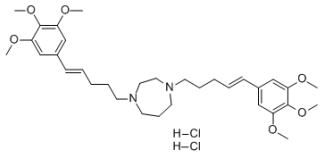
K-7174 dihydrochloride

Cat. No.:	HY-12743A
CAS No.:	191089-60-8
Molecular Formula:	$C_{33}H_{50}Cl_2N_2O_6$
Molecular Weight:	641.67
Target:	Others
Pathway:	Others

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

H_2O : 15 mg/mL (23.38 mM; Need ultrasonic and warming)

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	1.5584 mL	7.7922 mL	15.5843 mL
5 mM	0.3117 mL	1.5584 mL	3.1169 mL
10 mM	0.1558 mL	0.7792 mL	1.5584 mL

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

BIOLOGICAL ACTIVITY

Description

K-7174 dihydrochloride is a novel cell adhesion inhibitor; inhibits the expression of vascular cell adhesion molecule-1 (VCAM-1) induced by either IL-1β or TNF-α.IC50 value:Target: GATA-specific inhibitorin vitro: K-7174 inhibited the expression of vascular cell adhesion molecule-1 (VCAM-1) induced by either tumor necrosis factor alpha or interleukin-1beta, without affecting the induction of intercellular adhesion molecule-1 or E-selectin. K-7174 had no effect on the stability of VCAM-1 mRNA.K-7174 did not influence the binding to any of the following binding motifs: octamer binding protein, AP-1, SP-1, ets, NFkappaB, or interferon regulatory factor [1]. Addition of 10 microM K-7174 rescued these inhibitions of Epo protein production and promoter activity induced by IL-1beta, TNF-alpha, or L-NMMA, respectively [2]. K-7174 had the potential to induce endoplasmic reticulum (ER) stress evidenced by induction of GRP78 and CHOP.Other inducers of ER stress completely reproduced the effects of K-7174 including suppression of lipid accumulation, blockade of induction of adiponection and PPARgamma and maintenance of MCP-1 expression [3].in vivo: K-7174, one of proteasome inhibitory homopiperazine derivatives, exhibits a therapeutic effect, which is stronger when administered orally than intravenously, without obvious side effects in a murine myeloma model. Moreover, K-7174 kills bortezomib-resistant myeloma cells carrying a β5-subunit mutation in vivo and primary cells from a patient resistant to bortezomib [4].

CUSTOMER VALIDATION

- FASEB J. 2020 Mar;34(3):4462-4481.
- FEBS Open Bio. 2020 Aug 2.
- Research Square Preprint. 2020 Dec.
- Department of Molecular Medicine and Biopharmaceutical Sciences. 2020 May.
- bioRxiv. 2019 Sep.

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REFERENCES

[1]. Umetani M, et al. A novel cell adhesion inhibitor, K-7174, reduces the endothelial VCAM-1 induction by inflammatory cytokines, acting through the regulation of GATA. Biochem Biophys Res Commun. 2000 Jun 7;272(2):370-4.

[2]. Imagawa S, et al. A GATA-specific inhibitor (K-7174) rescues anemia induced by IL-1beta, TNF-alpha, or L-NMMA. FASEB J. 2003 Sep;17(12):1742-4.

[3]. Shimada T, et al. Unexpected blockade of adipocyte differentiation by K-7174: implication for endoplasmic reticulum stress. Biochem Biophys Res Commun. 2007 Nov 16;363(2):355-60.

[4]. Kikuchi J, et al. The novel orally active proteasome inhibitor K-7174 exerts anti-myeloma activity in vitro and in vivo by down-regulating the expression of class I histone deacetylases. J Biol Chem. 2013 Aug 30;288(35):25593-602.

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