**Abstract**

The role of secondary redox buffers is masked by the presence of the primary redox buffer (Glutathione) and the genes which can alter the levels of secondary redox buffers are not entirely known. In order to understand the role of NADPH which is a secondary redox buffer, a genetic screen was developed in the lab which can be used to detect changes in NADPH levels. Using this screen, knockout (deletion) and multicopy library approaches were used to identify novel genes that can alter NADPH levels. Through multicopy library approach, ATX2 (antioxidant) was identified that partially suppressed the growth defects of gsh1Δ. RTT01 (regulator of Ty1 transposition) gene was also found as a candidate gene which further needs to be confirmed. Through deletion studies, many genes were identified and one among those was FMP40 (Found in Mitochondrial Proteome) which was a putative protein of unknown function (although now, its function has been delineated). In the previous studies on FMP40 in the lab, it was observed that fmp40Δ was able to partially suppress the growth defects of cells having deletion of genes involved in glutathione biosynthesis (GSH1: gamma glutamylcysteine synthetase), mitochondrial NADPH generation (POS5: peroxide Sensitive) and iron-sulphur clusters transport (GRX5: glutaredoxin) in mitochondria. To get the further insights of FMP40 function, experiments were performed to confirm the localization, observe the effects of oxidative stress and to determine the potential substrates and interactors. All

of these strengthened the hypothesis that FMP40 acts as a consumer of mitochondrial
NAPDH which fits well with the delineated function of FMP40 and direct
interactions of FMP40 with GRX2, GRX5 and TRX3 are being investigated.