

Product Data Sheet

Product Name: Resorcinolnaphthalein

Cat. No.: GC11560

Chemical Properties

Cas. No. 41307-63-5

Chemical Name 3',6'-dihydroxy-spiro[1H,3H-naphtho[1,8-cd]pyran-1,9'-[9H]xanthen-3-one

SMILES Oc1ccc2c(c1)Oc1cc(O)ccc1C12OC(=O)c2cccc3cccc1c23

Formula $C_{24}H_{14}O_5$ M.Wt 382.4

Solubility $\leq 20\text{mg/ml}$ in ethanol; 20mg/ml in DMSO; 20mg/ml in dimethyl formamide

Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice. All other available size: ship with RT, or blue ice upon request.

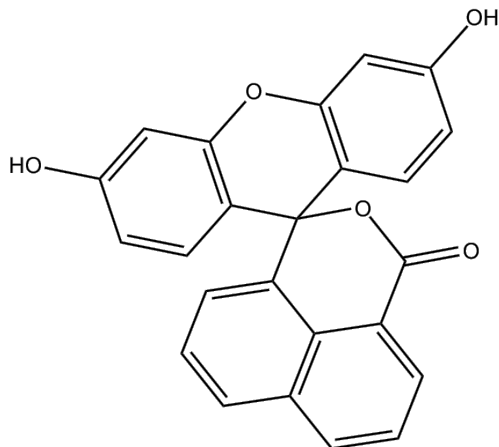
Caution: Product has not been fully validated for medical applications. For research use only.

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Structure



Background

Resorcinolnaphthalein is an Angiotensin-converting enzyme 2 (ACE2) activator [1].

Angiotensin-converting enzyme 2 (ACE2) has been involved in hydrolyzing angiotensin II and opposing its actions, and plays a protective role in the pathogenesis of pulmonary arterial hypertension (PAH) [2]. Targeted disruption of ACE2 in mice results in a severe cardiac contractility defect, increased angiotensin II levels, and upregulation of hypoxia-induced genes in the heart [2].

In vitro: Resorcinolnaphthalein enhanced ACE2 activity in a dose-dependent manner [1].

In vivo: Resorcinolnaphthalein caused significant activation of ACE2 in both normal and diseased rats in 7 days after treatment. Treatment with resorcinolnaphthalein prevented high PAP, right ventricular hypertrophy and neointimal formation. Resorcinolnaphthalein caused an improved endothelia-dependent vasorelaxation and decreased in proinflammatory cytokines, such as TNF- α , MCP-1, IL-6, and increased in anti-inflammatory cytokine IL-10 in the early stage of the pathogenesis [3]. Resorcinolnaphthalein can specifically and effectively enhanced ACE2 activity in rats [4]. ACE2 activation by resorcinolnaphthalein showed effects on pulmonary endothelial dysfunction and

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neointimal formation during the development of severe PAH in rats [4].

References:

- [1] Prada J A H, Ferreira A J, Katovich M J, et al. Structure-based identification of small-molecule angiotensin-converting enzyme 2 activators as novel antihypertensive agents[J]. Hypertension, 2008, 51(5): 1312-1317.
- [2] Crackower M A, Sarao R, Oudit G Y, et al. Angiotensin-converting enzyme 2 is an essential regulator of heart function[J]. Nature, 2002, 417(6891): 822-828.
- [3] Haber P K, Ye M, Wysocki J, et al. Angiotensin-converting enzyme 2-independent action of presumed angiotensin-converting enzyme 2 activators Novelty and significance[J]. Hypertension, 2014, 63(4): 774-782.
- [4] Li G, Liu Y, Zhu Y, et al. ACE2 activation confers endothelial protection and attenuates neointimal lesions in prevention of severe pulmonary arterial hypertension in rats[J]. Lung, 2013, 191(4): 327-336.

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