Power Struggles: How parasite mitochondria control infection in the host

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The apicomplexan mitochondrial electron transport chain (mETC) and ATP synthase are crucial for parasite energy metabolism and survival. They are highly divergent from the mammalian mETC, with parasite-specific subunits that could prove to be valuable drug targets for anti-parasitic treatment.

This project focuses on two of these subunits: the mETC complex IV subunit ApiCox10 and the ATP synthase subunit ATPTG11. Both subunits have a vital role in the formation of higher-order assemblies: Apicox10 is required to form associations between mETC complexes, "Supercomplexes". ATPTG11 is required for ATP synthase hexamerisation in *Toxoplasma gondii*.

Higher complex formation may enhance metabolic efficiency by improving electron diffusion between complexes, protection from reactive oxygen species or provide relevant structural features to the mitochondrial membrane. For example, *T. gondii's* ATP synthase's hexameric structure shapes the cristae of the mitochondria, enhancing ATP synthase metabolism.

Previous studies have shown that knockout of ApiCox10 prevents the formation of the III-IV supercomplex, resulting in minor growth defects in tachyzoites *in vitro*, and that a knockout of ATPTG11, which results in ATP synthase dimers rather than hexamers, shows similar results. However, the extent of the loss of metabolic activity in ATPTG11-KO culture, the effects on bradyzoite formation and health and parasite survival *in vitro* are yet to be explored and will be the focus of this project.

This poster summarises the focus of my first year of this project: The analysis and characterisation of the effects of the removal of higher-order assemblies from *T. gondii*'s mETC on parasite fitness *in vitro*.