# Small Molecules

#### Mifepristone

Progesterone and glucocorticoid receptor

antagonist

Catalog #100-0564 500 mg

100-0565 1 g



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TOLL FREE PHONE 1 800 667 0322 • PHONE +1 604 877 0713 INFO@STEMCELL.COM • TECHSUPPORT@STEMCELL.COM FOR GLOBAL CONTACT DETAILS VISIT OUR WEBSITE

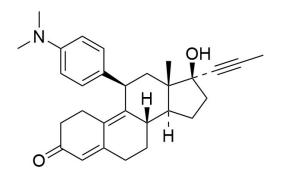
### **Product Description**

Mifepristone is a progesterone and glucocorticoid receptor antagonist ( $K_i = 0.64$  and 0.1 nM, respectively; Attardi et al.; Song et al.; von Geldern et al.). It has been reported that Mifepristone is used in the treatment of leiomyomata, endometriosis, breast cancer, and meningioma (Mahajan et al.).

Molecular Name:MifepristoneAlternative Names:RU-486CAS Number:84371-65-3Chemical Formula: $C_{29}H_{35}NO_2$ Molecular Weight:429.6 g/molPurity: $\geq 98\%$ 

Chemical Name: 11β-[4-(dimethylamino)phenyl]-17β-hydroxy-17-(1-propynyl)-estra-4,9-dien-3-one

Structure:



### **Properties**

Physical Appearance: A crystalline solid

Storage: Product stable at -20°C as supplied. Protect product from prolonged exposure to light. For long-term storage,

store with a desiccant. Stable as supplied for 12 months from date of receipt.

**Solubility:** • DMSO  $\leq$  45 mM

Absolute ethanol ≤ 45 mM

For example, to prepare a 10 mM stock solution in DMSO, resuspend 10 mg in 2.33 mL of DMSO.

Prepare stock solution fresh before use. Information regarding stability of small molecules in solution has rarely been reported, however, as a general guide we recommend storage in DMSO at -20°C. Aliquot into working volumes to avoid repeated freeze-thaw cycles. The effect of storage of stock solution on compound performance should be tested for each application.

Compound has low solubility in aqueous media. For use as a cell culture supplement, stock solution should be diluted into culture medium immediately before use. Avoid final DMSO concentration above 0.1% due to potential cell toxicity.

## **Small Molecules**

Mifepristone



## **Published Applications**

#### CANCER RESEARCH

- · Inhibits the expressions of cyclin-dependent kinase 1 (CDK1) and cyclin-dependent kinase 2 (CDK2), leading to cell cycle arrest and apoptosis in endometrial epithelial cells and stromal cells in adenomyosis (Che et al.).
- · Induces cell cycle arrest and apoptosis in antiestrogen-resistant breast cancer cells in vitro (Gaddy et al.).

### References

Attardi BJ et al. (2004) 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 88(3): 277–88.

Che X et al. (2020) A new trick for an old dog: The application of mifepristone in the treatment of adenomyosis. J Cell Mol Med 24(2): 1724–37.

Gaddy VT et al. (2004) Mifepristone induces growth arrest, caspase activation, and apoptosis of estrogen receptor-expressing, antiestrogen-resistant breast cancer cells. Clin Cancer Res 10(15): 5215–25.

Mahajan DK & London SN. (1997) Mifepristone (RU486): a review. Fertil Steril 68(6): 967-76.

Song L-N et al. (2004) Antiandrogen effects of mifepristone on coactivator and corepressor interactions with the androgen receptor. Mol Endocrinol 18(1): 70–85.

von Geldern TW et al. (2004) Liver-selective glucocorticoid antagonists: a novel treatment for type 2 diabetes. J Med Chem 47(17): 4213–30.

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## This product is hazardous. Please refer to the Safety Data Sheet (SDS).

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