MitoCPR - A stress response that maintains mitochondria homeostasis

All mitochondrial functions rely on the import of proteins into the organelle. This process is constantly challenged during specific types of cellular stress (e.g. oxidative stress) and is known to cause several diseases when impaired. Little was known about how cells respond to mitochondrial protein import defects or whether they can overcome this stress and recover mitochondrial functionality. We characterized a specific cellular response to import stress that we have termed the mitoCPR. We showed that the mitoCPR is important for mitochondrial homeostasis and function. We found that the mitoCPR maintains mitochondrial proteostasis and protein translocation by directing the removal and proteasomal degradation of stalled proteins from the mitochondrial surface, a function mediated by the ATPase Msp1 and its adaptor Cis1. This study provides important molecular insights into how cells cope with a stress occurring in both physiological and pathophysiological conditions to maintain mitochondrial and organismal health.

Refreshments will be available outside LSC3!!!