Small Molecules

FK-866

Nicotinamide phosphoribosyltransferase inhibitor

Catalog #100-0263	5 mg
100-0264	10 mg

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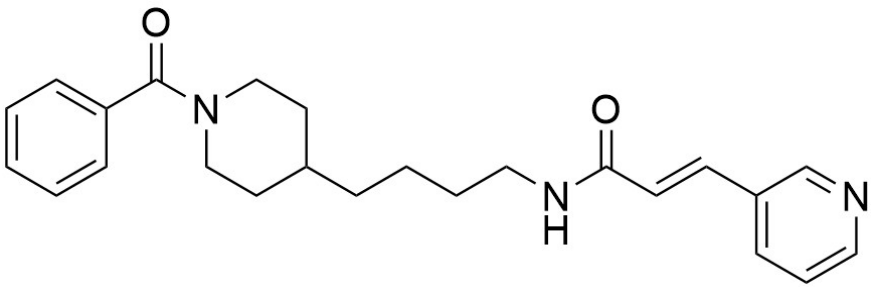
Product Description

FK-866 is a highly specific noncompetitive inhibitor of nicotinamide phosphoribosyltransferase (NAMPT), an enzyme that regulates NAD+ biosynthesis from the natural precursor nicotinamide (Cameron et al.; Hasmann & Schemainda). In hepatocarcinoma cells, FK-866 activates AMP-activated protein kinase (AMPK) and downregulates mammalian target of rapamycin (mTOR) signaling (Schuster et al.).

Molecular Name:	FK-866
Alternative Names:	K 22.175
CAS Number:	658084-64-1
Chemical Formula:	$C_{24}H_{29}N_3O_2$
Molecular Weight:	391.5 g/mol
Purity:	≥ 98%
Chemical Name:	N-[4-(1-benzoyl-4-piperidinyl)butyl]-3-(3-pyridinyl)-2E-propenamide
Structure:	



Properties

Physical Appearance:	A crystalline solid
Storage:	Product stable at -20°C as supplied. Protect from prolonged exposure to light. For long-term storage store with a desiccant. Stable as supplied for 12 months from date of receipt.
Solubility:	· PBS ≤ 1.2 mM · DMSO ≤ 60 mM · Absolute ethanol ≤ 100 mM For example, to prepare a 10 mM stock solution in DMSO, resuspend 1 mg in 255 µL of DMSO.

Prepare stock solution fresh before use. Information regarding stability of small molecules in solution has rarely been reported, however, as a general guide we recommend storage in DMSO at -20°C. Aliquot into working volumes to avoid repeated freeze-thaw cycles. The effect of storage of stock solution on compound performance should be tested for each application.

For use as a cell culture supplement, stock solution should be diluted into culture medium immediately before use. Avoid final DMSO concentration above 0.1% due to potential cell toxicity.

Published Applications

METABOLISM

· FK-866 can be used to deplete NAD+, a central metabolism cofactor (Cameron et al.; Hasmann & Schemainda; Jadeja et al.).

CANCER RESEARCH

· Depletes NAD+ and induces delayed cell death by apoptosis in HepG2 human liver carcinoma cells (IC50 = ~1 nM; Hasmann & Schemainda).

· Triggers dose-dependent cytotoxicity in multiple myeloma cells (Cea et al.).

· Induces autophagic death in neuroblastoma SH-SY5Y cells (IC50 = 0.93 nM; Billington et al.).

References

Billington RA et al. (2008) NAD depletion by FK866 induces autophagy. Autophagy 4(3): 385–7.

Cameron AM et al. (2019) Inflammatory macrophage dependence on NAD+ salvage is a consequence of reactive oxygen species-mediated DNA damage. Nat Immunol 20: 420–32.

Cea M et al. (2012) Targeting NAD+ salvage pathway induces autophagy in multiple myeloma cells via mTORC1 and extracellular signal-regulated kinase (ERK1/2) inhibition. Blood 120(17): 3519–29.

Hasmann M & Schemainda I. (2003) FK866, a highly specific noncompetitive inhibitor of nicotinamide phosphoribosyltransferase, represents a novel mechanism for induction of tumor cell apoptosis. Cancer Res 63(21): 7436–42.

Jadeja RN et al. (2018) Loss of NAMPT in aging retinal pigment epithelium reduces NAD+ availability and promotes cellular senescence. Aging 10(6): 1306–23.

Schuster S et al. (2015) FK866-induced NAMPT inhibition activates AMPK and downregulates mTOR signaling in hepatocarcinoma cells. Biochem Biophys Res Commun 458(2): 334–40.

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This product is hazardous. Please refer to the Safety Data Sheet (SDS).