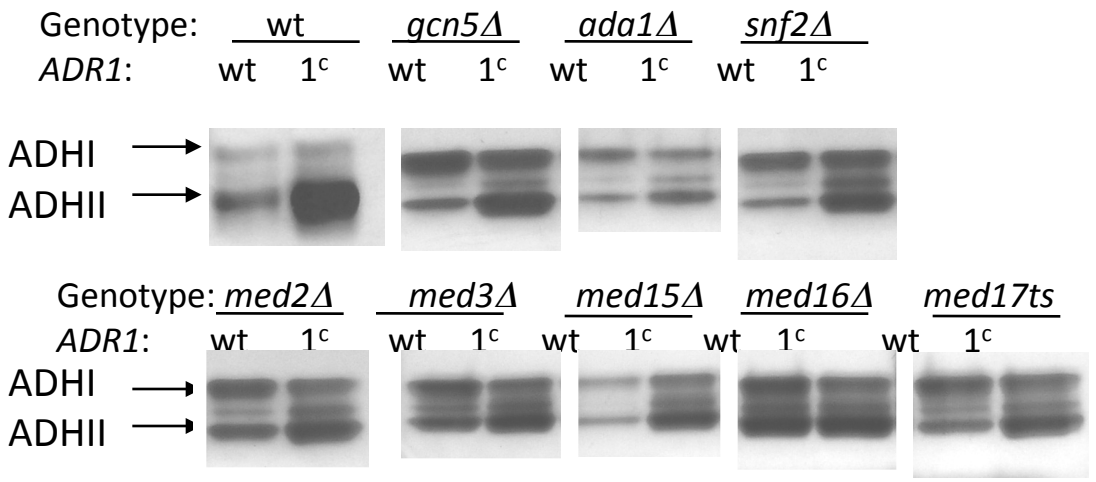
Supplementary Figure 1. Adr1^{c} partially suppresses defective derepression of *ADH2* in coactivator mutants.



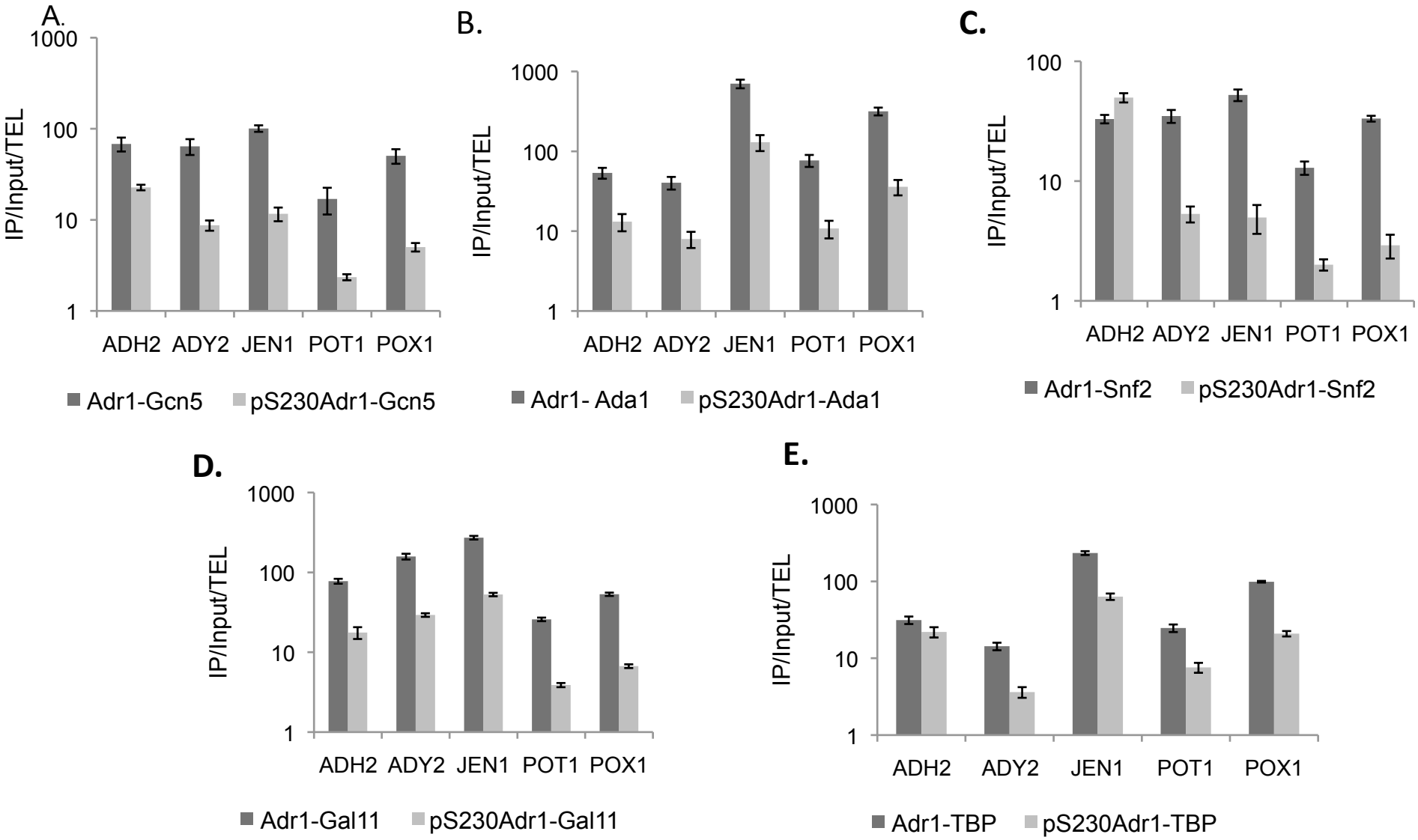
Legend to Supplementary Figure 1. **Adr1^{c} partially suppresses defective derepression of ADH2 in coactivator mutants.**

ADH in situ enzyme assays were performed as described in Dombek et al. (1997). ADHI is the constitutive isozyme of alcohol dehydrogenase. ADHII is the glucose-repressible isozyme. Cultures were grown in glucose and then shifted to low glucose (0.05%) for six hours. Cell extracts were prepared by vortexing with glass beads and 20 µg of protein was analyzed on a 6% non-denaturing PAGE. Wild-type is CKY13 (*adr1Δ::kanMX*).

Wild-type Adr1(wt) was expressed from pKD16 and Adr1^{c} (1^{c}; the S230A allele) from pKD14. The coactivator mutants of CKY13 are listed in Table 1.

Legend to Supplementary Figure 2. **Coactivator and polII recruitment by Adr1-HA (total Adr1) and Ser230-phosphorylated Adr1.** Recruitment of coactivator components was measured by sequential ChIP-qPCR in strains deleted for *ADR1* and carrying plasmid pKD16HA. Myc-tagged coactivator strains are listed in Table 1. Samples were collected under derepressing (DR; 3% glycerol) conditions and divided into two portions after breaking the cells. The first immunoprecipitations were for total Adr1 (using anti-HA antibody) and pSer230-phosphorylated Adr1 using anti-pSer230 antibody). In the second immunoprecipitation the recruitment of SAGA components Gcn5 (A) and Ada1 (B), Swi/Snf component Snf2 (C), Mediator component Gal11 (D), TATA binding protein (TBP; E), polII (F) and polII with CTD phosphorylated at Ser5 (G) was determined using specific antibodies. Error bars indicate standard deviations of two technical replicates. The experiment was repeated and similar results obtained.

Supplementary Figure 2. Coactivator and polII recruitment by Adr1 phosphorylated at Ser230.



Supplementary Figure 2. Coactivator and polII recruitment by Adr1 phosphorylated at Ser230.