

Dihydrotanshinone I Datasheet

4th Edition (Revised in July, 2016)

[Product Information]

Name: Dihydrotanshinone I

Catalog No.: CFN97435

Cas No.: 87205-99-0

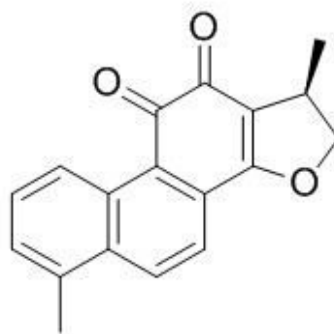
Purity: > 98%

M.F: C₁₈H₁₄O₃

M.W: 278.3

Physical Description: Red powder

Synonyms: 1,6-Dimethyl-1,2-dihydronaphtho[1,2-g]benzofuran-10,11-dione.



[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]

Dihydrotanshinone I (DI) and cryptotanshinone, constituents of a medicinal plant, *Salvia miltiorrhiza* Bunge, have antibacterial activity against a broad range of Gram positive bacteria, they generate superoxide radicals in *Bacillus subtilis* lysates, the superoxide radicals are important in the antibacterial actions of the agents.^[1]

Dihydrotanshinone I induces topoisomerase I-mediated DNA cleavage as strongly as camptothecin, but topoisomerase II-mediated DNA cleavage was not affected, dihydrotanshinone I 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 has cytotoxicity to a variety of tumor cells, it induces a potent cytotoxicity to human umbilical vein endothelial cells with an IC₅₀ value of approximately 1.28 µg/ml; it can 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 significantly impairs activation of extracellular signal-regulated kinase 1/2 (ERK1/2), p38 and stress-activated protein kinase/c-Jun NH₂-terminal kinase (JNK/SAPK), it suppresses the growth of HeLa cells in a xenograft tumor model, which could be correlated with its modulation of TNF-α production, suggests that dihydrotanshinone I could be a valuable candidate for the intervention of NF-κB-dependent pathological conditions such as inflammation and cancer.^[4]

Dihydrotanshinone I induces cell growth arrest during the S phase and subsequently, apoptosis, following its application to K562/ADR cells, exhibits cytotoxicity against various tumor cell lines.^[5]

Dihydrotanshinone I combines irradiation, which can enhance apoptotic effects in human cervical cancer by HPV E6 down-regulation and caspases activation.^[6]

Dihydrotanshinone I and cryptotanshinone exhibit strong inhibition towards human liver microsome (HLM)-catalyzed propofol glucuronidation, and dihydrotanshinone I exhibits strong inhibition towards UDP-glucuronosyltransferase (UGT) 1A7.^[7,8]

Dihydrotanshinone-I interferes with the RNA-binding activity of HuR affecting its post-transcriptional function.^[9]

Dihydrotanshinone I induces caspase and ROS dependent apoptosis and autophagy,

mediated by mitochondria in colon cancer; it could be a promising leading compound for the development of anti-tumor agent or be developed as an adjuvant drug for colon cancer therapy.^[10]

Dihydrotanshinone I has inhibitory effects on CYP4A11 and CYP4F3A mediated ω -hydroxylation of arachidonic acid.^[11]

[Solvent]

Chloroform, Dichloromethane, Diethyl ether, DMSO, Acetone, etc.

[HPLC Method]^[12]

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

[References]

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