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Escherchia Coli O157:H7 as an Important Cause of Food Borne Illness

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Abstract
Escherichia coli O157 is the most common member of a group of pathogenic *E. coli* strains and a cause for food borne illness. Transmission of *E. coli* O157:H7 to humans is principally via contamination of food by animal faeces, with cattle considered to be the primary reservoir. Typical illness as a result of an *E. coli* O157:H7 infection in humans can be life threatening, and susceptible individuals show a range of symptoms including haemolytic colitis, hemolytic-uremic syndrome, and thrombotic thrombocytopenia purpura. People of all ages are susceptible to infection with enterohemorrhagic *E. coli* O157:H7. However, the young and the elderly are more susceptible and are more likely to develop more serious symptoms. The enteric habitat of *E. coli* in animals provides easy access to animal-derived meats at slaughter and at points downstream in the food production process. An effective control program to substantially reduce *E. coli* O157:H7 infections will require the implementation of intervention strategies throughout the food continuum, from farm to table.
Keywords: *E. coli* O157:H7, Enterohemorrhagic, Haemolytic uraemic syndrome, Shiga-like toxin

Introduction

Food-borne diseases often follow the consumption of contaminated food-stuffs especially from animal products such as meat from infected animals or carcasses contaminated with pathogenic bacteria (Nouichi and Hamdi, 2009; Pal, 2012). One of the most significant food-borne pathogens that have gained increased attention in recent years is *E. coli* O157:H7. It is an enterohemorrhagic strain of the bacterium *Escherichia coli* and a cause of food borne illness (Pal, 2007). Typical illness as a result of an *E. coli* O157:H7 infection can be life threatening, and susceptible individuals show a range of symptoms including haemolytic colitis, hemolytic-uremic syndrome, and thrombotic thrombocytopenia purpura (Sima *et al*., 2009; Chileshe and Ateba, 2013).

Domestic and wild animals are the sources of *E. coli* O157, but the major animal carriers are healthy domesticated ruminants, primarily cattle and, to lesser extent, sheep, and possibly goat (Sima *et al*., 2009; Kiranmayi *et al*., 2010; Rahimi *et al*., 2012a). Transmission of *E. coli* O157:H7 to humans is principally via contamination of food by animal faeces, with cattle considered to be the primary reservoir (Hancock *et al*., 1997). Sporadic cases and outbreaks of human diseases caused by *E. coli* O157 have been linked to ground beef, raw milk, meat and dairy products, vegetables, unpasteurized fruit juices and water (Sima *et al*., 2009). There are also traceable links between human infection and ruminant faeces via water or direct contact (Licence *et al*., 2001; Strachan *et al*., 2001), and evidence that contact with animal faeces is a strong risk factor for sporadic *E. coli* O157:H7 infection (Locking *et al*., 2001).

The enteric habitat of *E. coli* in animals provides easy access to animal-derived meats at slaughter and at points downstream in the food production process (Olatoye e*t al*., 2012). Possible contamination of edible carcass tissue is the most significant challenge to food safety, and the extent and nature of such contamination are related to the *E. coli* O157:H7 status of the pre slaughter animal, and any processes which distribute the organism within or between carcasses during dressing operations (McEvoy *et al*., 2003).

The organism and its characteristics

Shiga-toxin producing E. coli

All shiga toxin producing *E. coli* (STEC) including serotype O157:H7 have the same morphology. They are Gram-negative, facultative anaerobic bacteria that belong to the *Enterobacteriaceae* family and the *Escherichia* genus (Xia, 2010; Farrokh *et al*., 2012). *Escherchia coli* O157:H7 produce shiga toxin which is an important cause of food borne illness in human and ruminants where they appear to be more frequently colonized by *E. coli* STEC than other animals, but the reason for this is unknown (Cornick *et al*., 2000).

Growth and inactivation

Serotype O157:H7 has been shown to grow well in broth media within the usual laboratory temperature range of 30-42°C and it survives freezing in ground beef quite well. At temperatures above 44-45°C serotype O157:H7 grows poorly and as these temperatures are often used for the detection of *E. coli* in food samples, such conditions probably will negatively impact on the recovery of this serotype from food (Hui *et al*., 2001). The other publication has also shown that *E. coli* O157 strains possess inherent genetic mechanisms which enable growth at low temperatures (<15 °C), compared to non-pathogenic *E. coli* (Vidovic *et al*., 2011).

Biochemical properties

E. coli can be differentiated from other members of the *Enterobacteriaceae* on the basis of a number of sugar-fermentation and other biochemical tests. Classically an important group of tests used for this purpose are known by the acronym IMViC. These tested for the ability to produce: indole from tryptophan (I); sufficient acid to reduce the medium pH below 4.4, the break point of the indicator methyl red (M); acetoin (acetylmethyl carbinol) (V); and the ability to utilise citrate (C) (Adams and Moss, 2008). The majority of *E. coli* O157:H7 strains can be distinguished from most *E. coli* by their inability to ferment sorbitol rapidly and by their lack of production of b-glucuronidase. Although rapid sorbitol-fermenting strains of *E. coli* O157:H7 have been associated with colitis and HUS in Germany, these strains are rarely isolated in the United States (Besser *et al*., 1999).

Acid and salt tolerance:

E.coli O157:H7 is a highly acid-resistant food-borne pathogen that survives in the acidic environment of stomach and to colonise the gastrointestinal tract (Price *et al*., 2004). Furthermore, it also increases the survival of STEC O157:H7 in acidic foods, enabling survival for extended periods, particularly at refrigeration temperature (Meng *et al*., 2007). Hence, contaminated cultured and fermented foods such as yoghurt and cheese have been implicated in sporadic cases and outbreaks (Baylis, 2009; Farrokh *et al*., 2012). The doubling time of *E. coli* O157: H7 increases by three fold in 4.5% NaCl in broth whereas at 6.5% a 36 hours lag was noted with a generation time of 31.7 hours and no growth occurred at ≥ 8.5% NaCl (Jay, 2000).

Carriage of a 60-MDa plasmid

E.coli O157:H7 isolates associated with human illness harbour a plasmid (pO157) of approximately 60 MDa that contains DNA sequences common to plasmids present in other serotypes of VTEC isolated from patients with haemorrhagic colitis. The plasmid is believed to play a role in the pathogenicity of disease (Fernandez, 2008; Tshabalala, 2011).

Epidemiology of enteric *E.coli* O157:H7

Distribution

The first STEC O157 infections were reported in 1982, when *E. coli* O157:H7 was involved in outbreaks associated with two fast food chain restaurants in the United States. These isolates were obtained from fecal samples taken from sporadic cases of hemorrhagic diarrhea submitted to public health or hospital laboratories for examination (Acha and Szyfres, 2001). Since then, ever-increasing numbers of cases and outbreaks due to STEC O157 have been reported worldwide. *E. coli O157:H7* was the causative agent of many out-breaks worldwide (Xia *et al.*, 2010). For instance, serotype O157:H7 has been isolated in outbreaks in Canada, Great Britain, and the United States. It has also been isolated in Argentina, Australia, Belgium, the former Czechoslovakia, China, Germany, Holland, Ireland, Italy, Japan, and South Africa. Reports from Africa (Effler *et al*., 2001) have shown that rates of O157:H7 infections but in countries lacking diagnostic capabilities might be underestimated (Tarr *et al.,* 2005). Annual incidence rates of 8 per 100,000 inhabitants or greater have been reported in the region of Scotland, Canada and USA (Constantiniu, 2002).

Susceptibility

Cattle are generally regarded as the main natural reservoir of O157 STEC. All ages of cattle are susceptible to colonization with O157 STEC, although peak shedding is observed in sub adult cattle from weaning to 24 months of age (Hussein and Sakuma, 2005; Joris *et al*., 2012). People of all ages are susceptible to infection with STEC. However, the young and the elderly are more susceptible and are more likely to develop more serious symptoms (FDA, 2012).

Mode of transmission

E. coli O157 is transmitted by food and water, directly from one person to another, and occasionally through occupational exposure. Most food borne outbreaks have been traced to foods derived from cattle, especially ground beef and raw milk (Constantiniu, 2002; Fairbrother and Nadeau, 2006; Gyles, 2007).

Among many foods and dairy products acted as vectors :-ground beef hamburgers; steak tenderised by injection; steak tartare; kebabs; ready-to-eat cold meats including poultry, pork, and beef products; salami and other fermented meat products; venison jerky; cheese; milk; butter; yoghurt; ice cream; apple juice; grapes; coleslaw; lettuce; spinach; radishes; alfalfa sprouts; and melons are mentioned (Pennington, 2010).

Outbreaks of O157 STEC most commonly occurred in restaurants, often due to cross-contamination during food preparation. Person-to-person transmission via the faecal-oral route has been an important mode of transmission, particularly since the early 1990s, and occurs mostly in child day care centres, individual homes, communities, and schools (Pennington, 2010).

Waterborne outbreaks of O157 STEC associated with recreational waters, such as lakes, swimming pools, and contaminated drinking water, have been increasingly reported since the early 1990s. Outbreaks associated with contaminated water tend to be larger in size and have been attributed to local well, municipal, and spring water systems. Since 1996, outbreaks resulting from a new transmission mode have been recognised, i.e. direct contact between humans and cows or calves at farms, fairs, or petting zoos. For the most part, the

modes of transmission in other industrialised countries appear to be similar to those observed in the USA (Effler *et al*., 2001; Fairbrother and Nadeau, 2006).

As more data become available from developing countries, other modes of transmission specific for the environmental, demographic, and farming conditions in these countries will certainly be elucidated. For instance, a large outbreak of bloody diarrhoea due to O157 STEC in South Africa in 1992 was the result of a combination of carriage of O157 STEC by pastured cattle, cattle deaths due to drought, and ensuing heavy rains resulting in contamination of surface waters (Effler *et al*., 2001; Fairbrother and Nadeau, 2006).

Carrier and sources of infection

Domestic and wild animals are sources of *E.coli* O157:H7 but the major animal carriers are healthy domesticated ruminants, primarily cattle and to a lesser extent, sheep, and possibly goats (Kiranmayi *et al*., 2010; Rahimi *et al*., 2012a). Faeces and hides of cattle are considered to be the main sources of *E. coli* O157 contamination of carcasses during slaughter (Elder *et al*., 2000; Aslam *et al*., 2003).

The main sources of *E.coli* O157:H7 infection in cattle are drinking water, feed, and the environment of the animal. The environment may be contaminated by cattle carrying the bacteria as well as by production animals of other species (e.g. sheep, goats, or pigs), by companion animals (e.g. dogs, cats, or horses), by wild animal species (e.g. deer), or by insects (e.g. flies). Infection may also occur through direct contact with other cattle or animals of other species (Fairbrother and Nadeau, 2006).

A plethora of fecal-contaminated food items including ground meat, unpasteurized dairy products, unpasteurized refreshments, fruits and vegetables (such as sprouts, lettuce, coleslaw) have been well-known vehicles for O157 STEC infections (Karmali, 2004; Schlundt *et al*., 2004, Caprioli *et al*., 2005). In addition, waterborne infections (Garcia-Aljaro *et al*., 2005), and infections associated with rural settings have been of growing importance (Karmali, 2004). In particular, environment-related exposures have been associated with O157 STEC infections during summer and fall (Karmali, 2004; Caprioli *et al*., 2005).

Pathogenesis and clinical features

Pathogenicity of *E. coli* O157:H7 is encoded by a variety of plasmid, bacteriophage and chromosomal genes (Kiranmayi *et al*., 2010). The key virulence factor for subset of EHEC is Stx which consists of five identical B subunits that are responsible for binding the holotoxin to the glycolipid globotriaosylceramide (Gb3) on the target cell surface, and a single A subunit that cleaves ribosomal RNA, causing protein synthesis to cease (Kaper *et al*., 2004).

The ability to produce shiga toxin was acquired from a bacteriophage presumably directly or indirectly from *Shigella* (Kiranmayi *et al*., 2010). The Stx family contains two subgroups -Stx1 and Stx2-that share approximately 55% amino acid homology (Kaper *et al*., 2004). The production of Shiga toxin is central to the pathogenesis of bloody diarrhoea and haemolytic uremic syndrome (Pennington, 2010). Stx is produced in the colon and travels by the bloodstream to the kidney, where it damages renal endothelial cells and occludes the microvasculature through a combination of direct toxicity and induction of local cytokine and chemokine production, resulting in renal inflammation. Stx also mediates local damage in the colon, which results in bloody diarrhoea, haemorrhagic colitis, necrosis and intestinal perforation (Kaper *et al*., 2004).

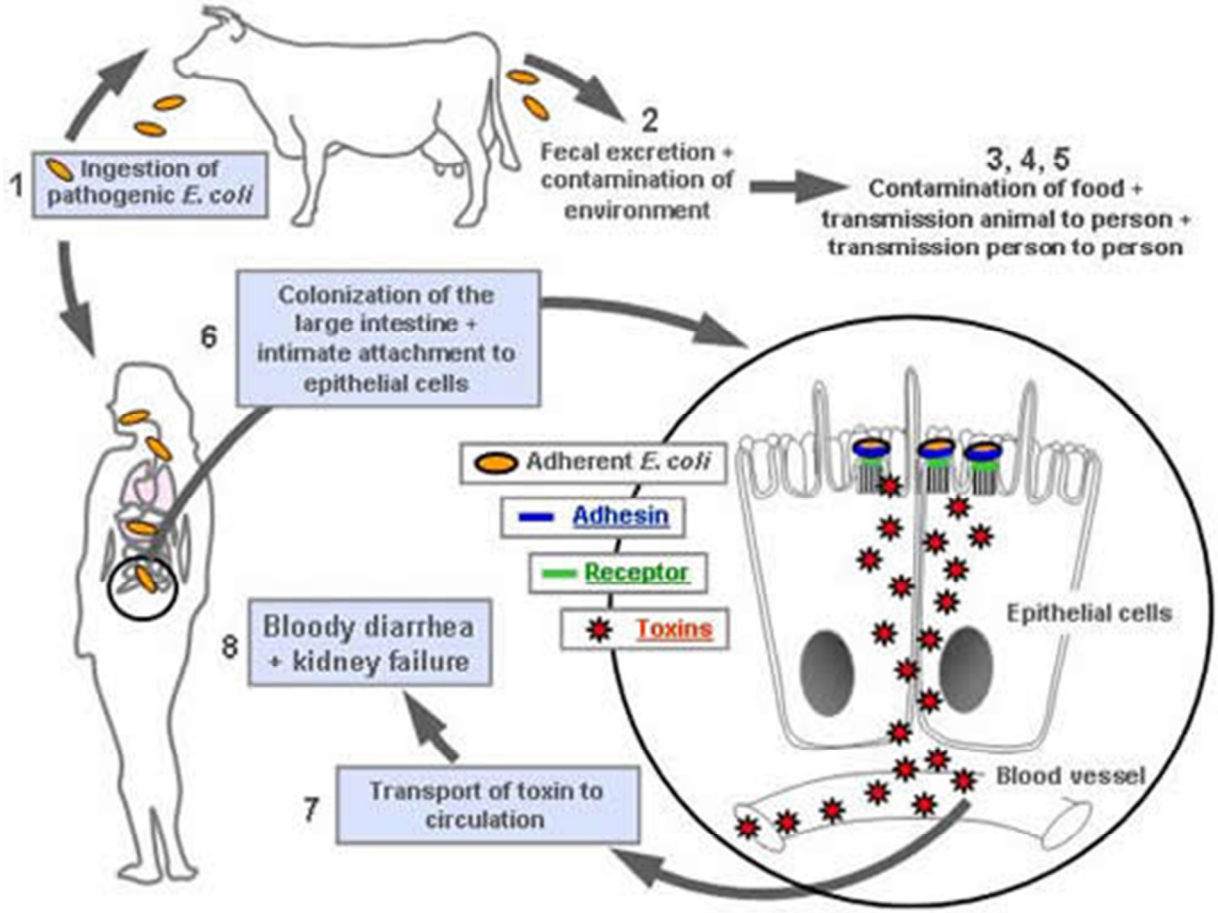

Figure 1: How zoonotic shiga toxin-producing *Escherichia coli* (STEC) cause bloody diarrhoea and haemolytic uraemic syndrome in humans (www.ecl-lab.ca).

Potentially pathogenic bacteria are ingested by cattle and other ruminants (1) and colonize the intestinal tract, but do not cause any disease in these animals. The bacteria are excreted in the feces and contaminate the environment, including the drinking and swimming water of the human population (2). There may also be contamination of foods such as fruits, vegetables, sprouts, lettuce, and raw milk and juice (3). There may be contamination of the carcass at slaughter, and bacteria will be mixed into ground beef. Persons in direct contact with animals, who are working on farms or in slaughter-houses, may also be contaminated by the bacteria (4). There may also be spread of bacteria from person to person (5). In humans, these bacteria colonize mostly the large intestine and cause similar attaching and effacing lesions (6) (Fig. 1).

Bacteria produce their own specific receptor which is injected into the host epithelial cell via a syringe-like bacterial apparatus. A bacterial adhesin then mediates a very intimate attachment of the bacteria to the cell receptors and bacterial signals stimulate effacement of the microvilli, or brush border, and reorganization of the cell cytoskeleton. The adherent bacteria produce a toxin which is transported across the epithelial cells to the circulation (7). This toxin acts on the endothelial cells of blood vessels, resulting in non-bloody to bloody diarrhea and abdominal cramps (8). There may be a complication of hemolytic uremic syndrome which may lead to acute kidney failure, especially in children (www.ecl-lab.ca).

The infective dose of *E. coli* O157:H7 is estimated to be very low, in the range of 10-100 cells. The infective dose of other STEC serotypes is suspected to be slightly higher (FDA, 2012).The pathogenicity of *Escherichia coli* O157:H7 is associated with a number of virulence factors, including shiga toxins (Stx1 and Stx2; encoded by the stx1 and stx2 genes), intimin (encoded by the eae gene) and the enterohaemolysin (encoded by the hlyA gene) (Manna *et al.,* 2006; Kiranmayi *et al*., 2010; Xia *et al.*, 2010).

The toxin is a 70.000 dalton protein composed of a single A subunit (32 kDal) and five B subunits (7.7 kDal). The A subunit has an N-glycosidase that inactivates the 28S ribosome, thus blocking protein synthesis. The B subunits provide tissue specificity by binding to globotriaosylceramide (Gb3) receptors on the surface of eukaryotic cells. Endothelial cells high in Gb3 receptors are the primary target, accounting for the toxin's affinity for colon and renal glomeruli, associated with HC and HUS. The toxin can also indirectly damage cells by releasing cytokines, such as tumour necrosis factor (Constantiniu, 2002).

Within the Stx2, there are additional antigenic variants. The Stx2v (variant)-producing *E. coli* is

associated with diseases in domestic animals, such as edema disease of swine. Enterohemorrhagic *E. coli* that commonly cause human illnesses produce Stx1, Stx2, or both. The presence of the Stx2 in these EHEC has a profound influence on the progression of the disease from hemorrhagic colitis to HUS. As is common for many bacterial toxins, Stx consists of 2 subunits. The Stx-A subunit contains the enzymatic activity responsible for inhibiting protein synthesis, and the B-subunit acts as a lectin, binding the toxins to intestinal epithelial and kidney endothelial cells. The Stx is believed to be the major factor contributing to the lesions in HUS, although the O157 lipopolysaccharide may also contribute to this disease syndrome (Sanchez *et al*., 2002).

The clinical manifestations of *E. coli* O157 and other VTEC serotypes infections range from symptom-free carriage to non-bloody diarrhoea, haemorrhagic colitis (a triade of severe abdominal pain, diarrhoea and frank red blood), HUS and death. The course of events in VTEC infection starts with the ingestion of the pathogen (Constantiniu, 2002). Haemolytic uremic syndrome is characterized by three features, acute renal failure, haemolytic anaemia (reduction in the number of red blood cells) and thrombocytopaenia (a drop in the number of blood platelets), sometimes preceded by a bloody diarrhoea. Thrombotic thrombocytopaenic purpura is a less common complication which is largely confined to adults. It is related to HUS but causes less kidney damage and includes fever and neurological symptoms resulting from blood clots in the brain (Adams and Moss, 2008).

Host responses to E.coli O157:H7 infection

Infection of the gastrointestinal tract of adult cattle, weaned calves and 5-day-old gnotobiotic calves by serotype O157:H7 is asymptomatic (Wray *et al*., 2000). Histological analysis of intestinal epithelia from calves and cattle infected with *E. coli* O157:H7 reveals intimate bacterial adherence in some but not all cases and a mild inflammatory response characterized by diffuse infiltration of neutrophils into the lamina propria (Stevens *et al*., 2002).

Serum antibody responses against the O157 lipopolysaccharide and Shiga toxin 1 have been detected in some but not all experimentally infected calves (Wray *et al*., 2000) and sheep (Cornick *et al*., 2000; Stevens *et al*., 2002).

It is likely that immunity plays a role in the susceptibility to infection with *E. coli* O157:H7, as evidenced by the increased rates of infection and HUS in young children and the elderly. Although antibodies to O157 LPS and shiga toxin 1 rise after acute infection, protective immunity has not been demonstrated in humans, and *E. coli* O157:H7 infection has caused recurrent hemorrhagic colitis and HUS in children without apparent immunodeficiencies (Besser *et al*., 1999).

Diagnosis

Detection of *E. coli O157:H7* is based on phenotypic differences from most other serotypes: its inability to ferment sorbitol on MacConkey sorbitol agar and absence of b-glucuronidase activity in most strains. Presumptive *E. coli* O157:H7 from these tests must then be confirmed serologically for which a latex agglutination kit is commercially available (Adams and Moss, 2008).

Identification of diarrhoeagenic *E. coli* can be based on detection of their associated virulence factors. For example, procedures are available to detect the ST and LT of ETEC serologically, and the LTI and Stx genes in ETEC and EHEC using gene probes and the polymerase chain reaction (PCR) (Adams and Moss, 2008).

Treatment

The use of antibiotics in the treatment of STEC infection is controversial (Panos *et al*., 2006; Ochoa *et al*., 2007). Some authors reported that antibiotics may have beneficial effects in STEC infection and reduce the risk of STEC-associated complications (Kurioka *et al*., 1999) while others reported an increase in the level of shiga toxin production and a greater risk of fatal complications following administration of antibiotics in STEC infection (Zhang *et al*., 2000; Wong *et al*., 2000). In vitro studies showing most strains are susceptible to various antibiotics, although certain antibiotics, at sublethal concentrations may increase the release of Shiga-like toxin which has been associated with the development of HUS. No clinical studies have indicated that antibiotics are effective in reducing the duration of *E. coli* infection or duration of bloody diarrhea **(**Collins and Green, 2010). In vitro data have demonstrated that ciprofloxacin or subinhibitory concentrations of trimethoprim-sulfamethoxazole induce shiga toxin production by *E. coli* O157:H7 (Besser *et al*., 1999).

Treatment of HUS is supportive, with particular attention to the management of fluids and electrolytes. With meticulous care, the mortality rate for HUS is approximately 4%. Numerous other treatment modalities have been tried but are of unproven efficacy. These include plasma infusion, plasma exchange, intravenous immunoglobulin, Shiga toxin inhibitors, prostacyclin, antithrombotic therapy, vitamin E, recombinant tissue plasminogen activator, and transfusion with P1-positive erythrocytes (Besser *et al*., 1999).

Control and prevention of E. coli O157:H7 infection

An effective control program to substantially reduce *E. coli* O157:H7 infections will require the implementation of intervention strategies throughout the food continuum, from farm to table. Promising intervention measures at the farm include competitive exclusion bacteria, bacteriophage, and targeted animal management practices addressing common points of contamination. Consumers also have a role in implementing intervention controls

in food handling and preparation. Unfortunately, many consumers eat high-risk foods, improperly handle and store foods, and ignore warnings regarding foods known to be unsafe (Sanchez *et al*., 2002).

Ground beef should be cooked until it is no longer pink. Meat from cattle, like that of other mammalian and avian species, can be contaminated by feces during slaughter and processing. Thus, all precautions should be taken to minimize this risk, and foods of animal origin should be well cooked before they are eaten. Personal hygiene, particularly hand washing after relieving oneself, is also important (Acha and Szyfres, 2001; Pal, 2007).

To control the risk of human infection through direct contact with farm animals, strict hygiene practices should be established, including controlling the movement of visitors to farms, restricting access to farm animals., making washing facilities readily available, providing a means of disinfection in case visitors come into contact with the animals, and segregating eating areas from areas where the animals are kept (Fairbrother and Nadeau, 2006).

The commonly accepted rules of herd management should be followed in animals. For calves, colostrum is important for the prevention of white scours, and for pigs, all unnecessary stress should be avoided during weaning in order to prevent edema (Acha and Szyfres, 2001).

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