PrP (106-126)

Cat. No.:	HY-P0305
CAS No.:	148439-49-0
Molecular Formula:	$C_{80}H_{138}N_{26}O_{24}S_2$
Molecular Weight:	1912.3
Sequence:	Lys-Thr-Asn-Met-Lys-His-Met-Ala-Gly-Ala-Ala-Ala-Ala-Gly-Ala-Val-Val-Gly-Gly-Leu-Gly
Sequence Shortening:	KTNMKHMAGAAAAGAVVGGLG
Target:	Others
Pathway:	Others
Storage:	Sealed storage, away from moisture Powder -80°C 2 years -20°C 1 year * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)

KTNMKHMAGAAAAGAVVGGLG

SOLVENT & SOLUBILITY

In Vitro	H_2O : 1 mg/mL (0.52 mM; Need ultrasonic and warming)
In Vivo	1. Add each solvent one by one: PBS Solubility: 100 mg/mL (52.29 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description	PrP (106-126) is a peptide corresponding to the prion protein (PrP) amyloidogenic region, and its biochemical properties resemble the infectious form of prion protein.
In Vitro	PrP (106-126) (100 μM) induces mTOR phosphorylation over time in N2a cells. PrP (106-126)-treated cells show significantly increased ROS production in comparison with that of PBS-treated control cells. Knockdown of PRAS40 enhances PrP (106-126)-induced apoptosis. PRAS40 alleviates PrP (106-126)-induced neuronal apoptosis via mTOR-AKT activation[1]. PrP (106-126) interacts selectively with porcine brain endothelial cells (PBEC) via their luminal side, and causes cumulative cell death, as shown by lactate dehydrogenase release, 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide reduction, Caspase 3 induction and direct cell counting. In addition, PrP (106-126), but not its corresponding scrambled peptide, produces a 50% reduction of the trans-endothelial electrical resistance, while the PBEC maintained confluency[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	The toxicity of PrP peptides is investigated on monolayers of PBEC. 100 000 PBEC/cm^2 are seeded on 96 [for 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT)] or 24-well plates [for lactate dehydrogenase (LDH)] pre-coated with rat tail collagen (27 μg/mL). The cell monolayers are treated with either PrP 106-126 wt or scr peptides. The PrP peptides are dissolved in double distilled water and then diluted in assay medium or in 5% newborn calf serum in Dulbecco-

modified Earl's medium without phenol red. Treatment duration with the PrP peptides varies from 24 h to 48 h and the peptide concentration is set to 100 μM at which it exerts optimal effects as shown in a dose-dependent experiment. This concentration is also chosen as similar concentrations of PrP 106-126 found to exert in-vitro effects on astrocytes, neurons, microglia and leukocytes. The MTT assay, which measures mitochondrial metabolism, is based on the conversion of the water-soluble MTT dye [3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide] to an insoluble purple formazan. This formazan is then solubilized in pure dimethylsulfoxide, and its concentration determined by optical density (550/630 nm). The LDH cell death assay is done using the CytoTox 96® Assay kit. The released LDH in culture supernatants is measured with a 30-min coupled enzymatic assay which results in the conversion of a tetrazolium salt, 2-p-iodophenyl-3-p-nitro-phenyl-5-phenyl tetrazolium chloride (INT) into a red formazan product. The LDH levels are measured at the end of each treatment period. Thus, 50 μL of the medium is removed at the end of the treatment with PrP peptides and transferred to a new 96-well plate to which the other compounds are added. The plates are analyzed using an Elisa reader with the 490 nm filter.

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REFERENCES

[1]. Yang W, et al. PRAS40 alleviates neurotoxic prion peptide-induced apoptosis via mTOR-AKT signaling. CNS Neurosci Ther. 2017 May;23(5):416-427.

[2]. Cooper I, et al. Interactions of the prion peptide (PrP 106-126) with brain capillary endothelial cells: coordinated cell killing and remodeling of intercellular junctions. J Neurochem. 2011 Feb;116(4):467-75.

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