Female germ cells comprise essentially immortal cell line which transmits the mitochondrial genome throughout generations. Sufficient number of functional mitochondria is known to be an important factor for the implantation rate and pregnancy outcome. It was estimated that up to 1: 200 babies is born with dysfunctions related to the mitochondria malfunctions. The presence of two types of mtDNA in the cell, the wild type mtDNA and the mutated mtDNA, is defined as heteroplasmy which provides a source of many human diseases associated with heart dysfunctions, obesity, diabetes and problems with learning, movement, or sight and hearing. Nowadays, the treatment and diagnosis of mitochondrial diseases is very difficult and is mainly limited to the symptomatic treatment. It is considered that the cells possess natural mechanisms preventing from accumulation of mtDNA mutations which act via "self-consumption" of damaged organelles in the process called autophagy (or mitophagy in case of type of autophagy restricted to mitochondria). However, this purification process is not always fully sufficient and it's efficiency may be additionally impaired during aging. In current research tasks, our group have been focused on the phenomenon of embryonic diapause which is the state of blastocyst dormancy in anticipation of the mother's signal for the embryo implantation. Our research confirmed that embryos undergoing embryonic diapause exhibit elevated autophagy. Thanks to this, the embryo is able to survive in the situation of the lack of nutrients from the mother due to the "consumption" of own unnecessary or damaged cellular structures. In our studies we are able to induce the state of diapause in mice. Long-term observations of such model make as the assumption that the mice embryos that underwent embryonic diapause exhibit higher pregnancy outcome and moreover extend lifespan expectancy compare to the standard pregnancies. As the chance of implantation and the rate of aging were often associated with the condition of the mitochondria, this led me to the hypothesis, that **autophagy during the embryonic diapause results in the removal of damaged mitochondria and in this way increases the viability of the offspring via reduction of mitochondrial mutation inheritance.** Because the studies will be carried out on the model diet induced obesity and control females whose offspring will undergo/ not undergo the embryonic diapause, this project will allow for;

a) an assessment of the risk of inheriting damaged mitochondria from obese mothers and determining the effects of this inheritance. For this purpose, the process of mitophagy in diapausing embryos will be investigated and then, the level of mutations that remained in the embryos undergone embryonic diapause will be evaluated.

b) examination whether the embryonic diapause could prevent from mutation inheritance. In this research task, the research will focus on the new-born offspring after the embryonic diapause. With the use of New Generation Sequencing, electron microscopy, Raman spectroscopy and fluorescence techniques the oocytes of the offspring will be examined in order to evaluate the general mitochondrial quality and mitochondrial mutations inheritance.