

Wouter De Coster

Neurodegenerative Brain Diseases Group
Department of Molecular Genetics, VIB
Laboratory of Neurogenetics, Institute Born-Bunge
University of Antwerp



BSc Biochemistry and Biotechnology, University of Antwerp, 2012
MSc Biochemistry and Biotechnology, University of Antwerp, 2014

Current Position

PhD student at University of Antwerp, VIB Department of Molecular Genetics

E-mail: wouter.decooster@molgen.vib-ua.be

Phone: +32 3 265 1039

Keywords

Frontotemporal Lobar Degeneration (FTLD) - non-coding genomic variation - whole genome sequencing - targeted resequencing - transcriptome sequencing - bio-informatics

Science

Frontotemporal lobar degeneration (FTLD) is a genetically, pathologically and clinically heterogeneous group of devastating neurological conditions caused by progressive degeneration of the frontal and temporal brain lobes. Clinical characteristics include changes in personality and behavior, language impairment and executive dysfunction. Dementia may become an eminent part of the syndrome in advanced stages. FTLD affects about 10 in 100,000 individuals.

The etiology and underlying disease mechanisms of FTLD are insufficiently understood and we lack effective disease-modifying therapeutics halting the progression of denervation or alleviating the symptoms. The underlying genetic defects remain to be discovered in about half of the families in which FTLD segregates. While previous research mainly focused on identifying pathogenic variations in protein coding genes, my research project will investigate the contribution of non-coding variation and structural variations, potentially with regulatory effects. The vast majority of the genome remains uninvestigated and this could in part explain the unidentified heritability in FTLD.

Using an integrated approach of transcriptome sequencing and whole genome sequencing my research aims to pinpoint causal regulatory variations. The contribution of these regulatory defects on transcription levels and splice patterns in FTLD will be explored and the initial insights in the downstream biological implications will provide us with opportunities for more rapid translation of these findings to development of cellular model systems for characterization of the biological implications of the identified mutations.