Clinical Microbiology-2013

OMICS Group Conferences
Accelerating Scientific Discovery

2nd International Conference on
Clinical Microbiology & Microbial Genomics

September 16-18, 2013 Las Vegas, USA

RNA modification enzymes: Role in modulation of biological and virulence characteristics of *Salmonella*

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Abstract

Our previous studies have shown that deletion of tRNA modification enzyme glucose-inhibited division gene (*gidA*) significantly attenuated *Salmonella enterica* serovar Typhimurium (STM) virulence. Transcriptome and proteome analyses indicated that expression of several virulence factors was significantly altered. Most importantly, *gidA* mutant mice immunized with the *gidA* mutant were protected against a lethal dose of wild-type (WT) STM. Further studies on the mechanistic basis of this protection indicated 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.

The GidB is a methyl transferase enzyme that is involved in the methylation of the 16S rRNA in bacteria. Deletion of *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 amino glycoside antibiotics streptomycin and neomycin. This study determined the methylation site for the WT STM to be at G527 of the 16S rRNA. A lack of methylation in the mutant confirmed that GidB is required for this methylation. Taken these data indicated that both GidA and GidB enzymes play a significant role in modulation of biological and virulence characteristics and alteration of antibiotic susceptibility in *Salmonella* under stress conditions.

Biography

Dr. 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.