

Amyloid Beta Protein

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Human Synthetic Amyloid Beta Peptide 1-42 (HFIP treated)
Catalog No. SPR-485

distributed in the US/Canada by:

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Product Name

Amyloid Beta Protein

Description

Human Synthetic Amyloid Beta Peptide 1-42 (HFIP treated)

Applications

WB, In vivo Assay, In vitro Assay

Concentration

N/A - dried peptide film

Conjugates

No tag

Nature

Synthetic

Species

Human

Expression System

N/A

Amino Acid Sequence

DAEFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVVIA

Purity

>98%

Other Resources

Protocol for re-suspension of Amyloid Beta Peptide 1-42

Protein Length

42 amino acids

Field Of Use

Not for use in humans. Not for use in diagnostics or therapeutics. For in vitro research use only.

Properties

Storage Buffer

Dry powder. See "Other Resources" for re-suspension instructions/protocol.

Storage Temperature

-80°C

Shipping Temperature

Blue Ice or 4°C

Purification

N/A

Cite This Product

Human Synthetic Amyloid Beta Peptide (StressMarq Biosciences Inc., Victoria BC CANADA, Catalog # SPR-485)

Certificate Of Analysis

Certified >98% pure using mass spec and HPLC.

Biological Description

Alternative Names

Abeta Protein, Abeta peptide, Amyloid beta peptide, Beta amyloid peptide, amyloid beta precursor protein peptide, APP

Research Areas

Alzheimer's Disease, Amyloid, Neurodegeneration, Neuroscience

Cellular Localization

Cell membrane, Intracellular Vesicles


Gene ID

351

Swiss Prot

P05067

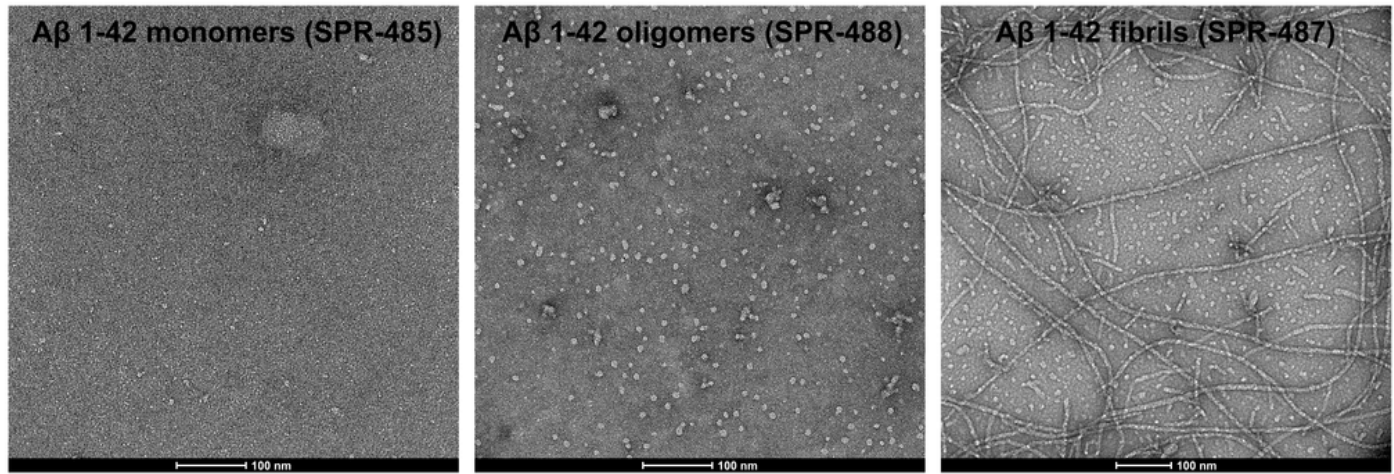
Scientific Background

Our amyloid beta peptide 1-42 (A β 42) is produced synthetically and treated with 1,1,1,3,3,3-Hexafluoro-2-propanol (HFIP) prior to drying which breaks down pre-formed fibrils and monomerizes the peptide, as previously published (1,2). Upon resuspension in DMSO/dH₂O, our A β 42 presents as a monomeric peptide without fibrils when observed under TEM, AFM and on a Western Blot with an anti-amyloid beta antibody. In contrast to A β 42 oligomer and fibril constructs, our A β 42 monomers were not toxic to primary rat cortical neurons. In the brain, amyloid beta peptide (A β) is generated by protease cleavage of amyloid precursor protein (APP), which aggregates into oligomers, protofibrils, fibrils and ultimately plaques in neurodegenerative diseases. The accumulation of A β plaques in the brain is considered a hallmark of Alzheimer's disease (AD), and most of the drugs tested for AD in the past 20 years have targeted amyloid beta accumulation (3). Soluble A β oligomers isolated from the brains of AD patients or those generated in vitro potently impaired synapse structure and function (4). A β oligomers generated in vitro were toxic to PC12 cells (2) and SH-SY5Y cells (5). A β was demonstrated to interact with tauopathies to affect neurodegeneration in AD patients (6) and accumulations of A β were shown to be associated with lower survival rates in Parkinson's disease patients with dementia (7). 

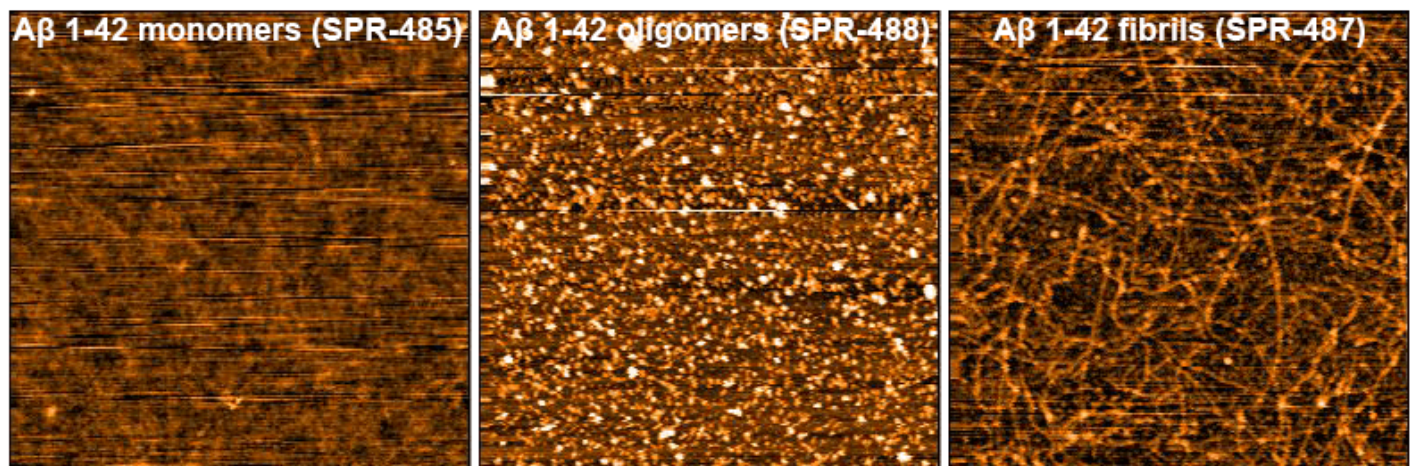
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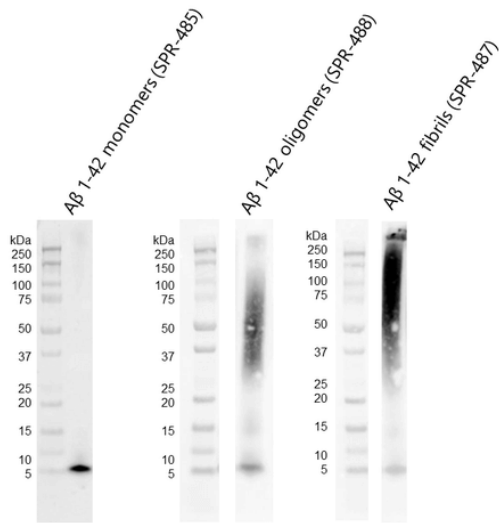
Product Images



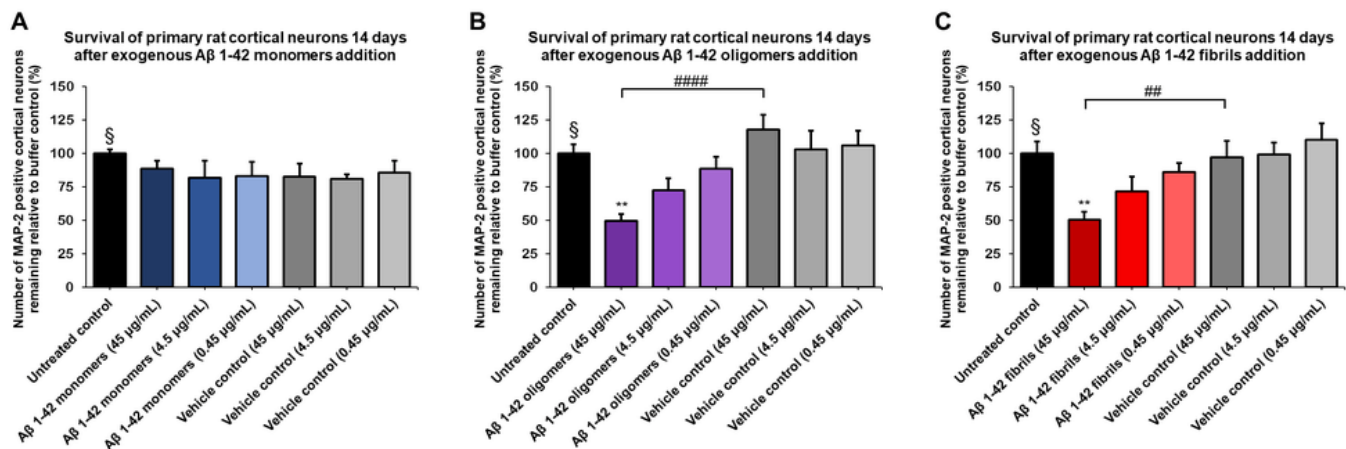
TEM of amyloid beta 1-42 monomers (SPR-485, left), oligomers (SPR-488, middle) and fibrils (SPR-487, right). Negative stain transmission electron microscopy images acquired at 80 Kv on carbon coated 400 mesh copper grids using phosphotungstic acid and uranyl acetate stain. Scale bar = 100 nm.



AFM of amyloid beta 1-42 monomers (SPR-485, left), oligomers (SPR-488, middle) and fibrils (SPR-487, right). Atomic force microscopy analysis of 1.0 mg/mL samples diluted to 0.1 mg/mL in dH₂O, mounted on freshly cleaved mica, washed, dried and analyzed with tapping mode. Representative images are 2.5 x 2.5 μm x-y with a z-range of 10 nm.



Western blot of amyloid beta 1-42 monomers (SPR-485, left), oligomers (SPR-488, middle) and fibrils (SPR-487, right) using anti-amyloid beta 6E10 antibody. Amyloid beta constructs at 160 pmol were run on 4-12% Bis-Tris SDS-PAGE, transferred to nitrocellulose in the presence of 0.02% v/v Tween-20, and blotted with 1:1000 mouse 6E10 primary antibody (Biolegend). Oligomers observed under TEM/AFM appear as distinct dimer/trimer bands at ~37-75 kDa on Western Blot with 6E10 antibody (middle). Fibrils observed under TEM/AFM appear as a distinct signal at greater than 100 kDa in the stacking gel (right).



Amyloid beta 1-42 oligomers (SPR-488) and fibrils (SPR-487) show a dose-dependent toxicity to primary rat cortical neurons, but not monomers (SPR-485). Survival of rat primary cortical neurons 14 days after treatment with different concentrations of (A) monomers, (B) oligomers or (C) fibrils quantified by MAP2 positive neurons and expressed as a percentage of control. Fibrils and respective vehicle controls were initially sonicated in a Bioruptor. Test conditions were run in the same plate as untreated control and vehicle controls, which consisted of buffer without amyloid beta 1-42 protein. Data expressed as mean \pm s.e.m. (n=6). A global analysis of the data was performed using a one-way ANOVA followed by Dunnett's test; ** p<0.01 stats vs control; ## p<0.01, ##### p<0.0001 stats vs vehicle control. § represents untreated control condition.


Product Citations (0)

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Export

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Orwa Aboud, Robert E Mrak, ..., W Sue T Griffin

Acta Neuropathologica Communications | 2013 Jul 29 | PubMed ID: 24252240 | [Read Article](#) 

"A β : Amyloid β ; AD: Alzheimer's disease; ApoE: Apolipoprotein E; AU: Arbitrary units; **β APP: Amyloid β precursor protein**; **Stress Marq**: 8-OH guanosine; IL-1: Interleukin-1." [More...](#) | [Article Share](#)

Currently there are no citations for this product.

Reviews

There are no reviews yet.