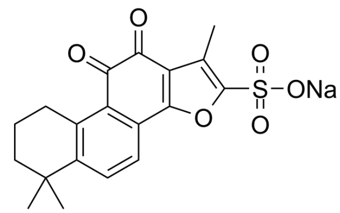
Tanshinone IIA sulfonate sodium

Cat. No.:	HY-N1370
CAS No.:	69659-80-9
Molecular Formula:	$C_{19}H_{17}NaO_6S$
Molecular Weight:	396.39
Target:	CRAC Channel
Pathway:	Membrane Transporter/Ion Channel
Storage:	4°C, sealed storage, away from moisture * The compound is unstable in solutions, freshly prepared is recommended.



SOLVENT & SOLUBILITY

In Vitro

DMSO : 62.5 mg/mL (157.67 mM; Need ultrasonic)
H_2O : 2 mg/mL (5.05 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	2.5228 mL	12.6138 mL	25.2277 mL
5 mM	0.5046 mL	2.5228 mL	5.0455 mL
10 mM	0.2523 mL	1.2614 mL	2.5228 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: ≥ 2.08 mg/mL (5.25 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: 2.08 mg/mL (5.25 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Tanshinone IIA sulfonate (sodium) is a derivative of tanshinone IIA, which acts as an inhibitor of store-operated Ca^{2+} entry (SOCE), and is used to treat cardiovascular disorders.

In Vitro

Sodium Tanshinone IIA sulfonate (12.5 μM) inhibits hypoxia-induced PKG and PPAR-γ downregulation in PASMCs and distal pulmonary arteries of rats. The suppressive effects of Sodium Tanshinone IIA sulfonate on TRPC1 and TRPC6 expression in hypoxic PASMCs can be prevented by specific knockdown PKG or PPAR-γ. The suppressive effects of Sodium Tanshinone IIA sulfonate on basal calcium concentration and SOCE in hypoxic PASMCs can be reversed by specific knockdown of PKG or PPAR-γ. PKG-PPAR-γ signaling axis participates in the suppressive effects of Sodium Tanshinone IIA sulfonate on proliferation in hypoxic PASMCs. PPAR-γ agonist promotes the protective role of Sodium Tanshinone IIA sulfonate on basal $[Ca^{2+}]i$ and SOCE in hypoxic PASMCs[2]. Sodium tanshinone IIA sulfonate inhibits the activity of CYP3A4 in a dose-dependent

	manner by the HLMs and CYP3A4 isoform. The K_M and Vmax values of STS are 54.8±14.6 μM and 0.9±0.1 nmol/mg protein/min, respectively, for the HLMs and 7.5±1.4 μM and 6.8±0.3 nmol/nmol P450/min, respectively, for CYP3A4. CYP1A2, CYP2A6, CYP2C9, CYP2D6, CYP2E1, and CYP2C19 show minimal or no effects on the metabolism of STS[3]. Sodium Tanshinone IIA sulfonate inhibits the activity of CYP3A4 in a dose-dependent manner in the HLMs and CYP3A4 isoform. Sodium Tanshinone IIA sulfonate primarily inhibits the activities of CYP3A4 in vitro, and Sodium Tanshinone IIA sulfonate has the potential to perpetrate drug-drug interactions with other CYP3A4 substrates[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Sodium Tanshinone IIA sulfonate (10 mg/kg, 20 mg/kg) and Donepezil shorten escape latency, increase crossing times of the original position of the platform, and increase the time spent in the target quadrant. Sodium Tanshinone IIA sulfonate decreases the activity of acetylcholinesterase (AChE) and increases the activity of choline acetyltransferase (ChAT) in the hippocampus and cortex of SCOP-treated mice. Sodium Tanshinone IIA sulfonate increases the activity of superoxide dismutase (SOD) and decreases the levels of malondialdehyde (MDA) and reactive oxygen species (ROS) in hippocampus and cortex[1]. Sodium Tanshinone IIA sulfonate prevention (30 mg/kg/day) alleviates hypoxia-induced characteristic changes in chronic hypoxia PH rat model[2]. Sodium Tanshinone IIA sulfonate (20, 10, and 5 mg/kg, i.p.) effectively prevents peritoneal adhesion without affecting anastomotic healing in the rats. Compared with the adhesion model group, the Sodium Tanshinone IIA sulfonate-treated groups show increased peritoneal lavage fluid tPA activity and tPA/PAI-1 ratio in the ischemic tissues with loared TGF-β1 and collagen I expressions in the ischemic tissues[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [1][2]	Mice[1] Male Kunming mice (KM, weighing 35-40 g) are maintained on standard laboratory conditions with free access to water and food. Mice are randomly divided into five groups: vehicle control group (CON, 0.9% saline, n=10), scopolamine group (SCOP, n=10), low dose Sodium Tanshinone IIA sulfonate group (Sodium Tanshinone IIA sulfonate L, SCOP 3 mg/kg + Sodium Tanshinone IIA sulfonate 10 mg/kg, n=10), high dose Sodium Tanshinone IIA sulfonate group (Sodium Tanshinone IIA sulfonate H, SCOP 3 mg/kg + Sodium Tanshinone IIA sulfonate 20 mg/kg, n=10), and Donepezil group (DON, SCOP 3 mg/kg + ARI 3 mg/kg, n=10). Mice are treated with saline, Sodium Tanshinone IIA sulfonate, and Donepezil, respectively, by gavage, once per day for two weeks. SCOP is injected from the eighth day for one week (intraperitoneally, IP). The SCOP is injected 0.5 h before the Morris water maze test. Rats[2] Male Sprague-Dawley rats (200-250 g) are randomly divided into four groups by the random number table: 1) normoxia control group, 2) normoxia + Sodium Tanshinone IIA sulfonate group, 3) hypoxia control group, and 4) hypoxia + Sodium Tanshinone IIA sulfonate group. Groups 1 and 2 are placed in normoxic condition and groups 3 and 4 in a hypoxic cabin with normal pressure, as previously reported, where the oxygen concentration is maintained at 10±1%, in a sustained hypoxic condition for 21 days. Groups 2 and 4, starting from the first day of hypoxia, are, respectively, intraperitoneally injected with 30 mg/kg tanshinone IIA sulfonate; meanwhile, groups 1 and 3 receive the same dose of saline. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Int Immunopharmacol. 2023 Apr 5;118:110111.
- J Cell Mol Med. 2020 Mar;24(6):3328-3335.
- Environ Toxicol. 2023 Nov 21.
- Research Square Preprint. 2020 Dec.
- Oxid Med Cell Longev. 2020 Apr 8;2020:5390482.

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REFERENCES

[1]. Xu QQ, et al. Sodium Tanshinone IIA Sulfonate Attenuates Scopolamine-Induced Cognitive Dysfunctions via Improving Cholinergic System. Biomed Res Int. 2016;2016:9852536

[2]. Jiang Q, et al. Sodium tanshinone IIA sulfonate inhibits hypoxia-induced enhancement of SOCE in pulmonary arterial smooth muscle cells via the PKG-PPAR-γ signaling axis. Am J Physiol Cell Physiol. 2016 Jul 1;311(1):C136-49.

[3]. Ouyang DS, et al. Kinetics of cytochrome P450 enzymes for metabolism of sodium tanshinone IIA sulfonate in vitro. Chin Med. 2016 Mar 22;11:11.

[4]. Chen D, et al. Sodium tanshinone IIA sulfonate and its interactions with human CYP450s. Xenobiotica. 2016 Dec;46(12):1085-1092. Epub 2016 Mar 2.

[5]. Lin S, et al. [Sodium tanshinone IIA sulfonate prevents postoperative peritoneal adhesions in rats by enhancing the activity of the peritoneal fibrinolytic system]. Nan Fang Yi Ke Da Xue Xue Bao. 2016 Feb;36(2):260-4.

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

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