Abstract

The deadly pathogen *Mycobacterium tuberculosis* has been characterized for its virulence in immense detail. Regardless of this, the capability of *M. tuberculosis* to combat extreme stress conditions inside the host cell for extended durations remains unclear. One of the adverse effects caused by host cell mechanisms to the invading *M. tuberculosis* is DNA damage. Several DNA repair mechanisms exist within the bacterium to counter the DNA damage. The most common type of damage occurring to the genetic material is double stranded breaks (DSBs). Two cardinal pathways exist for the double stranded break repair- Homologous recombination (HR) or Non-homologous end joining (NHEJ) repair pathway. Homologous recombination is more predominant in bacteria and yeast whereas injured mammalian cells are restored by NHEJ. A major intermediate of homologous recombination pathway is Holliday junction. A range of mechanisms act upon HJs for proper chromosome segregation. The process is termed as Holliday junction resolution. A class of enzymes acts on Holliday junction and resolves it into two DNA duplexes. These enzymes are called Holliday junction resolvases. For our study, a Holliday junction resolvase from *M. tuberculosis* termed as MtRuvX has been chosen. The crystal structure of MtRuvX confirms the presence of RNase H fold in the protein. Docking studies of MtRuvX conducted in this study indicate that the regions interacting with DNA and RNA could be similar. The ATP docking studies also show that there could be two ATP binding sites within the protein. The computational and structural analysis reported here could serve as the foundation for further directed experiments to comprehend the mechanistic details of enzymatic activity of MtRuvX and also its interactions with nucleic acids.