Arginine methylation regulates DNA polymerase beta

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Alterations in DNA repair lead to genomic instability and higher risk of cancer. DNA base excision repair (BER) corrects damaged bases, apurinic sites, and single-strand DNA breaks. Here, a regulatory mechanism for DNA polymerase beta (Pol beta) is described. Pol beta was found to form a complex with the protein arginine methyltransferase 6 (PRMT6) and was specifically methylated in vitro and in vivo. Methylation of Pol beta by PRMT6 strongly stimulated DNA polymerase activity by enhancing DNA binding and processivity, while single nucleotide insertion and dRP-lyase activity were not affected. Two residues, R83 and R152, were identified in Pol beta as the sites of methylation by PRMT6. Genetic complementation of Pol beta knockout cells with R83/152K mutant revealed the importance of these residues for the cellular resistance to DNA alkylating agent. Based on our findings, we propose that PRMT6 plays a role as a regulator of BER.

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