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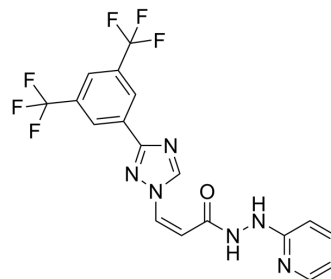
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Verdinexor

Cat. No.:	HY-15970
CAS No.:	1392136-43-4
Molecular Formula:	C ₁₈ H ₁₂ F ₆ N ₆ O
Molecular Weight:	442.32
Target:	CRM1
Pathway:	Membrane Transporter/Ion Channel
Storage:	<div>Powder</div> <div>-20°C 3 years</div> <div>4°C 2 years</div> <div>In solvent</div> <div>-80°C 6 months</div> <div>-20°C 1 month</div>



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (226.08 mM)
 * "≥" means soluble, but saturation unknown.

	Solvent Concentration	Mass	1 mg	5 mg	10 mg
Preparing Stock Solutions	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

- 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
- 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/CRM1 in 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 antiprogesterone/antiglucocorticoid activity and progesterone 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.

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

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