

Salvianolic acid A Datasheet

4th Edition (Revised in July, 2016)



Synonyms:

(2R)-3-(3,4-dihydroxyphenyl)-2-[(E)-3-[2-[(E)-2-(3,4-dihydroxyphenyl)ethenyl]-3,4-dihydro xyphenyl]-1-oxoprop-2-enoxy]propanoic acid.

[Intended Use]

- 1. Reference standards;
- 2. Pharmacological research;
- 3. Synthetic precursor compounds;
- 4. Intermediates & Fine Chemicals;
- 5. Others.

[Source]

The root of Salvia miltiorrhiza Bge.

[Biological Activity or Inhibitors]

Salvianolic acid A (SAA), the water-soluble phenolic acids in Salvia miltiorrhiza, has protection against cerebral lesion, defense from oxidative damage and improvement of remembrance; it also has antithrombotic effect, antiplatelet action and can modulate hemorheology without affecting coagulation system, the mechanisms underlying such activities may involve the induction of cAMP.^[1]

Salvianolic acid A possesses antioxidant activity, also has a significant protective effect against isoproterenol-induced myocardial infarction; it activates the Nrf2/HO-1 axis in RPE cells and protects against oxidative stress via activation of Akt/mTORC1 signaling. ^[2,3]

Salvianolic acid A (oral) can significantly improve glucose metabolism and inhibit oxidative injury as well as protect against impaired vascular responsiveness in STZ-induced diabetic rats.^[4]

Salvianolic acid A has protection on oxidative stress and liver injury induced by carbon tetrachloride in rats, which may mainly be related to its antioxidative effect.^{[5}

Salvianolic acid A inhibits platelet activation via the inhibition of PI3K, and attenuates arterial thrombus formation in vivo, suggests that SAA may be developed as a novel therapeutic agent for the prevention of thrombotic disorders.^[6]

Salvianolic acid A is a novel matrix metalloproteinase-9 inhibitor, can prevents cardiac remodeling in spontaneously hypertensive rats.^[7]

Salvianolic acid A inhibits PDGF-BB-activated HSC proliferation, partially through apoptosis induction, it exerts no direct cytotoxicity on primary hepatocytes and HSC-T6 cells under experimental concentrations. ^[8]

[Solvent]

Pyridine, DMSO, Methanol, etc.

[HPLC Method]^[9]

Mobile phase: Acetonitrile- 1% Acetic acid H2O=30:70;

Flow rate: 0.8 ml/min;

Column temperature: 30°C;

The wave length of determination: 280 nm.

[Storage]

2-8°C, Protected from air and light, refrigerate or freeze.

[References]

[1] Fan H, Fu F, Yang M, et al. Thromb. Res., 2010, 126(1):17-22.

[2] Wang S B, Tian S, Fan Y, et al. Eur. J. Pharmacol., 2009, 615(1-3):125-32.

[3] Zhang H, Liu Y Y, Jiang Q, et al. Free Radical Biol. Med., 2014, 69(4):219-28.

[4] Wang S B, Yang X Y, Tian S, et al. Life Sci., 2009, 85(13–14):499-504.

[5] Wu Z M, Wen T, Tan Y F, et al. Basic Clin. Pharmacol. Toxicol., 2007, 100(2):115-20.

[6] Huang ZS, Zeng CL, Zhu LJ, et al. J. Throm. Haemost., 2010, 8(6):1383-93.

[7] Jiang B, Li D, Deng Y, et al. Plos One, 2013, 8(3):e59621-e59621.

[8] Lin Y L, Lee T F, Huang Y J, et al. J. Pharm. Pharmacol., 2006, 58(7):933-9.

[9] Wang Z, Xu Y, Jiao R, et al. China Pharmacist, 2014(09):1473-5.

[Contact]

Address: S5-3 Building, No. 111, Dongfeng Rd., Wuhan Economic and Technological Development Zone, Wuhan, Hubei 430056, China Email: info@chemfaces.com Tel: +86-27-84237783 Fax: +86-27-84254680 Web: www.chemfaces.com Tech Support: service@chemfaces.com