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LSD1-LIKE1-mediated H3K4me2 demethylation is required for homologous recombination repair

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摘要	Homologous recombination is a key process for maintaining genome integrity and diversity. In eukaryotes, the nucleosome structure of chromatin inhibits the progression of homologous recombination. The DNA repair and recombination protein RAD54 alters the chromatin structure via nucleosome sliding to enable homology searches. For homologous recombination to progress, appropriate recruitment and dissociation of RAD54 is required at the site of homologous recombination; however, little is known about the mechanism regulating RAD54 dynamics in chromatin. Here, we reveal that the histone demethylase LYSINE-SPECIFIC DEMETHYLASE1-LIKE 1 (LDL1) regulates the dissociation of RAD54 at damaged sites during homologous recombination repair in the somatic cells of Arabidopsis (Arabidopsis thaliana). Depletion of LDL1 leads to an overaccumulation of RAD54 at damaged sites with DNA double-strand breaks. Moreover, RAD54 accumulates at damaged sites by recognizing histone H3 lysine 4 di-methylation (H3K4me2); the frequency of the interaction between RAD54 and H3K4me2 increased in the ldl1 mutant with DNA double-strand breaks. We propose that LDL1 removes RAD54 at damaged sites by demethylating H3K4me2 during homologous recombination repair and thereby maintains genome stability in Arabidopsis.
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