

Product Data Sheet

Product Name: Acitretin
Cat. No.: GC17094

Chemical Properties

Cas. No. 55079-83-9

Chemical Name (2E,4E,6E,8E)-9-(4-methoxy-2,3,6-trimethylphenyl)-3,7-dimethylnona-2,4,6,8-tetraenoic acid

SMILES CC1=CC(=C(C(=C1C=CC(=CC=CC(=CC(=O)O)C)C)C)C)OC

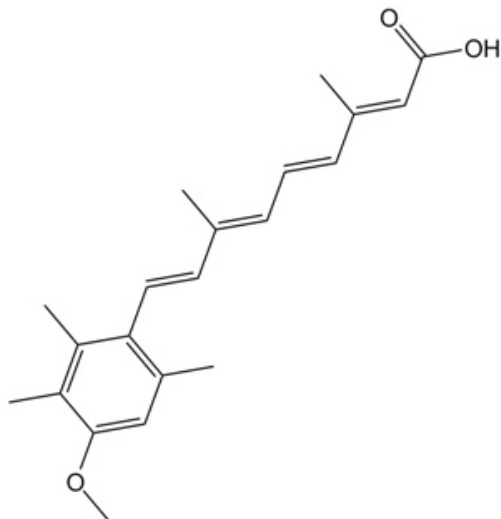
Formula $C_{21}H_{26}O_3$ M.Wt 326.43

Solubility $\geq 16.32\text{mg/mL}$ in DMSO 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



Protocol

Cell experiment [1]:

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Cell lines	HaCaT cells (human immortal keratinocyte cell line)
Preparation Method	HaCaT cells were cultured in DMEM supplemented with 100U/mL streptomycin and 10% heat-inactivated fetal bovine serum at 37°C, 5% CO ₂ . HaCaT cells were treated with 5µM Acitretin for 12, 24, 48 and 72 hours. Cell viability was assessed by MTS assay. STAT1, STAT3, SOCS1 and SOCS3 mRNA and protein expression were determined by Q-PCR and Western blot.
Reaction Conditions	5µM; 12-72 hours
Applications	Acitretin inhibited HaCaT cells proliferation, down-regulated STAT1, STAT3, SOCS1 and SOCS3 mRNA and protein expression.
Animal experiment [2]:	
Animal models	C57BL/6J mice
Preparation Method	Mice were intraperitoneally injected with 10mg/kg Acitretin or PBS for three consecutive days. Six hours after the final drug administration, mice were intraperitoneally injected with 10mg/kg lipopolysaccharide to induce sepsis. Twelve hours post-administration, mice were sacrificed and samples were collected. Survival analysis was performed by intraperitoneal injection of 30mg/kg lipopolysaccharide and monitoring for 96h. Lung function was assessed using whole-body plethysmography.
Dosage form	10mg/kg; i.p.; three consecutive days

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Applications	Acitretin reduced total cell counts, protein levels, neutrophils and macrophages accumulation, and IL-1 β levels in bronchoalveolar lavage fluid and serum, decreased lung wet-to-dry weight ratio, improved survival rate, dynamic lung compliance and airway resistance, attenuated inflammatory cell recruitment and structural damage in lung parenchyma, inhibited IL-1 β release, caspase-1 p20 production and GSDMD cleavage in alveolar macrophages, and reduced markers of liver, heart and kidney injury in mice.
References:[1] Qin X, Chen C, Zhang Y, et al. Acitretin modulates HaCaT cells proliferation through STAT1- and STAT3-dependent signaling. Saudi Pharm J. 2017;25(4):620-624.[2] Xu H, Xu H, Li W, et al. Modulating the NLRP3 Inflammasome: Acitretin as a potential treatment for Sepsis-induced acute lung injury. Int Immunopharmacol. 2025;153:114504.	

Background

Acitretin is a second-generation aromatic retinoid drug. Acitretin binds to Retinoic Acid Receptor (RAR) $\alpha/\beta/\gamma$ and retinoid X receptor (RXR) to regulate gene transcription. Acitretin reduces inflammation by downregulating the expression of inflammatory factors such as IL-6, MRP-8, and IFN- γ and inhibiting neutrophil chemotaxis. Acitretin can be used for research on keratinization disorders including severe plaque psoriasis, pustular psoriasis, erythrodermic psoriasis, Darier disease, pityriasis rubra pilaris, and congenital ichthyosis, as well as chemoprevention of non-melanoma skin cancer after organ transplantation^[1-4].

In vitro, SH-SY5Y cells were treated with 10 μ M Acitretin for 4 days. Acitretin promoted cell differentiation into neuron-like cells with discharge function. Acitretin upregulated the expression of β -tubulin III and NFH^[5]. HaCaT cells were treated with 5 μ M Acitretin for 12-72 hours. Acitretin inhibited cell proliferation, downregulated STAT1, STAT3, SOCS1, and SOCS3 mRNA and protein expression^[6]. SCL-1 cells were treated with 10 μ M Acitretin. Acitretin inhibited cell proliferation, induced cell apoptosis^[7].

In vivo, a psoriasis BALB/c mouse model received 400 μ L 0.3mg/mL Acitretin by gavage once daily for 7 days. Acitretin improved the psoriasiform dermatitis phenotype, reduced the total Psoriasis Area and Severity Index score, reduced IL-23a, IL-6, and Ki67 expression in skin lesions, increased

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Involucrin expression^[8]. A sepsis C57BL/6J mouse model received intraperitoneal injection of 10mg/kg Acitretin for 3 days. Acitretin reduced total cell counts and protein levels in bronchoalveolar lavage fluid, reduced the lung wet-to-dry weight ratio, attenuated alveolar wall thickening and immune cell infiltration, improved the survival rate, improved dynamic lung compliance and airway resistance^[9]. C57BL6/J OlaHsd mice received intraperitoneal injection of 10mg/kg Acitretin for 7 days. Acitretin increased liver triglyceride, total cholesterol, and free cholesterol content^[10].

References:

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- [4] Sauder MB, Cheung L, Beecker J. Acitretin-induced hepatitis: when to monitor cholestatic enzymes. J Cutan Med Surg. 2015 Mar-Apr;19(2):115-20.
- [5] Cai A, Liu N, Lin Z, et al. In Vitro Effects of Acitretin on Human Neuronal SH-SY5Y Cells. Neurochemical Research. 2023;48(1):72-81.
- [6] Qin X, Chen C, Zhang Y, et al. Acitretin modulates HaCaT cells proliferation through STAT1- and STAT3-dependent signaling. Saudi Pharm J. 2017;25(4):620-624.
- [7] Lin X, He C, Xiao T, et al. Acitretin induces apoptosis through CD95 signalling pathway in human cutaneous squamous cell carcinoma cell line SCL-1. J Cell Mol Med. 2009;13(9A):2888-2898.
- [8] Long K, Chen W, Mao M, et al. Role of acitretin in regulating glucose and lipid homeostasis in an imiquimod-induced psoriasis model mouse. J Cent South Univ(Med Sci). 2025;50(3):344-357.
- [9] Xu H, Xu H, Li W, et al. Modulating the NLRP3 Inflammasome: Acitretin as a potential treatment for Sepsis-induced acute lung injury. Int Immunopharmacol. 2025;153:114504.
- [10] Lauer A.A., Nguyen V.T.T., Janitschke D, et al. The Influence of Acitretin on Brain Lipidomics in Adolescent Mice-Implications for Pediatric and Adolescent Dermatological Therapy. Int. J. Mol. Sci. 2022, 23, 15535.

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