

Product Data Sheet

Product Name: Pregnenolone monosulfate

Cat. No.: GC36963

Chemical Properties

Cas. No. 1247-64-9

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

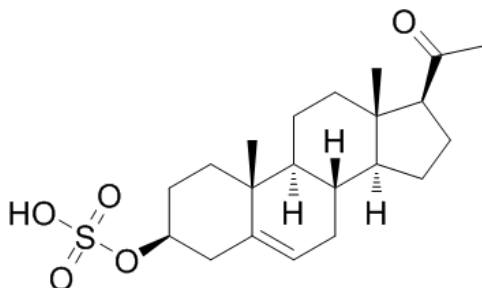
Formula $C_{21}H_{32}O_5S$ M.Wt 396.54

Solubility DMSO: 62.5 mg/mL (157.61 mM) Storage Store at -20°C, stored under nitrogen

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines GH3 cells (rat pituitary clonal cell line)

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GH3 cells were cultured as monolayers on glass coverslips in Dulbecco's modified Eagle medium supplemented with 10% fetal bovine serum at 37°C, 5% CO₂. Cells were loaded with Fura-2/AM (5μM) for 30 min and perfused with physiological buffer. Pregnenolone monosulfate (5–300μM) was applied via perfusion for 1 min.

Reaction Conditions
5–300μM; 1 min

Pregnenolone monosulfate induced a rapid, dose-dependent increase in intracellular Ca²⁺ ([Ca²⁺]_i) in GH3 cells, with an EC₅₀ of ~32μM. The [Ca²⁺]_i surge was abolished in Ca²⁺-free medium or in the presence of inorganic Ca²⁺ channel blockers (La³⁺, Co²⁺). Organic L-type voltage-gated Ca²⁺ channel (VGCC) blockers (nicardipine, D600) inhibited the response, indicating that the Ca²⁺ influx is primarily mediated through L-type VGCCs. Co-application with the VGCC agonist Bay K 8644 potentiated the effect, suggesting synergistic modulation of Ca²⁺ channels.

Animal experiment [2]:

Animal models C57BL/6-N mice (9–10 weeks old)

Mice were subcutaneously (s.c.) administered Pregnenolone monosulfate (6mg/kg) either 10 minutes before or 30 minutes after intraperitoneal (i.p.) injection of Δ9-tetrahydrocannabinol (THC; 0.3–10mg/kg). Behavioral tests (e.g., spontaneous alternation, social interaction, prepulse inhibition, locomotion, and "reality testing") were conducted at specified time points post-THC administration (45–120 minutes).

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Dosage form 6mg/kg; s.c.; Acute single injection.

Applications Pregnenolone monosulfate blocked a wide spectrum of THC-induced psychotic-like behaviors, including cognitive deficits, reduced prepulse inhibition, decreased social interaction, hyperlocomotion, and impaired "reality testing" (a measure of mental sensory representations).

References:

- [1] Büküşoğlu C, Sarlak F. Pregnenolone sulfate increases intracellular Ca^{2+} levels in a pituitary cell line. *Eur J Pharmacol.* 1996 Feb 29;298(1):79-85.
- [2] Busquets-Garcia A, Soria-Gómez E, Redon B, et al. Pregnenolone blocks cannabinoid-induced acute psychotic-like states in mice. *Mol Psychiatry.* 2017 Nov;22(11):1594-1603.

Background

Pregnenolone monosulfate is a key precursor to various steroid hormones, including steroid hormones, and possesses multiple biological functions^[1-2]. Pregnenolone monosulfate can be utilized in neuroscience research and related nervous system disorders^[3-4].

In vitro, Pregnenolone monosulfate (5-300 μM) was applied to GH3 pituitary cells for 1 minute, Pregnenolone monosulfate induced a rapid and transient increase in intracellular calcium ion ($[\text{Ca}^{2+}]_i$) levels. Pregnenolone monosulfate activated L-type voltage-gated calcium channels, mediating extracellular calcium influx^[5]. In neonatal rat cerebellar Purkinje cells (P4–10), Pregnenolone monosulfate (25 μM) enhanced glutamate release by promoting presynaptic calcium influx through transient receptor potential (TRP) channels, Pregnenolone monosulfate significantly increased AMPA receptor-mediated miniature excitatory postsynaptic currents (AMPA-mEPSCs)^[6].

In vivo, in dopamine transporter knockout (DAT-KO) mice (8-10 weeks old), Pregnenolone monosulfate (40 or 80mg/kg; administered either as a single injection or daily for 10 consecutive days via intraperitoneal injection). Pregnenolone monosulfate significantly suppressed hyperlocomotion and stereotypic behaviors, rescued prepulse inhibition deficits, and improved

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cognitive impairments in novel object recognition and social food preference tests. The therapeutic effects of Pregnenolone monosulfate were mediated through the AKT/GSK3 β pathway and increased expression of the hippocampal NMDA receptor NR1 subunit^[7]. In wild-type mice (9-10 weeks old), a single subcutaneous injection of Pregnenolone monosulfate (6mg/kg), Pregnenolone monosulfate antagonized Δ 9-tetrahydrocannabinol (THC)-induced psychotic-like behaviors, significantly blocked THC-induced cognitive impairments, sensory gating deficits, and reduced social interaction^[8].

References:

- [1] Vallée M, Vitiello S, Bellocchio L, et al. Pregnenolone can protect the brain from cannabis intoxication. *Science*. 2014 Jan 3;343(6166):94-8.
- [2] Ducharme N, Banks WA, Morley JE, 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] Shiels A. TRPM3_miR-204: a complex locus for eye development and disease. *Hum Genomics*. 2020 Feb 18;14(1):7.
- [4] Weaver CE Jr, Wu FS, Gibbs TT, et al. Pregnenolone sulfate exacerbates NMDA-induced death of hippocampal neurons. *Brain Res*. 1998 Aug 24;803(1-2):129-36.
- [5] Büküşoğlu C, Sarlak F. Pregnenolone sulfate increases intracellular Ca²⁺ levels in a pituitary cell line. *Eur J Pharmacol*. 1996 Feb 29;298(1):79-85.
- [6] Zamudio-Bulcock PA, Valenzuela CF. Pregnenolone sulfate increases glutamate release at neonatal climbing fiber-to-Purkinje cell synapses. *Neuroscience*. 2011 Feb 23;175:24-36.
- [7] Wong P, Sze Y, Chang CC, et al. Pregnenolone sulfate normalizes schizophrenia-like behaviors in dopamine transporter knockout mice through the AKT/GSK3 β pathway. *Transl Psychiatry*. 2015 Mar 17;5(3):e528.
- [8] Busquets-Garcia A, Soria-Gómez E, Redon B, et al. Pregnenolone blocks cannabinoid-induced acute psychotic-like states in mice. *Mol Psychiatry*. 2017 Nov;22(11):1594-1603.

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