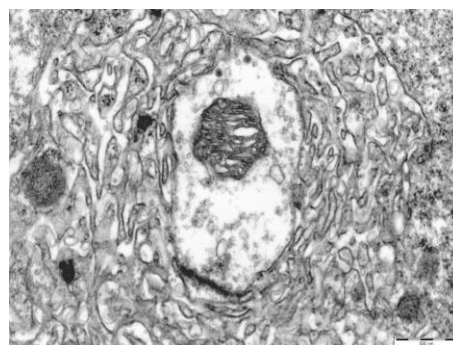
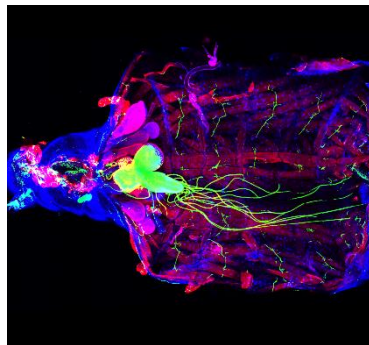
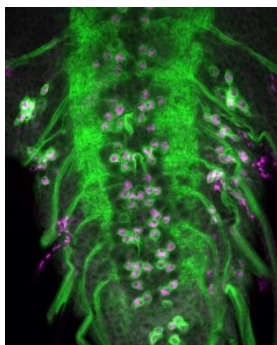


Postdoc position available in mitochondrial stress signalling



Application Deadline: 3rd November 2019

The post-doctoral researcher will use *Drosophila* models to investigate how mitochondrial dysfunction contributes to neurodegeneration. Mitochondrial dysfunction is strongly associated with Alzheimer's disease and other dementias (Hunt & Bateman 2018, *Febs Lett.* PMID 29086414). We identified hypoxia inducible factor-1 alpha as a key transcriptional regulator of the response to mitochondrial dysfunction in *Drosophila* (Cagin et al. 2015, *PNAS*, PMID: 26489648). Our studies have shown a number of stress signalling pathways are active in the *Drosophila* nervous system (Duncan et al. 2018, *PLoS Genetics*, PMID 30059502). Excitingly, we very recently showed that mitochondrial stress causes an increase in the metabolite 2-hydroxyglutarate (2-HG), which promotes neuronal dysfunction in *Drosophila* (Hunt et al. 2019, *J. Cell Biol.*, DOI: 10.1083/jcb.201904148).

The aim of this project is to determine how HIF-1a and 2-HG combine to cause neuronal dysfunction in the *Drosophila* nervous system. The postdoc will use genetic and biochemical methods to determine how 2-HG affects HIF-1a in *Drosophila* neurons. They will also identify genes that are mis-regulated by 2-HG and HIF-1a in neurons using RNA-seq and ChIP-seq. Overall, this project will answer fundamental questions about the response to mitochondrial dysfunction and identify new potential therapeutic strategies for neurological diseases.

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More details on the Bateman lab are here:

<https://www.kcl.ac.uk/ioppn/depts/bcn/Our-research/Neurodegeneration/bateman>

To apply:

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