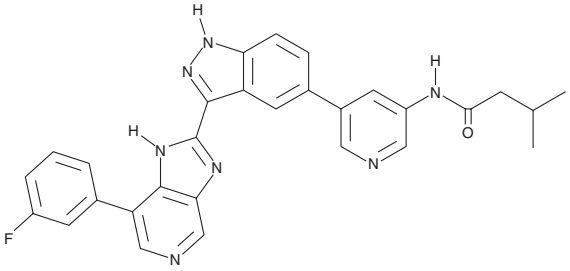
PRODUCT INFORMATION

Adavivint

Item No. 43927

CAS Registry No.:	1467093-03-3
Formal Name:	N-[5-[3-[7-(3-fluorophenyl)-3H-imidazo[4,5-c]pyridin2-yl]-1H-indazol-5-yl]-3-pyridinyl]-3-methyl-butanamide
Synonyms:	Lorecivivint, SM-04690
MF:	$C_{29}H_{24}FN_7O$
FW:	505.6
Purity:	≥98%
Supplied as:	A solid
Storage:	-20°C
Stability:	≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

Adavivint is supplied as a solid. A stock solution may be made by dissolving the adavivint in the solvent of choice, which should be purged with an inert gas. Adavivint is slightly soluble (0.1-1 mg/ml) in DMSO.

Description

Adavivint is an inhibitor of Wnt signaling (EC_{50} = 19.5 nM in a reporter assay using SW480 cells).[1] It inhibits CMGC family kinases, including CDC-like kinase 2 (Clk2) and dual-specificity tyrosine phosphorylation-regulated kinase 1A (DYRK1A; IC_{50}s = 7.8 and 26.9 nM, respectively), as well as Clk1, Clk3, Clk4, DYRK1B, glycogen synthase kinase 3β (GSK3β), and homeodomain-interacting protein kinase 1 (HIPK1), HIPK2, and HIPK3 (IC_{50}s = 16.8-239 nM).[2] Adavivint also induces activation of YES-associated transcriptional regulator (YAP) in a reporter assay using HEK293A cells (EC_{50} = 1.2 nM) in a Clk2-dependent manner.[3] It induces chondrogenesis in primary human bone marrow-derived mesenchymal stem cells (MSCs).[1] Adavivint inhibits the replication of pseudorabies virus in infected HeLa, BHK-21, Vero, and PK(15) cells (IC_{50}s = 0.16, 0.19, 0.22, and 0.35 µM, respectively).[4] Intra-arterial administration of adavivint (0.3 µg/animal) decreases disease severity in a surgically induced rat model of osteoarthritis.[1] Adavivint (50 mg/kg) also inhibits tumor growth in a patient-derived organoid xenograft mouse model of colorectal cancer.[5]

References

1. Deshmukh, V., Hu, H., Barroga, C., *et al*. A small-molecule inhibitor of the Wnt pathway (SM04690) as a potential disease modifying agent for the treatment of osteoarthritis of the knee. *Osteoarthritis Cartilage* **26(1)**, 18-27 (2018).
2. Deshmukh, V., O'Green, A.L., Bossard, C., *et al*. Modulation of the Wnt pathway through inhibition of CLK2 and DYRK1A by lorecivivint as a novel, potentially disease-modifying approach for knee osteoarthritis treatment. *Osteoarthritis Cartilage* **279(9)**, 1347-1360 (2019).
3. Bulos, M.L., Grzelak, E.M., Li-Ma, C., *et al*. Pharmacological inhibition of CLK2 activates YAP by promoting alternative splicing of AMOTL2. *bioRxiv [Preprint]* 1-17 (2023).
4. Wang, C., Wang, T., He, Q., *et al*. Inhibition of the canonical Wnt/β-catenin pathway interferes with macropinocytosis to suppress pseudorabies virus proliferation. *Vet. Microbiol.* **301**, 110373 (2025).
5. Xiang, Z., Wang, Y., Ma, X., *et al*. Targeting the NOTCH2/ADAM10/TCF7L2 axis-mediated transcriptional regulation of Wnt pathway suppresses tumor growth and enhances chemosensitivity in colorectal cancer. *Adv. Sci. (Weinh)* **12(3)**, e2405758 (2025).

CAYMAN CHEMICAL
1180 EAST ELLSWORTH RD
ANN ARBOR, MI 48108 · USA
PHONE: [800] 364-9897
[734] 971-3335
FAX: [734] 971-3640
CUSTSERV@CAYMANCHEM.COM
WWW.**CAYMANCHEM**.COM