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RESEARCH

Epidemiology of Shiga toxin-producing *Escherichia coli* O157:H7 in Africa in review

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Shiga toxin-producing *Escherichia coli* (STEC) O157:H7 is responsible for intestinal and extra-intestinal disease syndromes in human. Isolation of the pathogen from animals, food, clinical samples and environment has been reported from all continents. A review of STEC O157:H7 in Africa from a structured literature search of the PubMed electronic database is presented. It describes the epidemiological status of the pathogen on the aspects of source, transmission, pathogenesis, disease syndromes, diagnosis, disease burden and the challenges in treatment and control strategies. About a quarter of African countries have reported isolation of STEC O157:H7 either from humans, animals, food or the environment. Different methods have been used in detection of the pathogen. Most reported human infections do not show temporal relationships with reports of isolation of the pathogen from other sources such as animals, water or food. Lack of a direct link between isolates from humans and other sources makes it difficult to point out incident specific determinants and direction of transmission. The aim of this review is to give an insight into the features of STEC O157:H7 infection in Africa and draw the attention of various stakeholders to the public health threat of the pathogen for possible interdisciplinary and multi-sectoral joint efforts in the control strategies.

Keywords: Africa, *E. coli* O157:H7, HUS, Shiga toxin, STEC

Introduction

Escherichia coli strains that cause diarrhea in humans are either enterotoxigenic *E. coli* (ETEC), enteroaggregative *E. coli* (EAEC), enteropathogenic *E. coli* (EPEC), enteroinvasive *E. coli* (EIEC), diffusely adherent *E. coli* (DAEC) or verocytotoxigenic *E. coli* (VTEC).[1] One of the VTEC stains associated with diarrhea, bloody diarrhea, haemorrhagic colitis and haemolytic uremic syndrome (HUS) is the Shiga toxin-producing *Escherichia coli* (STEC) O157:H7.[2] Once described as a rare serotype causing human infection,[3] STEC O157:H7 is now widespread in food products[4–6] and the environment.[7,8] This prevalent nature and other biological characteristics, such as low infective dose,[7,9–11] ability to express different virulence factors,[9] long survival time in the environment[12] and the difficulty in treatment,[13] make STEC O157:H7 an enteric pathogen of major concern worldwide. This report reviews the epidemiology of the pathogen with focus on (1) distribution, (2) disease manifestation, (3) pathogenicity, (4) isolation and characterization, (5) treatment, (6) disease burden and (7) prone groups. The aim is draw the attention of public health stakeholder to this health problem in Africa so that multi-disciplinary joint efforts can be applied in the control strategies.

Methodology

A search algorithm with terms 'shiga-toxigenic *Escherichia coli*' OR 'shiga-toxigenic' AND '*Escherichia*' AND '*coli*' OR 'shiga-toxigenic *Escherichia coli*' OR 'STEC' AND 'O157' AND 'H7' AND 'Africa' was developed in a PubMed data base on 20 June 2015 to search for articles published in last 10 years. A total of 68 journal articles were initially obtained and 37 of them were selected for the report because they described subjects of interest. A search through the reference list of the selected articles provided additional references about the pathogen in relation to hosts, reservoirs, isolation techniques, virulence, pathogenicity, clinical signs, disease burden, treatment, control and threat to the immunocompromised population. Some references of non-African context are included in this review to cover general issues for a broader view of the pathogen.

The burden of STEC O157:H7 infection

Globally STEC causes 2 801 000 acute illnesses annually, with an incidence rate of 43.1 cases per 100 000 person-years. This burden leads to 3 890 cases of HUS and 230 deaths. Among those, a total of 10 200 cases of STEC infections occur in Africa with an incidence rate of 1.4 cases per 100 000 person-years. STEC O157:H7 contributes 10% to this burden.[14]

Reports of STEC O157:H7 Occurrence in Africa

Shiga toxin-producing *Escherichia coli* O157:H7 isolation has been reported from all zones of Africa (East, Central, South, North and West Africa) from humans, animals, food products and the environment. The first case of human infection was reported back in 1990 in Johannesburg, South Africa.[15]

In central Africa, the pathogen has been isolated in humans with haemorrhagic colitis in Bangui, Central African Republic in 1996, which led to mortalities.[13] In 1998, STEC O157:H7 isolation from humans was reported following an outbreak of bloody diarrhoea in Cameroun.[16]

In east Africa, isolation of the pathogen has been reported in Tanzania, Kenya and Ethiopia. A STEC O157:H7 prevalence of more than 7% was reported in patients with diarrhoea in Morogoro, Tanzania in 2006.[17] In 2012, the pathogen was isolated from cattle in the same area with a prevalence of 0.9%.[18] In Kenya, STEC O157:H7 was isolated from a two-year-old boy with haemorrhagic colitis.[19] Milk and cattle faeces subsequently tested positive for the pathogen in the same country.[6] STEC O157:H7 has been isolated from beef, mutton and chevon in Ethiopia at a prevalence of 8, 2.5 and 2%, respectively,[20] as well as goat and sheep faeces (4.7%), skin swabs (8.7%), carcass before washing (8.1%), carcass after washing (8.7%) and water samples (4.2%).[21]

Reports on STEC O157:H7 occurrence are available from Algeria, Morocco, Tunisia and Egypt (north Africa). A study in Algeria reported a prevalence of 7% from bovine carcasses.[5] In Morocco, a prevalence of 9.1% from dairy products and 11.1% in meat marketed in Rabat have been reported.[22] STEC O157:H7 was again isolated in Morocco from raw meat products at a proportion of 9%.[4] In 2011, a 1.9% prevalence from shellfish in Mediterranean coastline of Morocco was reported.[23] In Tunisia, 3.4% of isolates from human stool samples were shiga toxin-producing *E. coli* O157:H7.[24] Isolation of the pathogen from different sources has also been documented from Egypt. For instance, a survey in Egypt revealed that a prevalence of 6% from beef samples, 4% from chicken samples, 4% from lamb samples and 6% from milk samples was obtained in slaughterhouses, supermarkets and farmers' homes.[25]

In west Africa, much of the work reported on STEC O157:H7 has been from Nigeria. In Lagos, a prevalence of 6% from patients with diarrhoea has been documented.[26] In the city of Ibadan, STEC O157:H7 has been isolated from the faeces of cattle, sheep, goat and pig, and also from beef, chevon (goat) and pork with a prevalence of 5%.[27] In Zaria, the strain has been isolated from the diarrheal stool of children under the age of 5 years with a prevalence of 5.4% and from surface water at a proportion of 2.2%.[28] The STEC O157:H7 isolation in Nigeria provides evidence of occurrence of the pathogen in human, animals, meat and environment (water). A study in the coastal savannah zone of Ghana did not report on the isolation of *E. coli* O157:H7 in raw milk and milk products,[29] but this does not guarantee absence of the pathogen. Information on recovery of *E. coli* O157:H7 from other western African countries, including Mali, Niger, Guinea, Ivory Coast, Togo, Benin, Guinea Bissau, Sierra Leone, Liberia, Mauritania, Cape Verde and Burkina Faso, were not accessed. But given the similarity between environments, there is high chance that this pathogenic *E. coli* strain exists in these countries. The lack of reports on *E. coli* O157:H7 isolation in some African countries may be due to poor diagnostic facilities, especially in rural settings where infections may pass undiagnosed.

The southern African region is comprised of Zambia, Malawi, Mozambique, Zimbabwe, Botswana, Namibia, Swaziland, Lesotho and South Africa. In South Africa, a 10.3% prevalence of STEC O157:H7 from vegetable samples in Eastern Cape province was documented.[30] Meat and meat products from the same location carried the pathogen at a proportion of 2.8%.[31] Further studies reported a prevalence of 56.5% and 43.5% from stool of confirmed and non-confirmed HIV/AIDS patients, respectively, in the Eastern Cape province.[32] The STEC O157:H7 isolates from meat products (7.8%), water (8.6%), vegetables (10.3%), confirmed HIV/AIDS patients (56.5%) and non-confirmed HIV/AIDS patients (43.5%) were genetically related;[30] and, hence, provided evidence on the possible transfer of the pathogens between different study components. In the neighbouring country of Botswana, the prevalence of STEC O157:H7 in meat cubes, minced meat and fresh sausages in Gaborone were reported to be 5.22, 3.76 and 2.26%, respectively.[33] These findings from beef-product outlets put consumers at risk of infection. Home cooked food samples (maize flour porridge, fish, vegetables and beans) investigated for pathogenic bacteria, were found to be contaminated with STEC O157:H7 at a proportion of 8% in Lungwena, Malawi.[34] In Mozambique, the pathogen was reported to be one of the causes of diarrhoea in children at a proportion of 1.9%.[35] STEC O157:H7 was reported to also cause dysentery in HIV patients in Zimbabwe at a prevalence of 8%.[36]

Therefore, reports on isolation of pathogenic *E. coli* O157:H7 from all regions of the African continent (east, west, south, north and central) show that the pathogen is found throughout Africa. A total of 15 countries have reported recovery of pathogenic *E. coli* O157:H7 either from humans, animals, food products or the environment. Out of 30 reviewed cases, 10 (33.3%) come from human patients and the remaining 20 isolations (66.7%) belong to food stuffs,[8] cattle,[5] water[3] and others (2 sheep and goats, 1 vegetable and 1 shell fish) (Table 1).

Transmission of STEC O157:H7

STEC O157:H7 is an enteric pathogen that is transmitted to humans through ingestion of contaminated food, or hands to mouth.[7,37] Person-to-person contact can lead to transmission of the pathogen through the oral-faecal route.[9] The infectious dose that has caused disease symptoms in humans has been reported to be as low as 4 to 24 organisms.[7,11] Ruminants are said to be reservoirs, whereby cattle are regarded as principal sources of infections.[6,38–42] However other ruminant species, such as goats, sheep,[21,27] and buffaloes,[43] serve as a source of the pathogen, with the exception for camels.[44] Non-ruminant animals such as pigs[27,38,39] and pigeons[45] are also reported to carry this strain of pathogenic *E. coli*. Fish in contaminated water have been reported to harbour STEC O157:H7.[46] A single dose of 100 CFU is sufficient to infect cattle,[47] while sheep have been been infected by a single oral dose of 10^5 CFU.[48] These doses can be acquired by ingestion of as little as 0.1 g of manure containing 10^6 CFU/g.[48] Shedding of the pathogen in cattle is intermittent,[45,49] the duration of shedding by cattle is less than a month and shedding peaks occur during the months of summer.[49,50] Weaning calves are reported to shed more bacteria than other age groups.[50,51] These findings suggest that having negative results at a particular point in time does not indicate absence of STEC O157:H7. Moreover, the reported prevalence of STEC O157:H7 may be lower or higher than the real situation depending on the composition of cattle, by age, in the study.

Accidental ingestion of STEC O157:H7 following contact with infected animals or the contaminated environment has led to human infection.[7,52,53] Contaminated food products such as beef,[4,5,20,22,25,33] chevon, mutton,[20,21,25] milk and chicken may lead to human infection.[22,25] Marine environmental contamination has also posed a risk because of isolation of the pathogen from shellfish.[23] Convenient foods under poor preparation or handling have also been reported to play a role in propagation of this pathogen.[37] Moreover, inanimate objects such as soil,[7] water,[28,46] marine sediments[23] and manure[50] are a source of the pathogen. The risk is potentiated by the ability of the pathogen to survive harsh conditions, such as the low pH of dairy products,[54,55] or in manure for more than four months.[48] Generally, the risk factors for STEC O157:H7 infections include contact with animals and their environment and poor personal hygiene, such as not washing hands after handling animals or prior to eating.[7,52,53] These findings and reports call for hygiene observance after contact with animals, the suspected environment or during preparation of foods.

In Africa, evidence of STEC O157:H7 transmission between humans, animals and environment is not clear. From the reports in this review, isolation of STEC O157:H7 from humans was driven by the occurrence of disease syndromes, such as diarrhoea, while detection of the pathogen from animals, animal product and the environment was part of routine research work. There is no

temporal relationship in isolation from these two ends. Under such a scenario, it is difficult to establish events and direction of transmission, as well as to quantify the risk of pathogen transfer between humans, livestock and the environment. There is a need to investigate the possible sources and to quantify risk factors every time STEC O157:H7 is isolated from humans to ensure that prevention and control strategies are appropriate.

Isolation and characterisation of STEC O157:H7

Like any other member of the family Enterobacteriaceae, Shiga toxin-producing *Escherichia coli* O157:7 can be isolated on MacConkey agar, followed by conventional biochemical or serological tests to confirm that the isolates are *E. coli*. Isolation can also be done by use of sorbitol MacConkey agar whereby most STEC O157:H7 are distinguished from other strains by their inability to ferment sorbitol. Direct inoculation of a sample on sorbitol MacConkey agar has been employed, but has been proven to be less sensitive compared to immunomagnetic separation.[56,57] Some studies have employed both sorbitol MacConkey agar and immunomagnetic separation to maximise the chances of isolating the pathogen.[32] STEC O157:H7 strains should be distinguished from Non-O157:H7 strains, which also do not ferment sorbitol.[18] Either of these *E. coli* isolation options can be accomplished by performing an agglutination test using antibodies against a somatic antigen for O157:H7 and a flagella antigen for H7. Polymerase chain reaction (PCR) for the detection of shiga toxin-producing genes in *E. coli* O157:H7 remains a gold standard detection method.[57] Detection of the bacteria or toxins may take more than 24 h.[58] In some instances, DNA hybridisation has been performed to affirm additional virulence genes and phenotypic activities of shiga toxin-producing genes proven by Vero-cell cytotoxicity assay.

In the present review, sorbitol MacConkey agar was used in isolation of STEC O157:H7 in 21 out of 24 reports from Africa. An immunomagnetic separation technique was employed in seven reports, in which it was used together with sorbitol MacConkey agar. After isolation, the characterisation of *E. coli* O157:H7 was done by polymerase chain reaction (PCR) to detect the shiga toxin-producing genes (14 reports), O157 antisera for detection of somatic antigen O157 (18 reports) and dot plot DNA hybridisation was used to confirm PCR results (2 reports). Serotyping of O157:H7 antigens was performed in four studies, while Vero-cell cytotoxicity assays were performed to test for cytopathic effects on Vero-cell monolayers in six studies that are included in this review (Table 1). The use of molecular methods (PCR) to detect shiga toxin-producing genes in only 14 out of 24 (58%) studies in this review could have resulted in missed detection and under-reporting of STEC in Africa. All these STEC O157:H7 detection methods required more than 24 h to complete. Moreover, not many laboratories in Africa can afford these diagnostic procedures. There is a need to improve diagnostic facilities in Africa – even by starting with a few reference laboratories in each African country – which will enable quick and accurate detection of STEC O157:H7 infection. This will help to avoid inappropriate management of cases, such as use of antimicrobials which are easily accessed in Africa and often without prescription, for any enteric illness including STEC O157:H7 infection.

Table 1: Sources and methods of STEC O157 isolation and characterisation in African continent

Country	Source	Isolation and characterisation method*	Author
Central African Republic	Human	PCR	Germani *et al.* 1997
	Cattle, fish, water, environment	SMAC, anti O157 antisera and VCA	Tuyet *et al.* 2006
Cameroon	Human	SMAC, anti O157 antisera, O:H serotyping and VCA	Cunin *et al.* 1999
Tanzania	Human	SMAC, IMS, anti O157 antisera and PCR	Raji *et al.* 2008
	Cattle	SMAC, anti O157 antisera, PCR, DNA hybridization, O:H serotyping and VCA	Lupindu *et al.* 2014
Kenya	Human	PCR, DNA hybridization, VCA	Sang *et al.* 1996
	Cattle, food (milk)	SMAC, anti O157 antisera and PCR	Kang'ethe *et al.* 2007
Ethiopia	Beef, mutton and chevon	SMAC, O:H serotyping	Hiko *et al.* 2008
	Water, sheep, goats	IMS, SMAC, anti O157 antisera and PCR	Mersha *et al.* 2010
Algeria	Cattle (carcass)	SMAC, anti O157:H7 antisera, PCR, DNA hybridization	Chahel *et al.* 2006
Morocco	Food (Dairy and meat products)	IMS, SMAC, anti O157 antisera and PCR	Benkerroum *et al.* 2004
	Raw meat products	IMS, SMAC, anti O157 antisera and PCR	Beneduce *et al.* 2008
	Shellfish	SMAC, anti O157 antisera and PCR	Bennani *et al.* 2011
Tunisia	Human	SMAC, anti O157 antisera, PCR and VCA	Al-Gallas *et al.* 2006
Egypt	Beef, chicken, lamb and milk	SMAC and O:H serotyping	Abdul-Raouf *et al.* 1995
Nigeria	Human	SMAC, anti O157 antisera. VCA	Olorunshola *et al.* 2000
	Cattle, food, sheep, goat, pig	SMAC and anti O157 antisera	Ojo *et al.* 2010
	Human, water	SMAC, anti O157 antisera	Chigor *et al.* 2010
South Africa	Human, vegetable	IMS, SMAC and PCR	Abong'o *et al.* 2008
	Food	IMS, SMAC and PCR	Abong'o and Momba 2009
Botswana	Food (meat)	IMS, SMAC and anti O157 antisera	Magwira *et al.* 2004
Malawi	Maize porridge, vegetable, beans	SMAC, anti O157 antisera	Taulo *et al.* 2008
Mozambique	Human	PCR	Mandomando *et al.* 2007
Zimbabwe	Human	SMAC	Gwavava *et al.* 2001

*SMAC = sorbitol MacConkey agar, IMS = immunomagnetic separation, VCA = Vero-cell assay.

Pathogenicity of STEC O157:H7 infection

STEC O157:H7 possesses different virulence factors that are important in pathogenicity. The major virulence factor is the shiga toxin. Two forms of the toxin, stx1 and stx2 encoded by *stx1* and *stx2* genes are known[59] and reported to be responsible for haemorrhagic uremic syndrome (HUS).[60] The stx1 is divided into three subtypes (stx1a, stx1c and stx1d) while seven subtypes form the stx2 group (stx2a, stx2b, stx2c, stx2d, stx2e, stx2f and stx2g).[61] Of the two groups, subtypes of stx2 are associated with more severe HUS syndrome.[62] Shiga toxins, which are protein molecules, bind to eukaryotic surface cells and inhibit protein synthesis with the death of host cells as a consequence.[63] Intimin is another virulence factor which is coded by attaching and effacing the *eae* gene.[64] Intimin is reported to facilitate attachment of bacteria to intestinal epithelia during colonisation resulting into production of lesions and diarrhoea.[59,65,66] This virulence factor is also possessed by enteropathogenic *E. coli* (EPEC).[67] Enterohaemolysin is another virulence factor for STEC O157:H7. This protein toxin damages cell membranes of erythrocytes and is used as a surrogate tool in detection of shiga toxin-producing *E. coli*.[68–70] Although enterohaemolysin activity can easily be visualised on blood agar cultures, confirmation is usually achieved by PCR amplification of the *ehxA* gene.[59,68] Some other *E. coli* strains such as O26, O103, O111, O118, O128, O121, O45 and O145 can produce disease syndromes and have been reported to be enterohaemolysin-positive and produce shiga toxins.[68,70–72] The synergic effects of these virulence factors make STEC O157:H7 a potential pathogen to humans. All virulence genes, namely *stx1*, *stx2*, *eae* and *ehxA* genes, have been detected in humans, livestock, food products and the environment in eight different combinations as reported in 22 studies from Africa.[8] The most dominant combination was *stx1+stx2*. Cattle are the most common source of STEC O157:H7, as shown in Table 2. Therefore, it is important to consider the use of diagnostic approaches which target different genes so as to increase the sensitivity of STEC O157:H7-related studies.

Disease syndromes caused by STEC O157:H7

To date, STEC O157:H7 has been reported to cause intestinal and extra-intestinal disease symptoms in humans. Disease symptoms may take different forms such as diarrhoea,[17] haemorrhagic colitis[13,19] or haemolytic uremic syndrome.[13] Haemolytic uremic syndrome, which is characterised by thrombocytopenia, haemolytic anaemia and nephropathy, may come as a complication of STEC O157H7 infection following prolonged illness or sometimes disease management such as the use of antibiotics.[73] However, some humans do not show signs of disease despite infection and these are known as asymptomatic carriers.[24,74] Disease syndromes by STEC O157:H7 in Africa have been reported to take the form of an epidemic[13,16] whereby the 1992 outbreak in Swaziland and South Africa are reported to be the largest in Africa.[75] However, sporadic forms of the disease have posed a threat to public health as well.[19]

Table 2: STEC O157:H7 virulence factor combinations from studies in Africa

Gene combination	Source	Reports	Countries
stx1	Cattle feces, milk	2	Kenya
stx2	Cattle feces, human stool	2	CAR* and Kenya
stx1 + eae	Human stool	1	CAR*
stx1 + stx2	Cattle feces, water, fish, human stool, milk, beef, goat, sediment	6	CAR, Ethiopia, Egypt and Morocco
eae + ehxA	Cattle feces, pig	1	South Africa
eae + stx2 + ehxA	Cattle feces, cattle carcass	2	Tanzania and Algeria
stx1 + stx2 + eae	Human stool, beef	2	Cameroon and Morocco
stx1 + stx2 + eae + ehxA	Cattle feces, goat, sheep, pig, human stool	2	Nigeria, South Africa and Tunisia

*Central African Republic.

Treatment of STEC O157:H7 infection

Infections with shiga toxins-producing bacteria such as *Shigella dysenteriae* type I and STEC are controlled by the use of antibiotics and supportive therapies.[13,76] However, in complicated forms of infection, like with HUS, antibiotics are not effective.[13,76] Administration of antibiotics to patients infected with STEC O157:H7 is reported to increase the release of shiga toxins and thus increasing the risk of developing HUS.[13,73] This is thought to be due to the increased release of toxins following death of STEC.[73] The case is different, however, in *S. dysenteriae* type I infection where early antimicrobial therapy lowers the risk of developing HUS.[76] Therefore, it is important to establish the etiology of an enteric disease before administration of antibiotics because it may worsen the prognosis in case of a STEC infection. This demand presents a challenge in developing countries where diagnostics do not match the requirements and antibiotics are haphazardly used.[77,78]

Antimicrobial resistance in STEC O157:H7

Different studies in Africa have reported resistance of STEC O157:H7 to different antimicrobials. For instance, occurrence of multi-drug resistant STEC O157:H7 isolated from humans, animals and the environment has been reported in Egypt,[79] while isolation of multi-drug resistant STEC O157:H7 from cattle in South Africa have also been reported.[80] Similar results have been reported by Chigor *et al*. in Nigeria. Multi-drug resistance may seem of less importance since antimicrobials are not used to treat STEC O157:H7 infection, but there may be a contribution towards selection for resistance genes.

Control of STEC O157:H7 infection

Research on vaccination of reservoirs in an effort to reduce bacteria shedding has shown signs of success,[81] but the practicality of this approach is questionable due to the use of transgenic tobacco plant cells.[9] Some substances such as essential oils from *Cinnamomum zeylanicum* have shown bactericidal activities.[82] But, the above efforts plus dietary manipulations are not promising strategies. Thus, hygienic management of animal and food products remain better options in control of STEC transmission. Moreover, we suggest structuring of an inter-sectoral cooperation between the veterinary (where the main reservoir, cattle, belong) and medical profession (where patients are cared for). A platform for exchange of information and strategies can help in controlling the emergence and spread of the pathogen.

STEC O157:H7 special prone group

Shiga toxin-producing *E. coli* infect all sexes and ages, but many reported cases involve young and elderly people.[19,35] However, the susceptibility spectrum is broadening such that, apart from the usual prone groups of the young and elderly, immunocompromised people form part of a group at risk. Cases of STEC O157:H7 infections in people living with HIV/AIDS have been reported in Africa.[32,36] This poses a big challenge because

Africa has a large share in the global HIV/AIDS burden. Furthermore, complications of STEC O157:H7 infections, e.g. HUS, are aggravated by the use of antibiotics in HIV/AIDS patients and are essential to combat other opportunistic microorganism infections. Subsequently, there becomes imbalance between the desire to alleviate the effects of opportunistic pathogens and shiga-toxins in HIV/AIDS patients due to contrasting outcomes of antimicrobial use. Reports of antibiotic use, such as ciprofloxacin, meropenem, fosfomycin, chloramphenicol, azithromycin and rifaximin, in treatment of STEC O104:H4 infections without induction of shiga toxin release[83,84] are promising. More research on these antibiotics is required to ascertain the possibility of their use to treat STEC O157:H7 patients with HIV/AIDS.

STEC non-O157:H7

Although STEC O157:H7 is the most commonly reported cause of human gastroenteritis, STEC non-O157:H7 pose an increasing risk in public health. When isolation procedures do not specifically target O157:H7 strain, the proportion of STEC isolation skews towards non-O157:H7. In Africa most of the major worldwide-recognised non-O157 serotypes (O103, O111, O145 and O26) have been isolated from different parts of the continent. For instance, in Egypt STEC O26, O114, O125 and O158 have been isolated from humans, cattle, sheep, chickens and water.[85,86] In Tanzania, STEC O113 has been isolated from cattle faeces.[18] In South Africa, screening of STEC isolates from diarrhoeic human patients revealed isolation of STEC O4, O5, O21, O26, O84 and O111, in addition to O157.[72] In the same country, STEC O26 and O145 have been isolated from pig faeces.[87] These reports suggest that whenever STEC-related gastroenteritis is suspected, we should also consider other strains of STEC, not only O157, because failure to isolate O157:H7 may mislead the cause of illness. On the other hand, diagnosis of STEC-related gastroenteritis based on detection of shiga toxins could help in avoiding this discrimination.

The most recent and striking non-STEC O157-related HUS outbreak in German in 2011 was caused by O104:H4 strain. This strain had previously been isolated from diarrhoeic patients in Central African Republic in the mid-nineties.[88]

Conclusion

Isolation of STEC O157:H7 from animals and food products reported from almost all over Africa suggests a high risk for human infection. Lack of proper laboratory facilities, especially in rural settings of Africa, interferes with definitive diagnoses and, hence, patients are treated tentatively. As such, antibiotic prescribed to patients with gastroenteritis can be fatal especially in case of STEC O157:H7 infection. Additionally, difficulty in managing infection cases and time consuming diagnostic procedures call for preventive approaches rather than curative measures. Proper cattle and manure handling practices as well as public awareness on the epidemiology of the pathogen should be instituted. Vehicles of transmission, such as food products and water, should be decontaminated so as to prevent health implications due to STEC O157:H7 infection.

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