

Estradiol

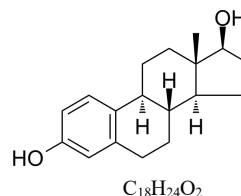
Catalog Number: C502820



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DESCRIPTION

Background	Estradiol is a steroid sex hormone vital to the maintenance of fertility and secondary sexual characteristics in females. Estradiol upregulates IL-6 expression through the estrogen receptor β (ER β) pathway ^{[1][2][3]} .		
Alias	β -Estradiol; E2; 17 β -Estradiol; 17 β -Oestradiol 雌二醇; 强力求偶素		
M. W t	272.38		
Formula	C ₁₈ H ₂₄ O ₂		
CAS No	50-28-2		
Storage	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month
Solubility	DMSO	≥ 100 mg/mL(283.71 mM)	
	Ethanol	14 mg/mL(51.40 mM)	
	H ₂ O	< 0.1 mg/mL(insoluble)	



BIOLOGICAL ACTIVITY

In Vitro

Estradiol causes new dendritic spines and synapses in hippocampal CA1 pyramidal cells. Estradiol increases NMDA receptor binding by 46% in parallel with dendritic spine and synapse density. Estradiol also elevates sensitivity of CA1 pyramidal cells to NMDA receptor-mediated synaptic input and such an effect is correlated with the estradiol-induced increase in dendritic spine density in the apical dendritic tree of these cells^[1]. Estradiol reduces Ba²⁺ entry reversibly via Ca²⁺ channels in acutely dissociated and cultured neostriatal neurons. Estradiol also reduces Ba²⁺ currents but is significantly less effective than Estradiol in rat neostriatal neurons^[2].

In Vivo

Estradiol functions in hippocampal synapse density during the estrous cycle in the adult rat^[4]. Estradiol reverses the ovariectomy-induced decrease in spine density. Estradiol in combination with progesterone enhances spine density for 2 to 6 h but decreases following estradiol alone^[5]. Estradiol (0.2 mg/kg; i.p.; C57BL/6 female mice) plus 10 or 20 mg/kg progesterone, enhances memory consolidation in young ovariectomized mice^[6].

REFERENCES

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