Supplemental Figures and Text

Figure S1

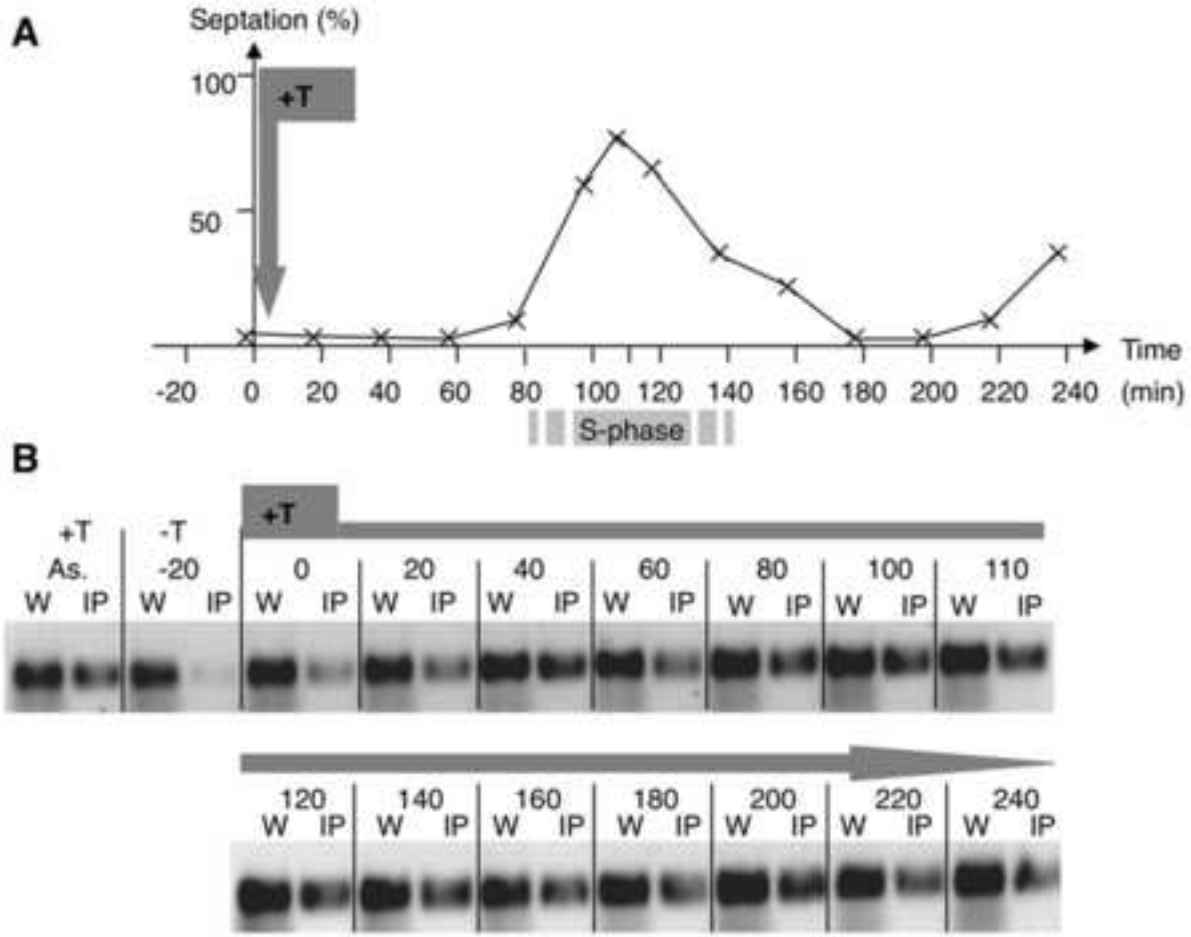

Figure S1. Lsd1 is enriched at *MPS1* independently of the cell cycle progression.

(A) Septation index of the synchronized *cdc25-11* conditional mutant expressing Lsd1-HA in the MT inducible background. Septation correlates with the S-phase period. At t=0 the cells are synchronously released into the cell cycle and thiamine is added (+T).
(B) The cells are grown in the absence of thiamine forcing transcription through the *MPS1* region to maintain a low level of imprint. The cells are blocked at the G2/M transition at 36°C for 3 hours and release into the cell cycle at 26°C. Then thiamine is introduced into the medium to allow imprinting establishment. Fractions of the cell population are taken every 20 min for ChIP analysis. +T and -T indicate the presence of thiamine into the medium. As: correspond to asynchronous cell population. W and IP indicate the DNA prepared from the whole cell extract and the Lsd1-HA IP material. In these experimental conditions, the imprint appears on the upper strand at *mat1* during S-phase (Holmes et al., 2005). Two independent experiments provided similar results, showing that transcription through *MPS1* is incompatible with Lsd1 binding and upon the addition of thiamine Lsd1 is rapidly recruited at *mat1* and remains stably associated with *mat1*, before Swi1 enrichment at *mat1* during the first replication (Holmes et al., 2005).

Figure S2

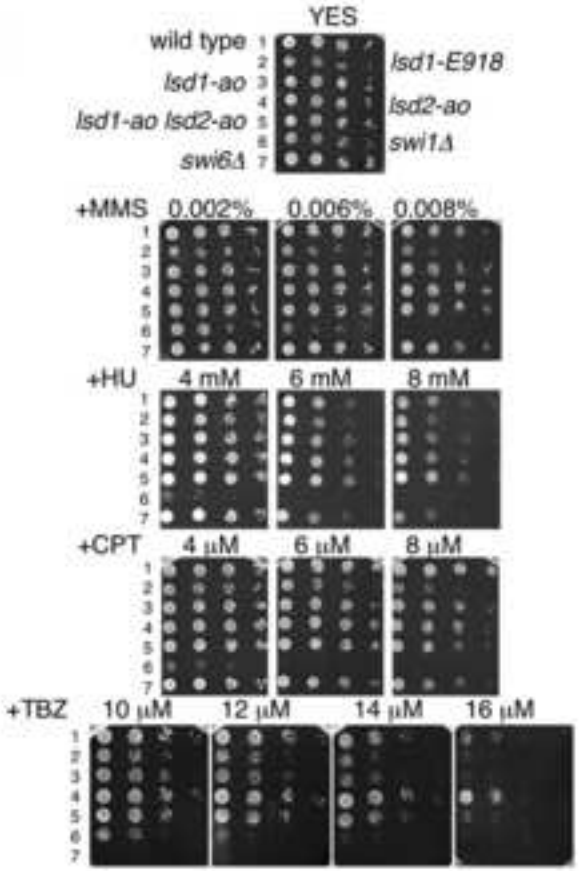

Figure S2: Unprogrammed replication fork blocks are not controlled by Lsd1/2.
In contrast to *swi1*, *lsd1* and *lsd2* functions are not required to promote cell survival in response to replication slow down induced by methyl methansulfonate (MMS), hydroxyurea (HU)-mediated depletion of dNTPs and camptothecin that covalently traps the topoisomerase I on DNA (CPT) together with the tubulin destabilizing drug, thiabendazole (TBZ). Very little or no effect was observed for the cells harboring *lsd1-E918* or amine oxidase mutations, as compared to the wild type and *swi1Δ* strains using these drugs, indicating that Lsd1 and Lsd2 amine oxidase activities, while required for Swi1/3 function at *MPS1*, are not required for Swi1/3 function at unprogrammed stalled replication forks. Interestingly, we found in the presence of TBZ, that lsd1-ao appears sensitive, although less than *swi6Δ* and *swi1Δ*, whereas *lsd2-ao* appears hyper-resistant and the double *lsd1-ao lsd2-ao* mutant appears similar to the wild type strain.
The strains used are indicated in the absence of drugs (YES) and are in the same order for each plate. All spot assays for the wild type and the mutant strains are indicated (serial dilutions: 300, 100, 30 and 10 cells).

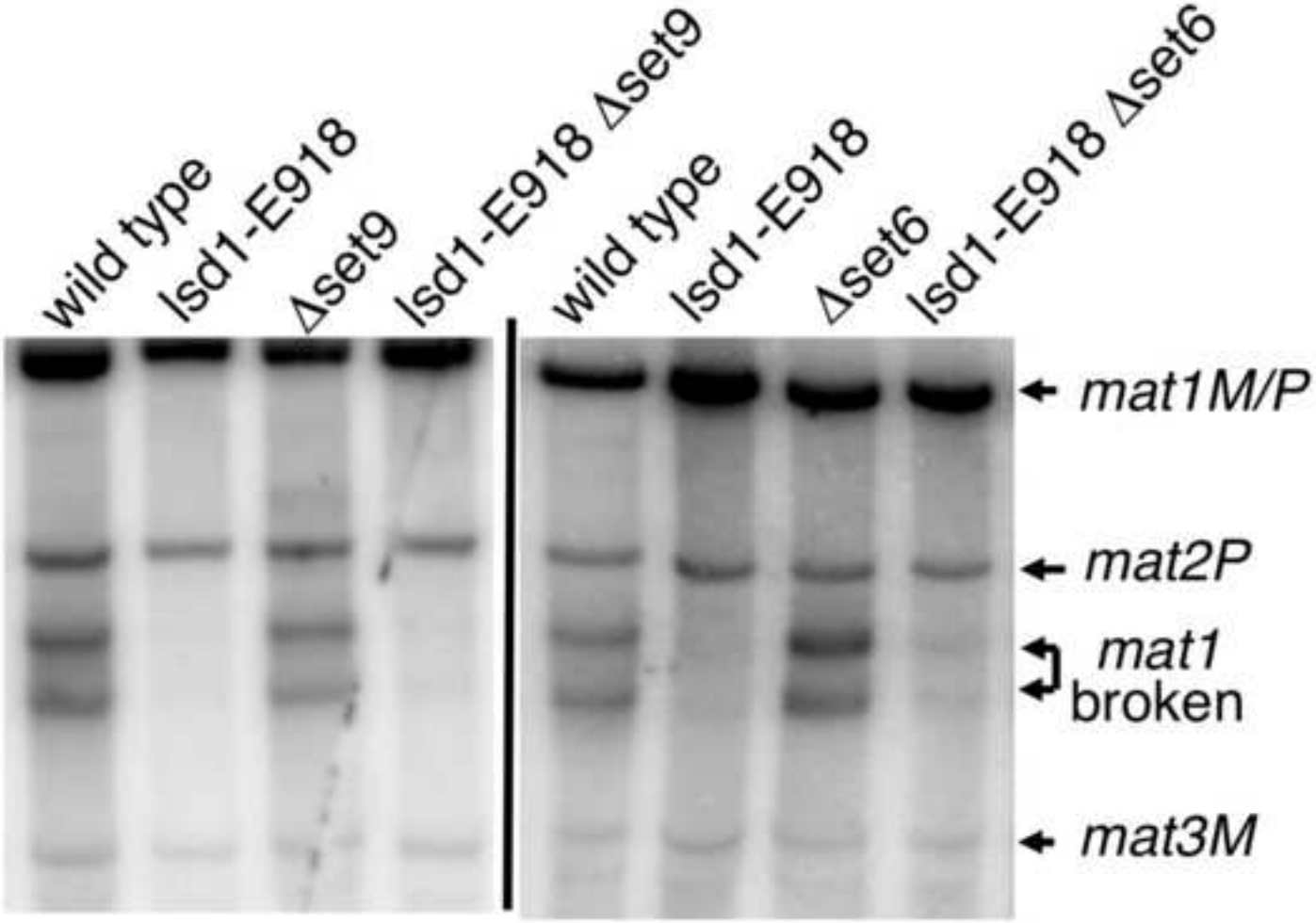

Figure S3: Imprinting levels in *lsd1-E918* and *methyltransferase* single and double mutants

Analysis of the DSB at *mat1*. The genomic DNA is analyzed by *Southern* blot, as in Figure 3. The relevant genotype of the strains, the sizes and identities of the DNA fragments are indicated.

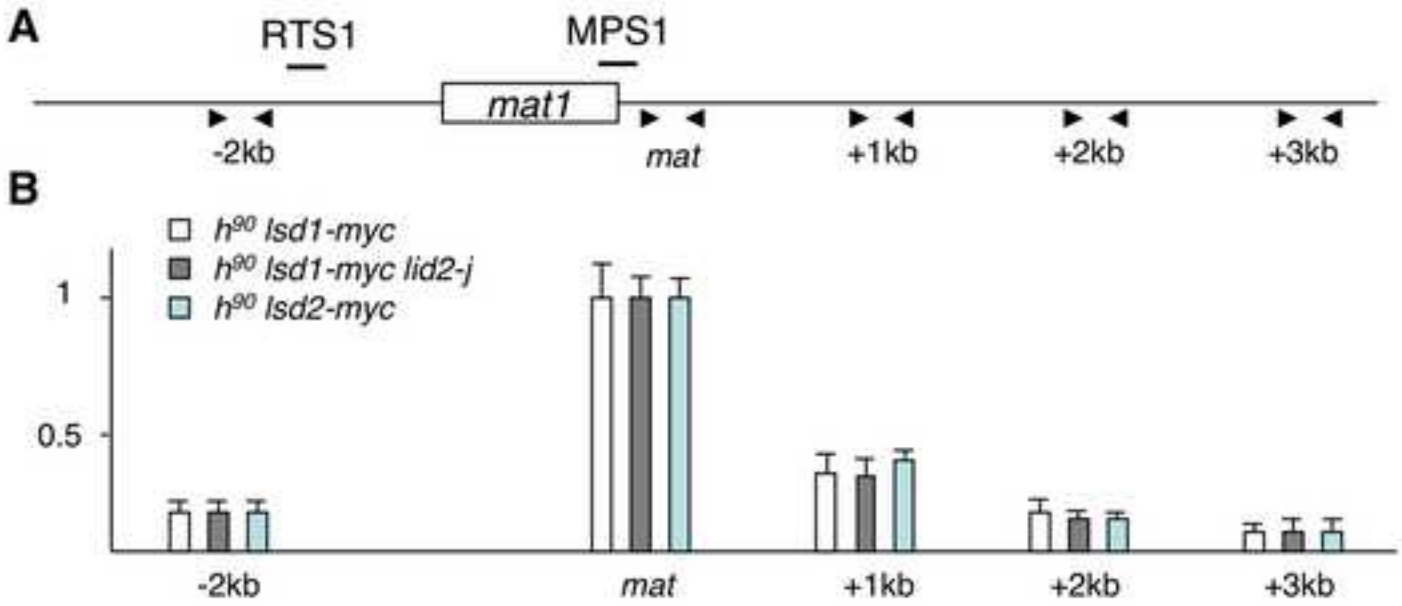

Figure S4: Lid2 is not required for Lsd1/2 recruitment at *mat1*

(**A**) Schematic representation of the *mat1* region. 5 pairs of PCR primers used in a ChIP assay are shown, along with *RTS1* and *MPS1*.
(**B**) Comparative quantification of Lsd1-myc and Lsd2-myc enrichment in wild-type and lid2-j mutant strains, by ChIP analyzed by qPCR.
The level of PCR amplification is set arbitrarily to 1 at *mat1*, showing that Lsd1 and Lsd2 are similarly enriched at *mat1*, independently of Lid2 activity.