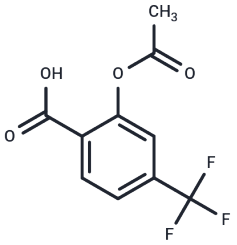
Data Sheet (Cat.No.T0705)

TargetMol

Triflusal

Chemical Properties



CAS No. : 322-79-2

Formula: C10H7F3O4

Molecular Weight: 248.16

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	Triflusal (UR1501) is a 2-acetoxy-4-trifluoromethylbenzoic acid and it is an aspirin chemically-related molecule but not a derivative.
Targets(IC50)	COX,PDE,PPAR
In vitro	The main Triflusal metabolite, HTB, preserves 6-keto-PGF1α synthesis in porcine aortic endothelial cells (PAEC) cells without a significant decline for up to 24 h even at the higher concentration. [1] Triflusal at 10 mM, 100 mM and 1 M decreases LDH efflux in rat brain slices after anoxia/reoxygenation by 24%, 35% and 49% respectively. Triflusal also reduces inducible NO synthase activity by 18%, 21% and 30%. [2]
In vivo	Triflusal (10 mg/kg i.v.) reduces platelet deposition on subendothelium-induced primary thrombus by about 68% in rabbits. Triflusal (10 mg/kg i.v.) reduces platelet deposition on a fresh thrombus formed over tunica media by about 48% in rabbits. Triflusal (40 mg/kg p.o.) reduces platelet deposition on a primary thrombus triggered by subendothelium and tunica media by 53% in rabbits. Triflusal (40 mg/kg p.o.) significantly reduces Cox-2 mRNA levels and protein levels without influence Cox-1 mRNA levels on the vascular wall in rabbits. [1] Triflusal (600 mg/day for 5 days) results in an increase in NO production by neutrophils and an increase in endothelial nitric oxide synthase (eNOS) protein expression in neutrophils in healthy volunteers. [3] Triflusal (300 mg, twice-daily orally) shows a more important increase in total walking distance and in pain-free walking distance over the basal values than those treated with placebo, together with an improvement of the symptomatology correlated with claudication in patients with chronic peripheral arteriopathy. Triflusal (300 mg, twice-daily orally) shows an increase in the peak-flow recorded through strain-gauge plethysmography in patients with chronic peripheral arteriopathy. [4] Triflusal (30 mg/kg) strongly decreases iNOS immunolabeling at both survival times analyzed, attenuating iNOS immunoreactivity in astroglial cells and infiltrated neutrophils in rats. Triflusal (30 mg/kg) decreases neuronal and microglial COX-2 expression at 10 and 24 hours after lesion and microglial and astroglial expression of IL-1beta and TNF-alpha at 24 hours after lesion in rats. [5]

Solubility Information

Solubility	Ethanol: 47 mg/mL (189.39 mM),Sonication is recommended. H2O: < 1 mg/mL (insoluble or slightly soluble), DMSO: 40 mg/mL (161.19 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 2.5 mg/mL (10.07 mM),Sonication is recommended. *Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.*

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	4.0297 mL	20.1483 mL	40.2966 mL
5 mM	0.8059 mL	4.0297 mL	8.0593 mL
10 mM	0.403 mL	2.0148 mL	4.0297 mL
50 mM	0.0806 mL	0.403 mL	0.8059 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Duran X, et al. J Thromb Haemost, 2008, 6(8), 1385-1392.
González-Correa JA, et al. Naunyn Schmiedebergs Arch Pharmacol. 2005 Jan;371(1):81-8.
De Miguel LS, et al. Eur J Clin Invest, 2000, 30(9), 811-817.
Auteri A, et al. Int J Clin Pharmacol Res, 1995, 15(2), 57-63.
Acarin L, et al. Stroke, 2002, 33(10), 2499-2505.