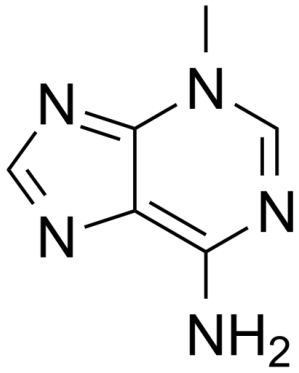
Data Sheet

Product Name:	3-Methyladenine
Cat. No.:	CS-5207
CAS No.:	5142-23-4
Molecular Formula:	C6H7N5
Molecular Weight:	149.15
Target:	Autophagy; Endogenous Metabolite; Mitophagy; PI3K
Pathway:	Autophagy; Metabolic Enzyme/Protease; PI3K/Akt/mTOR
Solubility:	H2O : 2 mg/mL (13.41 mM; Need ultrasonic); DMSO : 8.33 mg/mL (55.85 mM; Need ultrasonic)



BIOLOGICAL ACTIVITY:

3-Methyladenine is a **PI3K** inhibitor. 3-Methyladenine is a widely used inhibitor of **autophagy** via its inhibitory effect on class III PI3K. IC50 & Target: IC50: 25 μM (Vps34), 60 μM (PI3Kγ)[1] **In Vitro:** 3-Methyladenine (0-10 mM; 0-48 hours) induces caspase-dependent cell death in HeLa cells in a time-and dose-dependent manner[2].

3-Methyladenine (5 mM; 24 hours) suppresses autophagy in HeLa cells under both glucose-free conditions and normal conditions[2].

3-Methyladenine (5 mM; 0-48 hours) suppresses conversion of LC3-I to LC3-II (autophagy markers) between 12hours and 48 hours, confirms the inhibitory effects on autophagy[2].

3-Methyladenine induces cell death is independent of autophagy inhibition[2].

3-Methyladenine significantly shortens the duration of nocodazole-induced-prometaphase arrest[2].

In Vivo: 3-Methyladenine (1.5 mg/100 g; intraperitoneal injection; 3-24 hours) treatment alleviates sodium taurocholate-induced severe acute pancreatitis (SAP) in rats at both 12 hours and 24 hours[3].

3-Methyladenine inhibits autophagy of pancreatic acinar cells in sodium taurocholate-tnduced SAP[3].

3-Methyladenine also shows inhibitory effects on PI3K/Akt signaling pathway and NF-κB signaling pathway in sodium taurocholate-induced SAP[3].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: 3-Methyladenine is directly dissolved into the culture medium.[2]Cell viability is determined by a trypan blue exclusion assay. Cells are cultured in the medium 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 Administration:** [3]All rats are fasted for 12 h with free access to water prior to operation. After anesthesia by intraperitoneal (i.p.) injection of 2% sodium pentobarbital (0.25 mL/100 g), they are laid and fixed on the table, routinely shaven, disinfected, and draped. The rat SAP model is induced by 0.1 mL/min speed uniformly retrograde infusion of a freshly prepared 3.5% sodium taurocholate solution (0.1 mL/100 g) into the biliopancreatic duct after laparotomy. Equivalent volume of normal saline solution is substituted for 3.5% sodium taurocholate solution in the sham-operation (SO) control group. The incision is closed with a continuous 3-0-silk suture, and 2 mL/100 g of saline is injected into the back subcutaneously to compensate for the fluid loss. 180 rats are randomly divided into four groups: (1) Acanthopanax treatment group (Aca group, n = 45) where the rats are injected with 0.2% Acanthopanax injection at a dose of 3.5 mg/100 g 3 h after successful modeling via the vena caudalis once, knowing that this dosage is effective; (2) 3-Methyladenine treatment group (3-methyladenine group, n = 45) where the rats are injected with 100 nmol/μL 3-methyladenine solution at a dose of 1.5 mg/100 g 3 h after successful modeling via the intraperitoneal route once, knowing that this dosage is effective; (3) SAP model group (SAP group, n = 45) where these rats receive an equivalent volume of the normal saline instead of Acanthopanax injection 3 h after successful modeling via the vena caudalis once; (4) SO group (control, n = 45) where these

rats receive an equivalent volume of the normal saline instead of Acanthopanax injection 3 h after successful sham-operation via the vena caudalis once. The 45 animals in each of the four groups are equally randomized into 3, 12, and 24 h subgroups for postoperative observations[3].

References:

[1]. Miller S, et al. Finding a fitting shoe for Cinderella: searching for an autophagy inhibitor. Autophagy. 2010 Aug;6(6):805-7.

[2]. Hou H, et al. Inhibitors of phosphatidylinositol 3'-kinases promote mitotic cell death in HeLa cells. PLoS One. 2012;7(4):e35665.

[3]. Wang X, et al. Acanthopanax versus 3-Methyladenine Ameliorates Sodium Taurocholate-Induced Severe Acute Pancreatitis by Inhibiting the Autophagic Pathway in Rats.Mediators Inflamm. 2016;2016:8369704.

CAIndexNames:

3H-Purin-6-amine, 3-methyl-

SMILES:

NC1=C2N=CN=C2N(C)C=N1

Tel: 732-484-9848 Fax: 888-484-5008 E-mail: sales@ChemScene.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA