

Dihydrotanshinone I Datasheet

4th Edition (Revised in July, 2016)

[Product Information]

Name: Dihydrotanshinone I

Catalog No.: CFN90162

Cas No.: 20958-18-3

Purity: > 98%

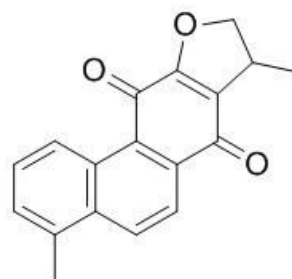
M.F: C₁₈H₁₄O₃

M.W: 278.30

Physical Description: Red powder

Synonyms:

4,8-dimethyl-8,9-dihydronaphtho[2,1-f]benzofuran-7,11-dione



[Intended Use]

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

[Source]

The roots of *Salvia miltiorrhiza*

[Biological Activity or Inhibitors]

Dihydrotanshinone I and cryptotanshinone, constituents of a medicinal plant, *Salvia miltiorrhiza* Bunge, have antibacterial activity against a broad range of Gram positive bacteria, they have non-selective inhibition against DNA, RNA, and protein syntheses in *B. subtilis*, suggest that superoxide radicals are important in the antibacterial actions of the agents.^[1]

Dihydrotanshinone I induces topoisomerase I-mediated DNA cleavage as strongly as camptothecin; and inhibits the catalytic activity of topoisomerase I by the formation of a cleavable complex and at least in part through the intercalation into DNA.^[2]

Dihydrotanshinone I (DI) has cytotoxicity to a variety of tumor cells, DI (with an IC₅₀ value of approximately 1.28 µg/ml) could inhibit angiogenesis through suppressing endothelial cell proliferation, migration, invasion and tube formation, indicating that DI has a potential to be developed as a novel anti-angiogenic agent.^[3]

Dihydrotanshinone I as an inhibitor of NF-κB activation through our research on *Salvia miltiorrhiza* Bunge, it significantly inhibits the expression of NF-κB reporter gene induced by TNF-α in a dose-dependent manner, also inhibits TNF-α induced phosphorylation and degradation of IκBα, phosphorylation and nuclear translocation of p65; it suppresses the growth of HeLa cells in a xenograft tumor model, which could be correlated with its modulation of TNF-α production, taken together, dihydrotanshinone I could be a valuable candidate for the intervention of NF-κB-dependent pathological conditions such as inflammation and cancer.^[4]

[Solvent]

Chloroform, Dichloromethane, Ethyl Acetate, DMSO, Acetone, etc.

[HPLC Method]^[5]

Mobile phase: Acetonitrile- H₂O= 55:45 ;

Flow rate: 1.0 ml/min;

Column temperature: Room Temperature;

The wave length of determination: 245 nm.

[Storage]

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

[References]

- [1] Lee D S, Lee S H, Noh J G, *et al. Biosci. Biotechn. Bioch.*, 2014, 63(12):2236-9.
- [2] Lee D S, Lee S H, Kwon G S, *et al. Biosci. Biotechn. Bioch.*, 1999, 63(8):1370-3.
- [3]Weipeng, Bian, Chen, *et al. Acta Bioch. Et. Bioph. Sin.*, 2008, 40(1):1-6.
- [4] Wang F, Ma J, Wang KS, *et al. Int. Immunopharmacol.*, 2015, 28(1):764-72.
- [5] Zhu D, Tan S. *China Pharmacist*, 2008, 11(03):301-3.

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