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Zuschläge

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Ferruginol

Cat. No.: HY-N6033

CAS No.: 514-62-5

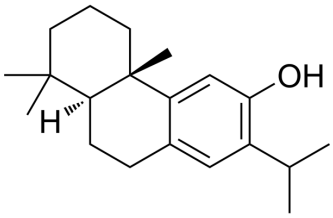
Molecular Formula: $C_{20}H_{30}O$

Molecular Weight: 286.45

Target: HSV; Apoptosis; EBV

Pathway: Anti-infection; Apoptosis

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Ferruginol ((+)-Ferruginol), a natural diterpenoid, is an inhibitor of the activation of Epstein-Barr virus early antigen (EBV-EA). Ferruginol inhibits the growth of thyroid cancer cells through the induction of mitochondrial apoptosis. Ferruginol has antitumor, cardioprotective, antioxidant, gastroprotective, and neuroprotective activities[1][2][3].

In Vitro

Ferruginol (0-160 μM; 24 hours) exerts potent antiproliferative action against thyroid cancer cells, and an IC_{50} of 12 μM for the MDA-T32 cell line. The toxic effects of Ferruginol are less pronounced against normal cells[1].

Ferruginol (0-24 μM; 24 hours) induces apoptotic cell death of MDA-T32 cells. Ferruginol increases Bax expression and decreases Bcl-2 expression dose-dependently[1].

Ferruginol (0-24 μM; 24 hours) inhibits the MAPK and PI3K/AKT signaling pathway of MDA-T32 cells[1].

Ferruginol (0-24 μM; 24 hours) also causes ROS mediated alterations in the MMP of MDA-T32 cells[1].

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

Cell Viability Assay[1]

Cell Line:	MDA-T32 cells
Concentration:	0-160 μM
Incubation Time:	24 hours
Result:	Exerted potent antiproliferative action against thyroid cancer cells.

Apoptosis Analysis[1]

Cell Line:	MDA-T32 cells
Concentration:	0 μM, 6 μM, 12 μM, and 24 μM
Incubation Time:	24 hours
Result:	Induced apoptotic cell death of MDA-T32 cells

Western Blot Analysis[1]

Cell Line:	MDA-T32 cells

Concentration:	0 μM, 6 μM, 12 μM, and 24 μM
Incubation Time:	24 hours
Result:	Blocked the MAPK and PI3K/AKT signaling pathway.

In Vivo

Ferruginol (20 mg/kg; p.o.; daily; for 4 weeks) exerts cardioprotection manifested as enhanced cardiac function and reduced structural damage and apoptosis. The transcriptome and other results revealed that Ferruginol facilitates PGC-1α-mediated mitochondrial biogenesis and fatty acid oxidation (MB and FAO) by increasing the expression of PGC-1α and concurrently promoting the expression of SIRT1-enhancing deacetylase SIRT1 deacetylating and activating PGC-1α[3].

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

Animal Model:	Male C57BL/6 mice (20 g, 8-10 weeks old) with Doxorubicin (DOX)-induced cardiotoxicity (DIC)[3].
Dosage:	20 mg/kg
Administration:	Administered intragastrically; daily; for 4 weeks
Result:	Relieved Doxorubicin-induced cardiac structural and functional lesion.

REFERENCES

[1]. Manabu Iwamoto, et al. Potential antitumor promoting diterpenoids from the stem bark of Thuja standishii. Planta Med. 2003 Jan;69(1):69-72.

[2]. Guoqing Luo, et al. Ferruginol Diterpenoid Selectively Inhibits Human Thyroid Cancer Growth by Inducing Mitochondrial Dependent Apoptosis, Endogenous Reactive Oxygen Species (ROS) Production, Mitochondrial Membrane Potential Loss and Suppression of Mitogen-Activated Protein Kinase (MAPK) and PI3K/AKT Signaling Pathways. Med Sci Monit. 2019 Apr 21;25:2935-2942.

[3]. Weili Li, et al. Ferruginol Restores SIRT1-PGC-1α-Mediated Mitochondrial Biogenesis and Fatty Acid Oxidation for the Treatment of DOX-Induced Cardiotoxicity. Front Pharmacol. 2021 Nov 24;12:773834.

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