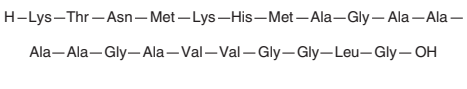
PRODUCT INFORMATION

Prion Protein (106-126) (trifluoroacetate salt)

Item No. 24556

Formal Name: L-lysyl-L-threonyl-L-asparaginyl-L-methionyl-L-lysyl-L-histidyl-L-methionyl-L-alanylglycyl-L-alanyl-L-alanyl-L-alanyl-L-alanylglycyl-L-alanyl-L-valyl-L-valylglycylglycyl-L-leucyl-glycine, trifluoroacetate salt



Synonym: PrP

MF: $C_{80}H_{138}N_{26}O_{24}S_2 \bullet XCF_3COOH$

FW: 1,912.3

Purity: ≥95%

Supplied as: A lyophilized powder

Storage: -20°C

Stability: ≥4 years

Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

Prion protein (106-126) (trifluoroacetate salt) is supplied as a lyophilized powder. A stock solution may be made by dissolving the prion protein (106-126) (trifluoroacetate salt) in water. The solubility of prion protein (106-126) (trifluoroacetate salt) in water is approximately1 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

Prion protein (106-126) is a peptide fragment of the cellular prion protein PrP^c.[1] It forms fibrils that are organized into β-strands, a behavior characteristic of amyloids. It is toxic to primary rat hippocampal neurons at a concentration of 80 µM following incubation for 10 days and induces DNA fragmentation and apoptosis. Prion Protein (106-126) (25 µM) activates ERK1 and 2, p38, and JNK1 and 2 kinases, induces apoptosis, increases NADPH oxidase-dependent production of reactive oxygen species (ROS), and decreases glutathione (GSH) levels in $1C11^{5\text{-HT}}$ serotonergic and $1C11^{NE}$ noradrenergic differentiated neuronal cells.[2] It forms channels in lipid bilayer membranes that are freely permeable to calcium, sodium, potassium, lithium, rubidium, cesium, and chloride ions at a concentration of 20 µM, and channel formation is significantly enhanced by aging and low pH.[3] *In vivo*, intraocular injection of prion protein (106-126) (1 mM) increases the number of terminal deoxynucleotidyltransferase-mediated dUTP nick end labeling (TUNEL) positive nuclei in mice 4 days post injection.[4]

References

1. Forloni, G., Angeretti, N., Chiesa, R., *et al.* Neurotoxicity of a prion protein fragment. *Nature* **362(6420)**, 543-546 (1993).
2. Pietri, M., Caprini, A., Mouillet-Richard, S., *et al.* Overstimulation of PrP^C signaling pathways by prion peptide 106-126 causes oxidative injury of bioaminergic neuronal cells. *J. Biol. Chem.* **281(38)**, 28470-28479 (2006).
3. Lin, M.-C., Mirzabekov, T., and Kagan, B.L. Channel formation by a neurotoxic prion protein fragment. *J. Biol. Chem.* **272(1)**, 44-47 (1997).
4. Bergström, A.-L., Cordes, H., Zsurger, N., *et al.* Amidation and structure relaxation abolish the neurotoxicity of the prion peptide PrP106-126 *in vivo* and *in vitro*. *J. Biol. Chem.* **280(24)**, 23114-23121 (2005).

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