6. CONCLUSION AND FUTURE PROSPECTIVE

We characterised *MoHPT1,* the only histidine phosphotransferase in *M. oryzae* B157, by using RNAi silencing technology. We analysed the function of *MoHPT1* in *M. oryzae* under stress conditions as well as light induced condition. Our results indicated that *MoHPT1* is an essential gene in *M. oryzae* B157. Further functional examination showed that *MoHPT1* is required for stress adaptation in response to oxidative stress, osmotic stress and cell wall stress. The results suggest that the stress adaptation to oxidative stress through *MoHPT1* is brought about by signalling of Hog1, while the stress adaptation to cell wall stress is brought about by MPS1 signalling. These observations open up an avenue to explore the mechanism of *MoHPT1* regulation of HOG1 and MPS1 pathway and to explore cross signalling between these two pthways. This study also reveals a novel role for *MoHPT1* in response to light. The existence of dual *MoHPT1* transcripts (of which the novel transcript is induced in presence of light) and the regulation of the PAS domain containing Histidine kinases influenced by *MoHPT1* provide a new area for scientific evaluation as to how and when *MoHPT1* light response is obligatory. Whether *MoHPT1* has any other functions related to circadian rhythms is yet another question to be followed up. The global transcriptome analysis of *MoHPT1* knock-down showed differential expression of genes which are required for *in planta* growth of the fungus. The up-regulated genes were mostly those which are expressed during biotrophic growth and down-regulated genes were mostly those required for necrotrphic growth. This observation suggests that *MoHPT1* has some important role in the transition from biotrophic to necrotrophic growth of the pathogen in the host. How this regulation is brought about by *MoHPT1* provides scope for

further investigation. On the whole this study has provided several valuable new insights into the functional role of *MoHPT1* in stress adaptation and pathogenicity.