Acolbifene

Cat. No.: HY-16023A

CAS No.: 182167-02-8

Molecular Formula: $C_{29}H_{31}NO_4$

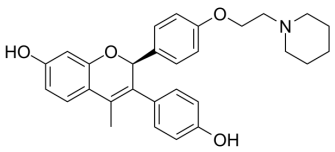
Molecular Weight: 457.56

Target: Estrogen Receptor/ERR

Pathway: Vitamin D Related/Nuclear Receptor

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 : 50 mg/mL (109.28 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.1855 mL	10.9275 mL	21.8551 mL
	5 mM	0.4371 mL	2.1855 mL	4.3710 mL
	10 mM	0.2186 mL	1.0928 mL	2.1855 mL

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

BIOLOGICAL ACTIVITY

Description

Acolbifene (EM-652), the active metabolite of EM800, is an orally active pure antiestrogen and selective estrogen receptor antagonist. Acolbifene (EM-652) inhibits estradiol (E2)-induced transcriptional activity of ERα (IC_{50} = 2 nM) and ERβ (IC_{50} = 0.4 nM). Acolbifene (EM-652) possesses potent and pure anticarcinogenic properties[1][2][3][4][5].

IC_{50} & Target

ERα	ERβ
2 nM (IC_{50}, E2-induced transcriptional activity)	0.4 nM (IC_{50}, E2-induced transcriptional activity)

In Vitro

Acolbifene (ACOL) does not affect pathways of cholesterol synthesis, supporting the involvement of the clearance-related receptors in its hypocholesterolemic action[2].

Acolbifene (EM-652) shows no agonistic activity on ERα and ERβ transcriptional function and blocks the estradiol (E2)-mediated activation of both ERα and ERβ[3].

Acolbifene (EM-652) shows the most potent inhibition of estradiol-stimulated cell proliferation in human breast cancer cancer cells (ZR-75-1, MCF-7, T-47D) and is devoid of any intrinsic estrogenic activity[4].

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

In Vivo

Acolbifene (ACOL) reduces food intake and strongly decreases cholesterolemia in rats fed a cholesterol-free diet[2].
Acolbifene (ACOL) reduces food intake (16%) and weight gain (45%, mainly fat) similarly in both dietary cohorts[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Female Sprague-Dawley rats (n = 42) initially weighing 175-200 g[2].
Dosage:	2.5 mg/kg.
Administration:	Oral gavage, once daily for 21 d.
Result:	Prevents tumor growth in rats.

CUSTOMER VALIDATION

- Int J Mol Sci. 2022 Oct 6;23(19):11892.

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REFERENCES

[1]. Wang T, et al. Recent advances in selective estrogen receptor modulators for breast cancer. Mini Rev Med Chem. 2009 Sep;9(10):1191-201.

[2]. Christian Lemieux, et al. The selective estrogen receptor modulator acolbifene reduces cholesterolemia independently of its anorectic action in control and cholesterol-fed rats. J Nutr. 2005 Sep;135(9):2225-9.

[3]. A Tremblay, et al. EM-800, a novel antiestrogen, acts as a pure antagonist of the transcriptional functions of estrogen receptors alpha and beta. Endocrinology. 1998 Jan;139(1):111-8.

[4]. Sylvain Gauthier, et al. Synthesis and structure-activity relationships of analogs of EM-652 (acolbifene), a pure selective estrogen receptor modulator. Study of nitrogen substitution. J Enzyme Inhib Med Chem. 2005 Apr;20(2):165-77.

[5]. F Labrie, et al. EM-652 (SCH 57068), a third generation SERM acting as pure antiestrogen in the mammary gland and endometrium. J Steroid Biochem Mol Biol. Apr-Jun 1999;69(1-6):51-84.

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