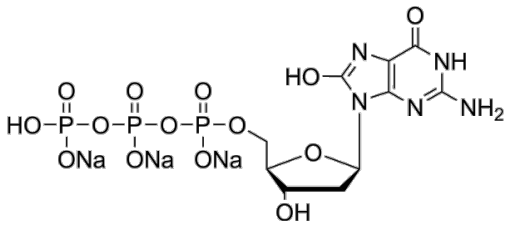
Product Data Sheet

Product Name: 8-Oxo-dGTP trisodium solution (100 mM)
Cat. No.: GC81392

Chemical Properties

Cas. No.			
Formula	$C_{10}H_{13}N_5Na_3O_{14}P_3$	M.Wt	589.13
Solubility		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.		
Structure			



Background

8-Oxo-dGTP (8-Oxo-Deoxyguanosine triphosphate) trisodium solution (100mM) is an oxidized guanine nucleotide formed by ROS-mediated oxidative modification of dGTP, and it also serves as a key substrate for 8-oxo-dGTP pyrophosphohydrolases (such as hMTH1 and E. coli MutT). 8-Oxo-dGTP trisodium solution (100mM) acts as a DNA mutagen, inserts into nascent DNA and pairs with adenine and cytosine, inducing A:T to C:G transversion mutations. Furthermore, 8-Oxo-dGTP trisodium solution (100mM) causes oxidative DNA base modification, strand breakage and S-phase arrest, and ultimately triggers AIF -mediated apoptosis and promotes spontaneous carcinogenesis in mth1-deficient mice. Accumulation of 8-Oxo-dGTP trisodium solution (100mM) in cells induces

genomic instability, but it exhibits a tumor-suppressive effect that reduces tumor incidence in mouse models instead. 8-Oxo-dGTP trisodium solution (100mM) is widely used in studies related to spontaneous carcinogenesis, Parkinson's disease, Alzheimer's disease, heart failure and tumor mechanisms [1] [2] [3] [4].

In Vivo, 8-oxo-dGTP (0.5 mg/kg; intravenous injection; once every 10 days; for 17 months) trisodium reduces the spontaneous tumor incidence rate to 19.4% in MTH1 knockout mice and to 14.7% in wild-type mice after 17 months of administration[4]. 8-oxo-dGTP (0.5 mg/kg; intravenous injection; once every 3 days; for 32 consecutive days) trisodium inhibits the growth of subcutaneous HeLa-shMTH1 xenografts in nude mice[4]. 8-oxo-dGTP (0.5-2.5 mg/kg; intravenous injection; once every 5 days; for 112 consecutive days) trisodium dose-dependently inhibits spontaneous intestinal adenoma formation[4].

In Vitro, 8-Oxo-dGTP trisodium solution (100mM) (100 μM) binds to wild-type hMTH1 with high affinity (Kd=0.08 μM), while the F27A and D119A mutations reduce the binding affinity, and the W117A mutation completely abolishes the binding activity[1]. 8-Oxo-dGTP trisodium solution (100mM) (2-8 mM; 24 h) inhibits the viability of HeLa-shGFP and HeLa-shMTH1 cells in a dose-dependent manner, with the inhibitory effects at concentrations of 6 mM and 8 mM being stronger than that of 50 μg/mL 5-FU[4]. 8-Oxo-dGTP trisodium solution (100mM) (2 mM, 6 mM; 8-24 h) increases the level of 8-oxo-dG in DNA of HeLa-shGFP cells in a dose- and time-dependent manner, and this elevation reaches comparable levels in HeLa cells with MTH1, OGG1 or MUTYH knockdown after 24 h of treatment[4]. 8-Oxo-dGTP trisodium solution (100mM) (6 mM; 24 h) induces S-phase cell cycle arrest in HeLa-shGFP and HeLa-shMTH1 cells, with a more pronounced effect in MTH1-knockdown HeLa cells; treatment with 6 mM 8-oxo-dGTP for 12 h also induces S-phase arrest in double thymidine-synchronized HeLa-shGFP and HeLa-shMTH1 cells[4]. 8-Oxo-dGTP trisodium solution (100mM) (2 mM, 6 mM; 24 h) induces apoptosis in HeLa-shGFP, HeLa-shMTH1, HeLa-shOGG1 and HeLa-shMUTYH cells via an AIF-mediated caspase-independent pathway, which is evidenced by elevated levels of cleaved PARP and nuclear translocation of AIF[4].

References:

[1]. Sakai Y, et al. A molecular basis for the selective recognition of 2-hydroxy-dATP and 8-oxo-dGTP by human MTH1. J Biol Chem. 2002;277(10):8579-8587.

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[2]. Bialkowski K, et al. A novel assay of 8-oxo-2′-deoxyguanosine 5′-triphosphate pyrophosphohydrolase (8-oxo-dGTPase) activity in cultured cells and its use for evaluation of cadmium (II) inhibition of this activity[J]. Nucleic acids research, 1998, 26(13): 3194-3201.
[3]. Tsutsui H, et al. 8-oxo-dGTPase, which prevents oxidative stress-induced DNA damage, increases in the mitochondria from failing hearts. Circulation. 2001;104(24):2883-2885.
[4]. Li J, et al. 8-oxo-dGTP curbs tumor development via S phase arrest and AIF-mediated apoptosis. Free Radic Biol Med. 2023;196:53-64.

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