Abiraterone acetate

Cat. No.: HY-75054

CAS No.: 154229-18-2

Molecular Formula: $C_{26}H_{33}NO_2$

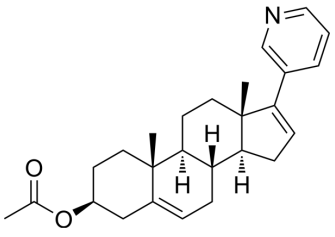
Molecular Weight: 391.55

Target: Cytochrome P450

Pathway: Metabolic Enzyme/Protease

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 : 10 mg/mL (25.54 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5540 mL	12.7698 mL	25.5395 mL
	5 mM	0.5108 mL	2.5540 mL	5.1079 mL
	10 mM	0.2554 mL	1.2770 mL	2.5540 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: ≥ 1 mg/mL (2.55 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1 mg/mL (2.55 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1 mg/mL (2.55 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Abiraterone acetate (CB7630) is an oral, potent, selective, and irreversible inhibitor of CYP17A1 with antiandrogen activity. Abiraterone acetate is a proagent form of Abiraterone (CB7598).

IC_{50} & Target

CYP17

In Vitro

Abiraterone (Abi) acetate is an ester prodrug of the anticancer agent Abiraterone, which shows IC_{50} values of 15 nM and 2.5 nM for the 17,20-lyase and 17α-hydroxylase (CYP17 is a bifunctional enzyme with both 17α-hydroxylase and 17,20-lyase

	activity). Abiraterone inhibits human 17,20-lyase and 17α-hydroxylase with IC_{50} of 27 and 30 nM respectively[1]. Significant inhibition of proliferation of the AR-positive prostate cancer cell lines LNCaP and VCaP with doses of Abiraterone ≥5 μM is confirmed[2]. Abiraterone inhibits recombinant human 3βHSD1 and 3βHSD2 activity with competitive K_i values of 2.1 and 8.8 μM. 10 μM Abiraterone is sufficient to completely block synthesis of 5α-dione and DHT in both cell lines.Treatment with Abiraterone significantly inhibited CRPC progression in the robustly growing subset, effectively putting a ceiling on tumor growth over 4 weeks of treatment (P<0.00001)[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Abiraterone (Abi) acetate prolongs survival in castration-resistant prostate cancer (CRPC). [^{3}H]-dehydroepiandrosterone (DHEA) depletion and Δ^4-androstenedione (AD) accumulation are inhibited by Abiraterone in LNCaP, with an IC_{50}<1 μM. The 0.5 mmol/kg/d Abiraterone treatment dose is previously shown to yield serum concentrations of about 0.5 to 1 μM. Xenograft tumor growth in the control group is widely variable, with some tumors growing slowly and only a subset of tumors exhibiting robust growth[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	LNCaP and VCaP cells are seeded in 96-well plates and grown in CSS-supplemented phenol red-free or FBS-supplemented media for 7 days. Cells are treated with Abiraterone (5 μM and 10 μM) at 24 and 96 hours after plating and cell viability is determined on day 7 by adding CellTiter Glo and measuring luminescence[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Mice[3] Male NOD/SCID mice 6 to 8 weeks of age are surgically orchiectomized and implanted with a 5 mg 90-day sustained release DHEA pellet to mimic CRPC with human adrenal physiology. Two days later, 7×10^6 LAPC4 cells are injected subcutaneously with Matrigel. Tumor dimensions are measured 2 to 3 times per week, and volume is calculated as length×width×height×0.52. Once tumors reach 300 mm^3, mice are randomly assigned to vehicle or Abiraterone treatment groups. Mice in the Abiraterone group are treated with 5 mL/kg intraperitoneal injections of 0.5 mmol/kg/d (0.1 mL 5% benzyl alcohol and 95% safflower oil solution) and control mice with vehicle only, once daily for 5 days per week over a duration of 4 weeks (n=8 mice per treatment). Statistical significance between Abiraterone and vehicle treatment groups is assessed by ANOVA based on a mixed-effect model. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Rep Med. 2024 Feb 20;5(2):101388.
- Cell Death Dis. 2022 Dec 12;13(12):1034.
- Cell Death Dis. 2021 Aug 12;12(8):787.
- Br J Cancer. 2017 Mar 28;116(7):937-943.
- JCI Insight. 2019 Sep 5;4(17):e122688.

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REFERENCES

[1]. Stein MN, et al. Androgen synthesis inhibitors in the treatment of castration-resistant prostate cancer. Asian J Androl. 2014 May-Jun;16(3):387-400.

[2]. Richards J, et al. Interactions of abiraterone, eplerenone, and prednisolone with wild-type and mutant androgen receptor: a rationale for increasing abiraterone

exposure or combining with MDV3100. Cancer Res. 2012 May 1;72(9):2176-82.

[3]. Li R, et al. Abiraterone inhibits 3β-hydroxysteroid dehydrogenase: a rationale for increasing drug exposure in castration-resistant prostate cancer. Clin Cancer Res. 2012 Jul 1;18(13):3571-9.

[4]. Lee GT, et al. Intracrine androgen biosynthesis in renal cell carcinoma. Br J Cancer. 2017 Mar 28;116(7):937-943.

[5]. A O'Donnell, et al. Hormonal impact of the 17α-hydroxylase/C17,20-lyase inhibitor abiraterone acetate (CB7630) in patients with prostate cancer. British Journal of Cancervolume 90, pages2317–2325 (2004)

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