# Lay summary

Our cells must recycle their «waste» to stay healthy, work properly, and adapt to survive. Autophagy is a cellular recycling process that helps cells break down and reuse their damaged, harmful, or unnecessary parts. Cells create membranous «bags» called autophagosomes to sequester the unwanted material. Once the «waste» is bagged, autophagosomes fuse with lysosomes, delivering their contents inside these acidic organelles. Lysosomes act like the cell's digestive system enzymatically degrading unwanted material like proteins, sugars, and fats into basic «building blocks». These are then used as fuel or recycled to build new parts for the cell. When autophagy is defective, unwanted material builds up, which can cause various diseases, including neurodegenerative disorders and cancer.

This thesis explores how the formation of autophagosomes is regulated. We studied the role of specific motor proteins, called dynamins, in controlling the transport of a key autophagy protein, Atg9. We also identified enzymes that regulate autophagy and are involved in adding or removing a special molecular tag named ubiquitin to proteins. This thesis also explores how degrading specific proteins by autophagy is important for the cell to adapt its metabolism when nutrient levels change. For this, we developed a new experimental tool that helps identify proteins degraded by autophagy at a specific time in baker’s yeast. We found proteins, including metabolic enzymes, that may be specifically degraded to adapt to nitrogen scarcity in this model organism.

Our findings improve our understanding of the regulation of autophagy and its role in metabolic adaptation.