Mitochondria are indispensable parts of our cells that contain molecular machinery called oxidative phosphorylation system, or in short – OXPHOS. It is a set of large complexes made of multiple proteins that produce energy. For this reason, mitochondria are often referred to as powerplants of the cell. To keep up energy production, our cells need to maintain these precious molecular machines in good shape. Next to bulk degradation of whole mitochondria via autophagy or mitophagy (self-eating or mito-eating, respectively), vigilant guards that live inside of mitochondria (intramitochondrial factors) are able to recognize and remove damaged part of the OXPHOS complexes – making place for newly produced ones. This constant exchange is critical for any protein of the cell, as time inevitably leads to accumulation of molecular damage. Appropriate repair of powerplant machinery becomes even more important during cell stress. This is when cells rely on the energy from OXPHOS for their survival and repair. In my project, I am interested in the mechanisms of OXPHOS quality control used by cells that experience severe difficulties. To model such conditions, I will use drugs that destroy cell DNA or allow for protein damage to appear at a faster rate. Such cells enter then a stress management programme called cellular senescence. Previous studies showed that mitochondria of senescent cells become inefficient at producing energy while mitophagy, instead of working at full speed, is switched off. This suggests that OXPHOS might indeed become damaged while intramitochondrial factors are left as the sole guards controllers of OXPHOS shape. The fate of OXPHOS and involvement of intramitochondrial factors in cellular senescence has not been studied so far. In my project, I want to check how fast the proteins that make up OXPHOS complexes are replaced and whether intramitochondrial factors manage to keep at least some OXPHOS functional under extreme stress. By employing a range of powerful techniques, next to tracking OXPHOS exchange, I will also check how much of the OXPHOS machinery is actually present in mitochondria, whether its composition changes as well as how efficient the complexes are at producing energy. Finally, I will set out to identify the leading actors that maintain OXPHOS function. Revealing these fascinating details about mitochondria and OXPHOS will not only improve our knowledge on how cells work but can become extremely beneficial from biomedical perspective. Cells enter senescence particularly frequently with ageing. The same is true during many anti-cancer therapies that succeed at destroying cancer cells but simultaneously, impair the function of healthy cells. Accumulation of high numbers of senescent cells drive the chronic diseases of ageing, such as cancer, heart diseases, neurodegeneration or diabetes as well as the side-effects of anti-cancer therapies. Therefore, I would like to understand whether the OXPHOS quality control is essential for the survival of senescent cells and whether it affects other processes that make senescent cells harmful. So far, scientists developed several strategies that eliminate senescent cells resulting in mice living healthier and longer. My study will help to understand whether the processes that keep OXPHOS fit are necessary for senescent cells – their disruption cause senescent cells to die or become less harmful. This will be critical to develop novel therapies against senescent cells, ameliorating various chronic diseases of the old age.