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MAIN FIELDS OF RESEARCH; ABSTRACT

Various cellular mechanisms cooperate to ensure faithful DNA replication and maintain genome stability. However, replication forks are frequently challenged by exogenous or endogenous DNA damage. This leads to transient slowing or stalling of replication forks, which is defined as replication stress. Homologous Recombination (HR) and the Fanconi Anemia (FA) pathways act in collaboration in order to secure fork stabilization and promote fork recovery. Human CtIP is most widely recognized for its essential function in DNA-end resection and homology-directed repair of DNA double-strand breaks. Furthermore, increasing evidence implicates CtIP as a critical factor for the maintenance of genome stability owing to its roles in transcriptional regulation, the DNA damage response and cell cycle checkpoint control. Recently, CtIP was reported to promote the recovery of stalled replication forks suggesting that CtIP also participates in the response to replication stress. However, our understanding of the role of CtIP in facilitating accurate DNA replication and preventing replication stress-induced genomic instability is still very limited. Using DNA fiber spreading, we are currently analyzing the functional relationship between CtIP and HR and FA proteins response to replication stress.

SPECIAL TECHNIQUES AND EQUIPMENT

Standard biochemistry and molecular biology techniques (western blot, immunoprecipitation, molecular cloning), cell culture, fluorescence microscopy, FACS, PFGE, DNA fibers assay, metaphase spreads.