol reduced cell viability and colony formation^[7].

In vivo, pregnant ICR mice received oral gavage of 200 μ g/kg Coumestrol once daily for 11 doses starting at 6.5 days post-coitum. Coumestrol decreased duodenal and jejunal alkaline phosphatase activity and reduced IAP, c-fos, and VEGF mRNA expression^[8]. High-fat diet-fed ovariectomized C57BL/6J mice received subcutaneous injection of 5mg/kg Coumestrol once daily for 10 weeks.

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Coumestrol prevented adipose tissue accumulation and reduced hepatic lipid accumulation^[9]. Cardiac dysfunction model C57BL/6 mice received oral gavage of 5mg/kg/day Coumestrol once daily for 2 weeks. Coumestrol reduced cardiac oxidative damage, cardiomyocyte apoptosis, and cardiac dysfunction, and decreased serum cTnT, CK-MB, and LDH levels^[10].

References:

- [1] Sun JS, Li YY, Liu MH, et al. Effects of coumestrol on neonatal and adult mice osteoblasts activities. J Biomed Mater Res A. 2007 Apr;81(1):214-23.
- [2] Wang T, Wang Y, Zhuang X, et al. Interaction of Coumarin Phytoestrogens with ER α and ER β : A Molecular Dynamics Simulation Study. Molecules. 2020 Mar 5;25(5):1165.
- [3] Whitten PL, Patisaul HB, Young LJ. Neurobehavioral actions of coumestrol and related isoflavonoids in rodents. Neurotoxicol Teratol. 2002 Jan-Feb;24(1):47-54.
- [4] Burroughs CD. Long-term reproductive tract alterations in female mice treated neonatally with coumestrol. Proc Soc Exp Biol Med. 1995 Jan;208(1):78-81.
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- [8] Kiriata Y, Horiguchi Y, Ueda M, et al. Effects of coumestrol administration to pregnant and lactating mice on intestinal alkaline phosphatase activity. Reprod Dev. 2010;56(5):645-654.
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- [10] Wu ZZ, Rao M, Xu S, et al. Coumestrol ameliorates doxorubicin-induced cardiotoxicity via activating AMPK α . Free Radical Res. 2020;54(11):882-897.

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