

Escherichia coli O157

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Contributor: Haiqing Sheng , Yansong Xue , Wei Zhao , Carolyn J. Hovde , Scott A. Minnich

Escherichia coli O157:H7 (O157) are noninvasive and weak biofilm producers; however, a subset of O157 are exceptions. O157 ATCC 43895 forms biofilms and invades epithelial cells. Tn5 mutagenesis identified mutation insertions that map within the curli *csgB* fimbriae locus to be responsible for both phenotypes. Screening of O157 strains for biofilm formation and cell invasion identify a bovine and a clinical isolate with those characteristics. A single base pair A to T transversion, intergenic to the curli divergent operons *csgDEFG* and *csgBAC*, is present only in biofilm-producing and invasive strains. Using site-directed mutagenesis, this single base change was introduced into two curli-negative/noninvasive O157 strains and modified strains to form biofilms, produce curli, and gain invasive capability. Transmission electron microscopy (EM) and immuno-EM confirmed curli fibers. EM of bovine epithelial cells (MAC-T) co-cultured with curli-expressing O157 show intracellular bacteria. The role of curli in O157 persistence in cattle was examined by challenging cattle with curli-positive and -negative O157 and comparing carriage. The duration of bovine colonization with the O157 curli-negative mutant was shorter than its curli-positive isogenic parent. These findings definitively demonstrate that a single base pair stably confers biofilm formation, epithelial cell invasion, and persistence in cattle.

E. coli O157

cattle

curli

biofilm

cell invasion

Enterohemorrhagic *Escherichia coli* (EHEC) cause human disease with symptoms ranging from self-limited watery diarrhea to life-threatening hemorrhagic colitis, and hemolytic uremic syndrome ^{[1][2]}. *E. coli* O157:H7 (O157) is the best studied EHEC serotype and is the predominant strain associated with disease outbreaks in North America, the United Kingdom, and Japan ^{[3][4][5]}. Cattle and other ruminants carry this pathogen with no apparent symptoms ^{[6][7]} and are the most common source for human infections ^{[8][9]}. O157 colonize at the bovine recto-anal junction (RAJ) and the bacteria persist in the feces of individual animals from a few days to several months ^{[10][11]}. Attachment to biological surfaces is a first critical step in colonization and is mediated by multiple bacterial factors. Surface-associated factors of O157 contributing to tissue adherence and persistence in the bovine host include O-antigen ^[12], fimbriae ^[13], adhesins such as intimin ^[14], and some autotransporters ^[15]. There is evidence that the duration of colonization and the bovine immune responses are strain/variant dependent ^{[16][17]}.

Curli fimbriae, comprised of polymerized amyloid protein, are expressed on the surface of many members of the Enterobacteriaceae and other Gammaproteobacteria ^[18]. Curli binds amyloid-specific dyes, such as Congo red and certain host proteins, including fibronectin, laminin, and plasminogen ^{[19][20]}. During infections, curli complexes with extracellular matrix DNA. In a mouse model for lupus erythematosus autoimmunity, these curli-DNA complexes interact with Toll-like receptors (TLRs) 2 and 9 on dendritic and macrophage cells resulting in the production of autoantibodies ^[21]. In most non-O157 *E. coli*, curli is regulated by σ s and synthesized at low temperature, in nutrient-deprivation, and/or in stationary phase, conditions that promote biofilm formation ^[22]. Curli synthesis

requires the expression of genes from two divergently transcribed operons, designated *csgDEFG* and *csgBAC*. Genes with identified functions include the regulator CsgD, the type VIII secretion machinery components CsgE-G, the curli major subunit CsgA, and the curli nucleation protein CsgB [23][24]. The intergenic region between *csgDEFG* and *csgBAC* is large and contains many putative binding sites for regulatory factors. CsgD is essential for the transcription of the curli operons [19]. Curli promotes biofilm adhesion to abiotic surfaces as well as to mammalian cells [25][26][27][28]. Although both operons are present in all sequenced O157 strains [29][30][31], the majority of O157 (approximately 95%) are curli-negative. This is because the prophage carrying Shiga toxin type-1 inserts into *mlrA*, a positive regulator of *csgD* [32]. The few curli-positive O157 strains produce curli constitutively, including at 37 °C, and have acquired a suppressor mutation overriding the normal requirement for *mlrA* [33][34].

O157 is a weak biofilm producer and is considered an extracellular pathogen [35][36]. However, some strains do not meet this general characterization. In a previous study, we showed that O157 strain 43895, an outbreak isolate from hamburger, produces biofilms at 37 °C, invades epithelial cells, and persists longer in cattle than a biofilm-negative strain [16]. Curli expression has been found in certain O157 strains [34], but the underlying mechanism has not been fully explored.

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