Title:	Metabolic regulation of the hematopoietic niche in Drosophila melanogaster
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Abstract:	Stem cells are identified as the source of virtually all differentiated cells that are replenished during the lifetime of an individual. Stem cells serve as a resource to take care, to maintain a critical balance between stem and differentiated cell population which is crucial for the long term maintenance of functional tissue types. They do so by choosing one of several alternate fates: self-renewal, commitment to differentiate and senescence or cell death. Microenvironment in which the stem cells reside and maintain their stemness is commonly known as the stem cell niche. A specific signaling pathway or a cell adhesion molecule allows the niche cells to maintain contact with stem cells and in the absence of this signal, the stem cells leave their niche and/or either divide, differentiate or apoptose. Question of how stem cells are maintained by niche has been actively investigated. But how the stem cell niche is maintained has not been investigated in detail. For investing into the question of stem cell niche maintenance we are probing Drosophila larval hematopoietic system, the lymph gland. Lymph gland, with its three very well characterized zones has been exploited to understand the Signaling network between niche, progenitor and differentiated cells. The three distinct zones namely, the outer peripheral zone, Cortical Zone or CZ houses the differentiating hemocytes, the Medullary Zone or MZ in which the stem like cells reside, and the third zone is the Posterior Signaling Centre or PSC that serves as the niche for the MZ cells. The small group of 40-45 PSC/niche cells control the fate of thousands of blood cell progenitors of the medullary zone intrigued us to hypothesize on the active metabolic nature of these Signaling cells and led us to probe into the role of mitochondrial genes in niche maintenance and functionality. We have undertaken a loss of function RNAi based screening for nuclear encoded mitochondrial genes in the niche. We got few candidate genes in which the niche morphology was perturbed i.e., either the niche cell number increased or decreased compared to the control niche number. In this scenario in which the niche was perturbed, we looked at the functional status of the aberrant niche by looking at the status of progenitor cells and the differentiating cells. We also tried to investigate the mechanism leading to the perturbation of niche cell number and functionality, by looking at the stress signals like the Reactive Oxygen Species or ROS generation in this mitochondrial loss of function background in the niche. It will be interesting to address whether this loss of function of the nuclear encoded mitochondrial genes in the hematopoietic niche has led to switching on an alternate metabolic pathway like aerobic glycolysis. Or as reported in some malignant and developmental scenarios, the niche cell is still able to keep the OXPHOS metabolic pathway on.
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