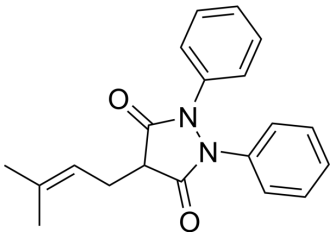
Feprazone

Cat. No.:	HY-114911
CAS No.:	30748-29-9
Molecular Formula:	$C_{20}H_{20}N_2O_2$
Molecular Weight:	320.39
Target:	COX; Reactive Oxygen Species; MMP
Pathway:	Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Feprazone (DA2370; Prenazone), an analogue of Phenylbutazone (HY-B0230), is a nonsteroidal anti-inflammatory agent with analgesic and antipyretic activities. Feprazone acts by inhibiting the activity of cyclooxygenase (COX)-2. Feprazone ameliorates free fatty acid (FFA)-induced oxidative stress by reducing the production of mitochondrial reactive oxygen species (ROS). Feprazone can decrease the expression of MMP-2 and MMP-9. Besides, Feprazone can suppress adipogenesis and increase lipolysis in differentiating 3 T3-L1 cells. Feprazone also can be used to research atherosclerosis and obesity[1][2][3].

IC_{50} & Target

COX, Reactive oxygen species, MMP[1]

In Vitro

Feprazone (2.5-10 μM; 48 h) rescues cell viability of FFAs-stimulated human aortic endothelial cells (HAECs)[1].
Feprazone (5, 10 μM; 24 h) reduces ROS production in HAECs to only 2.4- and 1.6-fold at 5 and 10 μM, respectively, while 300 μM FFA increases ROS production by 3.4-fold; also decreases the mRNA expression and secretion of cytokines CCL5, IL-6, and IL-8, as well as MMP-2 and MMP-9[1].
Feprazone (5, 10 μM; 6 h) decreases TLR4 and MyD88 activities, as well as reduces the phosphorylation of p65 and subsequent activation of NF-κB[1].
Feprazone (30 and 60 μM; 7 days) suppresses the adipogenesis in differentiating 3 T3-L1 cells; reduced the triglyceride content and increased lipolysis during 3 T3-L1 adipogenesis[3].

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

Cell Viability Assay[1]

Cell Line:	HAECs (stimulated with 300 μM FFAs)
Concentration:	2.5, 5 and 10 μM
Incubation Time:	48 h
Result:	Rescued cell viability to 81 and 93% of baseline at 5 and 10 μM, while FFAs reduced the cell viability to 63% of baseline.

RT-PCR[1]

Cell Line:	HAECs (stimulated with 300 μM FFAs)
Concentration:	5 and 10 μM

Incubation Time:	24 h
Result:	Decreased the mRNA expression and secretion of cytokines CCL5, IL-6, and IL-8 in a dose-dependent manner. Dose-dependently mitigated the VCAM-1 and ICAM-1 expression to only 1.7- and 1.8-fold, respectively, while FFA increased to 2.8- and 3.4-fold, respectively.

Western Blot Analysis[1]

Cell Line:	HAECs (stimulated with 300 μM FFAs)
Concentration:	5 and 10 μM
Incubation Time:	6 h
Result:	Decreased TLR4 and MyD88 expression, as well as reduced the phosphorylation of p65 and subsequent activation of NF-κB.

In Vivo

Significantly inhibited the adipocyte size, the visceral adipocyte tissue weights and the average bodyweights in HFD mice[3].

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Animal Model:	Male C57BL/6 N mice [high-fat diet (HFD) induced obesity model][3]
Dosage:	75 mg/kg
Administration:	(no described in the research)
Result:	The visceral adipocyte tissue weights of mice in the control, HFD, and HFD + Feprazone groups were 0.38, 3.51, and 2.37 g, respectively. The average bodyweights of mice in the control, HFD, and HFD + Feprazone groups were 29.6, 41.3, and 34.1 g, respectively.

REFERENCES

[1]. Song M, et al. Feprazone Prevents Free Fatty Acid (FFA)-Induced Endothelial Inflammation by Mitigating the Activation of the TLR4/MyD88/NF-κB Pathway. ACS Omega. 2021 Feb 9;6(7):4850-4856.

[2]. Fletcher MR, et al. Feprazone, a new anti-inflammatory agent. Studies of potency and gastrointestinal tolerance. Ann Rheum Dis. 1975 Apr;34(2):190-4.

[3]. Che L, et al. Feprazone Displays Antiadipogenesis and Antiobesity Capacities in in Vitro 3 T3-L1 Cells and in Vivo Mice. ACS Omega. 2021 Mar 7;6(10):6674-6680.

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

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