The World Health Organization predicts that between 2015 and 2050, the proportion of the world's population over 60 years old will nearly double. While advances in modern medicine contributed to this general increase in population life expectancy, new public health challenges have arisen. Age associated neurodegenerative disorders are now the fastest growing cause of death in the aging population.

Though disparate in their pathophysiology, neurodegenerative diseases, are collectively characterized by the accumulation of clumps of protein aggregates in the brain. We are especially interested in two of these diseases, amyotrophic lateral sclerosis (ALS) and frontal temporal lobar degeneration (FTLD). The accumulation of three proteins, called tau, FUS and TDP-43, are signatures of these two diseases. Interestingly, tau also forms aggregates, called tangles, in Alzheimer’s disease, the most common cause of dementia.

It is not yet known whether malfunction of one of these proteins could interfere with the other two to trigger the disease. This collaborative project will tackle this question by using complementary approaches in mouse and cell-based models to understand whether and how FUS, tau and TDP-43 interact in ALS and FTLD pathogenesis.