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THE EFFECT OF EBOLA OUTBREAK ON THE DEMOGRAPHY OF AFRICA

AN EMPIRICAL ANALYSIS OF MALAWI AND OTHER AFFECTED AFRICAN COUNTRIES

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BEING PARTIAL FULFILMENT OF MALAWI HOSPITALS HEALTH CAMPAIGN CONCEPT (MHCC) TOWARDS THE VISION 2020

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ABSTRACT

The research paper provides a summary of infection prevention and control (IPC) measures for those providing direct and non-direct care to patients with suspected or confirmed cases of Filovirus haemorrhagic fever (HF), including Ebola or Marburg haemorrhagic fevers, in health-care facilities (HCFs). It also includes some instructions and directions for those managing the implementation of IPC activities. These IPC measures should be applied not only by health-care professionals but by anyone in direct contact with patients (e.g., visitors, family members, volunteers), as well as by those not in contact with patients but potentially exposed the virus through contact with the environment (e.g., c leaners, laundry, house-keepers, security).

This document represents a rapid update of the WHO 2008 *"Interim Infection Control Recommendations for Care of Patients with Suspected or Confirmed Filovirus (Ebola, Marburg) Hemorrhagic Fever"*. This update is based upon review of WHO and other international reference documents being used in the current Ebola outbreak (see references) and international experts' consensus.

Therefore the research reveals that there is positive relationship between fatality of reported human virulence and death of reported human virulence but there exist a negative relationship between cases of reported human virulence and death of reported human virulence. It is pertinent that cases of reported human virulence are positively related to death of reported human virulence. This shows that cases show be reported earlier not to lead to death as Africa government should look critically to the trend and structure of this health hazard caused by the Ebola virus outbreak.

1.0 INTRODUCTION

Ebola virus[1] disease brings the most frightening of infectious disease syndromes to mind. Ebola virus disease is the kind of thing that horror writers dream about. The nonfiction book The Hot Zone by Richard Preston and the 1995 movie Outbreak, patterned after Ebola virus disease, are equally terrifying. Just imagine victims bleeding from their eyes, ears and nose and, at autopsy, the pathologist finding necrotic organs (Kaye, 2014).

Invariably, Ebola is a severe illness caused by Ebola virus. It is highly infectious, rapidly fatal, with a death rate of up to 90%, but can be prevented. It is spread through direct contact with body fluids like blood, saliva, urine, sperm, etc. of an infected person and by contact with contaminated surfaces or equipment, including linen soiled by body fluids from an infected person. The Ebola virus can be relatively easily eliminated with heat, alcohol-based products, and sodium hypochlorite (bleach) or calcium hypochlorite (bleaching powder) at appropriate concentrations.

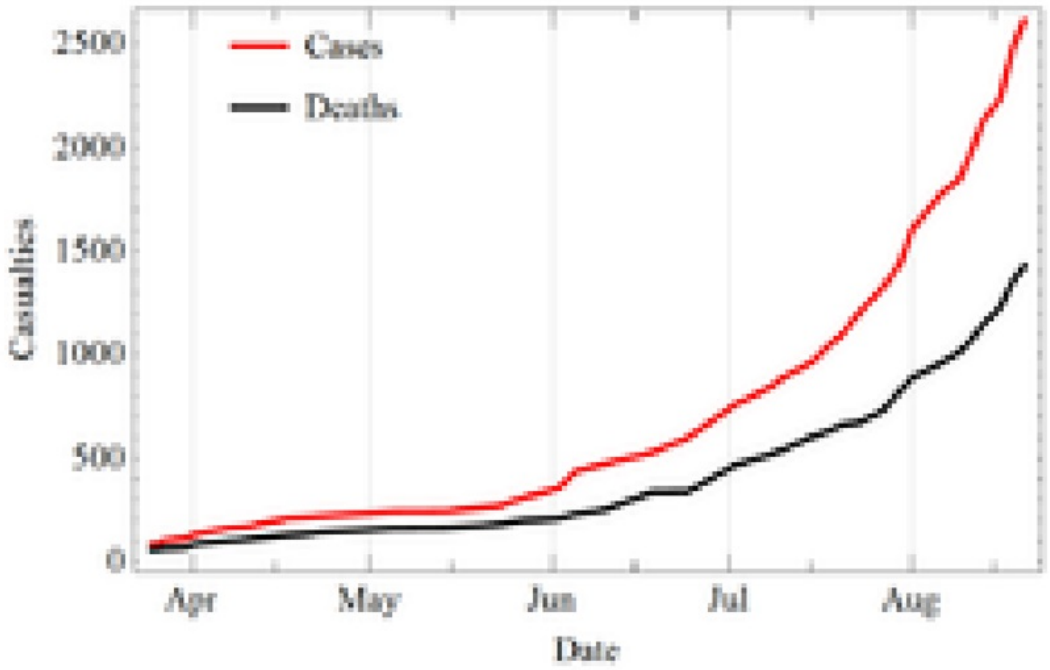
The first recognized outbreaks of Ebola virus disease occurred in 1976 in the Sudan and in Zaire (now the Democratic Republic of the Congo). Since then, there have been multiple outbreaks in central Africa, the largest of which occurred in 2000-2001 and involved 425 people. Outbreaks of Ebola virus disease have occurred primarily in remote villages near tropical rainforests. Historically, the disease has occurred most often in the Democratic Republic of the Congo and also Uganda, South Sudan and Gabon. Since 1976, there have been about 2,200 reported cases with 1,500 deaths (Kaye, 2014).

The disease is caused by members of a family of filoviruses called ebolavirus, and there are five distinct species: Bundibugyo ebolavirus, Tai Forest ebolavirus, Reston ebolavirus,Sudan ebolavirusand Zaire ebolavirus. All but Reston are restricted to Africa and is found in primates in the Philippines and appears not to be a human pathogen. The Zaire species is the most lethal, with a case fatality rate of up to 90%, and is the cause of the current outbreak that began in Guinea in February (Akharumere and Kaye, 2014).

This outbreak is the first-ever to occur this far west in Africa. As of early May, there have been 236 reported clinical cases with 158 deaths in Guinea. Some cases have occurred in neighboring Liberia. As with previous outbreaks, health care workers have been unfortunate victims of the epidemic, with at least 16 deaths (Kaye, 2014).

[1] Visit http://www.who.int/csr/disease/ebola/en/ for more details.

Figure 1.1 West Africa Ebola virus outbreaks 2014



Source: Wikipedia (2014)

In March 2014, the World Health Organization (WHO) reported a major Ebola outbreak in Guinea, a western African nation; it is the largest ever documented, and the first recorded in the region[2]. Researchers traced the outbreak to a two-year old child who died on 6 December[3]. As of 10 April 2014, WHO reported 157 suspected and confirmed cases in Guinea, 22 suspected cases in Liberia, and 8 suspected cases in Sierra Leone[4]. By 2014-07-31, they reported that the death toll had reached 826 people from 1440 cases[5]. On 8 August, the WHO declared the epidemic to be an international public health emergency. Urging the world to offer aid to the affected regions, the

[2] "Guidelines for Evaluation of US Patients Suspected of Having Ebola Virus Disease". *CDC*. 2014-08-01. Retrieved 2014-08-05

[3] Grady, Denise; Sheri Fink (2014-08-09). "Tracing Ebola's Breakout to an African 2-Year-Old". *The New York Times*.ISSN 0362-4331. Retrieved 2014-08-10.

[4] "Outbreak of Ebola in Guinea and Liberia". Centers for Disease Control and Prevention. Retrieved 2014-04-13.
World Health Organization (2014-04-07).
"Ebola virus disease, West Africa (Situation as of 7 April 2014) - Guinea". ReliefWeb.

[5] "Ebola virus disease, West Africa". World Health Organization Regional Office for Africa. 2014-07-31
"WHO raises global alarm over Ebola outbreak". *CBS*. Retrieved 2014-08-02.

Director-General said, "Countries affected to date simply do not have the capacity to manage an outbreak of this size and complexity on their own. I urge the international community to provide this support on the most urgent basis possible[6]." Further attempts to contain the outbreak were enacted by placing troops on roads to cordon off the infected areas and stop those who may be infected from leaving and further spreading the virus[7].

By mid-August 2014, 2,127 suspected cases including 1,145 deaths had been reported, however the World Health Organization has said that these numbers may be vastly underestimated[8]. By mid-August, Doctors without Borders reported the situation in Liberia's capital Monrovia as "catastrophic" and "deteriorating daily". They report that fears of Ebola among staff members and patients have shut down much of the city's health system which has resulted in leaving many people without treatment for other conditions[9]. On 16 August 2014, a quarantine center in West Point, Monrovia was attacked by protesters who distrust the government and health care workers and believe that the epidemic is a hoax. The attack caused a number of patients being monitored for Ebola to flee, while blood-soaked bedding and other infected items were removed. The incident was seen by medical officials as a disaster as it had the potential to accelerate the spread of the disease[10]. Tens of thousands of people in Liberia, Guinea, and Sierra Leone have been under quarantine, leaving them without access to food. The United Nations' World Food Programme has announced that it will deliver rations to 24,000 Liberian people affected by the epidemic.

Figure 1.2 Nigeria's temperature Airport Recorder Framework

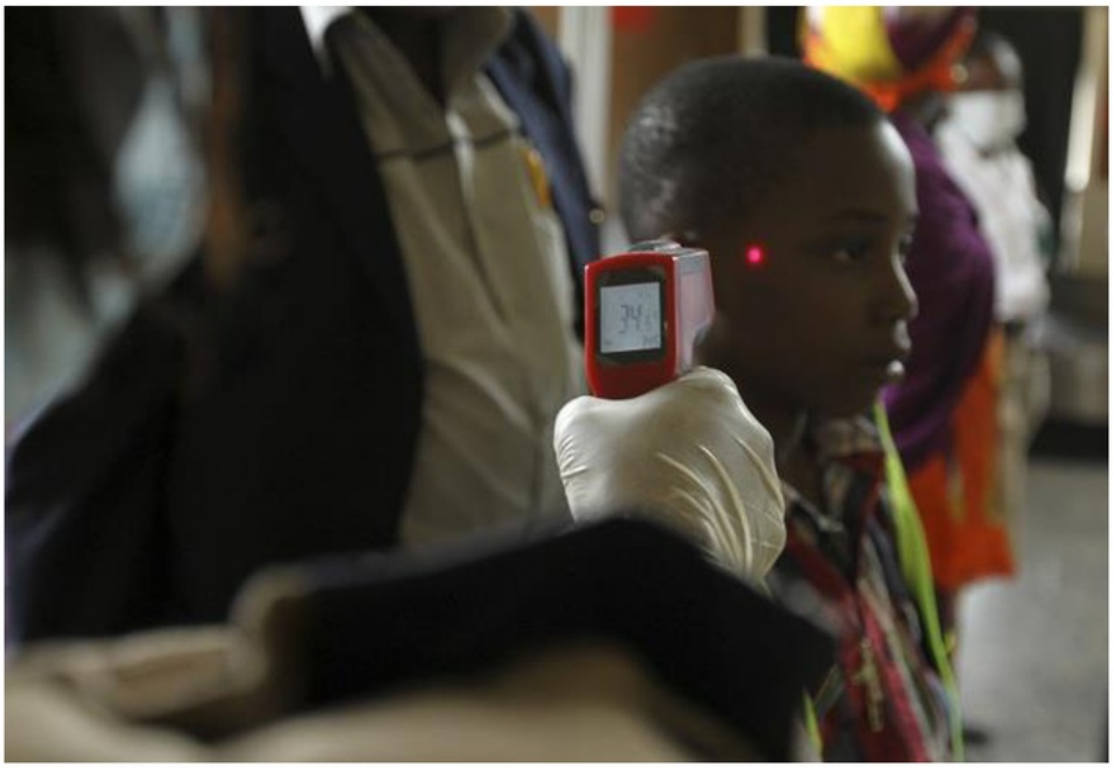
[6] "WHO raises global alarm over Ebola outbreak". *CBS*. Retrieved 2014-08-02.
"Ebola epidemic in West Africa declared a health emergency". *Big News Network.com*. Retrieved 2014-08-02.

[7] "Using a Tactic Unseen in a Century, Countries Cordon Off Ebola-Racked Areas". *New York Times*. Retrieved 2014-08-13.

[8] "Ebola virus disease". Retrieved 2014-08-15.

[9] "In Liberia's Ebola-Stricken Villages, Residents Face 'Stark' Choices". *n Liberia's Ebola-Stricken Villages, Residents Face 'Stark' Choices*. Common Dreams. 18 August 1014. Retrieved 20 August 2014.

[10] "Whole of West Point area at risk after Ebola quarantine centre attacked and looted". Liberia News.Net. 2014-08-17. Retrieved 2014-08-17.



Source: Afolandi Sotunde (2014)

However, Dr. J. Stephen Morrison describes the situation in Lagos, Nigeria, Africa's largest city, where new infections of the Ebola virus have recently been detected. Lagos is massive—21 million people with a massive population density (Global Health Policy Center at the Center for Strategic and International Studies, 2014)

Nigeria's coastal metropolis has a much different geography from the rural settings in which past Ebola crises have played out. Never before has a megacity played host to the virus. Keeping potentially infected individuals isolated and under surveillance—vital during an outbreak—is nearly impossible. It's feared that living conditions in crowded areas without the benefit of reliable sanitation could help spread the disease at rapid

A similar situation once unfolded in a U.S. metropolis—New York City. And that the lessons we learned from reining in that epidemic could offer some guidance for Lagos. Over a 14-year span during the 1980s and early 1990s, New York City battled its own pandemic of infectious disease: tuberculosis. According to The New England Journal of Medicine, the number of TB patients in NYC over the course of those years nearly tripled. By 1991, the city's infections accounted for 61 percent of all TB cases in America—despite the fact that NYC represented only 4 percent of the country's population at the time. Several strains of the disease even became drug resistant, hindering the ability of health workers to treat the infected. Ultimately, the epidemic cost the city over $1 billion. The motive is to establish the relationship between the determinants of

Ebola outbreak to demography of Africa and the impact of Ebola mortality cases on West Africa and Nigeria demographic transition with a view to enhancing the quality of population policy. It is on account of these that research of this nature would identify the trend and structure of Ebola outbreak and its impact on the demographic transition in Nigeria, West Africa, and Africa as a whole.

1.2 AIM AND OBJECTIVES

The aim of the research is to provide empirical evidences on the trends and structure of Ebola outbreak, its impact on Africa's population and demography as well as the impact of Nigeria's Ebola outbreak on West Africa's population and demography. Specifically, the following objectives are pursued:

- To undertake Ebola outbreak appraisal under Africa and other countries in the World, and their trends and structural growth in Malawi, and West Africa and African countries;
- To examine the determinants of Ebola outbreak within the demographic context of Africa;
- To assess the in-depth impact of Ebola outbreak and its adverse effect on the demography of Africa.

1.4 RESEARCH QUESTIONS

The following research questions are relevant to the issues being investigated in the study:

`1) Does Ebola outbreak appraisal under Africa and other countries in the World have any trend and structural growth in Malawi, and West Africa and African countries?

2) Does the determinants of Ebola outbreak have any significant impact on the demography of Africa?

3) Does the in-depth assessment of Ebola outbreak and its adverse effect have any significant effect on the demography of Africa?

Hypotheses

The following null hypotheses are tested in line with the above stated objectives:

1) Ebola outbreak has no transitional trends and structural growth in Africa and other countries in the World, also, in Nigeria and West African Countries.

2) Ebola outbreak has no impact on the demography of Africa in both the short-run and long-run.

3) The in-depth of Ebola outbreak and its adverse effect has no impact on the demography of Africa

2.0 LITERATURE REVIEW

This chapter will cover the conceptual framework of Ebola virus outbreak with a view to elaborate the rationale for various political stakeholders of PDP embark on the fight against this epidemic outbreak. It involves the overview of various researchers of Ebola virus and a structural framework of the virus.

2.1 CONCEPTUAL FRAMEWORK OF EBOLA VIRUS OUTBREAK

The earliest described outbreaks of a filovirus (Marburg [MBG] virus) were in 1967 in Germany and Yugoslavia. Cases of MBG virus infection occurred in South Africa in 1975, in Kenya in 1980, and in Kenya again in 1987. Epidemiologic surveys did not identify a reservoir; however, a biting insect was suspected in South Africa.

Ebola (EBO) epidemics were recorded in the Democratic Republic of the Congo (DRC) and Sudan in 1976; investigations did not discover the virus in insects or mammals. EBO reemerged with a single lethal case in Tandala, DRC, in 1977 and a new outbreak in Sudan in 1979. An outbreak due to a new subtype of the virus, EBO (subtype Reston [EBO-R] has occurred in a colony of cynomolgus mon- keys (Macaca fascicularis) in a quarantine facility in Reston, Virginia, in 1989. The same virus was responsible for three further epizootics among monkeys in the United States in 1990, as well as one outbreak in Italy in 1992. Investigations traced the source of all EBO-R outbreaks to a primate export facility
in the Philippines, but the mode of contamination of this facility was not determined. Although African green monkeys (Cercopithecus aethiops) from Uganda were the first animals known to be infected with filovirus, the cycle of these viruses in nature remains a mystery.

In November 1994, ethologists studying the behavior of a community of chimpanzees (Pan troglodytes verus) in the Tai National Park, Coˆte d'Ivoire, found 8 dead chimpanzees and noted the absence of other individuals. An epidemiologic survey was done to elucidate the cause of these deaths. Herein, we report the results of investigations that led to the identification of a new subtype of the virus, EBO (subtype Coˆte d'Ivoire [EBO-CI]), in the blood of a researcher who was probably infected during a chimpanzee necropsy

2..1.1 SIGNS AND SYMPTOMS OF EBOLA

Signs and symptoms of Ebola usually begin suddenly with an influenza-like stage characterized by fatigue, fever, headaches, joint, muscle and abdominal pain. Vomiting, diarrhea and loss of appetite are also common. Less common symptoms include the following: sore throat, chest pain, hiccups, breath and trouble swallowing. The average time between contracting the infection and the start of symptoms (incubation period) is 8 to 10 days, but it can vary between 2 and 21 days. Skin manifestations may include a maculopapular rash (in about 50% of cases). Early symptoms of EVD may be similar to those of malaria, dengue or other tropical fevers, before the disease progresses to the bleeding phase.

In 40–50% of cases, bleeding from puncture sites and mucous membranes (e.g.gastrointestinal tract, nose, vagina and gums) has been reported. In the bleeding phase, which typically starts 5 to 7 days after first symptoms internal and subcutaneous bleeding may present itself through reddening of the eyes and bloody vomit. Bleeding into the skin may create petechiae, purpura, ecchymoses and hematomas (especially around needle injection sites). Types of bleeding known to occur with Ebola virus disease include vomiting blood, coughing it up or blood in the stool. Heavy bleeding is rare and is usually confined to the gastrointestinal tract. In general, the development of bleeding symptoms often indicates a worse prognosis and this blood loss can result in death. All people infected show some symptoms of system involvement, including impaired blood clotting. If the infected person does not recover, death due to multiple organ dysfunction syndrome occurs within 7 to 16 days (usually between days 8 and 9) after first symptoms.

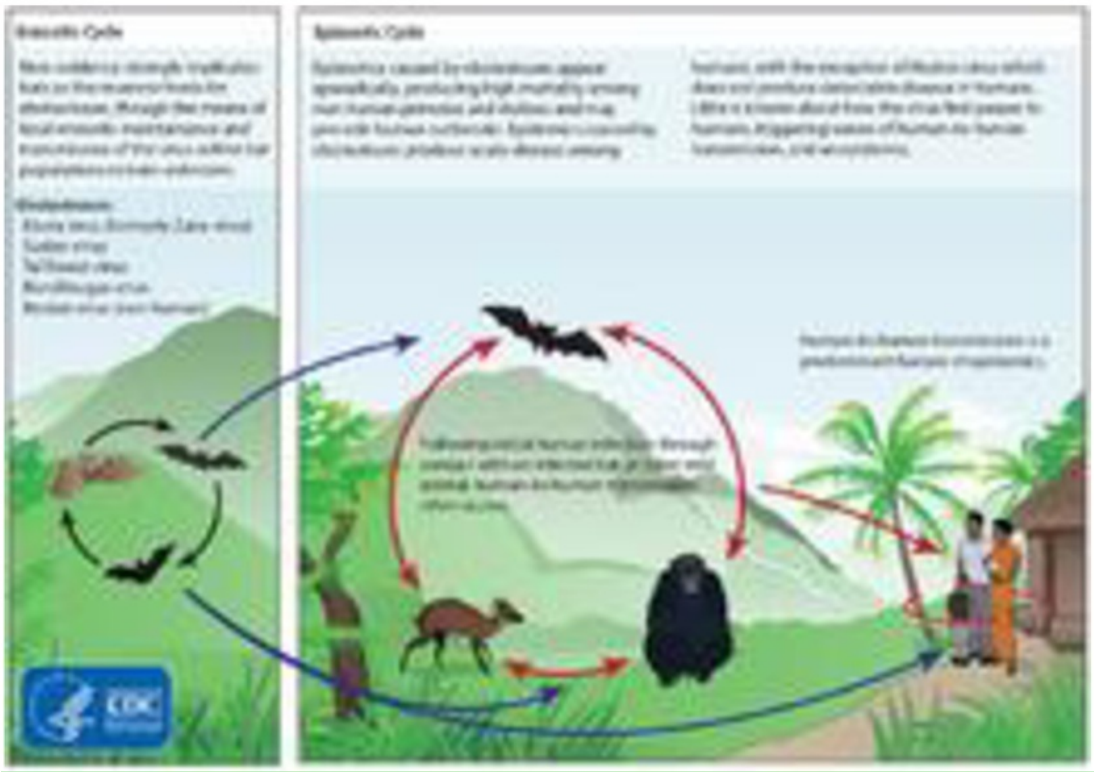
Figure 2.1 Symptoms of Ebola Framework

Source: "Ebola Hemorrhagic Fever: Signs and Symptoms". United States Centers for Disease Control and Prevention (2014)

\2.1.2 Causes, Transmission, Reservoir, and Virology of Ebola

EVD is caused by four of five viruses classified in the genus Ebolavirus, family Filoviridae, order Mononegavirales. The four disease-causing viruses are Bundibugyo virus (BDBV), Sudan virus (SUDV), Taï Forest virus (TAFV), and one called simply, Ebola virus (EBOV, formerly Zaire Ebola virus)). Ebola virus is the sole member of the Zaire ebolavirus species, and the most dangerous of the known Ebola disease-causing viruses, as well as being responsible for the largest number of outbreaks. The fifth virus, Reston virus (RESTV), is not thought to be disease-causing in humans. The five Ebola viruses are closely related to the Marburg viruses.

Figure 2.2 Life cycles of the Ebolavirus



Sources: Kuhn JH, Becker S, Ebihara H, Geisbert TW, Johnson KM, Kawaoka Y, Lipkin WI, Negredo AI, Netesov SV, Nichol ST, Palacios G, Peters CJ, Tenorio A, Volchkov VE, Jahrling PB (2010).

In addition, It is not entirely clear how Ebola is spread. EVD is believed to occur after an Ebola virus is transmitted to an initial human by contact with an infected animal's body fluids. Human-to-human transmission can occur via direct contact with blood or bodily fluids from an infected person (including embalming of an infected dead person) or by contact with contaminated medical equipment, particularly needles and syringes. The potential for widespread EVD infections is considered low as the disease is only spread by direct contact with the secretions from someone who is showing signs of infection. The quick onset of symptoms makes it easier to identify sick

individuals and limits a person's ability to spread the disease by traveling. Because dead bodies are still infectious local traditional burial rituals may spread the disease. Semen may be infectious in survivors for up to 50 days.

Medical workers who do not wear appropriate protective clothing may also contract the disease. In the past, hospital-acquired transmission has occurred in African hospitals due to the reuse of needles and lack of universal precautions.

Airborne transmission has not been documented during EVD outbreaks. They are, however, infectious as breathable 0.8– to 1.2-μm laboratory-generated droplets. The virus has been shown to travel without contact from pigs to nonhuman primates, although the same study failed to achieve transmission in that manner between primates.

Consequently, Bats drop partially eaten fruits and pulp, then land mammals such as gorillas and duikers feed on these fallen fruits. This chain of events forms a possible indirect means of transmission from the natural host to animal populations, which has led to research towards viral shedding in the saliva of bats. Fruit production, animal behavior, and other factors vary at different times and places that may trigger outbreaks among animal populations.

Moreover, Bats are considered the most likely natural reservoir of the EBOV; plants,arthropods, and birds have also been considered. Bats were known to reside in the cotton factory in which the first cases for the 1976 and 1979 outbreaks were employed, and they have also been implicated in Marburg virus infections in 1975 and 1980. Of 24 plant species and 19 vertebrate species experimentally inoculated with EBOV, only bats became infected. The absence of clinical signs in these bats is characteristic of a reservoir species. In a 2002–2003 survey of 1,030 animals including 679 bats from Gabon and the Republic of the Congo, 13 fruit bats were found to contain EBOV RNA fragments. As of 2005, three types of fruit bats (Hypsignathus monstrosus, Epomops franqueti, and Myonycteris torquata) have been identified as being in contact with EBOV. They are now suspected to represent the EBOV reservoir hosts. Antibodies against Ebola Zaire and Reston viruses have been found in fruit bats in Bangladesh, thus identifying potential virus hosts and signs of the filoviruses in Asia.

Between 1976 and 1998, in 30,000 mammals, birds, reptiles, amphibians andarthropods sampled from outbreak regions, no ebolavirus was detected apart from some genetic traces found in six rodents (Mus setulosus and Praomys) and one shrew (Sylvisorex ollula) collected from the Central African Republic. Traces of EBOV were detected in the carcasses of gorillas and chimpanzees during outbreaks in 2001 and 2003, which later became the source of human infections. However, the high lethality from infection in these species makes them unlikely as a natural reservoir.

Transmission between natural reservoir and humans is rare, and outbreaks are usually traceable to a single case where an individual has handled the carcass of gorilla, chimpanzee or duiker. Fruit bats are also eaten by people in parts of West Africa where they are smoked, grilled or made into a spicy soup.
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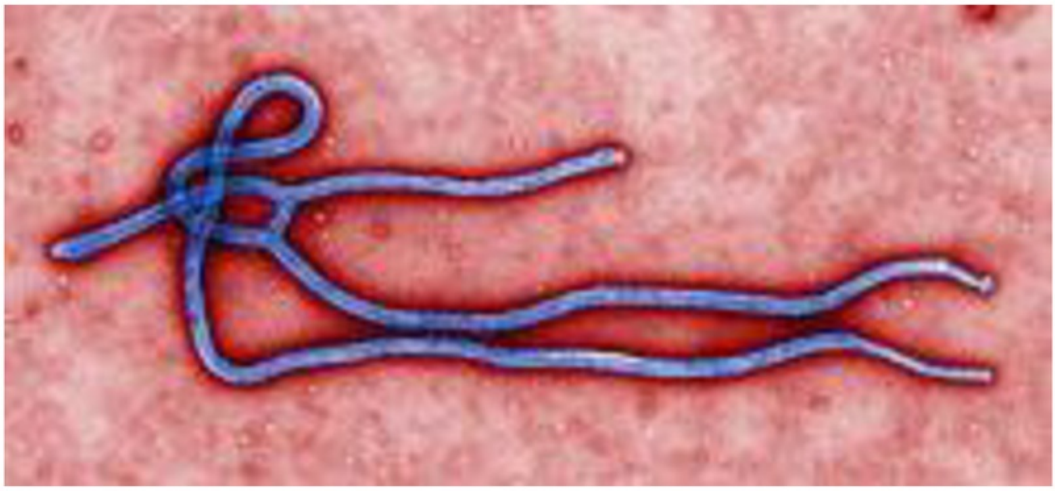
Figure 2.3 Bushmeat Reservoir Framework

Source: Williams E. "African monkey meat that could be behind the next HIV". *Health News - Health & Families*. The Independent. "25 people in Bakaklion, Cameroon killed due to eating of ape" (2014)

Note: Bush meat being prepared for cooking in Ghana, 2013 Human consumption of equatorial animals in Africa in the form of bushmeat has been linked to the transmission of diseases to people, including Ebola.

Like all mononegaviruses, ebolavirions contain linear nonsegmented, single-strand, non-infectious RNA genomes of negative polarity that possesses inverse-complementary 3' and 5' termini, do not possess a 5' cap, are not polyadenylated, and are not covalently linked to a protein. Ebolavirus genomes are approximately 19 kilobase pairs long and contain seven genes in the order 3'-UTR-*NP-VP35-VP40-GP-VP30-VP24-L*-5'-UTR. The genomes of the five different Ebola viruses (BDBV, EBOV, RESTV, SUDV, and TAFV) differ in sequence and the number and location of gene overlaps.

Figure 2.4 Genome Framework (Electron micrograph of an Ebola virus virion)



Source: Kiley MP, Bowen ET, Eddy GA, Isaäcson M, Johnson KM, McCormick JB, Murphy FA, Pattyn SR, Peters D, Prozesky OW, Regnery RL, Simpson DI, Slenczka W, Sureau P, van der Groen G, Webb PA, Wulff H (1982).

Also, like all filoviruses, ebolavirions are filamentous particles that may appear in the shape of a shepherd's crook or in the shape of a "U" or a "6", and they may be coiled, toroid, or branched. In general, ebolavirions are 80 nm in width, but vary somewhat in length. In general, the median particle length of ebolaviruses ranges from 974 to 1,086 nm (in contrast to marburgvirions, whose median particle length was measured at 795–828 nm), but particles as long as 14,000 nm have been detected in tissue culture.

Finally, Ebola virus life cycle begins with virion attachment to specific cell-surface receptors, followed by fusion of the virion envelope with cellular membranes and the concomitant release of the virus nucleocapsid into the cytosol. The viral RNA polymerase, encoded by the L gene, partially uncoats the nucleocapsid and transcribes the genes into positive-strandmRNAs, which are then translated into structural and nonstructural proteins. Ebolavirus RNA polymerase (L) binds to a single promoter located at the 3' end of the genome. Transcription either terminates after a gene or continues to the next gene downstream. This means that genes close to the 3' end of the genome are transcribed in the greatest abundance, whereas those toward the 5' end are least likely to be transcribed. The gene order is, therefore, a simple but effective form of transcriptional regulation. The most abundant protein produced is the nucleoprotein, whose concentration in the cell determines when L switches from gene transcription to genome replication. Replication results in full-length, positive-strand antigenomes that are, in turn, transcribed into negative-strand virus progeny genome copy. Newly synthesized structural proteins and genomes self-assemble and accumulate near the inside of the cell membrane. Virions bud off from the cell, gaining their envelopes from the cellular membrane they bud from. The mature progeny particles then infect other cells to repeat the cycle.The Ebola Virus genetics are difficult to study due to its virulent nature.

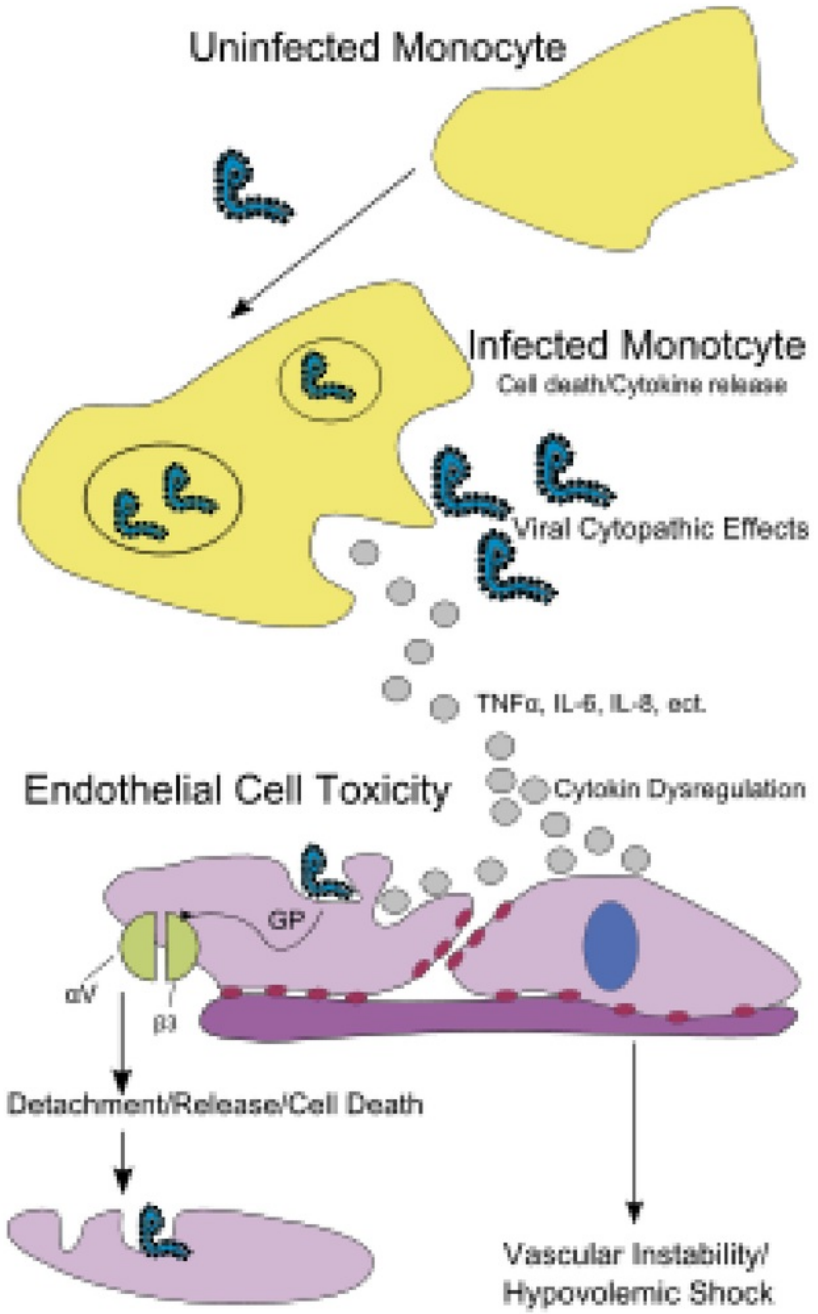
2.2 PATHOPHYSIOLOGY AND DIAGNOSIS

Endothelial cells, mononuclear phagocytes and hepatocytes are the main targets of infection. After infection, a secreted glycoprotein (sGP) known as the Ebola virus glycoprotein (GP) is synthesized. Ebola replication overwhelms protein synthesis of infected cells and host immune defenses. The GP forms a trimeric complex, which binds the virus to the endothelial cells lining the interior surface of blood vessels. The sGP forms a dimeric protein that interferes with the signaling of neutrophils, a type of white blood cell, which allows the virus to evade the immune system by inhibiting early steps of neutrophil activation. These white blood cells also serve as carriers to transport the virus throughout the entire body to places such as the lymph nodes, liver, lungs, and spleen.

The presence of viral particles and cell damage resulting from budding causes the release of cytokines (to be specific, TNF-α, IL-6, IL-8, etc.), which are the signaling molecules for fever and inflammation. The cytopathic effect, from infection in the endothelial cells, results in a loss of vascular integrity. This loss in vascular integrity is furthered with synthesis of GP, which reduces specific integrins responsible for cell adhesion to the inter-cellular structure, and damage to the liver, which leads tocoagulopathy.

The medical history, especially travel and work history along with exposure to wildlife are important to suspect the diagnosis of EVD. The diagnosis is confirmed by isolating the virus, detecting its RNA or proteins, or detecting antibodies against the virus in a person's blood. Isolating the virus by cell culture, detecting the viral RNA by polymerase chain reaction (PCR) and detecting proteins by enzyme-linked immunosorbent assay (ELISA) is effective early and in those who have died from the disease. Detecting antibodies against the virus is effective late in the disease and in those who recover. During an outbreak, virus isolation is often not feasible. The most common diagnostic methods are therefore real time PCR and ELISA detection of proteins, which can be performed in field or mobile hospitals. Filovirions can be seen and identified in cell culture by electron microscopy due to their unique filamentous shapes, but electron microscopy cannot tell the difference between the various filoviruses despite there being some length differences.

Figure 2.5 Pathogenesis schematic Framework



Source: Smith, Tara (2005). Ebola (Deadly Diseases and Epidemics). Chelsea House Publications

The genera Ebolavirus and Marburgvirus were originally classified as the species of the now-obsolete Filovirus genus. In March 1998, the Vertebrate Virus Subcommittee proposed in

the International Committee on Taxonomy of Viruses (ICTV) to change the Filovirus genus to theFiloviridae family with two specific genera: Ebola-like viruses andMarburg-like viruses. This proposal was implemented in Washington, DC, on April 2001 and in Paris on July 2002. In 2000, another proposal was made in Washington, D.C., to change the "-like viruses" to "-virus" resulting in today's Ebolavirus and Marburgvirus.

Rates of genetic change are 100 times slower than influenza A in humans, but on the same magnitude as those of hepatitis B. Extrapolating backwards using these rates indicates that Ebolavirus and Marburgvirus diverged several thousand years ago.

However, paleoviruses (genomic fossils) offiloviruses (Filoviridae) found in mammals indicate that the family itself is at least tens of millions of years old. Fossilized viruses that are closely related to ebolaviruses have been found in the genome of the Chinese hamster.

The symptoms of EVD are similar to those of Marburg virus disease. It can also easily be confused with many other diseases common in Equatorial Africa such as other viral hemorrhagic fevers, falciparum malaria, typhoid fever, shigellosis,rickettsial diseases such as typhus, cholera, gram-negative septicemia, borreliosis such as relapsing fever or EHEC enteritis. Other infectious diseases that should be included in the differential diagnosis include the following: leptospirosis,scrub typhus, plague, Q fever, candidiasis, histoplasmosis, trypanosomiasis, visceral leishmaniasis, hemorrhagic smallpox,measles, and fulminant viral hepatitis. Non-infectious diseases that can be confused with EVD are acute promyelocytic leukemia, hemolytic uremic syndrome, snake envenomation, clotting factor deficiencies/platelet disorders, thrombotic thrombocytopenic purpura, hereditary hemorrhagic telangiectasia, Kawasaki disease and even warfarinpoisoning.

2.3 Epidemiology

Ebola Virus Disease (EVD) Preparedness Plan

Since the Ebola Virus Disease Outbreak in West Africa in 2014, the Ministry of Health in Malawi (with technical guidance from WHO) started implementing a range of activities in preparedness of the Ebola Outbreak. The aims of the activities were to prevent Ebola from being transmitted to Malawi and to prepare the country to handle any Ebola case, should it be diagnosed. Specific activities included:

a. Development of Information Education and Communication (IEC) materials on Ebola and placement of IEC materials at strategic places such as airports, schools, colleges, and health facilities.
b. Radio phone-in programmes where listeners ask questions on Ebola.
c. Strengthening screening procedures at Chileka and Kamuzu international airports, particularly for passengers from Ebola affected countries.
    - According to a Ministerial statement on Ebola in parliament in 2014, renovations for the isolation room (quarantine room) at **Chileka International Airport** are underway. Beds, beddings and a fridge have been supplied. Orientation of Airport staff was done but they are still waiting for more Personal Protection Equipment (PPE) to be prepositioned at the airport. The PPEs are being procured through the Central Medical Stores.

- According to the same source, renovation of the quarantine rooms at **Kamuzu International Airport** is almost complete and beds and linen have been supplied. More PPE is being procured through Central Medical Stores. Airport Staff have been oriented on Ebola.

d. Development of Standard Operating Procedures (SOPs) including:
   - Surveillance at port of entry;
   - Case definition for Ebola Virus Disease;
   - Case management and treatment of cases;
   - SOP for collection, packaging and transportation of laboratory specimen;
   - SOP on handling and transportation of Ebola suspect/confirmed dead bodies;
   - Infection Prevention and Control measures; and
   - Training of Health Workers in Central Hospitals and District Health Offices. The training material is ready for orientations (Training Social workers in EVD psychosocial support).

e. Sensitisation meetings (on Ebola) with central hospitals, zonal and district health offices where it was agreed that:
   - Three Central Hospitals in (Mzuzu, Lilongwe and Blantyre have been designated as treatment centres for Ebola. But these centres should be in isolated buildings, away from the other patients at the hospital.
   - Since Ebola can also come to Malawi by land, 6 border districts are designated as Ebola management centres. These are the border districts especially those with ports of entry at: Mwanza, Dedza, Mchinji, Songwe, Kaporo (Karonga), Chitipa and Muloza (Mulanje)

f. Setting up Ebola Rapid Response Teams for Lilongwe, Blantyre, Mzuzu and in all border districts.

g. Training of laboratory personnel on international certification in sample packaging and transportation.

h. Procurement of PPE[11]

CURRENT STATUS OF HCWM IN MALAWI

It is acknowledged in the HCWM Strategic Plan of Action (2003-2008)[12] that there is no policy document or formal management procedures for health-care wastes in Malawi. Some of the important policies of sound management of health-care related waste include:

a. assignment of legal responsibility for safe management of waste disposal to the waste producers; and

b. high level of awareness on proper waste disposal among all health workers and general public and limited level of awareness of proper waste disposal among health workers and general public.

The disease typically occurs in outbreaks in tropical regions of Sub-Saharan Africa. From 1976 (when it was first identified) through 2013, the World Health Organization reported 1,716 confirmed cases. The largest outbreak to date is the ongoing 2014 West Africa Ebola virus

[11] Information on EVD preparedness plan for Malawi has been extracted from the Ministerial statement on health made in 2014.

[12] Health Care Waste Management Plan of Action. (July 2003-June 2008). Malawi Government. Ministry of Health and Population. Lilongwe.

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outbreak, which is affecting Guinea, Sierra Leone, Liberia and Nigeria As of 13 August, 2,127 cases have been identified, with 1,145 deaths.

The first identified case of Ebola was on 26 August 1976, in Yambuku, a small rural village in Mongala District in northern Democratic Republic of the Congo (then known as Zaire). The first victim, and the index case for the disease, was village school headmaster Mabalo Lokela, who had toured an area near the Central African Republic border along the Ebola river between 12–22 August. On 8 September he died of what would become known as the Ebola virus species of the ebolavirus. Subsequently a number of other cases were reported, almost all centered on the Yambuku mission hospital or having close contact with another case. 318 cases and 280 deaths occurred in the DRC. The Ebola outbreak was contained with the help of the World Health Organization and transport from the Congolese air force, by quarantining villagers, sterilizing medical equipment, and providing protective clothing. The virus responsible for the initial outbreak, first thought to be Marburg virus was later identified as a new type of virus related to Marburg, and named after the nearby Ebola river. Another ebolavirus, the Sudan virus species, was also identified that same year when an outbreak occurred in Sudan, affecting 284 people and killing 151.

The second major outbreak occurred in 1995 in the Democratic Republic of Congo, affecting 315 and killing 254. The next major outbreak occurred in Uganda in 2000, affecting 425 and killing 224; in this case the Sudan virus was found to be the ebolavirus species responsible for the outbreak. . In 2003 there was an outbreak in the Republic of Congo that affected 143 and killed 128, a death rate of 90%, the largest to date.

In August 2007, 103 people were infected by a suspected hemorrhagic fever outbreak in the village of Kampungu, Democratic Republic of the Congo. The outbreak started after the funerals of two village chiefs, and 217 people in four villages fell ill. The 2007 outbreak eventually affected 264 individuals and resulted in the deaths of 187.

On 30 November 2007, the Uganda Ministry of Health confirmed an outbreak of Ebola in the Bundibugyo District in Western Uganda. After confirmation of samples tested by the United States National Reference Laboratories and the Centers for Disease Control, the World Health Organization confirmed the presence of a new species of Ebolavirus, which was tentatively named Bundibugyo. The WHO reported 149 cases of this new strain and 37 of those led to deaths.

The WHO confirmed two small outbreaks in Uganda in 2012. The first outbreak affected 7 people and resulted in the death of 4 and the second affected 24, resulting in the death of 17. The Sudan variant was responsible for both outbreaks.

On 17 August 2012, the Ministry of Health of the Democratic Republic of the Congo reported an outbreak of the Ebola-Bundibugyo variant in the eastern region. Other than its discovery in 2007, this was the only time that this variant has been identified as the ebolavirus responsible for an outbreak. The WHO revealed that the virus had sickened 57 people and claimed 29 lives. The probable cause of the outbreak was tainted bush meat hunted by local villagers around the towns of Isiro and Viadana.

Ebola virus was first isolated in 1976 during outbreaks of Ebola hemorrhagic fever in the Democratic Republic of the Congo (then Zaire) and Sudan. The strain of Ebola that broke out in the Democratic Republic of the Congo had one of the highest case fatality rates of any human virus, 88%.

The name of the disease originates from the first recorded outbreak in 1976 in Yambuku, Democratic Republic of the Congo, which lies on the Ebola River. In late 1989, Hazelton Research Products' Reston Quarantine Unit in Reston, Virginia suffered a mysterious outbreak of fatal illness (initially diagnosed as Simian hemorrhagic fever virus(SHFV)) among a shipment of crab-eating macaque monkeys imported from the Philippines. Hazelton's veterinary pathologist sent tissue samples from dead animals to the United States Army Medical Research Institute of Infectious Diseases (USAMRIID) at Fort Detrick, Maryland, where a laboratory test known as an ELISA assay showed antibodies to Ebola virus. An electron microscopist from USAMRIID discovered filovirusessimilar in appearance to Ebola in the tissue samples sent from Hazelton Research Products' Reston Quarantine Unit.

Shortly afterward, a US Army team headquartered at USAMRIID went into action to euthanize the monkeys which had not yet died, bringing those monkeys and those which had already died of the disease to Ft. Detrick for study by the Army's veterinary pathologists and virologists, and eventual disposal under safe conditions.

Blood samples were taken from 178 animal handlers during the incident. Of those six animal handlers eventually reconverted. When the handlers did not become ill, the CDC concluded that the virus had a very low pathogenicity to humans. The Philippines and the United States had no previous cases of Ebola infection, and upon further isolation, researchers concluded it was another strain of Ebola, or a new filovirus of Asian origin, which they named Reston ebolavirus (REBOV) after the location of the incident.

The research has therefore been undertaken purposely to fill methodological and empirical review gap. This provides literature that could be useful to policy makers and academics in explaining the relationship between Ebola outbreak in Nigeria, West Africa and Africa and its impact on demographic transition in Africa.

3.0 THEORETICAL FRAMEWORK AND METHODOLOGY

In the literature review, we were able to establish the nexus between Ebola Virus and its outbreak through the direct approaches. It is pertinent to show that in this section, an economic growth model that captures the dynamic behaviour Ebola virus on the level of human mortality and other control variables.

However, it should be noted that a direct impact of Ebola outbreak on human mortality can be established on the basis of education and health. Thus, this brings the section to the adoption of the conventional "neoclassical" growth theory as modeled by Robert Solow (1956). He holds the view that economic growth as a result of the accumulation of physical capital and expansion of the

labour force, in conjunction with an "exogenous" factor technological progress that makes physical capital and labour productive.

Basically, the foregoing theoretical exposition leads to the augmented Solow model which is often formulated in a Cobb-Douglas production function. The model acknowledges the studies of Ayara (2003), Mankiw et al (1992), Pritchett (2001), Grammy and Assane (1996), and Akharumere (2014). Such a model is regarded important because it provides the appropriate framework for examining the contribution of human capital to economic growth.

Given the Cobb Douglas production function as:

$$Y = A\,L^{\alpha}K^{\beta} \qquad \text{----------} \qquad (3.1)$$

`Where Y is output level, K and L are capital and labour respectively and α and β are the labour and capital elasticity of output respectively. This production function is characterized by constant return to scale since, the capital stock K is composed of two components: physical capital K_p and Human Capital K_h.

Therefore capital stock K can be expressed as:

$$K = [K_p, K_h] \qquad \text{----------} \qquad (3.2)$$

Incorporating human capital into the growth process, the production function is modified as:

$$Y = A\,L^{1-\alpha-\beta}(K_p{}^{\alpha}, K_h{}^{\beta}) \qquad \text{----------} \qquad (3.3)$$

Where Y = Output; K_p = Physical Capital; K_h = Human Capital; A = Level of Technology; and L = Labour Force.

We assure $\alpha+\beta<1$, which implies that there is a decreasing return to capital. Therefore, defining y as the level of output per effective unit of labour, y can be stated as:

$$y = \frac{Y}{AL};$$

K_p as the stock of physical capital per effective unit of labour, thus,

$$k_p = \frac{K_p}{AL};$$ and

K_h as the stock of human capital per effective units of labour, such that,

$$k_h = \frac{K_h}{AL}.$$

If equation (3.3) is defined in terms of output per labour, then it becomes;

$$y = A^{1/1-\alpha-\beta}[K_p{}^{\alpha}, K_h{}^{\beta}] \qquad \text{----------} \qquad (3.4)$$

Taking the log of both side of the equation, we have;

$$\text{Log}(y) = \text{Log } A + \frac{\alpha}{1-\alpha-\beta}\text{Log}(K_p) + \frac{\beta}{1-\alpha-\beta}\text{Log}(K_h) \quad \text{----------} \quad (3.5)$$

However, let us assign the constant coefficient as;

$$a_1 = \frac{\alpha}{1-\alpha-\beta}$$

$$a_2 = \frac{\beta}{1-\alpha-\beta}$$

The corresponding equation becomes:

$$\text{`Log}(y) = a_0 + a_1 \text{Log}(K_p) + a_2 \text{Log}(K_h) + u_t \quad \text{----------} \quad (3.6)$$

Where u_t is the residual and thus taking the natural logarithm we have;

$$\text{`In}(Y_t) = a_0^* + a_1^* \text{In}(K_p^*) + a_2^* \text{In}(K_h^*) + u_t^* \quad \text{----------} \quad (3.7)$$

It is however imperative that equation (3.7) can be disaggregated to represent various equations for this research as follows:

$$C_t = \beta_0 + \beta_1 D_t + DUM_1 + \mu_t \quad \text{----------} \quad (3.8)$$

Alternatively, we can re-write the equation as:

$$D_t = \alpha_0 + \alpha_1 C_t + DUM_2 + \varepsilon_t \quad \text{----------} \quad (3.9)$$

Finally we can also re-write the equation as:

$$F_t = \theta_0 + \theta_1 C_t + \theta_2 D_t + DUM_3 + \rho_t \quad \text{----------} \quad (3.10)$$

Where C = Cases of Reported Human Virulence
D = Deaths of Reported Human Virulence
F = Fatality of Reported Human Virulence
DUM = Dummy
0 = Other Countries; 0 = Nigeria; 0 = African Countries
1 = African Countries; 1 = West African Countries; 1 = West African Countries
t = 1976 – 2014

In conclusion, a simple linear regression will be used to carryout the analysis such that the empirical finding can be deduced from the statistical summary output of the ANOVA tables.

4.0 Empirical Findings and Discussion

4.1 Ordinary Least Squares (OLS) Results

The summary of the results obtained from the ordinary least squares test statistics is stated below:

Table 4.1 Summary Output on OLS Result 1

SUMMARY OUTPUT

Regression Statistics	
Multiple R	0.994519
R Square	0.989068
Adjusted R Square	0.988157
Standard Error	54.4605
Observations	27

ANOVA

	df	*SS*	*MS*	*F*	*Significance F*
Regression	2	6440493	3220247	1085.74	2.91E-24
Residual	24	71182.7	2965.946		
Total	26	6511676			

	Coefficients	*Standard Error*	*t Stat*	*P-value*	*Lower 95%*	*Upper 95%*	*Lower 99.0%*	*Upper 99.0%*
Intercept	2.166667	22.2334	0.097451	0.923178	-43.7208	48.05416	-60.0188	64.35215
Dt	1.808743	0.039625	45.64705	7.7E-25	1.726962	1.890524	1.697915	1.91957
DUM	-22.4744	25.83966	-0.86977	0.393045	-75.8049	30.856	-94.7464	49.79753

RESIDUAL OUTPUT

Observation	*Predicted Ct*	*Residuals*	*Standard Residuals*
1	486.1402	-168.14	-3.21345
2	252.8124	31.18762	0.596049
3	-18.499	19.49904	0.37266
4	19.48456	14.51544	0.277415
5	2.166667	-2.16667	-0.04141
6	2.166667	1.833333	0.035038

PROBABILITY OUTPUT

Percentile	*Ct*
1.851852	0
5.555556	0
9.259259	0
12.96296	1
16.66667	1
20.37037	2

7	2.166667	0.833333	0.015926	24.07407	3
8	2.166667	-2.16667	-0.04141	27.77778	4
9	35.76325	16.23675	0.310312	31.48148	6
10	-20.3078	21.30778	0.407228	35.18519	17
11	431.8779	-116.878	-2.23374	38.88889	24
12	17.67582	19.32418	0.369318	42.59259	32
13	-18.499	20.49904	0.391772	46.2963	34
14	2.166667	-2.16667	-0.04141	50	35
15	61.08565	-1.08565	-0.02075	53.7037	37
16	384.8506	40.14939	0.767323	57.40741	52
17	153.3315	-31.3315	-0.5988	61.11111	60
18	211.2113	-68.2113	-1.30363	64.81481	77
19	32.14576	2.854238	0.054549	68.51852	122
20	-7.64658	24.64658	0.471038	72.22222	143
21	317.9271	-53.9271	-1.03064	75.92593	149
22	46.6157	102.3843	1.956739	79.62963	264
23	2.166667	3.833333	0.073262	83.33333	284
24	5.01462	26.98538	0.515737	87.03704	315
25	10.44085	13.55915	0.259139	90.74074	318
26	44.80696	32.19304	0.615264	94.44444	425
27	2560.768	54.23181	1.036462	98.14815	2615

Source: Own Composition

From the table above, it can be seen that one per cent increase in death of reported human virulence leads to 1.81 per cent increase in cases of reported human virulence. This indicates that cases of reported human virulence almost double the percentage of death of reported human virulence. This means that African governments should provide awareness campaign and the vaccine as there is enough time for intervention before death of the victims. Also, the trend of occurrence is negatively related to the cases of reported human virulence. This shows that one per cent increase in the trend and structure of Ebola outbreak will lead to 22.74 per cent decrease in the cases of reported human virulence. This is a clear indication that there will be a reduction in the cases of reported human virulence in the mere future if health and demographic policies are put in place by the African government all inclusive *ceteris paribus*.

Table 4.2 Summary Output on OLS Result 2

SUMMARY OUTPUT

Regression Statistics	
Multiple R	0.994566
R Square	0.989161

Adjusted R Square	0.988258
Standard Error	29.93766
Observations	27

ANOVA

	df	*SS*	*MS*	*F*	*Significance F*
Regression	2	1962995	981497.4	1095.099	2.63E-24
Residual	24	21510.33	896.2638		
Total	26	1984505			

	Coefficients	*Standard Error*	*t Stat*	*P-value*	*Lower 95%*	*Upper 95%*	*Lower 99.0%*	*Upper 99.0%*
Intercept	-1.18425	12.22203	-0.09689	0.923615	-26.4093	24.04078	-35.3685	33.00003
Ct	0.546575	0.011974	45.64705	7.7E-25	0.521862	0.571288	0.513084	0.580065
DUM	13.91286	14.14425	0.983641	0.335105	-15.2794	43.10514	-25.6477	53.47345

RESIDUAL OUTPUT

Observation	*Predicted Dt*	*Residuals*	*Standard Residuals*
1	186.5393	93.46066	3.249316
2	167.9558	-16.9558	-0.5895
3	13.27519	-12.2752	-0.42677
4	31.31215	-9.31215	-0.32375
5	-1.18425	1.184245	0.041172
6	1.002053	-1.00205	-0.03484
7	0.455479	-0.45548	-0.01584
8	-1.18425	1.184245	0.041172
9	41.15049	-10.1505	-0.3529
10	13.27519	-13.2752	-0.46153
11	184.8996	65.10038	2.263324
12	32.95187	-11.9519	-0.41553
13	13.82176	-12.8218	-0.44577
14	-1.18425	1.184245	0.041172
15	45.52309	-0.52309	-0.01819
16	245.0228	-21.0228	-0.73089
17	79.41072	16.58928	0.576754
18	90.88878	37.11122	1.290234
19	31.85872	-2.85872	-0.09939
20	22.02038	-15.0204	-0.52221

PROBABILITY OUTPUT

Percentile	*Dt*
1.851852	0
5.555556	0
9.259259	0
12.96296	0
16.66667	0
20.37037	0
24.07407	0
27.77778	1
31.48148	1
35.18519	7
38.88889	14
42.59259	17
46.2963	21
50	22
53.7037	29
57.40741	31
61.11111	36
64.81481	37
68.51852	45
72.22222	96

21	157.0243	29.97569	1.042155	75.92593	128
22	94.16823	-57.1682	-1.98755	79.62963	151
23	2.095203	-2.0952	-0.07284	83.33333	187
24	30.219	-16.219	-0.56388	87.03704	224
25	25.8464	-8.8464	-0.30756	90.74074	250
26	54.81486	-18.8149	-0.65413	94.44444	280
27	1442.021	-15.0213	-0.52224	98.14815	1427

Source: Own Composition

An alternative presentation of equation 3.8 as revealed in equation 3.9 indicated a unique result different from the initial regression. This summary output shows that one per cent increase in cases of reported human virulence would lead to 0.55 per cent increase in death of human virulence. This also justifies that the number of cases of report of human virulence doubles the number of death of reported human virulence. Preventive measure and awareness advert has to be putin place through media and other means of awareness campaign especially in the area of cleanliness and personal hygiene. There is the existence of goodness of fit as the coefficient of determination for both tables are 0.989068 and 0.989161 per cent respectively.

Table 4.3 Summary Output on OLS Result 3

SUMMARY OUTPUT

Regression Statistics	
Multiple R	0.818868
R Square	0.670545
Adjusted R Square	0.599947
Standard Error	11.15526
Observations	18

ANOVA

	df	*SS*	*MS*	*F*	*Significance F*
Regression	3	3545.841	1181.947	9.498135	0.001116
Residual	14	1742.159	124.4399		
Total	17	5288			

	Coefficients	*Standard Error*	*t Stat*	*P-value*	*Lower 95%*	*Upper 95%*	*Lower 99.0%*	*Upper 99.0%*
Intercept	60.95625	4.046469	15.06406	4.81E-10	52.27744	69.63506	48.91055	73.00195
Ct	-0.27794	0.060905	-4.56347	0.000442	-0.40857	-0.14731	-0.45924	-0.09663
Dt	0.45045	0.085869	5.245783	0.000124	0.266279	0.63462	0.194831	0.706068

DUM	87.05838	60.35777	1.442372	0.171195	-42.3962	216.5129	-92.6172	266.734

RESIDUAL OUTPUT

Observation	*Predicted Ft*	*Residuals*	*Standard Residuals*
1	98.69807	-10.6981	-1.05678
2	50.03993	2.960072	0.292404
3	61.41627	3.583727	0.35401
4	60.46745	-0.46745	-0.04618
5	86.01839	-7.01839	-0.6933
6	60.13201	-3.13201	-0.30939
7	64.55024	10.44976	1.032254
8	43.73358	9.266421	0.915361
9	70.29106	8.708941	0.860292
10	78.86876	11.13124	1.099573
11	64.29148	18.70852	1.848076
12	59.38446	-18.3845	-1.81607
13	71.81487	-0.81487	-0.08049
14	36.21021	-11.2102	-1.10737
15	58.36855	-13.3685	-1.32058
16	61.9434	9.056602	0.894635
17	55.77126	-8.77126	-0.86645
18	64	-2.8E-14	-2.8E-15

PROBABILITY OUTPUT

Percentile	*Ft*
2.777778	25
8.333333	41
13.88889	45
19.44444	47
25	53
30.55556	53
36.11111	57
41.66667	60
47.22222	64
52.77778	65
58.33333	71
63.88889	71
69.44444	75
75	79
80.55556	79
86.11111	83
91.66667	88
97.22222	90

Source: Own Composition

Loosely speaking, it can be seen that introducing fatality into the model causes the establishment of a relationship between cases of reported human virulence and death of reported human virulence. It indicated that one per cent increase if cases of reported human virulence will lead to 0.28 decrease in fatality of reported human virulence. This means that the ability for cases to be reported to the hospital leads to the reduction of fatal human virulence, This means that cases of Ebola virus outbreak should be reported if any victim is suspected in the neighbourhood. Also, the result indicated the one per cent increase in death of report human virulence will lead to 0.45 increases in fatality of reported human virulence. This means that death caused by victims infects their loved ones or during burial as there is a positive relationship between death of reported human virulence and fatality of reported human virulence. Finally, one per cent increase in the trend and structure of Ebola outbreak will lead to 87/06 per cent increase in fatality of reported human virulence.

5.0 Summary, Conclusion and Policy Recommendation

5.1 Summary and Conclusion

This research has examined the relationship between the determinants of Ebola outbreak and its impact on the demography of Africa using annual time series data between 1976 and 2014. It is carried out under the theoretical construct that if Ebola outbreak causes cases of reported human virulence and cases of reported human virulence causes fatality of reported human virulence and invariably death of reported human virulence in Malawi as it is in other African and neighboring countries then, by inference, a developed education and health sector will definitely bring about reduction of the Ebola virus outbreak and economic growth in Nigeria and Africa as a whole. In the research, the empirical continental relationship among the variables is examined using an econometric tool known as the ordinary least squares (OLS) analysis.

Therefore, occupational exposure rates are estimated based on the treatment of patients with Ebola virus disease (EVD) in a dedicated Ebola virus Treatment Centre (ETC). Treatment in other healthcare settings is not considered, since treatment in other settings is not recommended and the occupational exposure of healthcare and other workers in other settings may be highly variable. In small health care centres there may be a low ratio of health workers to EVD patients. For example the 1976 DRC outbreak and the Kikwit outbreak in 1995 started in healthcare settings with few healthcare staff and the number of EVD cases far exceeded the number of healthcare staff.

Conversely, in large urban hospitals, a single unrecognised EVD case may have contact with a large number of health care and ancillary staff: for example in outbreaks of highly pathogenic avian influenza A/H5N1, as many as 30 healthcare workers have been exposed to a single unrecognised case. The mean occupational exposure rates are based on current Médecins Sans Frontières standards for the EVD outbreak in West Africa. The following figures are per 10 ETC beds: 4 clinical teams that work 8 hour shifts (with one team on rest day). Each team is composed of 1 doctor, 3 nurses and 3 ancillary staff. In addition to these teams that work directly in the ward, 4 transport staff (not on team shift work) are also included. In total, this equates to a total of 3.2 healthcare workers per bed. All these values can be adjusted in the health budget.

5.2 POLICY RECOMMENDATION

The assignment has established the need for the Government of Malawi to move significantly in its efforts of eliminating TB/MDR-TB/XDR-TB and improving environmental, health and safety practices in both the mining and health sectors. It has been found in this study that the current situation of TB infection control measures in Malawi cannot guarantee safety among health-care workers, patients, and general population. The assessment has also shown that the current health care waste management procedures present high risk of infection hence need for improvement in management of health-care waste. The gap analysis on Ebola Virus Disease has shown that other stakeholders such as department of wildlife were not included in the EVD preparedness plan.

Sequel to the above findings, the research recommends the following key issues for policy consideration

- Government should adopt modern security technologies and infrastructures in the health sector by establishing the Ebola healthcare center for the manufacturing of vaccines and treatment of affected persons which we help to combat Ebola outbreak challenges ranging from cases of reported human virulence, fatality of reported human virulence to death of reported human virulence in the ECOWAS sub-region and Africa as a whole. It will also save cost of importation of vaccines and improve the human capacity building of the African population.

- Put suspected or confirmed cases in single *isolation rooms* with an adjoining dedicated toilet or latrine, showers, sink equipped with running water, soap and single-use towels, alcohol-based hand rub dispensers, stocks of personal protective equipment (PPE), stocks of medicines, good ventilation, screened windows, doors closed and restricted access;2 if isolation rooms are unavailable, *cohort* these patients in specific confined areas while rigorously *keeping suspected and confirmed cases separate* and ensure the items listed here for isolation rooms are readily available. Make sure that there is at least 1 meter (3 feet) distance between patient beds.

- Ensure that clinical and non-clinical personnel are assigned exclusively to HF patient care areas and that members of staff do not move freely between the HF isolation areas and other clinical areas during the outbreak.

- Restrict all non-essential staff from HF patient care areas.

- Stopping visitor access to the patient is preferred, but if this is not possible, limit their number to include only those necessary for the patient's well-being and care, such as a child's parent.
- .Do not allow other visitors to enter the isolation rooms/areas and ensure that any visitors wishing to observe the patient do so from an adequate distance (approximately 15 m or 50 feet). . Before allowing visitors to HF patients to enter the HCF, screen them for signs and symptoms of HF.

Africa is a fast economic growing continent and the government should put in place these various demographic health policies to ensure that economic development through guaranteeing well-being and productivity of the people as stipulated in Nigeria's vision 20:2020 and one vision be a reality and a plus to the nation Malawi and Africa as a whole.

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Appendix I

List of Ebola outbreaks

Date	Country	Species	Reported human virulence			Description
			Cases	Deaths	Fatality	
1976 Aug	Zaire	EBOV	318	280	88%	First recognition of Ebola virus disease. Occurred in Yambuku and surrounding areas. Disease was spread by close personal contact and by use of contaminated needles and syringes in hospitals/clinics.[10]
1976	Sudan	SUDV	284	151	53%	Occurred in Nzara, Maridi and the surrounding area. Disease was spread mainly through close personal contact within hospitals. Many medical care personnel were infected.[11]
1977	Zaire	EBOV	1	1	n/a	Noted retroactively in the village of Tandala.[12]
1979	Sudan	SUDV	34	22	65%	Occurred in Nzara, Maridi. Recurrent outbreak at the same site as the 1976 Sudan epidemic.[13]
1989	USA	RESTV	0	0	n/a	RESTV was introduced into quarantine facilities in Virginia and Pennsylvania by monkeys imported from the Philippines.[14]
1990	USA	RESTV	4[note 1]	0	n/a	RESTV was introduced once again into quarantine facilities in Virginia and Texas by monkeys imported from the Philippines. Four humans

Date	Country	Species	Reported human virulence			Description
			Cases	Deaths	Fatality	
						developed antibodies but did not get sick.[15]
1989–1990	Philippines	RESTV	3[note 2]	0	n/a	High mortality among crab-eating macaques in a primate facility responsible for exporting animals in the USA.[16] Three workers in the animal facility developed antibodies but did not get sick.[17]
1992	Italy	RESTV	0	0	n/a	RESTV was introduced into quarantine facilities in Siena by monkeys imported from the same export facility in the Philippines that was involved in the episodes in the United States. No humans were infected.[18]
1994	Gabon	EBOV	52	31	60%	Occurred in Mékouka and other gold-mining camps deep in the rain forest. Initially thought to be yellow fever; identified as Ebola hemorrhagic fever in 1995.[19]
1994	Ivory Coast[note 3]	TAFV	1	0	n/a	First and thus far only recognition of TAFV. Approximately one week after conducting necropsies on infected western chimpanzees in Taï National Park, a scientist contracted the virus and developed symptoms similar to those of dengue fever. She was discharged from a Swiss hospital two weeks later, and

Date	Country	Species	Reported human virulence			Description
			Cases	Deaths	Fatality	
						fully recovered after six weeks.[21]
1995	Zaire	EBOV	315	250	79%	Occurred in Kikwit and surrounding area. Traced to index case-patient who worked in forest adjoining the city. Epidemic spread through families and hospitals.[22]
1996 Jan–Apr	Gabon	EBOV	37	21	57%	Occurred in Mayibout area. A chimpanzee found dead in the forest was eaten by people hunting for food. Nineteen people who were involved in the butchery of the animal became ill; other cases occurred in family members.[19]
1996	South Africa	EBOV	2	1	n/a	A medical professional traveled from Gabon to Johannesburg, South Africa, after having treated Ebola virus-infected patients and thus having been exposed to the virus. He was hospitalized, and a nurse who took care of him became infected and died.[23]
1996 Mar	Philippines USA	RESTV	0	0	n/a	RESTV was introduced into a quarantine facility in Texas by crab-eating macaques from a monkey export facility in the Philippines. No human infections were identified.[24][25]

Date	Country	Species	Reported human virulence			Description
			Cases	Deaths	Fatality	
1996–1997 Jul–Jan	Gabon	EBOV	60	45	75%	Occurred in Booué area with transport of patients to Libreville. Index case-patient was a hunter who lived in a forest camp. Disease was spread by close contact with infected persons. A dead chimpanzee found in the forest at the time was determined to be infected.[19]
2000–2001	Uganda	SUDV	425	224	53%	Occurred in Gulu, Masindi, and Mbarara districts of Uganda. The three greatest risks associated with Ebola virus infection were attending funerals of Ebola hemorrhagic fever case-patients, having contact with case-patients in one's family, and providing medical care to Ebola case-patients without using adequate personal protective measures.[26]
2001–2002 Oct–Jul	Gabon Congo	EBOV	122	96	79%	Occurred over the border of Gabon and the Republic of the Congo. This was the first time that Ebola hemorrhagic fever was reported in the Republic of the Congo.[27]
2002–2003 Dec–Apr	Congo	EBOV	143	128	90%	Occurred in the districts of Mbomo and Kéllé in Cuvette Ouest Département.[28]
2003 Nov–Dec	Congo	EBOV	35	29	83%	Occurred in Mbomo and Mbandza villages located in

Date	Country	Species	Reported human virulence			Description
			Cases	Deaths	Fatality	
						Mbomo district, Cuvette Ouest Département.[29]
2004	Sudan	SUDV	17	7	41%	Occurred in Yambio county in Western Equatoria of southern Sudan. This outbreak was concurrent with an outbreak of measles in the same area, and several suspected EHF cases were later reclassified as measles cases.[30]
2007	DR Congo	EBOV	264	187	71%	Occurred in Kasai-Occidental Province. The outbreak was declared over on November 20. Last confirmed case on October 4 and last death on October 10.[31]
2007–2008 Dec–Jan	Uganda	BDBV	149	37	25%	First recognition of BDBV. Occurred inBundibugyo District in western Uganda.[2][3][4]
2008 Nov	Philippines	RESTV	6[note 4]	0	n/a	First recognition of RESTV in pigs. Strain closely similar to earlier strains. Six workers from the pig farm and slaughterhouse developed antibodies but did not become sick.[32][33]
2008–2009 Dec–Feb	DR Congo	EBOV	32	14	45%	Occurred in the Mweka and Luebo health zones of the Province of Kasai-Occidental.[34]

Date	Country	Species	Reported human virulence			Description
			Cases	Deaths	Fatality	
2012 Jun–Aug	Uganda	SUDV	24	17	71%	Occurred in the Kibaale District.[35]
2012 Jun–Nov	DR Congo	BDBV	77	36	47%	Occurred in Province Orientale.[36][37]
2013–2014 Dec–present	Guinea Liberia[note 5] Sierra Leone Nigeria	EBOV	2,615	1,427	64%	*Main article: 2014 West Africa Ebola virus outbreak* The most severe Ebola outbreak in recorded history in regards to both the number of human cases and fatalities.[40] It began in Guéckédou, Guinea[41] and spread to Sierra Leone and Liberia. Several cases have occurred in Nigeria, in travelers from infected areas, and subsequently in health care workers.[42]

Source: From Wikipedia, the free encyclopedia (2014)

Appendix II

Date	Country	Species	Ct	Dt	DUM
1976	Zaire	EBOV	318	280	1
1976	Sudan	SUDV	284	151	1
1977	Zaire	EBOV	1	1	1
1979	Sudan	SUDV	34	22	1
1989	USA	RESTV	0	0	0
1990	USA	RESTV	4	0	0
1989–1990	Philippines	RESTV	3	0	0
1992	Italy	RESTV	0	0	0
1994	Gabon	EBOV	52	31	1

1994	Ivory Coast	TAFV	1	0	1
1995	Zaire	EBOV	315	250	1
1996	Gabon	EBOV	37	21	1
1996	South Africa	EBOV	2	1	1
1996	Philippines and USA	RESTV	0	0	0
1996–1997	Gabon	EBOV	60	45	1
2000–2001	Uganda	SUDV	425	224	1
2001–2002	Gabon and Congo	EBOV	122	96	1
2002–2003	Congo	EBOV	143	128	1
2003	Congo	EBOV	35	29	1
2004	Sudan	SUDV	17	7	1
2007	DR Congo	EBOV	264	187	1
2007–2008	Uganda	BDBV	149	37	1
2008	Philippines	RESTV	6	0	0
2008–2009	DR Congo	EBOV	32	14	1
2012	Uganda	SUDV	24	17	1
2012	DR Congo	BDBV	77	36	1
2013–2014	Guinea, Liberia, Sierra leone and Nigeria	EBOV	2,615	1,427	1

Source: Own Composition

Note: 0 = Other continental countries; 1 = African countries

Appendix III

Date	Country	Species	Ft	Ct	Dt	DUM
1976	Zaire	EBOV	88	318	280	0
1976	Sudan	SUDV	53	284	151	0
1979	Sudan	SUDV	65	34	22	0
1994	Gabon	EBOV	60	52	31	0
1995	Zaire	EBOV	79	315	250	0
1996	Gabon	EBOV	57	37	21	0
1996–1997	Gabon	EBOV	75	60	45	0
2000–2001	Uganda	SUDV	53	425	224	0
2001–2002	Gabon and Congo	EBOV	79	122	96	0
2002–2003	Congo	EBOV	90	143	128	0
2003	Congo	EBOV	83	35	29	0

2004	Sudan	SUDV	41	17	7	0
2007	DR Congo	EBOV	71	264	187	0
2007–2008	Uganda	BDBV	25	149	37	0
2008–2009	DR Congo	EBOV	45	32	14	0
2012	Uganda	SUDV	71	24	17	0
2012	DR Congo	BDBV	47	77	36	0
2013–2014	Guinea, Liberia, Sierra leone and Nigeria	EBOV	64	2,615	1,427	1

Source: Own Composition

Note: 0 = Other African countries; 1 = West African countries

Appendix IV

DATE	COUNTRY	SPECIES	CT	DT	DUM
2013–2014	SIERRA LEONE	EBOV	646	273	0
2013–2014	GUINEA	EBOV	485	385	0
2013–2014	LIBERIA	EBOV	468	255	0
2013–2014	NIGERIA	EBOV	4	1	1

Source: Own Composition

Note: 0 = Other West African countries; 1 = Nigeria

Regression Result

SUMMARY OUTPUT

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Regression Statistics	
Multiple R	0.961028
R Square	0.923574
Adjusted R Square	0.770723
Standard Error	132.3449
Observations	4

ANOVA

	df	*SS*	*MS*	*F*	*Significance F*
Regression	2	211663.6	105831.8	6.042293	0.276452
Residual	1	17515.17	17515.17		
Total	3	229178.8			

	Coefficients	*Standard Error*	*t Stat*	*P-value*	*Lower 95%*	*Upper 95%*	*Lower 99.0%*	*Upper 99.0%*
Intercept	662.0002	411.4925	1.608778	0.354052	-4566.51	5890.508	-25532.3	26856.27
Dt	-0.42388	1.328596	-0.31904	0.80339	-17.3053	16.45754	-84.998	84.15022
DUM	-657.576	431.0088	-1.52567	0.369365	-6134.06	4818.91	-28094.2	26779.04

RESIDUAL OUTPUT

Observation	*Predicted Ct*	*Residuals*	*Standard Residuals*
1	546.2815	99.71849	1.305056
2	498.8072	-13.8072	-0.1807
3	553.9113	-85.9113	-1.12436
4	4	1.14E-13	1.49E-15

PROBABILITY OUTPUT

Percentile	*Ct*
12.5	4
37.5	468
62.5	485
87.5	646

Source: Own Composition

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