BC1618

Cat. No.: HY-134656

CAS No.: 2222094-18-8

Molecular Formula: $C_{24}H_{24}F_3NO_2$

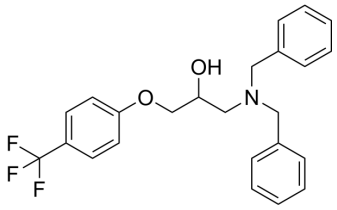
Molecular Weight: 415.45

Target: AMPK; Mitophagy; E1/E2/E3 Enzyme

Pathway: Epigenetics; PI3K/Akt/mTOR; Autophagy; Metabolic Enzyme/Protease

Storage:

Powder	-20°C	3 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (240.70 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.4070 mL	12.0351 mL	24.0703 mL
	5 mM	0.4814 mL	2.4070 mL	4.8141 mL
	10 mM	0.2407 mL	1.2035 mL	2.4070 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 5 mg/mL (12.04 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (6.02 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.02 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

BC1618, an orally active Fbxo48 inhibitory compound, stimulates Ampk-dependent signaling (via preventing activated pAmpkα from Fbxo48-mediated degradation). BC1618 promotes mitochondrial fission, facilitates autophagy and improves hepatic insulin sensitivity[1].

In Vitro

BC1618 enhances pAmpkα protein stability during CHX treatment[1].

BC1618 displays more than 1,000-fold enhanced activity to stimulate pAmpkα in cells than metformin[1].

BC1618 (0.1-2 μM, 16 h) induced dose- and time-dependent increases in pAmpkα and pACC protein levels are also confirmed

in human primary-like hepatocytes[1].
BC1618 (1 μM) effectively disrupts the interaction between Fbxo48 and pAmpkα, and has no effect on Fbxo48, Ampkα1 or Ampkα2 messenger RNAs[1].
BC1618 increases the abundance of a series of autophagic marker proteins during glucose depletion. BC1618 induces phosphorylation of the mTORC1 associated protein Raptor, reducing pS6 levels, all consistent with the known mTOR inhibitory effects exerted by activated Ampk[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	BEAS-2B cells.
Concentration:	0-2 μM.
Incubation Time:	16 h.
Result:	Induced pAmpkα and pACC protein levels dose-dependently.

In Vivo

BC1618 promotes mitochondrial fission, facilitates autophagy and improves hepatic insulin sensitivity in high-fat-diet-induced obese mice[1].
BC1618, appears to be ~1,000-fold more potent than metformin and is extremely well tolerated in mice[1].
BC1618 displays excellent oral bioavailability with a peak of 2,000 ng/mL within 0.5h and 500 ng/mL in plasma at 4h after an oral load of 20mg/kg[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	C57BL/6 mice[1].
Dosage:	2 or 10 mg/kg (challenged with LPS (3 mg/kg) for an additional 18 h).
Administration:	IP, once.
Result:	Reduced lung inflammation in endotoxin treated mice.

Animal Model:	C57BL/6 mice[1].
Dosage:	15 and 30 mg/kg/d.
Administration:	Drinking water for 3 months.
Result:	Exhibited no obvious toxicity.

CUSTOMER VALIDATION

- Research Square Preprint. 2023 Jun 15.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Yuan Liu, et al. A Fbxo48 inhibitor prevents pAMPKα degradation and ameliorates insulin resistance. Nat Chem Biol. 2021 Mar;17(3):298-306.

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA