Triflusal

Cat. No.: HY-B0531

CAS No.: 322-79-2

Molecular Formula: $C_{10}H_7F_3O_4$

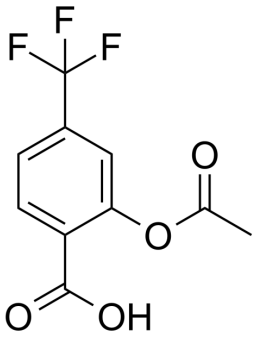
Molecular Weight: 248.16

Target: COX

Pathway: Immunology/Inflammation

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (402.97 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	4.0297 mL	20.1483 mL	40.2966 mL
	5 mM	0.8059 mL	4.0297 mL	8.0593 mL
	10 mM	0.4030 mL	2.0148 mL	4.0297 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 3 mg/mL (12.09 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 3 mg/mL (12.09 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 3 mg/mL (12.09 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Triflusal irreversibly inhibits the production of thromboxane-B2 in platelets by acetylating cycloxygenase-1.Target: COXTriflusal 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% [1].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 [2]. 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].	
IC_{50} & Target	COX-2 280 μM (IC_{50})	COX-2 160 μM (IC_{50}, in human blood)

REFERENCES

[1]. Fernández de Arriba A, et al. Inhibition of cyclooxygenase-2 expression by 4-trifluoromethyl derivatives of salicylate, triflusal, and its deacetylated metabolite, 2-hydroxy-4-trifluoromethylbenzoic acid. Mol Pharmacol. 1999 Apr;55(4):753-60.

[2]. Duran, X., et al., Protective effects of triflusal on secondary thrombus growth and vascular cyclooxygenase-2. J Thromb Haemost, 2008. 6(8): p. 1385-92.

[3]. De Miguel, L.S., et al., A 4-trifluoromethyl derivative of salicylate, triflusal, stimulates nitric oxide production by human neutrophils: role in platelet function. Eur J Clin Invest, 2000. 30(9): p. 811-7.

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

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