

Product Data Sheet

Product Name: Anisomelic acid

Cat. No.: GC78810

Chemical Properties

Cas. No. 59632-76-7

Formula $C_{20}H_{26}O_4$

M.Wt 330.42

Solubility

Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Background

Anisomelic acid is a human papillomavirus HPV16 E6 and HPV E7 depletor. Anisomelic acid directly interacts with HPV16 E6, promotes its ubiquitination and proteasomal degradation by recruiting E3 ubiquitin ligase, downregulates E6 and facilitates E7 degradation. Anisomelic acid induces endogenous mitochondrial apoptosis, activates caspase-3, causes cleavage of PARP, and triggers p53-independent endogenous apoptosis by depleting cIAP2. Anisomelic acid can be used in research related to HPV-positive cancers and cervical cancer [1] [2] [3].

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

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In Vivo, Anisomelic acid (2-20 mg/kg; topical administration; once daily for 5 consecutive days) inhibits cervical cancer tumor growth in a dose-dependent manner in the chick chorioallantoic membrane (CAM) model^[3].

In Vitro, Anisomelic acid (40 μ M; 24 h) induces proteasome-dependent degradation of exogenous HPV16 E6 in transfected SiHa cells by enhancing E6 ubiquitination^[1]. Anisomelic acid (20-40 μ M; 12-24 h) downregulates the HPV16-E6 and HPV16-E7 oncoproteins and stabilizes p53 and p21 in SiHa cervical cancer cells^[3]. Anisomelic acid (20-40 μ M; 12-24 h) mainly induces apoptosis in SiHa cervical cancer cells by activating caspase-9, accompanied by subsequent cleavage of caspase-3 and PARP, as well as delayed cleavage of caspase-8^[3]. Anisomelic acid (40 μ M; 12 h) downregulates the expression of E6 and cIAP2 in SiHa cervical cancer cells, and this process is mediated by the proteasomal degradation pathway^[3]. Anisomelic acid (10-40 μ M; 24 h) induces concentration-dependent cell death and apoptosis in NOK16 cells by downregulating HPV16 E6^[1]. Anisomelic acid (20 μ M; 24 h) induces apoptosis in human cervical cancer SiHa cells; among the formulations tested, anisomelic acid loaded onto folate-chitosan coated rod-like mesoporous silica nanoparticles induces the highest level of apoptosis, which is approximately 3.2 times that of free anisomelic acid^[2]. Anisomelic acid (20 μ M; 24 h) activates the apoptotic machinery in SiHa human cervical cancer cells via cleavage of procaspase-3 and PARP^[2]. Anisomelic acid (20-40 μ M; 48 h) induces dose-dependent apoptotic DNA fragmentation in SiHa cervical cancer cells^[3]. Anisomelic acid (20-40 μ M; 24 h) downregulates the expression level of cIAP2 protein in SiHa cervical cancer cells^[3]. Anisomelic acid (40 μ M; 48 h) preferentially induces apoptosis in HPV-positive cervical cancer cells (SiHa, CaSki, HeLa) compared with HPV-negative cervical cancer cells (c33a), breast cancer cells (MCF7, MDA-MB-231), and non-cancerous MEF cells^[3]. Anisomelic acid (20-40 μ M; 8-48 h) induces time- and dose-dependent mitochondrial membrane depolarization in SiHa cervical cancer cells^[3]. Anisomelic acid (20-40 μ M; 24 h) induces G2/M phase cell cycle arrest in synchronized SiHa cervical cancer cells^[3].

References:

[1]. Santos Silva M, et al. Anisomelic acid promotes proteasomal degradation of HPV16 E6 via E3 ligase recruitment: A mass spectrometry-based interactome study. Journal of proteomics. 2026 Jan

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[2]. Senthilkumar R, et al. Targeted delivery of a novel anticancer compound anisomelic acid using chitosan-coated porous silica nanorods for enhancing the apoptotic effect. Biomaterials science. 2015 Jan;3(1):103-11.

[3]. Paul P, et al. Novel action modality of the diterpenoid anisomelic acid causes depletion of E6 and E7 viral oncoproteins in HPV-transformed cervical carcinoma cells. Biochemical pharmacology. 2014 May 15;89(2):171-84.

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