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Foodborne pathogens like *Escherichia coli* O157: H7, is the leading causes of infectious gastrointestinal diseases, and death in severe conditions. During its journey, orally acquired food pathogen has to transit through the extremely acidic gastric compartment and cope with the extreme acidic stress (pH < 2.5). Therefore, the ability to endure the combined effect of HCl in gastric juice and of short chain fatty acids produced by the intestinal microbiota, is crucial for successful colonization of the gastrointestinal tract. Different food pathogen has have evolved different strategies to overcome acid stress. The glutamate-dependent acid resistance (GDAR) system is the most potent acid resistance system. The regulation of GDAR system is remarkably complex involving multiple regulatory circuits.

The MnmE protein, a GTPase involved in a tRNA modification, has been implicated in the regulation of the GDAR system. In the present study, to understand the role of MnmE and MnmG, another tRNA modifying enzyme, in the regulation of the glutamate decarboxylase gene (*gadA*), we constructed Δ*mnmG* and Δ*mnmE* deletion mutants of *E. coli* O157: H7 and gadA-lacZ translational fusions in K-12 strains. In O157:H7 strains, both the *mnmG* and *mnmE* deletion mutants were defective in the GDAR mechanism and lost their acid resistance at pH 2.0. The results of acid challenge test were further confirmed by westen blot analysis. It was obvious that expression of the GadA protein was completely suppressed in the *mnmG* and *mnmE* deletion mutants, confirming that these tRNA modifying genes serve as regulators for GadA expression. Gene fusion studies in K-12 strains implicated that *mnmE* and *mnmG* are not directly involved in translational regulation of GadA production. However, overexpression of the *gadE* gene, encoding an essential transcription factor for *gadA*, in the Δ*mnmG* and Δ*mnmE* mutants could not restore acid resistance ability of mutants. Therefore, to get further insight into the translational regulation of *gadA* gene, more studies are required.