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The chromatin remodeler Chd1 supports MRX and Exo1 functions in resection of DNA double-strand breaks

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Abstract:

DNA double strand breaks (DSBs) are among the most severe types of damage occurring in the genome because their faulty repair can result in chromosome instability, commonly associated with carcinogenesis. Efficient and accurate repair of DSBs relies on several proteins required to process them. However, eukaryotic genomes are compacted into chromatin, which restricts the access to DNA of the enzymes devoted to repair DNA DSBs. To overcome this natural barrier, eukaryotes have evolved chromatin remodeling enzymes that use energy derived from ATP hydrolysis to modulate chromatin structure. Here, we examine the role in DSB repair of the ATP-dependent chromatin remodeler Chd1, which is frequently mutated in prostate cancer. We find that Chd1 is important to repair DNA DSBs by homologous recombination (HR) because it promotes the association with a damaged site of the MRX complex and Exo1, which are necessary to initiate HR. This Chd1 function requires its ATPase activity, suggesting that Chd1 increases the accessibility to chromatin to initiate repair of DNA lesions.