The non-canonical SOS-system of DNA-repair and mutagenesis in *Acinetobacter baumannii*

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Acinetobacter baumannii is a Gram-negative bacterium which produces infections mainly in immunosuppressed patients and is characterized by its ability to rapidly develop antimicrobial resistance. In addition, one of the most striking features in comparison to most bacteria is the lack of LexA, the canonical transcriptional repressor of the SOS system. In order to study the atypical SOS system of this nosocomial pathogen, the transcriptional response of *A. baumannii* to the DNA-damaging agent mitomycin C (MMC) was studied using DNA microarray technology. Most of the 39 genes induced by MMC were related to either prophages or encoded proteins involved in DNA repair, mainly genes encoding predicted homologs to components of the error-prone DNA polymerase V (UmuDC). Electrophoretic mobility shift assays demonstrated that the product of the *A. baumannii* MMC-inducible *umuD* gene (*umuDAb*) specifically binds to a palindromic sequence present in its promoter region. Mutations in this palindromic region abolished UmuDAb protein binding. A comparison of the promoter regions of all MMC-induced genes identified four additional transcriptional units with similar palindromic sequences recognized and specifically bound by UmuDAb. Therefore, the UmuDAb regulon consists of at least eight genes, most of them encoding predicted error-prone DNA polymerase V components. Furthermore, inactivation of the *umuDAb* gene resulted in the deregulation of all DNA-damage-induced genes containing the described palindromic DNA motif, indicating that UmuDAb is the LexA analog in this bacterial species. Finally, to elucidate the role of the described UmuDC homologs in antibiotic resistance acquired through UV-induced mutagenesis, the three umuD homologs found were inactivated through the construction of *A. baumannii* knock-outs. Interestingly, all the mutants, and especially the *umuDAb* mutant, were less able to acquire resistance to rifampicin through the activities of their error-prone DNA polymerases. All these data suggest that non-canonical SOS system of *A. baumannii* provides an adaptation mechanism for this nosocomial pathogen.