Huntington´s disease, an autosomal dominant neurodegenerative disease, affects the cell in several toxical ways. One of them is accumulation of protein aggregates in cytoplasma, which could become a serious problem especially for long-lived cells such as neurons.
Autophagy (macroautophagy) is an important catabolic pathway, crucial for cell survival. If fully functional, it should eliminate protein aggregates and reduce the toxic effect on the cell. However, recent works show that this pathway might be defective, most probably in the cytoplasmic cargo recognition.
In my work I used a transgenic miniature pig model of Huntington´s disease to verify the hypothesis of autophagical dysfunction in individuals suffering from Huntington´s disease. I studied levels of autophagosomal markers – LC3 and p62 in mesenchymal stem cells after different autophagy stimulation treatments, and ammonium chloride was found the most effective. In addition I evaluated the effect of age of the animals on autophagic function, but no significant changes were identified, even if animal genotype was considered.
Moreover I had an opportunity to study proteins levels in three porcine brain tissues – cortex, cerebellum and striatum. Even though there is no significant diference, we can observe a trend of LC3 II and p62 increase in cerebellum and striatum. This leads us to the idea of changes in autophagosomal pathway occuring only slowly and that they will manifest in older animals.