Product Data Sheet

Product Name: Apamin
Cat. No.: GC14893

Chemical Properties

Cas. No.	24345-16-2		
Formula	$C_{79}H_{131}N_{31}O_{24}S_4$	M.Wt	2027.34
Solubility	Water: 1 mg/ml	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ 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	Apamin		

Protocol

Cell experiment [1]:

Cell lines	SH-SY5Y cells (human dopaminergic neuroblastoma cell line)
Preparation Method	SH-SY5Y cells were cultured in Dulbecco’s Modified Eagle’s Medium (DMEM) supplemented with 10% fetal bovine serum (FBS). Cells were treated with Apamin (0.5μg/mL) for 1 hour, followed by exposure to MPP^+ (3mM) for 12–24 hours.
Reaction Conditions	0.5μg/mL; 1h pretreatment

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA

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Applications	Apamin significantly attenuated MPP^+-induced cytotoxicity by restoring mitochondrial membrane potential and reducing apoptotic markers. Apamin suppressed calcium overload and downregulated SK2 channel (KCa2.2) expression, inhibiting CaMKII phosphorylation. Apamin also mitigated oxidative stress and endoplasmic reticulum stress, while blocking pro-inflammatory cytokine production via ERK/NF-κB/STAT3 pathway inhibition.

Animal experiment [2]:

Animal models	C57BL/6N male mice
Preparation Method	Mice were intraperitoneally injected with a single dose of lipopolysaccharide (LPS; 10mg/kg). Apamin (0.1mg/kg) was administered intraperitoneally 1 hour after LPS injection. Mice were sacrificed 24 hours post-LPS injection for renal and plasma analysis.
Dosage form	0.1mg/kg; i.p.; Single injection 1 hour post-LPS.

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Applications	Apamin significantly ameliorated LPS-induced acute kidney injury, reducing plasma creatinine and blood urea nitrogen (BUN) levels. Apamin attenuated renal tubular injury, including brush border loss and suppressed expression of injury markers. Apamin inhibited oxidative stress by downregulating NOX4 and enhancing HO-1 expression, reduced lipid peroxidation (4-HNE, MDA), and restored GSH/GSSG ratio. Apamin suppressed apoptosis and inhibited inflammation by reducing TNF-α and IL-6 levels, downregulating TLR4/NF-κB signaling, and attenuating immune cell infiltration and vascular adhesion molecule.

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References:
[1] Park J, Jang KM, Park KK. Effects of Apamin on MPP+-Induced Calcium Overload and Neurotoxicity by Targeting CaMKII/ERK/p65/STAT3 Signaling Pathways in Dopaminergic Neuronal Cells. Int J Mol Sci. 2022 Dec 3;23(23):15255.
[2] Kim JY, Leem J, Park KK. Antioxidative, Antiapoptotic, and Anti-Inflammatory Effects of Apamin in a Murine Model of Lipopolysaccharide-Induced Acute Kidney Injury. Molecules. 2020 Dec 3;25(23):5717.

Background

Apamin is a polypeptide neurotoxin derived from bee (Apis mellifera) venom[1-2]. Apamin is a highly selective small-conductance calcium-activated potassium channel (SK channel, especially the SK2 subtype; IC_{50}=0.06–0.4nM) blocker. Apamin enhances neuronal excitability and synaptic plasticity by preventing the hyperpolarization phase following neuronal action potentials, thereby influencing physiological functions such as learning and memory[3-4].

In vitro, Apamin (0.1μM) treatment of differentiated N1E 115 neuroblastoma cells for 2 minutes selectively blocks Ca^{2+}-dependent K^+ channels, increasing neuronal

excitability[5]. In SH-SY5Y human neuroblastoma cells pretreated with Apamin (0.5μg/mL) for 1 hour, followed by stimulation with 1-methyl-4-phenyl pyridinium (MPP^+; 3mM) for 12–24 hours, Apamin significantly inhibits the downregulation of tyrosine hydroxylase (TH) expression and abnormal aggregation of α-synuclein (αSYN), while reducing mitochondrial membrane potential disruption and apoptosis by blocking SK2 channel-mediated calcium overload[6].

In vivo, in a cholestatic liver fibrosis model induced by 3,5-Diethoxycarbonyl-1,4-Dihydrocollidine (DDC) diet, intraperitoneal injection of Apamin (0.1mg/kg) twice weekly for 4 weeks in 8-week-old C57BL/6 male mice significantly alleviates DDC-induced liver tissue damage and collagen deposition[7]. In a lipopolysaccharide (LPS; 10mg/kg)-induced acute kidney injury model, a single intraperitoneal injection of Apamin (0.1mg/kg) in 8-week-old C57BL/6N male mice significantly improves LPS-induced renal dysfunction and kidney tissue damage[8].

References:
[1] Gu H, Han SM, Park KK. Therapeutic Effects of Apamin as a Bee Venom Component for Non-Neoplastic Disease. Toxins (Basel). 2020 Mar 19;12(3):195.
[2] Laurindo LF, de Lima EP, Laurindo LF, et al. The therapeutic potential of bee venom-derived Apamin and Melittin conjugates in cancer treatment: A systematic review. Pharmacol Res. 2024 Nov;209:107430.
[3] Strong PN, Stocker M, Jenkinson DH. Apamin binding proteins and oligosaccharyltransferases. Toxicon. 1996 May;34(5):507-9.
[4] Lazdunski M, Fosset M, Hughes M, et al. The apamin-sensitive Ca2+-dependent K+ channel molecular properties, differentiation and endogenous ligands in mammalian brain. Biochem Soc Symp. 1985;50:31-42.
[5] Hugues M, Romey G, Duval D, et al. Apamin as a selective blocker of the calcium-dependent potassium channel in neuroblastoma cells: voltage-clamp and biochemical characterization of the toxin receptor. Proc Natl Acad Sci U S A. 1982 Feb;79(4):1308-12.
[6] Park J, Jang KM, Park KK. Effects of Apamin on MPP+-Induced Calcium Overload and Neurotoxicity by Targeting CaMKII/ERK/p65/STAT3 Signaling Pathways in Dopaminergic Neuronal Cells. Int J Mol Sci. 2022 Dec 3;23(23):15255.
[7] Kim JY, An HJ, Kim WH, et al. Apamin suppresses biliary fibrosis and activation of hepatic stellate cells. Int J Mol Med. 2017 May;39(5):1188-1194.
[8] Kim JY, Leem J, Park KK. Antioxidative, Antiapoptotic, and Anti-Inflammatory Effects

of Apamin in a Murine Model of Lipopolysaccharide-Induced Acute Kidney Injury. Molecules. 2020 Dec 3;25(23):5717.

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