**Supp. Figure 1. The enhancement of *spn1-K192N* by *JHD2* overexpression is reverted by a catalytically inactive histidine-427 to alanine mutation in Jhd2.**

Plate spot assays (as described in Figure 1) were used to compare the growth of the indicated strains on synthetic media with 2% dextrose (DEX) or 2% galactose (GAL). *PGAL-JHD2* is used to replace the endogenous *JHD2* locus so that cells grown in galactose overexpress *JHD2* and cells grown without galactose do not express JHD2. A H427A mutation was introduced into *PGAL-JHD2* which disrupts its histone demethylase activity. (A) Genetic interactions of *set2∆* and *PGAL-JHD2(H427A)* with *spn1-K192N* on GAL. (B) Genetic interactions of *set2∆* and *PGAL-JHD2(H427A)* with *spn1-K192N* on DEX.

**Supp. Figure 2. The suppression by *eaf3∆* is not due to loss of the NuA4 histone acetyltransferase complex.**

Plate spot assays (as described in Figure 1) were used to compare the growth of the indicated strains. Genetic interactions of *eaf7∆* and *jhd2∆* with *spn1-K192N*.

**Supp. Figure 3. The enhancement of *spn1-K192N* temperature sensitivity by *rph1∆* is reverted by *rco1∆.***

Plate spot assays (as described in Figure 1) were used to compare the growth of the indicated strains. Genetic interactions of *rco1∆* and *rph1∆* with *spn1-K192N*.