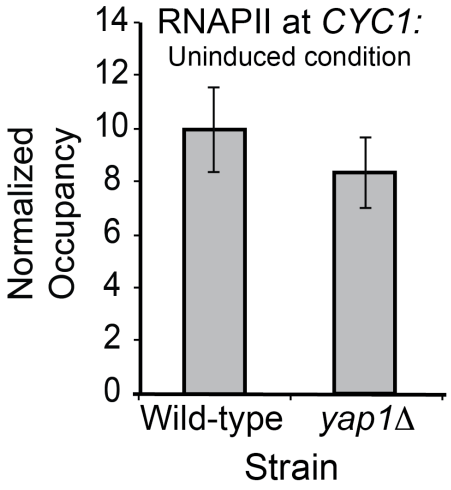
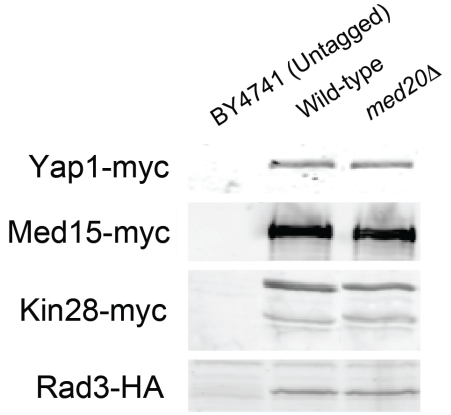
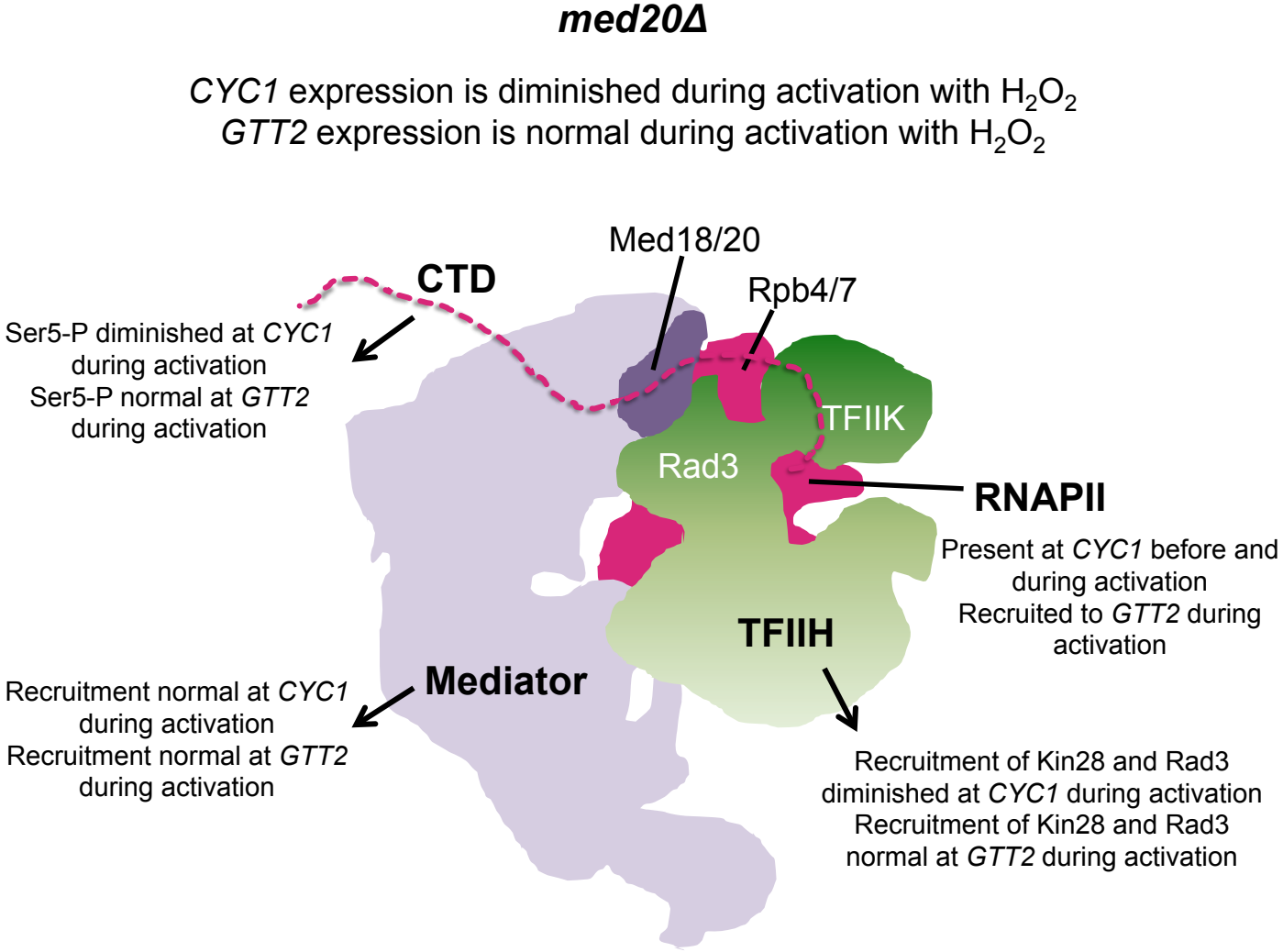
Supplemental Figures S1-S3 for Lee, et al. **The head module of Mediator directs activation of preloaded RNAPII in vivo**



Supplementary Figure S1. Yap1 is not required for preloading the *CYC1* promoter. Occupancy of RNAPII during growth in YP-Glucose (uninduced condition) in the wild-type and *yap1*Δ strains. Occupancy at a region proximal to the telomere on chromosome VI was subtracted from the occupancy at *CYC1* and the occupancy in the wild-type strain was set to 10. Bars represent the average ±SD of three samples processed independently.



Supplementary Figure S2. Expression of tagged derivatives in the wild-type and *med20*Δ strain backgrounds. Cell lysates from an untagged strain (BY4741), Yap1-myc, Med15-myc, Kin28-myc, and Rad3-HA in the wild-type and *med20*Δ strains were separated on a 10% gel. The blot was probed with anti-myc or anti-HA antibodies, and bands were detected with the Odyssey Infrared Imaging System (LI-COR). The *med20*Δ cell lysate was run on the same blot as the untagged and wild-type samples.



Supplementary Figure S3. Summary of the results for Mediator functions on activation of *CYC1* and *GTT2* in the *med20*Δ strain. In a wild-type strain, H_2O_2 induces both *CYC1* (preloaded) and *GTT2* (recruitment) expression**.** The Med18/20 heterodimer (dark purple) sits positioned near the Kin28-containing module (TFIIK) and Rad3 subunit of the TFIIH general transcription factor (green), and the Rpb4/7 heterodimer of RNAPII (pink). The putative location of the CTD is shown as a dashed pink line. In the absence of Med18/20 or Med19 (not shown due to unknown location), there is diminished occupancy and activity of TFIIH at *CYC1*, perhaps due to a change in the conformation of RNAPII, rendering binding of TFIIH less efficient. Removal of these subunits may also change the way the CTD is presented to the kinase module, resulting in decreased phosphorylation. The figure is based on structural data from references 61, 71, and 72.