Ufenamate

Cat. No.: HY-100009

CAS No.: 67330-25-0

Molecular Formula: $C_{18}H_{18}F_3NO_2$

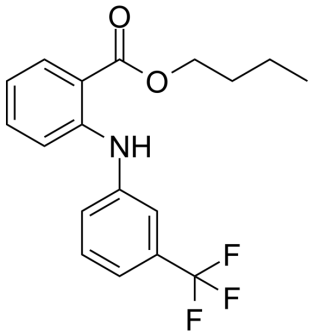
Molecular Weight: 337.34

Target: COX; Prostaglandin Receptor

Pathway: Immunology/Inflammation; GPCR/G Protein

Storage:

Pure form	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 44 mg/mL (130.43 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.9644 mL	14.8218 mL	29.6437 mL
	5 mM	0.5929 mL	2.9644 mL	5.9287 mL
	10 mM	0.2964 mL	1.4822 mL	2.9644 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

Ufenamate (Flufenamic acid butyl ester) is an anthranilic acid-based anti-inflammatory drug that can be used in the study of skin diseases such as acute and chronic eczema, contact dermatitis, diaper dermatitis, miliary rashes and atopic dermatitis. Ufenamate has a certain photoprotective effect, reduces the degree of skin erythema and swelling in the photoaging model, downregulates the expression level of COX-2 and can promote the healing of mouse skull defects by secreting BMP2[1][2][3][4].

IC_{50} & Target

COX-2

In Vivo

Ufenamat (2 μL/cm^2, ointment application, single dose) is not suitable for dry skin in rats[2].

Ufenamate (Ointment application, single dose) has a certain photoprotective effect, reduces the degree of skin erythema and swelling in the photoaging mice model and downregulates the expression level of COX-2[3].

Ufenamat (Ointment application, 6 and 8 weeks) promotes the healing of skull injuries in mice[4].

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

Animal Model:	UV-induced acute sunburn model, photoaging model and skin squamous cell carcinoma model in SKH-1 hairless mice[3]
Dosage:	Ointment application
Administration:	Ointment application; single dose; 30 min
Result:	Inhibited acute swelling and redness caused by UV irradiation and reduced COX-2 expression. At 12 weeks of irradiation, skin aging was alleviated, the collagen density of the dermis was higher, the expression of Bcl-2 was weakened, and the expression of Bax and Caspase 3 was enhanced. At 28 weeks of irradiation, the appearance of tumors was delayed.

Animal Model:	Intact and damaged skin of rats[2]
Dosage:	2μL/cm^2
Administration:	Ointment application; single dose
Result:	Applied on the damaged skin, contained in the epidermis was approximately 50% and 25% of that in the intact skin after LP and water, respectively, with the content being 5 times and 10 times higher than in the intact skin. Permeated UF five times more than LP in the intact skin. Reduced significantly the permeation of UF by both LP and water due to the defatting effect of SC.

Animal Model:	Skull defect mice[4]
Dosage:	Ointment application
Administration:	Ointment application; 6 weeks and 8 weeks
Result:	Promoted the formation of new bone in the defect area, and the effect was stronger after 8 weeks than at 6 weeks, improving the BV/TV and BMD in the defect area.

REFERENCES

[1]. Hayato Iino, et al. Penetration of Ufenamate into Intact, Stripped, or Delipidized Skin Using Different Vehicles. Bio Pharm Bull. 2015;38(10):1645-8.

[2]. Ting Lü, et al. Photoprotective effect of butyl flufenamate ointment on SKH-1 hairless mice. Chinese Journal of Dermatology ; (12): 711-715, 2013.

[3]. Fan Yang, et al. Topical Application of Butyl Flufenamate Ointment Promotes Cranial Defect Healing in Mice by Inducing BMP2 Secretion in Skin Mesenchymal Stem Cells. Cells. 2022 Nov 15;11(22):3620.

[4]. Iino H, et al. Penetration of Ufenamate into Intact, Stripped, or Delipidized Skin Using Different Vehicles. Biol Pharm Bull. 2015;38(10):1645-8.

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