Fluoxetine

Cat. No.: HY-B0102

CAS No.: 54910-89-3

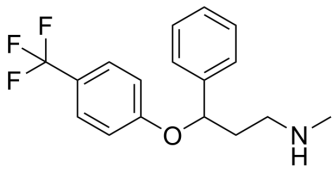
Molecular Formula: $C_{17}H_{18}F_3NO$

Molecular Weight: 309.33

Target: Serotonin Transporter; Autophagy

Pathway: Neuronal Signaling; Autophagy

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (323.28 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.2328 mL	16.1640 mL	32.3279 mL
	5 mM	0.6466 mL	3.2328 mL	6.4656 mL
	10 mM	0.3233 mL	1.6164 mL	3.2328 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 0.5% CMC-Na/saline water
   Solubility: 10 mg/mL (32.33 mM); Suspended solution; Need ultrasonic and warming and heat to 60°C
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (6.72 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (6.72 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (6.72 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Fluoxetine (LY-110140 free base) is a selective serotonin reuptake inhibitor (SSRI) class used for antidepressant research.

In Vitro

Fluoxetine blocks the downregulation of cell proliferation resulting from inescapable shock (IS) of hippocampal cell[1] .?Fluoxetine increases the number of newborn cells in the dentate gyrus of the hippocampus of adult rat. Fluoxetine also increases the number of proliferating cells in the prelimbic cortex[2].?Fluoxetine accelerates the maturation of immature neurons. Fluoxetine enhances neurogenesis-dependent long-term potentiation (LTP) in the dentate gyrus[3].?Fluoxetine, but not citalopram, fluvoxamine, paroxetine and sertraline, increases norepinephrine and dopamine extracellular levels in

	prefrontal cortex. Fluoxetine produces robust and sustained increases in extracellular concentrations of norepinephrine and dopamine after acute systemic administration[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Fluoxetine treatment also reverses the deficit in escape latency observed in animals exposed to inescapable shock in adult male Sprague-Dawley rats[1]. Fluoxetine (5 mg/kg) alone increases cell proliferation in the dentate gyrus. Coadministration (fluoxetine 5 mg/kg + olanzapine) also significantly increases the number of BrdU-positive cells compared with the control group[2]. Fluoxetine combined with Olanzapine produces robust, sustained increases of extracellular levels of dopamine ([DA](ex)) and norepinephrine ([NE](ex)) up to 361% and 272% of the baseline, respectively, which are significantly greater than either drug alone[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [2]	Male Sprague-Dawley rats weighing 250-300 g are housed under a 12-hour light/12-hour dark cycle (lights on at 7:00 am, lights off at 7:00 pm) and at constant temperature (25°C) and humidity and allowed free access to food and water. For chronic drug treatments, rats are administered fluoxetine (5 mg/kg/day) or saline by intraperitoneal (IP) injection once daily and olanzapine or vehicle in the drinking water for 21 days (vehicle-treated control, fluoxetine, and olanzapine alone) plus the combination of fluoxetine and olanzapine. For combination treatment, olanzapine is chosen because fluoxetine is known to interfere with the metabolism of olanzapine and raise the blood levels by up to 4-6 times. Olanzapine is dissolved in hydrochloric acid (HCl), then adjusted back to pH 6 with 1 N sodium hydroxide to make the stock solution of 3 mg/mL concentration. The same amount of vehicle solution is added to the water for the control animals. Fluid intake is measured three times per week, and drinking bottles are replenwashed with fresh drug solution. There are no differences in fluid intake among the treatment groups. For subchronic treatment, drugs are administered exactly the same way but for a total period of 7 days. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Med. 2019 Sep;25(9):1428-1441.
- Brain Behav Immun. 2021 Mar 15;S0889-1591(21)00114-8.
- J Neuroinflammation. 2023 May 10;20(1):112.
- J Neuroinflammation. 2017 Oct 30;14(1):210.
- Chemosphere. 2019 Jun;225:378-387.

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REFERENCES

[1]. Malberg JE, et al. Cell proliferation in adult hippocampus is decreased by inescapable stress: reversal by fluoxetine treatment. Neuropsychopharmacology. 2003 Sep;28(9):1562-71

[2]. Kodama M, et al. Chronic olanzapine or fluoxetine administration increases cell proliferation in hippocampus and prefrontal cortex of adult rat. Biol Psychiatry. 2004 Oct 15;56(8):570-80.

[3]. Wang JW, et al. Chronic fluoxetine stimulates maturation and synaptic plasticity of adult-born hippocampal granule cells. J Neurosci. 2008 Feb 6;28(6):1374-84.

[4]. Bymaster FP, et al. Fluoxetine, but not other selective serotonin uptake inhibitors, increases norepinephrine and dopamine extracellular levels in prefrontal cortex. Psychopharmacology (Berl). 2002 Apr;160(4):353-61

[5]. Zhang W, et al. Synergistic effects of olanzapine and other antipsychotic agents in combination with fluoxetine on norepinephrine and dopamine release in rat prefrontal cortex. Neuropsychopharmacology. 2000 Sep;23(3):250-62.

[6]. Avitsur R1. Increased symptoms of illness following prenatal stress: Can it be prevented by fluoxetine? Behav Brain Res. 2017 Jan 15;317:62-70.

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