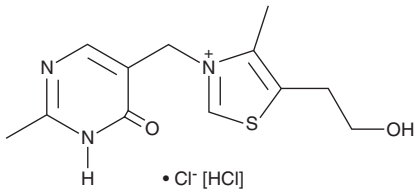
PRODUCT INFORMATION

Oxythiamine (chloride hydrochloride)

Item No. 29953

CAS Registry No.: 614-05-1
Formal Name: 3-[(1,6-dihydro-2-methyl-6-oxo-5-pyrimidinyl)methyl]-5-(2-hydroxyethyl)-4-methyl-thiazolium, monochloride, monohydrochloride
MF: $C_{12}H_{16}N_3O_2S$ • Cl [HCl]
FW: 338.3
Purity: ≥95%
UV/Vis.: λ_{max}: 221, 270 nm
Supplied as: A solid
Storage: -20°C
Stability: ≥2 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

Oxythiamine (chloride hydrochloride) is supplied as a solid. Aqueous solutions of oxythiamine (chloride hydrochloride) can be prepared by directly dissolving the solid in aqueous buffers. The solubility of oxythiamine (chloride hydrochloride) in PBS, pH 7.2, is approximately 10 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

Oxythiamine is a thiamine antimetabolite that has anticancer activities.[1-3] It is converted by thiamine pyrophosphokinase to oxythiamine pyrophosphate, a transketolase inhibitor.[1] Oxythiamine (5 mM) decreases transketolase activity in and is cytotoxic to MDA-MB-231 breast cancer cells.[4] It inhibits the nonoxidative synthesis of ribose and decreases RNA and DNA synthesis in MIA PaCa-2 pancreatic cancer cells when used at a concentration of 0.5 µM.[2] *In vivo*, oxythiamine (400 and 500 mg/kg per day) induces cell cycle arrest at the G_1 phase and apoptosis in an Ehrlich murine spontaneous adenocarcinoma model.[3] Oxythiamine, in combination with sorafenib, reduces tumor growth in an SMMC mouse xenograft model.[5] It has also been used to induce thiamine deficiency in mice.[6]

References

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2. Boros, L.G., Puigjaner, J., Cascante, M., *et al.* Oxythiamine and dehydroepiandrosterone inhibit the nonoxidative synthesis of ribose and tumor cell proliferation. *Cancer Res.* **57(19)**, 4242-4248 (1997).
3. Raïs, B., Comin, B., Puigjaner, J., *et al.* Oxythiamine and dehydroepiandrosterone induce a G1 phase cycle arrest in Ehrlich's tumor cells through inhibition of the pentose cycle. *FEBS Lett.* **456(1)**, 113-118 (1999).
4. Tseng, C.-W., Kuo, W.-H., Chan, S.-H., *et al.* Transketolase regulates the metabolic switch to control breast cancer cell metastasis via the α-ketoglutarate signaling pathway. *Cancer Res.* **78(11)**, 2799-2812 (2018).
5. Xu, I.M.-J., Lai, R.K.-H., Lin, S.-H., *et al.* Transketolase counteracts oxidative stress to drive cancer development. *Proc. Natl. Acad. Sci. USA* **113(6)**, E725-E734 (2016).
6. Cerecedo, L.R., Soodak, M., and Eusebi, A.J. Studies on thiamine analogues. I. Experiments in vivo. *J. Biol. Chem.* **189(1)**, 293-299 (1951).

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