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## Product Data Sheet

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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.

### Background

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.

PrP (106-126) (100  $\mu$ M) 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<sup>[1]</sup>. 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<sup>[2]</sup>.

### 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.

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

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