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Science

Amyotrophic lateral sclerosis (ALS) is a late-onset progressive neurodegenerative disease, mainly but not exclusively affecting motor neurons in the spinal cord, brain stem and cortex. This degeneration results into fasciculations, progressive muscle weakness and atrophy, spasticity and, ultimately, paralysis, and is fatal within 5 years after disease onset. Denervation of the respiratory muscles and the diaphragm is in most cases the fatal event. The course of disease is typically between 1 and 5 years after onset of symptoms. ALS has a prevalence of 4 to 6 per 100000 and the onset of disease is usually between 45 and 60 years of age. Currently, only one drug, riluzole, has shown to have a limited effect, as it modestly prolongs the survival.

The majority of patients have no familial history (sporadic ALS), while in 10% of cases an underlying genetic cause is present (familial ALS). A variety of disease causing mutations is known at the moment, such as mutations in superoxide dismutase 1 (SOD1), hexanucleotide repeats in C9orf72, mutations in fused in sarcoma/translocated in liposarcoma (FUS/TLS) and in the gene encoding TAR DNA binding protein 43 (TDP-43).

Much progress in the ALS research field has been made thanks to the mutant SOD1 overexpressing mouse model. These mice develop an adult-onset, progressive and fatal motor neuron degeneration, thereby recapitulating the human disease.
Studies using this mutant SOD1 mouse model have shown that motor neuron degeneration is non-cell autonomous. This means that also non-neuronal cells contribute to the death of motor neurons and the progression of the disease.

The aim of this project is to investigate how non-neuronal cells interact and contribute to the motor neuron degeneration seen in ALS.

Selected publications

Philips T, Bento-Abreu A, **Nonneman A**, Haeck W, Staats K, Geelen V, Hersmus N, Küsters B, Van Den Bosch L, Van Damme P, Richardson WD, Robberecht W. 2013. Oligodendrocyte dysfunction in the pathogenesis of amyotrophic lateral sclerosis. *Brain* 136(Pt 2):471-82