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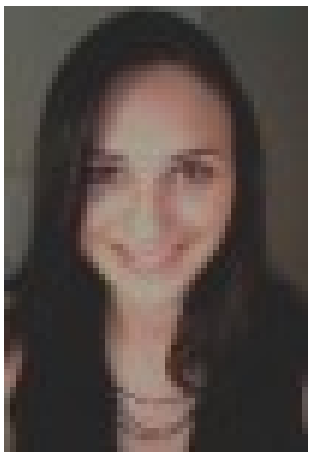

BSc in Biochemical Sciences with professional experience, University of Salford, 2004
MSc in Neuroscience, UCL -University College London, 2005
MPhil in Neurodegeneration, St George´s University London, 2007

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Science

MicroRNAs are important endogenous modulators of genes acting at the post-transcriptional level. miR-29 is a family of three closely related microRNAs encoded by two different genes with highest levels of expression in both the human and mouse brain. miR-29 has been implicated in a variety of biological and pathological processes (i.e. Alzheimer´s Disease (AD), a series of malignancies, different pathways of apoptosis, etc); however proof for the inferred functions of miR-29 *in vivo* is lacking. The data available are mostly correlative and based on *in vitro* cell biological experiments, hence proof for the inferred functions of miR-29 *in vivo* and moreover in the brain tissue is lacking. Previous work from our laboratory suggested a role for miR-29a/b-1 in sporadic Alzheimer´s Disease by targeting BACE-1/beta-secretase, which still awaits confirmation *in vivo*.

We are currently addressing this question by generating knockout mouse models, in which both miR-29 genes are inactivated. In addition to this, other pathways and proteins affected due to the loss of miR-29 in the brain, primarily in the context of AD will be explored.

Certainly, any phenotype observed in the genetically modified mouse models of miR-29 is further investigated in order to finally build a biological role for miR-29. Proteome studies will be performed to identify proteins that are differentially expressed in our models. Together and in parallel, microRNA expression will be assayed for possible identification

of compensatory actions. Biological relevance and confirmation will be processed accordingly.

Selected publications

Hébert SS, Horré K, Nicolaï L, Bergmans B, **Papadopoulou AS**, Delacourte A, De Strooper B (2009) MicroRNA regulation of Alzheimer amyloid precursor protein expression. Neurobiology of Disease 33: 422-428.

Hébert SS, Horré K, Nicolaï L, **Papadopoulou AS**, Mandemakers W, Silahtaroglu AN, Kauppinen S, Delacourte A, De Strooper B (2008) Loss of microRNA29a/b-1 in sporadic Alzheimer´s disease correlates with increased BACE-1/beta-secretase expression. Proc. Natl. Acad. Sci. USA 105(17): 6415-6420.