

Product Data Sheet

Product Name: ddhCTP trisodium solution (100 mM $\pm$ 3 mM)

Cat. No.: GC81395

Chemical Properties

Cas. No.

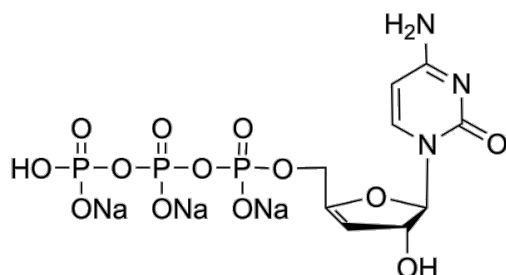
Formula $C_9H_{11}N_3Na_3O_{13}P_3$ M.Wt 531.09

Solubility H_2O : 3.33 mg/mL (6.27 mM; Need ultrasonic) Storage Store at $-20^{\circ}C$

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

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

Structure



Background

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

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ddhCTP trisodium solution (100 mM $\pm$ 3 mM) is an endogenously produced pyrimidine base analog with a K_d of 17.0 nM for LLDH and an IC_{50} of 55.8 μ M for GAPDH. By inhibiting key metabolic enzymes such as GAPDH, ddhCTP trisodium reduces glycolytic flux and shifts metabolic flow toward the pentose phosphate pathway, thereby regulating the redox balance of cells. As a competitive CTP analog, ddhCTP trisodium terminates RNA synthesis by flavivirus RdRps and SARS-CoV-2 RdRp, and inhibits Zika virus replication in vivo. ddhCTP trisodium can be used in studies related to viral infections, COVID-19 and Zika virus infections [1] [2].

In Vitro, ddhCTP (24-96 h) trisodium completely abolishes the extracellular levels of phenylacetic acid and 4-hydroxyphenyllactic acid in HEK293 cells expressing wild-type RSAD2^[1]. ddhCTP (72 h) trisodium modulates the levels of NAD⁺-dependent enzyme-related metabolites, including decreased taurine levels, increased 2-oxoglutarate levels, increased glyceraldehyde 3-phosphate levels, and increased levels of pentose phosphate pathway intermediates in hiPSC-derived macrophages expressing wild-type RSAD2 after stimulation with LPS and IFN- γ ^[1]. ddhCTP trisodium inhibits the NAD⁺-dependent forward activity of purified rabbit muscle GAPDH with an IC_{50} of 55.8 μ M, but does not affect the NADH-dependent reverse reaction^[1]. ddhCTP (72 μ M, 30 s) trisodium inhibits the NAD⁺-dependent activities of purified rabbit muscle LLDH and purified bovine heart MDH, but does not affect the NADH-dependent reverse reaction of LLDH^[1].

References:

- [1]. Honarmand Ebrahimi K, et al. ddhCTP produced by the radical-SAM activity of RSAD2 (viperin) inhibits the NAD⁺ -dependent activity of enzymes to modulate metabolism. FEBS Lett. 2020;594(10):1631-1644.
- [2]. Ciardullo G, et al. Viral RNA Replication Suppression of SARS-CoV-2: Atomistic Insights into Inhibition Mechanisms of RdRp Machinery by ddhCTP. J Chem Inf Model. 2024;64(5):1593-1604.

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