GENETICS

Supporting Information
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The Dot1 Histone Methyltransferase and the Rad9 Checkpoint Adaptor Contribute to Cohesin-Dependent Double-Strand Break Repair by Sister Chromatid Recombination in *Saccharomyces cerevisiae*

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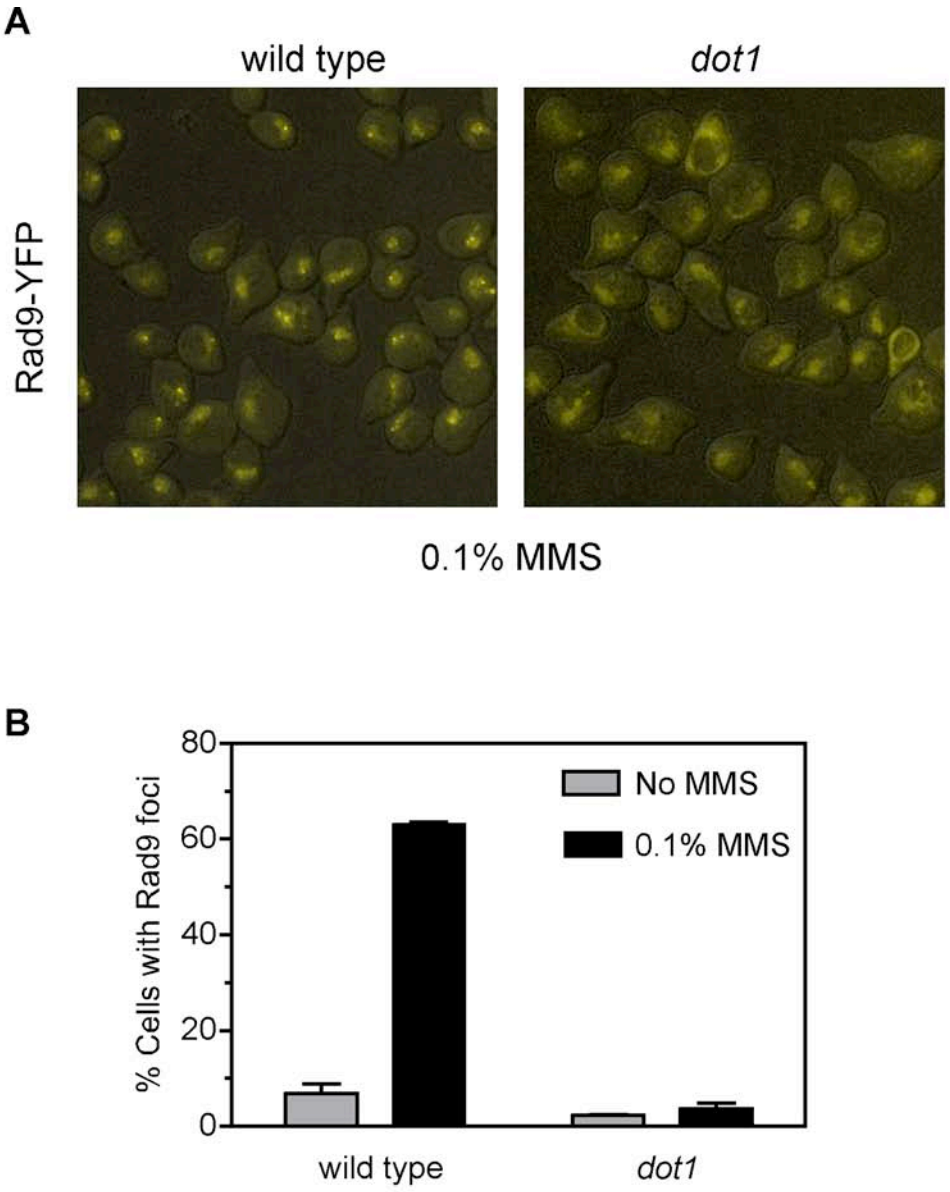

FIGURE S1.—Formation of MMS-induced Rad9 foci is impaired in the *dot1* mutant. (A) Deconvoluted fluorescence microscopy images of Rad9-YFP foci in wild type (W4638-2C) and *dot1* (YP759) cells arrested in G1 with α-factor and treated with 0.1% MMS for one hour. To outline the contour of the cells, an overlay of the Differential Interference Contrast (DIC) image with 10% transparency over the YFP image is shown. (B) The percentage of cells containing Rad9-YFP foci before and after MMS treatment is represented. Average and standard deviation of two independent experiments are shown. Between 300-500 cells were scored for each strain in each experiment.

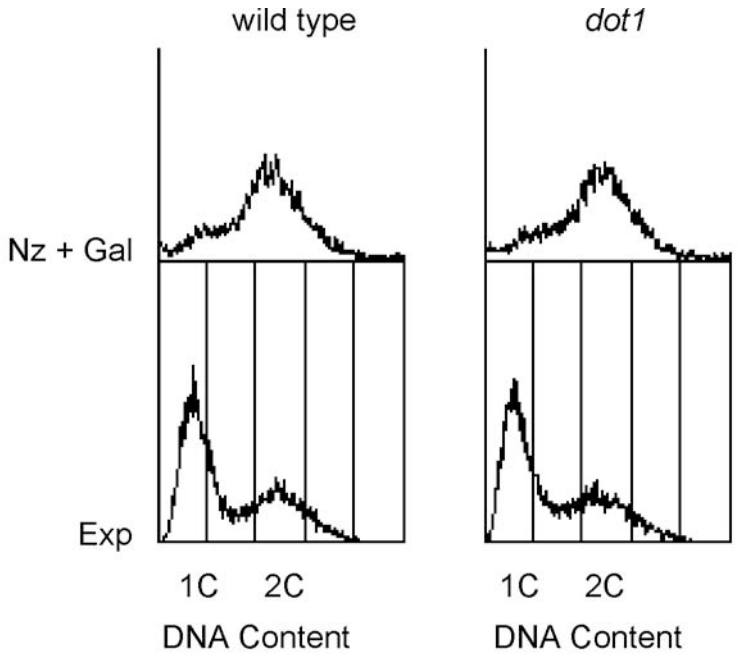

FIGURE S2.—FACS analysis of the DNA content of wild-type (CCG2876) and *dot1* (YP960) cells incubated under the conditions described in Figure 6 for ChIP analysis of Scc1 recruitment to the regions flanking a DSB at *MAT*. Exp: cells growing exponentially in glycerol-lactate. Nz + Gal: nocodazole-treated cells incubated in galactose for 4 hours to induce the HO cut.

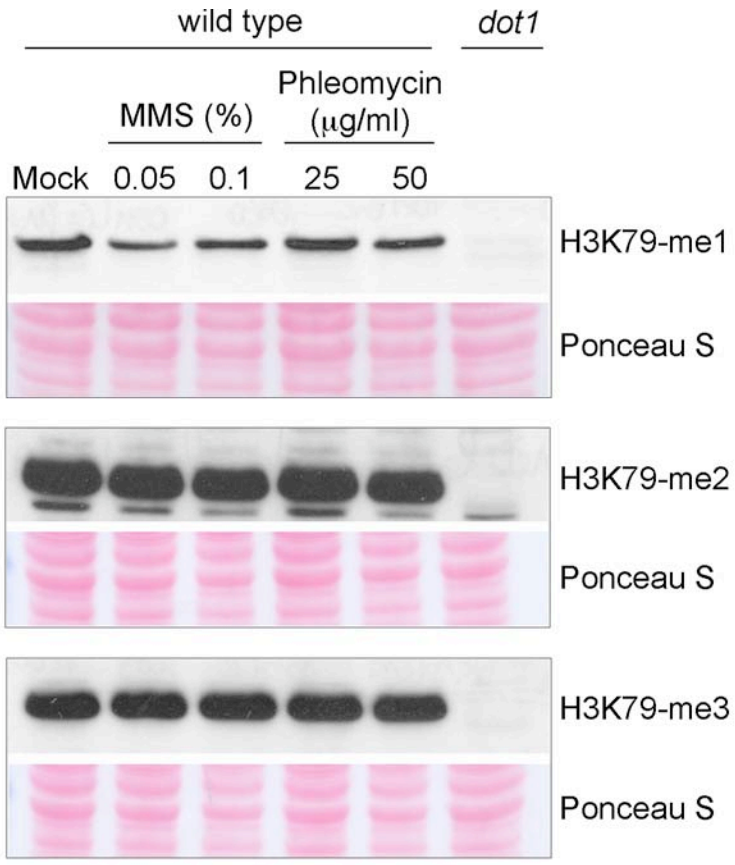

FIGURE S3.—The state of histone H3K79 methylation does not change upon global DNA damage. Wild-type cells were mock treated or treated for 1 hour with 0.05% or 0.1% MMS or with 25 μg/ml or 50 μg/ml of phleomycin, as indicated. Cell extracts were analyzed by western blot with antibodies that specifically recognize the mono-, di- or tri-methylated histone H3K79 (-me1, -me2 or -me3, respectively). The *dot1* mutant, which lacks H3K79 methylation, is included as a control. Staining of the membrane with Ponceau S is shown as a loading control.