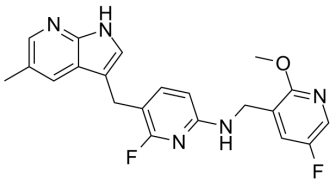
PLX5622

Cat. No.:	HY-114153
CAS No.:	1303420-67-8
Molecular Formula:	$C_{21}H_{19}F_2N_5O$
Molecular Weight:	395.41
Target:	c-Fms
Pathway:	Protein Tyrosine Kinase/RTK
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (126.45 mM; Need ultrasonic)
Ethanol : 3.33 mg/mL (8.42 mM; ultrasonic and warming and heat to 60°C)
H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.5290 mL	12.6451 mL	25.2902 mL
5 mM	0.5058 mL	2.5290 mL	5.0580 mL
10 mM	0.2529 mL	1.2645 mL	2.5290 mL

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

In Vivo

1. Add each solvent one by one: 5% DMSO >> 95% (0.5% HPMC/1% Tween-80 in water)
   Solubility: 6.5 mg/mL (16.44 mM); Suspended solution; Need ultrasonic and warming
2. Add each solvent one by one: 5% DMSO >> 95% (20% Ethoxylated hydrogenated castor oil in saline)
   Solubility: 5 mg/mL (12.65 mM); Clear solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 3.12 mg/mL (7.89 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.32 mM); Clear solution
5. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.32 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

PLX5622 is a highly selective brain penetrant and orally active CSF1R inhibitor (IC_{50}=0.016 μM; K_i=5.9 nM). PLX5622 allows for

	extended and specific microglial cells elimination, preceding and during pathology development. PLX5622 demonstrates desirable PK properties in varies animals. PLX5622 is predominantly administered via ad libitum diets with a dose of 1200 ppm[1][2].
IC_{50} & Target	IC50: 0.016 μM (CSF1R); Ki: 5.9 nM (CSF1R)[1][2]
In Vitro	PLX5622 (1-20 μM; 3 days) effectively depletes microglia without affecting oligodendrocytes or astrocytes in cerebellar slices. PLX5622 (4 μM; 3 days) causes a 30-40% reduction in NG2+ or PDGFRα+ cells, and this increased to 90-95% at 20 μM. No reduction of NG2+ or PDGFRα+ OPCs is observed in slices exposed to 1 μM or 2 μM PLX5622 despite robust (~95%) depletion of the microglial cells[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Pharmacodynamics of PLX5622 in preclinical studies PLX5622 (1200 ppm; chow; for 3 weeks or 3 days; adult C57/Bl6 wild type mice) leads to around 80% of microglia lost after 3 days of treatment and a 99% microglia loss after 3 weeks of treatment. PLX5622 (adult C57/Bl6 wild type mice aged 3 months; diet for 3 weeks) decreases microglia in cortex, striatum, cerebellum and hippocampus[4]. PLX5622 (50 mg/kg; intraperitoneal injection; once (neonatal rat) or twice (adult rat) a day; for a total of 14 days) depletes microglia by 80-90% within 3 days of treatment, which increases to > 90% by 7 days. After 14 days of PLX5622 treatment, microglia is depleted by > 96% in both neonates and adults while preserving baseline astrocyte quantity. (A single daily injection of 0.65% PLX5622 suspended in 5% dimethyl sulfoxide and 20% Kolliphor RH40 in 0.01 M PBS is sufficient for neonatal microglia depletion, adult depletion requires injections twice daily)[5]. PLX5622 (formulated in AIN-76A standard chow at 1200 mg/kg; for 28 days) leads to reduction in microglia throughout the CNS in 14-month-old 5xfAD mice[6]. Preparation of gavage dosing suspensions for PLX5622[1] PLX5622 is dissolved in DMSO at a concentration that is 20x the final dosing solution. The compound stock is protected from light. A fresh stock is made each week. The components of the diluent generally are prepared a day or more in advance because they take time to dissolve completely: a) 2% hydroxypropyl methyl cellulose (HPMC): 2.0 g powder was brought to 100 mL deionized water; b) 25% Polysorbate 80 (PS80): 25 g was brought to 100 mL deionized water. To make 100 mL diluent, add 25 mL of 2% HPMC stock (0.5% final) and 4 mL of 25% PS80 stock (1% final) to 71 mL deionized water to have final 100 mL. Final composition after mixing with compound: 0.5% HPMC, 1% PS80, 5% DMSO. On each dosing day, the compound stock is diluted 20-fold as follows: 19 volumes of diluent are measured into the tube, and 1 volume of the 20x compound/DMSO stock is added. The cap is closed and the content of the tube is mixed by inversion and placed in a sonicating water bath to make a uniform suspension. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nature. 2025 Jul;643(8071):509-518.
- Nature. 2021 Feb;590(7847):612-617.
- Cell. 2025 Apr 17;188(8):2159-2174.e15.
- Cell. 2023 Sep 28;186(20):4454-4471.e19.
- Immunity. 2024 Oct 9:S1074-7613(24)00458-8.

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REFERENCES

[1]. Spangenberg E, et al. Sustained microglial depletion with CSF1R inhibitor impairs parenchymal plaque development in an Alzheimer's disease model. Nat Commun. 2019 Aug 21;10(1):3758.

[2]. Lee S, et al. Targeting macrophage and microglia activation with colony stimulating factor 1 receptor inhibitor is an effective strategy to treat injury-triggered neuropathic pain. Mol Pain. 2018 Jan-Dec;14:1744806918764979.

[3]. Badimon A, et al. Negative feedback control of neuronal activity by microglia. Nature. 2020;586(7829):417-423.

[4]. Andrew J. Riquier, et al. Astrocytic response to neural injury is larger during development than in adulthood and is not predicated upon the presence of microglia, Brain, Behavior, & Immunity-Health, Volume 1, 2020, 100010, ISSN 2666-3546.

[5]. Liu Y, et al. Concentration-dependent effects of CSF1R inhibitors on oligodendrocyte progenitor cells ex vivo and in vivo. Exp Neurol. 2019;318:32-41.

[6]. Spangenberg EE, et al. Eliminating microglia in Alzheimer's mice prevents neuronal loss without modulating amyloid-β pathology. Brain. 2016;139(Pt 4):1265-1281.

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