ISSN No:-2456-2165

Understanding Covid-19 Pandemic using Ebola Virus Disease Death Transmission Power Spectral Density Model

A Dissertation Submitted to the School of Postgraduate Studies in Partial Fulfillment of the Requirement for the Degree of Masters in Statistics

[1]Ackmed Chebli

[2]Dr. Abdul Alimamy Kamara

Freetown Polytechnic

Fourah - Bay College

University of Sierra Leone

ISSN No:-2456-2165

DECLARATION

I confirm that this thesis presented for the degree of Masters in Statistics, has.

i) Been composed entirely by myself.
ii) Been solely the result of my own work.
iii) Not been submitted for any other degree or professional qualification

ISSN No:-2456-2165

CERTIFICATION

This is to certify that the thesis on Understanding Covid-19 Pandemic Using Ebola Virus Disease Death Transmission Power Spectral Density Model submitted by Ackmed Chebli, in Partial fulfillment of the requirements for the award of the Degree of Masters in Statistics is an original work carried out by him under our joint guidance. It is certified that the work has not been submitted anywhere else for the award of any other diploma or degree of this or any other University. We also certify that he complied with the Plagiarism Guidelines of the University.

Dr. Abdul Alimamy Kamara …….
……
Supervisor Date & Signature

DEDICATION

This work is dedicated to the Chebli family and all the people who helped me achieve this work.

ISSN No:-2456-2165

TABLE OF CONTENT

ISSN No:-2456-2165

ACKNOWLEDGEMENT

First of all, I will like to thank God the Almighty for giving me the strength and perseverance to complete my studies, secondly, I would like to the following persons who supported me and gave me the courage to complete this study.My Mother Marian Regina Babin and my daughters Naomi and Natasha Chebli who encouraged me to persevere.

Doctor Abdul Alimamy Kamaramy supervisor, for the guidance, support and motivation whenever I felt like giving up. I thank my God who supported me financially to complete my studies.My FriendMr. A. I. Turay who advised me to buy form to study at Fourah-Bay College University of Sierra Leone.

Thanks to all the respondents for participating in the research and taking time to respond to the questionnaire and to participate in the interviews; your time was sincerely appreciated.

MAY GOD BLESS YOU ALL.

ISSN No:-2456-2165

ABSTRACT

This study uses the Ebola virus disease (EVD) post-death transmission analytical power spectral density (PSD) prediction model to understand the dynamics of the COVID-19 pandemic. This is because the steady-state analysis and calculation of the Susceptible-Infected-Recovered-Dead (SIRD) analytical PSD model of the Ebola epidemic are equivalent to an asymptomatic infection (AI) SEIR model under the same conditions. This study aims to investigate the significant impact of the infection transmission routes, asymptomatic proportion, and latency period of the COVID-19 pandemic using an analytical PSD, AI-SEI model. The results show that the latent infections and asymptomatic proportion are challenging to identify using the infected equation (I(t)) to describe the disease dynamics. Despite the challenges, the observation shows the negative impact of asymptomatic infection transmission depends on the degree at which an infected individual becomes infectious (latency period).

Keywords: *COVID-19 Pandemic, Ebola Epidemic, Post-Death Transmission, Spectral Density.*

ISSN No:-2456-2165

CHAPTER ONE
INTRODUCTION

- *The Background of the Study:*

The 2019 coronavirus disease (COVID-19) is a severe respiratory viral infection in humans with a case fatality risk of 2.02% as of November 2021(Worldometer, 2021). COVID-19 is assumed to be a zoonotic disease, suspected of coming from bat species like the Ebola virus disease (EVD). In addition to the severe acute respiratory syndrome and Middle East Respiratory Syndrome, COVID-19 is the third coronavirus disease found to infect human populations(Perlman, 2020; C. Wang et al., 2020), and it has become a public health emergency of international concern and a pandemic(WHO (World Health Organization), 2020b). Unlike previous coronaviruses, up to the time of writing this paper, the outbreak has spread to 213 countries and territories and caused the highest numbers of infections and deaths (WHO (World Health Organization), 2020a). This problem has increased because the disease started in a densely populated area. A person with the virus and no symptoms of the disease (asymptomatic) can spread the virus(Anastassopoulou et al., 2020).

The spread of the COVID-19 pandemic requires an uninfected person to inhale air from an infected or asymptomatic person, either through breathing within 1.8 meters or direct contact with a respiratory droplet from coughing and sneezing(CDC, 2020a). The most noticeable symptoms are a high body temperature, coughing, difficulty breathing, a sore throat, a runny nose, and nausea (CDC, 2020b). Although there is a trial vaccine currently available to combat the spread of the virus, high percentages of those infected can recover from the illness globally (WHO (World Health Organization), 2020a).

Mathematical models are usually used to understand the dynamics of an infectious disease by separating the individuals in the population into compartments (Kermack & McKendrick, 1927). According to (Black & McKane, 2010b) and (Rozhnova & Nunes, 2010), contagious diseases such as COVID-19 have an incubation period before individuals who contracted the virus transmit it using the Susceptible-Exposed-Infectious-Recovered (SEIR) model. The susceptible is the population that has not yet been infected. The exposed are those infected but do not manifest symptoms. The infectious are the populations confirmed with the virus and those who overcome the illness.

In (Otunuga, 2017), the effects of stochastic fluctuations in the disease transmission rate were studied using latent (E) and infectious (I) infection transmissions, which are also applicable to the COVID-19 pandemic. The author discussed the stochastic fluctuation analysis using the Itó version in the sense of a Stratonovish stochastic SEI model. The author assumed the transmission rates would fluctuate around the mean value due to an external fluctuation. In doing so, the noise does not affect the visible infected states and the fluctuating terms are not adequate enough to show at which time the strength of the noise intensity is weak or strong.

Power spectral density (PSD) is a technique that has recently been used to show the strength of energy as a function of frequency. In other words, it shows at which frequencies the variations are weak. It has been used by several authors to understand the dynamics of an infectious disease (Black & McKane, 2010a, 2010b; Herrerías-Azcué & Galla, 2017a; Kamara et al., 2020a; Rozhnova & Nunes, 2009, 2010; Simões et al., 2008; R. H. Wang et al., 2012).

In (Kamara et al., 2020a), the authors proposed an analytical PSD model for the EVD epidemic using the SIRD model, where D is an infection compartment representing the population's individual deaths with the Ebola virus. Without intervention or recovered person reinfection, in terms of structures, the stochastic Fokker-Plank equation (SFPE) of the Ebola SIRD model (Kamara et al., 2020a) is equivalent to a SFPE asymptomatic infection SEIR (AL-SEIR) model. In other words, theoretically, the deterministic SID model of (Kamara et al., 2020a) and the AI-SEI model of (Otunuga, 2017) are equivalent under the same conditions. Therefore, the Ebola analytical post-death transmission model (Kamara et al., 2020a) can serve as a classical model to understand the dynamics of the current COVID-19 pandemic using PSD as a statistical tool to understand the dynamics of COVID-19.

In this research, we aim to study the significant impact of sensitive routes like infection transition, asymptomatic proportion, and latency period using an analytical PSD model, the AI-SEI model. The model was formulated by transforming the deterministic model in (Otunuga, 2017) to a Langevin equation (Black & McKane, 2010b; Rozhnova & Nunes, 2010). The asymptomatic infection periodicity effects around the persistence (endemic) state of the COVID-19 pandemic are studied. As there are more asymptomatic cases for the COVID-19 pandemic (WHO (World Health Organization), 2020c), there is a need to investigate the significant impact of asymptomatic infection.

- *Statements of the Problem:*

The 2019 coronavirus disease is a current health problem that was first discovered in Wuhan, China. Due to its asymptomatic mode of transmission, infection and mortality rates are very high globally. Because of this, there is a need to investigate the significant impact of the asymptomatic infection of the pandemic.

➢ *Aims:*

The main aim of this study is to show the significant impact of sensitive routes like infection transition, asymptomatic proportion, and latency period using an analytical PSD, AI-SEI model.

➢ *Objectives:*

The main objective of this work is to investigate whether, using an analytical AI-SEI-PSD model, the impact of

- The direct and indirect asymptomatic infections can be noticeable.
- The latency period can be noticeable.
- The population compartment of the model can explain the behavior of the disease.

➢ *Significant of the Research:*

The analysis of the SARS-covid2 epidemic is of paramount importance to understand the dynamics of the coronavirus spread. This can help health and government authorities take the appropriate measures and implement suitable policies aimed at fighting and preventing it.

➢ *Limitation of the Study:*

The COVID-19 pandemic has represented a challenge for humanity, which has sought to seek alternatives for the prevention, control, mitigation, and eradication of the disease. This has led to an 'explosion' of information from different parts of the world, which seeks to describe or propose alternatives for action in the face of the pandemic. Much of this information has a risk of bias or limitations of external validity, related to the availability of health technologies or the configuration of the health systems and services of the countries where the studies were carried out. As a limitation of this study, mobility, social distancing and self-quarantine have been applied. Moreover, health institutions advise people to practice good hygiene to keep from being infected. All these efforts have been made to reduce the transmission rate of the virus.

For the time being, COVID-19 infection is still on the rise. Government and research institutions scramble to seek antiviral treatment and vaccines to combat the disease. Several reports list possible drugs combination to apply, yet it is still unclear which drugs could combat the viral disease and which won't.

Several mathematical models have been proposed from various epidemiological groups. These models help governments as an early warning device about the size of the outbreak, how quickly it will spread, and how effective control measures may be. However, due to the limited emerging understanding of the new virus and its transmission mechanisms, the results are largely inconsistent across studies.

➢ *Definition of Terms:*

- CDC- Centre of disease control
- EVD- Ebola virus disease
- WHO- World health organization
- SEI- Susceptible exposed infection
- PSD- Power spectral density
- SFPE- Stochastic Fokker-plank equation
- SID- Susceptible infected disease
- VHF - Viral hemorrhagic fever

✓ *Pandemic* - Is an epidemic of an infectious disease that has spread across a large region.
✓ *Epidemic* - Is the rapid speed of disease to a large number of people in a given population within a short period of time.
✓ *Latent* - Is the time interval between when an individual or host is infected by a pathogen and when he/she becomes infector i.e. capable of transmitting pathogen to other susceptible individual.
✓ *Infectious Disease* - Infectious diseases are transmitted from person to person by direct or indirect contact. Certain types of viruses, bacteria, parasites, and fungi can all cause infectious disease. Malaria, measles, and respiratory illnesses are examples of infectious diseases.

➢ *Organization of the Study:*

This research is divided into five chapters starting with chapter one contains the following items: introduction, statement of the problem, Aims and Objectives, significant of the study, Limitation of the study, Definition of terms, organization of the study and Ethical consideration.

Chapter two present: Literature Review; overview, Description of the Epidemic model, Transmission for Covid-19, Covid-19 Pandemic Mathematical Model, Transmission of Ebola, Mathematical model of the Ebola Virus Disease and Power Spectral Density Modeling of Disease Transmission.

Chapter three; Research Methodology - Materials and methods, Model specification, the asymptomatic infection PSD-SEI Model.

Chapter four: Analysis and Outcomes; Analysis of the AI-SEI Numerically Simulation.

Chapter five: Findings thought and recommendation.

ISSN No:-2456-2165

CHAPTER TWO
LITERATURE REVIEW

- *Overview:*

This section presents different studies about COVID-19 and Ebola virus diseases. A mathematical formulation is a powerful method for developing and testing dynamic, complex multi-parameter models, such as those for the spread of a pandemic. A pandemic is an epidemic that develops in many countries or continents, whereas an epidemic disease influences enormous numbers of people within a community, population, or region.

However, statistical modeling helps in prediction, while epidemiology provides a method to understand why and how infections spread and how they might be prevented or restricted. For instance, when a new infectious disease emerges or there is an outbreak of a known infectious disease, epidemiologists are the scientists who collect, analyze, and interpret information to indicate interventions for halting further dissemination. Many infectious diseases do not respect national boundaries, color, creed, caste, communities, etc., initially affecting only one region of the world, rapidly disseminating to other regions and ultimately becoming pandemics like COVID-19. These diseases may have several types based on their nature of spread.

When an epidemic is spreading to a larger extent than the expected number of cases, it is called an outbreak. It may be one case in a new locality. If it is not registered soon, there may be an outbreak causing the epidemic.

Outbreaks of viral hemorrhagic fevers (VHFs) like Ebola and Lassa fever and other respiratory viruses, such as influenza or COVID-19, typically attract the attention of the media and politicians due to the potentially high rate of infectivity and mortality (Dicker, 2006). However, the statistical and empirical analysis in the discipline of epidemiology allows us to take information from individuals and aggregate it into logical groups (defined by the characteristics of the person, the environment, or time points) (Dicker, 2006). It may help us to logically understand where the infection has originated, how it might be disseminating, and thus the potential means for prevention and its containment in the restricted zones. Several mathematical models for epidemiological predictions of COVID-19 have been made. Most of them are based on the classical model of SIR (Susceptible–Infected–Recovered), initially described by (Kermack & McKendrick, 1927).

However, because this pandemic is unprecedented with multiple influencing factors, the mathematical models developed are not completely accurate and still need validation (Anirudh, 2020).In a comparative study (Anirudh, 2020), they evaluated the accuracy of several mathematical models used for the COVID-19 pandemic prediction. The majority of them are based on the SIR and its derivations, such as the Susceptible–Exposed–Infectious–Recovered (SEIR), Susceptible-Exposed-Infectious-Recovered-Undetectable (Liu et al., 2020), Simple Susceptible Infected-Recovered-Deaths (Fanelli & Piazza, 2020) to name few.

- *Description of the Epidemic Model:*

In 1927, Kermack and McKendrick introduced the idea of having a general procedure to analyze the spread of contagious epidemics (Kermack & McKendrick, 1927). This general procedure is known today as the "SIR model." The process of disease modelling begins with an interaction between an infected individual and a group of individuals who are at risk of being infected (susceptible). Several susceptible people will then become infected, thus joining the infected group. Infected people then either die or recover from the disease, and in both cases, they are removed from the infected compartment. When there is an epidemic, the number of susceptible populations decreases while the removed population increases without intervention(Dietz, 1982).

In constructing epidemic models, during an early phase of the model, demographic effects are ignored due to the fact that demographic time scales are ordinarily much longer than the disease timeframe. Then again, since endemics may endure for a longer time, it becomes important to include demographic effects in endemic models (Dietz, 1982).

The SEIR model is a modified SIR model that describes the incubation period between the infected period (entry of pathogen) and the stage of infectiousness (shedding of pathogen). It is very important to model the incubation period (E) explicitly since the individual during the incubation period has been exposed to a pathogen that may be active or dormant. It has been used in the study of epidemiological patterns and disease control since it was proposed by (Cooke & Van Den Driessche, 1996; Wei & Xue, 2020).

The SEIR model, like the SIR model, describes the progression of an epidemic by considering the total population to be equally likely to get infected. Here, immunity after infection is assumed, which means individuals do not return to the susceptible compartment after recovery (Kermack & McKendrick, 1927). These outbreaks recur until the majority of the populace develops immunity.

The formulation of the SEIR model was one of the first breakthroughs in mathematical epidemiology. It has aided in the investigation of the transmission dynamics of infections such as cerebrospinal meningitis, malaria, and hepatitis A and B (Side et al., 2017; Turner et al., 2015).

In (Weinstein et al., 2020) the authors studied the global dynamics of the SEIR model with varying population sizes and they also included latent and infection fraction of the population. In their study of the SEIR model they found the analytical solution to the model by recasting the SEIR model as a single second order nonlinear ordinary differential equation. Also (Upadhyay et al., 2019) proposed and studied the SEIR model for controlling highly contagious diseases. They recommended the use of the model in related studies. In their investigation to identify rational intervention strategies to control the dissemination of COVID-19 in India, (Mandal et al., 2020) employed the SEIR. More recently, (Mbogo & Odhiambo, 2021) studied the impact of social distancing and mass-testing in Kenya using a modified SEIR epidemic model. Furthermore, (Kamara et al., 2021) also used the SEIR model with latent infection to understand the dynamics of the COVID-9 pandemic.

- *Transmissions for COVID-19:*

Covid-19 is associated with high morbidity and mortality levels and neither has a known cure nor a vaccine. The disease is a respiratory infection that can be transmitted through droplets of different sizes. When the droplet particles are greater than five to ten micrometers (m) in diameter, they are referred to as respiratory droplets, and when they are less than five micrometers (5m) in diameter, they are referred to as droplet nuclei (Harko et al., 2014). According to current evidence, the COVID-19 virus is primarily transmitted between people through respiratory droplets and contact routes(Burke et al., 2020; Chamie et al., 2021; Chan et al., 2020; Huang et al., 2020; Q. Li et al., 2020). Droplet transmission occurs when a person is in close contact (within 1 m) with someone who has respiratory symptoms (e.g., coughing or sneezing) and is therefore at risk of having his/her mucosae (mouth and nose) or conjunctiva (eyes) exposed to potentially infective respiratory droplets. Transmission may also occur through fomites in the infected person's immediate environment (WHO, 2020).Therefore, transmission of the COVID-19 virus can occur by direct contact with an infected person and indirect contact with surfaces in the immediate environment or with objects used on the infected person (e.g., stethoscope or thermometer).

Airborne transmission is different from droplet transmission as it refers to the presence of microbes within droplet nuclei, which are generally considered to be particles of 5 m in diameter and can remain in the air for long periods of time and be transmitted to others over distances greater than 1 m. In an analysis of 75,465 COVID-19 cases in China, airborne transmission was not reported (Burke et al., 2020)

- *COVID-19 Pandemic Mathematical Model:*

COVID-19 Mathematical modeling is confidentially working to comprehend and predict how the infections spread. The use of mathematical models is to create a simplified depiction of infection spread in a population and to understand how an infection might increase in the future. These forecasts could help us make better use of public health resources such as vaccination programs, treatments, prevention, and interventions. The development of computational models used to simulate dynamical equations for coronavirus disease. Clinicians and administrators are accepting the conclusions drawn from modeling, often without realizing the data is simulated.

(Oke et al., 2021) co-operatively developed a mathematical model for understanding the transmission dynamics and control of COVID-19 in Nigeria, one of the main epicenters of COVID-19 in Africa. Rigorous analysis of the (Kermack & McKendrick, 1933) type compartmental epidemic model we developed, which takes the form of a deterministic system of nonlinear differential equations, it was revealed that the model has a continuum of disease-free equilibriums which is locally asymptotically stable whenever a certain epidemiological threshold, called the control reproduction, is less than unity. The epidemiological implication of this result is that the pandemic can be effectively controlled (or even eliminated) in Nigeria if the control strategies implemented can bring (and maintain) the epidemiological threshold to a value less than unity.

(Ahinkorah et al., 2020) Propose a compartmental mathematical model to predict and control the transmission dynamics of the COVID-19 pandemic in India with epidemic data up to April 30, 2020. They computed a basic reproduction number, which was used to further study the model simulations and predictions. They perform local and global stability analyses for the infection-free equilibrium point as well as an endemic equilibrium point with respect to the basic reproduction number. Moreover, this research shows the criteria of disease persistence. They further conducted a sensitivity analysis to determine the relative importance of model parameters to disease transmission. Their model simulation demonstrates that the disease transmission rate is more effective at mitigating the basic reproduction number. Based on estimated data, their model predicts that in about 60 days, the peak will be higher for COVID-19 in India, and after that, the curve will plateau, but the coronavirus diseases will persist for a long time.

(Kucharski et al., 2020) acknowledge many effects on humans that are ignored in deterministic models for COVID-19. In their paper, they formulated a stochastic susceptible–infected–recovered model and devoted full strength to the sufficient conditions for extinction and persistence. They also examine the threshold of the proposed stochastic COVID-19 model when the noise is small or large.

In (Hethcote, 2005), a mathematical model which explains the transmission dynamics of the COVID-19 pandemic in Bangkok, Thailand was discussed. The result shows that asymptomatically stable if the reproductive number is greater than one.

The mathematical model analysis of the model is supported by numerical simulations, and the results prove that the consistent use of face masks would go a long way towards reducing the COVID-19 pandemic.

(Liu et al., 2020) explain the outbreak of coronavirus disease 2019 (covid-19) with the help of a mathematical model using both ordinary differential equations (ODE) and fractional differential. This research uses the data of COVID-19 cases in Nigeria for the numerical simulation that has been fitted to the model. They brought in the consideration of both asymptomatic and symptomatic infected individuals with the fact that an exposed individual is either sent to quarantine first or moved to one of the infected classes, with the possibility that a susceptible individual can also move to a quarantined class directly. The proposed model was discovered to have two equilibrium points. One of the most important goals of the mathematical models was to predict disease epidemiology in advance and thus assist governmental authorities in developing public health policies to avoid or reduce overcrowding of health facilities during epidemics. The model showed a good relationship between the predicted number of new cases and the total number of inpatients and deaths associated with COVID-19 over the period of the country's five regions, highlighting the potential relevance of the model in the combat of the COVID-19 pandemic. Another important role of the mathematical models is to predict the duration and end of epidemics. They performed a preliminary analysis of the relationship between the sensitive parameters of the model and the period where the numbers of new cases start to decrease by 20% or more in the last 14 days.

(Fanelli & Piazza, 2020) propose a mathematical model to predict the evolution of COVID-19 and evaluate the impact of governmental decisions on this evolution, thus attempting to explain the pandemic's long duration among the twenty-six Brazilian states and capitals, as well as in the Federative Unit. The prediction was made based on the growth rate of new cases in a stable period, and the graphics were plotted with the significant governmental decisions to evaluate the impact on the epidemic curve in each Brazilian state and city.

(Z. Hu et al., 2020) reveal a control strategy preventing subclinical transmission that differed among countries. A stochastic transmission model was used to assess the potential effectiveness of control strategies in controlling the COVID-19 outbreak. Three strategies include a lack of prevention of subclinical transmission (Strategy A), partial prevention using testing with varying degrees of accuracy (Strategy B), and complete prevention by isolating all at-risk people (Strategy C, Taiwan policy). The high probability of containing COVID-19 in Strategy C is observed in different scenarios and has varied in the number of initial cases (5, 20, and 40), the reproduction number (1.5, 2, 2.5, and 3.5), the proportion of at-risk people being investigated (40%, 60%, 80%, to 90%), the delay from symptom onset to isolation (long and short), and the proportion of transmission that occurred before symptom onset (1%, 15%, and 30%). Strategy C achieved a probability of 80% under advantageous scenarios, such as low initial cases and high coverage of epidemiological investigation, but Strategies B and C rarely achieved that of 60%. Considering the unsatisfactory accuracy of current testing and insufficient resources, isolation of all at-risk people, as adopted in Taiwan, could be an effective alternative.

One of the early studies was conducted by (M. Y. Li et al., 1999) to unveil the causes of the rapid dissemination of the coronavirus disease. Their analysis was done by observing the reported data from December 2019 to January 2020 in Wuhan using a network dynamic meta population model. They estimated that about 86% of all infections are undocumented, with a 95% confidence interval of 82-90% (M. Y. Li et al., 1999). Thus, there were some undetected individuals present in the population who had been exposed to the virus and were facilitating its spread.(Sabbih et al., 2021) proposed the SEIS model to study the disease transmission dynamics, suspecting that individuals who had recovered from COVID-19 were still susceptible to reinfection. They also proposed a viral replication model to help comprehend virus-host cell interactions (Turner et al., 2015). (Yang & Wang, 2020) proposed the SEIR model in their study of the COVID-19 outbreak in Wuhan, the disease's epicenter at the time, based on current clinical diagnosis of reinfection cases. Through numerical simulation and mathematical analysis, they predicted that the coronavirus disease would persist and become epidemic.

In their investigation to identify rational intervention strategies to control the dissemination of COVID-19 in India, (Mandal et al., 2020) employed the SEIR. Their analysis was limited to quarantine and airport screening as intervention programs. They recommended more intervention programs to aid in the mitigation response needed.

More recently,(Mbogo & Odhiambo, 2021) studied the impact of social distancing and mass-testing in Kenya. Their analysis was conducted using the SEIHQRD model, a modified SEIR epidemic model. Their findings also suggested that a more dedicated effort from individuals and the government was required to decrease the infection rate in Kenya (Mbogo & Odhiambo, 2021). (kamara et al., 2021) also used the SEIR model with latent infection to understand the dynamics of the COVID-9 pandemic. They showed that the impact of the direct and latent infections of the COVID-19 pandemic is detrimental to the susceptible population. Though several studies have been done to understand the dynamics of the current pandemic, research on the total impact of awareness programs created is scarce.

(Kamara et al., 2021) also used the SEIR model with latent infection to understand the dynamics of the COVID-9 pandemic. They showed that the impact of the direct and latent infections of the COVID-19 pandemic is detrimental to the susceptible population.

- *Transmission of Ebola:*

Ebola virus disease (EVD) is one of the deadliest diseases in Africa. It was first discovered in 1976 in the Democratic Republic of Congo (DRC), previously known as Zaire, and has a potential fatality rate of up to 90%(Chowell et al., 2004; Ndanguza et al., 2013). While the virus has been found in other parts of the world, the majority of cases and outbreaks are African(Centers Disease Control and Prevention(CDC), 2018). Since 1976, African countries like Sudan, Gabon, Uganda, and the DRC have experienced more than three outbreaks each (Legrand et al., 2007).

There are six strains of the virus: Zaire, Sudan, and Tai Forest; Bundibugyo, Reston, and Bombali Ebola viruses. Of these, only the first four are known to cause disease in humans (Chowell et al., 2004; WHO, 2018a). The Reston virus can cause disease in nonhuman primates and the Bombali Ebola virus, which was discovered in Sierra Leone in 2018, has not yet been confirmed as causing disease in either animals or humans (Chowell et al., 2004).

However, post-death transmission plays an active role in infection transmission, causing susceptible populations to become infected either by a living host or by a host that died with the virus. (Weitz & Dushoff, 2015; WHO, 2018b). Almost all initial cases of EVD have resulted from spillover events. (2009), when an uninfected person meets a dead infected animal, such as a fruit bat or nonhuman primate (Herrerías-Azcué & Galla, 2017b; Judson et al., 2016; Rao et al., 2012) The transmission process then continued with human-to-human contact. The persistence of the virus in a community has been decisively linked by scholars to funeral practices(McKane & Newman, 2005; Van Kampen, 2007). Direct contact with the blood or bodily fluid of an infected individual showing signs and symptoms of the virus (e.g., vomit, high fever, diarrhea, generalized pain) can also cause transmission. Since the virus can survive outside of a living host for several days, it has the potential to spread through contaminated objects (Gillespie, 1976; Van Den Driessche & Watmough, 2002; Yamazaki, 2018). There is no confirmed source indicating where the virus originated, but various studies have proven that the African fruit bat is a known animal reservoir for the virus (Bermejo et al., 2006; Leroy et al., 2005). Most recently, there has been a vaccine for the Ebola virus. There were several research studies that were based on the Ebola vaccination.

- *The Mathematical Model of the Ebola Virus Disease:*

(Do & Lee, 2016; Fasina et al., 2014; K. Hu et al., 2015; Kiskowski et al., 2016; Lekone et al., 2006; Meakin et al., 2018; Nieddu et al., 2017; Salem & Smith, 2016; X.-S. Wang & Zhong, 2015; Weitz & Dushoff, 2015) collaborated on the Susceptible Exposed-Infectious-Recovered-Deceased (SEIRD) type model, which has been used to study the dynamics of EVD outbreaks with the Although an analytical application can give a better understanding of an epidemic model using a nonlinear differential equation, (Black & McKane, 2010a) further explain that numerical simulations are the most common computer application considered to study the dynamics of the disease with these models.(Meakin et al., 2018) used a numerical simulation on a simple stochastic meta population model to study the dynamics of the 2018 DRC Ebola outbreak with time-series data for three health care centers in Equateur province. (Do & Lee, 2016) provided analytical and numerical analyses of EVD dynamics without vital dynamics (that is, the birth and death processes are neglected) using the SLIRD model (Lis latently individual) to identify sensitive transmission areas in Nigeria during the 2014–2016 West Africa outbreak. They reported that safe burial was a targeted area that required effective intervention strategies to combat the spread of the disease. Their findings were reinforced by(Salem & Smith, 2016), who used the SEIRD model and provided a sensitivity analysis with a Partial Ranking Correlation Coefficient (PRCC) and Latin hypercube sampling. The major difference between these two studies is that, in (Levin-Sparenberg et al., 2015), transmission by latently infected deceased individuals was included in the model, whereas for Salem and Smith (2016), the model was deterministically analyzed with vital dynamics. Also,(Agusto et al., 2015) used PRCC to assess the impact of population-level on the basis of non-pharmaceutical control measures during the 2014 EVD outbreak in West Africa by incorporating the effects of traditional belief systems and customs, along with disease transmission within health-care settings and by Ebola-deceased individuals. The authors reported that in the absence of anti-Ebola public health interventions, traditional/cultural/custom belief systems through which the interaction between an uninfected individual and Ebola-deceased individuals is very high. It is one parameter that has the most influence on the Ebola transmission dynamics in Guinea.

(Weitz & Dushoff, 2015) reported that there was an identifiability problem when the SEIR was modelled with post-death transmission using epidemic growth rate data with the infective equation as a response function. This was accomplished by comparing the growth rates of the SEIR and SEIRD models using numerical simulation. It was shown that, for the latter, different transmission parameter values produced almost the same pattern of epidemic curves, indicating challenges in understanding the dynamics of the disease, as epidemiology parameters for EVD are difficult to identify.

(M.S.M. et al., 2012) shows that traditional funerals and burial costumes played a significant role in the spread of EVD during the 2000 Uganda and 1995 DRC Ebola epidemics. Augusto et al. combined traditional belief systems and customs with transmission within healthcare settings and transmission by Ebola human-deceased individuals to assess the effect of population-

level based on non-pharmaceutical preventive measures during the West African EVD outbreak. The authors showed that, besides the lack of anti-Ebola public health interventions, cultural (traditional or custom) beliefs were one of the parameters that showed the most influence on the transmission of Ebola in Guinea. Also, in (Rao et al., 2012), the authors incorporated disease relapses and reinfection to analyze the Ebola disease dynamics, showing that the model is locally asymptotically stable. They showed that the total number of new cases increases when both relapses and reinfection parameters increase.

(Altaf & Atangana, 2019) used the Caputo fractional derivative and showed how accurately the method could predict the Western African Ebola outbreak data. Also,(Nieddu et al., 2017)used the Hamiltonian method to describe the EVD dynamic with the hospital, community, and funeral transmission models, taking into account the transition from the Ebola virus reservoir (fruit bat) transmission in the model. The authors showed how reservoir transmission, post-death transmission, and stochastic population dynamics impacted the persistence and recurrence of the EVD in the human population.

However,(Berge et al., 2017) introduced a deterministic SIR-DP model and showed that the complete model has one endemic state and is locally asymptotically stable. They also showed that for the SIR-DP model to be globally stable at its endemicity levels, the shedding parameters of the model must be zero. (Yamazaki, 2018) included a diffusive term in the S, I, R, and P compartments of the Ebola partial differential equation model. This showed that the Ebola partial differential equation model is not well-posed and the stability analyses do not hold. Differential operators that possess crossover properties and fade out (Caputo, Caputo-Fabrizio, and Atangana-Baleanu) were used by Altaf and Atangana to inspect the dynamics of EVD in Africa using the SIR-DP model. Also, an extension was made to the SIR-DP model in Berge et al. (2018) by including the Ebola reservoir dynamics process and providing theoretical and numerical analyses to assess how the Ebola reservoir impacts the disease dynamics. Moreover, in (Kamara et al., 2020a), the SEIR was modelled with environmental transmission and also showed negative implications for the transmission from a contaminated environment.

- *Power Spectral Density Modeling of Disease Transmission:*

To understand the interplay between the deterministic and stochastic models and also to give a better description of the dynamics of an epidemic, an analytic power spectral density (PSD) has been considered; (Heida et al., 2013), purposed a parametric analytical definition for Power Spectral Density functions (PSD) coherent with a wide range of response spectra defined by several international seismic codes with different shapes. The reliability of such a powerful tool is then assessed through an extensive numerical campaign by comparing stochastic analysis and Monte-Carlo simulations.

(Heida et al., 2013) explain that the signals recorded from the extremities of Parkinson's disease patients showing rest and/or action tremor reveal a distinct high power resonance peak in the frequency band corresponding to tremor. They further said Parkinson's disease patients continuously balance between tremor and tremor suppression or compensation expressed by power shifts between the low frequency band and the tremor frequency band during rest and voluntary motor actions. This balance shows that the pathological trmor is either on or off, with the latter state not resembling that of a healthy subject. Deep brain stimulation can increase the spectral density of physiological, rest, and action tremors in Parkinson's disease patients treated with deep brain stimulation.

(Melek Manshouri, 2022) proposed that sound signals from the respiratory system be used as a proxy for human health. It was suggested that early diagnosis is of great importance because, if delayed, it exerts an irreversible effect on human health. The coronavirus pandemic, which is deeply shaking the world, has revealed the importance of this diagnosis even more. During the pandemic, it has become the focus of researchers to differentiate symptoms from similar diseases such as influenza. Among these symptoms, the difference in cough sound played a distinctive role in the research. Also, Clinical data collected under the supervision of doctors in a reliable environment was used as the dataset, consisting of 16 subjects suspected of COVID-19 with a specific patient demographic to undergo this research.

(Alonso et al., 2007) presented an analytic PSD model to investigate stochastic amplification in an epidemic model, considering internal and external transmission. It was shown that demographic noise could shift the damped oscillation of the deterministic period, and the oscillating infective curve of the PSD expression moved to a higher frequency as the transmission value increased. Moreover, a stochastic SIR model with seasonal forcing was also used with analytic PSD techniques in (Simões et al., 2008) to separate the connection between external forcing and stochasticity in an epidemic model. Also, to investigate the performance of the resonant fluctuation of the stochastic SIR model in (Simões et al., 2008), an analytical PSD solution was presented by relaxing the random mixing assumption and including a mixing network by(Simões et al., 2008). In (R. H. Wang et al., 2012), the environmental compartments are added to the SIR Model to show the impact of environmental transmission in an influenza outbreak. Also in (Kamara et al., 2020b), the death compartments were added to the SIR Model to show the impact of post-death transmission in an Ebola outbreak.

ISSN No:-2456-2165

CHAPTER THREE
RESEARCH METHODOLOGY

➢ *Materials and Methods:*

The aim of this study is to investigate the propagation of the COVID-19 pandemic using the (Kamara et al., 2020b) analytical post-death transmission for the Ebola model as a classical model. Indeed, the degree of disease propagation within a community (a country or a city) highly depends on parameters such as the infection rate and the latency period. Each parameter contributes to the evolution of the pandemic with a certain probability. The principal parameters affecting the spread rate are the focus of this research work. Hence, the aim of this work is to identify those parameters allowing the slow-down of COVID-19 propagation in order to maintain control of the disease dynamics.

➢ *Model Specification:*

- *The Asymptomatic Infection Model for SEI:*

In this section, we formulate the AI-SEI model as in (Otunuga, 2017), but assume birth and natural death are equal and disease-induced death only occurs in the infectious state. The model is used under the following assumptions:

✓ The population is constant but large.
✓ The only way a person can leave the susceptible state (*S*) is to become infected, either from the exposed (*E*) or visibly infected (*I*) state or die of natural causes.
✓ The only way a person can leave the *E* state is to show signs and symptoms of the illness or die of natural death.
✓ The only way a person can leave the state is to recover from the disease, die from natural death, or die as a result of the disease.
✓ A person who recovered (*R*) from the illness received permanent immunity.
✓ Age, sex, social status, and race do not affect the probability of being infected.
✓ Each member of the population has the same contact with one another equally.
✓ All births are in the susceptible state, and it is assumed that the birth and natural death rates are equal.
✓ In view of this, the rates of transition in the latent and infected periods are denoted as β_e and, $\beta_i$respectively. The susceptible population is increasing and decreasing at an equal rate, which is the rate when an exposed individual moves to an infectious state. The disease-induced death rates are referred to as the "recovery rate constant." Figure 1 represents the latent infection *SEIR* mode.

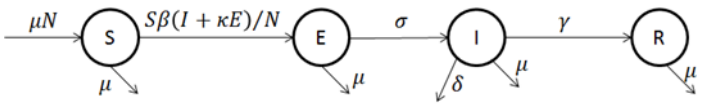

Fig 1 The Latent Infection SEIR Model Flow Diagram.

The total population is $= S + E + I$, and the modification of the AI-SEI model in (Otunuga, 2017) to a fixed population size model can give us a deterministic nonlinear differential equation which in proportional form is given as:

$$A_1 = \mu - \beta_e se - \beta_i si - \mu s,$$

$$A_2 = \beta_e se + \beta_i si - (\mu + \gamma)e$$

$$A_3 = \gamma e - (\mu + \alpha + \delta)i$$

Where $\beta_e = \kappa\beta$and$(s, e, i) = (1,2,3), A_1 = ds/dt\,, A_2 = de/dt,$ and$A_3 = di/dt\,(l = 1,2,3);$ and$s = S/N\,, e = E/N\,, i = I/N$, which is equivalent to the mean-field equation of (Otunuga, 2017). The dynamics of the outbreak is numerically investigated with this model in equation (1) using the Gillespie Stochastic Simulation Algorithm. The endemic state of the outbreak is of interest for this work, and it is the state when the outbreak continues to persist in the population as time increases. It is believed that the endemic equilibrium state occurs when $R_0 > 1$.

➢ *The Asymptomatic Infection PSD-SEI Model:*

Since in (kamara et al., 2021) it was showed that at the endemic equilibrium states the AI-SEI model has negative real part for all it eigenvalues we then considered a set of Langevin equation defines as:

$$\frac{dy_k}{dt} = \sum_{l=1}^{4} A_{kl}\, y_l + \zeta_k(t), \qquad (k, l = 1,2,3) \qquad (2)$$

Where$\zeta_k$represent the Gaussian white noise with zero mean andf_{kl} is the Jacobian matrix of the microscopic equations with respect to the endemic state$A_k(s^*, e^*, i^*)$. The cross-correlation structure is determined by the expansion.

$$\langle \zeta_k(t)\zeta_k(t')\rangle = \rho(t - t')B_{kl}$$

Where$B_{kl}$the noise covariance matrix is also evaluated at the endemic equilibrium states. Hence, following (Kamara et al., 2020a) the noise covariance matrix for the AI-SEI model of equation (1) is gives as

$$B_{11} = \mu + \beta_e se + \beta_i si + \mu s,$$

$$B_{22} = \beta_e se + \beta_i si + (\mu + \gamma)e$$

$$B_{33} = \gamma e + (\mu + \alpha + \delta)i$$

$$B_{12} = B_{21} = -s(e\beta_e + i\beta_i) \qquad (3)$$

$$B_{23} = B_{32} = -\gamma e$$

The Langevin equation of equation (2) is then transform to a linear system using Fourier series transformation since we are interested in cycles. In doing so we derive the power spectral density (PSD) that is correspond to the normalized fluctuation, independent of the community size N. From the Langevin equation above, taking temporary Fourier transform we get:

$$-i\omega\tilde{y}_k(\omega) = \sum_{l=1}^{4} A_{kl}\tilde{y}_l(\omega) + \bar{\zeta}_k(\omega) \qquad (4)$$

Whereω is the angular frequency, $\tilde{y}_l(\omega), (l = 1,2,3)$ are the stochastic corrections to the deterministic variables s, e and i. Also

$$\langle \bar{\zeta}_k(\omega)\bar{\zeta}_l(\omega')\rangle = B_{kl}(2\pi)\rho(\omega + \omega')$$

This leads us to three linear algebraic equations represented in matrix form as:

$$\begin{pmatrix} -i\omega - A_{11} & -A_{12} & -A_{13} \\ -A_{21} & -i\omega - A_{22} & -A_{23} \\ 0 & -A_{32} & -i\omega - A_{33} \end{pmatrix}\begin{pmatrix} \tilde{y}_1 \\ \tilde{y}_2 \\ \tilde{y}_3 \end{pmatrix} = \begin{pmatrix} \bar{\zeta}_1 \\ \bar{\zeta}_2 \\ \bar{\zeta}_3 \end{pmatrix}$$

However, it is easy to see that the Jacobean matrix (drift term ($A_{kl}\, matrix$) of the Langevin equation) and fluctuating matrix ($B_{kl}\, matrix$) structures in this work are the same as that of (Kamara et al., 2020a) . That is, Jacobean matrixes for drift term are

$$A_{kl} = \begin{pmatrix} Kamara\ et\ al.(2020) model \\ S & I & D \\ A_{11} & A_{12} & A_{13} \\ A_{21} & A_{22} & A_{23} \\ 0 & A_{32} & A_{33} \end{pmatrix} and\ A_{kl} = \begin{pmatrix} Propose\ model \\ S & E & I \\ A_{11} & A_{12} & A_{13} \\ A_{21} & A_{22} & A_{23} \\ 0 & A_{32} & A_{33} \end{pmatrix},$$

Where

$$A_{11} = \partial A_1/\partial s,$$

$$A_{12} = \partial A_1/\partial e,$$

$$A_{13} = \partial A_1/\partial i$$

$$A_{21} = \partial A_2/\partial s,$$

$$A_{22} = \partial A_2/\partial e,$$

$$A_{23} = \partial A_2/\partial i$$

$$A_{32} = \partial A_3/\partial e,$$

$$A_{33} = \partial A_3/\partial i$$

Also, the fluctuating matrixes are demonstrated as

$$B_{kl} = \begin{pmatrix} Kamara\ et\ al.(2020) model \\ S & I & D \\ B_{11} & B_{12} & 0 \\ B_{21} & B_{22} & B_{23} \\ 0 & B_{32} & B_{33} \end{pmatrix} and\ B_{kl} = \begin{pmatrix} Propose\ model \\ S & E & I \\ B_{11} & B_{12} & 0 \\ B_{21} & B_{22} & B_{23} \\ 0 & B_{32} & B_{33} \end{pmatrix}$$

Moreover, the focus is to use the infectious equation (I(t)) to describe the trajectories of epidemic curve which in this paper is the PSD death ($P_D(\omega)$) expression in (Kamara et al. 2020) as we can see from the matrixes. That is, the I(t) equation we are using to study the COVID-19 dynamic is not the same as the Ebola post-death transmission SIRD model in (Kamara et al., 2020a).

The analysis is done using the endemic equilibrium points ($E^* = (s^*, e^*, i^*)$) where the A_{kl} and B_{kl} parameters are defined using

$$s^* = \frac{1}{R_0},$$

$$e^* = \frac{\mu(R_0 - 1)}{(\mu + \gamma)R_0},$$

$$i^* = \frac{\mu\gamma(R_0 - 1)}{(\mu + \gamma)(\mu + \alpha + k)R_0} \qquad (5)$$

And

$$R_0 = \frac{1}{(\mu + \gamma)}\left(\beta_e + \frac{\gamma\beta_i}{(\mu + \alpha + k)}\right), \quad (6)$$

$R_0$is the basic reproduction number. It is well known that when $R_0 > 1$, the disease is in its endemic equilibrium state. Hence from (Kamara et al., 2020a) the real part of the $P_I(\omega)$ prediction model for our AI-SEI model is given as

$$P_I(\omega) = \frac{(\alpha_I - \mathrm{R}_I\omega^2 + B_{kk}\omega^4)}{\mathcal{D}(\omega)}, \qquad (5)$$

Where$k = 1,2,3$ and

$$\alpha_I = B_{11}\varepsilon p^2 + B_{22}\varepsilon q^2 + B_{33}\varepsilon r^2 - 2B_{12}\varepsilon_P\varepsilon_q - 2B_{23}\varepsilon_q\varepsilon_r$$

$$R_I = 2B_{23}A_{32}(A_{11} + A_{22}) + 2\left(B_{33}\varepsilon_r - B_{23}\varepsilon_q\right) - (B_{22}A^2{}_{32} + B_{33}(A_{11} + A_{22})^2),$$

$$\varepsilon_P = A_{21}A_{32};$$

$$\varepsilon_q = A_{11}A_{32}\,;$$

$$\varepsilon_r = A_{11}A_{22} - A_{12}A_{21}$$

$$D(\omega) = \omega^6 + (2L_2 - L_1^2)\omega^4 - (2L_1L_3 + L_2^2)\omega^2 - L_3^2$$

$$L_1 = A_{11} + A_{22} + A_{33},$$

$$L_2 = A_{11}(A_{22} + A_{44}) + A_{22}A_{44} - A_{24}A_{42} - A_{12}A_{21}$$

$$L_3 = A_{11}(A_{23}A_{32} - A_{22}A_{33}) + A_{21}(A_{12}A_{33} - A_{13}A_{32})$$

$$A_{11} = -\mu - e^*\beta_e - i^*\beta_i,$$

$$A_{12} = -s^*\beta_e ,$$

$$A_{13} = -s^*\beta_i ,$$

$$A_{33} = -(\mu + \alpha + k),$$

$$A_{21} = e^*\beta_e + i^*\beta_i,$$

$$A_{22} = s^*\beta_e - (\mu + \gamma),$$

$$A_{23} = s^*\beta_i ,$$

$$A_{32} = \gamma.$$

The parameter values are taken from the global COVID-19 pandemic literature as of 10 June 2020. We estimated the case fatality rate as the ratio of total confirmed cases(World Health Organization 2020, 2020)

$$\alpha = \frac{408025}{7145539} = 0.057.$$

The incubation period has a mean average of 5.2 days, and the recovery period is 5.8days (Li et al.2020), that is.

$$\gamma = \frac{1}{5.2} = 0.192,$$

$$k = \frac{1}{5.8} = 0.172$$

The natural death rate is assumed to be.

$$\mu = 0.00005,$$

The direct transmission as in (Lin et al., 2020)

$$\beta_i = 0.533$$

And $$\beta_e = \sigma\beta_i = 0.267,$$

$where\ \sigma = 0.5$is the proportion of asymptomatic infection rate of human (Chan et al., 2020), hence

$$R_o = 3.32$$

ISSN No:-2456-2165

CHAPTER FOUR
RESULT AND DISCUSSION

➢ *Analysis of the AI-SEI Numerically Stochastic Simulation:*

In this section, we illustrate numerically the stochastic simulation for equation (1) using the Gillespie Stochastic Simulation Algorithm to investigate the patterns of the susceptible, exposed, and visible infectious population. The model parameter values are used as obtained in the previous chapter, and we focus our analysis on a small settlement of approximately 1,000 people.

We investigate the endemic equilibrium, showing in Figure 2 that, as ***E*** and ***I*** increase, the ***S*** population declines. We also noticed that at a certain point in time, there is a decrease in the trajectories for the ***E*** and ***I*** states. The decrease trajectories for the E state are due to an increase in asymptomatic individuals to the visibly infectious state and natural death, whereas the decrease trajectories for the I state are due to an increase in recovered individuals and those who may have died of natural causes or from the virus.

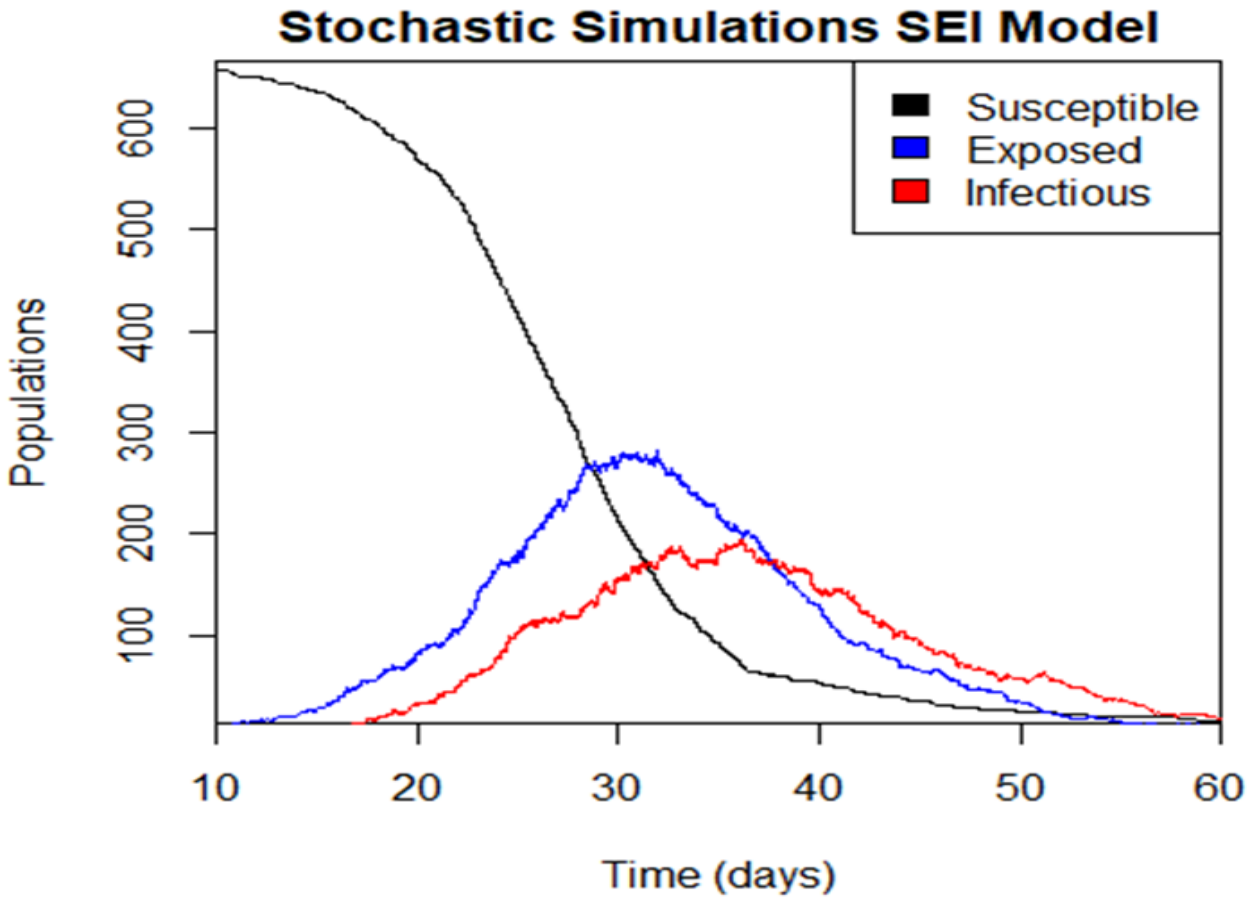

Fig 2 The Numerical Stochastic Simulation of the AI-SEI Model the Analytic AI-SEI Power Spectral Density Analysis**.**

➢ *Analysis of SEI Model Behavior in the Presence of Asymptomatic Infection:*

In this section, we only consider analysis using the analytic equation to observe how the SEI model trajectories behave with asymptomatic infection using R-software. As in Kamara et al. (2010), an insightful description of the parameters of the model is recognized if, for the best fit frequency ranges, the peak of the oscillating curve changes positively with different magnitudes. The parameter values are substituted manually into the expression and vary the infections, latency period, and asymptomatic proportion parameters as shown in the figures with a reduction of 5%.

In Figure 3, we observe that for a decreasing degree of the transition routes, Figures 3 and 4 show that the significant negative impact of asymptomatic and direct infection transmission cannot be recognized using the infective equation. The AI-SEI model infective curve pattern in this research is in agreement with the SEIRD model in (Weitz & Dushoff, 2015) and (Mouanguissa et al., 2021a), where it was shown that with two transmission routes, the SEIR model is very challenging to give insightful characteristics of an outbreak. As in (Weitz & Dushoff, 2015) and (Mouanguissa et al., 2021b), the same trajectories produce increasing values for the infectious and the new infection routes.

ISSN No:-2456-2165

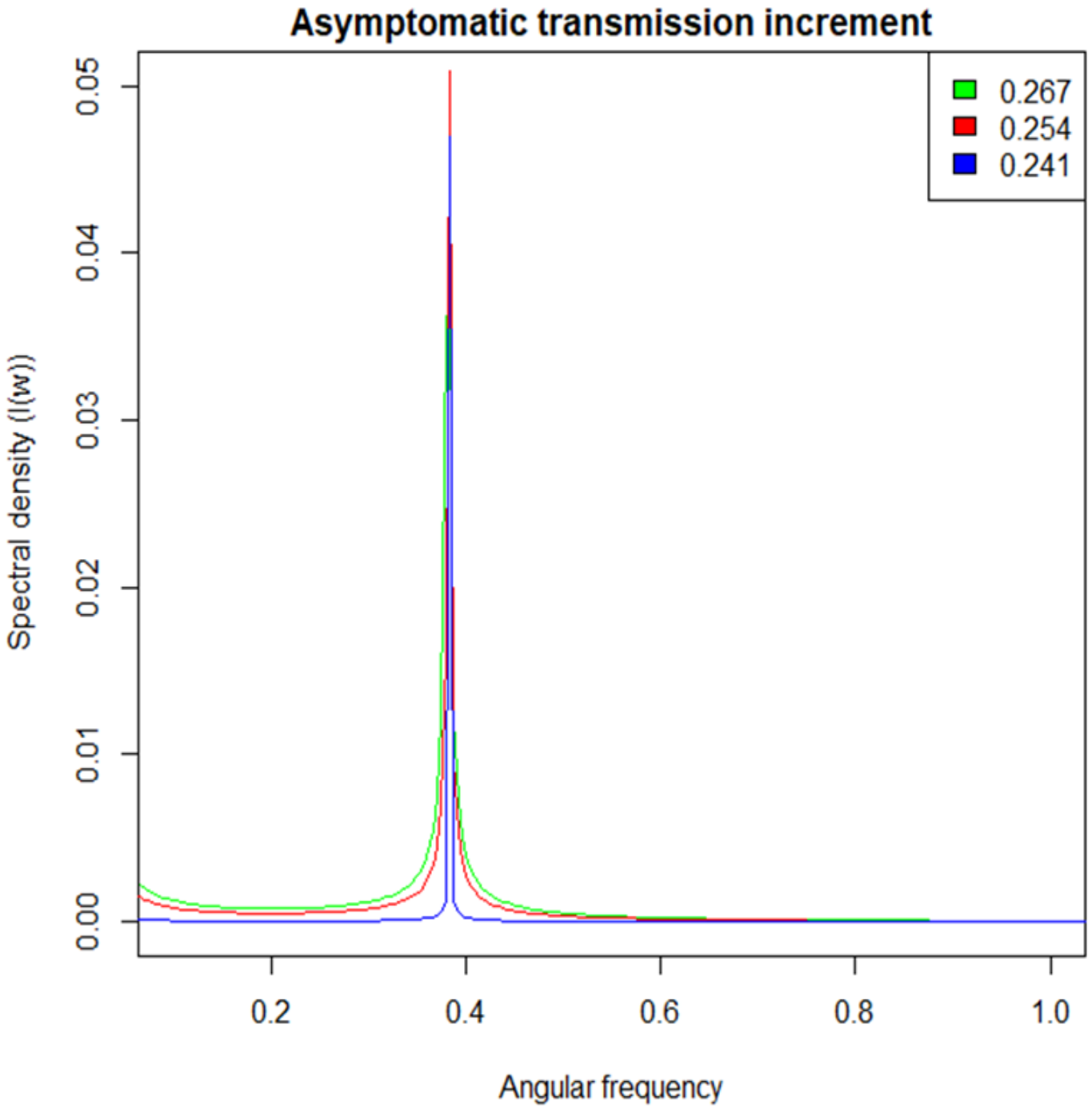

Fig 3 The Analytic Spectral Density Asymptomatic Transmissions SEI Model Analysis.

ISSN No:-2456-2165

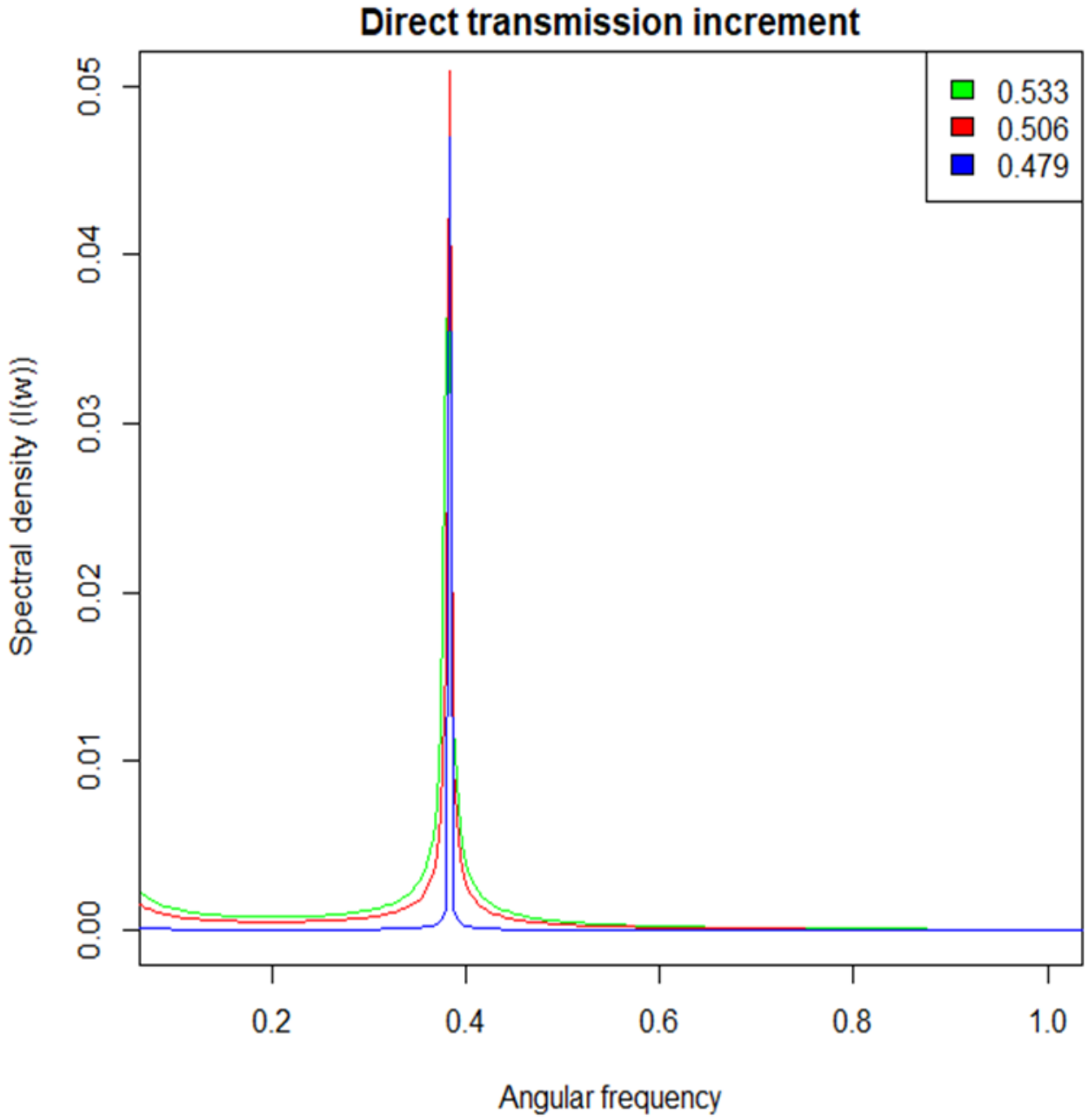

Fig 4 The Analytic Spectral Density Visible Infection Transmissions SEI Model Analysis.

However, we extend our analyses to differ from those of (Weitz & Dushoff, 2015)and (Mouanguissa et al., 2021b) to identify significant parameters in the model to give us an insightful characterization of the COVID-19 outbreak. It is observed that when the magnitude of the latency period is decreased, the spectral infective curve moves significantly. This means that the magnitude of the latency period determines whether or not an asymptomatic transmission has a significant negative impact.

ISSN No:-2456-2165

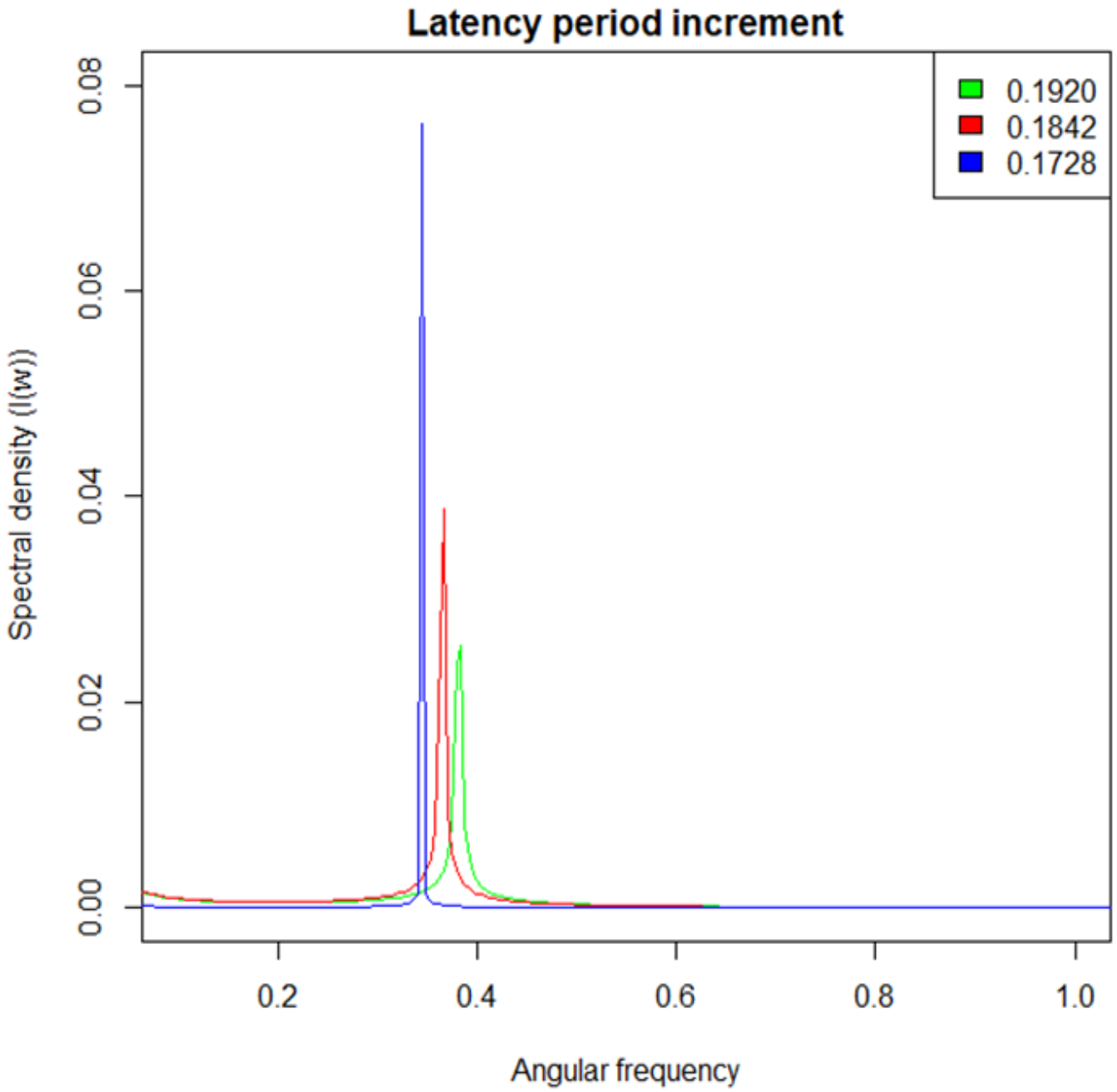

Fig 5 The Analytic Spectral Density Asymptomatic Latency Period Analysis.

ISSN No:-2456-2165

CHAPTER FIVE
SUMMARY, CONCLUSION AND RECOMMENDATIONS

- *Summary:*

In this paper, we used an Ebola virus disease analytical PSD post-death transmission model of the SIR class (Kamara et al.2020) to understand the dynamics of the COVID-19 pandemic. The model in this work uses the SEIR model to take into account latent and infectious infections, which makes the PSD-SIRD model calculation (Kamara et al.2020) equivalent to our PSD asymptomatic infection SEIR model but uses different prediction equations to investigate the trajectories patterns when the infected equation is of paramount interest. The analysis focuses on the steady-state solutions of the current COVID-19 as the data has shown that the basic reproduction number is greater than unity (Read et al., 2020; Zhao et al., 2020).

- *Conclusion:*

It is demonstrated that both visible and asymptomatic infection transmissions have a significant negative impact on the analytical PSD model, which is difficult to recognize. Because of this, estimating important parameters from an AI-SEI model has many implications and challenges in giving accurate predictions for the COVID-19 pandemic. This can be a result of the novelty of the disease, as there is more to learn about the disease's propagation. A better understanding of COVID-19 is necessary to improve the asymptomatic infection estimation that would alleviate this problem of identification. Because the asymptomatic infection is more scary for COVID-19, it is not appropriate to study the disease dynamic by neglecting it because it would miscalculate the parameters that show the severity of the disease (i.e., The magnitude of under-estimation depends on the rate of asymptomatic infection, which in turn depends on the time spent in the hospital, community, and infection risk. Such undervaluation would be concerning for the ongoing effort to develop an accurate model to understand the dynamics of COVID-19 and its preventable strategies.

A relevant feature identified in this research to prevent the asymptomatic infection spread is the reduction rate of those from the asymptomatic population progressing to an infectious population. Therefore, identifying all asymptomatic cases and providing treatment for them are significant response interventions to reduce the spread of asymptomatic infection and put an end to the COVID-19 pandemic. Other responses required vaccinations, individual behavioral changes, and intervention. Identifying all asymptomatic cases is believed to have contributed to the reduction of COVID-19 transmission in many initial hot spot countries like China. A better understanding of asymptomatic infection can help understand the COVID-19 pandemic and provide preventable strategies to control and eliminate the current pandemic.

- *Research Recommendation:*

Furthermore, for future research, the study can consider control strategies such as vaccination and isolation in order to reinvestigate the negative impact of the COVID-19 pandemic's visible direct and asymptomatic infection routes. Wavelet approach is another technique used in recent years to study the propagation of an infectious disease. We recommend that this approach be considered in future research.

REFERENCES

[1]. Agusto, F. B., Teboh-Ewungkem, M. I., & Gumel, A. B. (2015). Mathematical assessment of the effect of traditional beliefs and customs on the transmission dynamics of the 2014 Ebola outbreaks. *BMC Medicine*, *13*(1). https://doi.org/10.1186/s12916-015-0318-3
[2]. Ahinkorah, B. O., Seidu, A. A., Budu, E., Armah-Ansah, E. K., Agbaglo, E., Adu, C., Hagan, J. E., & Yaya, S. (2020). Proximate, intermediate, and distal predictors of under-five mortality in Chad: analysis of the 2014–15 Chad demographic and health survey data. *BMC Public Health*, *20*(1), 1–12. https://doi.org/10.1186/s12889-020-09869-x
[3]. Alonso, D., McKane, A. J., & Pascual, M. (2007). Stochastic amplification in epidemics. (Supplementary Material). *Journal of the Royal Society, Interface / the Royal Society*, *4*, 1–17. https://doi.org/: 10.1098/rsif.2006.0192
[4]. Altaf, K. M., & Atangana, A. (2019). Dynamics of ebola disease in the framework of different fractional derivatives. *Entropy*, *21*(303), 1–32. https://doi.org/10.3390/e21030303
[5]. Anastassopoulou, C., Russo, L., Tsakris, A., & Siettos, C. (2020). Data-based analysis, modelling and forecasting of the COVID-19 outbreak. *PLoS ONE*, *15*(3), 1–21. https://doi.org/10.1371/journal.pone.0230405
[6]. Anirudh, A. (2020). Mathematical modeling and the transmission dynamics in predicting the Covid-19 - What next in combating the pandemic. *Infectious Disease Modelling*, *5*. https://doi.org/10.1016/j.idm.2020.06.002
[7]. Berge, T., Lubuma, J. M. S., Moremedi, G. M., Morris, N., & Kondera-Shava, R. (2017). A simple mathematical model for Ebola in Africa. *Journal of Biological Dynamics*, *11*(1), 42–74. https://doi.org/10.1080/17513758.2016.1229817
[8]. Bermejo, M., Rodríguez-Teijeiro, J. D., Illera, G., Barroso, A., Vilà, C., & Walsh, P. D. (2006). Ebola outbreak killed 5000 gorillas. *Science*, *314*, 1564. https://doi.org/10.1126/science.1133105
[9]. Black, A. J., & McKane, A. J. (2010a). Stochastic amplification in an epidemic model with seasonal forcing. *Journal of Theoretical Biology*, *267*(1), 85–94. https://doi.org/10.1016/j.jtbi.2010.08.014
[10]. Black, A. J., & McKane, A. J. (2010b). Stochasticity in staged models of epidemics: Quantifying the dynamics of whooping cough. *Journal of the Royal Society Interface*, *7*(49). https://doi.org/:10.1098/rsif.2009.0514
[11]. Burke, R. M., Midgley, C. M., Dratch, A., Fenstersheib, M., Haupt, T., Holshue, M., Ghinai, I., Jarashow, M. C., Lo, J., McPherson, T. D., Rudman, S., Scott, S., Hall, A. J., Fry, A. M., & Rolfes, M. A. (2020). Active Monitoring of Persons Exposed to Patients with Confirmed COVID-19 — United States, January–February 2020. *MMWR. Morbidity and Mortality Weekly Report*, *69*(9). https://doi.org/10.15585/mmwr.mm6909e1
[12]. CDC, C. for D. C. and P. (2020a). *How COVID-19 Spreads*. Centers for Disease Control and Prevention. https://www.cdc.gov/coronavirus/2019-ncov/prevent-getting-sick/how-covid-spreads.html
[13]. CDC, C. for D. C. and P. (2020b). Transmission of Coronavirus Disease 2019 (COVID-19). *CDC Bulletin*.
[14]. Centers Disease Control and Prevention(CDC). (2018). Years of Ebola Virus Disease Outbreaks. *CDC Bulletin*. https://www.cdc.gov/vhf/ebola/history/chronology.html
[15]. Chamie, G., Marquez, C., Crawford, E., Peng, J., Petersen, M., Schwab, D., Schwab, J., Martinez, J., Jones, D., Black, D., Gandhi, M., Kerkhoff, A. D., Jain, V., Sergi, F., Jacobo, J., Rojas, S., Tulier-Laiwa, V., Gallardo-Brown, T., Appa, A., … Havlir, D. V. (2021). Community transmission of severe acute respiratory syndrome coronavirus 2 disproportionately affects the latinx population during shelter-in-place in san francisco. *Clinical Infectious Diseases*, *73*. https://doi.org/10.1093/cid/ciaa1234
[16]. Chan, J. F. W., Yuan, S., Kok, K. H., To, K. K. W., Chu, H., Yang, J., Xing, F., Liu, J., Yip, C. C. Y., Poon, R. W. S., Tsoi, H. W., Lo, S. K. F., Chan, K. H., Poon, V. K. M., Chan, W. M., Ip, J. D., Cai, J. P., Cheng, V. C. C., Chen, H., … Yuen, K. Y. (2020). A familial cluster of pneumonia associated with the 2019 novel coronavirus indicating person-to-person transmission: a study of a family cluster. *The Lancet*, *6736*(20), 30154–30159. https://doi.org/10.1016/S0140-6736(20)30154-9
[17]. Chowell, G., Hengartner, N. W., Castillo-Chavez, C., Fenimore, P. W., & Hyman, J. M. (2004). The basic reproductive number of Ebola and the effects of public health measures: The cases of Congo and Uganda. *Journal of Theoretical Biology*, *229*(1), 119–126. https://doi.org/10.1016/j.jtbi.2004.03.006
[18]. Cooke, K. L., & Van Den Driessche, P. (1996). Analysis of an SEIRS epidemic model with two delays. *Journal of Mathematical Biology*, *35*(2). https://doi.org/10.1007/s002850050051
[19]. Dicker, R. C. (2006). Principles of Epidemiology in Public Health Practice. *Cdc*, *May*.
[20]. Dietz, K. (1982). Overall Population Patterns in the Transmission Cycle of Infectious Disease Agents. In *Population Biology of Infectious Diseases*. https://doi.org/10.1007/978-3-642-68635-1_6
[21]. Do, T. S., & Lee, Y. S. (2016). Modeling the Spread of Ebola. *Osong Public Health and Research Perspectives*, *7*(1), 43–48. https://doi.org/10.1016/j.phrp.2015.12.012
[22]. Fanelli, D., & Piazza, F. (2020). Analysis and forecast of COVID-19 spreading in China, Italy and France. *Chaos, Solitons and Fractals*, *134*. https://doi.org/10.1016/j.chaos.2020.109761
[23]. Fasina, F., Shittu, A., Lazarus, D., Tomori, O., Simonsen, L., Viboud, C., & Chowell, G. (2014). Transmission dynamics and control of Ebola virus disease outbreak in Nigeria, July to September 2014. *Eurosurveillance*, *19*(40), 20920. https://doi.org/10.2807/1560-7917.ES2014.19.40.20920

[24]. Gillespie, D. T. (1976). A general method for numerically simulating the stochastic time evolution of coupled chemical reactions. *Journal of Computational Physics*, *22*, 403–434.
[25]. Harko, T., Lobo, F. S. N., & Mak, M. K. (2014). Exact analytical solutions of the Susceptible-Infected-Recovered (SIR) epidemic model and of the SIR model with equal death and birth rates. *Applied Mathematics and Computation*, *236*, 184–194. https://doi.org/10.1016/j.amc.2014.03.030
[26]. Heida, T., Wentink, E. C., & Marani, E. (2013). Power spectral density analysis of physiological, rest and action tremor in Parkinson's disease patients treated with deep brain stimulation. *Journal of NeuroEngineering and Rehabilitation*, *10*(1). https://doi.org/10.1186/1743-0003-10-70
[27]. Herrerías-Azcué, F., & Galla, T. (2017a). The effects of heterogeneity on stochastic cycles in epidemics. *Scientific Reports*, *7*(13008). https://doi.org/10.1038/s41598-017-12606-x
[28]. Herrerías-Azcué, F., & Galla, T. (2017b). The effects of heterogeneity on stochastic cycles in epidemics. *Scientific Reports*, *7*(1). https://doi.org/10.1038/s41598-017-12606-x
[29]. Hethcote, H. W. (2005). The Mathematics of Infectious Diseases. *SIAM Review*, *42*(4), 399–653. https://doi.org/10.1137/s0036144500371907
[30]. Hu, K., Bianco, S., Edlund, S., & Kaufman, J. (2015). *The Impact of Human Behavioral Changes in 2014 West Africa Ebola Outbreak The Impact of Human Behavioral Changes in 2014 West Africa Ebola Outbreak*. *March*. https://doi.org/10.1007/978-3-319-16268-3
[31]. Hu, Z., Cui, Q., Han, J., Wang, X., Sha, W. E. I., & Teng, Z. (2020). Evaluation and prediction of the COVID-19 variations at different input population and quarantine strategies, a case study in Guangdong province, China. *International Journal of Infectious Diseases*, *95*. https://doi.org/10.1016/j.ijid.2020.04.010
[32]. Huang, C., Wang, Y., Li, X., Ren, L., Zhao, J., Hu, Y., Zhang, L., Fan, G., Xu, J., Gu, X., Cheng, Z., Yu, T., Xia, J., Wei, Y., Wu, W., Xie, X., Yin, W., Li, H., Liu, M., … Cao, B. (2020). Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. *The Lancet*, *395*(10223). https://doi.org/10.1016/S0140-6736(20)30183-5
[33]. Judson, S. D., Fischer, R., Judson, A., & Munster, V. J. (2016). Ecological Contexts of Index Cases and Spillover Events of Different Ebolaviruses. *PLoS Pathogens*, *12*(8), e1005780. https://doi.org/10.1371/journal.ppat.1005780
[34]. Kamara, A. A., Mouanguissa, L. N., & Barasa, G. O. (2021). Mathematical modelling of the COVID-19 pandemic with demographic effects. *Journal of the Egyptian Mathematical Society*, *29*(1). https://doi.org/10.1186/s42787-021-00118-7
[35]. Kamara, A. A., Wang, X., & Mouanguissa, L. N. (2020a). Analytical solution for post-death transmission model of Ebola epidemics. *Applied Mathematics and Computation*, *367*. https://doi.org/10.1016/j.amc.2019.124776
[36]. Kamara, A. A., Wang, X., & Mouanguissa, L. N. (2020b). Analytical solution for post-death transmission model of Ebola epidemics. *Applied Mathematics and Computation*, *367*(124774). https://doi.org/10.1016/j.amc.2019.124776
[37]. kamara, A. A., Wang, X., & Tarus, S. K. (2021). Global analysis of an environmental and death transmission model for Ebola outbreak with perturbation. *Indian Journal of Pure and Applied Mathematics*. https://doi.org/10.1007/s13226-021-00085-w
[38]. Kermack, W. O., & McKendrick, A. G. (1927). Contributions to the mathematical theory of epidemics-I. *Proceedings of the Royal Society of London*, *115*, 700–721.
[39]. Kermack, W. O., & McKendrick, A. G. (1933). Contributions to the mathematical theory of epidemics. III. *Proceedings of the Royal Society of London*, *141*, 94–112. https://doi.org/10.1098/rspa.1933.0106
[40]. Kiskowski, M., Chowell, G., Kiskowski, M., & Chowell, G. (2016). Modeling household and community transmission of Ebola virus disease : Epidemic growth , spatial dynamics and insights for epidemic control. *Virulence*, *7*, 163–173. https://doi.org/10.1080/21505594.2015.1076613
[41]. Kucharski, A. J., Russell, T. W., Diamond, C., Liu, Y., Edmunds, J., Funk, S., Eggo, R. M., Sun, F., Jit, M., Munday, J. D., Davies, N., Gimma, A., van Zandvoort, K., Gibbs, H., Hellewell, J., Jarvis, C. I., Clifford, S., Quilty, B. J., Bosse, N. I., … Flasche, S. (2020). Early dynamics of transmission and control of COVID-19: a mathematical modelling study. *The Lancet Infectious Diseases*, *20*(5). https://doi.org/10.1016/S1473-3099(20)30144-4
[42]. Legrand, J., Grais, R. F., Boelle, P. Y., Valleron, A. J., & Flahault, A. (2007). Understanding the dynamics of Ebola epidemics. *Epidemiology and Infection*, *135*(4), 610–621. https://doi.org/10.1017/S0950268806007217
[43]. Lekone, P. E., Finkenstädt, B. F., Lekone, P. E., & Finkenstaidt, B. F. (2006). Statistical Inference in a Stochastic Epidemic SEIR Model with Control Intervention : Ebola as a Case. *Biometrics*, *62*(4), 1170–1177.
[44]. Leroy, E. M., Kumulungui, B., Pourrut, X., Rouquet, P., Hassanin, A., Yaba, P., Délicat, A., Paweska, J. T., Gonzalez, J. P., & Swanepoel, R. (2005). Fruit bats as reservoirs of Ebola virus. *Nature*, *438*, 575–576. https://doi.org/10.1038/438575a
[45]. Levin-Sparenberg, E., Gicquelais, R., Blanco, N., Ismail, M. D., Lee, K. H., & Foxman, B. (2015). Ebola: The Natural and Human History of a Deadly Virus By David Quammen. *American Journal of Epidemiology*, *181*(2). https://doi.org/10.1093/aje/kwu354
[46]. Li, M. Y., Graef, J. R., Wang, L., & Karsai, J. (1999). Global dynamics of a SEIR model with varying total population size. *Mathematical Biosciences*, *160*(1999), 192–213. https://doi.org/10.1016/S0025-5564(99)00030-9

[47]. Li, Q., Guan, X., Wu, P., Wang, X., Zhou, L., Tong, Y., Ren, R., Leung, K. S. M., Lau, E. H. Y., Wong, J. Y., Xing, X., Xiang, N., Wu, Y., Li, C., Chen, Q., Li, D., Liu, T., Zhao, J., Liu, M., … Feng, Z. (2020). Early Transmission Dynamics in Wuhan, China, of Novel Coronavirus-Infected Pneumonia. *The New England Journal of Medicine*. https://doi.org/10.1056/NEJMoa2001316

[48]. Lin, Q., Zhao, S., Gao, D., Lou, Y., Yang, S., Musa, S. S., Wang, M. H., Cai, Y., Wang, W., Yang, L., & He, D. (2020). A conceptual model for the coronavirus disease 2019 (COVID-19) outbreak in Wuhan, China with individual reaction and governmental action. *International Journal of Infectious Diseases*, *2020*(93), 211–216. https://doi.org/10.1016/j.ijid.2020.02.058

[49]. Liu, Z., Magal, P., Seydi, O., & Webb, G. (2020). A COVID-19 epidemic model with latency period. *Infectious Disease Modelling*, *5*. https://doi.org/10.1016/j.idm.2020.03.003

[50]. Mandal, S., Bhatnagar, T., Arinaminpathy, N., Agarwal, A., Chowdhury, A., Murhekar, M., Gangakhedkar, R., & Sarkar, S. (2020). Prudent public health intervention strategies to control the coronavirus disease 2019 transmission in India: A mathematical model-based approach. *Indian Journal of Medical Research*, *151*(2). https://doi.org/10.4103/ijmr.IJMR_504_20

[51]. Mbogo, R. W., & Odhiambo, J. W. (2021). COVID-19 outbreak, social distancing and mass testing in Kenya-insights from a mathematical model. *Afrika Matematika*, *32*(5–6). https://doi.org/10.1007/s13370-020-00859-1

[52]. McKane, A. J., & Newman, T. J. (2005). Predator-prey cycles from resonant amplification of demographic stochasticity. *Physical Review Letters*, *94*. https://doi.org/10.1103/PhysRevLett.94.218102

[53]. Meakin, S. R., Tildesley, M. J., Davis, E., & Keeling, M. J. (2018). *A metapopulation model for the 2018 Ebola outbreak in Equateur province in the Democratic Republic of the Congo*. *May*, 1–30.

[54]. Melek Manshouri, N. (2022). Identifying COVID-19 by using spectral analysis of cough recordings: a distinctive classification study. *Cognitive Neurodynamics*, *16*(1). https://doi.org/10.1007/s11571-021-09695-w

[55]. Mouanguissa, L. N., Kamara, A. A., & Wang, X. (2021a). Modeling 2018 Ebola virus disease outbreak with Cholesky decomposition. *Mathematical Methods in the Applied Sciences*, *44*(7). https://doi.org/10.1002/mma.7145

[56]. Mouanguissa, L. N., Kamara, A. A., & Wang, X. (2021b). Modeling 2018 Ebola virus disease outbreak with Cholesky decomposition. *Mathematical Methods in the Applied Sciences*, 1–14. https://doi.org/10.1002/mma.7145

[57]. M.S.M., van M., K.G.M., M., W.W., van S., J.-W., B. der S., L., R., A., T., M.J.M., B., van Mourik, M. S. M., Moons, K. G. M., van Solinge, W. W., Berkelbach-van der Sprenkel, J.-W., Regli, L., Troelstra, A., & Bonten, M. J. M. (2012). Automated Detection of Healthcare Associated Infections: External Validation and Updating of a Model for Surveillance of Drain-Related Meningitis. *PLoS ONE*, *7*(12).

[58]. Ndanguza, D., Tchuenche, J. M., & Haario, H. (2013). Statistical data analysis of the 1995 Ebola outbreak in the Democratic Republic of Congo. *Afrika Matematika*, *24*, 55–68. https://doi.org/10.1007/s13370-011-0039-5

[59]. Nieddu, G. T., Billings, L., Kaufman, J. H., Forgoston, E., & Bianco, S. (2017). Extinction pathways and outbreak vulnerability in a stochastic Ebola model. *Journal of the Royal Society Interface*, *14*. https://doi.org/10.1098/rsif.2016.0847

[60]. Oke, I. I., Oyebo, Y. T., Fakoya, O. F., Benson, V. S., & Tunde, Y. T. (2021). A Mathematical Model for Covid-19 Disease Transmission Dynamics with Impact of Saturated Treatment: Modeling, Analysis and Simulation. *OALib*, *08*(05). https://doi.org/10.4236/oalib.1107332

[61]. Otunuga, O. M. (2017). Global Stability of Nonlinear Stochastic SEI Epidemic Model with Fluctuations in Transmission Rate of Disease. *International Journal of Stochastic Analysis*, *2017*(6313620), 1–7. https://doi.org/10.1155/2017/6313620

[62]. Perlman, S. (2020). Another Decade, Another Coronavirus. *New England Journal of Medicine*. https://doi.org/10.1056/nejme2001126

[63]. Rao, F., Wang, W., & Li, Z. (2012). Stability analysis of an epidemic model with diffusion and stochastic perturbation. *Communications in Nonlinear Science and Numerical Simulation*, *17*(6), 2551–2563. https://doi.org/10.1016/j.cnsns.2011.10.005

[64]. Read, J. M., Bridgen, J. R., Cummings, D. A., Ho, A., & Jewell, C. P. (2020). Novel coronavirus 2019-nCoV: early estimation of epidemiological parameters and epidemic predictions. *MedRxiv*. https://doi.org/10.1101/2020.01.23.20018549

[65]. Rozhnova, G., & Nunes, A. (2009). Fluctuations and oscillations in a simple epidemic model. *Physical Review E - Statistical, Nonlinear, and Soft Matter Physics*, *79*(4). https://doi.org/10.1103/PhysRevE.79.041922

[66]. Rozhnova, G., & Nunes, A. (2010). Stochastic effects in a seasonally forced epidemic model. *Physical Review E - Statistical, Nonlinear, and Soft Matter Physics*, *82*. https://doi.org/10.1103/PhysRevE.82.041906

[67]. Sabbih, G. O., Korsah, M. A., Jeevanandam, J., & Danquah, M. K. (2021). Biophysical analysis of SARS-CoV-2 transmission and theranostic development via N protein computational characterization. In *Biotechnology Progress* (Vol. 37, Issue 2). https://doi.org/10.1002/btpr.3096

[68]. Salem, D., & Smith, R. (2016). A mathematical model of ebola virus disease: Using sensitivity analysis to determine effective intervention targets. *Simulation Series*, *48*(9). https://doi.org/10.22360/summersim.2016.scsc.008

[69]. Side, S., Irwan, Mulbar, U., & Sanusi, W. (2017). SEIR model simulation for Hepatitis B. *AIP Conference Proceedings*, *1885*. https://doi.org/10.1063/1.5002392

[70]. Simões, M., Telo Da Gama, M. M., & Nunes, A. (2008). Stochastic fluctuations in epidemics on networks. *Journal of the Royal Society Interface*, *5*(22), 555–566. https://doi.org/10.1098/rsif.2007.1206
[71]. Turner, A., Jung, C., Tan, P., Gotika, S., & Mago, V. (2015). A comprehensive model of spread of malaria in humans and mosquitos. *Conference Proceedings - IEEE SOUTHEASTCON*, *2015-June*(June). https://doi.org/10.1109/SECON.2015.7132968
[72]. Upadhyay, R. K., Pal, A. K., Kumari, S., & Roy, P. (2019). Dynamics of an SEIR epidemic model with nonlinear incidence and treatment rates. *Nonlinear Dynamics*, *96*(4). https://doi.org/10.1007/s11071-019-04926-6
[73]. Van Den Driessche, P., & Watmough, J. (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. In *Mathematical Biosciences*.
[74]. Van Kampen, N. G. (2007). Stochastic processes in physics and chemistry. In *North-Holland Personal Library* (Third edit). Elsevier.
[75]. Wang, C., Horby, P. W., Hayden, F. G., & Gao, G. F. (2020). A novel coronavirus outbreak of global health concern. In *The Lancet* (pp. 470–473). https://doi.org/10.1016/S0140-6736(20)30185-9
[76]. Wang, R. H., Jin, Z., Liu, Q. X., van de Koppel, J., & Alonso, D. (2012). A simple stochastic model with environmental transmission explains multi-year periodicity in outbreaks of avian flu. *PLoS ONE*, *7*(2). https://doi.org/10.1371/journal.pone.0028873
[77]. Wang, X.-S., & Zhong, L. (2015). Ebola outbreak in West Africa: real-time estimation and multiple-wave prediction. *Mathematical Biosciences and Engineering*. https://doi.org/10.3934/mbe.2015.12.1055
[78]. Wei, F., & Xue, R. (2020). Stability and extinction of SEIR epidemic models with generalized nonlinear incidence. *Mathematics and Computers in Simulation*, *170*. https://doi.org/10.1016/j.matcom.2018.09.029
[79]. Weinstein, S. J., Holland, M. S., Rogers, K. E., & Barlow, N. S. (2020). Analytic solution of the SEIR epidemic model via asymptotic approximant. *Physica D: Nonlinear Phenomena*, *411*. https://doi.org/10.1016/j.physd.2020.132633
[80]. Weitz, J. S., & Dushoff, J. (2015). Modeling post-death transmission of Ebola: challenges for inference and opportunities for control. *Scientific Reports*, *5*(8751). https://doi.org/10.1038/srep08751
[81]. WHO. (2018a). *Health Emergency Information and Risk Assessment EBOLA VIRUS DISEASE Democratic Republic of Congo External Situation Report 1*. 1–6. http://apps.who.int/iris/bitstream/handle/10665/272509/SITREP-EVD-DRC-20180511.pdf?ua=1
[82]. WHO, W. H. O. (2018b). *Ebola virus disease 2,Information, Health Emergency Assessment, Risk*. 1–11.
[83]. WHO, W. H. O. (2020). Report of the WHO-China Joint Mission on Coronavirus Disease 2019 (COVID-19). *WHO*, *2019*(February), 1–40.
[84]. WHO (World Health Organization). (2020a). Novel Coronavirus (2019-nCoV)Situation Report - 142. In *WHO Bulletin*. https://www.who.int/docs/default-source/coronaviruse/situation-reports/20200610-covid-19-sitrep 142.pdf?sfvrsn=180898cd_6
[85]. WHO (World Health Organization). (2020b). Statement on the meeting of the International Health Regulations (2005) Emergency Committee regarding the outbreak of novel coronavirus (2019-nCoV). In *WHO Newsletter*.
[86]. WHO (World Health Organization). (2020c). *WHO Director-Generals opening remarks at the mission briefing on COVID-19 - 26 February 2020*. Https://Www.Who.Int/Dg/Speeches/Detail/Who-Director-General-S-Opening-Remarks-At-the-Media-Briefing-on-Covid-19---11-March-2020. https://doi.org/11 March 2020
[87]. World Health Organization 2020. (2020). Report of the WHO-China Joint Mission on Coronavirus Disease 2019 (COVID-19) 16-24 February 2020. *Geneva: World Health Organization*.
[88]. Worldometer. (2021). *Sierra Leone COVID_ 4,042 Cases and 79 Deaths*.
[89]. Yamazaki, K. (2018). Threshold dynamics of reaction–diffusion partial differential equations model of Ebola virus disease. *International Journal of Biomathematics*, *11*(8). https://doi.org/10.1142/s1793524518501085
[90]. Yang, C., & Wang, J. (2020). A mathematical model for the novel coronavirus epidemic in Wuhan, China. *Mathematical Biosciences and Engineering*, *17*(3). https://doi.org/10.3934/mbe.2020148
[91]. Zhao, S., Lin, Q., Ran, J., Musa, S. S., Yang, G., Wang, W., Lou, Y., Gao, D., Yang, L., He, D., & Wang, M. H. (2020). Preliminary estimation of the basic reproduction number of novel coronavirus (2019-nCoV) in China, from 2019 to 2020: A data-driven analysis in the early phase of the outbreak. *International Journal of Infectious Diseases*, *92*, 214–217. https://doi.org/10.1016/j.ijid.2020.01.050