L-Thyroxine sodium

Cat. No.: HY-18341B

CAS No.: 55-03-8

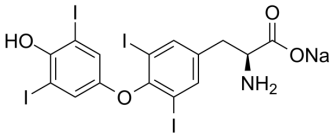
Molecular Formula: $C_{15}H_{10}I_4NNaO_4$

Molecular Weight: 798.85

Target: Thyroid Hormone Receptor; Endogenous Metabolite

Pathway: Vitamin D Related/Nuclear Receptor; Metabolic Enzyme/Protease

Storage: 4°C, sealed storage, away from moisture and light
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 62.5 mg/mL (78.24 mM; Need ultrasonic)
0.5 M NaOH : 25 mg/mL (31.29 mM; ultrasonic and warming and adjust pH to 11 with NaOH and heat to 60°C)
H_2O : 14 mg/mL (17.53 mM; ultrasonic and adjust pH to 12 with NaOH)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.2518 mL	6.2590 mL	12.5180 mL
5 mM	0.2504 mL	1.2518 mL	2.5036 mL
10 mM	0.1252 mL	0.6259 mL	1.2518 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (2.60 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.08 mg/mL (2.60 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

L-Thyroxine sodium (Levothyroxine sodium) is a synthetic hormone for the research of hypothyroidism. DIO enzymes convert biologically active thyroid hormone (Triiodothyronine,T3) from L-Thyroxine (T4)[1].

IC_{50} & Target

Human Endogenous Metabolite

In Vivo

Deiodinases (DIOs), which catalyse the conversion of thyroxine (pro-hormone) to the active thyroid hormone, are associated with thyroid stimulating hormone (TSH) levels. DIO1 and DIO2 catalyze activation of thyroid hormone secretion in contrast to DIO3 playing role inactivation of the secretion. Activities of DIO1 and DIO2 play pivotal role in the negative feedback regulation of pituitary TSH secretion[1]. L-Thyroxine (T4) and Triiodothyronine (T3) hormones are known to modulate the

expression of ionic channels, pumps and regulatory contractile proteins. Moreover, thyroid hormones have been shown to influence calcium homeostasis and flux responsible for excitation and contractility, with L-Thyroxine and Triiodothyronine modulating its pharmacological control and secretion. In rats fed 12 weeks with the iodine-free diet, a significant decrease in the levels of both Triiodothyronine and L-Thyroxine is observed when compared to the control group fed with standard diet ($p<0.001$). In the group treated with low doses of L-Thyroxine, an increase in L-Thyroxine levels is observed ($p=0.02$) while Triiodothyronine levels remain virtually similar to the control group ($p=0.19$). Rats treated with high doses of L-Thyroxine display a significant increase in both Triiodothyronine and L-Thyroxine circulating concentrations compared to the non-treated hypothyroid group ($p<0.001$ and $p=0.004$, respectively) and a significant increase in L-Thyroxine levels when compared to the control values ($p=0.03$)[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Immunol. 2024 Mar 18.
- Cell Rep Med. 2023 May 24;101061.
- J Drug Deliv Sci Technol. 2023 Sep 28, 105008.
- Stem Cell Rev Rep. 2021 Jun;17(3):999-1013.
- Sci Rep. 2022 Jul 4;12(1):11259.

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REFERENCES

[1]. Arici M, et al. Association between genetic polymorphism and levothyroxine bioavailability in hypothyroid patients. Endocr J. 2018 Mar 28;65(3):317-323.

[2]. Corriveau S, et al. Levothyroxine treatment generates an abnormal uterine contractility patterns in an in vitro animalmodel. J Clin Transl Endocrinol. 2015 Sep 9;2(4):144-149.

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