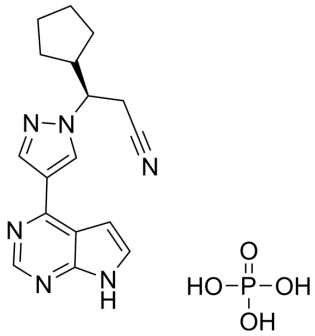
Ruxolitinib phosphate

Cat. No.:	HY-50858
CAS No.:	1092939-17-7
Molecular Formula:	$C_{17}H_{21}N_6O_4P$
Molecular Weight:	404.36
Target:	JAK; Autophagy; Mitophagy
Pathway:	Epigenetics; JAK/STAT Signaling; Protein Tyrosine Kinase/RTK; Stem Cell/Wnt; Autophagy
Storage:	4°C, sealed storage, away from moisture * In solvent : -80°C, 2 years; -20°C, 1 year (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 31 mg/mL (76.66 mM)

H_2O : 10 mg/mL (24.73 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.4730 mL	12.3652 mL	24.7304 mL
5 mM	0.4946 mL	2.4730 mL	4.9461 mL
10 mM	0.2473 mL	1.2365 mL	2.4730 mL

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

In Vivo

1. Add each solvent one by one: 0.5% MC >> 0.5% Tween-80
   Solubility: 10 mg/mL (24.73 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 5% DMSO >> 40% PEG300 >> 5% Tween-80 >> 50% saline
   Solubility: ≥ 2.75 mg/mL (6.80 mM); Clear solution
3. Add each solvent one by one: 5% DMSO >> 95% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.75 mg/mL (6.80 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (5.14 mM); Clear solution
5. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (5.14 mM); Clear solution
6. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.14 mM); Clear solution

BIOLOGICAL ACTIVITY

Description	Ruxolitinib phosphate (INCB018424 phosphate) is a potent JAK1/2 inhibitor with IC_{50}s of 3.3 nM/2.8 nM, respectively, showing more than 130-fold selectivity over JAK3.			
IC_{50} & Target	JAK2 2.8 nM (IC_{50})	JAK1 3.3 nM (IC_{50})	Tyk2 19 nM (IC_{50})	JAK3 428 nM (IC_{50})
In Vitro	Ruxolitinib (INCB018424) potently and selectively inhibits JAK2V617F-mediated signaling and proliferation. Ruxolitinib inhibits the growth of HEL cells with EC_{50} of 186 nM. Ruxolitinib markedly increases apoptosis in Ba/F3-EpoR-JAK2V617F cell system, and inhibits hematopoietic progenitor cell proliferation in primary MPN patient samples[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.			
In Vivo	Ruxolitinib (180 mg/kg, p.o.) reduces the tumor burden of mice inoculated with JAK2V617F-expressing cells without causing anemia or lymphopenia[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.			

PROTOCOL

Cell Assay [1]	Cells are seeded at 2000/well of white bottom 96-well plates, treated with compounds from DMSO stocks (0.2% final DMSO concentration), and incubated for 48 hours at 37°C with 5% CO_2. Viability is measured by cellular ATP determination using the Cell-Titer Glo luciferase reagent or viable cell counting. Values are transformed to percent inhibition relative to vehicle control, and IC_{50} curves are fitted according to nonlinear regression analysis of the data using PRISM GraphPad. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Mice are fed standard rodent chow and provided with water ad libitum. Ba/F3-JAK2V617F cells (10^5 per mouse) are inoculated intravenously into 6- to 8-week-old female BALB/c mice. Survival is monitored daily, and moribund mice are humanely killed and considered deceased at time of death. Treatment with vehicle (5% dimethyl acetamide, 0.5% methocellulose) or Ruxolitinib (INCB018424) begins within 24 hours of cell inoculation, twice daily by oral gavage. Hematologic parameters are measured using a Bayer Advia120 analyzed, and statistical significance is determined using Dunnett testing. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Med. 2018 Aug;24(8):1143-1150.
- Nature. 2023 Mar;615(7950):158-167.
- Nature. 2022 Sep;609(7928):785-792.
- Cell. 2021 Apr 15;184(8):2167-2182.e22.
- J Hematol Oncol. 2021 Jun 24;14(1):97.

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REFERENCES

[1]. Quintas-Cardama A, et al. Preclinical characterization of the selective JAK1/2 inhibitor INCB018424: therapeutic implications for the treatment of myeloproliferative neoplasms. Blood, 2010, 115(15), 3109-3117.

[2]. Fleischman AG, et al. The CSF3R T618I mutation causes a lethal neutrophilic neoplasia in mice that is responsive to therapeutic JAK inhibition. Blood. 2013 Nov 21;122(22):3628-31.

[3]. de Bock CE, et al. HOXA9 Cooperates with Activated JAK/STAT Signaling to Drive Leukemia Development. Cancer Discov. 2018 May;8(5):616-631.

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