

Abstract

Homologous recombination (HR) is an essential mechanism for accurate repair of DNA double-strand breaks (DSBs). However, HR must be tightly controlled because excessive or unwanted HR events can lead to genome instability, which is a prerequisite for premature aging and cancer development. A critical step of HR is the loading of RAD51 molecules onto single-stranded DNA regions generated in the vicinity of the DSB, leading to the formation of a nucleoprotein filament. Several DNA helicases have been involved in the regulation of the HR process. One of these is human FBH1 (F-box DNA helicase 1) that is a member of SF1 superfamily of helicases. As a unique DNA helicase, FBH1 additionally possesses a conserved F-box motif that allows it to assemble into an SCF complex, an E3 ubiquitin ligase that targets proteins for degradation. FBH1 has been implicated in the restriction of nucleoprotein filament stability. However, the exact mechanism of how FBH1 controls the RAD51 action is still not certain. In this work, we revealed that FBH1 actively disassembles RAD51 nucleoprotein filament. We also show that FBH1 interacts with RAD51 and RPA physically *in vitro*. Based on these data, we propose a potential mechanism of FBH1 antirecombinase function.