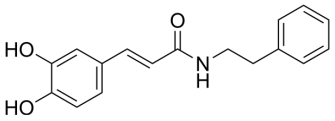
KS370G

Cat. No.:	HY-114683		
CAS No.:	105955-01-9		
Molecular Formula:	$C_{17}H_{17}NO_3$		
Molecular Weight:	283.32		
Target:	Others		
Pathway:	Others		
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 (352.96 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.5296 mL	17.6479 mL	35.2958 mL
	5 mM	0.7059 mL	3.5296 mL	7.0592 mL
	10 mM	0.3530 mL	1.7648 mL	3.5296 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.82 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.82 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

KS370G is an orally active hypoglycemic and cardiovascular protective agent. KS370G improves left ventricular hypertrophy and function in pressure-overload mice heart. KS370G reduces renal obstructive nephropathy[1][2].

In Vivo

KS370G (1 mg/kg; oral; once daily for 8 weeks) improves left ventricular function and inhibited cardiac hypertrophy through the decrease of the phosphorylation of ERK, AKT and GSK3β in pressure-overload mice heart[1].

KS370G (10 mg/kg; oral; once daily for 13 days) attenuates unilateral ureteral obstruction-induced renal fibrosis by the reduction of inflammation and oxidative stress in mice[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Pressure-overload ICR mice model[1]
Dosage:	1 mg/kg
Administration:	Oral gavage, once daily for 8 weeks
Result:	Inhibited cardiac hypertrophy and improved cardiac function induced by pressure overload. Decreased the plasma levels of atrial natriuretic peptide and lactate dehydrogenase. Significantly reduced pressure overload-induced increase of α-SMA and phosphorylation of ERK, AKT and GSK3β. Reduced cardiac collagen accumulation.

Animal Model:	Male ICR mice, unilateral ureteral obstruction (UUO) model[2]
Dosage:	10 mg/kg
Administration:	Oral, once daily for 13 days
Result:	Significantly attenuated collagen deposition in the obstructed kidney and inhibited UUO-induced renal fibrosis markers expression, including fibronectin, type I collagen, vimentin, and α-smooth muscle actin (α-SMA). Significantly lowered the expression of renalinflammatory chemokines/adhesion molecules and monocyte cells marker (MCP-1, VCAM-1, ICAM-1 and CD11b). Reduced renal malondialdehyde levels and reversed the expression of renal antioxidant enzymes (SOD and catalase) after UUO. Significantly inhibited UUOinduced elevated plasma AngII and TGF-β1 levels, TGF-β1 protein expression and Smad3 phosphorylation.

REFERENCES

[1]. Weng YC, et al. KS370G, a synthetic caffeamide derivative, improves left ventricular hypertrophy and function in pressure-overload mice heart. Eur J Pharmacol. 2012 Jun 5;684(1-3):108-15.

[2]. Chuang ST, et al. KS370G, a caffeamide derivative, attenuates unilateral ureteral obstruction-induced renal fibrosis by the reduction of inflammation and oxidative stress in mice. Eur J Pharmacol. 2015 Mar 5;750:1-7.

[3]. Chuang ST, et al. KS370G, a caffeamide derivative, attenuates unilateral ureteral obstruction-induced renal fibrosis by the reduction of inflammation and oxidative stress in mice. Eur J Pharmacol. 2015 Mar 5;750:1-7.

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