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# Interaction between the HSV-1 Glycoprotein B and the antimicrobial peptide Amyloid-β

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Unravelling the mystery of Alzheimer's disease (AD) requires urgent resolution given the worldwide increase of the aging population. There is a growing concern that the current leading AD hypothesis, the amyloid cascade hypothesis, may not stand up to validation with respect to emerging new data. Indeed, several paradoxes are being discussed in the literature, for instance, both the deposition of the amyloid-β peptide (Aβ) and the intracellular Neurofibrillary Tangles (NFTs) could occur within the brain without any cognitive pathology. Thus, these paradoxes suggest that something more fundamental could be at play in the onset of the disease and other key and related pathomechanisms must be investigated. The present study follows our previous investigations on the infectious hypothesis, which posits that some pathogens are linked to late onset AD. Our studies also build upon the finding that Aβ is a powerful antimicrobial agent, produced by neurons in response to viral infection, capable of inhibiting pathogens as observed in in vitro experiments. Herein, we ask what the molecular mechanisms in play are when Aβ neutralizes infectious pathogens. To answer this question, we probed at nanoscale lengths with FRET (Förster Resonance Energy Transfer), the interaction between Aβ peptides and glycoprotein B (responsible of virus-cell binding) within the HSV-1 virion. We concluded that Aβ insert into viral membrane, close to gB, and participate in virus neutralization.

## References

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## Biography

Tamas Fulop is a Professor at the Universite de Sherbrooke, Quebec, Canada. He has over 350 publications and his/her publication H-index is 65 and has been serving as an Editor in Chief for Gerontology and on the editorial boards of numerous reputed Journals.

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