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Bafilomycin A1(Baf-A1)

Technical Data

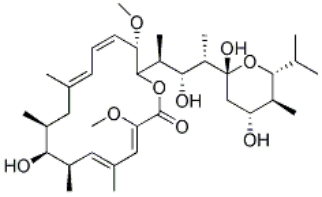
Molecular Weight	622.83	**Storage**	3 years -20°C powder 2 years -80°C in solvent
Formula	$C_{35}H_{58}O_9$		
CAS No.	88899-55-2	**Synonyms**	N/A
Chemical Name	Oxacyclohexadeca-3,5,11,13-tetraen-2-one, 8-hydroxy-16-[(1S,2R,3S)-2-hydroxy-1-methyl-3-[(2R,4R,5S,6R)-tetrahydro-2,4-dihydroxy-5-methyl-6-(1-methylethyl)-2H-pyran-2-yl]butyl]-3,15-dimethoxy-5,7,9,11-tetramethyl-, (3Z,5E,7R,8S,9S,11E,13E,15S,16R)-		
Solubility (25°C) *	In vitro		
	In vivo（should be freshly prepared each time）		

* <1 mg/ml means slightly soluble or insoluble.
* Please note that Selleck tests the solubility of all compounds in-house, and the actual solubility may differ slightly from published values. This is normal and is due to slight batch-to-batch variations.

Biological Activity

Description	Bafilomycin A1 is a vacuolar **H^+-ATPase** inhibitor with **IC50** of 0.44 nM. Bafilomycin A1 is found to inhibit **autophagy** while induces **apoptosis**.
Targets	H+-ATPase [1] (Cell-free assay) 0.44 nM
In vitro	Bafilomycin A1 is a toxic macrolide antibiotic derived from Streptomyces griseus. Bafilomycin A1 inhibits the short circuit current induced by the outer mantle epithelium (OME). The IC50 and maximum inhibition dose of Bafilomycin A1 are 0.17 μM and 0.5 μM, respectively. [2] In addition, Bilomycin A1 inhibits the acid influx with an IC50 value of 0.4 nM. Bafilomycin A1 inhibits the acidification dose-dependently resulting in a lower quenching, and thus a higher fluorescence. [3] Bafilomycin A1 prevents the vacuolization of Hela cells induced by H. pylori, with an inhibitory concentration giving 50% of maximal (ID50) of 4 nM. Bafilomycin A1 is also very efficient in restoring vacuolated cells to a normal appearance. [4] Bafilomycin A1 also affects the transport of endocytosed material from early to late endocytic compartments. Bafilomycin not only dissipates the low endosomal pH but also blocks transport from early to late endosomes in HeLa cells. [5] Bafilomycin A1 at doses of 0.1-1 μM completely inhibits the acidification of lysosomes revealed by the incubation with acridine orange in BNL CL.2 and A431 cells. [6] When Bafilomycin A1 is added to Hanks' balanced salt solution, endogenous protein degradation is strongly inhibited and

Chemical Structure



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	numerous autophagosomes accumulated in H-4-II-E cells. Bafilomycin A1 also prevents the appearance of endocytosed HRP in autophagic vacuoles. [7]
In vivo	Bafilomycin A1 (1 μM and 0.1 μM) completely inhibits the resorptive activity of cultured osteoclasts. [8] Bafilomycin A1 dose-dependently inhibits the rate of Na+ uptake in young tilapia with a Ki of 0.16 μM. [9]
FeaturesS1413	

Protocol *(Only for Reference)*

Kinase Assay: [2]

ATPase enzyme activity assays	The ATPase enzyme assay medium contains 6 mM $MgSO_4$, 50 mM HEPES (pH 7.4), 200 mM Na_2SO_3 (V-ATPase activator), 0.5 mM sodium ortho-vanadate (P-ATPase inhibitor), 0.5 mM sodium azide (F-ATPase inhibitor) and 3 mM Na_2ATP. This medium (1.0 mL), with or without the addition of the V-type ATPase inhibitor bafilomycin A1, is incubated with the filtered homogenate (0.1 mL) for 60 minutes at 23–25 °C. The reaction is stopped by the addition of 1 mL of TCA 3%. Spectrometric blanks are prepared as for the enzyme assay with the exception that the tissue sample is added after the acid. Phosphate analysis is accomplished by adding 2 mL of 1-butanol and 0.2 mL molybdate solution (5 g ammonium molybdate, 22 mL H_2SO_4 to 100 mL). After vortexing for 15 seconds the solution is neutralised with 0.5 mL citrate solution (100 g/500 mL, pH 7.0) and again vortexed for 15 seconds. The solution is then centrifuged (2000 × g; 3 minutes) to separate the butanol phase and the absorbance of this phase is read at 400 nm. Standards of orthophosphate are prepared (0.1 μM–2.0 μM) and treated in the same way as the enzyme activity assays. Enzyme activity is expressed in μmol of orthophosphate liberated per hour and per milligram of protein. V-ATPase activity is considered to be the difference between the total ATPase activity measured in the presence of Na_2SO_3, sodium orthovanadate and sodium azide and the ATPase activity measured in the presence of these reagents and of the specific V-ATPases inhibitor Bafilomycin A1.

Cell Assay: [4]

Cell lines	HeLa cells
Concentrations	~20 nM
Incubation Time	20 hours
Method	H. pylori bacteria extract is treated with inhibitors, before addition to HeLa cells, as follows: DCCD 10 mM for 1 hour at 30 ºC; NBD-Cl 100 μM for 1 hour at 30 ºC and the reaction is blocked with glycine 10 mM final concentration; NEM 275 μM for 1 hour at 30 ºC and the reaction is blocked by addition of β-mercaptoethanol 275 mM; Mg-ATP 14 μM for 1 hour at 0 ºC; 100 μM KNO_3 and 14 μM Mg-ATP for 1 hour at 30 μM; $NaCO_3$ 100 μM, pH 11 for 1 hour at 0 ºC. The bacterial extract is then added to cell with a 40-fold dilution at a final concentration of 0.65 mg/mL. Controls are HeLa cells incubated with untreated bacterial extracts and cells treated with inhibitor Bafilomycin A1 under the same conditions as bacterial extracts, at the same concentrations or after a 40-fold dilution. The vacuotating activity of the bacterial extracts is assayed.

Animal Study: [9]

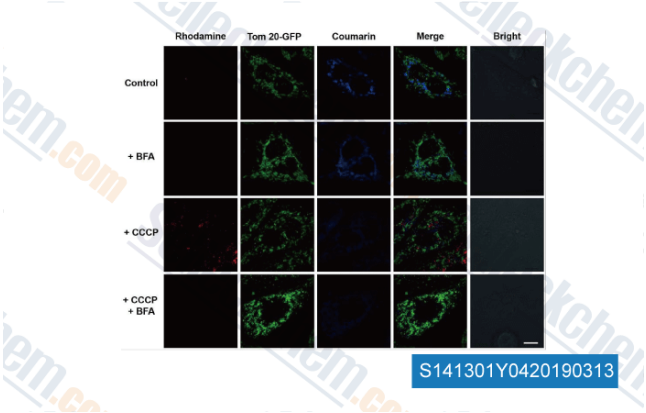
Animal Models	Young (approximately 9 days old) Mozambique tilapia, Oreochromis mossambicus
Dosages	10 μM
Administration	--

References:

[1] al-Fifi ZI, et al. Insect BiochemMol Biol, 1998, 28(4), 201-211.
[2] Oliveira PF, et al. Comp Biochem Physiol A Mol Integr Physiol, 2004, 139(4), 425-432.
[3] S?rensen MG, et al. J Bone Miner Res, 2007, 22(10), 1640-1648.
[4] Papini E, et al. Mol Microbiol, 1993, 7(2), 323-327.
[5] Bayer N, et al. J Virol, 1998, 72(12), 9645-9655.
[6] Yoshimori T, et al. J Biol Chem, 1991, 266(26), 17707-17712.
[7] Yamamoto A, et al. Cell Struct Funct, 1998, 23(1), 33-42.

[8] Sundquist KT, et al. J Bone Miner Res, 1994, 9(10), 1575-1582.
[9] Fenwick JC, et al. J Exp Biol, 1999, 202 Pt 24, 3659-3666.
[10] Kanzawa T, et al. Cell Death Differ, 2004, 11(4), 448-457.

Customer Product Validation



Data from [Data independently produced by , , Chem Sci, 2017, 8(3):1915-1921]
Mitochondrial depolarization mediated relocation of mitochondrial RC-TPP into lysosomes and the ensuing lysosomal acidity triggered rhodamine fluorescence. RC-TPP-loaded Tom20-GFP+ HeLa cells were treated without or with CCCP (20 μM) in the presence or absence of BFA (200 nM). Colocalization of Tom20-GFP (in green) and mitochondrial RC-TPP (in blue) is shown in cyan. Scale bar: 10 μm.

Data from [Data independently produced by , , Cancer Lett, 2018, 431:85-95]
A549 cells and SK-1 cells co-treated with Baf A1 (100 nM) and SFN-NAC (30 μM) for 24 h, the expression of LC3 I and LC3 II was determined by Western blot.

Bafilomycin A1(Baf-A1) has been referenced in publications.

Pharmacological targeting of MCL-1 promotes mitophagy and improves disease pathologies in an Alzheimer's disease mouse model [Nat Commun, 2020, 11(1):5731]	PubMed: 33184293
Inhibition of Vps34 Reprograms Cold Into Hot Inflamed Tumors and Improves anti-PD-1/PD-L1 Immunotherapy [Sci Adv, 2020, 29;6(18):eaax7881]	PubMed: 32494661
Crosstalk between HSPA5 arginylation and sequential ubiquitination leads to AKT degradation through autophagy flux. [Autophagy, 2020, 10.1080/15548627.2020.1740529]	PubMed: 32164484
Stabilization of MORC2 by Estrogen and Antiestrogens Through GPER1- PRKACA-CMA Pathway Contributes to Estrogen-Induced Proliferation and Endocrine Resistance of Breast Cancer Cells [Autophagy, 2020, 16(6):1061-1076]	PubMed: 32401166
ATF4 links ER stress with reticulophagy in glioblastoma cells [Autophagy, 2020, 1-17]	PubMed: 33111629
Oncogenic role of MIR516A in human bladder cancer was mediated by its attenuating PHLPP2 expression and BECN1-dependent autophagy. [Autophagy, 2020, 1:1-15]	PubMed: 32116109
The Dynein Adaptor RILP Controls Neuronal Autophagosome Biogenesis, Transport, and Clearance. [Dev Cell, 2020, 20;53(2):141-153 e4]	PubMed: 32275887
Auto-ubiquitination of NEDD4-1 Recruits USP13 to Facilitate Autophagy through Deubiquitinating VPS34. [Cell Rep, 2020, 25;30(8):2807-2819 e4]	PubMed: 32101753
LSD1 contributes to programmed oocyte death by regulating the transcription of autophagy adaptor SQSTM1/p62. [Aging Cell, 2020, 19(3):e13102]	PubMed: 32074399
Differential Expression of MAGEA6 Toggles Autophagy to Promote Pancreatic Cancer Progression [Elife, 2020, 9;9:e48963]	PubMed: 32270762

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