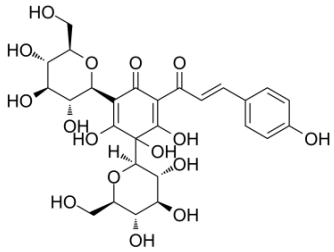
Hydroxysafflor yellow A

Cat. No.:	HY-N0567
CAS No.:	78281-02-4
Molecular Formula:	$C_{27}H_{32}O_{16}$
Molecular Weight:	612.53
Target:	Others
Pathway:	Others
Storage:	4°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 34 mg/mL (55.51 mM)

H_2O : 33.33 mg/mL (54.41 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.6326 mL	8.1629 mL	16.3257 mL
	5 mM	0.3265 mL	1.6326 mL	3.2651 mL
	10 mM	0.1633 mL	0.8163 mL	1.6326 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 (4.08 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (4.08 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Hydroxysafflor yellow A is a flavonoid derived and isolated from traditional Chinese medicine Carthamus tinctorius L., possesses anti-tumor activity. IC50 value:Target: in vitro: HYSA could inhibit LPS-induced VSMCs proliferation and migration, accompanied by the downregulated levels of several key pro-inflammatory cytokines, including TNF-α, IL-6, and IL-8. We further showed that HYSA inhibited LPS-induced upregulation of TLR-4 expression as well as the activation of Rac1/Akt pathway [1]. HSYA protected EC viability against LPS-induced injury (P<0.05). LPS-induced NF-κB p65 subunit DNA binding (P<0.01) and nuclear factor of kappa light polypeptide gene enhancer in B-cells inhibitor -α (I-κB-α) phosphorylation was inhibited by HSYA. HSYA attenuated LPS triggered ICAM-1 and E-selectin mRNA levels elevation and phosphorylation of p38 MAPK or c-Jun N-terminal kinase MAPK [2]. HSYA inhibited the proliferation of 3T3-L1 preadipocytes and cell viability greatly decreased in a dose and time dependent manner. HSYA (1 mg/l) notably reduced the amount of intracellular lipid and

triglyceride content in adipocytes by 21.3 % (2.13 ± 0.36 vs 2.71 ± 0.40, P < 0.01) and 22.6 % (1.33 ± 0.07 vs 1.72 ± 0.07, P < 0.01) on days 8 following the differentiation, respectively [3]. in vivo: HSYA treatment ameliorated serum biochemical indicators by reducing the levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), hyaluronan (HA), laminin (LN), and type III precollagen (III-C) in rats [4].

CUSTOMER VALIDATION

- Acta Pharm Sin B. 2021 Jan;11(1):143-155.
- Pharmacol Res. 2020 May;155:104751.
- Am J Transl Res. 2020 Aug 15;12(8):4781-4794.
- Cell Biochem Biophys. 2020 Dec;78(4):511-520.

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REFERENCES

[1]. Yang G, et al. Hydroxysafflor yellow A inhibits lipopolysaccharide-induced proliferation and migration of vascular smooth muscle cells via Toll-like receptor-4 pathway. Int J Clin Exp Med. 2015 Apr 15;8(4):5295-302.

[2]. Zhu HJ, et al. Hydroxysafflor yellow A (HYSA) inhibited the proliferation and differentiation of 3T3-L1 preadipocytes. Cytotechnology. 2015 Mar 7.

[3]. He Y, et al. Protective effects of hydroxysafflor yellow A (HSYA) on alcohol-induced liver injury in rats. J Physiol Biochem. 2015 Mar;71(1):69-78.

[4]. Jin M, et al. Hydroxysafflor yellow A attenuate lipopolysaccharide-induced endothelium inflammatory injury. Chin J Integr Med. 2015 May 27.

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