

Review

The challenges of foodborne pathogens and antimicrobial chemotherapy: A global perspective

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Accepted 8 March, 2013

Foodborne diseases remain a major cause of morbidity and mortality in the general population, particularly in vulnerable groups. These diseases emanate from either the toxin of the “disease-causing” microbe, or by the human body’s reactions to the microbe. More than 250 different types of viruses, bacteria, parasites, toxins, metals, and prions are associated with foodborne diseases in humans, which are mostly manifested as acute gastroenteritis that could be self-limiting. However, in severe cases, antimicrobial therapy is prerequisite. Antimicrobial resistance is a major challenge in the management of severe foodborne illness; since antimicrobial use in animals selects for resistant foodborne pathogens that may be transmitted to humans as food contaminants. This article presents a comprehensive review on the very important and selected foodborne pathogens and associated illnesses, as well as treatment and control measures.

Key words: Foodborne diseases, viruses, parasites, bacteria, fungi, antimicrobial therapy.

INTRODUCTION

The battle against foodborne diseases is facing new challenges due to the globalization of the food market, climate change and changing patterns of human consumption as fresh and minimally processed foods are currently preferred (Schelin et al., 2011). As food is biological in nature, it is capable of supporting the growth of microorganisms and foodborne diseases result from the ingestion of contaminated foods and food products (Guyader and Atmar, 2008). More than 250 different types of viruses, bacteria, parasites, toxins, metals, and prions are associated with foodborne diseases in humans (Schmidt et al., 2009). Although viruses are more responsible for more than 50% of all foodborne illnesses; generally hospitalizations and deaths associated with foodborne infections are due to bacterial agents. The infections range from mild gastroenteritis to life-threatening neurologic, hepatic, and renal syndromes

caused by either toxin from the “disease-causing” microbe, or by the human body’s reaction to the microbe itself (Teplitzki et al., 2009).

Food poisoning is divided into three types: ‘infection’, ‘intoxication’, and ‘intermediate’ (Schmidt et al., 2009). ‘Infection’ is caused by the oral ingestion of viable microorganisms in adequate amounts to build up infection and the commencement of symptoms is normally delayed, reflecting the time required for an infection to develop. Examples of food-poisoning that cause infection are enteric viruses, *Salmonella*, *Campylobacter* and *Vibrio* species. ‘Intoxication’ on the other hand, is caused by the ingestion of toxins that have been pre-formed in the food. Therefore, there is no necessity for live organisms to be present and the onset of the symptoms is rapid. Examples are *Bacillus cereus* and *Staphylococcus aureus*. The ‘intermediate’ food

poisoning occurs when live bacteria are ingested and subsequently produce a toxin in the host, as in the case of *Clostridium perfringens* food poisoning (Teplitski et al., 2009).

Foodborne diseases remain a major source of morbidity and mortality in the general population, mainly in susceptible groups, such as infants, the elderly and the immunocompromised (WHO, 2011). According to the World Health Organization (WHO), up to 1.5 billion cases of diarrhoea and more than three million deaths that occur in children every year are as a result of food and water contamination (WHO, 2007), and in the United States of America (USA) it is estimated that, foodborne diseases result in 76 million illnesses, 325,000 hospitalizations and 5000 deaths each year (Mead et al., 1999; WHO, 2007). In France, it is estimated that these pathogens cause 10,200 - 17,800 hospitalizations yearly (Vaillant et al., 2005). The developing world are not spared, in South East Asia, approximately one million children below the age of five years die each year from diarrheal diseases due to contaminated food and water (WHO, 2000). Several devastating foodborne outbreaks have been reported on the African continent; in 2004, Kenya experienced an acute aflatoxicosis outbreak which was attributed to maize whereas in 2007, Angola registered 400 cases of bromide poisoning, associated with the use of sodium bromide as cooking salt (WHO, 2005).

The prevalence of antimicrobial resistance among foodborne pathogens is reported to have increased (Yucel et al., 2005; Nyenje et al., 2012a), probably as a result of selection pressure created by the use of antimicrobials in animal health. Our recent study on antimicrobial susceptibility of *Listeria ivanovii* and *Enterobacter cloacae* isolates from food samples signified an alarming multi-drug resistance of at least four or more of the test antibiotics (Nyenje et al., 2012a). In this study we therefore present a comprehensive review on some selected and important foodborne pathogens and associated illnesses, as well as treatment and control measures in an effort to throw more light on the danger they pose to the community.

AETIOLOGY, PATHOGENICITY AND EPIDEMIOLOGY OF FOODBORNE ILLNESSES

Viruses

Viruses are very small microorganisms, ranging in size from 0.02 to 0.4 micrometres in diameter and cause a wide range of diseases in plants, animals and humans (Koopmans and Duizer, 2004). They are recognized as the most common pathogens transmitted via food; for example in the USA, viruses account for 67% of food related illnesses, compared to 9.7% and 14.2% for *Salmonella* and *Campylobacter*, respectively (Vasickova

et al., 2005).

Viruses are intracellular organisms, which only replicate within living cells of the host; therefore the number of viral particles in food does not increase and sensory features of the contaminated and non-contaminated food will be identical (Koopmans and Duizer, 2004). Foodborne viruses include, rotaviruses, noroviruses, enteric adenoviruses, hepatitis A virus (HAV), enteroviruses, human astroviruses, aichiviruses, toroviruses, coronaviruses and picobirnaviruses (Chitimbar et al., 2012). However, overwhelming majority of cases are due to norovirus and HAV (Koopmans and Duizer, 2004).

Noroviruses

Noroviruses (NoVs) are non-enveloped single stranded RNA viruses belonging to the family *Caliciviridae*. Five genogroups exist, GI to GV, of which GI, GII and GIV are recognized to infect humans; genotype GII.4 is widespread in outbreaks (Koopmans, 2008). NoVs are considered the major cause of epidemic gastroenteritis in all age groups, leading to over 267,000,000 annual infections worldwide (Barrabeig et al., 2010). Furthermore, it is estimated that about 900,000 cases of pediatric gastroenteritis in industrialized nations and 1.1 million episodes and 218,000 deaths in developing nations are caused by NoVs (Patel et al., 2008).

Noroviruses are acid resistant; hence they pass through the stomach and replicate in the small intestines where the infected individuals (symptomatic and asymptomatic) shed the virus in both fecal matter and vomitus, and can also be transmitted through contaminated water or food (Lopman et al., 2002). Person-to-person transmission is by far the most common route of infection. For example, a surveillance study in New Zealand conducted between 2001-2007 reported that of the total outbreaks, 19.9% were associated with environmental sources, 17.6% with foodborne infection and 61.0% with person-to-person transmission (Lim et al., 2010). Likewise, norovirus outbreaks resulting from contamination by an infected food-handler, water, both directly (example consumption of tainted water) or indirectly (example via washed fruits, by swimming or canoeing in recreational waters) has been documented (Atmar and Estes, 2006).

Norovirus infections are mostly manifested as gastroenteritis; characterised by acute onset of nausea (81%), vomiting (54%), diarrhoea (85%) and abdominal cramps (72%). Constitutional symptoms such as fever (51%), rigors, muscle and joint pain and headache are also common (Simmons et al., 2001). In healthy individuals, the symptoms are generally mild and self-limiting however, in vulnerable groups, more serious illnesses have been reported (Teunis et al., 2008). Immunity to norovirus infection seems to be short-lived, in the order of several months, after this period, individuals

appear to become susceptible to the same strain of virus (Glass et al., 2009). Although the pathogenesis is not clearly understood, some studies noted lesion on the small intestinal mucosa and, inflammation, blunting of the villi, shortening of the microvilli and dilation of the endoplasmic reticulum; these conditions lead to abnormal gastric motor function, believed to be the cause of associated nausea and vomiting (Lopman et al., 2002; Glass et al., 2009).

Hepatitis A virus (HAV)

HAV is a non-enveloped single-stranded RNA virus. It is a member of the Hepatovirus genus, belonging to the *Picornaviridae* family. There is only one serotype of HAV and six genotypes; genotypes I, II and III cause acute hepatitis in humans and immunity after infection is lifelong (Fiore, 2004). Despite being endemic worldwide, areas with low socio-economic standards have high incidence rates of HAV; exemplified by immunological surveys where almost 90% of children are infected before the age of 10 years, though most infections are asymptomatic (Nainan et al., 2006).

The first largest outbreak occurred in China in 1988 where 3 million people were infected after consumption of clams harvested from a sewage-polluted area (Cuthbert, 2001). Other outbreaks associated with oyster, mussels, green onions, lettuce, strawberries in Australia, Brazil, Italy and Spain have been reported (Coelho et al., 2003; Wheeler et al., 2005). In most of these outbreaks, sewage was the source of pollution.

HAV infections result in a number of symptoms including: Fever, anorexia, nausea and abdominal discomfort, followed within a few days by jaundice. HAV infection may also cause liver damage, usually from the host's immune response to the infection of the hepatocytes. In some cases, the liver damage may lead to death. The virus has a low case fatality rate of 0.3% but increases with age and underlying chronic liver diseases (Nainan et al., 2006). The exact pathogenesis of HAV is unclear; however, it is believed that once the virus has been acquired, it enters and replicates in the small intestines (Kumar et al., 2010). This primary replication is followed by a viremic stage and transportation to the liver where the virus is further replicated in the hepatocytes (Koopmans et al., 2008). The virus is secreted into the bile canaliculi from where it passes back into the intestinal tract, and the infected individuals (asymptomatic and symptomatic) will shed the virus in the faeces in high titers. HAV is not cytolytic and hepatic damage is immune-mediated (Pinto et al., 2010).

Bacterial agents

Foodborne bacterial agents are the leading cause of

severe and fatal foodborne illnesses. Of the many thousands different bacteria species, more than 90% of food-poisoning illnesses are caused by species of *Staphylococcus*, *Salmonella*, *Clostridium*, *Campylobacter*, *Listeria*, *Vibrio*, *Bacillus*, and Enteropathogenic *Escherichia coli* (Nyenje et al., 2012b). For instance in France, in the last decade of the 20th century, *Salmonella* was the most frequent cause of bacterial foodborne illness (5,700 - 10,200 cases), followed by *Campylobacter* (2,600–3,500 cases) and *Listeria* (304 cases) (Vaillant et al., 2005). In South Africa, species of *Listeria*, *Enterobacter* and *Aeromonas* were the most prevalent bacteria in ready-to-eat foods (Nyenje et al., 2012b).

***Salmonella* spp.**

Salmonella enterica is a Gram-negative bacillus (Akoachere et al., 2009a); it is a common cause of human bacterial gastroenteritis worldwide, and food animals are important reservoir for non-typhoidal *Salmonella* spp. (Skov et al., 2007). Human salmonellosis are mostly caused by *Salmonella* serovar Typhimurium and serovar Enteritidis (Galanis et al., 2006), although prevalence of other serovars especially serovar Schwarzengrund in Denmark and USA has also been reported (Oslen et al., 2001; Vugai et al., 2004). Mostly, salmonellosis is asymptomatic but in symptomatic cases fever, diarrhoea, abdominal cramps and nausea may be experienced often controlled within a week. However, the organisms may be excreted in the faeces for many weeks after symptoms subside (FDA/CFSAN, 2003).

Salmonellosis represents an important public health problem globally. For example in Denmark, foodborne cases of salmonellosis cost the country about \$ 10.4 – \$25.5 million in 2001 and in the USA, the organism is responsible for an estimated 1.4 million cases, 16,000 hospitalizations and more than 500 deaths annually at an estimated annual cost of about \$2.3 billion (Wegener et al., 2003). Salmonellosis also affects countries economically, for instance, in 2010; over 550 million eggs were recalled due to possible *Salmonella* contamination, resulting in one of the largest massive recalls. Other products recalled included headcheese, pickles, salami, raw tuna, frozen dinners, alfalfa sprouts, lettuce, tomatoes, and olives (Linscott, 2011).

Salmonella can enter the food supply chain to cause illness in three main ways: Food animals harbour the bacteria in their intestines, making meats, poultry, eggs, and milk important vehicles for salmonellosis (Liu et al., 2011). It can also be introduced into the environment, through manure and litter subsequently, contaminating farm produce in particular fruits and vegetables which are eaten raw or with minimal cooking. Cross-contamination can also occur in food service environments or homes,

often between raw poultry and ready-to-eat products (McEntire et al., 2004). Nevertheless, most studies have documented foods of animal origin as the major vehicles for salmonellosis, which man nurtures through mishandling (Zhao et al., 2001; Akoachere et al., 2009a). The CDC estimates that 75% of *Salmonella enteritidis* cases result from the consumption of raw or undercooked eggs; this is because the microorganism is localized inside eggs, making thorough cooking imperative (FDA/CFSAN, 2003). In another study, it was demonstrated that *Salmonella* can colonise the avian reproductive tract, persist in the ovary and oviduct and survive in hen's eggs (Gantois et al., 2009). Worthy to note is the fact that there is an increasing trend of salmonellosis outbreaks associated with fresh produce and many such crops are produced in the developing countries where manure is frequently used as a natural fertilizer and some studies suggest that some *Salmonella* spp. have now evolved to attach to and colonise vegetables (Klerks et al., 2007; Franz and van Bruggen, 2008).

Staphylococcus aureus

Staphylococci are Gram-negative and catalase positive, cocci that are ubiquitous in the environment being found in the air, dust, sewage, water, environmental surfaces, humans and animals. The species of this organism are classified into two, based on their ability to produce coagulase. Coagulase-positive *Staphylococci* (CPS), in particular *S. aureus* is the pathogenic strain that produces enterotoxin responsible for food poisoning while some strains of Coagulase-negative *Staphylococci* (CNS) are used in the fermentation of meat and milk-based products (Becker et al., 2001). Although some studies have reported the existence of certain CNS enterotoxins producing strains (Zell et al., 2008; Even et al., 2010), the subject has always been controversial because very little information is available about food poisoning caused by CNS (Hennekinne et al., 2010).

Staphylococcal food poisoning (SFP) occurs from the ingestion of foods containing preformed staphylococcal enterotoxins (Loir et al., 2003). In most cases food handlers carrying enterotoxin-producing *S. aureus* in their noses or hands are the main source of food contamination due to improper handling and subsequent storage at temperatures which permit growth of *S. aureus* and production of the enterotoxin (Argudin et al., 2010). Various food types have been implicated in SFP and they differ widely from one country to another, probably due to differing food habits (Loir et al., 2003). For instance, in the UK and the USA, meat or meat-based products are the food vehicles mostly involved (Genigeorgis, 1998), while in France, milk-based products are commonly involved than in other countries (De Buyser et al., 2001). Salted food products, such as ham, have also been

implicated in Japan (Qi and Miller, 2000). Outbreaks of SFP have been reported worldwide; in Brazil, 42 cases of SFP occurred following a meal at a restaurant (Carmo et al., 2003); while in 2009, six food poisoning outbreaks caused by Staphylococcal enterotoxin type E were reported in France and the source of the outbreak was soft cheese made from unpasteurised milk (Ostyn et al., 2010). Two *S. aureus* outbreaks from restaurants in the USA in 2003 and 2005 were traced to carrots, green peppers, and leeks (Loir et al., 2003).

Staphylococcal enterotoxins (SEs), are superantigenic toxins (SAGs) that cause food poisoning and toxic shock syndrome in humans throughout the world (Balaban and Rasooly, 2000). SAGs belong to the broad family of pyrogenic toxin superantigens (SEA), the toxins that induce emesis (Schelin et al., 2011). SEAs are able to evade antigen recognition by interacting with major histocompatibility complex (MHC) class II molecules on the surface of antigen presenting cells (APC), and with T-cell receptors (TCR) on specific T-cell subsets (Thomas et al., 2007). This interaction leads to activation of a large number of T-cells followed by proliferation and massive release of chemokines and pro-inflammatory cytokines that may lead to adverse effects such as lethal toxic shock syndrome (Balaban and Rasooly, 2000). It is suggested that SEA triggers emesis by stimulating the vagus nerve in the abdominal viscera, which transmits the signal to the vomiting centre in the brain (Hu et al., 2007). In addition, SEs are able to penetrate the gut lining and activate the immune system which responds by releasing inflammatory mediators including histamine, leukotrienes, and neuroenteric peptide that causes vomiting (Shupp et al., 2002).

Methicillin resistant *Staphylococcus aureus* (MRSA) strains are now reportedly been isolated in livestock (LA-MRSA) and various foods especially meat and milk, posing a threat over a potential spread of MRSA to consumers via the food chain (Voss et al., 2005; Scott et al., 2010). Contamination of meat with MRSA is mostly as a result of cross contamination from the colonized body sites of the animal to the carcass, through the environment of processing facilities or by people involved in the handling of carcasses or meat (Weese et al., 2010). MRSA bears the *mecA* gene which alters penicillin binding proteins (PBP) having low affinity for all beta-lactam antimicrobials (Scott et al., 2010). Hence, transmission of these MRSA strains through the food will contribute to the growing problem of antimicrobial resistance.

***Campylobacter* species**

Campylobacter species are Gram-negative, non-sporeforming rods. Most species require a microaerobic atmosphere for optimal growth; however, some species grow aerobically or anaerobically. An atmosphere

containing increased hydrogen appears to be a growth requirement for other species such as *C. sputorum*, *C. concisus*, *C. mucosalis*, *C. curvus*, *C. showae*, *C. rectus*, *C. gracilis*, and *C. hominis* (Nachamkin, 2003).

Campylobacters are a leading cause of bacterial enteritis worldwide and can be transmitted directly from animal to person, through ingestion of fecally contaminated water, food, or by direct contact with animal feces or contaminated environmental surfaces. Species of the organism are primarily zoonotic, with a variety of animals implicated as reservoirs of infection, including a diverse range of domestic and wild animals and birds (Butzler, 1984; Altekruise et al., 1999). In addition, water and the environment play a significant but poorly understood role in the epidemiology of campylobacteriosis (Koenraad et al., 1997).

Several epidemiological studies in different countries have identified sources of *Campylobacter* enteritis in man to include animals, food, water, and milk products (Oporto et al., 2007; Esteban et al., 2008). Reports of *Campylobacter* enteritis in developing countries (Padungton and Kaneene, 2005; Uaboi-Egbenni et al., 2008), underscores an urgent need to explore prevalence rates, antibiograms and haemolytic activities in animals because of the zoonotic nature of infections and for proper planning of effective prevention and control measures (Raji et al., 1997; Oporto et al., 2009).

A prevalence study in the Basque County (Northern Spain) identified 28.3% (34/120) of ovine and 18.0% (37/206) of bovine farms positive for *C. jejuni* (Oporto et al., 2007), and even higher rates (38.2%, 13/34) in free-range poultry farms (Esteban et al., 2008). In the United States, requirements for reporting incidence of culture-confirmed infections vary by state. The active food-borne disease surveillance program FoodNet (www.FoodNet.gov) provides uniform reporting from a panel of sentinel sites, giving an accurate incidence of diagnosed infections. The reported incidence of *Campylobacter* infections in the United States has been declining for several years, from 24.7 cases per 100,000 persons in 1997 to 12.7 cases per 100,000 in persons in 2005, a rate lower than reported for salmonellosis (Shepard et al., 2004; Fitzgerald et al., 2009).

Although the majority of *Campylobacter* infections are self-limiting, complicated cases may warrant antimicrobial therapy. Antimicrobial susceptibility data show an increase in the number of fluoroquinolone-resistant and, to a lesser extent, macrolide-resistant *Campylobacter* strains causing human infections (Gibreel and Taylor, 2006; Anderson et al., 2006). Antimicrobial treatment is indicated for systemic *Campylobacter* infections in immune-suppressed patients and for severe or long-lasting infections (Allos, 2001). Erythromycin is considered the drug of choice for treating *Campylobacter* gastroenteritis, and ciprofloxacin and tetracycline are used as alternative drugs (Nachamkin et al., 2000); however resistance of these species has been reported

to these antibiotics (Pezzotti et al., 2003; Oporto et al., 2009).

Isolates of *C. jejuni* and *C. coli* with resistance to various antimicrobial agents have been reported in both developed and developing countries (Hart and Kariuki, 1998; Van Looveren et al., 2001). A significant increase in the prevalence of resistance to macrolides among *Campylobacter* spp. has been reported since the 1990s, and this is recognized as an emerging public health problem (Altekruise et al., 1999). It has been suggested that resistance to macrolides is mainly found in isolates of animal origin; especially *C. coli* from pigs and also *C. jejuni* from chickens (Van Looveren et al., 2001).

Listeria species

Listeria species are Gram-positive, intracellular rods that are ubiquitously found in diverse environments such as soil, water, various food products, animals, and humans (Swaminathan and Smidt, 2007). *L. monocytogenes* is one of the deadly human foodborne pathogens responsible for listeriosis, a rare but fatal disease with a mortality rate of 20-30% in newborns, the elderly and immunocompromised individuals; however, some studies have also implicated *Listeria ivanovii* (*L. ivanovii*), albeit rarely (Guillet et al., 2010; Nyenje et al., 2012b). The organism is persistent in food industries, because it survives food-processing technologies that rely on acidic or salty conditions, and, unlike many pathogens, can continue to multiply slowly at low temperatures, allowing for growth even in properly refrigerated foods (Swaminathan and Smidt, 2007).

Apart from consumption of contaminated food, infection can also be transmitted, directly from infected animals to humans, as well as between humans and from mother to child in the uterus or during passage through the infected birth canal (Jacobson, 2008; Allerberger and Wagner, 2010). Different food sources particularly poultry, red meat and meat products have been associated with outbreaks as well as sporadic cases (Billie et al., 2006; Pichler et al., 2009; El-Malek et al., 2010). Other studies reported vegetable products especially cabbage as a source of contamination and retrospective study, revealed that sheep manure from a flock diagnosed earlier with listeriosis was used to fertilize the crop (Schlech et al., 1983; Rocourt et al., 2001).

Listeriosis can be manifested in two forms, invasive or non-invasive (febrile gastroenteritis); in immunocompetent individuals, non-invasive listeriosis develops as a typical febrile gastroenteritis (watery diarrhea, nausea and headache) while in immunocompromised adults, such as the elderly and patients receiving immunosuppressive agents, listeriosis can manifest as septicaemia or meningoencephalitis (Longhi et al., 2004). On the other hand perinatal listeriosis acquired by the foetus from its infected mother

via the placenta can lead to abortion, birth of a stillborn foetus or a baby with generalized infection, and sepsis or meningitis in the neonate (Allerberger and Wagner, 2010).

Pathogenesis of *Listeria* starts with the entry of the bacterium through the intestines to the liver where it replicates until the infection is contained by the cell-mediated immune response. The mechanism by which *Listeria* causes diarrhoea is not entirely clear. However, it is believed that diarrhoea results from direct invasion of the intestinal mucosal epithelium by the organism (Schuppler and Loessner, 2010).

It is alleged that in normal individuals, the continual exposure to listerial antigens contributes to the maintenance of anti-*Listeria* memory T-cells (Werbrouck et al., 2006). However, in debilitated and immunocompromised patients, there is unrestricted proliferation of the organism resulting in prolonged low-level bacteraemia; subsequently, the bacteria invade target organs (the brain and gravid-uterus). *L. monocytogenes* and *L. ivanovii* are facultative intracellular parasites hence they are able to survive in macrophages and invade a number of non-phagocytic cells such as epithelial, hepatocytes and endothelial cells (Ramaswamy et al., 2007).

Vibrio parahaemolyticus

V. parahaemolyticus is the leading cause of acute gastroenteritis in humans after consumption of contaminated raw or undercooked seafood; it can also cause severe infections in the immune-compromised (Hiyoshi et al., 2010). The organism was identified initially as a cause of foodborne illness in Osaka, Japan in 1951 where it caused 272 illnesses and 20 deaths; semidried juvenile sardines were the source of infection (Nair et al., 2007). Since then, the organism has been reported to account for half of all food poisoning cases in Japan and identified as a common cause of sea-foodborne illness in many Asian countries (Alam et al., 2002; Su and Liu, 2007). Similarly, high incidences of *V. parahaemolyticus* gastroenteritis have been noted in the USA in the summer season (Daniels et al., 2000; Potasman et al., 2002). In contrast to Asian countries, *V. parahaemolyticus* infection is less common in European countries (Nair et al., 2007). However, sporadic outbreaks have been reported in countries such as Spain, Italy and France (Di Pinto et al., 2008; Ottaviani et al., 2010). A small number of studies on the prevalence of *V. parahaemolyticus* in sea foods in particular shrimps have been reported on the African continent mostly from West Africa (Ndip et al., 2002; Eja et al., 2008; Adeleye et al., 2010).

Infection with the bacterium is manifested in three major syndromes: gastroenteritis, wound infections, and septicaemia with gastroenteritis being the commonest.

Although the gastroenteritis is self limited, the organism may cause septicaemia that is life threatening in individuals having underlying medical conditions such as liver, heart and kidney diseases or immune disorders (Yeung and Boor, 2004). The exact virulence mechanisms of *V. parahaemolyticus* are not known; in particular, specific mechanisms that contribute to the ability of strains lacking recognized virulence factors (example, *tdh* and *trh*) to mount an infection are unknown (Han et al., 2007). Studies have linked the virulence of this organism to the presence of a thermostable direct hemolysin (TDH) and TDH -related hemolysin (TRH) (Boyd et al., 2008). It is believed that TDH and TRH act on cellular membranes as a pore-forming toxin that alters ion balance in the intestinal cells thereby leading to secretory response and diarrhoea observed in gastroenteritis (Nair et al., 2007). TDH has also been associated with multiple biological activities including haemolysis, enterotoxicity, cytotoxicity and cardiotoxicity hence it has been considered a major virulence factor of this organism (Raimondi et al., 2000; Park et al., 2004).

Escherichia coli

E. coli is a Gram-negative rod, a member of the family *Enterobacteriaceae*, and a successful gut coloniser in many host species (Tarr et al., 2005). Classification of *E. coli* strains is based on specific virulence factors and phenotypic traits; these include enterohaemorrhagic *E. coli* (EHEC) or Shiga toxin-producing *E. coli* (STEC) strains that produce verocytotoxin or shiga-like toxin, the causative agent of haemorrhagic colitis (HC) and haemolytic-uremic syndrome (HUS); enterotoxigenic *E. coli* (ETEC) strains which produces enterotoxin causing diarrhoea; enteroinvasive *E. coli* (EIEC) strains, the causative agent of dysentery-like illnesses; enteroaggregative *E. coli* (EAEC), which do not secrete heat-labile enterotoxins but adhere to mucosal cells in an aggregative pattern, and diffusely adherent *E. coli* (DAEC) strains that adhere to the surface of epithelial cells (Tarr et al., 2005; Gyles, 2006; Wu et al., 2011). However, foodborne outbreaks have been particularly associated with EHEC and EAEC strains (Tarr et al., 2005; Wu et al., 2011).

Among the EHEC strains, *E. coli* O157:H7 has been widely recognized as the major cause of foodborne illness (Saghaian et al., 2006). The first devastating outbreak of EHEC (*E. coli* O157:H7) occurred in Japan, in which 2764 confirmed cases were reported; the source was radish sprouts (Michino et al., 1999). Ever since, outbreaks and sporadic cases have been reported globally; in most of these outbreaks, contaminated meat, meat products, unpasteurized milk and leafy green vegetables and fruits fertilized with contaminated animal manure was the source of contamination (Sartz et al., 2008; CDC, 2011).

In 2011, an unusual outbreak of enterohemorrhagic gastroenteritis and haemolytic uremic syndrome (HUS) related to infections with shiga toxin-producing *E. coli* O104:H4 (STEC O104:H4) occurred; first reported in Germany followed by France before spreading to other European countries and North America (Wu et al., 2011); raw tomatoes, cucumber and leaf salad was the source of contamination (Frank et al., 2011). This was the first and largest outbreak of infections due to *E. coli* serotype O104:H4 worldwide with 3167 enterohemorrhagic gastroenteritis and 908 HUS cases which claimed 50 lives (WHO, 2011).

Studies on the virulence of *E. coli* O104:H4 strain suggests that they have unique properties of both EHEC and EAEC genes, encoding the production of shiga-toxin (stx) and resistance to multi-antibiotics (Brzuszkiewicz et al., 2011; Ruggenenti and Remuzzi, 2011). The pathogenesis of EAEC strains includes: bacteria adherence to the intestinal mucosa using aggregative adherence fimbriae (AAF); the fimbriae allow bacteria to adhere to each other in a "stacked-brick" pattern and produce mucus, hence forming a biofilm on the surface of enterocytes; followed by the release of toxins and elicitation of inflammatory response, mucosal toxicity, and intestinal secretion (Bielaszewska et al., 2011; Frank et al., 2011). Hence *E. coli* O104:H4 is a typical EAEC strain that forms AAF to enhance bacteria attachment to the intestinal wall and STEC/EHEC that produces shiga-toxin.

Virulence factors of *E. coli* can be encoded by mobile genetic elements such as plasmids and bacteriophages which can be transferred horizontally. This is exemplified by the findings of Brzuszkiewicz et al. (2011), who reported that, the *E. coli* O104:H4 strain acquired the stx-producing gene from the stx-phage which is characteristic for EHEC strains, and speculated that the enhanced adherence factor may have facilitated the absorption of stx-toxin which resulted in the higher percentage of HUS cases (Bielaszewska et al., 2011); the combination of the virulence factors (AAF, stx, extended-beta-lactamases) and formation of the stacked-brick pattern may have led to a stronger gut colonisation and release of toxins (Ruggenenti and Remuzzi, 2011).

Parasites

Parasitic foodborne diseases are generally under-recognised; however they are becoming more common in humans worldwide, with infections in childhood, pregnancy and those related to HIV/AIDS being of major importance (Khan et al., 2007; Dorny et al., 2009). Associated morbidity and mortality are high, with more than 58 million cases of childhood protozoal diarrhoea reported per year, with an estimated financial management of US\$ 150 million (Savioli et al., 2006). Parasites of concern include *Giardia lamblia*, *Entamoeba*

histolytica, *Cryptosporidium parvum*, *Toxoplasma gondii*, *Trichinella spiralis* etc (Doyle, 2003).

Cryptosporidium

Cryptosporidium are protozoan members of the Phylum Apicomplexa, affecting a wide variety of vertebrate hosts. In humans, *C. hominis* (anthroponotic origin) and *C. parvum* (zoonotic origin) are responsible for more than 90% of cryptosporidiosis which accounts for more than 3.1 million deaths each year among children less than 15 years of age (Fayer, 2004). *C. hominis* is more prevalent in North and South America, Australia, and Africa, whereas *C. parvum* causes more human infections in the USA and Europe, especially in the UK (Tumwine et al., 2005; Samie et al., 2006; Morse et al., 2007; Omoruyi et al., 2011).

In developing countries, cryptosporidiosis is most prevalent during early childhood, with as many as 45% of children experiencing the disease before the age of 2 years (Valentiner-Branth et al., 2003); exemplified by most sub-Saharan countries, where cryptosporidiosis prevalence peaks among children aged 6 - 12 months and decreases thereafter. Experimental studies revealed that repeated exposure to *C. parvum* promotes an immunoglobulin G (IgG) response that imparts partial protection against subsequent infection and illness (Chappell et al., 1999). Cryptosporidiosis is chronic and life threatening among immunocompromised individuals but self-limiting in immunocompetent individuals (Paul and Gordon, 2002). It accounts for up to 6 and 24% of all diarrheal diseases in immunocompetent and in persons with AIDS respectively worldwide (Bialek et al., 2002; Omoruyi et al., 2011).

The parasite is transmitted through the faecal-oral route or indirectly via contaminated water supply, food or environment (Miler et al., 2006). The ingested parasite in the form of oocysts, excyst in the gastrointestinal tract and release infective sporozoites, which attach to the apical membrane of the host epithelial cells where they mature into merozoites by asexual reproduction. The merozoites are released into the intestinal lumen where they can either infect other epithelial cells or mature into gametocytes which later releases the oocysts which are excreted in faeces into the environment to start another life cycle (Morgan et al., 2002).

Toxoplasma gondii

Toxoplasmosis is a widely prevalent disease caused by *T. gondii*, an obligate intracellular parasite that forms cysts in mammalian cells. *T. gondii* infects approximately one third of the global population and a wide range of other mammalian and avian species (Marawan et al., 2008). The major sources of human infection are the

ingestion of tissue cysts in raw or undercooked meat, food or water contaminated with sporulated oocysts or by transplacental transmission (Jiménez-Coello et al., 2012). In the USA, toxoplasmosis is the second leading cause of foodborne illness related deaths and fourth leading cause of foodborne illness related hospitalizations (Scallan et al., 2011). It is also noted that South America, Asia and Africa have a higher prevalence of sero-positive individuals; high temperature and humid conditions in these continents may favour the persistence of viable sporulated oocysts in the environment (Ayi et al., 2009; Mercier et al., 2010; Jiménez-Coello et al., 2012).

The life cycle is complex involving two hosts; an intermediate host, usually warm-blooded animals and a definitive host (domestic cats). There are three infectious stages: tachyzoites, bradyzoites contained in tissue cysts, and sporozoites contained in sporulated oocysts (Alayande et al., 2012). The transmission cycle starts, with the shedding of oocysts by the definitive host in the faeces. The oocysts sporulate and become infective within a few days in the environment. Intermediate hosts get infected after ingesting water or food contaminated with the cat faeces. In the gut, oocysts transform into tachyzoites which later migrates to other parts of the body via the bloodstream and further develop into tissue cyst (bradyzoites) in skeletal, ocular muscle and neural tissue where they can persist for many decades (Tenter et al., 2000). The mechanism of this persistence is unknown; however, some investigators believe that tissue cysts break down periodically, with bradyzoites transforming to tachyzoites that reinvade host cells and again transform to bradyzoites within new tissue cysts (Tenter et al., 2000).

Trichinella spiralis

Trichinella spp. are the causative agents of human trichinellosis, a zoonotic disease caused by the ingestion of raw or undercooked meat containing larvae of *Trichinella* nematodes. The most common sources of human infection are pig meat, wild game and horse meat. There are at least eight recognised species of *Trichinella*; the most commonly isolated species is *Trichinella spiralis* (*T. spiralis*) (Dorny et al., 2009). Trichinellosis affects as many as 11 million people worldwide (Hernandez-Bello et al., 2008). Numerous trichinellosis outbreaks have been noted in Asia, particularly in China and Thailand. From 1964 to 2003, China experienced 247 deaths from trichinellosis, and in Thailand 97 people died from 1962 to 2005 (Kaewpitoon et al., 2006). Between 2004 -2005, 5690 cases and 5 deaths were documented from 147 outbreaks, with wild game (bear, cougar or wild boar meat) and commercial pork products identified as the source of infection (Dupouy-Camet, 2009). In 2003, an outbreak in Poland involving 124 people was reported to have been caused by infected wild boar meat (Doyle et

al., 2003).

The ingested *Trichinella* larvae encyst in muscle tissue and rapidly develop into adults in the intestine, where they mate and produce newborn larvae. The new borne larvae then migrate from the intestines through the lymphatic system to the blood stream, before invading striated skeletal muscle cells to complete the cycle (Movsesijan and Milosavijevic, 2010). The clinical signs of acute trichinellosis in humans are characterised by two phases. The first phase, whose symptoms include nausea, diarrhoea, vomiting, fatigue, fever and abdominal discomfort, is often asymptomatic. Headaches, fevers, chills, cough, eye swelling, aching joints and muscle pains, itchy skin, diarrhoea, or constipation follow the first symptoms, and if the infection is heavy, patients may experience difficulty coordinating movements, and have heart and breathing problems (Dupouy-Camet, 2009; Bruschi, 2012).

Giardia intestinalis

Giardia intestinalis (also known as *G. lamblia* or *G. duodenalis*) are ubiquitous enteric protozoan pathogens that infect humans, domestic animals and wildlife worldwide. Giardiasis is linked to the socioeconomic level of a country, with prevalence ranging between 2 - 7% in most industrialized regions and reaching 40% in developing countries; most of these infection occur in children (Júlio et al., 2012). A study in a refugee camp of Guma in Nigeria noted a prevalence of 40% in children (Nyamngee et al., 2009), while in Rwanda, a 60% prevalence was reported among children under the age of 5 years (Ignatius et al., 2012). In the USA *Giardia* is responsible for approximately 2.4 million infections annually, and they primarily occur in children day care centres (Furness et al., 2000). The infection results from the ingestion of the cyst in fecally contaminated food or water or through person-to-person and to a lesser extent, animal-to-person transmission. The parasite has a two-stage life cycle: a reproductive trophozoite and an environmentally resistant cyst stage. An ingested cyst passes into the duodenum, where encystation occurs, releasing four trophozoites which multiply rapidly via asexual reproduction and colonise the small intestine. It is during the trophozoite stage that clinical symptoms occur, as a result of damage to the mucous membrane (Dawson et al., 2005).

Although often asymptomatic, *Giardia* infections may lead to acute or chronic diarrhoea with abdominal cramping, dehydration, nausea and/or vomiting, malabsorption, weight loss, and fatigue. The pathogenesis of *Giardia* is not completely understood due to the extensive variation seen in disease expression. Nonetheless, several pathogenic mechanisms have been implicated in giardial diarrhoea, including reduction in intestinal disaccharides and protease activities, disruption

of microvillous brush border, villus shortening or atrophy, crypt hyperplasia, increased epithelial permeability, mucosal inflammation, bacterial overgrowth and intestinal hyper-motility (Adam, 2001; Mank, 2001; Koot et al., 2009).

Entamoeba histolytica

Entamoeba histolytica (*E. histolytica*) is an important cause of diarrhoea in people in tropical and subtropical countries. Cases in the U.S. generally occur in immigrants, travellers returning from endemic areas, and in persons living in states along the border with Mexico. According to the WHO, *E. histolytica* is the second leading parasitic cause of death (after malaria) and has been estimated to infect 50,000,000 people worldwide of whom 40,000 - 100,000 die yearly (Babiker et al., 2009). Although most cases of *E. histolytica* remain asymptomatic, they excrete a large number of cysts in their faeces hence serving as a source of infection (Doyle, 2003). In Mexico, it was observed that 340 asymptomatic carriers excreted an average of nearly 4,000 *Entamoeba* cysts/g of faeces (Garrido et al., 2002). Man is major reservoir of *E. histolytica*, passing virulent cysts that are transmitted chiefly by ingestion of contaminated food or water or through an infected food handler and also occurs when produce is freshened or crops are irrigated with contaminated water. Other vectors such as flies, cockroaches and other insects may also transfer cysts from faeces to foods (Babiker et al., 2009).

Human infection usually begins with the ingestion of the cyst in food or water contaminated with human faecal material. Cysts survive the acidic pH of the stomach and pass into the intestine where the cysts undergo excystation and mature into trophozoites which are passed to the colon. In the intestine, many of the trophozoites encyst and both trophozoites and cysts are excreted along with the faeces; cysts can survive for prolonged periods outside the host while the trophozoites survive only for a few hours. Infections can range from non-invasive intestinal diseases which are often asymptomatic to invasive where the trophozoites penetrate the intestinal mucosa to other organs and produce an extraintestinal amoebiasis which are usually more serious and life threatening (Acker and Mirelman, 2006).

Fungi

Filamentous fungi and moulds are able to produce an enormous number of secondary metabolites, including antibiotics and mycotoxins. The term mycotoxin refers to those secondary metabolites which, at a low concentration, are toxic to humans and animals (Sánchez-Hervás et al., 2008).

Worthy of note is the fact that the existence of mycotoxin-producing fungi in plants is not always favourable to contamination with mycotoxins. In order for fungi to produce these secondary metabolites, they have to be stressed by some factor, such as nutritional imbalance, drought or water excess (Dutton, 2009). Mycotoxins have been implicated as causative agents of human foodborne intoxication, as well as human hepatic and extra-hepatic carcinogenesis (Wild and Gong, 2010); clinical symptoms include diarrhoea, liver and kidney damage, pulmonary oedema, vomiting, haemorrhaging and tumours (Bryden, 2012).

The most frequent toxigenic fungi are *Aspergillus*, *Penicillium* and *Fusarium* species (Sánchez-Hervás et al., 2008). The foodborne mycotoxins of greatest significance in Africa and other tropical developing countries are the fumonisins (FB), aflatoxins (AFs) and trichothecenes (Wagacha and Muthomi, 2008). These toxins contaminate various food stuffs, including maize, cereals, groundnuts and tree nuts feed during production, harvest, storage or processing (Sánchez-Hervás et al., 2008); mycotoxins can also occur in milk, meat and their products as a result of animals consuming mycotoxin contaminated feeds (Wild and Gong, 2010). The toxins frequently occur in maize, a staple food in most parts of Africa, Asia and Latin America; hence, their contamination translates to high-level chronic exposure in these countries (Wild and Gong, 2010).

TREATMENT

Foodborne illnesses are mostly manifested as acute gastroenteritis and are usually self limiting. However, in others, fluid replacement and supportive care may be essential. Oral rehydration is indicated for patients who are mildly to moderately dehydrate; whilst in more severe dehydration, intravenous therapy may be administered (Mølbak, 2005).

Worthy to note is the fact that many anti-diarrheal agents have potentially serious adverse effects in infants and young children; therefore their routine use in this age group is discouraged (CDC, 2004). For severe cases, extraintestinal disease, or for immunocompromised persons, antimicrobial therapy is prerequisite and should be based on: clinical signs and symptoms; organism detected in clinical specimens; antimicrobial susceptibility tests. Such information can also support public health surveillance of infectious disease and antimicrobial resistance trends in the community (CDC, 2004; Gilbert et al., 2004; Foley and Lynne, 2008).

A number of drugs have been recommended for severe extraintestinal foodborne disease; they include fluoroquinolones or third-generation cephalosporins, ampicillin, gentamicin, sulfamethoxazole/ trimethoprim (Mølbak, 2005; Akoachere et al., 2009b). Since fluoroquinolones are reported to cause damage to the

cartilage in children under the age of 16 years, third-generation cephalosporins including ceftriaxone are an alternative therapy for this age group (Gilbert et al., 2004).

Listeriosis is another deadly disease where intravenous antimicrobial therapy is paramount. Drugs like ampicillin, penicillin, erythromycin, sulfamethoxazole/ trimethoprim are employed for invasive diseases whereas tetracycline, doxycycline and gentamicin are commonly used (Ruiz-Bolivar et al., 2011). We recently reported marked susceptibility of *L. ivanovii* and *E. cloacae* isolates from food to chloramphenicol, ciprofloxacin, streptomycin and trimethoprim/sulfamethoxazole; hence these drugs could be considered in the treatment of infections caused by these organisms in the study area (Nyenje et al., 2012a). On the other hand, viral infections are managed by supportive care whereas parasitic infections can be treated with metronidazole in cases of *E. histolytica* and *G.lambli*a; spiramycin or primethamine plus sulfadiazine in toxoplasmosis while cryptosporidiosis in adult is treated using paromomycin and in children, nitazoxanide is administered (CDC, 2004; Rossignol, 2010).

The treatment of severe cases of food poisoning (SFP and botulism) involves supportive therapy, with mechanical ventilation where pulmonary involvement is expected. Administration of immune globulin to neutralize circulating botulism toxin can also assist (Arnon et al., 2006). Foodborne illnesses have fatal consequences in vulnerable groups, interestingly some recommended prophylactic treatment have also shown positive effects against foodborne pathogens. For example, sulfamethoxazole/trimethoprim (SXT) presently used in many transplant centres to prevent *Pneumocystis pneumonia*, is also effective against *L. monocytogenes* and *T. gondii*. SXT also appears to reduce the incidence of *Salmonella* infections after transplant, although resistance has occurred in some *Salmonella* species (Safdar and Armstrong, 2003; Conter et al., 2009; Morvan et al., 2010).

Antimicrobial resistance is a major challenge in the management of severe foodborne illness; even though much of the resistance experienced in human medicine is accredited to improper use of antibiotics in humans, antimicrobial use in animals selects for resistant foodborne pathogens that may be transmitted to humans as food contaminants (Mesa et al., 2006). Worthy of note is the fact that most of these foodborne pathogens are of food-animal origin notably, *Campylobacter*, *Salmonella*, *Listeria*, *E. coli* O157, *T. gondii*. In the USA, 14% of chicken samples contained ciprofloxacin-resistant *C. jejuni* two years after the introduction of fluoroquinolones use in poultry-farming which also saw an increase of quinolone-resistant *C. jejuni* in human infections during the same period, suggesting that chickens were the possible reservoir (Collignon et al., 2009); therefore it is likely that the proper use of other antimicrobials in food

animals may equally reduce the resistance to these drugs.

Outbreaks of a multidrug resistance *Salmonella* Typhimurium DT104 strain (Chloramphenicol, ampicillin, streptomycin, sulfonamides, chloramphenicol, and tetracycline) have been documented (Davis et al., 2007; Sofos, 2008). *Salmonella* spp resistant to nalidixic acid, gentamicin, kanamycin, sulfamethoxazole/trimethoprim and ciprofloxacin has also been reported (Savadkoobi and Kacho, 2007; De Paula et al., 2010).

A few outbreaks involving community acquired MRSA in SFP have been reported (Jones et al., 2002). In France two MRSA strains were isolated from food incriminated in SFP (Kerouanton et al., 2007). MRSA colonize a number of animals including humans where they may serve as a reservoir for MRSA, leading to its persistence and spread in the community (Lee, 2006). It should also be noted that the resistance of MRSA infections are not only related to beta-lactam antibiotics but also to other antibacterial drugs such as fluoroquinolones, gentamicin, clindamycin, erythromycin and trimethoprim/sulfamethoxazole (Kuint et al., 2007; Huang et al., 2007).

PREVENTION AND CONTROL

Most of the foodborne pathogens are ubiquitous in nature; as such there is a possibility of cross-contamination between one or several products during processing. Preventive measures, such as the pasteurization of milk and dairy products, irradiation, healthier manufacturing processes, and attention to clean water have helped to minimize cases of tuberculosis, typhoid fever, and cholera that were once common causes of foodborne illnesses (Linscott, 2011).

Internationally, countries are now adopting the Hazard Analysis and Critical Control Point (HACCP) system; endorsed by the Codex Alimentarius Commission as a tool that can help prevent known hazards and reduce the risks that may occur at specific points in the food chain. Since the approach enforces procedural governance and rigorous documentation practices, HACCP serves not only as a model to assess risk, but also as an effective means to communicate risk control (WHO, 2007). In addition, quality systems such as good agricultural practices (GAP) are recently recommended for farms, to provide basis for the development of best practices in the production of horticultural products (example fruits, vegetables, potatoes, salads, etc.) (Codex Alimentarius Commission, 1997; Kokkinakis and Fragkiadakis, 2006).

Studies have demonstrated the effectiveness of HACCP and GAP implementation; In Greece, tomatoes produced with GAP exhibited low microbial levels when compared with those grown routinely. The study also found that salads from establishments applying HACCP methodology were safe. Hence it was concluded that the

application of GAP and HACCP are critical for the quality of the products (Kokkinakis and Fragkiadakis, 2006). Similarly, in Jordan, low bacterial plate count was noted from home-made jam after applying HACCP system (Al-Saed et al., 2012).

GAP such as early harvesting; proper drying, sanitation, proper storage and insect management among others are some of the strategies employed to prevent fungal infections. Other possible interventions include biological control, chemical control and breeding for resistance strains (Wagacha and Muthomi, 2008). Dorner and Cole (2002) reported a reduction of 74 - 99.9% in aflatoxin contamination in peanuts in the USA upon introduction of atoxigenic strains of *A. flavus* and *A. parasiticus* into the soil of developing crops. Other biological control addresses the potential use of microorganisms as mycotoxin binders in the gastrointestinal tract of both humans and animals, thereby reducing the potential deleterious effects of exposure to these toxins (Kabak and Dobson, 2009).

At consumer's level, foodborne illnesses can be reduced by proper cooking of meat, poultry and eggs to temperatures that will kill bacteria; although, spores are heat resistant; steaming under pressure, grilling, roasting and frying of foods can destroy the vegetative cells and spores (Linscott, 2011). Additionally, refrigerating leftovers promptly and storing foods at recommended temperatures; avoiding cross-contamination of cooked and raw foods; washing of utensils and surfaces before and after use with hot, soapy water; and frequently washing hands and/or using gloves when preparing food are all recommended (Gashaw et al., 2008).

Since most foodborne viral infections are transmitted faeco-orally through infected persons who handle food that is not heated or ready-to-eat foods, emphasis should be on stringent personal hygiene during preparation. In addition to adequate heating, ultraviolet light or strong oxidizing agents can also inactivate viruses (Doyle, 2003). Water treatment for public consumption is a safe and highly effective preventative measure; additionally, the effective treatment of sewage minimizes the spread of enteric disease-causing organisms. For this reason, use of municipal water supplies is recommended for all food-handling facilities (Linscott, 2011).

CONCLUSION

Globally, foodborne illnesses are accountable for significant morbidity and mortality. It results from consumption of food contaminated with the pathogen, poisonous chemicals or toxins (Teplitski et al., 2009). These illnesses also play a key role in emerging and re-emerging infections as well as escalating antibiotic resistance trends. A range of new pathogens have emerged due to changing dynamics of the food industry. Therefore, it is imperative to monitor and investigate

foodborne illnesses in order to control and prevent further outbreaks; various surveillance systems are established in different parts of the world for the early detection and control of outbreaks. Nevertheless the incidences of established outbreaks of foodborne illness in most developing countries are underreported (Mans et al., 2010), which could be partly due to lack of resources for investigation. A considerable proportion of the population including the immunocompromised, as well as pregnant women, infants, and the elderly are at risk of foodborne disease; hence a concerted action to diagnose, report, treat and control foodborne illnesses is imperative globally.

ACKNOWLEDGEMENT

We are grateful to the Govan Mbeki Research and Development Centre, University of Fort Hare, South Africa for funding.

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