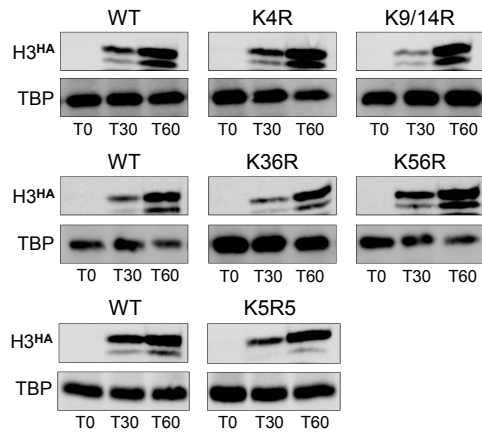
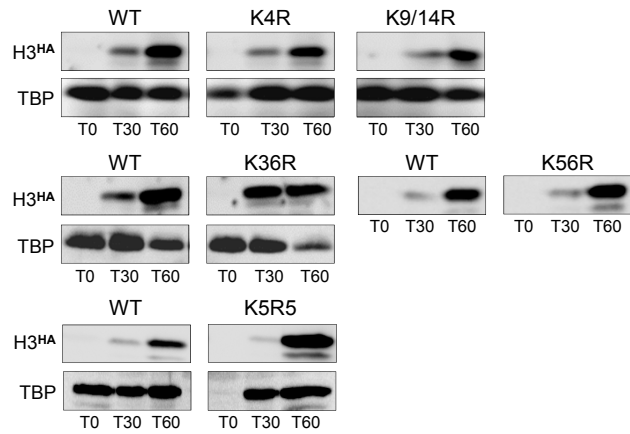


Western blots for Figure 1

First experiment

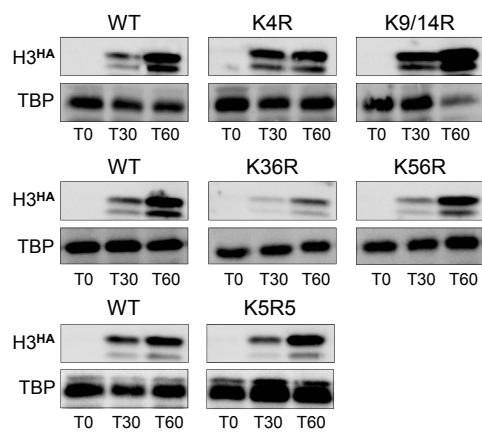


Second experiment

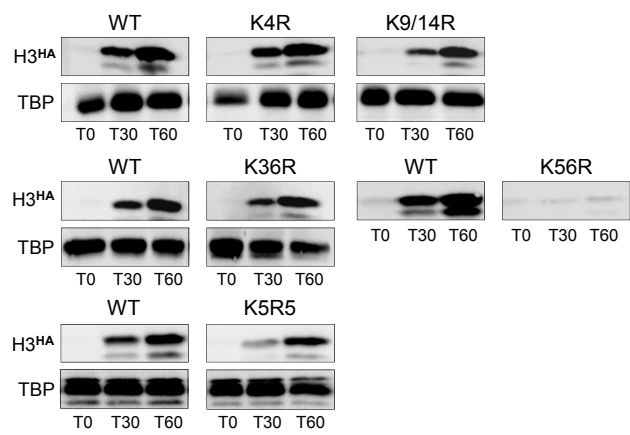


Western blots for Figure 2

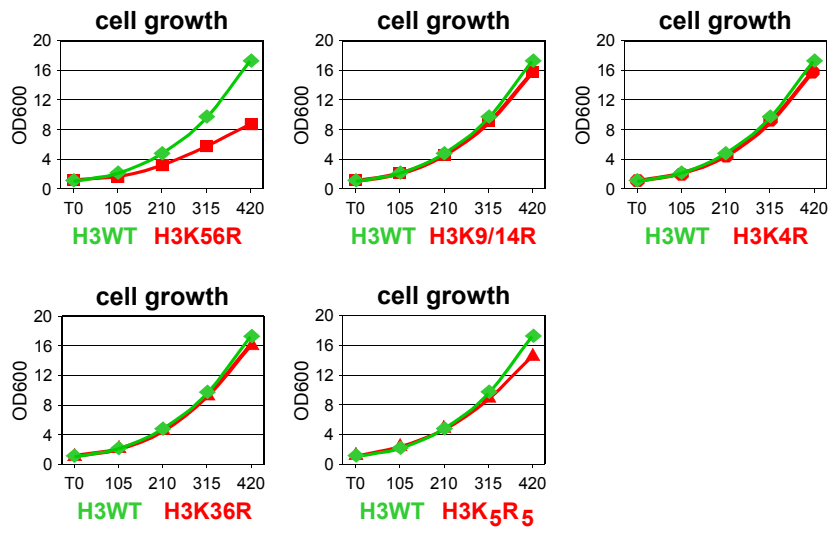
First experiment



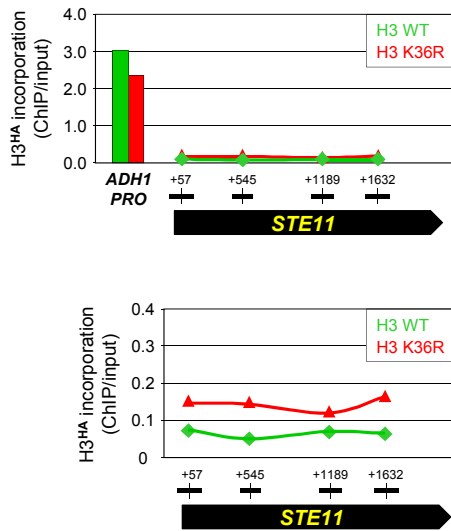
Second experiment

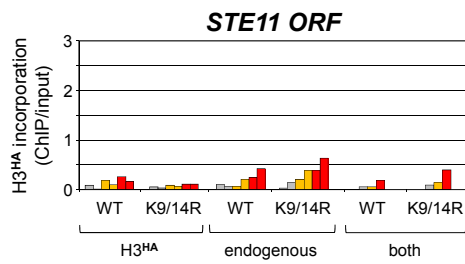
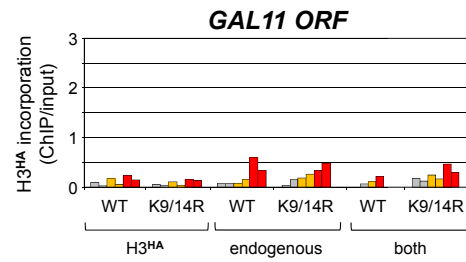
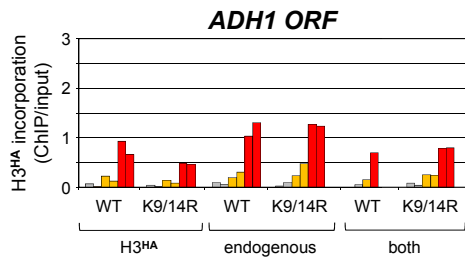
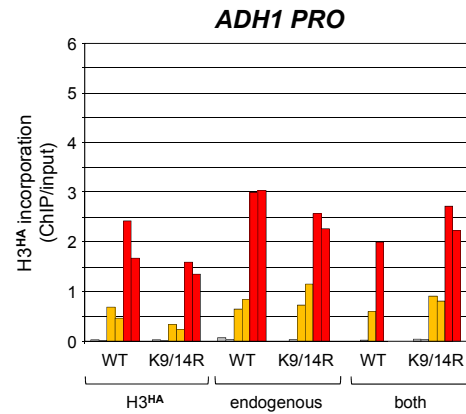
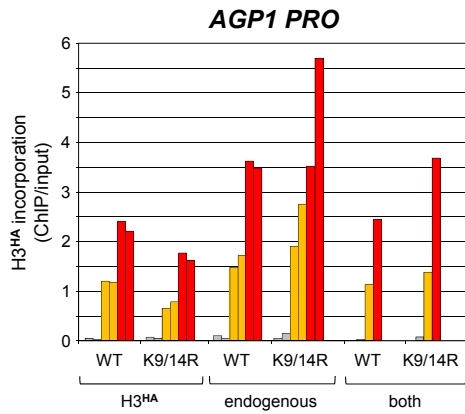
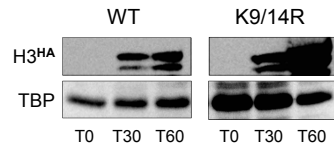
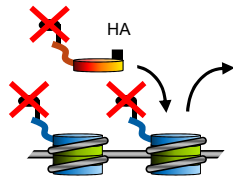


A



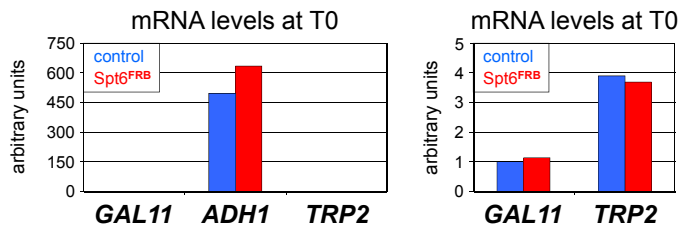
B



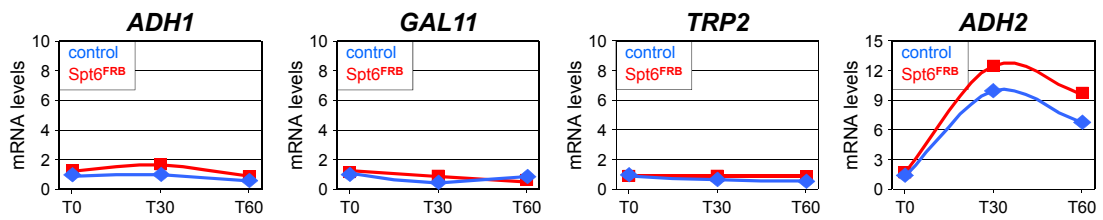




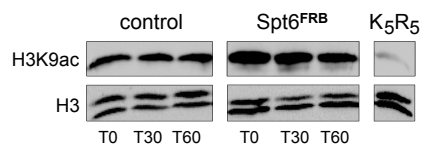
A



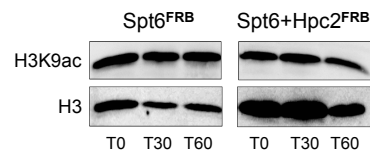
B



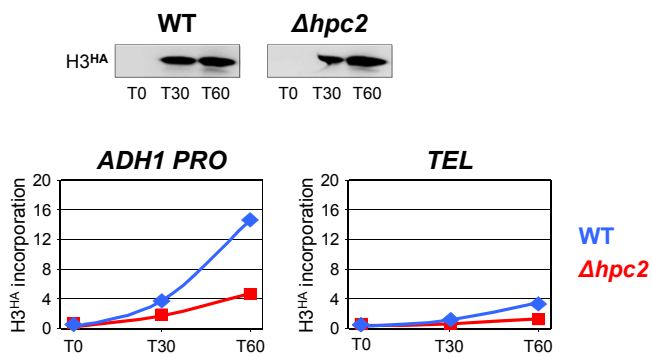
C

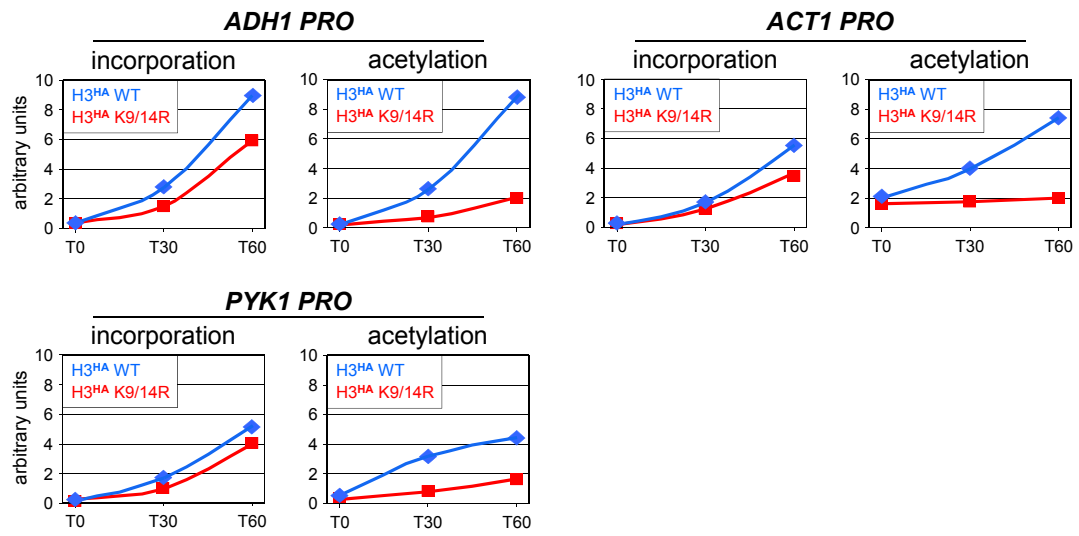


D



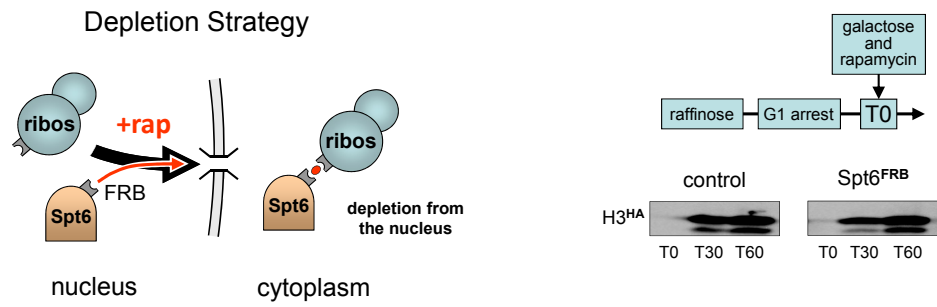
E



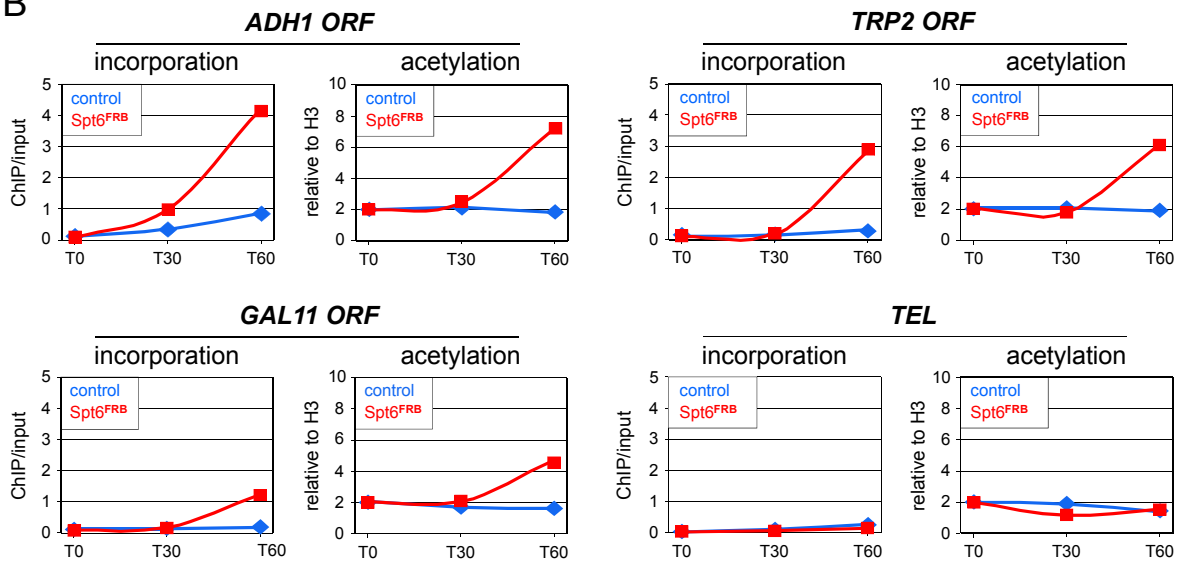


Duplicate of Figure 3

A

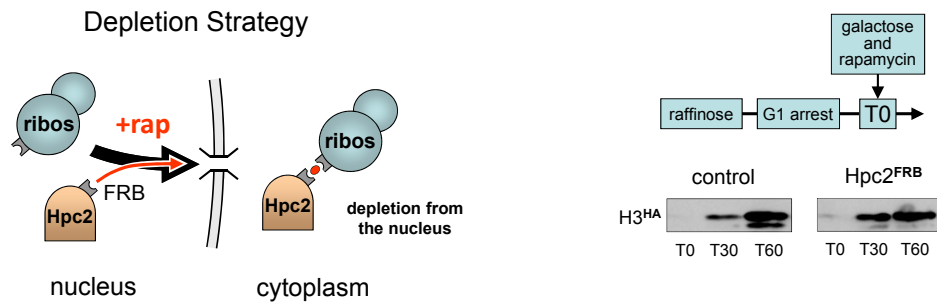


B

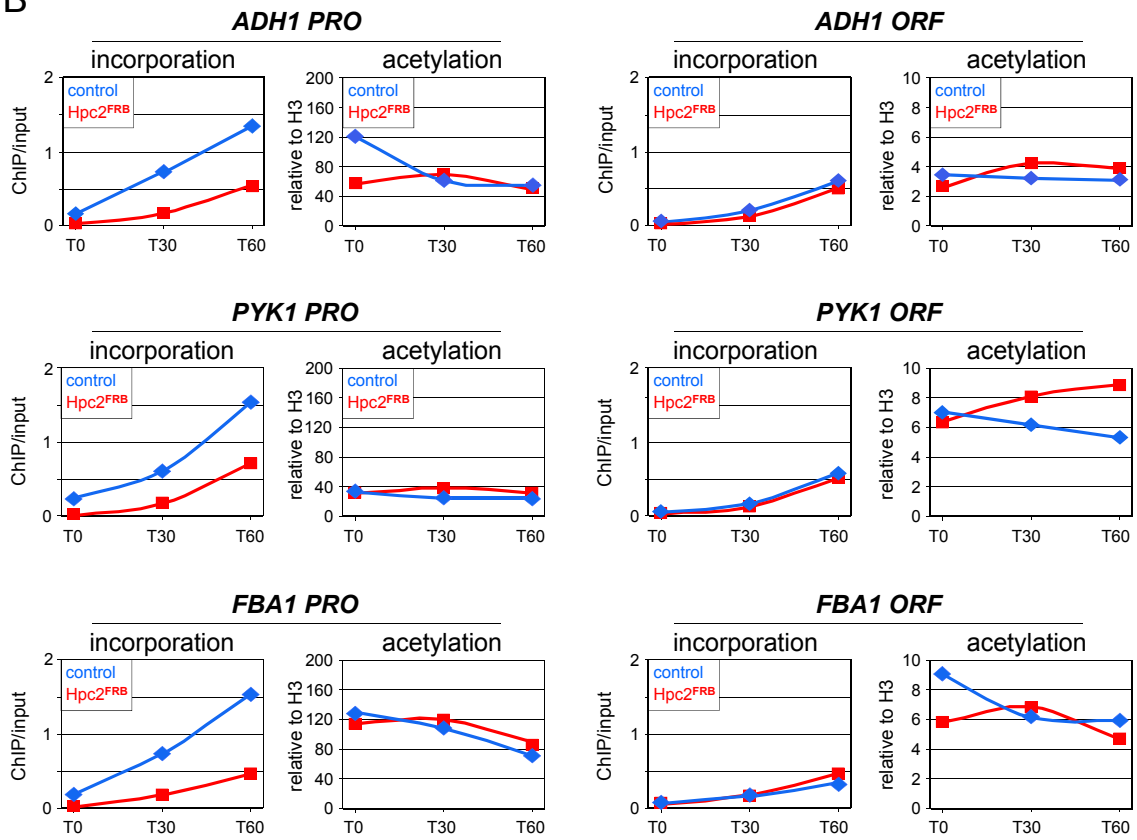


Duplicate of Figure 4

A

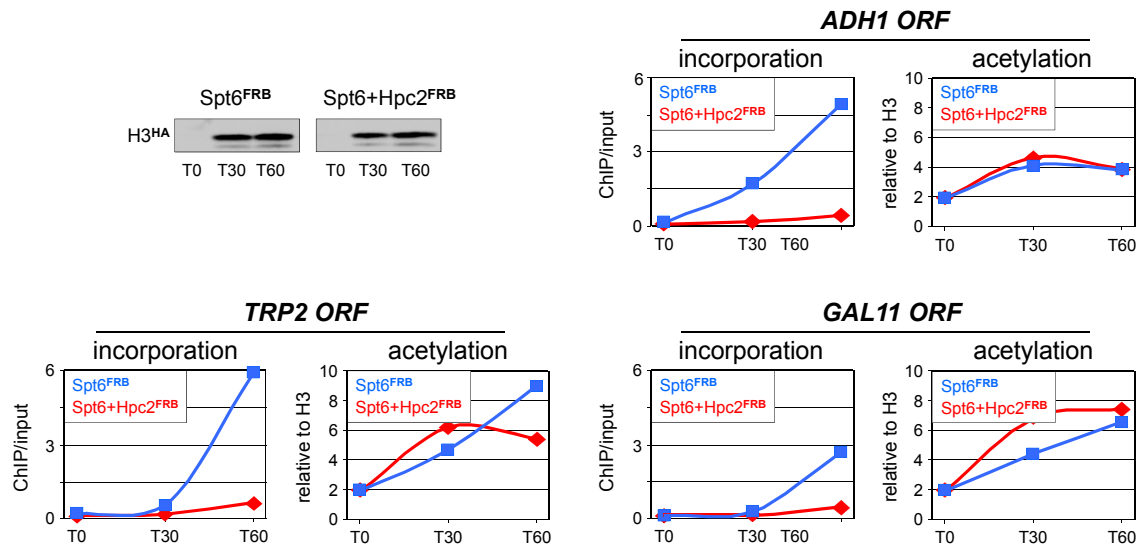


B

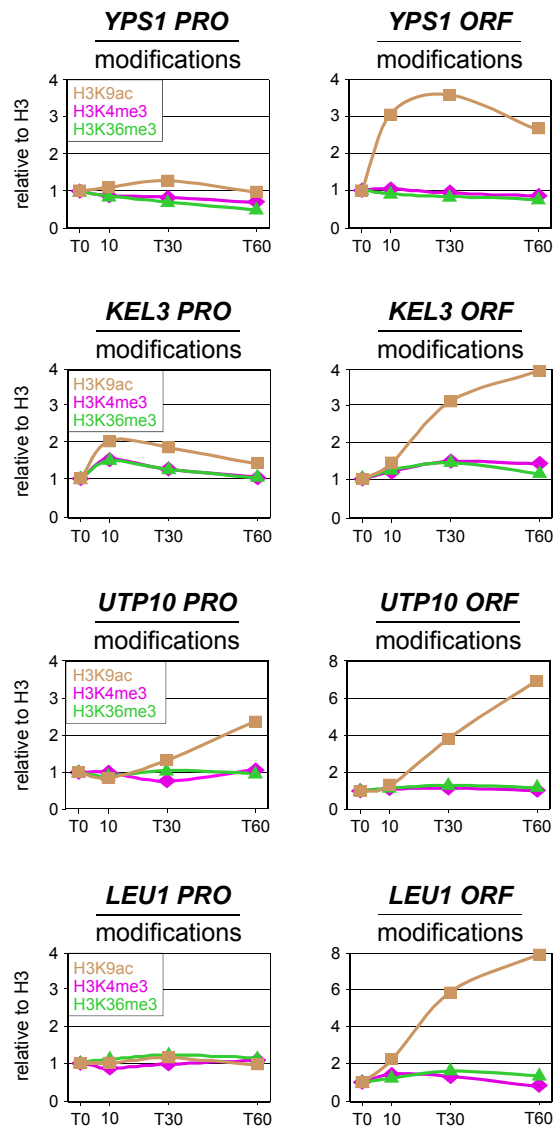


Duplicate of Figure 5





Duplicate of Figure 6



Duplicate of Figure 7

## Supplementary Figure Legends

**Figure S1.** Western blot analysis using anti-HA antibodies to control for galactose activation of the H3<sup>HA</sup> proteins in Figures 1 and 2. TBP serves as a loading control. Note that the grouped panels are from the same gel and exposure for each protein and are directly comparable.

**Figure S2.** Cell growth and turnover of histone H3 mutants.

(A) Growth rates measured by OD<sub>600</sub> of the yeast strains expressing the indicated modification mutants as the sole source of histone H3. The values are relative to those measured at T0 for each strain (ranging from OD<sub>600</sub> = 0.04 to 0.05), which was given a value of 1.

(B) H3<sup>HA</sup> incorporation in a wild-type (WT) or H3K36R chromatin background was measured at 60 min after galactose induction over the *STE11* coding region reported to show increased histone H3K56 acetylation, a mark of newly deposited histones, in a strain deleted for the Set2 enzyme that methylates H3 at residue K36 (28). The midpoints of the amplicons relative to the initiator ATG are indicated on the locus map below the graphs. The arrow indicates the direction of transcription. The results for *ADHI PRO* in the upper panel are from Figure 2 and serve as a comparison. Note the different scales in the two panels.

**Figure S3.** Histone turnover in a K9/14R double mutant background.

Same experiment as in Figure 2 but with both the HA-tagged ( $H3^{HA}$ ) and the chromatin histones carrying a K9/14R mutation (both). The results with only the HA-tagged ( $H3^{HA}$ ) or the chromosomal (endogenous) histone mutated are from Figures 1 and 2 and serve as a comparison.

**Figure S4.** Histone turnover in a K56R double mutant background.

Same experiment as in Figure 2 but with both the HA-tagged ( $H3^{HA}$ ) and the chromatin histones carrying a K56R mutation (both). The results with only the HA-tagged ( $H3^{HA}$ ) or the chromosomal (endogenous) histone mutated are from Figures 1 and 2 and serve as a comparison.

**Figure S5.** mRNA levels, total H3K9ac levels, and  $H3^{HA}$  incorporation in the absence of histone chaperones.

(A) The relative mRNA levels for the indicated genes were measured at T0 in the Spt6-anchor-away strain ( $Spt6^{FRB}$ , red bars) and isogenic control strain (control, blue bars) under the same experimental conditions as described in Figure 4. Quantification was by oligo-dT primed real-time RT-PCR analysis using total RNA prepared by the hot-phenol extraction method. The RT-PCR signals were normalized to the signal obtained for the 18S rRNA in each strain to correct for variation between samples. Results are relative to those for *GAL11* in the control strain, which was arbitrarily set to 1.

(B) Relative mRNA levels during the time course experiment shown in Figure 4. The values at T0 in the control strain were set to 1 for each locus to facilitate comparison. *ADH2* is included as an example of a gene showing increased expression in these experiments.

(C and D) Western blot analysis of global H3K9 acetylation in the time course experiments presented in Figures 4 and 6. A strain expressing the K<sub>5</sub>R<sub>5</sub> acetylation mutant as sole source of histone H3 was used as a control. Note that the grouped panels are from the same gel and exposure for each protein and are directly comparable.

(E) Histone H3 incorporation in the absence of histone chaperone Hpc2. The time course of H3<sup>HA</sup> incorporation after galactose induction at T0 was monitored at the indicated chromosomal locations in exponentially growing wild-type cells (blue lines) and in cells lacking Hpc2 (red lines; kindly provided by Françoise Stutz, Geneva University). Shown on top is a Western blot analysis of H3<sup>HA</sup> expression. The two panels are from the same gel exposure and are directly comparable. Note the marked decrease in H3<sup>HA</sup> incorporation at the *ADHI PRO* (left panel) in the mutant.