Abstract

The genome is constantly threatened by various damaging agents and maintaining its integrity is crucial for all organisms. Several repair pathways have been implicated in the removal of different types of lesions from DNA. Among them, homologous recombination (HR) plays a key role in repair of double-strand breaks. HR is a highly important repair mechanism which has to be tightly regulated to prevent excessive HR events. These events could interfere with other DNA repair pathways, generate toxic intermediates, or block the progression of the replication fork. Therefore, it is not surprising that cells have evolved mechanisms that counteract inappropriate HR events. As it has been shown recently, cells possess DNA helicases capable of preventing excessive recombination. A novel human DNA helicase, hFBH1, belonging to the superfamily I has been shown to function as pro- and anti-recombinase. Similar to the two members of RecQ family, BLM and RECQL5, FBH1 disrupts Rad51 from nucleofilament. However, FBH1 might also promote initiation of HR. The FBH1 helicase possesses additional high conserved F-box motif which allows it to act within a Skp1-Cullin-F-box, SCF, complex as ubiquitin ligase and target proteins for degradation.