SAPIENZA UNIVERSITÀ DI ROMA

DIPARTIMENTO DI SCIENZE BIOCHIMICHE A. ROSSI FANELLI

Dottorato di Ricerca in Biochimica

AVVISO DI SEMINARIO

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The RarA protein of Escherichia coli: How DNA gaps are generated and what happens in them

ABSTRACT

The replication fork often encounters barriers in the form of bound proteins and DNA lesions. Some types of DNA lesions lead to replication fork stalling or collapse, and require repair before replication can continue. However, some lesions are simply skipped over. The replication fork disengages from the template, and replication is re-initiated downstream, leaving the lesion behind in a gap. The existence of lesion-containing gaps behind the replication fork was discovered in the 1960s. However, our understanding of the role of such gaps in DNA repair has been constrained due to technological limitations in detecting and monitoring the formation and resolution of genomic gaps.

The RarA protein of Escherichia coli is a member of a highly conserved DNA repair protein family (yeast homologue = Mgs1; human homologue = WRNIP1). It is a AAA+ ATPase in the clamp loader clade. RarA protein acts directly at the replisome to create post replicative gaps in the lagging strand. This protein function is both unprecedented and unanticipated. Gap formation requires the ATPase and β-clamp interaction functions of RarA. In vivo, it is likely that RarA promotes gap formation to bring about lesion skipping, leaving lesions behind the replication fork in gaps. There are three processes that can repair lesions within gaps. These are recombinational DNA repair, translesion DNA synthesis, and replication template switching. A number of additional (and until recently enigmatic) E. coli proteins, including the Uup and RadD proteins, have specialized roles in gap repair. The DNA repair that is focused on DNA gaps plays an important role in the induction of DNA damage responses in all cells, and is also a source of considerable genome instability and mutagenesis.

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