



**Cyrusbioscience**

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## **Mifepristone, >98%**

**Product No.:** 101-84371-65-3

**Package:** 5g

**Synonym:** 11 $\beta$ -(4-Dimethyl-amino)-phenyl-17 $\beta$ -hydroxy-17-(1-propynyl)-estra-4,9-dien-3-one, RU-486

**Targets:** Bcl-2; Progesterone receptor(T47D cells)/0.2nM; Glucocorticoid receptor(A549 cells)/2.6nM

**Purity:** >98%

**Storage:** -20°C

### **Introduction:**

Mifepristone is an antagonist of glucocorticoid, progesterone, and androgen receptors. It is selective for these receptors over the mineralocorticoid receptor (MR), estrogen receptor  $\alpha$  (ER $\alpha$ ), and ER $\beta$ .

It also inhibits synthetic androgen R1881-stimulated reporter transcription in a concentration-dependent manner.<sup>2</sup> Mifepristone (10  $\mu$ M) inhibits growth of 4-OHT-resistant MCF-7 breast cancer cells *in vitro*. It also inhibits tumor growth in an SKOV3 ovarian cancer nude mouse xenograft model when administered at doses of 0.5 or 1 mg per day.

### ***In vivo:***

Mifepristone can impair the growth of SK-OV-3 tumors in immunosuppressed mice at 0.5 mg/day and 1 mg/day. Mifepristone inhibits the prostate weight significantly in the highest doses *in vivo*, and inhibits growth of the prostate gland produced by dihydrotestosterone (DHT) to a greater extent than the induction of atrophy and cell death in rats.

### ***In vitro:***

Mifepristone inhibit corticoid-induced transcription from a glucocorticoid response element (GRE)-linked luciferase reporter gene in the human lung carcinoma cell line A549. Moreover, Mifepristone also blocks progesterone induction of alkaline phosphatase activity in the human breast cancer cell line T47D. Mifepristone inhibits ovarian cancer cell growth of SK-OV-3 and OV2008 with IC<sub>50</sub> of 6.25  $\mu$ M and 6.91  $\mu$ M, respectively. A recent study shows that Mifepristone induces caspase-1 over expression both in differentiated and undifferentiated caspase-1-embryonic stem cells.

### **Application:**

- To induce damage against the proliferative and secretory phase of endometrial stromal cells
- To establish a preterm birth (PTB) mice model in order to study the difference in cervical ripening mechanism between term and PTBs
- To activate geneswitch gal4 in flies
- To study the effects of sex steroids on prostaglandin secretion