

Critical environmental factors that influence the presence of *vibrio cholera* in the setting of Nairobi

Juliana Wanjiru Maina

PhD. Department, Food Science and Nutrition, Jomo Kenyatta University of Agriculture and Technology, Kenya

Abstract

Cholera happens to be among the illnesses that are considered major public health threats owing to the numerous incidences of deaths that have resulted from it across the globe. This study aimed to investigate the critical environmental factors that influence the presence of *Vibrio cholera* in the setting of Nairobi. This goal was accomplished by focusing on the review of information on the existing literature. The findings of the study revealed that virulence factors of the bacterium, water, temperature, pH, salinity, and conditions of hygiene. Salinity conditions where sodium chloride concentrations ranging from 0.2%-3%, electrical conductivity condition ranging from 119 to 1090 $\mu\text{S cm}^{-1}$, temperatures range of 17.9°C to 40°C, and pH conditions of between pH 5.0 and pH 9.88 were noted to be optimal for the survival of *V. cholerae*. Some of the measures that can be embraced to address cholera outbreak in Nairobi are improving infrastructure associated with water and sanitation, protecting the environment, conducting medical surveys, and adhering to hygiene measures. Embracing programmes aimed at instilling behavioural change and treatment of water prior to discharge into water bodies such as rivers are also encouraged.

Keywords: vibrio chorea, survival, factors, Sub-saharan Africa

Introduction

Cholera happens to be among the illnesses that are considered major public health threats owing to the numerous incidences of deaths that have resulted from it across the globe. According to Jutla *et al.* (2013)^[8], the 2010 Haiti earthquake reaffirmed the fact that cholera is a significant threat to public health. However, News Medical Life Sciences (2017)^[16] asserts that African records the highest cases of cholera deaths in the world. Even though *Vibrio cholerae* is considered autochthonous to marine setting and that its eradication is futile, hydroclimatology prevention and prediction can be employed in addressing it. Gaffga *et al.* (2007)^[7] defines Cholera as an acute, diarrheal illness that results from the infection of the human intestine with the toxic bacterium *Vibrio cholera* sero-group O1 or O139. Cholera infection can be severe, mild, or asymptomatic. On the other hand Bertuzzo *et al.* (2008)^[1] define cholera as an acute diarrheal infection resulting from the *Vibrio cholerae* bacterium, which triggers illness exclusively in humans. According to Gaffga *et al.* (2007)^[7], approximately 1 in every 20 cholera infected individuals possesses severe illness characterized by leg cramps, vomiting, and profuse watery diarrhea. In these individuals, rapid body fluid loss often results into hypovolemic shock, electrolyte disturbances, and dehydration, which can lead to death within hours in the absence of treatment. In relation to this, Moore *et al.* (2014)^[15] argue that inexpensive and simple treatment mechanisms such as intravenous and vigorous oral fluid as well electrolyte replacement can be employed to treat the illness and enhance recovery within a few days. Gaffga *et al.* (2007)^[7] argue that these measures can result into the reduction of the entire mortality rate from 50 percent to less than 1 percent even within the makeshift centres of treatment in the rural areas. On

the other hand, News Medical Life Sciences (2017)^[16] asserts that the availability of cholera treatment centres along with quick and supportive care can result into the reduction of fatality rate from about 30% to almost 0%. According to Bertuzzo *et al.* (2008)^[1], the toxigenic *Vibrio cholera*, which is epidemic cholera's causal agent, can persist in an indefinite manner in aquatic, riverine, and estuarine settings. Since 1817, epidemic cholera has been witnessed in endemic regions in Asia within seven pandemic waves, which have involved much of the globe (Moore *et al.* 2014)^[15]. Nonetheless, incidences of cholera outbreak have been more pronounced in Africa than in other regions of the world. For instance, Democratic Republic of Congo registered as the world's highest incidences of cholera outbreak between 2002 and 2007 (Piarroux *et al.* 2009)^[21]. In El Nino years the cumulative numbers of cholera incidences across Africa in its entirety were noted to be almost the same relative to non-El Nino years (News Medical Life Sciences, 2017)^[16]. However, the geographical distribution of diseases was essentially different. The El Nino conditions within the equatorial Pacific area robustly impact weather conditions across the globe, including decreasing rainfall within the drier regions of southern and northern Africa, and ensuring rainfall increase in East Africa (News Medical Life Sciences, 2017)^[16]. In the period between 2000 and 2014, also known as El Nino years, the incidence of cholera increased threefold in areas such as East Africa, which registered the strongest association between cholera and El Nino (News Medical Life Sciences, 2017)^[16]. In certain regions within the central West Africa, significantly limited cholera cases were recorded, but with insignificant change in patterns of rainfall. During this period, it was noted that approximately 117 million individuals residing in regions that witnessed a rise in cholera incidences

in a period of additional rainfall (Bertuzzo *et al.* 2008) ^[1]. Researchers also note that this period was associated with about 30,000 fewer cholera incidences in southern Africa where limited rainfall relative to normal situations (Moore *et al.* 2014) ^[15]. In the recent years, East Africa has continued to record increased cases of cholera outbreak, especially in nations such as Kenya and Tanzania (Moore *et al.* 2014) ^[15]. For instance, in Tanzania, the period between 2015 and 2016, which was an El Nino year, was noted to record the largest outbreak of cholera since 1997 (News Medical Life Sciences, 2017) ^[16].

The acquisition of *V. cholerae* occurs from environmental sources where its persistence transpires between outbreaks of the illness (Piarroux *et al.* 2009) ^[21]. Thus, it can be noted that the survival or presence and multiplication of *Vibrio cholera* are dependent on the presence of certain environmental survival. In relation to this, it is evident that the prevalence of Cholera outbreak in East Africa can be attributed to such environmental aspects or conditions. However, it is significant to note that *Vibrio cholera* is capable of surviving in different niches of the environment. This capability is majorly attributed to the bacterium's evolution of various adaptive responses that enable it to survive stressors like fluctuations in salinity, deprivation, and temperature, and to repel predation by bacteriophage and heterotrophic protists (Bertuzzo *et al.* 2008) ^[1]. This study aims to investigate the critical environmental factors that influence the presence of *Vibrio cholera* in the Setting of Nairobi. The paper's focus is on the Nairobi setting in Kenya. The primary limitation of this study is that there are limited secondary sources that focus on the environmental aspects influencing the existence of *Vibrio cholerae* in Nairobi, Kenya. As such, the peer reviewed sources employed in this article are those that focus on the Sub-Saharan Africa of which Kenya is part. According to Piarroux *et al.* (2009) ^[21], cholera continues to be a significant public health issue, which calls for adequate consideration and effective interventions. Bertuzzo *et al.* (2008) ^[1] add that is significant that environmental management within the developing nations should focus on the effecting of improvements in access to sanitation and hygiene, and water supply. Therefore, identifying the environmental aspects that can influence the survival of *Vibrio cholera* in Nairobi will contribute significantly to shaping the direction for policy implementation and interventions in public health care to address the issue of Cholera in Nairobi, Kenya in the future.

Overview of Cholera

Cholera is an acute intestinal infection causing profuse watery diarrhea or voluminous watery stools, often accompanied by vomiting, circulatory collapse and shock. Piarroux *et al.* (2009) ^[21] assert that many infections are associated with milder diarrhea or have no symptoms at all. If left untreated, 25-50% of severe cholera cases can be fatal. According to Bertuzzo *et al.* (2008) ^[1], people who are more likely to be exposed to cholera include healthcare personnel treating cholera patients, cholera response workers, and travelers in an area of active cholera transmission who cannot or do not always follow safe food and water precautions and personal hygiene.

Structure and Classification

Vibrios are highly motile Gram-negative, highly motile curved rods or comma-shaped with a single polar flagellum. They tolerate alkaline media that kill most intestinal commensals, but they are sensitive to acid. Of the vibrios that are clinically significant to humans, *Vibrio cholerae* O group 1, the agent of cholera, is the most important (Bertuzzo *et al.* 2008) ^[1]. *Vibrio cholerae* was first isolated in pure culture by Robert Koch in 1883, although it had been seen by other investigators, including Pacini, who is credited with describing it first in Florence, Italy, in 1854 (Piarroux *et al.* 2009) ^[21]. Numerous free-living vibrios are known, some potentially pathogenic. Until 1992, cholera was caused by only two serotypes, Inaba (AC) and Ogawa (AB), and two biotypes, classical and El Tor, of toxigenic O group 1 *V. cholerae* (Bertuzzo *et al.* 2008) ^[1]. These organisms may be identified by agglutination in O group 1-specific antiserum directed against the lipopolysaccharide component of the cell wall and by demonstration of their enterotoxigenicity (Piarroux *et al.* 2009) ^[21]. In 1992, cholera caused by serogroup O139 (synonym "Bengal" the 139th and latest serogroup of *V. cholerae* to be identified) emerged in epidemic proportions in India and Bangladesh. This serovar is identified by 1) absence of agglutination in O group 1 specific antiserum; 2) by agglutination in O group 139 specific antiserum; and 3) by the presence of a capsule.

Pathogenesis

Cholera is transmitted by the fecal-oral route. Vibrios are sensitive to acid, and most die in the stomach. Surviving virulent organisms may adhere to and colonize the small bowel, where they secrete the potent cholera enterotoxin (CT, also called "cholera toxin") (Bertuzzo *et al.* 2008) ^[1]. This toxin binds to the plasma membrane of intestinal epithelial cells and releases an enzymatically active subunit that causes a rise in cyclic adenosine 5'-monophosphate (cAMP) production. The resulting high intracellular cAMP level causes massive secretion of electrolytes and water into the intestinal lumen

Epidemiology

Cholera is endemic or epidemic in areas with poor sanitation; it occurs sporadically or as limited outbreaks in developed countries. In coastal regions it may persist in shellfish and plankton. Long-term convalescent carriers are rare (Bertuzzo *et al.* 2008) ^[1]. Enteritis caused by the halophile *V. parahaemolyticus* is associated with raw or improperly cooked seafood.

Literature Review

Nkoko *et al.* (2011) ^[18] consider *Vibrio cholerae* a chemoheterotrophic bacterium with the capability of executing both fermentative and respiratory metabolism, beginning from a range of organic substrates. Nkoko *et al.* (2011) ^[18] add that *V. cholerae* is a halo-tolerant microorganism, which is majorly isolated from sites of the environment, where sodium chloride (NaCl) concentrations are estimated to fall between 0.2% and 3%. According to Bhattacharya *et al.* (2009) ^[2], the optimal growth temperature for *V. cholerae* varies from 30°C to 40°C, while its optimal pH is 8. It is vital to note that the environmental resources and

conditions exploited by *Vibrio cholerae* in nature along with its interactions with other abiotic substrates and living organisms influence the ecological niche of this bacterium. In relation to this, *V. cholerae* persists in the environment due its ability to assume various forms of survival. This bacterium releases the chitinase enzyme and can bind to chitin that forms the primary component of crustacean shells. According to Olago *et al.* (2007)^[20], *V. cholerae* is always found in marine setting attached to the zooplankton's chitinous exoskeleton. Olago *et al.* (2007)^[20] assert that this attachment has the capability of enhancing the existence of the bacterium by offering a source of nitrogen and carbon along with the surface for biofilms' formation. In addition, the production of the toxin-coregulated pili (TCP) contributes significantly to the chitinous surfaces' colonization due to the fact the bacterium's TCP mutants cannot establish biofilms on chitins or micro-colonies. As such, it can be argued that the *V. cholerae* that produce TCP possess a fitness advantage within the environment relative to those unable to produce it (Piarroux, 2011)^[18].

The association of chitin with *V. cholerae* offers micro-organism adaptation to stresses of the environment, food availability, and protection from predators. Piarroux (2011)^[18] argues that *Vibrio cholerae* possess the selective benefit in their potential to enter a latent stage, which is termed considered viable, but non-culturable (VBNC). According to Piarroux (2011)^[18], the detection of non-culturable cholera in water during the inter-epidemic stages can only be accomplished when immunochemical and molecular techniques are embraced. In relation to this, it can be noted that the isolation of the culturable *Vibrio cholera* O1 is rare (Piarroux, 2011)^[18]. Molecular techniques have clearly shown the existence of toxigenic *Vibrio cholerae* O1 within the marine setting during inter-epidemic stages a viable state. However, the bacterium has been shown to be in a non-cultural state and as masses of structured biofilms (Olago *et al.* 2007)^[20]. Carbon, which happens to be abundant in brackish and marine water systems, plays a significant role in the establishment of biofilms by engaging in the direct stabilization intercellular interactions.

Cholera has been linked to bathing in river water contaminated by *V. cholerae*. As such, rivers contaminated by the bacterium often transmit the illness from one social group or community to the other, which in turn contributes to the spread of cholera (Piarroux, 2011)^[18]. Nonetheless, sudden, widespread cholera outbreaks always arise from supplies of contaminated public water. Therefore, water plays an essential role in the epidemiology and transmission of cholera be it sea water, waste water, or surface water (Olago *et al.* 2007)^[20]. Therefore, the practice of directly discharging effluent into water bodies continues to pose a significant threat, as it increases the organic load, which in turn leads to the depletion of the dissolved content of oxygen in the receiving water mass. Piarroux (2011)^[18] asserts that the employment of waste and sewage water in many urban settings in Africa is always considered the primary source of irrigation in such regions. Thus, consuming vegetables and fruits, which have been irrigated with polluted water, serves as the probable mechanism of ingesting the toxigenic *Vibrio cholerae*.

According to Okoh & Igbinosa (2010)^[19], *V. cholerae* is an

autochthonous microbial organism that inhabits brackish water, coastal areas, and estuarine ecosystems, and can stay in the non-culturable form within the aquatic setting for years. Okoh & Igbinosa (2010)^[19] argue the bacterium's long lasting persistence results into the reduction of pathogen reliance on host-to-host interaction for the processes of transmission and propagation, which enables the bacterium to attain higher levels of virulence (i.e. forms having the ability to produce enormous number of infectious particles for the infected host within a short time (Okoh & Igbinosa, 2010)^[19]. This situation may account for cases of violent epidemics coupled by long non-host stages. *V. cholera* micro-organisms have the ability to build up enormous multicellular structures upon solid surfaces. According to Okoh & Igbinosa (2010)^[19], the amount of cells within a biofilm may attain as high as 1.0×10^9 cells for every clump, which in many instances, can constitute an infectious pathogen dose. In study conducted by Mendelsohn & Dawson (2008)^[13], researchers demonstrated that environmental water, which tests apparently negative for virulent *V. cholerae* by traditional enrichment techniques, but possess conditionally viable environmental cells (CVEC), is introducible into rabbit ileal loops. This water can yield strains of *V. cholerae* after in-vivo incubation for 18 hours. In the same manner, humans who consumer clumps of temporarily viable environmental cells may experience the resuscitation of *Vibrio cholerae* and amplification of this strain (Okoh & Igbinosa, 2010)^[19]. Such persons can become index cholera incidences that could commence cholera epidemic.

Climate variability and change have resulted into growing concerns about the impacts of climate on health. Fleming *et al.* (2007)^[6] assert that the combination of environmental degradation and climate change has established ideal or suitable conditions for the resurgence, emergence, and spread of infectious illnesses, thereby leading to the deaths of millions of individuals annually. In relation to this, researchers have made significant steps in investigating the environmental aspects that influence the presence of *Vibrio cholerae* in Sub-Saharan Africa. In a study executed by Rebaudet *et al.* (2013)^[22], the researchers argued that cholera is driven directly by variations induced by the climate within the coast marine reservoirs of *Vibrio cholerae*. In this study, the findings revealed that cholera outbreaks often intensify within densely inhabited slum quarters prior to spreading to adjacent areas or regions. Moreover, frequent seasonality associated with the incidence of cholera was noted to be driven by rainfall-induced infection of unprotected sources of water via sewage and latrine overflow, as well as human operations such as travelling (Rebaudet *et al.* 2013)^[22]. The researchers concluded that the application of the cholera paradigm in Africa continues to be questionable. Rebaudet *et al.* (2013)^[22] add that surveys with microbiological assessments of prospective genotyping and water samples of clinical and environmental strains of *Vibrio cholerae* are required to comprehend cholera determinants in Africa, and better target the measures for prevention and control. Luquero *et al.* (2011)^[12] conducted a study on cholera epidemic in Guinea-Bissau where they established that high incidences of cholera outbreak were evident in market places and intersections where runoff amasses waste. Such findings were essential in reinforcing and guiding the control and prevention measures

for cholera (Luquero *et al.* 2011) ^[12].

In study conducted on the presence of *Vibrio cholerae* in Rift Valley Lakes (George and Albert) and Lake Victoria, Kaddumukasa *et al.* (2012) ^[9] focused on the measurement of environmental aspects such as electrical conductivity, temperature, and pH. The findings of this study revealed that the pH ranged from 7.76 to 9.36, 8.68 to 9.85, and 6.6 to 9.88 at Butiaba, Kayanza, and Ggaba respectively. On the other hand, the temperatures were noted to range from 17.9 to 32.3 °C, 22.5 to 29 °C, and 18.2 to 30.5 °C at Butiaba, Kayanzi, and Ggaba respectively. The findings on electrical conductivity varied from 129.2 to 984 $\mu\text{S cm}^{-1}$, 658 to 1090 $\mu\text{S cm}^{-1}$, and 119 to 218 $\mu\text{S cm}^{-1}$ at Butiaba, Kayanzi, and Ggaba respectively (Kaddumukasa *et al.* 2012) ^[9]. It is significant to note that Ggaba, Butiaba, and Kayanzi represented lakes Victoria Albert, and George. In addition, Kaddumukasa *et al.* (2012) ^[9] enrichment techniques were employed to identify culture *Vibrio cholerae* on TCBS (Thiosulfate-citrate-bile salts-sucrose) culture media. The findings revealed that 75% of the samples ($n = 90$) tested positive for *Vibrio cholerae*. The presence of *V. cholerae* was positively related with the quality parameters of water over the 10-month duration of study (Kaddumukasa *et al.* (2012) ^[9]. The detection of *V. cholerae* was also noted to be more frequent during the warmer or dry season relative to the wet season. According to Kaddumukasa *et al.* (2012) ^[9], the findings of this study revealed that water bodies are natural harbours for *V. Cholerae*.

Jutla *et al.* (2015) ^[10] focused on the investigation of hydroclimatic conditions associated with cholera in Zimbabwe. In this study, Jutla *et al.* (2015) ^[10] argued that improved comprehension of the association between climatic processes and pathogenic abundance enables the prediction of cholera outbreak to be an accomplishable goal. This study focused on the city of Chitungwiza, which is located with the Mashinaland East province. The findings of this study revealed that climatic conditions played a significant role in triggering the outbreak of cholera and that these conditions related to anomalies of precipitation and temperature. As such, the researchers concluded that poor sanitation conditions along with elevated temperatures and heavy rainfall initiate cholera outbreak. Jutla *et al.* (2015) ^[10] also asserted that hydroclimatic processes derived from the satellite can be employed to capture conditions of the environment associated with the cholera epidemic.

Kazaji (2015) ^[11] focused on the investigation of the environmental factors responsible for the prevalence of cholera between 2008 and 2009 in Vhembe District of Limpopo, in South Africa. The findings of this study revealed that the absence of hygienic practices, absence of safe food handling and preparation, inadequate sanitation system, and lack of access to safe or clean drinking water. Kazaji (2015) ^[11] recommended prevention and control of outbreak of cholera, as well as case management.

In a study conducted by Ngwa *et al.* (2016) ^[9], the researchers identified that the cholera epidemiology differs significantly between climatic subzones such as tropical humid, Sudano-Sahelian, Equatorial Monsoon, and Guinea Equatorial. For instance, the northern Sudano-Sahelian was noted to register the highest number of cholera incidences in the rainy season,

particularly between July and September. On the other hand, the Equatorial Monsoon subzone registered incidences of cholera outbreak throughout the year, with the least cases recorded in the peak rainfall, particularly between July and September (Ngwa *et al.* 2016) ^[9].

Piarroux *et al.* (2009) ^[21] executed a study that was aimed to comprehend the cholera epidemiology in the Democratic Republic of Congo (DRC) and ensure advancements in the strategy for fighting cholera. This research enabled the researchers to precisely determine the mechanisms of cholera epidemic on various scales by identifying the source zones of the illnesses and population groups serving as agents of the spread. The findings of this study revealed that measures such as improvement of sanitation and water infrastructure, protection of the environment, medical surveys, and hygiene awareness can be employed to address the cholera epidemic in an effective manner. Cummings *et al.* (2010) ^[3] conducted a case-control research during the outbreak of cholera among semi-nomadic pastoralists within the Karamoja sub-region located in northeast Uganda. Some of the interventions identified in this study were programmes associated with intensive health education for ensuring behavioural change. Other interventions were advancements in sanitation and water infrastructure. In epidemic cholera areas, elevated air temperatures often create environmental conditions, which are favorable for the growth of bacteria (Mintz & Guerrant, 2009) ^[14]. When such conditions are accompanied by above normal rainfall and appropriate mechanisms of transmission such as inaccessibility of safe water and damaging of sanitation infrastructure, which contribute to the mixing of flood waters with sewers, cholera epidemic often results.

De Magny *et al.* (2007) ^[5] conducted a study on *Vibrio cholerae* with a focus on the oceanographic and environmental variables. In this study, the researchers focused on the investigation of the combined effect of iron and PH on the existence of *Vibrio cholerae* in water within a laboratory setting. The findings of the study also revealed that none of the six strains of *Vibrio cholerae* employed in the experiment did not live at pH 5.0, and that the existence of strains was enhanced with increasing the pH.

Reimer *et al.* (2011) ^[23] conducted a study that focused on the investigation of the role of environmental or ecological factors in the presence and transmission of *Vibrio cholerae*. The findings of this study revealed that temperature and pH plays a significant role in the existence of the bacterium. For instance, the findings showed that temperature about 25°C and a pH 7.0 were essential for the survival of *Vibrio cholerae*. In relation these findings, Reimer *et al.* (2011) ^[23] argued that elevated air temperatures establish favourable ecological conditions for the growth of *Vibrio cholerae*. Reimer *et al.* (2011) ^[23] also noted that when such temperatures are followed by above normal rainfall in conjunction with appropriate mechanisms of transmission such as damage of sanitation infrastructures and inadequate safe water, outbreak of cholera are often evident. Reimer *et al.* (2011) ^[23] proceeded to calculate the odds associated with cholera occurrence during below and above average air temperatures along with the corresponding odds ratios (ORs) and appropriate confidence intervals (CIs). These findings revealed that the odds associated with the occurrence of

cholera were substantially higher when air temperature existed above the climatological average. Moreover, the outcomes of the OR calculations indicated that the cholera epidemic rose 6-fold in the event that the temperatures of air existed above the climatological average.

According to Deen *et al.* (2008)^[4] the population dynamics of *Vibrio cholera* within the environment are robustly regulated by environmental aspects such as salinity, temperature, and water. In Sub-Saharan Africa the association of climate, human health, and cholera has been established in greater detail by employing satellite remote sensing, which has offers a robust proof that cholera epidemics are linked to climate (Deen *et al.* 2008)^[4]. The role played by the environment in spread and transmission of the cholera illness and of the link to chitin is supported by the finding that certain virulence factors employed by pathogens during the process of transmission or infection may derive from the role they play within their natural habitat (Safa *et al.* 2008)^[24]. In relation to this, Safa *et al.* (2008)^[24] argue that the ligands (i.e. MSHA and TCP) generated by *Vibrio cholerae* that partake in the process of intestinal colonization are involved in formation of a biofilm and chitin binding on surfaces containing chitin. Therefore, these findings indicate that *Vibrio cholera* clones, which have the ability to colonize the intestinal tract, have the possibility of persisting in biofilms within the environment. As such, the evolutionary process of virulence within a bacterium like *Vibrio cholera* may be influenced by factors, which are external to the host along with the mechanism associated with virulence likely mirror adaptive mechanisms functioning within the environment.

Diagnosis

The diagnosis is suggested by strikingly severe, watery diarrhea. For rapid diagnosis, a wet mount of liquid stool is examined microscopically. The characteristic motility of vibrios is stopped by specific antisomatic antibody (Safa *et al.* 2008)^[24]. Other methods are culture of stool or rectal swab samples on TCBS agar and other selective and nonselective media; the slide agglutination test of colonies with specific antiserum; fermentation tests (oxidase positive); and enrichment in peptone broth followed by fluorescent antibody tests, culture, or retrospective serologic diagnosis (Deen *et al.* 2008)^[4]. More recently the polymerase chain reaction (PCR) and additional genetically-based rapid techniques have been recommended for use in specialized laboratories (Ngwa *et al.* 2016)^[17].

Control

Cholera control in Africa continues to face certain challenges. According to Safa *et al.* (2008)^[24], some of the challenges faced in cholera control are long lasting endemicity, lack of preparedness, and complexity in cholera prevention. Reimer *et al.* (2011)^[23] argue that three critical elements in cholera surveillance and control are preparedness, response, and prevention. The first step of preparedness involves the strengthening of the available surveillance systems by assessing the true burden, identifying trends over time, identifying vulnerable populations and high risk areas, strain tracking, and identifying early warning associated with cholera outbreak. The next step involves extended

surveillance, which transcends counting deaths and cases. This process encompasses the collection of economic and demographic data, description of socio-behavioral patterns, collection of data on water access, quality, and sources, collection of data on seasonal and rainfall patterns, and description of waste management practices and sanitation conditions (Mintz & Guerrant, 2009)^[14]. The next step involves a multisectoral coordinated approach. This strategy encompasses environmental management to ensure the prevention of cholera occurrence. Environmental management focuses on health education, food safety, proper hygiene, adequate sanitation, and safe water (Mintz & Guerrant, 2009)^[14]. Preparedness and response are also essential components of the multisectoral approach to prevention of cholera. Measures embraced in this step are development of action plan, provision of health education, emergency stock, training, standard guidelines, and ensuring access to good health care.

Conclusion and Recommendations

The findings of the existing literature present several factors influencing the survival of *Vibrio cholerae* in Nairobi, which in this case is taken to be Sub-Saharan Africa. These factors are virulence factors of the bacterium, temperature, pH, salinity, and conditions of hygiene. Whereas certain critical environmental factors influence the existence of *V. cholerae* in a setting such as Nairobi, the persistence of this bacterium in such an environment can also be attributed to its ability to assume different survival forms. For instance, the findings of the existing literature reveal that this bacterium produces the chitinase enzyme and has the ability to bind to chitin that serves as a primary component of crustacean shells. The attachment enhances the bacterium's capability to exist by providing carbon and nitrogen sources, as well as a surface for the establishment of a biofilm. The findings of the literature also reveal that the survival of *V. cholerae* is enhanced by conditions of salinity where sodium chloride concentrations ranging from 0.2%-3% are considered ideal. In relation to this, the bacterium's optimal survival electrical conductivity condition was noted to range from 119 to 1090 $\mu\text{S cm}^{-1}$. Thus, the presence of saline conditions in the city of Nairobi provides a good survival ground for *V. cholerae*. Water has also been noted to be a key contributor to the existence of *V. cholerae* in any setting including Nairobi. Since the bacterium is autochthonous to aquatic environment, the presence of water contributes significantly to its optimal survival. As such, the consumption of contaminated water as well as vegetables and fruits that have been irrigated by water contaminated by the bacterium results into cholera illness. In relation to this, it can be argued that the spread of cholera illness in Nairobi is more pronounced during the rainy seasons. Apart from depending on the presence of water for survival, the findings from the literature above also indicate that *V. cholerae* survives under a range of temperature and pH conditions. The general range of temperatures ideal for the survival of the bacterium is 17.9°C to 40°C. Thus, the prevalence of cholera disease in Nairobi can be higher in warmer seasons than in can be in seasons characterized by lower temperatures (i.e. temperatures below 17.9°C). *Vibrio cholerae* is also noted to have the capability of surviving under a range of pH conditions. From the findings of the existing literature, the

optimal pH range for the existence of this bacterium is between pH 5.0 and pH 9.88. As such, these findings reveal that the presence of cholera illness in Nairobi is attributed to presence of water, saline conditions, optimal temperatures, and pH. It is also significant to note that there are different strains of *V. cholerae*, which are suited to different temperature and pH conditions. The existing literature reveals certain measures that can be adopted to address the issue of cholera within the setting of Nairobi. Some of the measures that can be embraced to address cholera outbreak in Nairobi are improving infrastructure associated with water and sanitation, protecting the environment, conducting medical surveys, and adhering to hygiene measures. Programmes aimed at instilling behavioural change should also be encouraged as they contribute largely to addressing issue associated with poor hygiene, which enhance the survival and transmission of *V. cholerae*. Moreover, treatment of water prior to discharge in water bodies such as rivers is also encouraged. Such an undertaking ensures that the presence of the bacterium in the water is eliminated, which in turn makes the consumption of water or even vegetables and fruits irrigated by river water safe.

References

- Bertuzzo E, Azaele S, Maritan A, Gatto M, Rodriguez-Iturbe I, Rinaldo A. On the space-time evolution of a cholera epidemic. *Water Resources Research*, 2008, 44(1).
- Bhattacharya S, Black R, Bourgeois L, Clemens J, Cravioto A, Deen JL, *et al.* The cholera crisis in Africa. *Science*, 2009; 324(5929):885-885.
- Cummings MJ, Wamala JF, Eyura M, Malimbo M, Omeke ME, Mayer D, *et al.* A cholera outbreak among semi-nomadic pastoralists in northeastern Uganda: epidemiology and interventions. *Epidemiology & Infection*. 2012; 140(8):1376-1385.
- Deen JL, Von Seidlein L, Sur D, Agtini M, Lucas ME, Lopez AL, *et al.* The high burden of cholera in children: comparison of incidence from endemic areas in Asia and Africa. *PLoS neglected tropical diseases*. 2008; 2(2):173.
- De Magny GC, Guégan JF, Petit M, Cazelles B. Regional-scale climate-variability synchrony of cholera epidemics in West Africa. *BMC infectious diseases*, 2007; 7(1):20.
- Fleming G, Van der Merwe M, McFerren G. Fuzzy expert systems and GIS for cholera health risk prediction in southern Africa. *Environmental Modelling & Software*. 2007; 22(4):442-448.
- Gaffga HN, Tauxe VR, Intz DE. Cholera: A New Homeland in Africa? *American Journal of Tropical Medicine and Hygiene*. 2007; 77(4):705-713.
- Jutla A, Whitcombe E, Hasan N, Haley B, Akanda A, Huq A, *et al.* Environmental factors influencing epidemic cholera. *The American journal of tropical medicine and hygiene*. 2013; 89(3):597-607.
- Kaddumukasa M, Nsubuga D, Muyodi FJ. Occurrence of Culturable *Vibrio cholerae* from Lake Victoria, and Rift Valley Lakes Albert and George, Uganda. *Lakes & Reservoirs: Research & Management*. 2012; 17(4):291-299.
- Jutla A, Aldaach H, Billian H, Akanda A, Huq A, Colwell R. Satellite based assessment of hydroclimatic conditions related to cholera in Zimbabwe. *PLoS one*. <http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0137828>. 2015; 10(9):0137828.
- Kazaji D. *Factors contributing to the prevalence of cholera during 2008 to 2009 in Vhembe District of Limpopo Province, South Africa* (Doctoral dissertation, University of Limpopo), 2015.
- Luquero FJ, Banga CN, Remartínez D, Palma PP, Baron E, Grais RF. Cholera epidemic in Guinea-Bissau (2008): the importance of place. *PLoS one*. 2011; 6(5):19005.
- Mendelsohn J, Dawson T. Climate and cholera in KwaZulu-Natal, South Africa: The role of environmental factors and implications for epidemic preparedness. *International journal of hygiene and environmental health*. 2008; 211(1):156-162.
- Mintz ED, Guerrant RL. A lion in our village—the unconscionable tragedy of cholera in Africa. *New England Journal of Medicine*. 2009; 360(11):1060-1063.
- Moore S, Thomson N, Mutreja A, Piarroux R. Widespread epidemic cholera caused by a restricted subset of *Vibrio cholerae* clones. *Clinical Microbiology and Infection*. 2014; 20(5):373-379.
- News Medical Life Sciences Cholera incidence in Africa increases during El Niño, Study Reveals. Retrieved From <https://www.newsmedical.net/news/20170411/Cholera-incidence-in-Africa-increases-during-El-Nino-study-reveals.aspx>, 2017.
- Ngwa MC, Liang S, Kracalik IT, Morris L, Blackburn JK, Mbam LM, *et al.* Cholera in Cameroon, 2000-2012: Spatial and Temporal Analysis at the Operational (Health District) and Sub Climate Levels. *PLoS neglected tropical diseases*. 2016; 10(11):0005105.
- Nkoko DB, Giraudoux P, Plisnier PD, Tinda AM, Piarroux M, Sudre B, *et al.* Dynamics of cholera outbreaks in Great Lakes region of Africa, 1978–2008. *Emerging infectious diseases*. 2011; 17(11):2026.
- Okoh AI, Igbino EA. Antibiotic susceptibility profiles of some *Vibrio* strains isolated from wastewater final effluents in a rural community of the Eastern Cape Province of South Africa. *BMC microbiology*. 2010; 10(1):143.
- Olago D, Marshall M, Wandiga SO, Opondo M, Yanda PZ, Kangalawe R, *et al.* Climatic, socio-economic, and health factors affecting human vulnerability to cholera in the Lake Victoria basin, East Africa. *AMBIO: A Journal of the Human Environment*. 2007; 36(4):350-358.
- Piarroux R, Bompangue D, Oger PY, Haaser F, Boinet A, Vandeveld T. From research to field action: example of the fight against cholera in the Democratic Republic of Congo. *Field Actions Science Reports*. 2009; 2(1):69-77.
- Rebaudet S, Sudre B, Faucher B, Piarroux R. Cholera in coastal Africa: a systematic review of its heterogeneous environmental determinants. *The Journal of infectious diseases*. 2013; 208(1):98-106.
- Reimer AR, Van Domselaar G, Stroika S, Walker M, Kent H, Tarr C, *et al.* Comparative genomics of *Vibrio cholerae* from Haiti, Asia, and Africa. *Emerging infectious diseases*. 2011; 17(11):2113.

24. Safa A, Sultana J, Cam PD, Mwansa JC, Kong RY. *Vibrio cholerae* O1 hybrid El Tor strains, Asia and Africa. *Emerg Infect Dis.* 2008; 14(6), 987-8.