Bavo Heeman

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BSc Biomedical Sciences, Catholic University of Leuven, 2003
MSc Biomedical Sciences, Catholic University of Leuven, 2005
PhD Biomedical Sciences, Catholic University of Leuven, 2011

Current position
Research Associate, VIB Department of Molecular Genetics, since July 2012

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Keywords
Neurodegeneration – Parkinson's disease - Frontotemporal lobar degeneration – Alzheimer's disease - Animal models – Cell & Molecular biology – Epigenetics - Transcriptomics

Science
Parkinson's disease (PD) is the most common neurodegenerative movement disorder, affecting more than 1% of the population at 65 years of age, increasing steeply to 4-5% in 85-year-olds. PD has long been considered as a non-genetic disorder of „sporadic" origin with ageing and exposure to environmental factors as main risk factors for disease development. However, over the last decade several disease-causing mutations were identified in monogenically inherited PD genes (SNCA, PRKN, PINK1, DJ-1, LRRK2,…).
During my PhD, I have focused on unraveling the function of the (mitochondrial) kinase PINK1. Since clinical mutations in this gene are known to largely abrogate its kinase activity, I opted to silence PINK1 expression in cell culture as well as in mice by RNA interference and by the generation of a genetic knockout. Using unbiased automated live single-cell imaging, I have demonstrated a key role for PINK1 in the maintenance of mitochondrial homeostasis and energy metabolism under physiological conditions. In addition, I have shown that *in vivo* depletion of PINK1 sensitizes the nigrostriatal pathway to 6-OHDA toxicity and that PINK1 knockdown transgenic mice recapitulate several behavioral deficits reminiscent of PD.
Having recently joined the neurodegenerative brain diseases (NBD) group, I will be focussing on unravelling the functional role of – by our research group - recently identified disease-causing mutations in genes that are involved in Parkinson's disease (PD), Frontotemporal lobar degeneration (FTLD) and Alzheimer's disease (AD). More specifically, I will focus on the development and optimization of functional assays to elucidate the biological pathway(s) affected in these diseases. In addition, I aim to investigate the effect of the novel identified mutations on gene expression and gene regulation by transcriptome and epigenomic analysis, respectively.

Research projects and fellowships
Research Foundation Flanders (FWO). PhD fellowship
Period: 01.10.2005 – 30.09.2009
Title: 'Cell biological and *in vivo* study of the role of PINK1 in the pathogenesis of Parkinson's disease.'
Role: Fellow

Selected Publications

Valsecchi,F., Monge,C., Forkink,M., de Groof,A.J., Benard,G., Rossignol,R., Swarts,H.G., van Emst-de Vries,S.E., Rodenburg,R.J., Calvaruso,M.A., Nijtmans,L.G., **Heeman,B.,** Roestenberg,P., Wieringa,B., Smeitink,J.A., Koopman,W.J., Willems,P.H.: Metabolic consequences of NDUFS4 gene deletion in immortalized mouse embryonic fibroblasts. Biochim Biophys Acta 1817 (10): 1925-36 (2012)

Oliveras-Salvá,M., Van Rompuy,A-S., **Heeman,B.**, Van den Haute,C., Baekelandt,V.: Loss-of-function rodent models for Parkin and PINK1. Journal of Parkinson's Disease 1: 229-251 (2011)

Heeman,B., Van den Haute,C., Aelvoet,S.A., Valsecchi,F., Rodenburg,R.J., Reumers,V., Debyser,Z., Callewaert,G., Koopman,W.J., Willems,P.H., Baekelandt,V.: Depletion of PINK1 affects mitochondrial metabolism, calcium homeostasis and energy maintenance. Journal of Cell Science 124(Pt 7): 1115-25 (2011)

Van der Perren,A., Toelen,J., Carlon,M., Van den Haute,C., Coun,F., **Heeman,B.**, Reumers,V., Vandenberghe,L.H., Wilson,J.M., Debyser,Z., Baekelandt,V.: Efficient and stable transduction of dopaminergic neurons in rat substantia nigra by rAAV 2/1, 2/2, 2/5, 2/6.2, 2/7, 2/8 and 2/9. Gene Therapy 18(5): 517-27 (2011)

All publications