

Amyloid-neuroinflammatory cascade in neurodegenerative diseases: role of S100 proteins

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The formation and deposition of amyloid oligomers and fibrils defined as an amyloid cascade plays central role in neurodegenerative diseases such as Alzheimer's and Parkinson's disease. The triggering and exacerbating factors of amyloid depositions, however, remain poorly understood. Neuroinflammation is increasingly recognized as an important factor, which may lead to amyloid formation and neurodegeneration. We were focused on the role of inflammatory S100A8 and S100A9 proteins in the amyloid cascade in vitro, in cell model and in ex vivo tissues from Alzheimer's disease, traumatic brain injury and prostate. S100A9 appeared to be as amyloidogenic as Abeta peptide- the major amyloid peptide in Alzheimer's disease, in vitro conditions and S100A9 coaggregates with Abeta in the Alzheimer's tissues. S100A9 can be primarily aggregating protein in traumatic brain injury, which may lead to Alzheimer's development at the later stages. As inflammatory conditions lead to significant rise of S100 protein concentrations, these can be triggering factors leading to amyloid aggregation and amyloid cytotoxicity in various tissues in the body, especially during age-related degenerative diseases.

Host: Cláudio M. Gomes
Protein Folding and Misfolding Laboratory

When: 25 June 2015 12h00

Where: Building C1, room 1.3.33 A

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