Norethindrone

Cat. No.: HY-B0554

CAS No.: 68-22-4

Molecular Formula: $C_{20}H_{26}O_2$

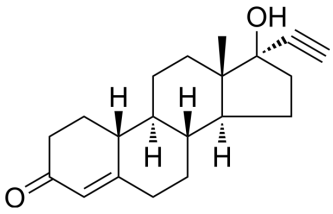
Molecular Weight: 298.42

Target: Progesterone Receptor; Bacterial

Pathway: Vitamin D Related/Nuclear Receptor; Anti-infection

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 : 25 mg/mL (83.77 mM; ultrasonic and warming and heat to 60°C)

H_2O : 2.7 mg/mL (9.05 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.3510 mL	16.7549 mL	33.5098 mL
5 mM	0.6702 mL	3.3510 mL	6.7020 mL
10 mM	0.3351 mL	1.6755 mL	3.3510 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 (8.38 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.38 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.38 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Norethindrone is a female progestin approved by FDA for the treatment of endometriosis, uterine bleeding caused by abnormal hormone levels, and secondary amenorrhea.

IC_{50} & Target

Progesterone Receptor[1]

In Vivo

Norethindrone acetate could be a cost-effective alternative with relatively mild side effects in the treatment of symptomatic

endometriosis. Subjects treated with norethindrone acetate obtain dysmenorrhea and noncyclic pelvic pain relief[1]. Norethindrone acetate alone is a well-tolerated, effective option to manage pain and bleeding for all stages of endometriosis. Post-Norethindrone acetate bleeding scores are improved regardless of prior hormonal regimen, and post-Norethindrone acetate pain scores improved in all patients except for those previously prescribed GnRH-agonist plus add-back[2]. Norethindrone acetate shows low acute toxicity in experimental animals and is consistent with the lack of toxicity seen in humans. Administration of norethindrone acetate alone to rodents at several multiples of the human dose results in no treatment related mortality, hematological changes, behavioral changes, or organ pathology[3]. Norethindrone acetate administration leads to significant and proportional reductions of the concentrations of triglycerides, cholesterol and phospholipids of plasma lipoproteins of density <1.006 of rats fed a high carbohydrate diet. Norethindrone acetate (0.1 mM) also significantly inhibits the incorporation of both palmitate and glycerol into triglycerides of isolated hepatocytes from fed rats[4].

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

PROTOCOL

Animal Administration [4]

Rats: Female Sprague-Dawley rats (200-210 g), 6 of which serve as controls, are individually caged in an animal room illuminated from 9:00 a.m. to 9:00 p.m. Each of the 7 rats receiving norethindrone acetate is fed 35 μg/day for 2 weeks. Water and the rat chow which is high in carbohydrate are available ad libitum[4].

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

CUSTOMER VALIDATION

- Cancer Cell Int. 2021 Jun 5;21(1):291.

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REFERENCES

[1]. Muneyyirci-Delale O, et al. Effect of norethindrone acetate in the treatment of symptomatic endometriosis. Int J Fertil Womens Med. 1998 Jan-Feb;43(1):24-7.

[2]. Kaser DJ, et al. Use of norethindrone acetate alone for postoperative suppression of endometriosis symptoms. J Pediatr Adolesc Gynecol. 2012 Apr;25(2):105-8.

[3]. Maier WE, et al. Pharmacology and toxicology of ethinyl estradiol and norethindrone acetate in experimental animals. Regul Toxicol Pharmacol. 2001 Aug;34(1):53-61.

[4]. Cheng DC, et al. Norethindrone acetate inhibition of triglyceride synthesis and release by rat hepatocytes. Atherosclerosis. 1983 Jan;46(1):41-8.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA