

PRODUCT INFORMATION

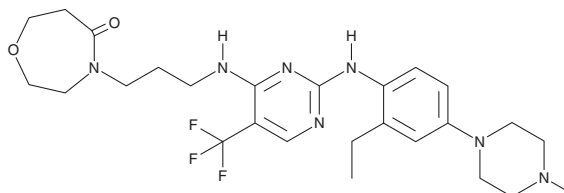


DCC-3116

Item No. 45313

CAS Registry No.: 2543673-19-2
Formal Name: 4-[3-[[[2-ethyl-4-(4-methyl-1-piperazinyl)phenyl]amino]-5-(trifluoromethyl)-4-pyrimidinyl]amino]propyl]tetrahydro-1,4-oxazepin-5(2H)-one

Synonym: Inlexisertib
MF: C₂₆H₃₆F₃N₇O₂
FW: 535.6
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

DCC-3116 is supplied as a solid. A stock solution may be made by dissolving the DCC-3116 in the solvent of choice, which should be purged with an inert gas. DCC-3116 is soluble (≥10 mg/ml) in ethanol and DMSO..

Description

DCC-3116 is an inhibitor of unc-51-like autophagy activating kinase 1/2 (ULK1/2).¹ It decreases phosphorylation of the ULK1 substrate ATG13 in NCI H2122, Calu-1, and NCI H358 non-small cell lung cancer (NSCLC) cells, which express the activating K-Ras^{G12C} mutation, at 2 μM. It also reduces basal autophagy and autophagy induced by the K-Ras^{G12C} inhibitor sotorasib (AMG 510; Item No. 29465) in the same cells. DCC-3116 (2 μM) inhibits the proliferation of Calu-1 and NCI H358 cells, with a synergistic effect when used in combination with sotorasib in NCI H2122 and Calu-1, but not NCI H358, cells. It cooperatively increases sotorasib-induced reductions in tumor volume in NCI H2122 and Calu-1 mouse xenograft models.

Reference

1. Ghazi, P.C., O'Toole, K.T., Srinivas Boggaram, S., *et al.* Inhibition of ULK1/2 and KRASG12C controls tumor growth in preclinical models of lung cancer. *Elife*. **13**, RP96992 (2024).

WARNING

THIS PRODUCT IS FOR RESEARCH ONLY - NOT FOR HUMAN OR VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

SAFETY DATA

This material should be considered hazardous until further information becomes available. Do not ingest, inhale, get in eyes, on skin, or on clothing. Wash thoroughly after handling. Before use, the user must review the complete Safety Data Sheet, which has been sent via email to your institution.

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