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Phenotypic and immunologic characteristics of *Salmonella* lacking RNA modification enzymes

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Abstract

Deletion of tRNA modification enzymes *gidA* and *mnmE* significantly attenuated *Salmonella* virulence. Transcriptome and proteome analyses indicated that expression of several virulence factors was significantly altered. Mice immunized with the *gidA* mutant were protected against a lethal dose of wild-type (WT). The mechanistic basis of protection afforded by immunization of mice with the *gidA* mutant was investigated using flow cytometry and several immunological techniques. Data from this study indicated that both humoral and cellular immunity played a role with the humoral immune response potentially being the main mechanism of protection.

GidA together with MnmE thought to catalyze modification of tRNA which is required for correct translation during gene expression. Examination of relative contribution of GidA and MnmE in modulation of *Salmonella* virulence indicated various degree of attenuation and that GidA and MnmE bind together and alters *Salmonella* tRNA modification.

GidB, a methyltransferase enzyme, was identified as being responsible for methylation of 16S ribosomal RNA (rRNA) in *Escherichia coli* and other bacteria. Deletion of *Salmonella gidB* gene indicated a compromised overall bacterial fitness, significantly reduced motility and showed a filamentous morphology under the stress of nalidixic acid. Most importantly, deletion of *gidB* conferred high-level resistance to the aminoglycoside antibiotics streptomycin and neomycin. This study determined the methylation site for the WT *Salmonella*. Taken together, these data indicated that GidA, GidB and MnmE enzymes play a significant role in modulation of biological and virulence characteristics and alteration of antibiotic susceptibility in *Salmonella* under stress conditions.

Biography

Amin Fadl obtained his PhD from University of Connecticut, Storrs and completed a postdoctoral training at the University of Texas Medical Branch. Currently he is an Assistant Professor of Microbiology at the University of Wisconsin-Madison. His research focuses on the molecular pathogenesis, immune and inflammatory responses, and host-pathogen interaction of *Salmonella*. He has published more than 44 papers in reputed journals.