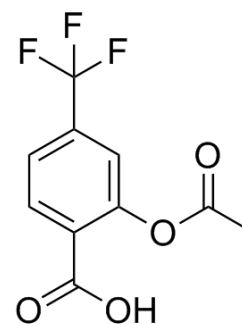


Triflusal

Cat. No.:	HY-B0531		
CAS No.:	322-79-2		
Molecular Formula:	C ₁₀ H ₇ F ₃ O ₄		
Molecular Weight:	248.16		
Target:	COX		
Pathway:	Immunology/Inflammation		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

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

Preparing Stock Solutions	Solvent Concentration	Mass		
		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

- Add each solvent one by one: **10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline**
Solubility: ≥ 3 mg/mL (12.09 mM); Clear solution
- Add each solvent one by one: **10% DMSO >> 90% (20% SBE-β-CD in saline)**
Solubility: ≥ 3 mg/mL (12.09 mM); Clear solution
- 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 cyclooxygenase-1. Target: COX. 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% [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	COX-2
	280 μM (IC ₅₀)	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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