Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

2-Methoxyestradiol

Catalog No: tcsc0176

Available Sizes

Size: 10mg

Size: 50mg

Size: 100mg

Specifications

CAS No:
362-07-2

Formula:
$C_{19}H_{26}O_3$

Pathway:
Cell Cycle/DNA Damage;Cytoskeleton;Apoptosis

Target:
Microtubule/Tubulin;Microtubule/Tubulin;Apoptosis

Purity / Grade:
>98%

Solubility:
H2O :

Alternative Names:
2-ME2;NSC-659853

Observed Molecular Weight:
302.41

Product Description

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2-Methoxyestradiol is an **angiogenesis** inhibitor and **apoptosis** inducer with potent antineoplastic activity. 2-Methoxyestradiol also destablize **microtubules**.

IC50 & Target: IC50: 1.2 μM (tubulin/microtubule, in living interphase MCF7 cells)[1]

In Vitro: 2-Methoxyestradiol (5-100 μM) inhibits assembly of purified tubulin in a concentration-dependent manner, with maximal inhibition (60%) at 200 μM 2-Methoxyestradiol (2ME2). However, with microtubule-associated protein-containing microtubules, significantly higher 2-Methoxyestradiol concentrations are required to depolymerize microtubules, and polymer mass is reduced by only 13% at 500 μM 2-Methoxyestradiol. 4 μM 2-Methoxyestradiol reduces the mean growth rate by 17% and dynamicity by 27%. In living interphase MCF7 cells at the IC_{50} for mitotic arrest (1.2 μM), 2-Methoxyestradiol significantly suppresses the mean microtubule growth rate, duration and length, and the overall dynamicity, consistent with its effects in vitro, and without any observable depolymerization of microtubules. 2-Methoxyestradiol induces G_2-M arrest and apoptosis in many actively dividing cell types while sparing quiescent cells. 2-Methoxyestradiol binds to tubulin at or near the colchicine site, it inhibits microtubule assembly, and high concentrations have been shown to depolymerize microtubules in cells. 2-Methoxyestradiol induces G_2-M arrest and apoptosis in many actively dividing also blocks mitosis and inhibits endothelial cell migration[1]. 2-Methoxyestradiol (2-ME) decreases the HIF-1α and HIF-2α nuclear staining in cells cultured under hypoxia. The HIF-1α and HIF-2α mRNA levels are significantly lower when cells are exposed to 2-Methoxyestradiol under normoxia and hypoxia. 2-Methoxyestradiol is an anti-angiogenic, anti-proliferative and pro-apoptotic agent that suppresses HIF-1α protein levels and its transcriptional activity. A significant decrease in the growth rate is found in the 10 μM 2-Methoxyestradiol-treated A549 cells in comparison with the DMSO-treated cells (66.2±7.2 and 101.2±2.3%, respectively; p=0.04) at 96 h. 2-Methoxyestradiol at a concentration of 10 μM is used for the apoptosis and HIF-1α and HIF-2α expression assays, due to the significance found for this concentration when cells are incubated under normoxic conditions at 72 h. A significant increase in apoptosis is observed in cells treated with 10 μM 2-Methoxyestradiol in a normoxic condition in comparison with cells under lower O_2 concentration (5.8±0.2%; p=0.003)[2].

In Vivo: To investigate the effect of 2ME2 on uveitis development, C57BL/6 mice are randomly assigned into two groups and immunized with IRBP peptide. 2ME2 group starts 2-Methoxyestradiol (15 mg/kg) intraperitoneally from day 0 to day 13 while control group is given with vehicle. The disease score of 2-Methoxyestradiol (2ME2) group is 0.30±0.30, significantly lower than that of control group 2.09±0.28 (p[3]. Treatment with 2-Methoxyestradiol (60-600 mg/kg/d) results in a dose-dependent inhibition of tumor growth. The percentage of cells with strong pimonidazole-positive staining (+++) is significantly decreased in the 2-Methoxyestradiol-treated group (36.0% for 60 mg/kg/d and 0% for 200 and 600 mg/kg/d) compare with the vehicle-treated group (86.5%). This may be attributed to the dramatic inhibition of tumor growth in a dose-dependent manner following 2-Methoxyestradiol treatment[4].