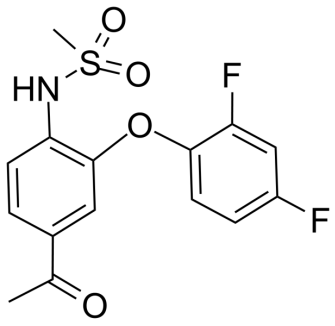
FK 3311

Cat. No.:	HY-14445
CAS No.:	116686-15-8
Molecular Formula:	$C_{15}H_{13}F_2NO_4S$
Molecular Weight:	341.33
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 (292.97 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.9297 mL	14.6486 mL	29.2972 mL
5 mM	0.5859 mL	2.9297 mL	5.8594 mL
10 mM	0.2930 mL	1.4649 mL	2.9297 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: ≥ 2.5 mg/mL (7.32 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.32 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

FK 3311 (COX-2 Inhibitor V) is a selective inhibitor of COX-2 with antiinflammatory agent.

IC_{50} & Target

COX-2

In Vitro

Cyclooxygenase (COX) is an intracellular enzyme that converts arachidonic acid into prostaglandin (PG)G2 and PGH2[1]. The racemic mixtures and the (R)- and (S)-isomers of the 2 metabolites were inactive in the PGE2 test. IC50 values were more than 100 uM for (2 and 5), compared to 1.6 uM for FK 3311 (COX-2 Inhibitor V). Antiinflammatory activity was assessed by inhibition of adjuvant-induced arthritis, and analgesic activity was determined in the acetic acid-induced writhing assay. Following p.o. administration of 10 mg/kg, racemic (2) and its optical isomers showed activity comparable to FK-3311 (76%

	inhibition) in the adjuvant arthritis test, whereas racemic (5) showed very weak activity, and (R)- and (S)-(5) were not tested. With regard to analgesic effects, FK-3311 and racemic (2) showed 81 and 62% inhibitions, respectively, at a dose of 100 mg/kg p.o. The (R)- and (S)-isomers of (2) and racemic (5) all showed 46% inhibition of writhing syndrome. (R)- and (S)-(5) were less active showing 16 and 20% inhibitions, respectively[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	L-PVR, CO, PaO(2), and WDR were significantly better in the FK group than in the control group. Histological tissue edema was mild, and PMN infiltration was significantly reduced in the FK group compared to the control group. The serum TxB(2) levels were significantly lower in the FK group than in the control group, while 6-keto-PGF(1alpha) levels were not significantly reduced. Two-day survival rate was significantly better in the FK group than in the control group[2]. Survival rate was significantly better and serum GOT levels 30 min after reperfusion were significantly lower in the FK high-dose group compared to the other two groups. Four hours after reperfusion, GPT levels and liver tissue flow were significantly better in the FK high-dose group compared to the control. Both 30 min and 4 hr after reperfusion, serum TxB(2) levels were significantly lower in the FK high-dose group compared to the control[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Nakamura K, Ochi T, Matsuo M. [Stereoselective synthesis and pharmacological properties of metabolites of new antiinflammatory agent. 4'-Acetyl-2'-(2,4-difluorophenoxy)methanesulfonanilide (FK3311)]. Yakugaku Zasshi. 1995 Nov;115(11):928-36.

[2]. Sunose Y, Takeyoshi I, Tsutsumi H, Effects of FK3311 on pulmonary ischemia-reperfusion injury in a canine model. J Surg Res. 2001 Feb;95(2):167-73.

[3]. Oshima K, Yabata Y, Yoshinari D, The effects of cyclooxygenase (COX)-2 inhibition on ischemia-reperfusion injury in liver transplantation. J Invest Surg. 2009 Jul-Aug;22(4):239-45.

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