Product Datasheet

Deucravacitinib

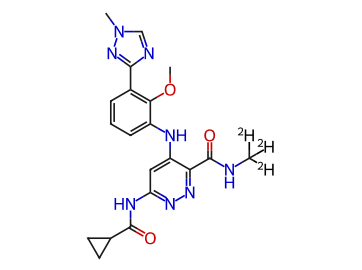
Catalog No. **BP-01903** · CAS **1609392-27-9** · Form **free base** · Physical state **solid** · Color **Off-white to light yellow** · Version **1.0** · Revision **2026-09-04**

FOR RESEARCH USE ONLY. NOT FOR HUMAN OR VETERINARY USE.

Target	Research area
JAK; Interleukin Related	**Inflammation/Immunology**

Molecular formula	$C_{20}H_{22}N_8O_3$
Molecular weight	**425.4590**

STRUCTURE



DESCRIPTION

BMS-986165 is a highly selective, orally bioavailable allosteric TYK2 inhibitor for the treatment of autoimmune diseases. BMS-986165 inhibits IL-12/23 and type I IFN pathways.

BIOLOGICAL ACTIVITY

In Vitro

Deucravacitinib potently inhibits TYK2-dependent IFNα-induced STAT5 phosphorylation in human CD3+ T cells, with an IC50 of 2 nM.

Deucravacitinib potently inhibits TYK2-dependent IL-23-induced STAT3 phosphorylation in human CD161+ CD3+ T cells, with an IC50 of 9 nM.

Deucravacitinib potently inhibits TYK2-dependent IFNα-induced STAT5 phosphorylation in human whole blood with an IC50 of 13 nM, while exhibiting high functional selectivity for signaling pathways dependent on JAK2, JAK1 and JAK3.

Biological Activity

Description

Deucravacitinib (BMS-986165) is an orally active allosteric inhibitor of tyrosine kinase 2 (TYK2), with an IC50 of 0.2 nM and a Ki of 0.02 nM against the JH2 domain of TYK2, and it exhibits selectivity over other JAK subtypes and most of the kinome. Deucravacitinib blocks IL-23, IL-12, p-STAT1/3 and Type I IFN signaling, and inhibits Th17/Th1-mediated psoriasis inflammation. Deucravacitinib can be used in research related to moderate-to-severe plaque psoriasis, inflammatory bowel disease and systemic lupus erythematosus[1][2].

IC50 & Target[2]

Tyk2

0.2 nM (IC50)

JAK1

1 nM (IC50)

IL-23

IL-12

STAT1

STAT3

All JAK Isoforms :

JAK1

JAK2

JAK3

Tyk2

JAK

All STAT Isoforms :

STAT3

STAT5

STAT6

STAT1

All Interleukin Related Isoforms :

IL-1

IL-2

IL-3

IL-4

IL-5

IL-6

IL-8

IL-10

IL-12

IL-13

IL-15

IL-17

IL-23

IL-7

IL-33

IL-22

IL-18

In Vitro

Deucravacitinib potently inhibits TYK2-dependent IFNα-induced STAT5 phosphorylation in human CD3+ T cells, with

an IC50 of 2 nM[2].

Deucravacitinib potently inhibits TYK2-dependent IL-23-induced STAT3 phosphorylation in human CD161+ CD3+ T cells, with an IC50 of 9 nM[2].

Deucravacitinib potently inhibits TYK2-dependent IFNα-induced STAT5 phosphorylation in human whole blood with an IC50 of 13 nM, while exhibiting high functional selectivity for signaling pathways dependent on JAK2, JAK1 and JAK3[2].

MedChemExpress (MCE) has not independently confirmed the accuracy of these methods. They are for reference only.

Parmacokinetics

Species	Dose	Route	Cmax	AUC	Bioavailability	CL	Vss	T1/2	MRT
Mice[2]	1 mg/kg	i.v.	/	/	/	13.2 mL/min/kg	2.9 L/kg	4.2 h	3.6 h
Mice[2]	10 mg/kg	p.o.	7.5 μM	36.4 μM·h	122 %	/	/	/	/
Dog[2]	2 mg/kg	i.v.	/	/	/	6.8 mL/min/kg	2.3 L/kg	4.6 h	5.5 h
Dog[2]	10 mg/kg	p.o.	6.9 μM	73.6 μM·h	128 %	/	/	/	/
Monkey[2]	2 mg/kg	i.v.	/	/	/	4.8 mL/min/kg	2 L/kg	5.3 h	7 h
Monkey[2]	10 mg/kg	p.o.	5.9 μM	75.7 μM·h	87 %	/	/	/	/

In Vivo

Deucravacitinib (7.5-30 mg/kg; p.o.; twice daily; for 9 consecutive days) exhibits dose-dependent efficacy in a mouse IL-23-driven psoriasis model.

Deucravacitinib (50 mg/kg; p.o.; twice daily) almost completely inhibits body weight loss and significantly reduces histological damage in a mouse model of anti-CD40-induced colitis.

Deucravacitinib (30 mg/kg; p.o.; once daily; for 3 consecutive months) is well tolerated in a mouse lupus model and exerts significant efficacy in preventing nephritis.

SOLUBILITY

Soluble in DMSO

REFERENCES

[1]. Wrobleski ST, et al. Highly Selective Inhibition of Tyrosine Kinase 2 (TYK2) for the Treatment of Autoimmune Diseases: Discovery of the Allosteric Inhibitor BMS-986165. J Med Chem. 2019 Jul 18.

[2]. Catlett I, et al. SAT0226 A first-in-human, study of BMS-986165, a selective, potent, allosteric small molecule inhibitor of tyrosine kinase 2. Annals of the Rheumatic Diseases 2017;76:859.