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Print

3-Methyladenine (3-MA)

Technical Data

Molecular Weight	149.15	**Storage**	3 years -20°C powder 2 years -80°C in solvent
Formula	$C_6H_7N_5$		
CAS No.	5142-23-4	**Synonyms**	NSC 66389
Chemical Name	3-methyl-3H-purin-6-amine		
Solubility (25°C) *	In vitro	Water	30 mg/mL **warmed with 50ºC water bath** (201.13 mM)
		DMSO	7 mg/mL **warmed with 50ºC water bath** (46.93 mM)
		Ethanol	4 mg/mL (26.81 mM)
	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.

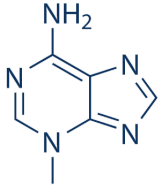
Preparing Stock Solutions

Volume / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	6.7047 mL	33.5233 mL	67.0466 mL
5 mM	1.3409 mL	6.7047 mL	13.4093 mL
10 mM	0.6705 mL	3.3523 mL	6.7047 mL
50 mM	0.1341 mL	0.6705 mL	1.3409 mL

Biological Activity

Description	3-Methyladenine (3-MA, NSC 66389) is a selective PI3K inhibitor for **Vps34** and **PI3Kγ** with **IC50** of 25 μM and 60 μM in HeLa cells; blocks class I PI3K consistently, whereas suppression of class III PI3K is transient, and also blocks **autophagosome** formation. 3-Methyladenine (3-MA) is successfully used to suppress **mitophagy**. **Solutions of 3-MA are best fresh-prepared by heating.**
Targets	Vps34 [1] (HeLa cells) / PI3Kγ [1] (HeLa cells)

Chemical Structure



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	25 μM	60 μM
In vitro	The slight preference for Vps34 prevention by 3-Methyladenine probably arises from a hydrophobic ring specific to Vps34, which encircles the 3-methyl group of 3-Methyladenine. [1] 3-Methyladenine has been reported to cause cancer cell death under both normal and starvation conditions. 3-Methyladenine could also suppress cell migration and invasion independently of its ability to inhibit autophagy, implying that 3-Methyladenine possesses functions other than autophagy suppression. 3-Methyladenine elicits caspase-dependent cell death that is independent of autophagy inhibition. Treatment with 5 mM 3-Methyladenine reduces the percentage of glucose-starved HeLa cells displaying GFP-LC3 puncta to 23%. The levels of LC3-I are increasing and the levels of LC3-II are decreasing between 12 and 48 hours in cells that are treated with 3-Methyladenine. Conversion of LC3-I to LC3-II is suppressed by 3-Methyladenine. Treatment of HeLa cells with 3-Methyladenine at 2.5 mM or 5 mM for one day does not affect cell viability, whereas treatment with 10 mM 3-Methyladenine for one day causes a 25.0% decrease in cell viability. Treatment of cells with 2.5, 5 or 10 mM 3-Methyladenine for two days causes 11.5%, 38.0% and 79.4% decrease in viability, respectively. 3-Methyladenine decreases cell viability in a time- and dose-dependent manner. 3-Methyladenine significantly shortens the duration of nocodazole-induced-prometaphase arrest. [2] Suppression of autophagy by 3-Methyladenine inhibits SU11274-induced cell death. [3] Prolonged treatment with 3-Methyladenine (up to 9 hours) induces significant LC3 I to II conversion in wild type MEFs. Prolonged treatment with 3-Methyladenine, but not wortmannin, markedly increases GFP-LC3 punctuation/aggregation. 3-Methyladenine-induced LC3 conversion and free GFP liberation are ATG7-dependent. 3-Methyladenine treatment leads to evident increase of p62 protein level. 3-Methyladenine increases the p62 level even in Atg5−/− MEFs as well as in cells with DOX-mediated deletion of ATG5. 3-Methyladenine inhibits class I and class III PI3K in different temporal patterns. 3-Methyladenine-induced LC3 I to LC3 II conversion is dramatically compromised in Tsc2−/− cells compared with wild type cells.3-Methyladenine disrupts the anti-autophagic function of mTOR complex 1. [4]	
In vivo	3-Methyladenine blocks autophagy through its effect on class III phosphatidylinositol 3-kinase (PI3K). 3-Methyladenine treatment does not alter the degree of hemorrhage compared with the subarachnoid hemorrhage (SAH) group. 3-Methyladenine pretreatment significantly aggravates neurological symptoms when compared with the SAH + vehicle group. Autophagy is decreased when 3-Methyladenine treatment is applied. Conversely, cleaved caspase-3 is markedly up-regulated in the SAH + 3-Methyladenine group. In line with the up-regulation of cleaved caspase-3 expression, the number of TUNEL-positive cells in the right cortex is significantly increased in the SAH + 3-Methyladenine group compared with the SAH + vehicle group. [5]	
FeaturesS2767		

Protocol *(Only for Reference)*

Kinase Assay: [4]

Protein degradation assay	HeLa cells are radiolabeled for 24 hours with 0.05 mCi/mL l-[U-^{14}C]valine. At the end of the labeling period, cells are rinsed three times with PBS. Cells are incubated for the designated times in either full medium or EBSS with or without the presence of 10 mM 3-Methyladenine.

Cell Assay: [2]

Cell lines	HeLa cell line
Concentrations	1-10 mM
Incubation Time	24, 48 or 72 hours
Method	Cell (such as HeLa cell) viability is determined by a trypan blue exclusion assay. Briefly, after treated with 3-Methyladenine, both adherent and floating cells are collected and suspended in phosphate buffered saline (PBS, pH 7.4) at a final density of 1-2 × 10^6/mL. An equal volume of 0.4% trypan blue solution (w/v, in PBS) is added to the cell suspension and mixed thoroughly. After incubation at room temperature for 3 min, cell counting is performed using a hemacytometer.

Animal Study: [5]

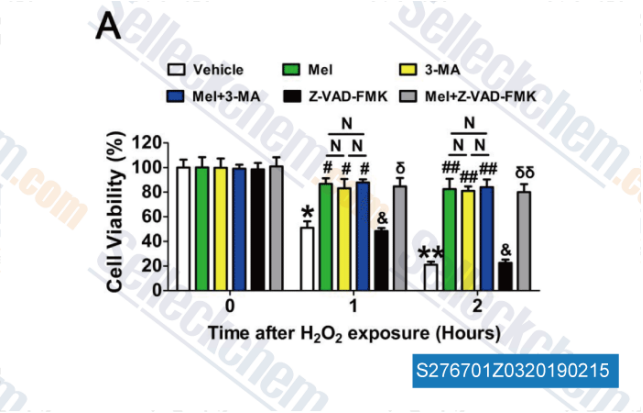
Animal Models	Adult male Sprague–Dawley rats weighing 300-350 g

Dosages	400 nM
Administration	Intracerebral ventricular

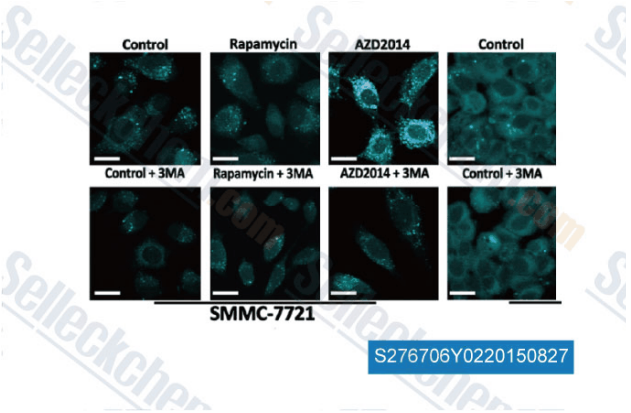
References:

[1] Miller S, et al. Autophagy. 2010, 6(6), 805-807.
[2] Hou H, et al. PLoS One. 2012, 7(4), e35665.
[3] Liu Y, et al. J Pharmacol Sci. 2012, 118(4), 423-432.
[4] Wu YT, et al. J Biol Chem. 2010, 285(14), 10850-10861.
[5] Jing CH, et al. Neuroscience. 2012.

Customer Product Validation



Data from [Data independently produced by , , Redox Biol, 2018, 18:138-157]
granulosa cells (GCs) with 24 h of melatonin (10 μM) treatment were rinsed in PBS, and then exposed to H2O2 (200 μM) for 2 h. The autophagy inhibitor 3-MA (10 mM), or the apoptosis inhibitor Z-VAD-FMK (50 μM) were added 1 h prior to H2O2 incubation. Cell viability was determined using the CCK-8 assay. Data represent mean ± S.E; n = 3 in each group. *P < 0.05 (**P < 0.01) vs. vehicle group at 0 h. # Represents P < 0.05 (## Represents P < 0.01) vs. H2O2-only-treated cells. & Represents P > 0.05 vs. H2O2-only-treated cells. N, not significant, P > 0.05. δ Represents P < 0.05 (δδ Represents P < 0.01) vs. Z-VAD-FMK-treated cells.



Data from [Data independently produced by , , Am J Cancer Res, 2015, 5(1): 125-139]
MDC-labeled vacuoles were induced by AZD2014 and inhibited by autophagy inhibitor (3-MA). SMMC-7721 cells were treated with AZD2014 or rapamycin at concentrations of 100 and 600 nM, respectively, for 48 hours in the presence or absence of 3-MA, and then stained with MDC. Cells were immediately observed under a confocal microscope. Cells in the control group were treated with DMSO. bars, 20 μm.

3-Methyladenine (3-MA) has been referenced in publications.

Irradiated Tumor Cell-Derived Microparticles Mediate Tumor Eradication via Cell Killing and Immune Reprogramming [Sci Adv, 2020, 25;6(13):eaay9789]	PubMed: 32232155
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
The role of the key autophagy kinase ULK1 in hepatocellular carcinoma and its validation as a treatment target. [Autophagy, 2020, 10.1080/15548627.2019.1709762]	PubMed: 31986961
1-phenyl 2-thiourea (PTU) activates autophagy in zebrafish embryos. [Autophagy, 2020, 22:1-10]	PubMed: 32286915
Autophagy triggers CTSD (cathepsin D) maturation and localization inside cells to promote apoptosis. [Autophagy, 2020, 23:1-23]	PubMed: 32324083
HIF-1α-Mediated Mitophagy Determines ZnO Nanoparticle-Induced Human Osteosarcoma Cell Death both In Vitro and In Vivo [ACS Appl Mater Interfaces, 2020, 12(43):48296-48309]	PubMed: 33054172

Ultrafast Low-Temperature Photothermal Therapy Activates Autophagy and Recovers Immunity for Efficient Antitumor Treatment. [ACS Appl Mater Interfaces, 2020, 29;12(4):4265-4275] PubMed: 31903741

KPNB1-mediated nuclear translocation of PD-L1 promotes non-small cell lung cancer cell proliferation via the Gas6/MerTK signaling pathway [Cell Death Differ, 2020, 10.1038/s41418-020-00651-5] PubMed: 33139930

LSD1 contributes to programmed oocyte death by regulating the transcription of autophagy adaptor SQSTM1/p62. [Aging Cell, 2020, 19(3):e13102] PubMed: 32074399

Zinc oxide nanoparticles effectively regulate autophagic cell death by activating autophagosome formation and interfering with their maturation [Part Fibre Toxicol, 2020, 17(1):46] PubMed: 32948194

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