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Produktinformation

- Forschungsprodukte & Biochemikalien
- Zellkultur & Verbrauchsmaterial
- Diagnostik & molekulare Diagnostik
- Laborgeräte & Service

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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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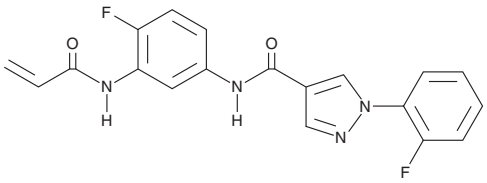
PRODUCT INFORMATION

EN6

Item No. 28715

CAS Registry No.:	1808714-73-9
Formal Name:	N-[4-fluoro-3-[(1-oxo-2-propen-1-yl) amino]phenyl]-1-(2-fluorophenyl)-1H-pyrazole-4-carboxamide
MF:	$C_{19}H_{14}F_2N_4O_2$
FW:	368.3
Purity:	≥95%
UV/Vis.:	λ_{max}: 268 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥2 years



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

Laboratory Procedures

EN6 is supplied as a crystalline solid. A stock solution may be made by dissolving the EN6 in the solvent of choice, which should be purged with an inert gas. EN6 is soluble in organic solvents such as DMSO and dimethyl formamide (DMF). The solubility of EN6 in these solvents is approximately 15 and 30 mg/ml, respectively.

EN6 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, EN6 should first be dissolved in DMF and then diluted with the aqueous buffer of choice. EN6 has a solubility of approximately 0.09 mg/ml in a 1:10 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

EN6 is an activator of autophagy.[1] It inhibits mammalian target of rapamycin complex 1 (mTORC1) signaling by binding covalently to cysteine 277 of the ATP6V1A catalytic subunit of the lysosomal vacuolar ATPase (v-ATPase) and impairing v-ATPase coupling with Rag GTPases. It is selective for inhibition of mTORC1 over mTORC2 signaling. EN6 increases lysosome acidification and increases cellular clearance of TDP-43 protein aggregates, which are a pathological feature of several neurodegenerative diseases, in a v-ATPase-dependent manner in U2OS osteosarcoma cells. It also inhibits mTORC1 signaling and enhances autophagy in mouse skeletal muscle and heart when administered at a dose of 50 mg/kg.

Reference

1. Chung, C.Y.-S., Shin, H.R., Berdan, C.A., *et al.* Covalent targeting of the vacuolar H^+-ATPase activates autophagy via mTORC1 inhibition. *Nat. Chem. Biol.* **15**, 776-785 (2019).

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