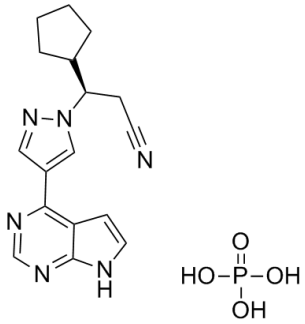
Data Sheet

Product Name:	Ruxolitinib (phosphate)
Cat. No.:	CS-0326
CAS No.:	1092939-17-7
Molecular Formula:	$C_{17}H_{21}N_6O_4P$
Molecular Weight:	404.36
Target:	Autophagy; JAK
Pathway:	Autophagy; Epigenetics; JAK/STAT Signaling; Stem Cell/Wnt
Solubility:	DMSO: ≥ 31 mg/mL; H_2O: 5.4 mg/mL (Need ultrasonic or warming)



BIOLOGICAL ACTIVITY:

Ruxolitinib (phosphate) is the first potent **JAK1/2** inhibitor with **IC_{50}** values of 3.3 nM/2.8 nM, more than 130-fold selectivity for JAK1/2 versus JAK3.

IC50 & Target: IC50: 3.3 nM (JAK1), 2.8 nM (JAK2)

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].

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].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: Ruxolitinib phosphate is dissolved in 0.2% DMSO.[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. **Animal Administration:** Ruxolitinib phosphate is dissolved in vehicle (5% dimethyl acetamide, 0.5% methocellulose).[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.

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.

Caution: Product has not been fully validated for medical applications. For research use only.

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