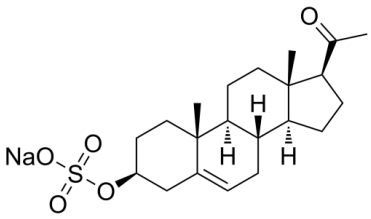
Data Sheet

Product Name:	Pregnenolone monosulfate (sodium)
Cat. No.:	CS-0033053
CAS No.:	1852-38-6
Molecular Formula:	$C_{21}H_{31}NaO_5S$
Molecular Weight:	418.52
Target:	Autophagy; Cannabinoid Receptor; Endogenous Metabolite; TRP Channel
Pathway:	Autophagy; GPCR/G Protein; Membrane Transporter/Ion Channel; Metabolic Enzyme/Protease; Neuronal Signaling
Solubility:	DMSO : 100 mg/mL (ultrasonic);H_2O : 5 mg/mL (ultrasonic;warming;heat to 60°C)



BIOLOGICAL ACTIVITY:

Pregnenolone monosulfate sodium (3β-Hydroxy-5-pregnen-20-one monosulfate sodium) is a powerful neurosteroid, the main precursor of various steroid hormones including steroid ketones. Pregnenolone monosulfate sodium acts as a signaling-specific inhibitor of **cannabinoid CB1 receptor**, inhibits the effects of tetrahydrocannabinol (THC) that are mediated by the **CB1 receptors**. Pregnenolone monosulfate sodium can protect the brain from cannabis intoxication[1][2]. Pregnenolone monosulfate sodium is also a **TRPM3 channel** activator, and also can weakly activate **TRPM1 channels**[3]. IC50 & Target: Cannabinoid CB1 receptor[1] ***In Vitro:*** CB1 receptor stimulation increases brain Pregnenolone levels, which in turn exerts a negative feedback on the activity of the CB1 receptor antagonizing most of the known behavioral and somatic effects of THC. Pregnenolone likely acts as a signaling-specific negative allosteric modulator binding to a site distinct from that occupied by orthosteric ligands. Pregnenolone does not modify agonist binding but only agonist efficacy[1].

The effect of THC is significantly attenuated when slices are pre-treated with Pregnenolone 100 nM (15.1±1.8 % of inhibition). These effects are likely due to a pre-synaptic action of Pregnenolone. Thus, Pregnenolone blocks the increase in paired-pulse ratio (PPR) induced by THC but does not modify either the amplitude or the decay time of miniature EPSC (mEPSC)[1]. ***In Vivo:*** Pregnenolone administration (2-6 mg/kg) blocks THC-induced food-intake in Wistar rats and in C57BL/6N mice, and blunts the memory impairment induced by THC in mice, but it does not modify these behaviors ***per se***. Injections of Pregnenolone (2 and 4mg/kg) before each self-administration session reduce the intake of WIN 55,212-2 and reduce the break-point in a progressive ratio schedule[1].

PROTOCOL (Extracted from published papers and Only for reference)

Animal Administration: Pregnenolone is dissolved in Tween 80 (1 drop/3 mL) and DMSO (2.5%), diluted in saline solution[1].[1]Mice and Rats[1]

Adult male Wistar rats (weighing 320-340g), **Sprague Dawley male rats** (weighing 330-350g), **C57BL/6N mice** (2-3 months) and **CD1 mice** (weighing 25-30 g at the beginning of the experiments) are used. Pregnenolone is **injected subcutaneously (sc)**. The injection volumes are **1 mL/kg** of body weight for **rats** and **10 mL/kg** for **mice**[1].

References:

[1]. Vallée M, et al. Pregnenolone can protect the brain from cannabis intoxication. Science. 2014 Jan 3;343(6166):94-8.

[2]. Ducharme N, et al. Brain distribution and behavioral effects of progesterone and pregnenolone after intranasal or intravenous administration. Eur J

Pharmacol. 2010 Sep 1;641(2-3):128-34.

[3]. Alan Shiels. TRPM3_miR-204: a complex locus for eye development and disease. Hum Genomics. 2020 Feb 18;14(1):7.

CAIndexNames:

Pregn-5-en-20-one, 3-(sulfooxy)-, sodium salt (1:1), (3β)-

SMILES:

CC([C@H]1CC[C@@]2([H])[C@]3([H])CC=C4C[C@@H](OS(=O)(O[Na])=O)CC[C@]4(C)[C@@]3([H])CC[C@]12C)=O

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