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Raman spectroscopy to probe the amyloid proteins involved in Alzheimer's disease

Alzheimer's disease (AD) is the most common neurodegenerative disorder and cause of dementia. The disease is pathophysiologically characterized by aggregated amyloid protein, as Aβ (Aβ). The disease is identified pathologically by amyloid plaques composed of aggregated amyloid peptide, neurofibrillary tangles composed of aggregated, hyperphosphorylated tau protein and neuron loss.

Our group aim at understanding the process of aggregation of $A\beta_{1\text{-}42}$ and Tau proteins involved in Alzheimer disease, and their interaction with membrane. Tip-Enhanced Raman Spectroscopy is very relevant to probe at the nanoscale the morphology and associated structure with the different species detected during the assembly of the $A\beta_{1\text{-}42}$ peptide and Tau protein. TERS allows an analysis of the surface of oligomers or fibers at the scale of the single object. [1-3]

We are also interested to determine what damage is created within the tissue in the vicinity of the amyloid plaques of the Aβ1-42 peptide. To probe this damage, we chose to study brain sections of mice and humans with severe Alzheimer's disease by vibrational microscopy methods. These methods require no labeling and are non-destructive. Fourier transform infrared and Raman imaging on Alzheimer's diseased mice and human brain tissue were performed. Our finding suggests the accumulation of hemes in the senile plaques of both murine and human samples. We compared the Raman signature of the plaques to the ones of various hemeoproteins and to the hemin-Aβ-42 complex. The detected Raman signature of the plaques does not allow identifying the type of heme accumulating in the plaques [4]. With the same approach, by FTIR and Raman imaging, we evidenced a reorganization of phospholipids in brain tissue from AD diseased tissues of mice with severe AD.

References

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