Ulipristal acetate

Cat. No.: HY-16508

CAS No.: 126784-99-4

Molecular Formula: $C_{30}H_{37}NO_4$

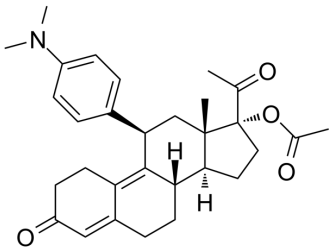
Molecular Weight: 475.62

Target: Progesterone Receptor; Autophagy

Pathway: Vitamin D Related/Nuclear Receptor; Autophagy

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 33.33 mg/mL (70.08 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.1025 mL	10.5126 mL	21.0252 mL
	5 mM	0.4205 mL	2.1025 mL	4.2050 mL
	10 mM	0.2103 mL	1.0513 mL	2.1025 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: ≥ 2.5 mg/mL (5.26 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.26 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ulipristal acetate (CDB-2914) is an orally active, selective progesterone receptor modulator (SPRM). Ulipristal acetate stimulates the autophagic response selectively in leiomyoma cells. Ulipristal acetate has the potential for benign gynecological conditions treatment, such as uterine myoma[1][2].

In Vitro

Ulipristal acetate (0.1-5 μM; 96 hours) stimulates autophagy in leiomyoma cells. Ulipristal-induced expression changes of the autophagic markers LC3 and p62/SQSTM1. Ulipristal up-regulates Atg7 protein in leiomyoma cells[2].
Ulipristal acetate blocks activin A modulation of fibronectin and vascular endothelial growth factor A (VEGF-A) mRNA expression in cultured myometrial and leiomyoma cells[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo	Ulipristal and CDB-4124 have significant antiprogestational activity in vivo[5]. Ulipristal acetate decreases incidences of fibroadenomas and adenocarcinomas in the mammary gland in all treated groups. Ulipristal acetate exposure [AUC(0-24h)] at the highest dose in rats is 67 times human therapeutic exposure at 10 mg/day. In mice, no tumor of any type increases at Ulipristal acetate exposures up to 313 times of therapeutic exposure. Ulipristal acetate-related findings in mice are limited to organ weight changes in the liver, pituitary, thyroid/parathyroid glands, and epididymis as well as minimal panlobular hepatocellular hypertrophy in male and female mice receiving 130 mg/kg/day[6]. Ulipristal acetate (1 mg/kg and 5 mg/kg) increases the frequency with which pathologists assessed the endometrium as being thickened compared to controls in a dose-dependent manner. There is a slight decrease in secretory differentiation with increasing dose of Ulipristal acetate, with small decreases in frequency of sub- and supra-nuclear vacuolation[7]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [5]	The study consisted of four groups, each comprising four female cynomolgus monkeys. The groups eitherreceive ASV (control), or Ulipristal acetate at dose levels of 1, 5, or 25 mg/kg for 39 weeks. Two additional animals are allocated to the control and high dose groups for an 8-week post-dose recovery period. At randomization, there is no statistically significant difference between treatment groups in mean body weight. The vehicle or Ulipristal acetate is administered to all groups by oral gavage for 273 consecutive days at a dose volume of 2 mL/kg. Following the dosing or recovery period, animals are euthanized by intravenous administration of sodium pentobarbital followed by exsanguination of the femoral vessels. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Int J Cancer. 2021 Dec 22.
- Hum Reprod. 2015 Apr;30(4):800-11.

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REFERENCES

[1]. Jadav SP, et al. Ulipristal acetate, a progesterone receptor modulator for emergency contraception. J Pharmacol Pharmacother. 2012 Apr;3(2):109-11.

[2]. Del Bello B, et al. Autophagy up-regulation by ulipristal acetate as a novel target mechanism in the treatment of uterine leiomyoma: an in vitro study. Fertil Steril. 2019 Dec;112(6):1150-1159.

[3]. Hild SA, et al. CDB-2914: anti-progestational/anti-glucocorticoid profile and post-coital anti-fertility activity in rats and rabbits. Hum Reprod. 2000 Apr;15(4):822-9.

[4]. Ciarmela P, et al. Ulipristal acetate modulates the expression and functions of activin a in leiomyoma cells. Reprod Sci. 2014 Sep;21(9):1120-5.

[5]. Attardi BJ, et al. In vitro antiprogestational/antiglucocorticoid activity and progestin and glucocorticoid receptor binding of the putative metabolites and synthetic derivatives of CDB-2914, CDB-4124, and mifepristone. J Steroid Biochem Mol Biol.

[6]. Pohl O, et al. Carcinogenicity and chronic rodent toxicity of the selective progesterone receptor modulator ulipristal acetate. Curr Drug Saf. 2013 Apr;8(2):77-97.

[7]. Pohl O, et al. A 39-week oral toxicity study of ulipristal acetate in cynomolgus monkeys. Regul Toxicol Pharmacol. 2013 Jun;66(1):6-12.

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

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