Verdinexor

Cat. No.: HY-15970

CAS No.: 1392136-43-4

Molecular Formula: $C_{18}H_{12}F_6N_6O$

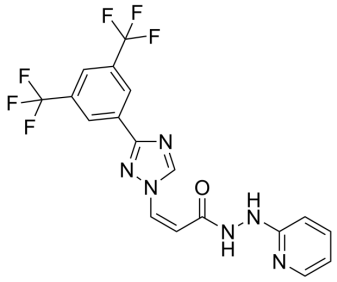
Molecular Weight: 442.32

Target: CRM1

Pathway: Membrane Transporter/Ion Channel

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 (226.08 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.2608 mL	11.3040 mL	22.6081 mL
	5 mM	0.4522 mL	2.2608 mL	4.5216 mL
	10 mM	0.2261 mL	1.1304 mL	2.2608 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 (5.65 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.65 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Verdinexor(KPT-335) is a novel, orally bioavailable selective inhibitor of nuclear export (SINE), inhibits nuclear export protein Exportin 1(XPO1/CRM1) against canine tumor cell lines; also reduce influenza virus replication in vitro and in vivo.IC50 value:Target: SINE; XPO1/CRM1in vitro: potently and selectively inhibit vRNP export and effectively inhibited the replication of various influenza virus A and B strains in vitro, including pandemic H1N1 virus, highly pathogenic H5N1 avian influenza virus, and the recently emerged H7N9 strain [1]. KPT-335 inhibited proliferation, blocked colony formation, and induced apoptosis of treated cells at biologically relevant concentrations of drug. Additionally, KPT-335 downregulated XPO1 protein while inducing a concomitant increase in XPO1 messenger RNA. Lastly, KPT-335 treatment of cell lines upregulated the expression of both protein and mRNA for the tumor suppressor proteins p53 and p21, and promoted their nuclear localization [3].in vivo: Prophylactic and therapeutic administration of verdinexor protected mice against disease pathology

following a challenge with influenza virus A/California/04/09 or A/Philippines/2/82-X79, as well as reduced lung viral loads and proinflammatory cytokine expression, while having minimal toxicity [1]. A dose expansion study was performed in 6 dogs with NHL given 1.5 mg/kg KPT-335 Monday/Wednesday/Friday; CB was observed in 4/6 dogs with a median TTP for responders of 83 days (range 35-354 days). Toxicities were primarily gastrointestinal consisting of anorexia, weight loss, vomiting and diarrhea and were manageable with supportive care, dose modulation and administration of low dose prednisone; hepatotoxicity, anorexia and weight loss were the dose limiting toxicities [2]. Inhibition of XPO1 with KPT-335 attenuated cyst growth in vivo in the PKD1 mutant mouse model Pkd1v/v [4].

CUSTOMER VALIDATION

- J Exp Clin Cancer Res. 2021 Aug 12;40(1):255.
- Methods Mol Biol. 2018;1711:351-398.

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REFERENCES

[1]. Munuce MJ, et al. Effects of ulipristal acetate on sperm DNA fragmentation during in vitro incubation. Eur J Contracept Reprod Health Care. 2013 Oct;18(5):355-63.

[2]. 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.

[3]. 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.

[4]. 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. 2004 Mar;88(3):277-88.

[5]. 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.

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