**DESCRIPTION FOR THE GENERAL PUBLIC**

In the abstract world letting your imagination run wild, the mitochondria may resemble little monsters overcrowded with ,,energy". Why like this? Because for many centuries, mitochondria were known mainly from ,,energy production" and to be more precise – ATP production, which constitutes peculiar ,, nourishment" for the cell. Generally speaking, in nowadays there is no such well technologically advanced power station like mitochondrium. However, life is not so colorful because a ,,power stations" carry a potential risk, which in case of mitochondria is reactive oxygen species (ROS) production. Excessive ROS production is extremely harmful for the cell because it causes oxidative stress (the state which may damage the cells and in some cases induce cell death called "apoptosis"). Therefore, from point of view of our research, which we would like to conduct, ROS production and apoptosis are processes that involve also a small adaptor p66Shc protein constituting aim of our studies. As a both processes exert disorders in cancer cells (often having beneficial influence on cancer cells simultaneously making cancer biology extremely complicated) and p66Shc protein is involved in ROS production and apoptosis, examination the impact of p66Shc protein among others on mitochondria bioenergetics and the status of antioxidant defense system in breast cancer cells seems to be justified. In order to increase the value of our studies it should be mentioned that p66Shc protein may also act as negative regulator of proliferation what in the context of cancer cells (exerting fast cell growth) seems to be extremely promising. Within this project we would like to explore the role of p66Shc protein in human breast cancer cell lines with the use of cancerous and noncancerous breast cell lines. Particular attention will be paid to changes in the p66Shc level and their impact on cellular response of breast cancer cell lines to an anti-cancer chemotherapy drug - doxorubicin. Modifications in the p66Shc level were obtained by us with the use of genetic modifications (changes in the ,,bricks" building DNA) concerning the increase of p66Shc level in cancer cells as well as total removal of this protein. In order to confirm the significance of our project, we are going to evaluate the impact of mutation at the ,,region" of p66Shc protein responsible for its oxidative stress response (it is Serine 36 residue). Taking into consideration the fact that phosphorylation at Serine 36 residue (Ser36) is caused by oxidative stress-related factors simultaneously contributing to the increase of ROS production (the vicious cycle), examination of this ,,functionality" of p66Shc protein may constitute the novel approach to better understanding of cancer biology. Therefore, deeper insights into the cellular response of breast cancer cells with a modified p66Shc level to chemotherapeutic treatment (doxorubicin) may enable us to answer the question of whether the p66Shc adaptor protein may act as an apoptosis mediator or stress sensor in cancer cells increasing cancer cell death.