UNIVERSITY OF WARSAW | CeNT CENTRE OF NEW TECHNOLOGIES

invites to a seminar by

Prof. Mariusz R. Więckowski

Laboratory of Bioenergetics and Biomembranes,
Nencki Institute of Experimental Biology

What can we foretell from mitochondrial parameters?

19th of April 2018 at 12 p.m.

Venue: Centre of New Technologies, Banacha 2C,
Lecture Hall 0142 (Ground floor)

Host: Marta B. Wiśniewska

Very often defects in mitochondrial function are associated with several pathologies, which in turn can lead to various diseases. Improper function of the mitochondrial respiratory chain can lead to decreased ATP production, what is often considered as a main cause of the observed symptoms. However, growing evidence has suggested a direct relationship between development and progression of mitochondrial disorders and the presence of oxidative stress. Increased ROS production may lead to oxidation of DNA, lipids and proteins and thus can affect fundamental cellular processes. Whether mitochondria are the primary source of ROS production remains under debate, but mitochondria are clearly the predominant target of the damaging effects of ROS. The aim of our studies is complex characterization of mitochondrial respiratory chain function and the related parameters responsible for or involved in mitochondrial defect-mediated cellular dysfunction. Our studies address mitochondrial bioenergetics and oxidative stress to elucidate how these parameters participate in the pathogenesis of different mitochondrial disorders. We want to determine which mitochondrial parameters are significant contributors to the development of mitochondrial disorder. We have found that mitochondrial bioenergetic parameters, ROS production and the status of antioxidant defence system show unique multifactorial profiles characteristic for fibroblasts from controls and patients with different mutations in mtDNA (MTND1, MTND3, MTND5 and MTATP6) and nDNA (SURF1; SCO2, DGUOK, PDHA1 as well as in the huntingtin gen). These unique profiles of the parameters describe detected similarities, differences and dependencies between measured parameters. We believe that such comprehensive analysis will suggest potential therapeutic strategies in which mitochondrial physiology or ROS production is modulated to alleviate the consequences of mitochondrial diseases.