



Supplementary Figure 5. Induction of *CTA1* provides acquired resistance to H_2O_2 in *C. glabrata*. To test if pre-inducing *CTA1* is sufficient to provide ASR for H_2O_2 , we replaced the endogenous *CTA1* promoter with the promoter of the *C. glabrata* *MET3* gene. When grown in SC medium lacking methionine and cysteine (-MC), *CTA1* was induced to a comparable level as in the endogenous *CTA1pr-CTA1* strain under phosphate starvation at 45 minutes (A). Dots represent the mean of at least 3 biological replicates, and the error bars the 95% confidence interval by bootstrapping. The line is the LOESS fit to the data. The endogenous *CTA1pr-CTA1* has a basal expression level that is higher than the *MET3pr-CTA1* (0 min). We also confirmed that the -MC media itself did not provide ASR in the wild type *C. glabrata* cells (B, top). Note that in this set of ASR experiments, all SC medium containing H_2O_2 also lacked methionine and cysteine (C). This allows the *MET3pr-CTA1* to be induced during the secondary stress, mimicking what the wild type strain experiences during the H_2O_2 stress. Using this strain, we found that inducing *CTA1* during the primary stress significantly enhanced the survival of *C. glabrata* cells during the secondary oxidative stress, i.e., providing ASR (B, bottom two rows, -MC vs Mock).