Cancer cells generally present abnormal bioenergetic properties including an elevated glucose uptake, a high glycolysis and a poorly efficient oxidative phosphorylation system. However, the determinants of cancer cells metabolic reprogramming remain unknown. The main question in this project was how environmental conditions *in vivo* can influence functioning of mitochondrial OXPHOS, because details of mitochondrial bioenergetics of cancer cells are poorly documented. We have combined two conditions, namely glucose and oxygen deprivation, to measure their potential interaction. We examined the impact of glucose deprivation and oxygen deprivation on cell survival, overall bioenergetics and OXPHOS protein expression. As a model, we have chosen a human breast carcinoma (HTB-126) and appropriate control (HTB-125) cultured cells, as large fraction of breast malignancies exhibit hypoxic tumor regions with low oxygen concentrations and poor glucose delivery. The results demonstrate that glucose presence or absence largely influence functioning of mitochochondrial oxidative phosphorylation. The level of mitochondrial respiration capacity is regulated by glucose; by Crabtree effect, by energy substrate channeling towards anabolic pathways that support cell growth and by mitochondrial biogenesis pathways. Both oxygen deprivation and glucose deprivation can remodel the OXPHOS system, albeit in opposite directions. As an adaptative response to hypoxia, glucose inhibits mitochondrial oxidative phosphorylation to the larger extent than in normoxia. We concluded that the energy profile of cancer cells can be determined by specific balance between two main environmental stresses, glucose and oxygen deprivation. Thus, variability of intratumoral environment might explain the variability of cancer cells´ bioenergetic profile. In the second part of thesis, I discuss consequences of findings of UCPn transcripts in the studied mouse and rat tissues. In the third part of the thesis, we tried to elucidate another apoptotic pathway connected to mitochondria, independent of caspases.