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The Effect of an E. coli topA66 Mutation on SOS and Antibiotic Response

The adaptive force of bacterial mutagenesis allows microbes to dodge even the most advanced bactericide. Topoisomerases are effective targets for treatment from anticancer to antibacterial chemotherapy.The objective of this research was to explore the effects of stifling mutagenesis (SOS response) in E. coli. Microdilutions comparing growth of wild type and topA66 mutant to antibiotics found that a mutation in the topA66 gene leads to increased sensitivity. Since topoisomerases are vital components of SOS transcription, a luciferase assay was performed to confirm the decreased SOS response levels in the topA66 mutant. Colony counts determined a decreased mutation rate for the topA66 mutant verses wild type strain after norfloxacin treatment indicating that decreased SOS response levels may result in lower inclinations to develop multi-drug resistance. This study, dealing with a novel topA mutation, supports previous research and shows that topoisomerase I has the potential to be a target for antibiotics.