Chlorotrianisene

Cat. No.: HY-B2158

CAS No.: 569-57-3

Molecular Formula: $C_{23}H_{21}ClO_3$

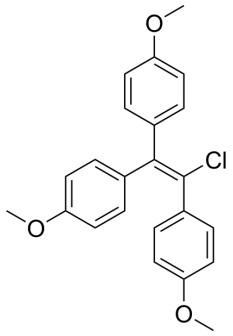
Molecular Weight: 380.86

Target: Estrogen Receptor/ERR; COX

Pathway: Vitamin D Related/Nuclear Receptor; Immunology/Inflammation

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (262.56 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6256 mL	13.1282 mL	26.2564 mL
	5 mM	0.5251 mL	2.6256 mL	5.2513 mL
	10 mM	0.2626 mL	1.3128 mL	2.6256 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (6.56 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.56 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Chlorotrianisene is a long-acting non-steroidal estrogen and an orally active estrogen receptor modulator. Chlorotrianisene exhibits antiestrogenic activity. Chlorotrianisene potently inhibits the enzyme COX-1 and inhibits platelet aggregation in whole blood[1][2][3].

IC_{50} & Target

Estrogen receptor[1]
COX-1[2]

In Vitro

Comparison of intracellular estrogen receptor (ER) affinities of Chlorotrianisene with respective rat uterine cytosolic ER affinities has initially suggested the potential for activation of ER as a mechanism of growth stimulation. Chlorotrianisene exhibits concentration dependent cell growth stimulation with an EC_{50} of 28 nM and a K_i of 500 nM in MCF-7 cells[1].

	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	The incubation of Chlorotrianisene with rat liver microsomes and NADPH generates a reactive intermediate which binds covalently to proteins. Intermediate may inactivate the uterine estrogen receptors (ER). The incubation of Chlorotrianisene with rat liver microsomes and NADPH in the presence of rat uteri, under conditions which generate intermediate, markedly decreased the binding capacity of the ER for [^{3}H]estradiol (E2)[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Ruenitz PC, et al. Estrogenic tamoxifen derivatives: categorization of intrinsic estrogenicity in MCF-7 cells. J Steroid Biochem Mol Biol. 1997 Nov-Dec;63(4-6):203-9.

[2]. Lounkine E, et al. Large-scale prediction and testing of drug activity on side-effect targets. Nature. 2012 Jun 10;486(7403):361-7.

[3]. Kupfer D, et al. Inactivation of the uterine estrogen receptor binding of estradiol during P-450 catalyzed metabolism of chlorotrianisene (TACE). Speculation that TACE antiestrogenic activity involves covalent binding to the estrogen receptor. FEBS Lett. 1990 Feb 12;261(1):59-62.

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