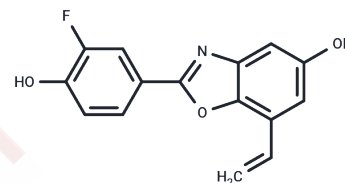


Prinaberel

Chemical Properties

CAS No. :	524684-52-4
Formula:	C ₁₅ H ₁₀ FNO ₃
Molecular Weight:	271.24
Storage:	Keep away from moisture Powder: -20°C for 3 years In solvent: -80°C for 1 year <i>Actual storage temperature shall be subject to the COA.</i>



Biological Description

Description	Prinaberel (ERB 041) is a selective and potent estrogen receptor β (ER β) agonist with anticancer activity that restores or increases ER β expression and reduces cancer cell proliferation in mouse squamous cell carcinoma and human carcinoma cells. Prinaberel exhibits anti-inflammatory activity, inhibits the NF κ B pro-inflammatory signaling pathway, and induces apoptosis of ovarian cancer cells. study endometriosis.
Targets(IC50)	Apoptosis, Estrogen Receptor/ERR, Estrogen/progestogen Receptor, Wnt/beta-catenin
In vitro	Treatment of human squamous cell carcinoma (SCC) cells with Prinaberel (0-60 μ M for 24 h) promoted cell differentiation, blocked the cell cycle, and reduced cell colony formation. In A431 cells, Prinaberel significantly decreased the expression of inflammation-regulating proteins such as p-NF κ Bp65, iNOS, and COX-2. Prinaberel inhibited the phosphorylation levels of PI3K and AKT, as well as enhanced the expression of E-cadherin, and reduced the migration ability of A431 cells. [2] Prinaberel (0.01-10 μ M) inhibited cell proliferation in a dose- and time-dependent manner. Prinaberel (10 μ M, 48 h) induced apoptosis in ovarian cancer cells (SKOV-3 cells). [3]
In vivo	In the SKH-1 nude mouse model, Prinaberel (2 mg/mouse, topical application, 30 minutes prior to UVB irradiation, weekly for 30 weeks) inhibited the development of squamous cell carcinoma. Prinaberel inhibited cell proliferation and angiogenesis, while promoting apoptosis, in UVB-induced skin tumors. Prinaberel also inhibited UVB. Prinaberel also inhibited the pro-inflammatory signaling pathway in UVB-induced skin tumors and reduced tumor invasiveness by modulating the PI3K-AKT pathway and the WNT signaling pathway. [2]

Solubility Information

Solubility	DMSO: 30 mg/mL (110.6 mM), Sonication is recommended. ($<$ 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 5 mg/mL (18.43 mM), Sonication is recommended. <i>Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and</i>

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In vivo Formulation	<i>used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.</i>
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Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	3.6868 mL	18.4339 mL	36.8677 mL
5 mM	0.7374 mL	3.6868 mL	7.3735 mL
10 mM	0.3687 mL	1.8434 mL	3.6868 mL
50 mM	0.0737 mL	0.3687 mL	0.7374 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Hinsche O, et al. Estrogen receptor β selective agonists reduce invasiveness of triple-negative breast cancer cells. *Int J Oncol.* 2015 Feb;46(2):878-84.

Chaudhary SC, et al. Erb-041, an estrogen receptor- β agonist, inhibits skin photocarcinogenesis in SKH-1 hairless mice by downregulating the WNT signaling pathway. *Cancer Prev Res (Phila).* 2014;7(2):186-198.

Chan KKL, et al. Estrogen receptor modulators genistein, daidzein and ERB-041 inhibit cell migration, invasion, proliferation and sphere formation via modulation of FAK and PI3K/AKT signaling in ovarian cancer. *Cancer Cell Int.* 2018;18:65. Published 2018.

Xiu-li W, et al. ERB-041, a selective ER beta agonist, inhibits iNOS production in LPS-activated peritoneal macrophages of endometriosis via suppression of NF-kappaB activation. *Mol Immunol.* 2009 Jul;46(11-12):2413-8.

Malamas MS, et al. Design and synthesis of aryl diphenolic azoles as potent and selective estrogen receptor-beta ligands. *J Med Chem.* 2004;47(21):5021-5040.

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