BCHM421/422

Project Outline: Mitochondria are commonly understood as the energy source for the cell, using acetyl-CoA to produce ATP. While mitochondria are typically drawn as oval-shaped tubes containing cristae, these organelles are much more dynamic, splitting, fusing and moving through the cell as a large network. As cells become energetically stressed, mitochondria re-model and part of this may be to interact properly with the cell autophagy pathway.

During autophagy, cells degrade a portion of their cytoplasm as a last resort to meet nutritional requirements. Autophagy also plays a critical role in degrading damaged mitochondria, a process termed mitophagy. Mitophagy is crucial for cell homeostasis since accumulation of damaged leaky mitochondria will very quickly poison the cell. In the current model, cells faced with nutrient starvation trigger mitochondrial fusion to thereby evade mitophagy and maintain cellular energy levels. We have been investigating the mechanisms that coordinate mitochondrial fusion and autophagy during starvation stress. This project aims to identify the autophagy mechanisms that signal to the mitochondrial fusion pathway. Findings could suggest potential targets for improved mitochondrial function and reduced rates of cellular aging.

Supervisor: Edmond Chan

Project title: Regulation of mitochondria fusion machinery by nutrient stress

Keywords: Mitochondria, Cell metabolism, Signal transduction, Protein kinase

Project goals: To define the signalling events linking autophagy kinases to mitochondrial membrane dynamics

Experimental approaches: You will learn how to culture mammalian cells. These cell models will be treated to different nutrient stress conditions or control stimulants. Signalling events will be measured using immuno-blotting protein biochemistry. Mitochondrial fusion will be measured using microscopy and computer-based analysis of mitochondria patterns.

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