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Targeting soluble oligomers associated with Alzheimer with antibody-secreting mesenchymal stem cells

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Although there is currently no effective treatment for Alzheimer's disease (AD), significant advances have been made in suppressing its progress, using conventional antibodies that block aggregation of the abnormally folded proteins. The major limitation in targeting plaques and aggregates associated with Alzheimer in the CNS is the blood-brain barrier (BBB), which restricts the access of biologics and drugs to the brain parenchyma. To that end, we used anti-oligomer PRIOC-antibody-secreting mesenchymal stem cells (MSCs) modified by Zinc Finger Nuclease (ZFN) technology. Tg2576 and htau mice models were used to assess the therapeutic efficacy of a sustained local delivery of PRIOC mAbs *in situ*. This led to substantial reduction of Aβ plaques and aggregate formation/reduction and neuropathology and improvement of cognitive deficits in these animals. Our preliminary results indicate that this strategy might be useful in reducing Aβ burden and neuropathology recognized in Alzheimer patients.

Biography

Mourad Tayebi was awarded a PhD in Neuroimmunology from Imperial College. He worked as Senior Research Fellow at the University of Sydney before returning to the UK and accepting an academic position at the Royal Veterinary College leading a team of scientists with a specific focus on protein misfolding diseases. He was offered a Faculty position at UT Health where he led a team of scientists interested in investigating the molecular mechanisms underlying protein misfolding/aggregation. His research focuses on refining the therapeutic strategies through developing novel antibodies that would specifically recognize the oligomeric forms of misfolded proteins. His team developed a panel of camelid antibodies able to transmigrate across the blood-brain barrier and enter the cell membrane of neurons.

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