Title:	Studying Mitochondrial Role in Cell Growth Regulation in Drosophila melanogaster
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Abstract:	Cell growth is the process of increase in mass and size of a cell, which is coordinated through various cell autonomous and non-autonomous factors. The role of many such factors like nutrients, growth factors, signaling cascades, transcription factors have been quite well studied. The process of cell growth is quite energy demanding and requires extensive metabolic activity. Yet, there are no reports about how mitochondrial function is linked with cell growth regulation. The focus of this project is to elucidate the mechanistic basis of cell growth regulation by mitochondrial function. Mitochondrial role in many cellular events like cell cycle regulation, differentiation, and apoptosis is well established and it has been shown to be affecting cell signaling pathways by modulating its outputs e.g. ATP, Reactive Oxygen Species (ROS) etc. We have launched a genome wide loos-of-function screen for nuclear encoded mitochondrial proteins to identify genes which can modify overgrowth phenotype associated with Cyclin D-CDK4 overexpression in adult eyes of Drosophila. We have identified three classes of overgrowth modifier; enhancers, suppressors and no change. Thus, we establish that attenuating mitochondrial function can modulate cell growth in positive as well as negative manner. We are doing further cellular level analysis of candidate genes using larval fat body. We have identified a gene mitochondrial acyl carrier protein (mtACP1), a subunit of complex I which when knocked down leads to decrease in cell size. Further analysis shows that mtACP1 knock down cells have high levels of ROS as well as JNK pathway is getting activated in these cells. We have found that in mtACP1 knock down cells, JNK pathway is mediating retardation in cell size. This suggests that attenuating mitochondrial function could affect cell size regulation mediated through JNK pathway.
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