I-INTRODUCTION

Hemorrhagic diarrhea: Bloody colitis (inflammation of the bowel). The diarrhea is severe with painful abdominal cramps, gross blood in the stool, caused mostly by Escherichia coli, usually by the strain *E. coli* O157:H7. and *Shigella*. (*William and Melissa 2008*)

Most of the *E. coli* are normal inhabitants of the small intestine and colon (non-pathogenic). Nevertheless, these non-pathogenic *E. coli* can cause disease if they spread outside of the intestines, for example, into the urinary tract, or into the blood stream (sepsis). Other *E. coli* strains (enterovirulent *E. coli* strains or EEC) cause "poisoning" or diarrhea even though they usually remain within the intestine by producing toxins or intestinal inflammation. (*Hudault, et al.*, 2001)

There are four to six groups of E. coli strains enterohemorrhagic E. coli (EHEC), enterotoxigenic E. coli (ETEC), enteropathogenic E. coli (EPEC), enteroinvasive E. coli (EIEC), enteroadherent E. coli (EAEC), enteroaggregative E. coli (EAggEC) (\it{Todar} , $\it{2005}$)

E. coli O157:H7 can cause a bloody diarrhea due to toxins it secretes when it infects human intestinal tracts. (Rendón, et al., 2007).

E. coli O157:H7 can cause additional complications in children and the elderly; renal failure, anemia, and dehydration especially for children termed Hemolytic-uremic syndrome (HUS) and spontaneous bleeding, organ failures, and mental changes in the elderly. Some of these patients develop disabilities or die. (*Karamali, et al. 1989*)

Diagnosis is definitively made when *E. coli* O157:H7 is isolated, usually from the patient's stool, and identified as serotype O157 by immunologic tests. (*Chart, et al., 1991*)

Shigella is abacteria closely related to E. coli and Salmonella. During infection, it typically causes dysentery. Phylogenetic studies indicate that Shigella is more appropriately treated as subgenus of Escherichia, and that moreover certain strains generally considered E. coli— such as EHEC O157:H7— are better placed in Shigella (Hale and Keusch, 1996).

Shigella infection is typically via ingestion (fecal—oral contamination); depending on age and condition of the host, as few as 100 bacterial cells can be enough to cause an infection . *Shigella* causes

dysentery that results in the destruction of the epithelial cells of the intestinal mucosa in the cecum and rectum. (*Levinson*, 2006)

Verotoxin (VT) or shiga like toxin, is a toxin generated by some strains of *E. coli* It is named for its similarity to the AB5-type Shiga toxin produced by the bacteria *Shigella dysenteriae*. (*Beutin, et al., 1993*) There are two types of verotoxin, known as (VT1) and (VT2) (*O'Brien and Holmes, 1987*)

Some strains of *Shigella* produce enterotoxin and shiga toxin, similar to the verotoxin of *E. coli* O157:H7 and other verotoxin - producing *E. coli*. Both shiga toxin and verotoxin are associated with causing HUS. (*Hale and Keusch 1996*).

The toxin acts on the vascular endothelium. The B subunits of the toxin bind to a component of the cell membrane known as Gb3. Binding of the subunit B to Gb3 causes induction of narrow tubular membrane invaginations, which drives formation of inward membrane tubules for the bacterial uptake into the cell. When the protein is inside the cell, the A subunit interacts with the ribosome's to inactivate them. The A subunit of Shiga toxin is an N-glycosidase that modifies the RNA component of the ribosome to inactivate it and so bring a halt to protein synthesis leading to the death of the cell. The vascular endothelium has to continually renew itself, so this killing of cells leads to a breakdown of the lining and to hemorrhage. The first response is commonly a bloody diarrhea. This is because Shiga toxin is usually taken in with contaminated food or water. (Römer, et al., 2007).