Apoptosis Inducing Factor (AIF), a nuclear encoded phylogenetically conserved mitochondrial inter-membrane space flavoprotein plays a vital role in caspase independent cell death. In response to cell death stimuli, it undergoes mitochondrio-nuclear translocation resulting in DNA fragmentation. Nevertheless, AIF is also requisite for cell survival, wherein it plays a part in the maintenance of mitochondrial function and structure in an organized manner. Although it performs crucial cellular functions, its underlying mechanism of regulating mitochondrial homeostasis is still not clear. The present study aims to understand the role of AIF in cell survival, growth and development by its down-regulation and overexpression in an interesting unicellular eukaryote, *Dictyostelium discoideum* which shows multicellularity upon starvation. Constitutive *AIF* down-regulated (dR) cells showed slower growth profile and delay in multicellular development. Under oxidative stress, *AIF* dR cells exhibited early mitochondrial membrane depolarization followed by AIF translocation from mitochondria to nucleus and necrotic cell death as compared to paraptoptic cell death of control cells.

Also, *AIF* dR cells demonstrated compromised oxidative phosphorylation along with high intracellular ROS, oxidative DNA damage and calcium levels and lower ATP content. Interestingly, constitutive *AIF* dR cells showed amelioration in cellular growth and in the activity of the ETC complexes upon an antioxidant treatment upon antioxidant treatment, strengthening AIF's role as ROS regulator. Moreover, constitutive *AIF* dR cells manifested reduced transcript levels of the various subunits of ETC. It also affected mtDNA content and mitochondrial fusion-fission mechanism which consequently caused morphometric mitochondrial alterations. Constitutive *AIF* overexpressed (OE) cells also showed higher cellular ROS and mitochondrial fission genes transcript levels along with reduced mitochondrial fusion genes transcript levels and mtDNA content. Thus, the outcomes of the current study provide a paradigm where AIF is implicated in cell survival by maintaining mitochondrial bioenergetics, morphology and fusion-fission mechanism in *D. discoideum*, an evolutionarily significant model organism for mitochondrial diseases.