The term ‘mitochondrial retrograde signalling’ typically refers to the fact that in many organisms these organelles have been shown to use specific signalling pathways to convey information on their developmental and physiological status to the nucleus and modulate the expression of nuclear genes accordingly. It is not known if similar signalling pathways exist in trypanosomes.

We are using a pleomorphic *Trypanosoma brucei* strain devoid of mitochondrial DNA (akinetoplastic, or AK cells) to investigate the presence of retrograde signalling in these organisms. As *T. brucei* differentiates from the slender bloodstream forms to the procyclic form it undergoes dramatic remodelling of its morphology and metabolism, including mitochondrial activity. Respiratory complexes III (cIII, cytochrome bc_1 complex) and IV (cIV, cytochrome C oxidase), composed of both mitochondrially and nuclearly-encoded subunits, are repressed in slender forms but fully active in procyclic forms. mRNAs for nuclearly encoded cIII and cIV subunits become detectable in transitional stumpy forms and the corresponding proteins and complexes only become detectable after differentiation into PCF forms. Using northern blotting we now show that AK *T. brucei*, lacking the mitochondrial encoded cIII and cIV subunits, still upregulate cIV subunit COX VI mRNA upon differentiation into stumpy forms, potentially indicating a lack of communication between mitochondrion and nucleus. We are currently comparing the global transcriptome of wild type and AK *T. brucei* during differentiation from slender to stumpy forms to obtain a comprehensive view of the potential effects of mitochondrial dysfunction on nuclear gene expression in these parasites.