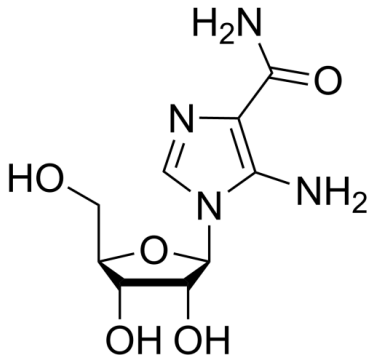
Data Sheet

Product Name:	AICAR
Cat. No.:	CS-1951
CAS No.:	2627-69-2
Molecular Formula:	$C_9H_{14}N_4O_5$
Molecular Weight:	258.23
Target:	AMPK; Autophagy; Endogenous Metabolite; Mitophagy; YAP
Pathway:	Autophagy; Epigenetics; Metabolic Enzyme/Protease; PI3K/Akt/mTOR; Stem Cell/Wnt
Solubility:	H_2O : 65 mg/mL (ultrasonic;warming);DMSO : 125 mg/mL (ultrasonic)



BIOLOGICAL ACTIVITY:

AICAR (Acadesine) is an adenosine analog and a **AMPK** activator. AICAR regulates the glucose and lipid metabolism, and inhibits proinflammatory cytokines and iNOS production. AICAR is also an **autophagy**, **YAP** and **mitophagy** inhibitor[1][2]. IC50 & Target:AMPK[1] ***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]. ***In Vivo:*** Fourteen-week-old male, lean (L; 31.3 g body wt) wild-type and ob/ob (O; 59.6 g body wt) mice are injected with the AMP-activated kinase (AMPK) activator AICAR (A) at 0.5 mg/g per day 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].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: AICAR is dissolved in DMSO and stored, and then diluted with appropriate medium before use[1].[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]. **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 per day one time 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.

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.

CAIndexNames:

1H-Imidazole-4-carboxamide, 5-amino-1-β-D-ribofuranosyl-

SMILES:

OC[C@@H]1[C@@H](O)[C@@H](O)[C@H](N2C(N)=C(N=C2)C(N)=O)O1

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