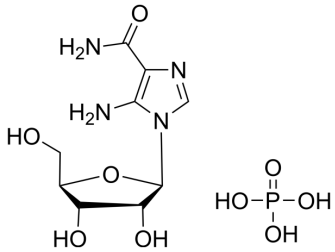
AICAR phosphate

Cat. No.:	HY-13417A
CAS No.:	681006-28-0
Molecular Formula:	$C_9H_{17}N_4O_9P$
Molecular Weight:	356.23
Target:	AMPK; Autophagy; Mitophagy; YAP; Endogenous Metabolite
Pathway:	Epigenetics; PI3K/Akt/mTOR; Autophagy; Stem Cell/Wnt; Metabolic Enzyme/Protease
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

H_2O : 100 mg/mL (280.72 mM; Need ultrasonic)

Preparing Stock Solutions

Mass / Solvent / Concentration	1 mg	5 mg	10 mg
1 mM	2.8072 mL	14.0359 mL	28.0718 mL
5 mM	0.5614 mL	2.8072 mL	5.6143 mL
10 mM	0.2807 mL	1.4036 mL	2.8072 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 33.33 mg/mL (93.56 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (5.84 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (5.84 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.84 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

AICAR phosphate (Acadesine phosphate) is an adenosine analog and a AMPK activator. AICAR phosphate regulates the glucose and lipid metabolism, and inhibits proinflammatory cytokines and iNOS production. AICAR phosphate is also an autophagy, YAP and mitophagy inhibitor[1][2].

IC_{50} & Target	AMPK	Autophagy	Mitophagy	Human Endogenous Metabolite

In Vitro	HepG2 cells are treated with various concentrations of AICAR (0.1-1.0 mM) for 12, 24, and 48 h, respectively. The expression level of IR-β significantly decreases with 0.25, 0.5, and 1.0 mM of AICAR at 48 h to 50%, 53%, and 46% of the control, respectively[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Fourteen-week-old male, lean (L; 31.3 g body wt) wild-type andob/ob (O; 59.6 g body wt) mice are injected with the AMP-activated kinase (AMPK) activator AICAR (A) at 0.5 mg*g body wt^{-1}*day^{-1} or saline control (C) for 14 days. At 24 h after the last injection (including a 12-h fast), all mice are killed, and the plantar flexor complex muscle (gastrocnemius, soleus, and plantaris) is excised for analysis. Muscle mass is lower in OC (159±12 mg) than LC, LA, and OA (176±10, 178±9, and 166±16 mg, respectively) mice, independent of a body weight change[3]. The kidney weight is significantly higher in the untreated group when compared with both the exercise and AICAR (0.5 mg/g body wt) groups. The heart weight is higher in the exercise group than in the other groups, whereas the liver weight is significantly higher in the AICAR-treated group when compared with the exercise and untreated groups[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	HepG2 cells (5×10^5 cells) are plated in 6-well culture plate dishes and then are incubated in the serum-free media for 12 h before transfection. One microgram of plasmid is transfected with FuGENE6 Transfection Reagent. After 5 h of transfection, the culture media are removed and then media supplemented with or without AICAR (0.1-1.0 mM) are added to each well. The stimulation media are changed every 24 h[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2][3]	Mice[2] Fourteen-week-old lean ($Lep^{ob/+}$ or $Lep^{ob/+}$) and ob/ob ($Lep^{ob}/Lep^{ob/}$) male mice are uesd. After the 14-day experimental treatment (24 h after AICAR injection, including a 12-h fast), the plantar flexor complex muscle is cleanly (tendon-to-tendon) excised from an anesthetized mouse. The muscle is quickly weighed and then processed for histology or frozen in liquid nitrogen and stored at -80°C. The anesthetized mice are killed by transection of the diaphragm and removal of the entire heart, after blood collection via needle puncture directly into the heart. AICAR or saline (control) is injected subcutaneously into the lateral distal portion of the back. AICAR is administered at 0.5 mg*g body wt^{-1}*day^{-1} one time per day for 14 days. Saline (control) is injected in volumes identical to those used for AICAR treatment in a manner identical to that of AICAR treatment. Body weight is measured prior to death. Rats[3] Male 5-week-old ZDF rats are either subcutaneously injected with a single dose of AICAR (0.5 mg/g body wt) or underwent a single bout of treadmill running (60 min, speed of 25 m/min at a 5% incline). Untreated ZDF rats serve as controls (n=5 in each group). One hour after the subcutaneous AICAR injection or immediately after treadmill running, rats are killed by cervical dislocation. To avoid any effect of muscle spasm and hypoxia, red and white gastrocnemius muscles are removed within seconds and immediately freeze clamped for later determination of AMPK activity. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cancer Cell. 2020 Sep 14;38(3):350-365.e7.
- Signal Transduct Target Ther. 2023 Mar 6;8(1):95.
- Signal Transduct Target Ther. 2022 Feb 14;7(1):40.
- Cell Metab. 2019 Jul 2;30(1):157-173.e7
- Cell Death Differ. 2021 Apr;28(4):1222-1236.

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REFERENCES

[1]. Nakamaru K, et al. AICAR, an activator of AMP-activated protein kinase, down-regulates the IR expression in HepG2 cells. Biochem Biophys Res Commun. 2005 Mar 11;328(2):449-54

[2]. Drake JC, et al. AICAR treatment for 14 days normalizes obesity-induced dysregulation of TORC1 signaling and translational capacity in fasted skeletal muscle. Am J Physiol Regul Integr Comp Physiol. 2010 Dec;299(6):R1546-54.

[3]. Pold R, et al. Long-term AICAR administration and exercise prevents diabetes in ZDF rats.Diabetes. 2005 Apr;54(4):928-34.

[4]. Giri S, et al. 5-aminoimidazole-4-carboxamide-1-beta-4-ribofuranoside inhibits proinflammatory response in glial cells: a possible role of AMP-activated protein kinase. J Neurosci. 2004 Jan 14;24(2):479-87.

[5]. Ajaybabu V Pobbati, et al. A combat with the YAP/TAZ-TEAD oncoproteins for cancer therapy. Theranostics. 2020 Feb 18;10(8):3622-3635.

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

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