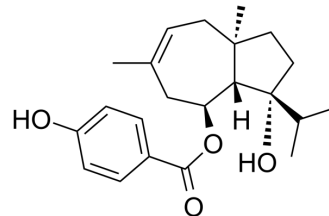


Ferutinin

Cat. No.:	HY-125703
CAS No.:	41743-44-6
Molecular Formula:	C ₂₂ H ₃₀ O ₄
Molecular Weight:	358.47
Target:	Estrogen Receptor/ERR; Apoptosis
Pathway:	Others; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	<p>Ferutinin, a natural terpenoid compound, is an estrogen receptor ERα agonist and estrogen ERβ-receptor agonist/antagonist with IC₅₀s of 33.1 nM and 180.5 nM, respectively. Ferutinin acts as an electrogenic Ca²⁺-ionophore that increases calcium permeability of lipid bilayer membranes, mitochondria. Ferutinin possesses estrogenic, antitumor, antibacterial and antiinflammatory activities^{[1][2]}.</p>	
IC₅₀ & Target	ER α 33.1 nM (IC ₅₀)	ER β 180.5 nM (IC ₅₀)
In Vitro	<p>Ferutinin manifested antiproliferative activity, inducing apoptosis in several cell types: MCF-7 estrogen-dependent cancer cells, leukemia T-cell line (Jurkat), human and mouse colon carcinoma cells (Caco-2, CT26, HT29), as well as bladder (TCC) cancer cells. Ferutinin potentiates bone mineralization, and is proposed to be used as an antiosteoporosis phytoestrogen^[2]. Ferutinin considerably increases the permeability of artificial and cellular membranes to Ca²⁺-ions and produces apoptotic cell death in different cell lines in a mitochondria-dependent manner. Ferutinin alone (10-60 μM) also dose-dependently dissipated membrane potential. In the presence of Ca²⁺-ions, Ferutinin (10-60 μM) induces considerable depolarization of the inner mitochondrial membrane^[2].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>	

REFERENCES

- [1]. Kazuhiro Ikeda, et al. Terpenoids found in the umbelliferae family act as agonists/antagonists for ER(alpha) and ERbeta: differential transcription activity between ferutinine-liganded ER(alpha) and ERbeta. *Biochem Biophys Res Commun*. 2002 Feb 22;291(2):354-60.
- [2]. Tatsiana Ilyich, et al. Ferutinin Induces Membrane Depolarization, Permeability Transition Pore Formation, and Respiration Uncoupling in Isolated Rat Liver Mitochondria by Stimulation of Ca²⁺-Permeability. *J Membr Biol*. 2018 Aug;251(4):563-572.

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

Tel: 609-228-6898

Fax: 609-228-5909

E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA