

## **Project 1.10 Exploring the impact of nanoplastics on cellular injury: investigating the role of mitochondrial potassium channels**

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**Laboratory:** Intracellular Ion Channels, the project will be implemented in cooperation with Department of Physics and Biophysics, Institute of Biology, Warsaw University of Life Sciences-SGGW  
**www:** <https://infraredmito.nencki.edu.pl/>

### **Background:**

Nanoplastics (NPs) pose significant global health risks, contributing to conditions like Crohn's disease, intestinal cancer, and inflammation. Mitochondrial dysfunction plays a key role in NP-induced cytotoxicity and is associated with diseases such as muscle disorders, neurodegeneration, and intestinal illnesses. Recent research highlights the importance of mitochondrial potassium (mitoK) channels, which regulate K<sup>+</sup> permeability in the inner mitochondrial membrane. Activation of mitoK channels protects against cell death during myocardial infarction and cerebral hypoxia/reperfusion, offering cardioprotective and neuroprotective effects.

Since intestinal epithelial tissue is a primary site of NP deposition, mitoK channels in epithelial cells may play a crucial cytoprotective role against NP-induced damage. Preliminary findings indicate functional mitoK channels in epithelial cells and altered ion transport in the presence of NPs. We suspect that mitoK channels in epithelial cells might play a critical role in cytoprotection against NP-induced injury. However, knowledge about mitoK channels in epithelial tissues remains limited.

### **Aim:**

The project aims to identify and characterize mitoK channels in human intestinal epithelial cells and investigate their role in protecting against damage caused by nanoplastics (NPs) or oxidative stress. Key objectives include the biophysical, electrophysiological, and pharmacological characterization of mitoK channels, along with the evaluation of ion transport changes in response to NPs. The project also seeks to identify potassium channel proteins at the molecular level and generate new cell lines expressing or lacking selected mitoK channels. We will also study the modulation of mitochondrial bioenergetics and cellular electrical properties of the cell monolayer by modulators of mitoK channels and nanoplastics.

Additionally, the research will explore the effects of NPs on cell survival, oxidative stress, apoptosis, cell cycle regulation, and DNA damage/repair mechanisms. A particular focus will be on the role of mitoK channels under NP-induced stress conditions. These studies are expected to enhance understanding of endogenous cytoprotective mechanisms in epithelial cells, potentially leading to novel therapeutic strategies for treating epithelial tissue injuries and intestinal disorders linked to NPs.

### **Requirements:**

- we are seeking a motivated, creative, and collaborative individual with a background in chemistry, biophysics, biology, biotechnology, or a related field;
- candidates should possess basic knowledge of biochemical, techniques and/or molecular biology and biophysical methods;
- additionally, a strong command of both spoken and written English is required.