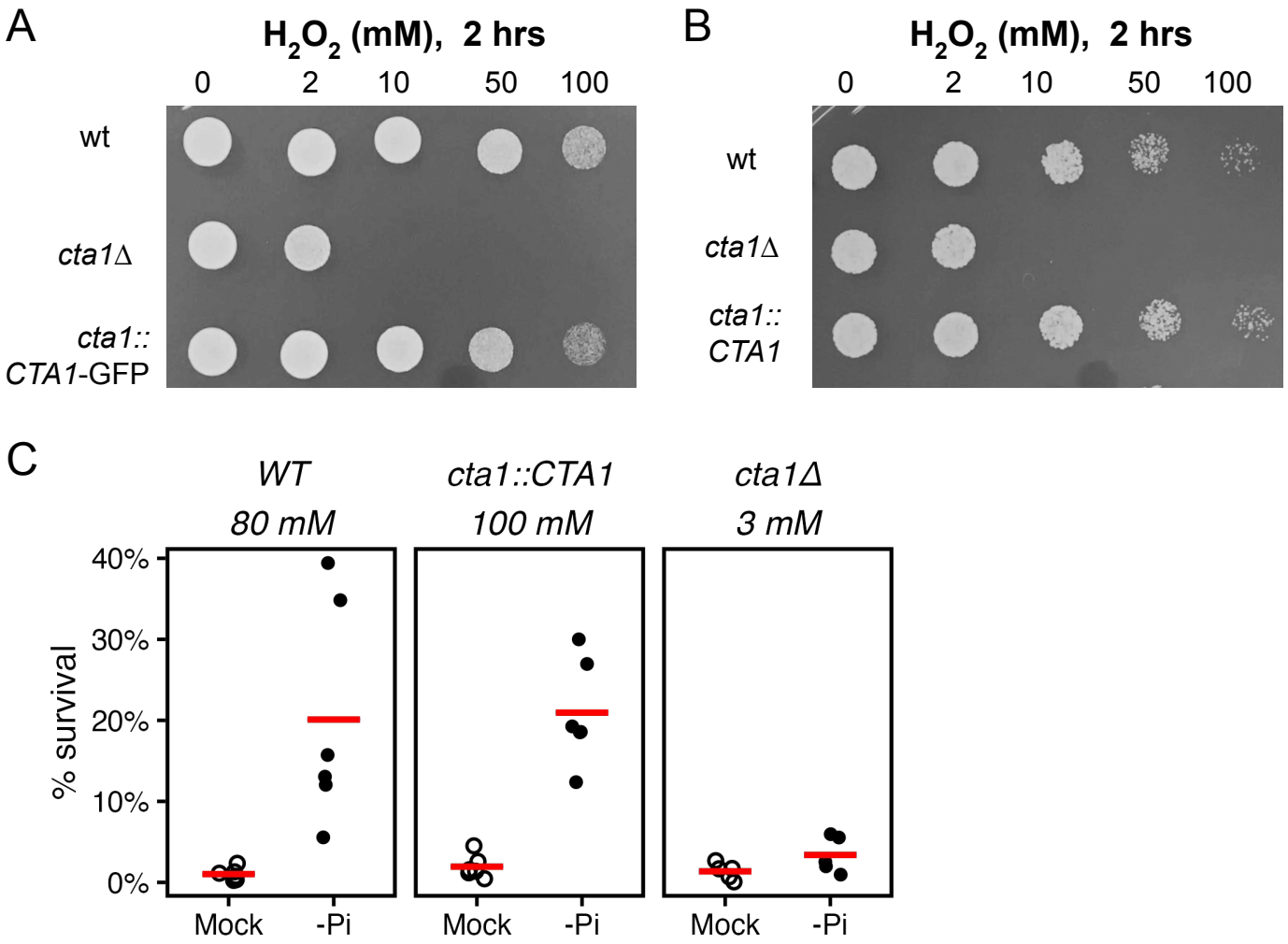

Supplementary Figure 4. *CTA1* complement strain rescues basal H_2O_2 survival and ASR defects in *cta1*Δ. Putting either *CTA1*-GFP (A) or the untagged *CTA1* (B) back to the endogenous locus in the *cta1*Δ background restored the resistance to H_2O_2 compared with the wild type strain. Each strain was treated at the indicated concentrations of H_2O_2 for 2 hours, then spotted onto YPD plates and incubated at 30°C for 48 hours (A) and 20hrs (B). (C) ASR in the wild type, *CTA1* complement (*cta1::CTA1*) and *cta1*Δ strains. The experiment was conducted similarly as in Fig. 3 for wild type and *cta1*Δ strains. Concentrations of H_2O_2 were calibrated to achieve a similar basal survival rates (open circles, Kruskal-Wallis rank sum test for differences among the three groups P = 0.38). ASR-scores for the three strains are (with 95% CI and Wilcoxon signed-rank test P-values in the parenthesis): wild type 23.9 ([14.9, 36.5], P=0.048); *cta1::CTA1* 15.4 ([9.5, 21.3], P=0.048); *cta1*Δ 5.1 ([2.0, 10.6], P=0.093). The difference in ASR-score is significant between *cta1*Δ and wild type, but not between *cta1::CTA1* and wild type (Mann-Whitney U test P = 0.034 and 0.48, respectively, Bonferroni-corrected).