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Monash Biomedicine Discovery Institute
Cancer Program

OTHER PROGRAM AFFILIATIONS



Metabolic Disease
and Obesity



Infection and Immunity

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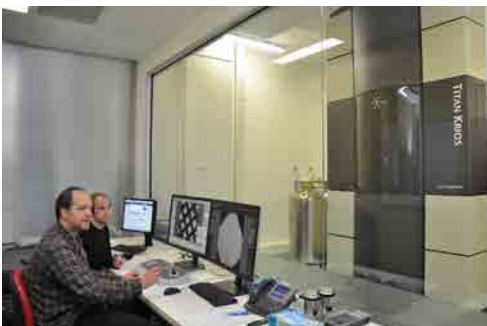
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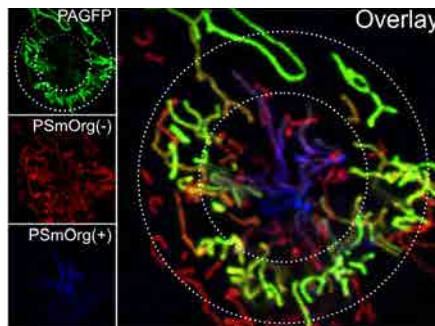
Our lab is focused on the regulation of autophagy, a major intracellular degradation process. In cancer, autophagy plays complex roles and can suppress tumours, but also helps tumour cells survive in other cases. Autophagy delivers cellular and cytoplasmic structures to the lysosome, where they are degraded. This process is tightly linked to cellular metabolism and is an evolutionary conserved survival mechanism that helps cells cope with nutrient starvation. We have recently discovered a link between metabolic control and the Serine/Threonine kinase ULK1, a key regulator of autophagy. We aim to develop a detailed understanding of how these regulatory networks are causing changes in intracellular membrane trafficking during autophagy.

Research Projects

1. High-resolution imaging of the mitophagy pathway
2. Autophagy and Cancer



Dr Georg Ramm working with the Titan Krios cryo-EM



A tricolour mitochondrial fusion assay developed by PhD student Ben Padman in the lab

Selected significant publications:

1. Oorschot VMJ, Sztal TE, Bryson-Richardson RJ, **Ramm G**. 2014. Immunocorrelative Light and Electron Microscopy on Tokuyasu Cryosections. *Methods in Cell Biology* 124, 241-257.
2. Padman BS, Bach M, Lucarelli G, Prescott M, **Ramm G**. 2013. The protonophore CCCP interferes with lysosomal degradation of autophagic cargo in yeast and mammalian cells. *Autophagy* 9,1862-75.
3. Bach M, Larance M, James DE, **Ramm G**. 2011. The serine/threonine kinase ULK1 is a target of multiple phosphorylation events. *Biochem J* 440, 283-91.
4. Yip FMF, **Ramm G**, Larance M, Wagner MC, Guilhaus M, James DE. 2008. Phosphorylation of the Myosin Motor Myo1c Is Required For Insulin-Stimulated GLUT4 Translocation in Adipocytes. *Cell Metab* 8, 384-98. (Cover story & Preview *Cell Metab* 8, 344-6).
5. Ng Y, **Ramm G**, Lopez JA, James DE. 2008. Rapid activation of Akt2 is sufficient to stimulate GLUT4 translocation in 3T3-L1 adipocytes. *Cell Metab* 7, 348-56.