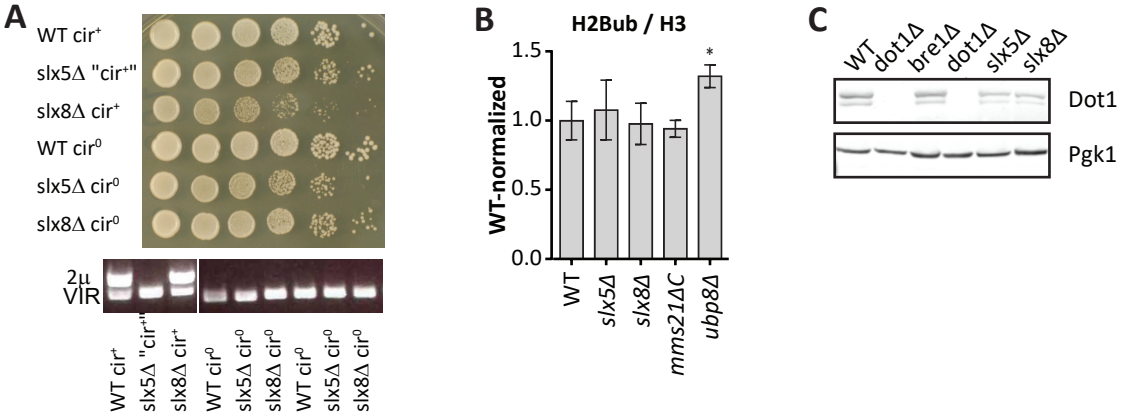

Figure 4 -figure supplement 2. Excluding mechanisms of H3K79me regulation by Slx5, Slx8 and Mms21.
A) Growth assay after serial dilution on solid media of strains before and after removal of the (endogenous) 2µ plasmid. PCR shows that the plasmid was indeed lost after curing, and that in this case the plasmid had already spontaneously been lost in the *slx5Δ* strain. Removal of the over-replicated 2µ plasmid relieves the growth defect in *slx5Δ* and *slx8Δ* strains, as described previously (Burgess et al., 2007). B) Mini Epi-ID experiment for ubiquitinated H2B on the indicated strains. H2Bub/H3 was normalized to wild-type. 3-9 replicates each. Comparison with wild-type was done using one-way ANOVA, *ubp8Δ* is shown for comparison. WT and *ubp8Δ* data is the same as in Figure 2G. H2Bub level at the UpTag was unaltered in the SUMO-ubiquitin pathway mutants, suggesting that H3K79me regulation occurs through another mechanism. C) Immunoblot analysis of Dot1 levels in the indicated strains, Pgk1 serves as a loading control. Slx5 and Slx8 do not regulate Dot1 expression level, suggesting that H3K79me regulation occurs through another mechanism.