# E coli: a foodborne illness

Escherichia coli, or E. coli, is a versatile bacterium which has many different strains with different adaptations. Most strains are harmless and found as normal flora within the intestinal tract of humans and warm-blooded animals. (Meers, et al., 1995) There are, however, some strains, for example O157: H7, which can cause disease, and are usually sourced from food contamination. (Semenova, et al., 2010). The following is a discussion focused on E. coli as a food-borne disease.

## Epidemiology

E. coli has a background of irregular cases, with occasional outbreaks. E. coli O157: H7 is the most important strain in relation to public health; however, other strains are also frequently involved. (World Health organisation, n. d.). Enteropathogenic E. coli is considered a potential food-borne pathogen, infections of this type are reported to come from chicken and beef. Both enterotoxigenic E. coli and enteroaggregative E. coli may also be associated with food-borne outbreaks however they are infrequent. (Aidara-Kanec, et al., 2010)

The first recorded incident was in 1982, there were outbreaks of severe bloody diarrhea in North America. Outbreaks then increased dramatically and spread out to many other countries, (Aidara-Kanec, et al., 2010) and has been reported in Autralia, Canada, Japan, Europe, United States, Europe and Africa. (World Health organisation, 2002) 33% of outbreaks are food-borne in Britain. (Aidara-Kanec, et al., 2010)

E. coli O157: H7 has been the reason for an increasing number of disease outbreaks associated with the consumption of fresh fruit and vegetables

during the last decade. This pathogen, can enter the vegetable production chain via contaminated manure and water and then further infect cattle which are considered the main source of infection (Semenova, et al., 2010). Most epidemics of e. coli O157: H7 have been associated with the consumption of undercooked ground beef or unpasteurised milk or fruit juice contaminated with feaces. (Bauman. R., 2007) Outbreaks have also been sourced from alfalfa sprouts, unpasteurized fruit juice, lettuce, and cheese curd. (World Health organisation, 2002)

## Microbes

E. coli is a gram negative, anaerobic, rod-shaped, (Atlas. R. M., 1984) and motile (Chiodini. P. L., 2008) bacterium from then entrobacteriaceae family. (Atlas. R. M., 1984). Can be found with or without a capsule. (Chiodini. P. L., 2008). Its optimal growth is 37°C. (Atlas. R. M., 1984) Figure 1. Shows a diagram of the components and shape of E. coli. Here it shows the flagellum, which it uses for its motility. Also shown, but not labelled, are the fimbrae and pili, which allow for asdhesion and communication. (Chiodini. P. L., 2008)

E. coli can be detected by many methods. It can be grown on selective media such as Rainbow agar and Sorbital MacConkey agar (Ashtonc, et al., 2008), as shown in figure 2. MacConkey agar allows the growth of gram negative bacteria and also gives lactose fermenting bacteria a pink appearance. (Bauman. R., 2007). Another method developed involves using a growth medium and plotting it with a 5 h two dot assay on nitrocellulose filter disks . This enrichment allows viable bacteria to grow for a short period prior to detection in a visual immunoassay. (Ashtonc, et al., 2008). E. coli can also be identified bichemically; it undergoes rapid lactose fermentation, it

does not utilize citrate, it does not oxidase, and it is able to metabololize nitrate to nitrite. (Bauman. R., 2007).

## Pathogenicity

There are 6 distinct groups of E. coli with different pathogenic mechanisms; enteropathogenic e. coli(EPEC), enterotoxigenic e. coli (ETEC), enterohemorrhagic e. coli (EHEC), enteroaggregative e. coli (EAEC), and diffuse-aggregative e. coli (DAEC). (Chiodini. P. L., 2008)

Virulence factors identified within E. coli, particularly in strains which cause diarrhea are endotoxins, adhesins, and a capsule. (Chiodini. P. L., 2008)

Endotoxins are found in all strains. (Chiodini. P. L., 2008) They bind to proteins on cells lining the intestinal tract. A portion of the toxin then enters the cell and triggers a series of chemical reactions that cause the loss of sodium, chloride, potassium, bicarbonate, and water from the cells, producing watery diarrhea, cramps, nausea, and vomiting. (Bauman. R., 2007)

The adhesins, the P fimbriae or pili allow for attachment and colonisation in the affected area.

The capsule present in some strains, may be associated with adhesion and also provides the cell with protection from being detected by the host’s defense cells. (Chiodini. P. L., 2008)

ETEC possess colonization factors and EPEC produce bundle-forming pili, intimin and an associated protein, this allows the bacteria to attach to epithelial cells of the small intestine, this disrupts the microvillus and in turn

causes diarrhea. EHEC isolates produce a verotoxin. After the bacterium has attached to the mucosa of the large intestine, the toxin produced affects the intestinal epithelium, resulting in diarrhea. Verotoxin receptorcan be found on renal epithelium. While there are many serotypes of EHEC, the most common one is O157: H7. EIEC attach specifically to the mucosa of the large intestine. They invade the cells via endocytosis by using plasmid-associated genes. Inside the cell, they enter the endocytic vacuole, where they grow and spread to adjacent cells, causing tissue damage, inflammation, and cell death, resulting in blood and mucus in stools. EAEC have a specific attachment pattern to tissue culture cells. The pattern is an aggregative formation which looks like ‘ stacked bricks’. These organisms act in the small intestine to cause persistent diarrhea. Their aggregative adherence ability is due to plasmid-associated fimbriae. DAEC produce an alpha hemolysin and cytotoxic necrotizing factor 1. They are also known as diffuse-adherent or cell-detaching E. coli. (Chiodini. P. L., 2008)

E. coli O157: H7, is now the most dominant strain of pathogenic E. coli in developed countries. Disease can occur after the ingestion of as little as 10 organisms. E. coli O157: H7 produces type III secretion system through which it injects 2 types of proteins into intestinal cells. One type of the proteins can disrupt the cell’s metabolism; the other can stick into the cell’s cytoplasmic membrane, where they act as receptors for the attachment of other E. coli O157: H7 bacteria. This attachment enables this strain of e. coli to displace strains of the normal flora. E. coli O157: H7 also produces shiga-like toxin, which inhibits protein synthesis in host cells and spreads throughout the body by attaching to the surfaces of neutrophils, they then cause widespread

death of host cells and tissues. Antimicrobial drugs induce E. coli O157: H7 to increase its production of shiga-like toxin, making the disease worse. (Bauman. R., 2007)

E. coli has the ability to evolve into completely new strains. With a mixture of changes in genes and swapping of virulent factors between current strains, this bacterium is growing into more new strains, which are growing even more resistant and virulent. (Aidara-Kanec, et al., 2010)

## Symtoms and disease

E. coli food poisoning has a medium incubation period ranging from 6 to 48 hours, however it is possible for a period of up to 5 days to occur. (Meers, et al., 1995)

Someimes infection does not show any sign of disease, however it is more common to see a wide range of human diseases (Ashtonc, et al., 2008), including gastroenteritis (Meers, et al., 1995), bloody and non-bloody diarrhea, hemorrhagic colitis, hemolytic uremic syndrome, occasional kidney failure, (Ashtonc, et al., 2008), meningitis, abdominal cramps, enterocolitis (Atlas. R. M., 1984) and sometimes death. (Ashtonc, et al., 2008)

The most common syptom, Diarrhoea, results from a newly-aquired strain of E. coli invading the intestinal mucous membrane or producing an enterotoxin. (Meers, et al., 1995)The enterotoxins produced by E. coli cause a loss of fluids from intestinal tissues. Proper replacement of body fluids and electrolytes can normally ensure the infection is not fatal. (Atlas. R. M., 1984)

## Prevention and treatment

There is currently no vaccine available against E. coli (Chiodini. P. L., 2008), and many strains are resistant to antimicrobials, therefore treatment for these diseases are limited. Although some strains can be treated with antimicrobials, in most cases it is usually just the symptoms which are treated, for example the diarrhea is treated with fluid and electrolyte replacement (Meers, et al., 1995).

Prevention is therefore very important in controlling cases and outbreaks of E. coli. It is important to have control measures throughout the stages of the food chain (World Health organisation, n. d.). Clean water supply and adequate systems for sewage disposal (Chiodini. P. L., 2008), can reduce contamination via water supply. Testing the animals preslaughter for virulent E. coli can reduce the large numbers of new pathogens in the slaughtering environment. Hygienic slaughtering practices can be implemented to reduce contamination of carcasses by faeces, and food sources can also have reduced risk of contamination from good hygienic practises. However, to further eliminate E. coli from foods, the only effective method is to introduce a bactericidal treatment, such as heating or irradiation. Also in retailers and at home contamination can be reduced by simply keeping raw and cooked foods separate and cooking thoroughly; cooking above 70°C should kill virulent strains of E. coli. (World Health organisation, n. d.).

## Conclusion

Food-borne E. coli infections can cause serious outbreaks and can cause serious illness and sometimes fatality. Treatment is limited, so prevention by controlling food processing is essential for controlling these outbreaks.

However, E. coli is very versatile, adaptive and with its ability to communicate and transfer it’s DNA, new strains are being created, and becoming resistant to current treatment and control methods. More research in this area is needed before full control can be gained.