

Understanding the Detection of Shiga Toxin Producing Escherichia Coli: Virulence Factors, Pathogenicity Islands or Serotypes?



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Abstract

Shiga toxin-producing Escherichia coli (STEC) are important food borne pathogens that can cause severe disease in children by producing two toxins (Stx1 and Stx2). This bacteria harbor several additional virulence factors and have been classified in different serotypes that have been used as screening for detection of STEC. As it is not possible to define when a STEC strain is pathogenic, it is important to incorporate the risk assessment.

Abbreviations: STEC: Shiga Toxin-producing Escherichia coli; HUS: Hemolytic Uremic Syndrome; LEE: Locus of Enterocyte Effacement; LAA: Locus of Adhesion and Auto Aggregation; FSIS: Food Safety and Inspection Service; HACCP: Hazard Analysis and Critical Control Points; EFSA: European Food Safety Authority

Introduction

Shiga toxin-producing Escherichia coli (STEC) are important food borne pathogens that can cause severe disease in children, including a life-threatening complication such as bloody diarrhea and hemolytic uremic syndrome (HUS) [1]. The damage is produced by Shiga toxins encoded by stx1 and stx2 genes carried by lysogenic phages that infect the bacteria [2]. STEC must intimately adhere to epithelial cells through adhesions to deliver toxins efficiently to the host organism. Intimin, encoded by eae gene, is the best characterized adherence protein inducing a characteristic histopathological lesion defined as "attaching and effacing" (A/E). A pathogenicity island called the locus of enterocyte effacement (LEE) encodes proteins necessary for the formation of the A/E lesion, including intimin [1]. The presence of both eae and stx genes has been associated with enhanced virulence [3]. Nevertheless, the presence of LEE is not essential for pathogenesis, considering that some LEE negative STEC have been associated with severe disease in humans.

The mechanism used by LEE negative STEC strains to adhere and colonize to epithelial cells is poorly understood, although the Protein Sea has been involved in the cells adherence [4]. In a recent

study in STEC LEE negatives, Montero, Velasco [5] identified a member of the Heat-resistant agglutinin family and characterized this antigen named Hem agglutinin from Shiga toxin-producing E. coli (Hes). They show that hes and other genes such as asiha, pagC, ag43 were integrated in each of the four modules present in the new Island named Locus of Adhesion and Auto aggregation (LAA) whose presence is associated with severe disease. They have proposed hes as a genetic marker of LAA. According to serotype, STEC is classified in O157: H7, recognized as the most important serotype associated with human infection, and non-O157 serotypes such as O91, O104, O113, and others that have been involved in human disease [6]. For instance, in 2011, the Food Safety and Inspection Service (FSIS) of USDA declared the "Big Six" (O26, O45, O103, O111, O121, and O145) as adulterants through a Federal Register Notice and announced plans to start the Hazard Analysis and Critical Control Points (HACCP) verification testing for raw beef trim and ground beef [7].

According to a European Food Safety Authority report (EFSA, 2009), the major serotypes or serogroups of concern are O157:H7, O26, O103, O145, O111, and O9 [8]. Under the new rules, any

meat that contains these sero groups cannot be sold as raw products. Karmali, Mascarenhas [9] proposed to classify STEC into five seropathotypes, according to their reported frequencies in human illness. Seropathotype A (O157:H7/NM), seropathotype B (O26:H11, O103:H2, O111: NM, O121:H19, and O145: NM), seropathotype C (O91:H21 and O113:H21), seropathotype D and E (multiple). But, Scheutz [10] argued that this classification should not be used because it associates serotype with illness instead of the virulence profile, and proposes a new classification of STEC: HUS-inducing and/or epidemic outbreak potential STEC, human diarrhea inducing STEC, and animal-associated STEC.

Cattle are the main reservoir of STEC but other ruminant species such as, sheep, goats, and deer also act as reservoirs, shedding these bacteria through their feces, maintaining these pathogens among cattle herds, contaminating the environment and derived foods [11]. For public health investigation of STEC infection, in clinical and/or food samples the presence of the stx genes should be screened by PCR. However, the detection of these genes without the corresponding strains isolation is considered a presumptive diagnosis [12]. Risk analysis of food category considering the habits of consumer groups and the geographical and temporal relationship with human and food strains is necessary to determine the microbiological criteria for each foodstuff [13]. Efforts for risk assessment and stringent monitoring system are necessary to give an insight into the real clinical implications of virulence genes, serotypes and new genetic markers allowing the classification of STEC strains more efficiently.

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