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Zuschläge

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SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

F. +43(0)1 489 3961-7

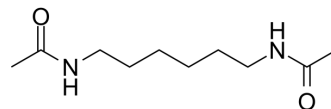
mail@szabo-scandic.com

www.szabo-scandic.com

[linkedin.com/company/szaboscandic](https://www.linkedin.com/company/szaboscandic) 

Hexamethylene bisacetamide

Cat. No.:	HY-124284
CAS No.:	3073-59-4
Molecular Formula:	C ₁₀ H ₂₀ N ₂ O ₂
Molecular Weight:	200.28
Target:	Apoptosis; p38 MAPK; Akt; NF-κB; Notch; Bcl-2 Family; MDM-2/p53; Epigenetic Reader Domain
Pathway:	Apoptosis; MAPK/ERK Pathway; PI3K/Akt/mTOR; NF-κB; Neuronal Signaling; Stem Cell/Wnt; Epigenetics
Storage:	4°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro	H ₂ O : 100 mg/mL (499.30 mM; Need ultrasonic)					
	DMSO : 25 mg/mL (124.83 mM; ultrasonic and warming and heat to 60°C)					
	Preparing Stock Solutions	Solvent Concentration	Mass	1 mg	5 mg	10 mg
		1 mM		4.9930 mL	24.9650 mL	49.9301 mL
		5 mM		0.9986 mL	4.9930 mL	9.9860 mL
10 mM			0.4993 mL	2.4965 mL	4.9930 mL	
Please refer to the solubility information to select the appropriate solvent.						
In Vivo	1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: ≥ 2.5 mg/mL (12.48 mM); Clear solution					
	2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.5 mg/mL (12.48 mM); Clear solution					
	3. Add each solvent one by one: 10% DMSO >> 90% corn oil					
	Solubility: ≥ 2.5 mg/mL (12.48 mM); Clear solution					

BIOLOGICAL ACTIVITY

Description	Hexamethylene bisacetamide (HMBA) is a differentiation inducer and selective bromine domain inhibitor that can differentiate across the blood-brain barrier. Hexamethylene bisacetamide can induce tumor cell differentiation and inhibit cell proliferation, showing antitumor activity. Hexamethylene bisacetamide induces apoptosis by Notch1, Bcl-2 and p53 signaling pathways. In addition, Hexamethylene bisacetamide improves the obesity phenotype of mice ^{[1][2][3][4][5]} .
In Vitro	Hexamethylene bisacetamide (2.5-10 mM; 15 days) significantly inhibits the proliferation of SHG-44 cells in a dose-

dependent manner, but the inhibitory effect is reversible^[2].

Hexamethylene bisacetamide (5-10 mM; 15 days) changes the morphology of SHG-44 cells and increases the degree of cell differentiation^[2].

Hexamethylene bisacetamide (1-20 mM; 30-480 minutes) inhibits the activation of MAPK, Akt signaling pathways and NF - κ B in TNF α -treated A549 cells^[3].

Hexamethylene bisacetamide (5 mM; 2 h-7 days) induces apoptosis through Notch1, Bcl - 2 and p53 signaling pathways in Molt4 cells^[4].

Hexamethylene bisacetamide (0.1 mM; 2 h) regulates the expression of neuropeptides through MYH9 and ACTG1 in hypothalamic cells^[5].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay^[2]

Cell Line:	Human malignant glioma cell line SHG-44
Concentration:	2.5, 5, 7.5 and 10 mM
Incubation Time:	15 days
Result:	Reduced cell proliferation compared with the control group.

Western Blot Analysis^[3]

Cell Line:	A549 cells
Concentration:	10 mM
Incubation Time:	30, 60, 120 and 240 min
Result:	Inhibited the phosphorylation levels of p44/p42 MAPK and MEK1/2. Inhibited the phosphorylation levels of Akt.

Western Blot Analysis^[4]

Cell Line:	Molt4 cells
Concentration:	5 mM
Incubation Time:	2 h, 4 h, 8 h, 24 h, 48 h, 5 days and 7 days
Result:	Reduced the levels of Notch1.

In Vivo

Hexamethylene bisacetamide (1000 mg/kg intraperitoneal injection; 500-1000 mg/kg intravenous injection; 400 nmoles 2 μ L intracerebroventricular injection; 7 days) has an improved effect in mouse obesity model^[5].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Mice with diet-induced obese (DIO) model ^[5]
Dosage:	1,000 mg/kg (i.p.); 500 and 1,000 mg/kg (i.v.); 400 nmoles 2 μ L (ICV)
Administration:	Intraperitoneal injection (i.p.); 7 days Intravenous injection (i.v.); 7 days Intracerebroventricular injection (ICV); 7 days
Result:	Ameliorated obese phenotype in DIO mice, reduced weight gain and food intake and

	improved metabolic parameters in whatever injection way. Did not cause sickness behaviors in DIO mice in whatever injection way. Regulate hypothalamic neuropeptide expression in whatever injection way.
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REFERENCES

- [1]. Li XN, et al. Modulation effects of hexamethylene bisacetamide on growth and differentiation of cultured human malignant glioma cells. J Neurosurg. 1996 May;84(5):831-8.
- [2]. Dey A, et al. Hexamethylene bisacetamide (HMBA) simultaneously targets AKT and MAPK pathway and represses NF kappaB activity: implications for cancer therapy. Cell Cycle. 2008 Dec;7(23):3759-67.
- [3]. Cecchinato V, et al. Hexamethylene bisacetamide inhibits malignant phenotype in T-ALL cell lines. Leuk Res. 2008 May;32(5):791-7.
<https://pubmed.ncbi.nlm.nih.gov/17964649/>
- [4]. Nilsson LM, et al. Muralidharan SV, Demir D, Welin M, Bhadury J, Logan DT, Walse B, Nilsson JA. Cancer Differentiating Agent Hexamethylene Bisacetamide Inhibits BET Bromodomain Proteins. Cancer Res. 2016 Apr 15;76(8):2376-83.
- [5]. Park S, et al. HMBA ameliorates obesity by MYH9- and ACTG1-dependent regulation of hypothalamic neuropeptides. EMBO Mol Med. 2023 Dec 7;15(12):e18024.

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

Tel: 609-228-6898

Fax: 609-228-5909

E-mail: tech@MedChemExpress.com

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