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Produktinformation

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- Diagnostik & molekulare Diagnostik
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- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

F. +43(0)1 489 3961-7

mail@szabo-scandic.com

www.szabo-scandic.com

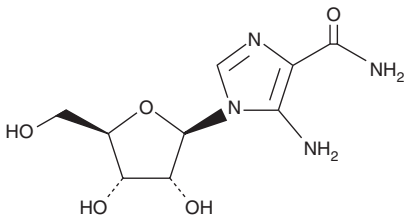
linkedin.com/company/szaboscandic

PRODUCT INFORMATION

AICAR

Item No. 10010241

CAS Registry No.:	2627-69-2
Formal Name:	5-amino-1-β-D-ribofuranosyl-1H-imidazole-4-carboxamide
Synonyms:	Acadesine, AICA-Riboside, NSC 105823
MF:	$C_9H_{14}N_4O_5$
FW:	258.2
Purity:	≥98%
UV/Vis.:	λ_{max}: 268 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥2 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

AICAR is supplied as a crystalline solid. A stock solution may be made by dissolving the AICAR in the solvent of choice. AICAR is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide, which should be purged with an inert gas. The solubility of AICAR in these solvents is approximately 1, 20, and 10 mg/ml, respectively.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. Organic solvent-free aqueous solutions of AICAR can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of AICAR in PBS, pH 7.2, is approximately 2.5 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

AICAR is an activator of AMP-activated protein kinase (AMPK).[1] It increases AMPK kinase activity in isolated hepatocytes (EC_{50} = ~500 µM) and reduces HMG-CoA reductase activity and fatty acid and sterol synthesis in these cells. AICAR (0.5 mM) inhibits insulin-stimulated glucose uptake to 62% of control cells and reduces GLUT4 translocation by 2.5-fold in 3T3-L1 adipocytes.[2] In astrocyte-enriched glial cells, AICAR (1 mM) prevents increases in protein levels of inducible nitric oxide synthase (iNOS), COX-2, and manganese superoxide dismutase (MnSOD) induced by LPS and amyloid-β (Aβ) (25-35) and expression of TNF-α, IL-1β, and IL-6 induced by LPS and Aβ42.[3] AICAR inhibits autophagy in rat hepatocytes (IC_{50} = 0.3 mM) and induces apoptosis in rat β cells in a concentration-dependent manner.[4,5]

References

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2. Salt, I.P., Connell, J.M.C., and Gould, G.W. *Diabetes* **49(10)**, 1649-1656 (2000).
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4. Samari, H.R. and Seglen, P.O. *J. Biol. Chem.* **273(37),** 23758-23763 (1998).
5. Kefas, B.A., Heimberg, H., Vaulont, S., *et al. Diabetologia* **46(2),** 250-254 (2003).

CAYMAN CHEMICAL
1180 EAST ELLSWORTH RD
ANN ARBOR, MI 48108 · USA
PHONE: [800] 364-9897
[734] 971-3335
FAX: [734] 971-3640
CUSTSERV@CAYMANCHEM.COM
WWW.**CAYMANCHEM**.COM