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Commentary

Intestinal Infections Caused by *Escherichia coli*

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DESCRIPTION

Gram-negative bacillus *Escherichia coli* (*E.coli*) are recognised to be a component of healthy intestinal flora but can also cause intestinal and extra intestinal disease in humans. Numerous distinctive *E.coli* strains have been found, causing illnesses ranging from mild, identity gastroenteritis to renal failure and septic shock. *E.coli's* virulence makes it easier for it to overcome host defense mechanisms and develop antibiotic resistance. *E.coli* infections that result in extra intestinal illness will be differentiated from those that resulting in intestinal infection. The pathogenic *E.coli* syndromes will be used to characterise intestinal diseases, such as Entero Toxigenic *Escherichia Coli* (ETEC), Entero Hemorrhagic *Escherichia Coli* (EHEC), also referred to as Shiga toxin-producing *Escherichia coli* (STEC), Entero Invasive *Escherichia Coli* (EIEC), Entero Pathogenic *Escherichia Coli* (EPEC). *E.coli* is a component of the commensal gut flora. The most prevalent gram-negative *bacterium* in the human gastrointestinal tract is *E.coli*, and it is not pathogenic in this environment. *E.coli*, however, it can also result in pneumonia, bacteremia, peritonitis, Urinary Tract Infections (UTI's), and other conditions when identified beyond the gastrointestinal system. Numerous nosocomial infections, such as pneumonia related to ventilation systems and catheter-associated urinary tract infections are caused by *E.coli*. In addition to raw meats, *E.coli* can also be found in water, on vegetables, and soil. When undercooked food is ingested by humans, pathogenic strains can lead to intestinal disease.

Both intestinal disease and infection beyond the intestine are brought on by *Escherichia coli*. One of five subtypes of *E.coli* that cause intestinal sickness can be characterized by their O and H antigens. A repeating polysaccharide chain found in the Lipopolysaccharide (LPS) outer membrane determines the O antigen, while the flagellum determines the H antigen.

In environments with low resources, ETEC (Entero Toxigenic *Escherichia Coli*) is widely found in food and water, and and it can cause diarrhoea. Ingestion of about 100,000,000 organisms is required to make a healthy individual sick. It is the primary agency responsible for traveler's diarrhoea. In situations with insufficient resources, ETEC also plays a key role in the dehydrating diarrheal disease that affects infants and children. The first *E. coli* pathotype to be identified as the main cause of watery diarrhoea in newborns and children is EPEC, which develops sporadic and epidemic infections. The most common method of contracting an EPEC-related diarrheal sickness is through ingestion, while it can also be transmitted from person to person.

E.coli infection comes from bacterial consumption and *E.coli's* innate ability to get through host immune system. The cell envelope for gram-negative bacteria is made up of an outer membrane, peptidoglycan cell wall, and inner cytoplasmic cell membrane. In the case that the Lipopolysaccharide (LPS), which makes up the outer membrane, is lysed, a toxic reaction will occur. Each strain of pathogenic *E.coli* has unique virulence factors that are encoded on plasmids, transposons, and bacteriophages.

ETEC (Entero Toxigenic *Escherichia Coli*) produces colonising fimbriae that allow the bacteria to adhere to the gut wall. When attached, ETEC produces secretory toxins that are encoded on plasmids known as heat-Labile Toxin (LT) and/or heat-Stable Toxin (ST). Intestinal crypt cells secrete chloride as a result of LT's stimulation of adenylate cyclase, which raises intracellular Cyclic Adenosine Mono-Phosphate (cAMP) levels. Additionally, this mechanism prevents the absorption of sodium chloride *via* intestinal villi. Watery diarrhoea is the result of this process' free water discharge into the intestinal lumen. As a result of ST's stimulation of guanylate cyclase, there is an increase in intracellular cyclic Guanosine Mono-Phosphate (cGMP), which causes chloride to be secreted and sodium chloride absorption to be blocked, resulting in diarrhoea.

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