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Structural Analysis of *E. coli* β-sliding Clamp 148-152 Ala Mutant and its Role in DNA Polymerase V-dependent Translesion DNA Synthesis. Vivian Cody, Jim Pace, Edward Snell, Hauptman-Woodward Medical Research Inst. Buffalo, NY 14203, Mark D. Sutton, Laurie Sanders, Sarah Ponticelli, Jill Duzen, Robert Maul, Univ. at Buffalo, Buffalo, NY 14214.

Damaged bases in the DNA that are not repaired prior to replication can act as potent blocks to polymerization, leading to replication fork arrest. We previously described a mutant form of the *E. coli* β sliding clamp protein bearing alanine substitutions in place of residues 148-152 (β-148-152) that was severely impaired for pol IV-dependent translesion synthesis *in vivo*. We have solved the crystal structure of the mutant β-148-152 clamp protein. Data were measured to 1.75Å resolution for the homodimeric *E. coli* β-148-152 mutant that crystallizes in a triclinic lattice. The current model reveals that the loop encompassing the mutations adopts an alternative conformation from the wild type protein. In addition, BIAcore and gel filtration chromatography data for the interactions of wild type and β-148-152 clamp proteins with various *E. coli* pols indicate that residues 148-152 define a surface of the clamp that is critically important for the proper functioning of some, but not all, *E. coli* pols. These data suggest that these residues play a vital role in DNA polymerase switching. Supported in part by GM66094 (MDS).