THE IMPACT OF ACID STRESS ON *ESCHERICHIA COLI* O157:H7 VIRULENCE

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Abstract

Escherichia coli O157:H7 infection is a leading cause of hemorrhagic colitis, and hemolytic uremic syndrome. Many opportunities for acid stress exposure exist for this food and waterborne pathogen, including gastric acid shock. Yet little is known how this affects *E. coli* O157:H7 virulence. The effect of various acid shock protocols on *E. coli* O157:H7 survival, verotoxin production, and the ability to adhere to host epithelial cells was examined. Brief acid shock alone at pH 3.0 decreased the host cell adhesion capability by a factor of 4.3-4.8, yet when the acid shock was preceded by adaptation at pH 5.0, a 1.6-3.2 fold enhanced adhesion of surviving organisms to epithelial cells relative to unstressed organisms was observed. However, acid stress did not affect verotoxin production. Pretreatment of acid stressed bacteria with erythromycin eliminated the acid-induced adhesion enhancement, suggesting that protein synthesis is a requirement for the enhanced adhesion observed with acid-adapted acid-shocked *E. coli* O157:H7. Real time PCR analysis of locus for enterocyte effacement (LEE)-encoded virulence factors, intimin and EspA, revealed no significant upregulation for the acid stress treatments associated with the increased host cell adhesion. On the contrary, elevated mRNA levels for both intimin and EspA were observed for bacteria subjected to brief acid shock alone even though the host-cell adhesion was significantly decreased with these treatments. These results suggest that complex regulation mechanisms for LEE-encoded virulence factors exist and that *E. coli* O157:H7 virulence can be enhanced after acid stress through increased adhesion to host epithelial cells.