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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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Sudoxicam

Cat. No.: HY-106628

CAS No.: 34042-85-8

Molecular Formula: $C_{13}H_{11}N_3O_4S_2$

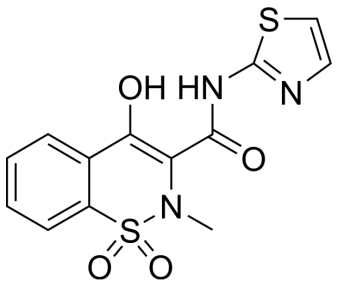
Molecular Weight: 337.37

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 : 83.33 mg/mL (247.00 mM; ultrasonic and warming and heat to 80°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.9641 mL	14.8205 mL	29.6410 mL
5 mM	0.5928 mL	2.9641 mL	5.9282 mL
10 mM	0.2964 mL	1.4821 mL	2.9641 mL

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

BIOLOGICAL ACTIVITY

Description

Sudoxicam is a reversible and orally active COX antagonist and a non-steroidal anti-inflammatory drug (NSAID) from the enol-carboxamide class. Sudoxicam has potent anti-inflammatory, anti-edema and antipyretic activity[1][2][3].

IC_{50} & Target

COX[3]

In Vitro

Sudoxicam demonstrates NADPH-dependent covalent binding to human liver microsomes. With addition of glutathione (GSH) in microsomal incubations, about half of the covalent incorporation of Sudoxicam is blocked by addition of GSH[1].
Metabolite identification studies on [14C]-Sudoxicam in NADPH-supplemented human liver microsomes indicated that the primary route of metabolism involved a P450-mediated thiazole ring scission to the corresponding acylthiourea metabolite (S3), a well-established pro-toxin[1].
In vitro, Sudoxicam underwent the oxidative thiazole-open biotransformation, resulting in the formation of an acylthiourea and the subsequent hydroxylated metabolite[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Sudoxicam (1-10 mg/kg; oral administration; daily; for 7 days; rats) treatment effective reduces plasma inflammation units,

reduces the swelling of inflamed hind-paws and restores toward normal the daily gain in body weight[2].
In the intact rat, Sudoxicam significantly inhibited edema formation at doses as low as 0.1 mg/kg, p.o[2].
Sudoxicam inhibits the erythema caused by ultraviolet irradiation in the guinea pig. Sudoxicam (3.3 mg/kg, i.p.) is capable of counteracting the pyrexia induced by the intraperitoneal injection of typhoid/paratyphoid vaccine in rats, maintaining body temperature about that of uninjected control rats[2].
The plasma half-life of Sudoxicam ranged between 8 hours (monkey), 13 hours (rat), and 60 hours (dog)[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Rats injected with ,i>M. butyrieum[2]
Dosage:	1 mg/kg, 3.3 mg/kg, 10 mg/kg
Administration:	Oral administration; daily; for 7 days
Result:	Were both effective in reducing plasma inflammation units, in reducing the swelling of inflamed hind-paws.

REFERENCES

[1]. Obach RS, et al. In vitro metabolism and covalent binding of enol-carboxamide derivatives and anti-inflammatory agents sudoxicam and meloxicam: insights into the hepatotoxicity of sudoxicam. Chem Res Toxicol. 2008 Sep;21(9):1890-9.

[2]. Wiseman EH, et al. Anti-inflammatory and pharmacokinetic properties of sudoxicam N-(2-thiazolyl)-4-hydroxy-2-methyl-2H-1,2-benzothiazine-3-carboxamide 1,1-dioxide. Biochem Pharmacol. 1972 Sep 1;21(17):2323-34.

[3]. Zhi-Yi Zhang. Sudoxicam. Handbook of Metabolic Pathways of Xenobiotics. September 2014.

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