To survive in adverse conditions cells must respond properly to the changing environment. One kind of cell response to stress is the formation of so-called stress granules. Stress granules are clusters of RNAs and proteins that aggregate in the cytoplasm and allow the cells to survive unfavorable conditions by sequestration of mRNAs stalled at the translation initiation stage. These dynamic structures that do not contain a membrane are created in response to stresses including oxidative stress, osmotic stress, and heat shock with the first one induced by incubation with sodium arsenite being the most studied one.

In this project, we plan to study the molecular mechanisms governing KHNYN function. Our previous results clearly show that a poorly described protein named KHNYN is a negative regulator of stress granules, i.e. when KHNYN is expressed in the cells only about 50% of them can form stress granules when they are treated with sodium arsenite while 100% of control cells form stress granules in the same conditions. Additionally, our results indicate that overexpression of this protein causes cell death. To discover the mechanism of action of the KHNYN protein, we plan to employ high-throughput transcriptomics and proteomics approaches combined with advanced microscopy imaging analyses. The results obtained in this project will allow for the preparation of a wide-view mechanistic image of KHNYN role in stress response.