One of the main environmental stressors of the 21st century, having a huge impact on our health, is plastic pollution. Global production of plastic exceeds 320 million tons per year, over 40% of which is used as a single-use packing, resulting in plastic waste. In combination with wind, wave action and abrasion, degraded plastic fragments into micro- and nano-sized. Micro/nanoplastics are thus considered as a main priority pollutants which are listed in the Stockholm Convention for their potential adverse health effects. The human body is exposed to micro/nanoplastics through various exposure routes such as for example air, food and water. The average amount of annual exposure via inhalation is 55 000 particles, which exceeds the 49 000 particles ingested from food and beverages. Most human exposure to airborne micro/nanoplastic may occur indoors. This has important consequences because Europeans and Americans spend on average 90% of their time indoors, whether at home or at work. The fate of inhaled micro/nanoplastic and their uptake in lung tissue is one of many unknowns. Long-term exposure to airborne microplastics can cause a variety of diseases, including asthma, fibrosis, pneumothorax, chronic bronchitis and lung cancer.

A lot of basic questions about the connection between air plastic pollutants and human health still remain unanswered, therefore we decided **to investigate how environmental pollution, in the form of nanoplastic dust, influence mitochondrial adaptation.**

Mitochondria are important cellular organelles responsible for energy production and regulation of cellular metabolism, and are considered an attractive therapeutic target, because as a first within the cell respond to stress. It is also the first place in the cell that responds to stress. Under changing environmental demands, reprogrammed mitochondrial signaling plays a key role in maintaining the metabolic flexibility of the cell. We want to find out how a healthy and cancerous epithelial cell of the lungs responds to the stress caused by nanoplastic particles by activating the retrograde signaling cascade mitochondria-nucleus-mitochondria.

In our previous study, we analyzed the effect of the mitochondrial retrograde signaling of neurodegenerative diseases, with chronic mitochondrial stress, such as Amyotrophic Lateral Sclerosis (ALS) and Alzheimer disease, and demonstrated different ways of adaptation of mitochondria to altered conditions. Given the importance of mitochondria for cell health, it can be postulated that studying the function, signaling pathways and physiology of mitochondria in a cell may lead to therapeutic intervention.

To study the adaptation of mitochondrial function to chronic stress, we will focus on identifying and assessing several elements, especially of the stress response pathway, like mitochondrial plasticity and dynamics, namely mitochondrial biogenesis, mitophagy, and mitochondrial network morphology. Appropriate bioinformatics tools will allow the integration of many types of mitochondrial parameters and the comparison of mitochondrial responses to environmental stress in healthy and cancer cells.