Sarah Herdewyn

Laboratory of Neurobiology
Experimental Neurology
Department of Neurosciences
Vesalius Research Center - VIB
K.U.Leuven

Bachelor and master degree in medicine (MD), KULeuven, Belgium, 2009
Neurologist in training (Master after master) since 2009 at UZ Leuven, Belgium
FWO-aspirant/PhD-student lab of Neurobiology since 2010, University KULeuven, Belgium

e-mail: sarah.herdewyn@vib-kuleuven.be
phone: 0032 (0)16 330754

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Science
Frontotemporal lobar degeneration (FTLD) and amyotrophic lateral sclerosis (ALS) are two neurodegenerative disorders that overlap at the clinical, genetic and pathological level. Although the majority of patients with FTLD or ALS present without a positive familial history, familial forms of both disorders are not rare.
Clinically, some patients with FTLD suffer from motor neuron degeneration as seen in ALS and some patients with ALS have cognitive impairments. Loss of function mutations in the gene encoding the growth factor
progranulin (PGRN), which result in reduced PGRN protein levels, are a common cause of familial FTLD with or without ALS. Missense mutations in the DNA/RNA binding protein TAR DNA binding protein 43 (TDP-43) are a cause of ALS and are also encountered in some patients with FTLD. At the pathological level, most forms
of sporadic and familial FTLD (including patients with PGRN mutations) or ALS have inclusions of TDP-43. PGRN and TDP-43 thus play a central role in the pathogenesis of FTLD and ALS. In the current research project, the interplay between PGRN and TDP-43 will be studied using in vitro and in vivo models. Better understanding of the mechanisms of neurodegeneration linked to PGRN and TDP-43 will hopefully lead to
better treatments.