Aldometanib

Cat. No.: HY-148189

CAS No.: 2904601-67-6

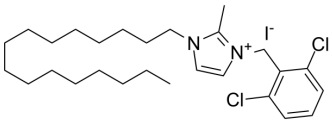
Molecular Formula: $C_{27}H_{43}Cl_2IN_2$

Molecular Weight: 593.45

Target: AMPK

Pathway: Epigenetics; PI3K/Akt/mTOR

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (168.51 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.6851 mL	8.4253 mL	16.8506 mL
	5 mM	0.3370 mL	1.6851 mL	3.3701 mL
	10 mM	0.1685 mL	0.8425 mL	1.6851 mL

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

BIOLOGICAL ACTIVITY

Description

Aldometanib (LXY-05-029) is an orally active aldolase inhibitor. Aldometanib can activate lysosomal adenosine monophosphate-activated protein kinase (AMPK) and decreases blood glucose. Aldometanib can be used for the research of metabolic homeostasis[1].

In Vitro

Aldometanib (0-1000 nM; 2 h) activates AMPK by preventing aldolase from binding to FBP to engender a pseudo-starvation signal[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	Mouse primary hepatocytes, MEFs cells
Concentration:	0-1000 nM
Incubation Time:	2 h
Result:	Activated AMPK in mouse embryonic fibroblasts (MEFs) and mouse primary hepatocytes cells.

Immunofluorescence[1]

Cell Line:	MEFs cells
Concentration:	5 nM
Incubation Time:	2 h
Result:	Inhibited TRPVs and induces AXIN lysosomal translocation.

In Vivo

Aldometanib (oral; 0-10 mpk) reduces blood glucose in lean mice[1].
?Aldometanib (oral; 2-10 mpk; twice daily; for a week) reduces blood glucose and alleviates fatty liver in obese hyperglycaemic mice[1].
?Aldometanib alleviates fatty liver and nonalcoholic steatohepatitis[1].
?Aldometanib (oral; 2mpk; twice-daily; for a month) alleviates liver fibrosis in NASH mice[1].
?Aldometanib (oral; 0-50 μM; 0-50 days) extends lifespan in C. elegans via the lysosomal pathway[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Lean mice[1]
Dosage:	0-10 mpk
Administration:	Oral
Result:	Decreased fasting blood glucose and improved glucose tolerance, promoted muscular TBC1D1 phosphorylation and glucose uptake.

Animal Model:	Obese hyperglycaemic mice[1]
Dosage:	2-10 mpk
Administration:	Oral, twice daily, for a week
Result:	Decreased blood glucose, lowered blood glucose in a muscular AMPK-dependent manner reduced hepatic TAG, improved insulin sensitivity, increased glucose disposal rates, inhibited TAG synthesis in liver and primary hepatocytes, decreased fat mass.

Animal Model:	NASH mice[1]
Dosage:	2 mpk
Administration:	Oral, twice-daily, for a month
Result:	Decreased histological scores used to describe the features of NASH, reduced apoptosis rate of hepatic cells, inhibited inflammatory responses in the liver of NASH mice and improved glucose tolerance of NASH mice.

Animal Model:	C. elegans[1]
Dosage:	0-50 μM
Administration:	Oral, 0-50 days
Result:	Promoted oxidative stress resistance and mitochondrial functions in C. elegans.

Animal Model:	C57BL/6 mice[1]
Dosage:	100 μg/mL
Administration:	Oral
Result:	Extended lifespan, elevated NAD⊠ levels and mitochondrial oxidative respiration, rejuvenated muscle function in aged mice.

CUSTOMER VALIDATION

- Life Metabolism. 2023 Mar 1.

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REFERENCES

[1]. Chen-Song Zhang, et al. The aldolase inhibitor aldometanib mimics glucose starvation to activate lysosomal AMPK. Nat Metab. 2022 Oct 10.

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