JBSNF-000088

Cat. No.: HY-112584

CAS No.: 7150-23-4

Molecular Formula: $C_7H_8N_2O_2$

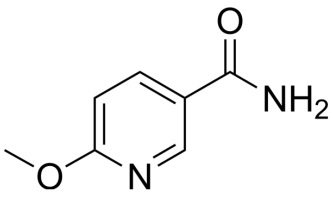
Molecular Weight: 152.15

Target: Others

Pathway: Others

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (657.25 mM)

H_2O : 1 mg/mL (6.57 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	6.5725 mL	32.8623 mL	65.7246 mL
5 mM	1.3145 mL	6.5725 mL	13.1449 mL
10 mM	0.6572 mL	3.2862 mL	6.5725 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 2.94 mg/mL (19.32 mM); Clear solution; Need ultrasonic and warming and heat to 60°C
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (13.67 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (13.67 mM); Suspended solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (13.67 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

JBSNF-000088 (6-Methoxynicotinamide), a analog of nicotinamide (NA), is a potent and orally active Nicotinamide N-methyltransferase (NNMT) inhibitor with IC_{50}s of 1.8 μM, 2.8 μM, and 5.0 μM for human NNMT, monkey NNMT and mouse NNMT, respectively. JBSNF-000088 inhibits NNMT activity, reduces MNA levels and drives insulin sensitization, glucose

	modulation and body weight reduction in animal models of metabolic disease[1].
IC_{50} & Target	IC50: 1.8 μM (human NNMT), 2.8 μM (monkey NNMT) and 5.0 μM (mouse NNMT)[1]
In Vitro	JBSNF-000088 (6-Methoxynicotinamide) has IC_{50} values are 1.6 and 6.3?μM for U2OS or differentiated 3T3L1 cells[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	JBSNF-000088 (6-Methoxynicotinamide) (50?mg/kg; oral route of administration for four weeks) shows statistically significant reduction in body weight (%) and leads to a statistically significant reduction in fed blood glucose on day 21[1]. ?JBSNF-000088 (50?mg/kg; oral gavage administration; twice daily for four weeks) leads to a statistically significant improvement in oral glucose tolerance on day 28 with glucose tolerance being normalized[1]. ?JBSNF-000088 (1 mg/kg; intravenous administration; for 4 hours) results in low plasma clearance of 21?mL/min?kg and the volume of distribution at steady state of 0.7?L/kg, a very short plasma half-life of 0.5?hours upon intravenous administration [1]. ?JBSNF-000088 (10 mg/kg; oral gavage; for 4 hours) results in a Cmax of 3568 ng/mL with a T_{max} value of 0.5?hours, indicating rapid absorption in the intestine, and half-life of 0.4?hours by oral gavage. The oral bioavailability is found to be approximately 40%[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Mice with high fat diet (HFD)-induced obesity[1]
Dosage:	50 mg/kg
Administration:	Oral route of administration for four weeks; oral gavage administration and twice daily for four weeks
Result:	Showed statistically significant reduction in body weight (%) and led to a statistically significant reduction in fed blood glucose on day 21 by oral route of administration. Led to a statistically significant improvement in oral glucose tolerance on day 28 with glucose tolerance being normalized by oral gavage administration.

Animal Model:	C57BL/6 mice[1]
Dosage:	1 mg/kg (Intravenous administration);10 mg/kg (oral gavage)(Pharmacokinetic Study)
Administration:	Intravenous administration and oral gavage; for 4 hours
Result:	Resulted in low plasma clearance of 21 mL/min□kg and the volume of distribution at steady state of 0.7 L/kg, a very short plasma half-life of 0.5 h upon intravenous administration. Resulted in a Cmax of 3568 ng/mL with a T_{max} value of 0.5 hours, indicating rapid absorption in the intestine, and half-life of 0.4 hours by oral gavage.

CUSTOMER VALIDATION

- Chemosphere. 2021, 129727.
- Am J Physiol Cell Physiol. 2023 May 22.
- Am J Physiol Cell Physiol. 2021 Aug 11.

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REFERENCES

[1]. Kannt A, et al. A small molecule inhibitor of Nicotinamide N-methyltransferase for the treatment of metabolic disorders. Sci Rep. 2018 Feb 26;8(1):3660.

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