Title:
NANOPARTICLES AGAINST ALZHEIMER’S DISEASE: PEG-PACA
NANOPARTICLES ARE ABLE TO LINK THE Aβ-PEPTIDE AND INFLUENCE
ITS AGGREGATION KINETIC

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Abstract: (Your abstract must use Normal style and must fit in this box. Your abstract should be no longer than 300 words. The box will "expand" over 2 pages as you add text/diagrams into it.)
INTRODUCTION:
Alzheimer’s Disease (AD) is a neurodegenerative disorder characterized by a progressive loss of cognitive functions and characteristic pathological changes in the brain, evidenced by the formation of extracellular β-amyloid (Aβ) peptide aggregates and tangles of hyperphosphorylated Tau. Actually, Aβ peptide oligomers/aggregates are strongly considered as the main cause of neuronal cell degeneration. Despite all scientific efforts, efficient pharmacotherapeutic options for prevention and treatment of this disease are still lacking. One of the possible solutions could arise from the field of nanotechnology and especially from colloidal nanomedicines. Indeed, in our laboratory, biodegradable and “PEGylated” poly[(hexadecyl cyanoacrylate)-co-methoxypoly(ethylene glycol) cyanoacrylate] (P(HDCA-co-MePEGCA)) nanoparticles (NPs), have shown impressive results in vivo regarding their long-circulating behaviour and their ability to reach the central nervous system (CNS) by crossing the blood-brain barrier [1]. The aim of this study was to prepare stable PEG-PHDCA NPs, to study their ability to link/adsorb the Aβ peptide 1-42 and to influence its aggregation kinetic.

METHODS:
The ability of our P(HDCA-co-MePEGCA) NPs to slow down or disrupt the aggregation process has been investigated in vitro through kinetic studies performed with the Aβ 1-42 peptide or corresponding oligomers by capillary electrophoresis (CE) [2], surface plasmon resonance, confocal microscopy studies and Thioflavin T assays.

RESULT AND DISCUSSION:
CE experiments have shown that these NPs were able to link the Aβ 1-42 peptide both under its monomeric and soluble oligomeric forms, which was further confirmed by surface plasmon resonance and confocal microscopy. The ability of these NPs to influence Aβ 1-42 peptide aggregation was also confirmed by Thioflavin T assays. Since the NPs strongly affect the aggregation of Aβ 1-42, their effect on Aβ 1-42 uptake and toxicity is currently being investigated.

CONCLUSION:
These findings open an avenue to new therapeutic strategies in the field of Alzheimer’s disease.
REFERENCES:

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