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Science

After Alzheimer's disease, frontotemporal lobar degeneration (FTLD) is second most common dementia-type in individuals under 65 years. Degeneration in prefrontal and anterior temporal areas leads to variable psychiatric problems (changes in personality and social conduct), and/or disturbances in language with impaired word retrieval and/or comprehension. Behavioral symptoms disrupt normal functioning of the patient, with loss of social and professional activities. In few patients with FTLD, motor neuron degeneration (MND) resulting in progressive muscle weakness can be observed. The course is progressive with devastating effects on patients and their families. FTLD is a familial disorder in up to 30-40% of cases.

In 2006, mutations in progranulin on chromosome 17q were found which take account for 40% of familial FTLD cases. These mutations are inherited mostly in an autosomal dominant way and most of these mutations are null mutations although some missense mutations have been observed as well. Until now all hypotheses regarding the pathogenic mechanism suggest shortage of progranulin through a haploinsufficiency mechanism as underlying disease mechanism in patients with progranulin mutations. This PhD project aims at improving current insight into the biology of progranulin and at identifying the mechanisms underlying the neurodegeneration induced by mutations in the progranulin gene.