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SEIR Childhood Disease Model With Constant Vaccination Strategy

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Declaration

This research thesis is my original work and has not been presented for a degree award in any other university.

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Nomenclature

S	Susceptible.
E	Exposed group to the disease.
I	Infected.
R	Vaccinated and recovered.
N	Total population.
P	Vaccination rate at birth.
β_1	Susceptible contact rate of disease from exposed group.
β_2	Susceptible contact rate of disease from infected group.
μ	Natural death rate for all the groups.
γ	Progression rate from infected to recovered group due to treatment.
π	Proportion of susceptible that are vaccinated at birth.
R_v	Vaccination reproduction number.
R_0	Basic reproduction number.
α	The death rate due to disease infection.
δ	Progression rate from exposed to infected group.
P_c	Critical vaccination proportion point.
E^0	Disease-free equilibrium.
E^u	Endemic equilibrium.

Abbreviations

ADM Adomain Decomposition Method.

RK4 Fourth order Runge-Kutta integration Method.

WHO World health organization.

UNICEF United nations children's fund.

OVP Oral polio vaccine.

IVP Inactivated polio vaccine.

MCV Measles-containing vaccine.

SIR Susceptible, infective and recovered population.

SIV Susceptible, infective and vaccinated population.

KEMRI Kenya medical research institute.

EE Endemic Equilibrium.

DFE Disease-free equilibrium.

LAS Locally Asymptotically stable.

SEIR Susceptible, exposed, infective and recovered population.

SIRV Susceptible, exposed, infective and vaccinated population.

HAM Homotopy analysis method.

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Dedication

This research work is dedicated to God Almighty and my family.

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Abstract

Childhood diseases are increasingly becoming the most common form of infectious diseases. These diseases include measles, mumps, Influenza, smallpox, chicken pox, Rubella, Polio etc., which take special interest to children under-five years who are born highly susceptible. Childhood vaccination programs and campaigns have yielded in high levels of permanent immunity against childhood diseases. Childhood diseases have several characteristics which make them well fit for mathematical modeling such as a relatively short incubation and infectious periods and confer permanent immunity when vaccinated. In this study, a SEIR model that monitors the temporal transmission dynamics of a childhood disease in the presence of preventive vaccine was formulated and analyzed. We normalized the governing model. Maple was used in carrying out the simulations. Semi-numerical Adomain Decomposition method was used to compute an approximate solution of the non-linear system of differential equations governing the model. The results obtained by Adomain Decomposition method are compared with the pure numerical classical fourth order Runge-Kutta integration method to gauge it's effectiveness in describing the transmission dynamics of the model. Graphical results were presented and discussed to illustrate the solution of the problem. The achieved results reveals that the disease will die out within the community if the vaccination coverage is above the critical vaccination proportion, P_c.

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Chapter 1

Introduction

1.1 Background of the study

Mathematical models are considered to be viable frameworks for describing the temporal dynamics and control spread of infectious diseases. Models in modern days play a crucial role in policy making, planning for emergency outbreaks risk assessment, evaluation of control programs and optimizing control methods (Al-Sheikh (2012)).

Childhood diseases are increasingly becoming the most common form of infectious diseases. These diseases include measles, mumps, Influenza, smallpox, chicken pox, Rubella, Polio etc., (as in Hethcote (2000); Zhang et al. (2015); May et al. (1979); Krämer et al. (2009); Bai and Ren (2015); Arafa et al. (2012); Makinde (2007)) which take special interest to children under-five years who are born highly susceptible. Children under five are frequently in close contact with others, at home, at school and playground hence such childhood diseases can spread faster. According to UNICEF (2012), it is estimated that out of 6.9 million deaths in children under-five in 2011, infectious diseases such as measles, poliomyelitis, diarrhea etc. account for almost two-thirds (64%) of the deaths as in UNICEF (2012) and Organization et al. (2010) .

Childhood diseases have several characteristics which make them well fit for mathematical modeling such as a relatively short incubation and infectious periods, confer permanent immunity etc. as shown by May et al. (1979) in Table 1.1.

Table 1.1: Characteristics of some common infectious childhood diseases, adopted from (May et al. (1979))

Disease	Incubation	Infectiousness	Infectiousness	Immunity	Mortality rate
Measles	$9-12$ days	$5-7$ days	high	permanent	low-high
Smallpox	$12-14$ days	10 days	medium	permanent	high
Rubella	$17-20$ days	14 days	medium	permanent	low
Mumps	$10-20$ days	7 days	medium	permanent	low
Whooping cough	$7-10$ days	$14+$ days	high	permanent	medium
Poliomyelitis	$5-20$ days	$long$	high	permanent	medium
Chicken pox	$13-17$ days	$20-30$ days	high	permanent	low

As in most childhood diseases, there is a time delay between an individual becoming infected and developing disease symptoms. The exposed group only have the disease virus in the body due to exposure to the virus but have not developed the disease symptoms physically (Krämer et al. (2009); Yano et al. (2016)). We have both the exposed and infected groups being infectious, but the exposed group is less infectious than the infected group since the infected individuals are already showing the symptoms of the disease.

The disease is in the incubation period at the exposed group, an illustrative example is incubatory carriers (individuals who are going to show symptoms, but transmit infection before their symptoms begin) such as measles infected individuals who start transmitting the virus through nasal and throat secretions a day or two before any rashes are observed. For inapparent infections, individuals never develop any symptoms but they transmit infections to others. These occur in diseases such as poliomyelitis and Hepatitis A, children under-five years are effective spreaders since transmission is mainly through feces regardless of the presence of symptoms Yano et al. (2016). In Influenza, the onset of symptoms begins one day after the infected individuals have become infectious i.e. the incubation period is one day longer than the latent period (Krämer et al. (2009)).

Vaccination is in most cases effective and safe strategy against most childhood infectious diseases Yano et al. (2016), an illustrative example of vaccine-preventable childhood disease is Poliomyelitis.

This disease affects the central nervous system of an infected person with attributes of being highly contagious, an incurable viral infection that paralyzes a child permanently or leads to deaths shortly after infection. Significant steps have been taken towards global elimination of poliovirus. In 2013, sudden outbreaks were witnessed in Somalia, Ethiopia, Kenya, Syria and Cameroon. This occurrence was due to imports of polio virus from endemic countries. Kenya reported 14 polio cases in 2013 as in Organization et al. (2010) and UNICEF (2015).

To combat the outbreak polio immunization programs were implemented, thanks to Kenyan ministry of health with the support of UNICEF that helped to immunize children under-five in most parts of the country including refugee camps and slums which are more vulnerable UNICEF (2015). An estimated 530,000 children under-five in Kenya were vaccinated in December 2013 with oral polio vaccine (OPV), in addition to 120,000 children under the age of five who received inactivated polio vaccine (IPV) (Organization et al. (2010); WHO (2015a)).

World Health Organization (WHO) advice OPV to be the vaccine of choice for routine child immunization in many countries. However, for endemic countries and in countries vulnerable to re-infection, WHO advice an OPV dose at a birth of a child followed closely by three primary doses of OPV with at least one IPV dose as demonstrated by UNICEF (2015) and Organization et al. (2010)).

Figure 1.1: Picture showing children receiving inactivated polio vaccine (IPV) and oral polio vaccine (OPV) in Daadab refugee camp in Kenya on 26 February 2014 and Pakistan, adopted from UNICEF and WHO.

A medical officer at the world Health Organization, **Dr. Okiror**, remarked "*This time we are including the injectable polio vaccine, when IPV is combined with OPV, or polio drops, the immunity*

of the target group improves a lot. We want to make sure that there is better immunity gained among the children" UNICEF (2015).

Figure 1.2 shows that polio remains endemic in two countries-Afghanistan and Pakistan as of December 2015, this implies that unless poliovirus is eradicated completely in these countries, all countries worldwide remain vulnerable to infection, especially developing countries with weak public health institutions and immunization programs and those countries that have trade links or make visits to the endemic countries (Organization et al. (2010)).

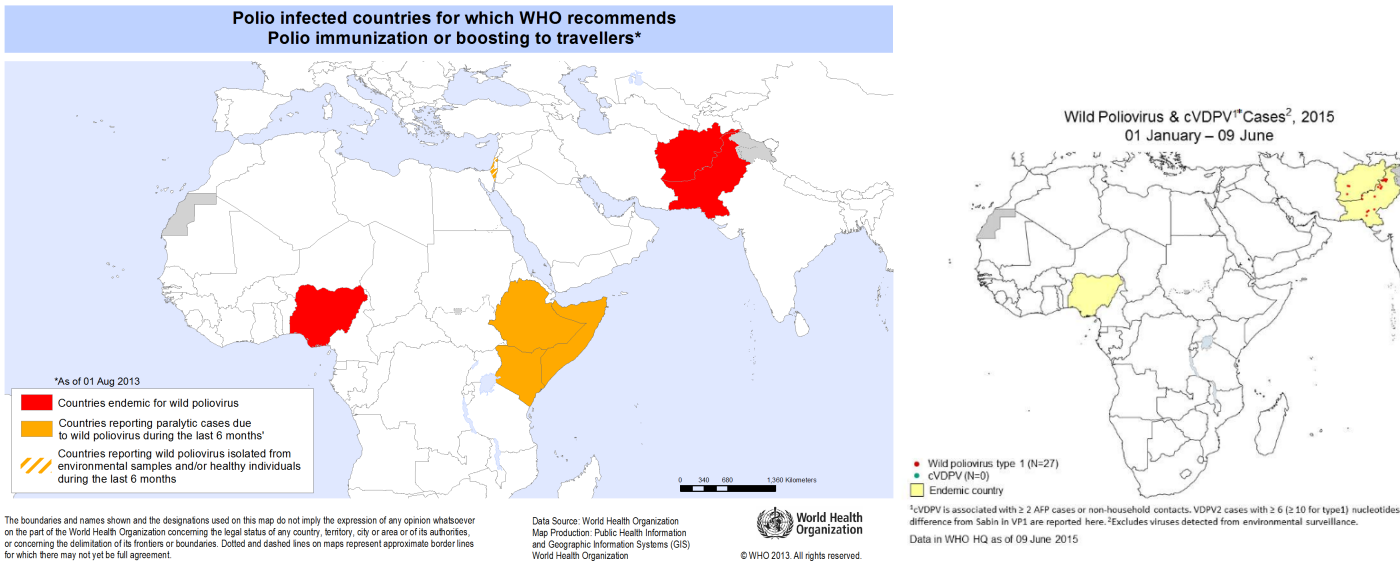

Figure 1.2: Map showing polio endemic countries in 2013 and 2015

Measles is described by WHO (2015a) as one of the most deadly of all childhood diseases. Measles is a highly contagious vaccine-preventable viral disease. The mode of transmission is by droplets or person-person contact with infective persons. Measles is an infectious disease which has been observed to be endemic in many countries worldwide both developing and industrialized countries. Measles is estimated to have caused $145,700$ deaths in 2013 with a confidence interval of $81,100 - 335,400$. Surprisingly, more than half of the deaths witnessed in Africa, as given by (WHO (2015b) and WHO (2015a)).

Prevention strategies that can lower measles child mortality rate in developing nations include the administration of at least two doses of measles-containing vaccine (MCV1) to all children, these will help under-five to acquire long lasting immunity against measles. Vaccinations have increasingly been

effective in controlling the spread of Measles, thus there is a need for optimizing vaccination coverage levels in many countries. WHO report of 2015, estimates 85% vaccination coverage of children with one dose of measles-containing vaccine in 2014 globally, up from 73% coverage recorded in 1990 as in WHO (2015a).

Table 1.2: Shows the estimates of vaccination coverage with the first and second doses of measles-containing vaccine (MCV) administered through routine immunization services, reported cases and incidence by who regions, 2000 and 2014 (adopted from WHO (2015b) and **WHO (2015a)**).

	2000						2014						
WHO REGION	Coverage with 1st dose of MCV (%)	Member states with coverage	Coverage with 2nd dose of MCV (%)	No. of reported measles cases	Estimated measles deaths		Coverage with 1st dose of MCV (%)	Member states with coverage	Coverage with 2nd dose of MCV (%)	No. of reported measles cases	Measles incidence	Estimated measles deaths	
		≥ 90%			No.	95% confidence interval		≥ 90%				No.	95% confidence interval
African	53	9	5	520102	342800	225400-574200	73	30	11	73914	78	48000	41600-165000
Americas	93	63	45	1754	<100	-	92	77	51	1817	1.9	<100	-
Eastern Mediterranean	72	57	28	38592	54300	322000-91100	77	57	66	18129	29	13900	9500-38400
European	91	60	49	37421	300	100-2200	94	83	84	14176	19	100	0-1800
South-East Asia	63	30	3	78558	138500	102100-185900	84	45	59	28403	18	46900	27900-80800
South-East Asia (excluding India)	78	33	9	39723	52700	32700-81300	85	50	78	3426	12	8100	2700-25400
India	56	-	0	38835	85800	69400-104700	83	-	51	24977	20	38800	25300-55400
Western Pacific	85	43	2	177052	10800	5400-53600	97	74	93	131043	71	6100	800-63300
Total	72	44	15	853479	546800	365200-907000	85	63	56	267482	40	114900	53700-330000

As shown in Table 1.2, the vaccination coverage with the second dose of measles-containing vaccine is estimated to be 56% globally as compared to 15% recorded in 2000. The level of vaccines coverage is 93% as seen in Western Pacific region, in comparison to 11% in the African region (WHO (2015b,a)).

Another illustrative example of a childhood vaccine-preventable disease is the mumps infection (Ma et al. (2013); WHO (2015a)). A SEIR model of invasion of mumps in China was studied by Ma et al. (2013). They estimated the model parameters using the demographic and epidemiological data in China ranging from 2005 to 2010. Mumps is attributed to be a respiratory childhood disease which is viral and spreads by secretions of mouth and noses. It then matures to develop swelling

of the parotid salivary gland as described by WHO (2015a). The main symptoms of mumps are the swollen, painful and sore salivary glands in the face. The infectiousness period of mumps lasts for 10 to 14 days. The susceptible people will attain permanent immunity after they receive vaccinations or are recovered from the infection. It is estimated that 121 countries worldwide have received and administered mumps vaccine by the end of 2014 as shown by WHO (2015a) and Ma et al. (2013). The SEIR model best describes the infection of mumps and other childhood diseases due to its incubation period and permanent immunity when vaccines and treatment are administered (Ma et al. (2013)).

Childhood diseases are continually becoming endemic in many parts of the world and causes thousands of child deaths yearly. For instance, currently polio is endemic in Afghanistan and Pakistan and measles being persistent in many countries globally with outbreaks that result to deaths occurring in developing nations. Births and deaths are often ignored in the formulation of their models since the "clock of the disease" are much shorter than the demographic changes. To formulate a plausible model for a disease which may be persistent we need to take into account deaths due to diseases. The possibility of eliminating the endemic scenario of the disease and controlling spread or eradication of the disease in a population is a worthy field for research (Brauer et al. (2001); Allen et al. (2008); Anderson et al. (1992)).

The occurrence of the endemic nature in measles is due to the inflow of susceptible individuals into the population, and a plausible model must consider births to the population and deaths due to diseases in the model. To include births and deaths in an infectious disease model we assume a varying population size so that births remain unequal to deaths as described by Makinde (2007); May et al. (1979); Hethcote (2000). This is vital in developing regions such as African and Asian regions with a very high mortality rate for childhood diseases such as measles. The inclusion of realistic control methods in mathematical models is significant in order to ascertain the intervention of public health authorities. The control strategies available to control the spread of infectious diseases include pharmaceutical interventions e.g. Drugs, vaccines etc. and non-pharmaceutical interventions such as social distancing, quarantine or isolation (Anderson et al. (1992); Hethcote (1976); May et al. (1979)). Vaccination is the most effective preventive strategy in most cases. Low levels of vaccine coverage among children with an inconsistent supply of preventive vaccines are considered to be the main reasons that contribute to the re-emergence of many childhood diseases, this implies

that a combination of high vaccination coverage levels with the application of constant vaccination strategy may lead to total eradication of childhood vaccine-preventable diseases. Since vaccination is considered quite often to be the most effective strategy against childhood diseases, the need to understand optimal ways to control the spread of these diseases, therefore, presents itself stronger than ever in the twenty-first century (May et al. (1979)).

In this study, a SEIR epidemic model for childhood diseases with a preventive vaccine that poses long-life immunity to the disease was studied. The focus is to use this model to predict the optimal vaccine coverage required to assure that the disease is controlled or dies out. The four-dimensional model monitors the temporal dynamics of the susceptible individuals, exposed individuals who are also infectious, recovered individuals and infectious individuals.

1.2 Statement of the Problem

Childhood diseases are some of the most common infectious diseases worldwide. So far, efforts to control the spread of childhood diseases through vaccination campaigns has resulted in a high decline in child mortality rates caused by diseases like measles, polio, mumps etc. Presently the incidence has been significantly lowered by vaccination. Childhood diseases still poses a problem for public health authorities with outbreaks reported in a number of developing regions. Although, there have been a number of studies over the years on childhood diseases, but the emphasis has been on assuming the population size to be a constant hence disregarding deaths due to the diseases. Recent studies have shown that using varying population size so that birth rates remain unequal to deaths to model childhood diseases and applying different numerical methods can be described successfully by (Makinde (2007, 2009); Moghadas and Gumel (2003); Hethcote (2000); May et al. (1979); Arafa et al. (2012); Yano et al. (2016)). As in many diseases, there is a time delay between an individual becoming infected and developing the symptoms of the disease physically. The exposed individuals may present more risk for transmission of disease than the infected since their contacts are unaware of the infection. The exposed individuals have an incubation period with the exposed group being less infectious than the infected group. Thus, in this study, we introduce susceptible contact rate,

β_1 of disease from exposed group (Exposed individuals can be infectious) in addition to susceptible contact rate, β_2 of disease from an infective group. Since vaccination is considered in most cases as an effective and safe strategy against childhood diseases, the development of a framework that would predict the optimal vaccine coverage level needed to prevent the spread of these diseases is crucial.

1.3 Justification

The study focuses on the temporal dynamics of childhood diseases in a varying population size. Childhood diseases are endemic and so many children under-five years are born highly susceptible, especially in regions with weak economies and poor healthy policies and the successes of vaccination as a preventive strategiy have been a boon to mankind. Low levels of vaccine coverage level among children with an inconsistent supply of preventive vaccines are considered to be the main reasons that contribute to the re-emergence of many childhood diseases. Childhood diseases remain a major public health problem and needs informed policy making in the healthy sector on intervention strategies including optimizing vaccine coverage in the population. In view of the above, the development of a framework that would predict the optimal vaccine coverage needed to prevent the spread of these diseases is crucial. This study formulates a mathematical model that can be used to analyze most childhood diseases effectively. It contributes to the field of mathematical epidemiology since it modifies the SEIR model to include more realistic variables. It develops a framework that would predict the optimal vaccine coverage level needed to prevent the spread of these diseases. It also acts as a basis for further research by students and researchers on modelling childhood diseases.

1.4 Objectives

1.4.1 General objective

The main objective of this study is to examine and analyze a SEIR model that monitors the temporal dynamics of a childhood disease in the presence of preventive vaccine.

1.4.2 Specific objectives:

1. To propose a SEIR epidemic model for childhood diseases.
2. To determine the existence and stabilities of the disease-free equilibrium point and endemic equilibrium point.
3. To apply Adomain Decomposition method and compare its results with the classical Runge-Kutta method to determine its effectiveness in describing childhood disease transmission dynamics.
4. To perform a sensitivity analysis of the model variables to determine the effect of each parameter on the control or die out of the childhood disease.

1.5 Limitations of the study

1. Uncertainty in the parameters since the incubation period varies
2. The SEIR model does not work with all infectious diseases.
3. In this study, we only consider the temporal transmission dynamics of the childhood diseases in a community with varying population thus the spatial diffusion spread of disease is not considered in this study.

1.6 Scope of the Thesis

Chapter one has an introduction to a mathematical role in epidemiology modeling and SEIR model. We give an overview of the research problem, justification and the objectives of this thesis. In **Chapter two,** we present a literature review of some epidemic mathematical models. We present

the most important contributions on modeling childhood diseases and other infectious diseases, and some methods of qualitative and quantitative analysis and interpretation. Also, we present and discuss some research work related to incorporating vaccination and treatment into models. In **Chapter three,** we provide some mathematical concepts that are used in this thesis. We highlight some definitions, theorems and notation about dynamical systems and stability analysis, and related theories which analyze such systems. We present some computations using the semi-numerical Adomain Decomposition method and a pure numerical Fourth order Runge-Kutta integration method applied on the normalized system of non-linear differential equations. We also display a bifurcation diagram by using Maple (Shahin (2014)). In **Chapter four,** we present and discuss the graphical and tabular computational results. **Chapter five** is devoted to conclusions and recommendations of the study.

In the next chapter, we highlight some of the recent studies that have been conducted on childhood infectious diseases.

Chapter 2

Literature Review

2.1 Introduction

Childhood diseases have become a serious health problem globally and researchers have focused their attention on developing mathematical models that play a significant role in describing the temporal dynamics of infectious diseases and understanding how vaccination programs can be used to control the spread of childhood diseases. A number of studies have been conducted to highlight the intervention strategies and control of the infectious diseases.

2.2 Literature relevant to this study

Moghadas and Gumel (2003) presented a modified SEIR model for childhood diseases with waning immunity. A qualitative and quantitative study for the deterministic model was done. Using the Jacobian approach and Routh-Hurwitz criteria it was established that the disease-free and endemic equilibrium are locally asymptotically stable for $R_0 < 1$ and $R_0 > 1$ respectively. Construction of a suitable Lyapunov function for the disease-free equilibrium was done and shows that the global stability was asymptotic. The local and global stability analysis confirmed that the disease free is stable given that the vaccination coverage level exceeds the critical point, p_c. A robust semi-explicit second-order finite difference method was used to construct approximate solutions in the SEIR model. Numerical simulations based on the estimated parameter values proved that the numerical scheme

was unconditionally convergent to the disease-free equilibrium when $R_0 < 1$ and converging to the endemic point when $R_0 > 1$.

Makinde (2007) presented a SIR model for childhood diseases with varying population size using constant vaccination strategy. A qualitative study was done to analyze the stability of the model. Using the Jacobian approach it was found that the disease-free equilibrium point is locally asymptotically stable if the reproduction number $R_v < 1$ and global stability determined using the Bendixon-Dulac argument for $R_v < 1$. Then the SIR model's endemic equilibrium was determined to be locally asymptotically stable when reproduction number $R_v > 1$. The analysis revealed that there is a critical vaccination proportion $p_c = {}^{(\beta-\gamma-\pi)}/_{\beta}$ above which the disease-free equilibrium is stable i.e. $p > p_c$. To successfully prevent disease, the vaccination proportion should be large enough. Adomain Decomposition method was used to construct approximate non-perturbative solutions of the differential equations developed in the SIR model. The numerical simulations were performed using a set of reasonable parameter values that considered presence and absence of infection, vaccination reproduction number less than unity and greater than unity and also used vaccination proportion less and greater than critical value. The results suggest that vaccination has a positive impact on the decrease of the number of infectives, an increase in the recovered individuals and a decrease in the number of susceptible. The use of varying population size is realistic and assures that the study is feasible in any given changing population. However, the study failed to consider deaths due to fatal childhood diseases such measles, Influenza etc. A plausible model for a disease that may be fatal needs to consider deaths due to diseases. The exposed individuals, when considered, can push such models to further realism.

Arafa et al. (2012) presented a fractional order SIR model for childhood diseases with constant vaccination strategy. Fractional-order was introduced to derive the SIR model. The analysis of the model showed that there is a critical vaccination proportion p_c above which the disease-free equilibrium is stable. To successfully prevent disease, the vaccination proportion should be large enough ($p > p_c$). Homotopy Analysis Method was implemented to examine the impact of vaccination on the transmission dynamics of a childhood disease described by the fractional SIR model. The results achieved by HAM are compared with the classical fourth order Runge–Kutta method to gauge its effectiveness. The numerical simulations were performed using a set of reasonable parameter values.

The results suggest that the disease will persist within the population if the vaccination coverage level is below a certain threshold.

Makinde (2009) presented a mathematical model that described the dynamics of infectious diseases with waning immunity. A qualitative study was done to analyze stability of the model. It was found that the disease-free equilibrium point is locally and globally asymptotically stable if the reproduction number $R_v < 1$. Then the SIR model's endemic equilibrium is locally asymptotically stable when reproduction number is $R_v > 1$. The analysis reveals that disease eradication depends on vaccination coverage level as well as the efficacy of the vaccine. To successfully prevent disease, the vaccination proportion should be large enough. Adomain decomposition method coupled with Padé approximation and He's Variational iteration method was used to construct approximate non-perturbative solutions of the non-linear system of equations developed in the SIV model. The numerical simulations were performed using a set of reasonable parameter values. The results suggested the reliability and efficacy of the non-pertubative methods. In addition, the simulations supported the analytic findings and thus illustrated possible behavior scenarios of the SIV model.

Ongau et al. (2014) presented a mathematical model of the control of measles by vaccination: A case of Kisii County, Kenya. A qualitative study was done to analyze stability of the SEIR model. It was found that the disease free equilibrium point is locally and globally asymptotically stable if the reproduction number $R_v < 1$. Routh-Hurwitz criteria of stability was used to determine the SEIR model's endemic equilibrium local stability. The analysis reveals that elimination of measles is by exceeding the level of mass vaccination. To successfully prevent disease, the mass vaccination should be large enough. The numerical simulations were performed using a set of reasonable parameter values obtained from research institutions. The results suggest that mass vaccination has a positive impact on the decrease of the number of infected individual and Herd Immunity for measles in the county was found to be 93.75%.

Bakare (2015) studied optimal control of vaccination and treatment for a SIR epidemic model. A qualitative study was undertaken to establish the existence and stability of equilibria. It was found that in the absence of infected immigrants the disease-free equilibrium exists and is locally and globally stable. Sensitivity indices of the basic reproduction number to the parameters in the model were computed which revealed the most sensitive parameters to be the transmission rate and the recruitment

rate. Optimal control analysis was done. The existence, uniqueness and necessary conditions of the control were established. They characterized the controls and obtained the optimality system. The resulting optimality system numerically solved. The optimal control analysis revealed that a combination of vaccination and treatment strategies were crucial in driving infectious diseases like measles and other childhood diseases towards eradication.

Yang et al. (2010) presented a SIRV epidemiological model with vaccination and varying population. A qualitative study was done to determine the existence and stability of equilibria. The study confirmed the existence and local stability of Disease free and endemic equilibrium. The global dynamics of the model were investigated. The main results shows that the global dynamics of the model with the proportionate transformed system, the correlation between the fractions and population size in disease eradication and persistence and the impact of different vaccination strategies on the control of disease. Numerical simulations done for the system revealed that the disease eradication occurs when the vaccination rate $\phi > \phi_{1c}$ and disease persists when $\phi < \phi_{1c}$.

Sun and Hsieh (2010) studied a SEIR model with vaccination strategy that incorporates distinct incidence rates for infected and exposed individuals. A qualitative study revealed that that the diseases free equilibrium is locally and globally asymptotically stable if $R_0 < 1$. This study took into account the effectiveness of the vaccine. It also revealed that the model system has a unique endemic equilibria which is locally stable. By using a suitable Lyapunov function and applying the Lasalle's invariance set theorem , they proved the global stability for disease-free equilibrium. The compound matrix theory was used to provide the sufficient conditions for global stability of the endemic equilibrium. Direct numerical simulations using estimated parameters revealed that there is a periodic solution, when the model has three equilibrium points.

Zhang et al. (2015) studied the global dynamics of a SEIR epidemic model with discontinuous treatment strategies. A qualitative study was done to determine the feasibility region, positivity of model variables and stability of equilibria. The study revealed that the disease free and the endemic equilibrium are locally asymptotically stable if $R_0 \leq 1$ and $R_0 > 1$ respectively. Lyapunov stability theory was used to establish the global stability of Disease free equilibrium, together with the basic reproductive number, R_0 which proved to be a sharp threshold value that determines the dynamics of the model. Numerical simulation using estimated parameter values revealed that strengthening

treatment measures after infective individuals reach some level is significant to disease control. The study also reveals that a die out of the disease in a finite time contrary to the continuous treatment in SEIR model which leads to die out of disease asymptotically.

Concluding this chapter we can observe that the models of Makinde (2007); Arafa et al. (2012) and Makinde (2009) are very similar to the model we present in this study. We extend the work of Makinde Makinde (2007) from a SIR model to a SEIR model and study the temporal dynamics. However, the mathematical model we formulate in this research thesis advances from previous studies by incorporating an infectious exposed group of children and we compare the effectiveness of the Adomain Decomposition method with the classical fourth order Runge-Kutta integration method to gauge its effectiveness in describing the temporal dynamics of childhood diseases with preventive vaccines as a control strategy. The main question to be addressed is whether vaccination coverage can influence disease spreading and inform health authorities on prevention and eradication strategies.

In the next chapter, we discuss the methodologies used in analysis and discussion of the governing model.

Chapter 3

Methodology

3.1 Introduction

In this Section, we highlight some basic mathematical tools and methods. The definitions and theory from dynamical systems associated with monitoring and analysis of the temporal dynamics of epidemiological modeling that are used in this study are presented. For more information, we refer the reader to the books of Martcheva (2015), Perko (2013),Allen et al. (2008), Brauer et al. (2001) and Boyce et al. (2001).

Definition 3.1. (**Dynamical Systems**) A dynamical system is a system which evolves with time and satisfies the following:

(i) Its future depends only on the past phenomena or the present time.

(ii) For a deterministic system ,each given initial condition at a fixed time (i.e. at a present instant) is going to correspond at each later time to one and only one possible future state (Perko (2013); Boyce et al. (2001)).

Definition 3.2. (**Basic Reproduction Number**) The basic reproduction number is defined as the average number of Secondary infections generated by one infective introduced into a completely susceptible population to the disease (Yang et al. (2010); Martcheva (2015)).

Definition 3.3. (**Equilibrium points**) An equilibrium point of a system of differential equations $X^{'} = f(s, e, i, r)$ ($f(s, e, i, r)$ is a differentiable function) is a point (s_e, e_e, i_e, r_e) such that $s(t) = s_e$,$e(t) = e_e$,$i(t) = i_e$ and $r(t) = r_e$ is a constant solution of the system of differential equations i.e. (s_e, e_e, i_e, r_e) is a point at which $s^{'}(t) = 0, e^{'}(t) = 0, i^{'}(t) = 0$ and $r^{'}(t) = 0$ (Perko (2013); Boyce et al. (2001)).

Definition 3.4. (**Stability of equilibrium points**) Suppose we consider a system of differential equations $X^{'} = f(s, e, i, r)$ with an equilibrium point (s_e, e_e, i_e, r_e). The equilibrium point is said to be stable when a small change or perturbations of the populations of the equilibrium values, results in the populations remain in the neighborhood of equilibrium points as time increases (Perko (2013); Boyce et al. (2001)).

Definition 3.5. (**Asymptotic stability**) A critical point $x^* = (s_e, e_e, i_e, r_e)$ is said to be **asymptotically stable** if it is stable and if there exists a ξ_0, with $0 < \xi_0 < \xi$, such that if a solution $x = f(t, s, e, i, r)$ satisfies:
$\| f(0) - x^* \| < \xi_0$, then $\lim_{t\to\infty} f(t, s, e, i, r) = x^*$ (Boyce et al. (2001)).

Definition 3.6. (**Global asymptotic stability**) An equilibrium point x^* is said to be **globally asymptotically stable** if it is asymptotically stable and that $\lim_{t\to\infty} f(t, s, e, i, r) = x^*$ for almost all conditions, not just those initial conditions that are close to the critical point (Perko (2013); Boyce et al. (2001)).

Definition 3.7. (**Bifurcation Diagram**) This is a way to study how a differential equation depends on a parameter. In this study, we will study how the governing differential equations of model depend on the parameter, R_v, the vaccination reproduction number (Perko (2013)).

Definition 3.8. (**Normalized forward sensitivity index**) The normalized forward sensitivity index of a variable, P, which depends differentially on a parameter, K, defined as: $Z_K^P = \frac{\partial P}{\partial K} \times \frac{K}{P}$ (Chitnis et al. (2008); Bakare (2015)).

The sensitivity indices serve as determinants of the significance of each parameter in the dynamics and prevalence of the diseases. They measure the change in model variables when a parameter changes (Chitnis et al. (2008)).

Routh-Hurwitz criteria

Theorem 3.9. *Suppose we consider the kth-degree polynomial with real constant coefficients i.e.* $P(\lambda) = \lambda^k + a_1\lambda^{k-1} + ... + a_{k-1}\lambda + a_k$

then, we define k Hurwitz matrices using the coefficients of the characteristic polynomial,

$$H_1 = (a_1),\ H_2 = \begin{pmatrix} a_1 & 1 \\ a_3 & a_2 \end{pmatrix} \text{ and } H_k = \begin{pmatrix} a_1 & 1 & 0 & 0 & . & . & . & 0 \\ a_3 & a_2 & a_1 & 1 & . & . & . & 0 \\ a_5 & a_4 & a_3 & a_2 & . & . & . & 0 \\ . & . & . & . & & & & . \\ . & . & . & . & . & . & . & . \\ . & . & . & . & & & & . \\ 0 & 0 & 0 & 0 & . & . & . & a_k \end{pmatrix},$$

where $a_i = 0$ if $i > k$. *All roots of the polynomial* $P(\lambda)$ *are negative or will have negative real part iff determinants of all Hurwitz matrices are positive i.e.* $DetH_i > 0, i = 1, ..., k$ (Boyce et al. (2001); Martcheva (2015)).

Lyapunov stability theorem

Theorem 3.10. *If a function* $V(s, e, i)$ *is globally positive definite and radially unbounded and its time derivative is globally negative,*

i.e. $V^{'}(s, e, i) < 0$, *for all* $(s, e, i) \neq (s^*, e^*, i^*)$ *, then the equilibrium* (s^*, e^*, i^*) *is globally stable (see Boyce et al. (2001); Martcheva (2015))*

Krasovkii-Lasalle Theorem

Theorem 3.11. *We consider the autonomous system* $X^{'} = f(s, e, i)$, *where* $x^* = (s^*, e^*, i^*)$ *is an equilibrium i.e.* $f(s^*, e^*, i^*) = 0$. *We suppose there exists a continuously differentiable function* $V : \mathbb{R}^n \to \mathbb{R}$ *and that this function is positive definite in the entire space and radially unbounded and satisfying,*

$V^{'}(s, e, i) \leq 0$, $\forall t$ *and all* $(s, e, i) \in \mathbb{R}^3$.

Then, the invariant set $\Gamma = \{(s, e, i) \in \mathbb{R}^3 : V^{'} = 0\}$,

if Γ *contains only the equilibrium* x^*, *then the equilibrium is globally stable (Martcheva (2015); Boyce et al. (2001)).*

3.2 Model Derivation

A SEIR model is formulated and analyzed to study the temporal transmission dynamics of childhood diseases in a varying population size. The model has four epidemiological compartments: The susceptible S, an exposed group E, an infected group I, and a removed group R, denoting the vaccinated and recovered group who poses permanent immunity to the disease. This SEIR model assumes the efficacy of the vaccine to be 100% and death rates μ due to causes other than the disease in the classes remain unequal to births, so that a population size N is realistically not constant. Citizens are born into the population with the proportion of susceptible that are vaccinated as P (with $0 < P < 1$) and assume the rest are susceptible. We assume the population is uniform and mixes homogeneously and the vaccination rate at birth each year as π . A susceptible individual will move into the exposed group through contact with an infected individual, approximated by an average contact rate β_2 or through contact with an exposed individual, approximated by an average contact rate β_1 . An exposed individual Progresses from exposed to the infective group at a rate δ. An infected individual progresses from infected to the recovered group due to treatment at a rate γ. We also assume α to be death rate due to disease infection. The resulting differential equations for the model are

$$\begin{cases} \frac{dS}{dt} = (1-P)\pi N - (\frac{\beta_1 E + \beta_2 I}{N})S - \mu S, \\ \frac{dE}{dt} = (\frac{\beta_1 E + \beta_2 I}{N})S - (\delta + \mu)E, \\ \frac{dI}{dt} = \delta E - (\alpha + \gamma + \mu)I, \\ \frac{dR}{dt} = P\pi N + \gamma I - \mu R, \end{cases} \tag{3.1}$$

satisfying $S(0) = S_{0,}E(0) = E_{0,}I(0) = I_0$and $R(0) = R_0$,

with the relation $N = S + E + I + R$ and assuming $\mu, \beta_{1,}\beta_{2,}\pi, \alpha, \delta, \gamma$ are all positive constant parameters.

Adding the equations of model system (3.1) we have,

$$\frac{dN}{dt} = (\pi - \mu)N - \alpha I. \tag{3.2}$$

We are now dealing with a varying population size, as demonstrated by Makinde (2007), with deaths due to fatal diseases. A flow chart for the process is drawn in Fig.(3.1).

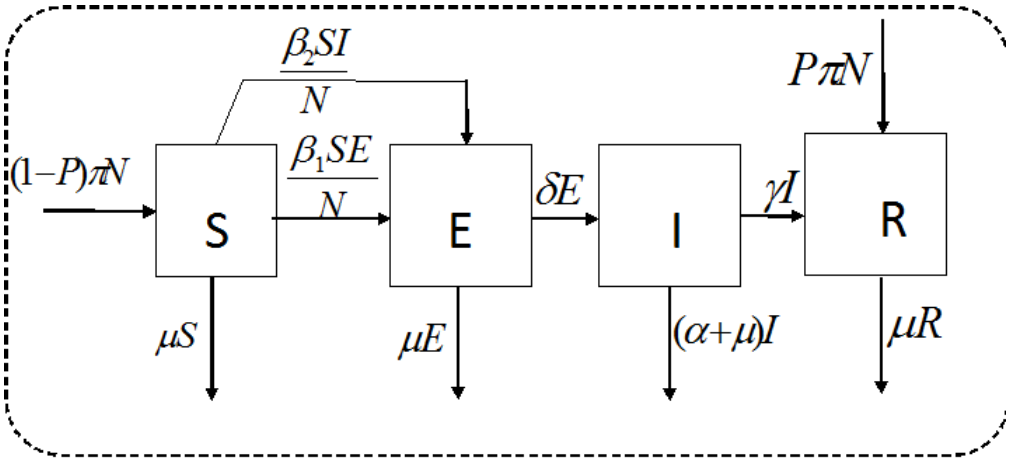

Figure 3.1: SEIR model flow chart

For simplicity we scaled the compartments by population N using the new variables, $s = {}^{S}/_{N}, e = {}^{E}/_{N}, i = {}^{I}/_{N}$ and $r = {}^{R}/_{N}$.

The population is now normalized with $s + e + i + r = 1$ and we formulate the new system,

$$\begin{cases} \frac{ds}{dt} = (1-P)\pi - (\beta_1 e + \beta_2 i)s - \pi s + \alpha i s, \\ \frac{de}{dt} = (\beta_1 e + \beta_2 i)s - (\delta + \pi)e + \alpha i e, \\ \frac{di}{dt} = \delta e - (\alpha + \gamma + \pi)i + \alpha i^2, \\ \frac{dr}{dt} = P\pi + (\gamma + \alpha)i - \pi r. \end{cases} \tag{3.3}$$

We observe that the variable r does not appear in the first three equations of system (3.3). Thus, we can analyze the system qualitatively by studying the subsystem,

$$\begin{cases} \frac{ds}{dt} = (1-P)\pi - (\beta_1 e + \beta_2 i)s - \pi s + \alpha i s, \\ \frac{de}{dt} = (\beta_1 e + \beta_2 i)s - (\delta + \pi)e + \alpha i e, \\ \frac{di}{dt} = \delta e - (\alpha + \gamma + \pi)i + \alpha i^2, \end{cases} \tag{3.4}$$

where r can be determined from $r = 1 - s - e - i$ or $\frac{dr}{dt} = p\pi + (\gamma + \alpha)i - \pi r$.

Remark. We remark that $\beta_1 < \beta_2$ so that the exposed group is less infectious than the infected group. This is natural since the infected individuals are already showing the symptoms of the disease hence they are more infectious than exposed group.

3.3 The Model Properties

3.3.1 Feasibility Region

In this section, we seek a region in which the solutions of the model system are non-negative and uniformly bounded.

Let $(S, E, I, R) \in \mathbb{R}^4_+$ be any solution with initial conditions $S(0) = S_{0,}E(0) = E_{0,}I(0) = I_0, R(0) = R_0 \geq 0$,

with relation $N = S + E + I + R$.

Adding the differential equations of the system (3.1) , we have

$$\frac{dN}{dt} = (\pi - \mu)N - \alpha I. \tag{3.5}$$

or

$$\frac{dN}{dt} \leq (\pi - \mu)N. \tag{3.6}$$

On integrating equation (3.6), we have

$$N \leq N_0 \exp(\pi - \mu)t, \tag{3.7}$$

where N_0 is the initial population computed at the initial conditions $(S_{0,}E_{0,}I_0, R_0)$.

Hence, as $t \to \infty$ in equation (3.7), the population size N is such that $0 \leq S + E + I + R \leq N_0 \exp(\pi - \mu)t$ or $0 \leq s + e + i + r \leq 1$.

This implies, all the feasible solutions of the system (3.1) in $\mathbb{R}^4_+$ are confined in the region,

$$\Gamma = \left\{ s, e, i, r \in \mathbb{R}^4_+ : 0 \leq s + e + i + r \leq 1 \right\}. \tag{3.8}$$

Thus, we have the following result.

Theorem 3.12. *The solutions of the governing model system (3.3) are uniformly bounded in a set* Γ*, where* Γ *is defined in equation (3.8).*

Lemma 3.13. *The region* $\Gamma \subset \mathbb{R}^4_+$ *is positively invariant with respect to the model system (3.1) with initial conditions in* $\mathbb{R}^4_+$.

3.3.2 Positivity of Model Variables

In this subsection, we seek a model that is biologically and mathematically feasible. Thus, we prove that all the model variables and parameters are non-negative at all time since the model deals with a population as demonstrated by (May et al. (1979)).

Lemma 3.14. *Let the initial conditions be* $\{\{s(0), e(0), i(0), r(0) \geq 0\} \in \mathbb{R}^4_+\}$ *then, the solution set* $\{s(t), e(t), i(t), r(t)\}$ *of the normalized model system (3.3) is positive for all* $t > 0$.

Proof. From the differential equations of system (3.3) we have,

$$\frac{ds}{dt} = (1 - P)\pi - (\beta_1 e + \beta_2 i)s - \pi s + \alpha i s, \tag{3.9}$$

or

$$\frac{ds}{dt} \geq -(\beta_1 e + \beta_2 i)s - \pi s, \tag{3.10}$$

$$\frac{ds}{s} \geq (-(\beta_1 e + \beta_2 i + \pi))\, dt, \tag{3.11}$$

on integrating equation (3.11), we have

$$\ln s(t) \geq -(\pi t + \beta_1 \int_0^t e\, dt + \beta_2 \int_0^t i\, dt), \tag{3.12}$$

$$s(t) \geq K \exp(-(\pi t + \beta_1 \int_0^t e\, dt + \beta_2 \int_0^t i\, dt)), \tag{3.13}$$

when $t = 0$ we obtain,

$$s(t) \geq s(0) \exp(-(\pi t + \beta_1 \int_0^t e\, dt + \beta_2 \int_0^t i\, dt)) \geq 0, \tag{3.14}$$

since

$$(\pi t + \beta_1 \int_0^t e\, dt + \beta_2 \int_0^t i\, dt) > 0$$

.

$$\frac{de}{dt} = (\beta_1 e + \beta_2 i)s - (\delta + \pi)e + \alpha i e, \tag{3.15}$$

or

$$\frac{de}{e} \geq -(\delta + \pi)\, dt, \tag{3.16}$$

on integrating equation (3.16) yields,

$$\ln e(t) \geq -(\delta + \pi)t, \tag{3.17}$$

$$e(t) \geq C\exp(-(\delta+\pi)t), \tag{3.18}$$

at $t = 0$ we have,

$$e(t) \geq e(0)\exp(-(\delta+\pi)t) \geq 0, \tag{3.19}$$

since

$$(\delta+\pi)t > 0$$

.

$$\frac{di}{dt} = \delta e - (\alpha+\gamma+\pi)i + \alpha i^2, \tag{3.20}$$

or

$$\frac{di}{i} \geq -(\alpha+\gamma+\pi)\,dt, \tag{3.21}$$

on integrating equation (3.21) yields,

$$\ln i(t) \geq -(\alpha+\gamma+\pi)t, \tag{3.22}$$

$$i(t) \geq D\exp(-(\alpha+\gamma+\pi)t), \tag{3.23}$$

at $t = 0$ we have,

$$i(t) \geq i(0)\exp(-(\alpha+\gamma+\pi)t) \geq 0, \tag{3.24}$$

since

$$(\alpha+\gamma+\pi)t > 0$$

.

$$\frac{dr}{dt} + \pi r = P\pi + (\gamma+\alpha)i, \tag{3.25}$$

The integrating factor $\varphi(t) = \exp(\pi t)$, multiplying both sides of equation (3.25) by the integrating factor gives,

$$\frac{d}{dt}(r\varphi(t)) = (P\pi + (\gamma+\alpha)i)\varphi(t), \tag{3.26}$$

on integrating we have,

$$r(t) = Pt + (\gamma+\alpha)\frac{\int_0^t i\varphi(t)\,dt}{\varphi(t)} \geq 0, \tag{3.27}$$

since

$$Pt + (\gamma + \alpha)\frac{\int_0^t i\varphi(t)\,dt}{\varphi(t)} > 0$$

.

Hence, $s(t), e(t), i(t)$ and $r(t)$ are positive for all $t \geq 0$. □

3.4 Equilibrium Points and Stability Analysis

To undertake stability analysis of the model, we first compute the equilibrium points of the normalized system (3.3) of differential equations. We determine the Disease-free equilibrium point (i.e. in absence of infection) and Endemic equilibrium (i.e. in presence of infection). The study considered a sub-system (3.4).

3.4.1 Disease-free equilibrium (DFE)

At the disease-free equilibrium, we consider the case when the disease dies out which implies that $e = 0, i = 0$, the model has disease-free equilibrium, where $s = s_0, e_0 = 0$ and $i_0 = 0$ which is obtained by setting the sub-system to zero. i.e. $\frac{ds}{dt} = \frac{de}{dt} = \frac{di}{dt} = 0$,

$$\begin{cases} (1-P)\pi - (\beta_1 e_0 + \beta_2 i_0)s_0 - \pi s_0 + \alpha i_0 s_0 = 0, \\ (\beta_1 e_0 + \beta_2 i_0)s_0 - (\delta + \pi)e_0 + \alpha i_0 e_0 = 0, \\ \delta e_0 - (\alpha + \gamma + \pi)i_0 + \alpha i_0^2 = 0. \end{cases} \tag{3.28}$$

Substituting $e_0 = 0$ and $i_0 = 0$ into equations in system(3.28) we have,

$(1-P)\pi - \pi s_0 = 0$, which implies $s_0 = 1 - P$.

Thus, the disease-free equilibrium solution $E^0 = (s_0, e_0, i_0) = (1 - P, 0, 0)$.

3.4.2 Vaccination Reproduction number

This is defined as the average number of secondary infections generated by an infective individual introduced into a susceptible population to the disease with vaccination as a control measure (Van den Driessche and Watmough (2002)). This is the threshold value that determines the spread or die out of

childhood diseases in a susceptible population (Martcheva (2015)).

We compute the vaccination reproduction number, R_v using the Jacobian approach at the disease-free equilibrium and we pose the condition that all the eigenvalues of the corresponding characteristic equation must have negative real parts i.e. trace of Jacobian $(TrJ < 0)$ and determinant of Jacobian $(DetJ > 0)$ (Martcheva (2015); Boyce et al. (2001)). The vaccination reproduction number serves as a threshold parameter that predicts whether an infection dies out or keeps persisting in the community (Martcheva (2015)). The Jacobian of the governing model system is given by,

$$J = \begin{bmatrix} a & -\beta_1 s & (\alpha - \beta_2)s \\ \beta_1 e + \beta_2 i & b & \beta_2 s + \alpha e \\ 0 & \delta & c \end{bmatrix}, \tag{3.29}$$

where $a = -(\beta_1 e + \beta_2 i) - \pi + \alpha i$, $b = \beta_1 s - (\delta + \pi) + \alpha i$ and $c = -(\alpha + \gamma + \pi) + 2\alpha i$.

The Jacobian of the system at the disease-free equilibrium $E^0 = (1 - P, 0, 0)$ is given by,

$$J_{E^0} = \begin{bmatrix} -\pi & -\beta_1(1-P) & (\alpha - \beta_2)(1-P) \\ 0 & \beta_1(1-P) - (\delta + \pi) & \beta_2(1-P) \\ 0 & \delta & -(\alpha + \gamma + \pi) \end{bmatrix}. \tag{3.30}$$

We consider $\mid J_{E^0} - \lambda I \mid = 0$ which yields,

$$(-\pi - \lambda) \begin{vmatrix} \beta_1(1-P) - (\delta + \pi) - \lambda & \beta_2(1-P) \\ \delta & -(\alpha + \gamma + \pi) - \lambda \end{vmatrix} = 0, \tag{3.31}$$

which implies $\lambda_1 = -\pi$,

or

$$\lambda^2 + [(\alpha + \gamma + \pi) - \beta_1(1-P) + (\delta + \pi)]\lambda - \beta_1(\alpha + \gamma + \pi)(1-P) +$$

$$(\delta + \pi)(\alpha + \gamma + \pi) - \delta\beta_2(1-P) = 0. \tag{3.32}$$

where

$$TrJ_{E_0} = (\alpha + \gamma + \pi) - \beta_1(1-P) + (\delta + \pi)$$

and

$$DetJ_{E_0} = -\beta_1(\alpha+\gamma+\pi)(1-P) + (\delta+\pi)(\alpha+\gamma+\pi) - \delta\beta_2(1-P).$$

We now apply the conditions that are sufficient to guarantee the eigenvalues of equation (3.32) to have negative real parts i.e.($TrJ_{E^0} < 0$) and $DetJ_{E_0} > 0$). Using $DetJ_{E_0} > 0$ gives,

$$-\beta_1(\alpha+\gamma+\pi)(1-P) + (\delta+\pi)(\alpha+\gamma+\pi) - \delta\beta_2(1-P) > 0. \tag{3.33}$$

This condition results to the threshold parameter R_v given by,

$$R_v = \frac{\beta_1(\alpha+\gamma+\pi)(1-P) + \delta\beta_2(1-P)}{(\delta+\pi)(\alpha+\gamma+\pi)}. \tag{3.34}$$

Using the Jacobian approach, the conditions for $Re\lambda < 0$ are $TrJ_{E^0} < 0$ and $DetJ_{E_0} > 0$ (Martcheva (2015)). It is easy to see that the condition $R_v < 1$, clearly implies that $DetJ_{E_0} > 0$. We note that if $R_v > 1$, then

$$\beta_1(\alpha+\gamma+\pi)(1-P) + \delta\beta_2(1-P) > (\delta+\pi)(\alpha+\gamma+\pi) \Rightarrow DetJ_{E_0} < 0 \tag{3.35}$$

Hence, the necessary and sufficient conditions for the eigenvalues to have negative real parts are satisfied when $R_v < 1$. Thus, the Jacobian approach (Boyce et al. (2001); Martcheva (2015)) shows that the disease-free equilibrium is locally asymptotically stable if $R_v < 1$ and unstable if $R_v > 1$. Hence, we have achieved the following result.

Lemma 3.15. *If $R_v < 1$, the disease-free equilibrium of the model subsystem (3.4) is locally asymptotically stable and unstable if $R_v > 1$.*

From equation (3.34) we can obtain the critical vaccination proportion (P_c) when $R_v = 1$ given by,

$$P_c = 1 - \frac{1}{R_0}, \tag{3.36}$$

where

$$R_0 = \frac{\beta_1(\alpha+\gamma+\pi) + \delta\beta_2}{(\delta+\pi)(\alpha+\gamma+\pi)},$$

R_0 is the basic reproductive number.

This implies, $R_v < 1 \Leftrightarrow P > P_c$ and $R_v > 1 \Leftrightarrow P < P_c$.

3.4.3 Global stability of disease free equilibrium

We note that there are no established procedures for calculating a Lyapunov function, and often finding a Lyapunov function is tedious and tricky when using trial and error approach (Martcheva (2015)). We consider the following Lyapunov function:

$$V = (\alpha + \gamma + \pi)e + \beta_2 i. \tag{3.37}$$

We note that V is positive definite since $V = 0$ at E^0 and $V > 0$ otherwise. Moreover, V is also radially unbounded. Therefore, V is a Lyapunov function. Its derivative is given by,

$$V^{'} = (\alpha + \gamma + \pi)[(\beta_1 e + \beta_2 i)s - (\delta + \pi)e + \alpha i e] + \beta_2[\delta e - (\alpha + \gamma + \pi)i + \alpha i^2], \tag{3.38}$$

or

$$V^{'} = \beta_1(\alpha + \gamma + \pi)es + \beta_2(\alpha + \gamma + \pi)is - (\delta + \pi)(\alpha + \gamma + \pi)e + (\alpha + \gamma + \pi)\alpha ie\}+$$

$$\beta_2\delta e - \beta_2(\alpha + \gamma + \pi)i + \beta_2\alpha i^2, \tag{3.39}$$

or

$$V^{'} = \{\beta_1(\alpha + \gamma + \pi)s + \beta_2\delta - (\delta + \pi)(\alpha + \gamma + \pi)\}e + \{\beta_2(\alpha + \gamma + \pi)s+$$

$$(\alpha + \gamma + \pi)\alpha e - \beta_2(\alpha + \gamma + \pi) + \beta_2\alpha i\}i, \tag{3.40}$$

or

$$V^{'} \leq e\{\frac{R_v}{(1-P)} - 1\}(\delta + \pi)(\alpha + \gamma + \pi) \leq 0, \tag{3.41}$$

with $P \neq 1$and for all $(e, i) \geq 0$.

$$\Rightarrow V^{'} < 0, \tag{3.42}$$

if

$$R_v < 1 \tag{3.43}$$

.

Clearly, $V^{'} = 0$ if $e = 0, i = 0$. Therefore, the maximum invariant set contained in the set $\{(s, e, i) \in \Gamma : V^{'} = 0\}$ is reduced to the disease free equilibrium. Being in a compact invariant set, the global asymptotic stability of E^0 follows from Lassalle's invariance principle (Boyce et al. (2001); Martcheva (2015); Shuai and van den Driessche (2013); Brauer et al. (2001)). Therefore, the following result was achieved.

Lemma 3.16. *If $R_v < 1$ the Disease-free equilibrium $E^0 = (1 - P, 0, 0)$ is Globally asymptotically stable on the closed set Γ.*

3.4.4 Existence of endemic equilibrium

Theorem 3.17. *Suppose that $R_v > 1$. Then the model subsystem (3.4) has a unique endemic equilibrium $E^u = (s^*, e^*, i^*)$.*

Proof. In the presence of infection $(e \neq 0, i \neq 0)$ the model has endemic equilibrium $E^u = (s^*, e^*, i^*)$ obtained by setting the subsystem (3.4) to zero.

$$\begin{cases} (1-P)\pi - (\beta_1 e^* + \beta_2 i^*)s^* - \pi s^* + \alpha i^* s^* = 0, \\ (\beta_1 e^* + \beta_2 i^*)s^* - (\delta + \pi)e^* + \alpha i^* e^* = 0, \\ \delta e^* - (\alpha + \gamma + \pi)i^* + \alpha i^{*2} = 0, \end{cases} \tag{3.44}$$

with $s^* > 0$,$e^* > 0$ and $i^* > 0$.

The variables (s^*, e^*) can be determined uniquely as follows:

From the third equation of (3.44) we have,

$$e^* = \frac{(\alpha + \gamma + \pi)i^* - \alpha i^{*2}}{\delta} \tag{3.45}$$

and the second equation of (3.44) yields,

$$s^* = \frac{[(\delta + \pi) - \alpha i^*][(\alpha + \gamma + \pi) - \alpha i^*]}{\beta_1(\alpha + \gamma + \pi) + \delta\beta_2 - \alpha\beta_1 i^*}. \tag{3.46}$$

Adding equations of subsystem (3.44) yields,

$$(1-P)\pi - \pi(s^* + e^* + i^*) + \alpha i^* s^* + \alpha i^* e^* + \alpha i^{*2} - \gamma i^* - \alpha i^* = 0, \tag{3.47}$$

or

$$(\pi - \alpha i^*)(1 - s^* - e^* - i^*) = \gamma i^* + P\pi, \tag{3.48}$$

which results to the following range of i^*,

$$0 < i^* < \min\{1, \frac{\pi}{\alpha}\}. \tag{3.49}$$

We refer the reader to (Li and Muldowney (2000)) for a proof of (3.49).

Adding the first two equations of system (3.44) we have,

$$(1 - P)\pi + (-\pi + \alpha i^*)s^* + (-(\delta + \pi) + \alpha i^*)e^* = 0, \tag{3.50}$$

substituting equations (3.45) and (3.46) into equation (3.50) we obtain,

$$(1 - P)\pi + (-\pi + \alpha i^*)\frac{[(\delta + \pi) - \alpha i^*][(\alpha + \gamma + \pi) - \alpha i^*]}{\beta_1(\alpha + \gamma + \pi) + \delta\beta_2 - \alpha\beta_1 i^*} +$$

$$(-(\delta + \pi) + \alpha i^*)\frac{(\alpha + \gamma + \pi)i^* - \alpha i^{*2}}{\delta} = 0, \tag{3.51}$$

or

$$\beta_1(1 - P)\pi\alpha i^* + (\pi - \alpha i^*)[(\delta + \pi) - \alpha i^*][(\alpha + \gamma + \pi) - \alpha i^*] + \frac{1}{\delta}[\beta_1(\alpha + \gamma + \pi) + \delta\beta_2 -$$

$$\alpha\beta_1 i^*][(\delta + \pi) - \alpha i^*][(\alpha + \gamma + \pi) - \alpha i^*]i^* = [\beta_1(\alpha + \gamma + \pi) + \delta\beta_2](1 - P)\pi. \tag{3.52}$$

Dividing equation(3.52) by $\pi(\delta + \pi)(\alpha + \gamma + \pi)$ gives,

$$\frac{\beta_1(1 - P)\alpha}{(\delta + \pi)(\alpha + \gamma + \pi)}i^* + (1 - \frac{\alpha}{\pi}i^*)(1 - \frac{\alpha}{\delta + \pi}i^*)(1 - \frac{\alpha}{\alpha + \gamma + \pi}i^*) +$$

$$\frac{1}{\delta}(1 - \frac{\beta_1(1 - P)\alpha}{R_v(\delta + \pi)(\alpha + \gamma + \pi)}i^*)(1 - \frac{\alpha}{\delta + \pi}i^*)(1 - \frac{\alpha}{\alpha + \gamma + \pi}i^*)i^* = R_v. \tag{3.53}$$

We have that i^* satisfies,

$$f(i^*) = R_v \tag{3.54}$$

with

$$f(i^*) = \frac{\beta_1(1 - P)\alpha}{(\delta + \pi)(\alpha + \gamma + \pi)}i^* + (1 - \frac{\alpha}{\pi}i^*)(1 - \frac{\alpha}{\delta + \pi}i^*)(1 - \frac{\alpha}{\alpha + \gamma + \pi}i^*) +$$

$$\frac{1}{\delta}(1 - \frac{\beta_1(1 - P)\alpha}{R_v(\delta + \pi)(\alpha + \gamma + \pi)}i^*)(1 - \frac{\alpha}{\delta + \pi}i^*)(1 - \frac{\alpha}{\alpha + \gamma + \pi}i^*)i^* \tag{3.55}$$

and R_v is as defined in (3.34).

The roots of $f(i^*)$ are $i_1^* = \frac{\pi}{\alpha}$, $i_2^* = \frac{\delta+\pi}{\alpha}$, $i_3^* = \frac{\alpha+\gamma+\pi}{\alpha}$ and $i_4^* = \frac{R_v(\delta+\pi)(\alpha+\gamma+\pi)}{\beta_1(1-P)\alpha}$. We note that the roots i_2^*, i_3^* and i_4^*all lie outside $[0, \frac{\pi}{\alpha}]$ when $R_v > 1$.

We observe that

$$f(0) = 1 \tag{3.56}$$

and

$$f(\frac{\pi}{\alpha}) = \omega R_v + K > R_v, \tag{3.57}$$

where

$$\omega = \frac{\pi^2}{\alpha\delta(1-P)} + \frac{\pi(\alpha+\delta+\gamma+2\pi)}{\alpha\delta(1-P)} + \frac{1}{\delta}\frac{(\delta+\pi)(\alpha+\gamma+\pi)}{(1-P)} - \frac{\pi^3}{\alpha\delta(\alpha+\gamma+\pi)}$$

and

$$K = \frac{1}{\alpha\delta(\delta+\pi)(\alpha+\gamma+\pi)}\{\frac{\delta\pi^3\beta_2}{(\alpha+\gamma+\pi)} + \pi^2(\beta_1(\alpha+\delta+\gamma+2\pi) - \delta\pi) + \pi[\beta_1(\delta+\pi)(\alpha+\gamma+\pi) -$$

$$\alpha\delta(\alpha+\delta+\gamma+2\pi)] + \delta\beta_1(1-P)\pi\alpha - \alpha\delta[\pi(\delta+\pi) + \pi(\alpha+\gamma+\pi) + (\delta+\pi)(\alpha+\gamma+\pi)]\} + 1.$$

Also,

$$f(1) = \varpi R_v + L > R_v, \tag{3.58}$$

where

$$\varpi = \frac{\alpha^2}{\pi\delta(1-P)} + \frac{\alpha(\alpha+\delta+\gamma+2\pi)}{\pi\delta(1-P)} + \frac{\alpha}{\delta\pi}\frac{(\delta+\pi)(\alpha+\gamma+\pi)}{(1-P)} - \frac{\alpha^3}{\pi\delta(\alpha+\gamma+\pi)}$$

and

$$L = \frac{1}{\delta\pi(\delta+\pi)(\alpha+\gamma+\pi)}\{\frac{\delta\alpha^3\beta_2}{(\alpha+\gamma+\pi)} + \alpha^2(\beta_1(\alpha+\delta+\gamma+2\pi) - \delta\pi) + \alpha[\beta_1(\delta+\pi)(\alpha+\gamma+\pi) -$$

$$\alpha\delta(\alpha+\delta+\gamma+2\pi)] + \delta\beta_1(1-P)\pi\alpha - \alpha\delta[\pi(\delta+\pi) + \pi(\alpha+\gamma+\pi) + (\delta+\pi)(\alpha+\gamma+\pi)]\} + 1.$$

The above observations implies that, when $R_v > 1$, the line $f(i) = R_v$ has exactly one intersection

point $(i^*, f(i^*))$ with the graph of $f(i)$ as demonstrated by Figure 3.2. □

The endemic equilibrium satisfies the following polynomial,

$$P(i^*) = A{i^*}^4 + B{i^*}^3 + C{i^*}^2 + Di^* + E = 0, \tag{3.59}$$

where

$$A = \frac{\alpha^3}{\pi\delta(\delta+\pi)(\alpha+\gamma+\pi)}\left[\frac{\delta\beta_2}{\alpha+\gamma+\pi} - \frac{R_v(\delta+\pi)}{(1-P)}\right],$$

$$B = \frac{\alpha^2}{\delta\pi}[\frac{R_v}{1-P} + \frac{\beta_1(\delta+\alpha+\gamma+2\pi)-\delta\pi}{(\delta+\pi)(\alpha+\gamma+\pi)}],$$

$$C = \frac{\alpha}{\delta\pi}[\frac{R_v(\alpha+\delta+\gamma+2\pi)}{1-P} + \frac{\beta_1(\delta+\pi)(\alpha+\gamma+\pi)-\delta\alpha(3\pi+\delta+\alpha+\gamma)}{(\delta+\pi)(\alpha+\gamma+\pi)}],$$

$$D = \frac{\alpha}{\delta\pi}\{\frac{R_v}{1-P}(\delta+\pi)(\alpha+\gamma+\pi)+\frac{\delta\beta_1(1-P)\pi-\delta[\pi(\delta+\pi)+\pi(\alpha+\gamma+\pi)+(\delta+\pi)(\alpha+\gamma+\pi)]}{(\delta+\pi)(\alpha+\gamma+\pi)}\},$$

and

$$E = 1 - R_v.$$

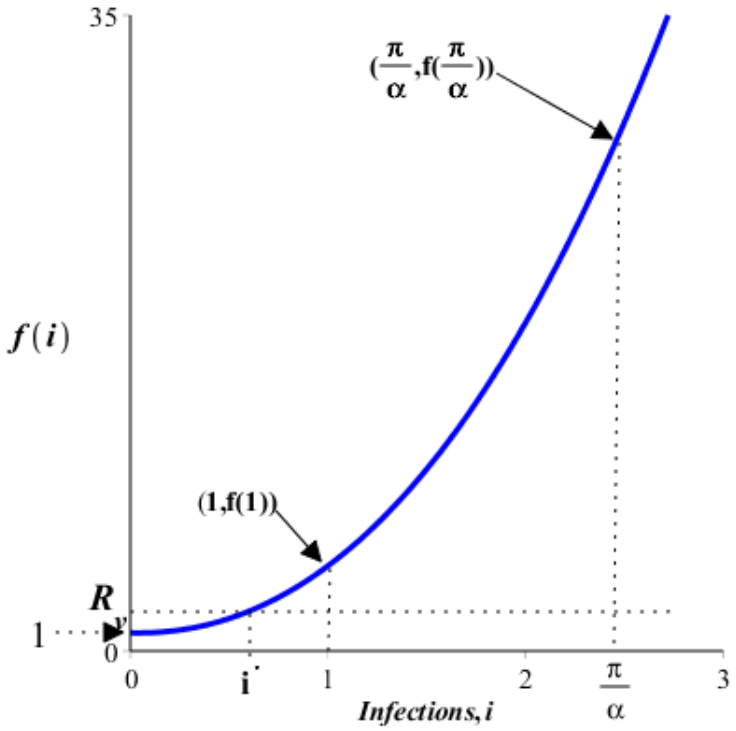

Figure 3.2: The existence and uniqueness of i^* in the interval $0 < i^* < \min\{1, \frac{\pi}{\alpha}\}$

3.4.5 Local stability of endemic equilibrium

Theorem 3.18. *If $R_v > 1$, then the endemic equilibrium $E^u = (s^*, e^*, i^*)$ of the governing model (3.4) is locally asymptotically stable.*

Proof. We note that $R_v > 1$ implies that $\beta_2 > \alpha$. The Jacobian of (3.3) at $E^u = (s^*, e^*, i^*)$ is given by,

$$J_{E^u} = \begin{bmatrix} -\frac{(1-P)\pi}{s^*} & -\beta_1 s^* & (\alpha - \beta_2)s^* \\ \beta_1 e^* + \beta_2 i^* & -\beta_2 \frac{s^* i^*}{e*} & \beta_2 s^* + \alpha e^* \\ 0 & \delta & -\frac{(\delta e^* - \alpha i^{*2})}{i^*} \end{bmatrix}. \tag{3.60}$$

We show the stability of the matrix J_{E^u} by verifying the Routh-Hurwitz conditions, that is, all the roots of the resulting characteristic equation must have negative real part.

The resulting polynomial is,

$$\mid J_{E^u} - \lambda I \mid = \lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 = 0, \tag{3.61}$$

where,

$$A_1 = \beta_2 \frac{s*i*}{e*} + (\alpha + \gamma + \pi) + \frac{(1-P)\pi}{s*},$$

$$A_2 = \beta_2 \frac{s^* i^*}{e*}[(\alpha + \gamma + \pi) + \frac{(1-P)\pi}{s^*} + \frac{(\alpha + \gamma + \pi)(1-P)\pi}{s^*}] -$$

$$(\delta\beta_2 s^* + \delta\alpha e^* + \beta_1^2 s^* e^* + \beta_1\beta_2 s^* i^*),$$

and

$$A_3 = \beta_2 \frac{s^* i^*}{e*}(\alpha + \gamma + \pi) + (\alpha + \gamma + \pi)(\beta_1^2 s^* e^* +$$

$$\beta_1\beta_2 s^* i^*) - \delta(\alpha - \beta_2)s^*(\beta_1 e^* + \beta_2 i^*) - \delta\beta_2 s^* - \delta\alpha e^*.$$

Clearly, $A_1 > 0$ and if $R_v > 1$ then $A_3 > 0$ and $A_1A_2 - A_3 > 0$.

$$A_1A_2 - A_3 = \{[\beta_2 \frac{s*i*}{e*} + (\alpha + \gamma + \pi) + \frac{(1-P)\pi}{s*}][\beta_2 \frac{s^* i^*}{e*}((\alpha + \gamma + \pi) + \frac{(1-P)\pi}{s^*} +$$

$$\frac{(\alpha + \gamma + \pi)(1-P)\pi}{s^*}) - (\delta\beta_2 s^* + \delta\alpha e^* + \beta_1^2 s^* e^* + \beta_1\beta_2 s^* i^*)] - (\beta_2 \frac{s^* i^*}{e*}(\alpha + \gamma + \pi) +$$

$$(\alpha+\gamma+\pi)(\beta_1^2 s^* e^* + \beta_1\beta_2 s^* i^*) - \delta(\alpha-\beta_2)s^*(\beta_1 e^* + \beta_2 i^*) - \delta\beta_2 s^* - \delta\alpha e^*)\}. \tag{3.62}$$

Hence, by Routh-Hurwitz criteria as in Martcheva (2015); Boyce et al. (2001), we have that the eigenvalues of J_{E^u} have negative real parts when $R_v > 1$. This shows that the endemic equilibrium E^u, which exists if $R_v > 1$ is locally asymptotically stable. □

3.4.6 Global stability of Endemic equilibrium

Theorem 3.19. *If $R_v > 1$, the endemic equilibrium E^u of the model subsystem (3.4) is globally asymptotically stable.*

Proof. We determine the global stability of the endemic equilibrium E^u, by defining the following Lyapunov function:

$$V(s^*, e^*, i^*) = (s - s^* - s^* \log\frac{s^*}{s}) + (e - e^* - e^* \log\frac{e^*}{e}) + (i - i^* - i^* \log\frac{i^*}{i}). \tag{3.63}$$

We observe that V is positive definite since $V = 0$ when $(s, e, i) = (s^*, e^*, i^*)$ and $V > 0$ otherwise; We also note that V is radially unbounded. Hence, V is a Lyapunov function.

Next, we prove that the derivative of V with respect to t is negative. The derivative of V, by direct calculation along the solution of system (3.3) is,

$$V^{'} = (\frac{s-s^*}{s})s^{'} + (\frac{e-e^*}{e})e^{'} + (\frac{i-i^*}{i})i^{'}. \tag{3.64}$$

Substituting the expressions of the model $(s^{'}, e^{'}, i^{'})$ into the equation (3.64) we have,

$$V^{'} = (\frac{s-s^*}{s})[(1-P)\pi - (\beta_1 e + \beta_2 i)s - \pi s + \alpha i s] + (\frac{e-e^*}{e})[(\beta_1 e + \beta_2 i)s -$$

$$(\delta+\pi)e + \alpha i e] + (\frac{i-i^*}{i})[\delta e - (\alpha+\gamma+\pi)i + \alpha i^2], \tag{3.65}$$

or

$$V^{'} = F - G, \tag{3.66}$$

where F represents the positive terms and G the negative terms of equation (1.5.30). Hence, if $F < G$ in equation (3.66), then we have that $V^{'} \leq 0$. We note that $V^{'} = 0$ if and only if $s = s^*, e = e^*, i = i^*$.

Thus, the largest compact invariant set in $E^u = \{(s^*, e^*, i^*) \in \Gamma : V^{'} = 0\}$ is the singleton E^u where E^u is the endemic equilibrium of the model system (3.3). By Lasalle's Invariant principle Martcheva (2015); Boyce et al. (2001); Shuai and van den Driessche (2013); Brauer et al. (2001), it follows that E^u is globally asymptotically stable in Γ if $F < G$. □

3.5 Sensitivity Analysis

Uncertainties when estimating certain parameter values demands that we investigate how sensitive the vaccination reproductive number R_v is with respect to its parameters. We determine the changes occurring in childhood disease temporal dynamics by computing the sensitivity indices of the basic reproduction Number , with respect to the parameter values in the model. The sensitivity indices serve as determinants of the significance of each parameter in the dynamics and prevalence of the diseases. They measure the change in model variables when a parameter changes (Chitnis et al. (2008); Bakare (2015)). Sensitivity analysis is essential in determining certain key parameter values that are highly sensitive and influence the outcome of a disease outbreak and must be targeted by control strategies (Chitnis et al. (2008)).

The sensitivity indices of R_v to parameter values for the SEIR model were computed using the following estimated parameter values; $\beta_1 = 0.2$, $\beta_2 = 0.3$, $\alpha = 0.04$, $\gamma = 0.1$, $\delta = 0.1$, $\pi = 0.1$, $P = 0.3846$.

Using the normalized forward sensitivity index (Chitnis et al. (2008); Bakare (2015)) of a variable,p, which depends differentially on a parameter,q, defined as:

$$Z_q^p = \frac{\partial p}{\partial q} \times \frac{q}{p}. \tag{3.67}$$

From (3.67), we derived an expression for the sensitivity index of R_v given by $Z_q^{R_v} = \frac{\partial R_v}{\partial q} \times \frac{q}{R_v}$ with respect to each of the parameters contained in R_v. Some illustrative examples of sensitivity index of R_v with respect to β_1 and P are given by; $Z_{\beta_1}^{R_v} = \frac{\partial R_v}{\partial \beta_1} \times \frac{\beta_1}{R_v} = 0.615385$ and $Z_P^{R_v} = \frac{\partial R_v}{\partial P} \times \frac{P}{R_v} = -0.624959$.

The other indices are given by: $Z_{\beta_2}^{R_v}, Z_\alpha^{R_v}, Z_\gamma^{R_v}, Z_\delta^{R_v}$ and $Z_\pi^{R_v}$ are computed using the same approach.

Table 3.1: Sensitivity indices of R_v.

Parameter	Parameter description	sensitivity indices
π	Proportion of susceptible that are vaccinated at birth	-0.660256
P	Vaccination rate at birth	-0.624959
β_1	Susceptible contact rate of disease from exposed group	0.615385
β_2	Susceptible contact rate of disease from infected group	0.384615
γ	Progression rate from infected to recovered group	-0.160256
δ	Progression rate from exposed to infected group	-0.115385
α	The death rate due to disease infection	-0.064103

3.5.1 Interpretation of sensitivity indices

Table 3.1 shows the numerical values of sensitivity indices of R_v to the parameter values for the SEIR model. The parameters are arranged from the most sensitive to least. The most sensitive parameters are the proportion of susceptible that are vaccinated at birth π, followed by vaccination coverage, P and then susceptible contact rate of disease from exposed group, β_1 while the least sensitive parameter is the death rate due to disease infection, α . From Table 3.1, we see that when the parameters β_1 and β_2 are increased while keeping the other parameters constant, the computational value of R_v increases. Hence, they increase the persistence of the childhood diseases in the community since they have positive indices. Also, the parameters $\alpha, \delta, \gamma, P$ and π decreases the computational value of R_v when increased while holding the other parameters constant. Thus, they decrease the endemicity of the disease as they have negative indices.

3.6 Bifurcation Diagram

To analyze a bifurcation diagram, we considered the computed equilibrium points for the differential equations obtained in the model i.e. the disease-free and endemic equilibrium. We illustrate the phenomenon of Bifurcation (Hethcote (2000); Martcheva (2015)) by considering the polynomial (3.59) resulting from the endemic equilibrium. The estimated parameter values of Table **4.1** are used to plot

the diagram.

We observe that equation (3.59) has a unique positive solution when $R_v > 1$, since the constant term is negative. We considered equation (3.59) to define a curve of i^* against R_v in the positive quadrant.

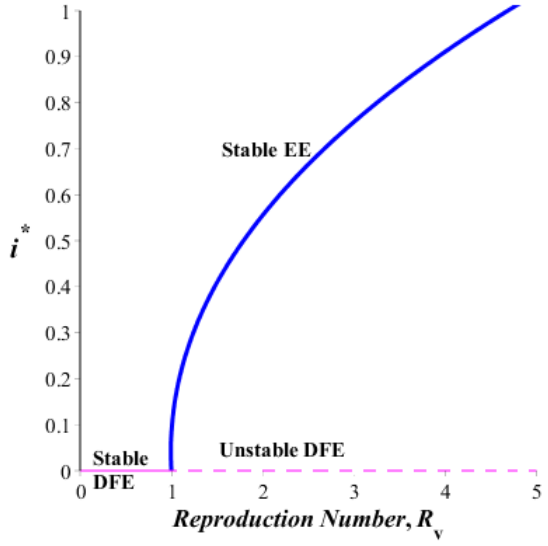

Figure 3.3: Forward Bifurcation diagram for SEIR childhood disease model.

Figure 3.3 clearly shows the existence of a unique locally asymptotically stable equilibrium when $R_v < 1$, confirming that the governing model (3.3) undergoes the phenomenon of forward bifurcation (Martcheva (2015); Hethcote (2000)).

The diagram exhibits a globally stable disease-free equilibrium when $R_v < 1$ and an unstable state if $R_v > 1$, while it is evident that a unique stable endemic equilibrium emerges from the bifurcation point $R_v = 1$ and increases rapidly when $R_v > 1$. It is clear that the disease-free state exists for all R_v while an endemic equilibrium only exists for $R_v > 1$. An increase in R_v through the critical reproduction number i.e. $R_v = 1$ results to a transcritical bifurcation.

3.7 Methods of Solution

3.7.1 Adomain Decomposition Method

In this subsection, the Semi-numerical solution of the considered model problem was completely determined using Adomain Decomposition Method (Jiao et al. (2002); Haldar (2015); Wazwaz (1999,

2000); Cherruault (1989); Cherruault and Adomian (1993)). Adomain Decomposition Method was used to construct approximate solutions of the normalized system of Equations.

The method is efficient since it can be applied directly to all types of differential and integral equations, linear or nonlinear, homogeneous or inhomogeneous, with constant coefficients or with variable coefficients. The approach is capable of reducing the size of computation work while maintaining high levels of accuracy of the semi-numerical solution (Cherruault (1989); Jiao et al. (2002); Wazwaz (1999)).

We rewrite the system of differential equations (3.3) in operator form as follows:

$$\begin{cases} Ls = (1-P)\pi - (\beta_1 e + \beta_2 i)s - \pi s + \alpha i s, \\ Le = (\beta_1 e + \beta_2 i)s - (\delta + \pi)e + \alpha i e, \\ Li = \delta e - (\alpha + \gamma + \pi)i + \alpha i^2, \\ Lr = P\pi + (\gamma + \alpha)i - \pi r, \end{cases} \tag{3.68}$$

where

$$L = \frac{d}{dt}, \tag{3.69}$$

and

$$L^{-1}(.) = \int_0^t (.)\, dt. \tag{3.70}$$

By applying the inverse operator L^{-1} on both sides of system (3.68) yields,

$$s(t) = s(0) + (1-P)\pi t - \beta_1 \int_0^t es\, dt - (\beta_2 - \alpha) \int_0^t is\, dt - \pi \int_0^t s\, dt, \tag{3.71}$$

$$e(t) = e(0) + \beta_1 \int_0^t es\, dt + \beta_2 \int_0^t is\, dt - (\delta + \pi) \int_0^t e\, dt + \alpha \int_0^t ie\, dt, \tag{3.72}$$

$$i(t) = i(0) + \delta \int_0^t e\, dt - (\alpha + \gamma + \pi) \int_0^t i\, dt + \alpha \int_0^t i^2\, dt, \tag{3.73}$$

$$r(t) = r(0) + P\pi t + (\gamma + \alpha) \int_0^t i\, dt - \pi \int_0^t r\, dt. \tag{3.74}$$

Applying Adomain Decomposition Method, the solutions of equations (3.71)-(3.74) are found as the sum of the following series

The linear terms:

$$s = \sum_{n=0}^{\infty} s_n, e = \sum_{n=0}^{\infty} e_n, i = \sum_{n=0}^{\infty} i_n, r = \sum_{n=0}^{\infty} r_n. \tag{3.75}$$

And we approximate the non-linear terms as follows:

$$\begin{cases} si = \sum_{n=0}^{\infty} A_n(s_0, ..., s_n, i_0, ..., i_n), ei = \sum_{n=0}^{\infty} B_n(e_0, ..., e_n, i_0, ..., i_n), \\ se = \sum_{n=0}^{\infty} C_n(s_0, ..., s_n, e_0, ..., e_n), i^2 = \sum_{n=0}^{\infty} D_n(i_0, ..., i_n). \end{cases} \tag{3.76}$$

where

$$A_n = \frac{1}{n!}\left[\frac{d^n(\sum_{k=0}^{\infty} s_k\lambda^k)(\sum_{k=0}^{\infty} i_k\lambda^k)}{d\lambda^n}\right]_{\lambda=0},$$

$$C_n = \frac{1}{n!}\left[\frac{d^n(\sum_{k=0}^{\infty} s_k\lambda^k)(\sum_{k=0}^{\infty} e_k\lambda^k)}{d\lambda^n}\right]_{\lambda=0},$$

$$B_n = \frac{1}{n!}\left[\frac{d^n(\sum_{k=0}^{\infty} e_k\lambda^k)(\sum_{k=0}^{\infty} i_k\lambda^k)}{d\lambda^n}\right]_{\lambda=0},$$

and

$$D_n = \frac{1}{n!}\left[\frac{d^n(\sum_{k=0}^{\infty} i_k\lambda^k)^2}{d\lambda^n}\right]_{\lambda=0}.$$

The Adomain polynomials are the non-linear functions A_n, B_n, C_n and D_n. Substituting the Adomain polynomials and the linear terms in equtions (3.71)-(3.74) we have,

$$\sum_{n=0}^{\infty} s_n = s(0) + (1-P)\pi t - \beta_1 \int_0^t \sum_{n=0}^{\infty} C_n\, dt - (\beta_2 - \alpha)\int_0^t \sum_{n=0}^{\infty} A_n\, dt - \pi \int_0^t \sum_{n=0}^{\infty} s_n\, dt, \tag{3.77}$$

$$\sum_{n=0}^{\infty} e_n = e(0) + \beta_1 \int_0^t \sum_{n=0}^{\infty} C_n\, dt + \beta_2 \int_0^t \sum_{n=0}^{\infty} A_n\, dt - (\delta + \pi)\int_0^t \sum_{n=0}^{\infty} e_n\, dt + \alpha \int_0^t \sum_{n=0}^{\infty} B_n\, dt, \tag{3.78}$$

$$\sum_{n=0}^{\infty} i_n = i(0) + \delta \int_0^t \sum_{n=0}^{\infty} e_n\, dt - (\alpha + \gamma + \pi)\int_0^t \sum_{n=0}^{\infty} i_n\, dt + \alpha \int_0^t \sum_{n=0}^{\infty} D_n\, dt, \tag{3.79}$$

$$\sum_{n=0}^{\infty} r_n = r(0) + P\pi t + (\gamma + \alpha)\int_0^t \sum_{n=0}^{\infty} i_n\, dt - \pi \int_0^t \sum_{n=0}^{\infty} r_n\, dt. \tag{3.80}$$

Using equations (3.77)-(3.80) we define the following initial conditions and recursive relationship:

$$s_0 = s(0) + (1-P)\pi t, e_0 = e(0), i_0 = i(0), r_0 = r(0) + P\pi t, \tag{3.81}$$

$$s_{n+1} = -\beta_1 \int_0^t C_n\, dt - (\beta_2 - \alpha)\int_0^t A_n\, dt - \pi \int_0^t s_n\, dt, \tag{3.82}$$

$$e_{n+1} = \beta_1 \int_0^t C_n\, dt + \beta_2 \int_0^t A_n\, dt - (\delta + \pi) \int_0^t e_n\, dt + \alpha \int_0^t B_n\, dt, \tag{3.83}$$

$$i_{n+1} = \delta \int_0^t e_n\, dt - (\alpha + \gamma + \pi) \int_0^t i_n\, dt + \alpha \int_0^t D_n\, dt, \tag{3.84}$$

$$r_{n+1} = (\gamma + \alpha) \int_0^t i_n\, dt - \pi \int_0^t r_n\, dt. \tag{3.85}$$

We compute the adomain polynomials as follows:

$A_0 = s_0 i_0, A_1 = s_0 i_1 + s_1 i_0, A_2 = s_0 i_2 + s_1 i_1 + s_2 i_0, A_3 = s_0 i_3 + s_1 i_2 + s_2 i_1 + s_3 i_0, A_4 = s_0 i_4 + s_1 i_3 + s_2 i_2 + s_3 i_1 + s_4 i_0, A_5 = s_0 i_5 + s_1 i_4 + s_2 i_3 + s_3 i_2 + s_4 i_1 + s_5 i_0, ...,$

$B_0 = e_0 i_0, B_1 = e_0 i_1 + e_1 i_0, B_2 = e_0 i_2 + e_1 i_1 + e_2 i_0, B_3 = e_0 i_3 + e_1 i_2 + e_2 i_1 + e_3 i_0, B_4 = e_0 i_4 + e_1 i_3 + e_2 i_2 + e_3 i_1 + e_4 i_0, B_5 = e_0 i_5 + e_1 i_4 + e_2 i_3 + e_3 i_2 + e_4 i_1 + e_5 i_0, ...,$

$C_0 = s_0 e_0, C_1 = s_0 e_1 + s_1 e_0, C_2 = s_0 e_2 + s_1 e_1 + s_2 e_0, C_3 = s_0 e_3 + s_1 e_2 + s_2 e_1 + s_3 e_0, C_4 = s_0 e_4 + s_1 e_3 + s_2 e_2 + s_3 e_1 + s_4 e_0, C_5 = s_0 e_5 + s_1 e_4 + s_2 e_3 + s_3 e_2 + s_4 e_1 + s_5 e_0, ...,$

$D_0 = i_0^2, D_1 = 2 i_0 i_1, D_2 = 2 i_0 i_2 + i_1^2, D_3 = 2 i_0 i_3 + 2 i_1 i_2, D_4 = 2 i_0 i_4 + 2 i_1 i_3 + i_2 i_2, D_5 = 2 i_0 i_5 + 2 i_1 i_4 + 2 i_2 i_3, ...$

substituting equations (3.81)-(3.85) and the Adomain polynomials into equation (3.77)-(3.80), by use of Maple we approximate the solution as follows:

$$s_N = \sum_{n=0}^{N} s_n, e_N = \sum_{n=0}^{N} e_n, i_N = \sum_{n=0}^{N} i_n, r_N = \sum_{n=0}^{N} r_n, \tag{3.86}$$

where

$$s(t) = \lim_{N \to \infty} (s_N), e(t) = \lim_{N \to \infty} (e_N), i(t) = \lim_{N \to \infty} (i_N), r(t) = \lim_{N \to \infty} (r_N). \tag{3.87}$$

3.7.2 Fourth order Runge-Kutta integration method

The numerical solution of the considered model problem was completely determined using the classical fourth order Runge-Kutta Method (Atkinson (2008); Christodoulou (2009)).

The pure numerical technique was used to solve the differential equations of system (3.3) of the form:

$\frac{ds}{dt} = f(t, s, e, i)$, $\frac{de}{dt} = f(t, s, e, i)$, $\frac{di}{dt} = f(t, e, i)$ and $\frac{dr}{dt} = f(t, i, r)$ satisfying $s(0) = s_0, e(0) = e_0, i(0) = i_0$ and $r(0) = r_0$.

We let $h = t_{n+1} - t_n$, $n = 0, 1, 2, ...$ so that the Taylor series of $s(t_{n+1}) = s_{n+1}$ about s_n is given by,

$$s_{n+1} = s_n + hf(t_n, s_n) + \frac{1}{2!}h^2 f^{'}(t_n, s_n) + ..., \tag{3.88}$$

with $f(t, s, e, i, r)$.

Then, the RK4 integration method (Atkinson (2008); Christodoulou (2009)) for the equation (3.88) yields,

$$s_{n+1} = s_n + \frac{h}{6}(k_1 s + 2k_2 s + 2k_3 s + k_4 s), \tag{3.89}$$

$$e_{n+1} = e_n + \frac{h}{6}(k_1 e + 2k_2 e + 2k_3 e + k_4 e), \tag{3.90}$$

$$i_{n+1} = i_n + \frac{h}{6}(k_1 i + 2k_2 i + 2k_3 i + k_4 i), \tag{3.91}$$

$$r_{n+1} = r_n + \frac{h}{6}(k_1 r + 2k_2 r + 2k_3 r + k_4 r), \tag{3.92}$$

where

$k_1 s = fs(t_n, s_n, e_n, i_n)$,

$k_1 e = fe(t_n, s_n, e_n, i_n)$,

$k_1 i = fi(t_n, e_n, i_n)$,

$k_1 r = fr(t_n, i_n, r_n)$,

$k_2 s = fs(t_n + \frac{h}{2}, s_n + \frac{h}{2} * k_1 s, e_n + \frac{h}{2} * k_1 e, i_n + \frac{h}{2} * k_1 i)$,

$k_2 e = fe(t_n + \frac{h}{2}, s_n + \frac{h}{2} * k_1 s, e_n + \frac{h}{2} * k_1 e, i_n + \frac{h}{2} * k_1 i)$,

$k_2 i = fi(t_n + \frac{h}{2}, e_n + \frac{h}{2} * k_1 e, i_n + \frac{h}{2} * k_1 i)$,

$k_2 r = fr(t_n + \frac{h}{2}, i_n + \frac{h}{2} * k_1 i, r_n + \frac{h}{2} * k_1 r)$,

$k_3 s = fs(t_n + \frac{h}{2}, s_n + \frac{h}{2} * k_2 s, e_n + \frac{h}{2} * k_2 e, i_n + \frac{h}{2} * k_2 i)$,

$k_3 e = fe(t_n + \frac{h}{2}, s_n + \frac{h}{2} * k_2 s, e_n + \frac{h}{2} * k_2 e, i_n + \frac{h}{2} * k_2 i)$,

$k_3 i = fi(t_n + \frac{h}{2}, e_n + \frac{h}{2} * k_2 e, i_n + \frac{h}{2} * k_2 i)$,

$k_3 r = fr(t_n + \frac{h}{2}, i_n + \frac{h}{2} * k_2 i, r_n + \frac{h}{2} * k_2 r)$,

$k_4s = fs(t_n + h, s_n + h * k_3s, e_n + h * k_3e, i_n + h * k_3i)$,

$k_4e = fe(t_n + h, s_n + h * k_3s, e_n + h * k_3e, i_n + h * k_3i)$,

$k_4i = fi(t_n + h, e_n + h * k_3e, i_n + h * k_3i)$,

$k_4r = fr(t_n + h, i_n + h * k_3i, r_n + h * k_3r)$,

with $fs = \frac{ds}{dt}$, $fe = \frac{de}{dt}$, $fi = \frac{di}{dt}$ and $fr = \frac{dr}{dt}$.

The error in this method is proportional to h^5, this implies that the accuracy of the method can be improved by using small values of h (Atkinson (2008); Christodoulou (2009)).

The next chapter will display achieved results in tabular form and graphically and analyse the transmission dynamics of diseases based on the study findings.

Chapter 4

Results and Discussion

This chapter focuses on the display of findings in tabular form, graphically and its analysis. We estimate the parameters, implement the ADM scheme and fourth order Runge-Kutta integration scheme to plot the figures showing the effects of various parameter variation on each group of the population. This analysis was used to illustrate our results on stability, as well as numerical simulation and graphical representation of the system of normalized non-linear differential equations.

4.1 Estimation of parameters

The estimated parameter values and initial conditions of variables used for computations and numerical simulation purposes are shown in the **Table 4.1**,

Table 4.1: Effects of vaccination coverage on disease transmission, $P_c = 0.3846$.

Case	s_0	e_0	i_0	r_0	β_1	β_2	γ	α	π	δ	P	R_v	Comments
1	0.4	0.2	0.1	0.3	0.2	0.3	0.1	0.04	0.1	0.1	0.7	0.4875	LAS (E^0)
2	0.4	0.2	0.1	0.3	0.2	0.3	0.1	0.04	0.1	0.1	0.5	0.8125	LAS (E^0)
3	0.4	0.2	0.1	0.3	0.2	0.3	0.1	0.04	0.1	0.1	0.3	1.1375	Unstable (E^0)
4	0.4	0.2	0.1	0.3	0.2	0.3	0.1	0.04	0.1	0.1	0.1	1.4625	Unstable (E^0)

4.2 Computational Results

In this section, we study the effects of parameter variation on each group of the population using estimated parameter values and plotting their figures to demonstrate their impact on disease transmission and control. We also plot their phase portrait. Therefore, we monitor the effect of vaccination coverage on the dynamics of a childhood disease as shown by the SEIR model equations in (3.3) using the semi-numerical Adomian Decomposition Method in comparison with the classical Fourth order Runge-Kutta method to gauge its effectiveness.

The parameter values in Table 4.1 were used in the numerical simulations of the qualitative results obtained in the study.

Table 4.2: Comparison between ADM solution and 4th Order R-K numerical solution for Case-1, ($P = 0.7, P_c = 0.3846$)

ADM Solution					4^{th} order R-K solution			
t	s	e	i	r	s	e	i	r
0.1	0.396380	0.202877	0.099658	0.305370	0.396396	0.198871	0.099638	0.305370
0.3	0.389262	0.208615	0.099083	0.315939	0.389403	0.196567	0.098906	0.315936
0.5	0.382300	0.214333	0.098645	0.326284	0.382687	0.194204	0.098162	0.326273
0.7	0.375491	0.220034	0.098340	0.336414	0.376240	0.191791	0.097405	0.336384
1.0	0.365552	0.228559	0.098114	0.351223	0.367050	0.188088	0.096244	0.351137
1.5	0.349689	0.242709	0.098310	0.374935	0.352944	0.181745	0.094242	0.374654
2.0	0.334653	0.256809	0.099151	0.397527	0.340236	0.175257	0.092157	0.396885

Table 4.2 depicts case-1 and shows the comparison between ADM solution and fourth order Runge-Kutta numerical solution. The results for the case $P > P_c$ shows that the adomain series solution only agreed with the fourth order Runge-Kutta solution for very small values of time, t, i.e. $t = 0.1$ and 0.3. We note that the ADM series only agree with RK4 solutions as $t \to 0$ i.e. for very small values of time,t. Furthermore, as the values of time increases the Adomain solution diverge and does not agree with the fourth order Runge-Kutta numerical solution.

We observe that the fourth order Runge-Kutta solution converges to the Disease-free equilibrium.

It is noteworthy that unlike the ADM solution which fails to converge as time increases , the fourth order Runge-Kutta method gave results that are in agreement with the qualitative analysis results of the model as demonstrated by **Lemma 3.15** and **3.16**. In order to improve the ADM series radius of convergence, several series summations and enhancement methods such as Padé Approximation Technique (Makinde (2009)) may be applied. By this manipulation and improvement, we gain more information that describes the mathematical behavior of the solution (Makinde (2009)).

The Results obtained by using classical fourth order Runge-Kutta method are shown in **Figures 4.1, 4.2, 4.3, 4.4 and 4.5.**

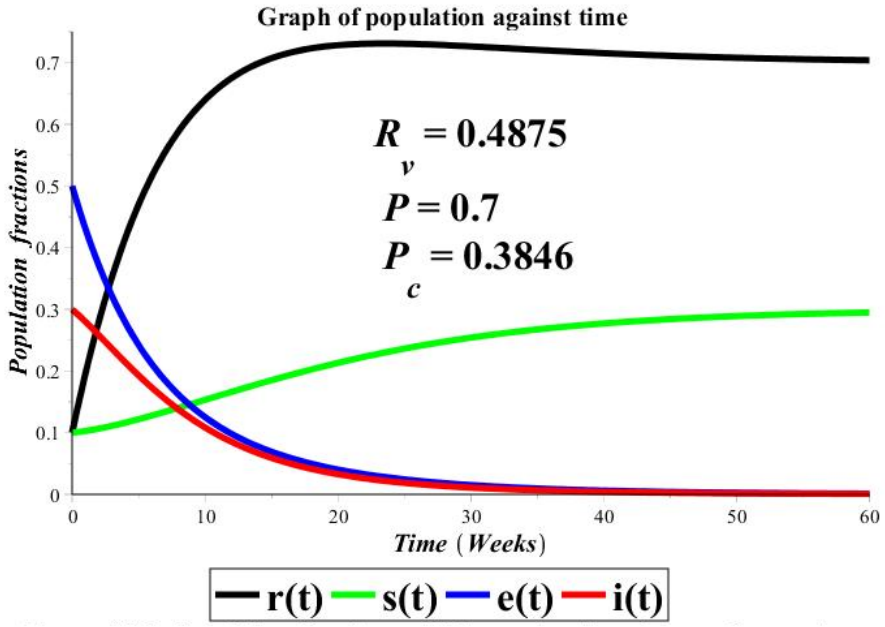

Figure 4.1: Population fractions with increasing time for case 1 scenario.

Figure 4.1 Describes case-1 and demonstrates the impact of high vaccination coverage ($P > P_c$) on the temporal dynamics of the population fractions with increasing time. The Susceptible group gradually increases by a small amount as time increases and asymptotically attains a steady state. The increase is due to the recruitment of susceptible children/new-born babies. The gradual small increase is due to high vaccination coverage i.e. $P = 0.7$ which led to low recruitment rates of susceptible children/newborn babies. The population fractions of the exposed and infective groups display a sharp decrease as time increases and finally attains a disease free state i.e. $e = 0$ and $i = 0$. The rapid decrease of exposed and infective populations is due to the high vaccination coverage and recovery due to the treatment of infected children. The recovered group displays a sharp increase

as time increases, it attains a peak and a slightly decreases to a steady state. The rapid rise is due to recruitment of more children/newborn babies into the recovered group through vaccination, in addition to the removed/treated children that confer permanent immunity. The slight decrease is due to losses by natural deaths. It is noteworthy that the disease-free equilibrium state is attained when we have a high vaccination coverage ($P > P_c$) and the entire population attains disease-free state asymptotically.

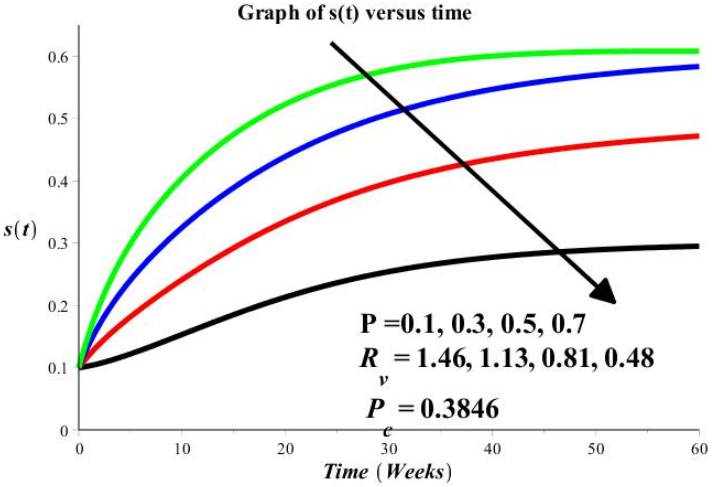

Figure 4.2: Effects of increasing vaccination (P) on susceptible population

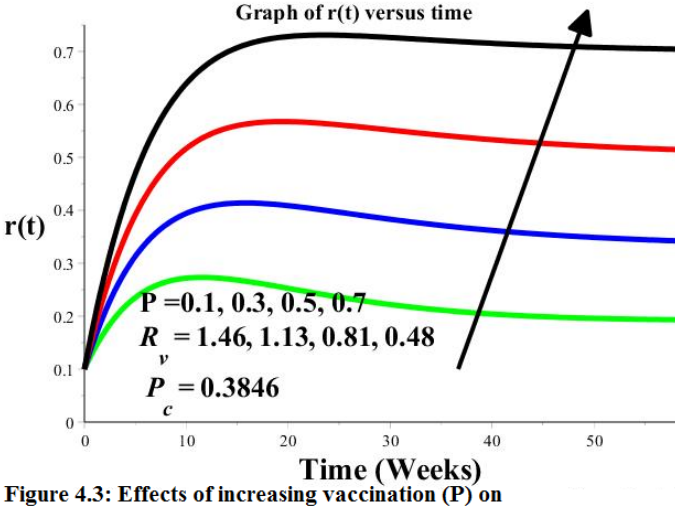

Figure 4.3: Effects of increasing vaccination (P) on removed/vaccinated population

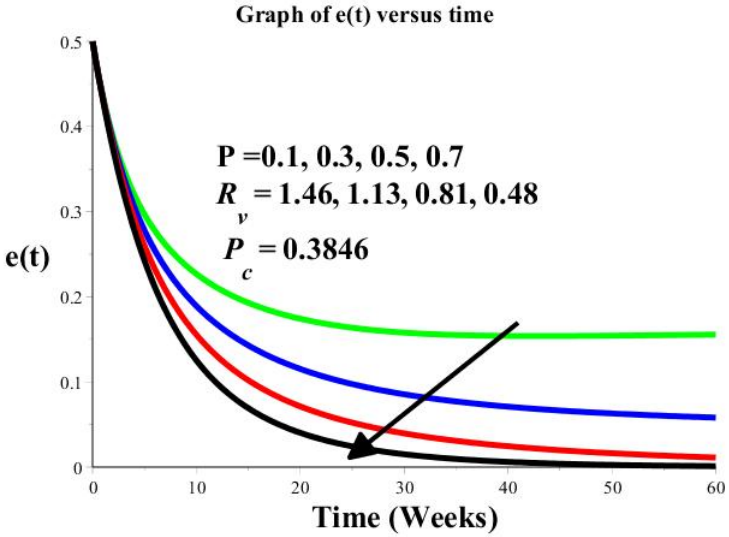

Figure 4.4: *Effects of increasing vaccination (P) on exposed population*

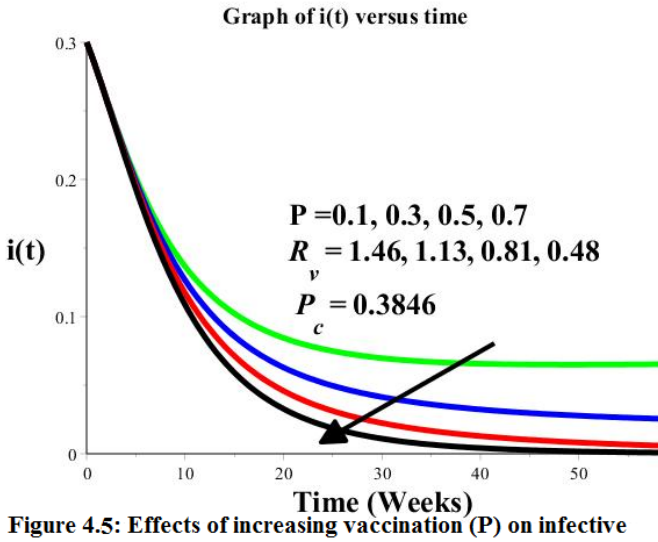

Figure 4.5: Effects of increasing vaccination (P) on infective population

Figure 4.2 Displays Case $1-4$ and illustrates the effects of varying vaccination coverage on susceptible population proportion. We observe that an increase in vaccination proportion results in a decrease in the number of susceptible children/newborn babies i.e. $s(t)$. The decrease is due to the low recruitment of susceptible children/new-born babies as the vaccine coverage is increased.

Figure 4.3 Displays Case $1-4$ and illustrates the effects of varying vaccination coverage on the removed/vaccinated population. An increase in the vaccination coverage led to an increase in the recovered population $(r(t))$, it attains a peak and a steadily decreases to a steady state. The increase is due to recruitment of more children / newborn babies into the $r(t)$ group through vaccination and treatment.

Figure 4.4 Displays Case $1-4$ and illustrates the effects of varying vaccination coverage on exposed population proportion. An increase in the vaccination coverage led to a decrease in the exposed population $(e(t))$. **Figure 4.5** Displays Case $1-4$ and illustrates the effects of varying vaccination coverage on infective population fraction. An increase in the vaccination coverage led to a decrease in the infective population $(i(t))$. The decrease of exposed and infective populations is due to the increased vaccination coverage which leads to low recruitment and recovery due to the treatment of infected children.

The **Figures 4.4** and **4.5** confirms that the exposed $(e(t))$ and infective population $(i(t))$ experience a rapid decrease on application of high vaccination coverage ($P > P_c$) to attain a disease-free state i.e. if P is large enough then the disease will die out in the population.

4.2.1 Phase portrait

In this subsection, we study the phase portrait of the model differential equations of system (3.3). The phase portrait displays the set of solutions plotted as trajectories which trace the path of each solution on the phase plane. We use the phase portrait to visualize how the solutions of the model system of differential equations behave as time increases (Boyce et al. (2001); Martcheva (2015)). The plotted phase portraits are demonstrated in **Figures 4.6, 4.7, 4.8, 4.9** and **4.10**,

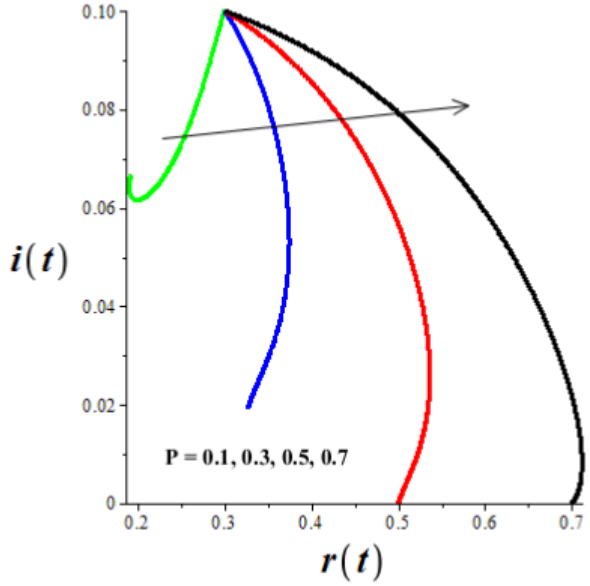

Figure 4.6: Phase portrait of infected and removed population with increasing vaccination (P)

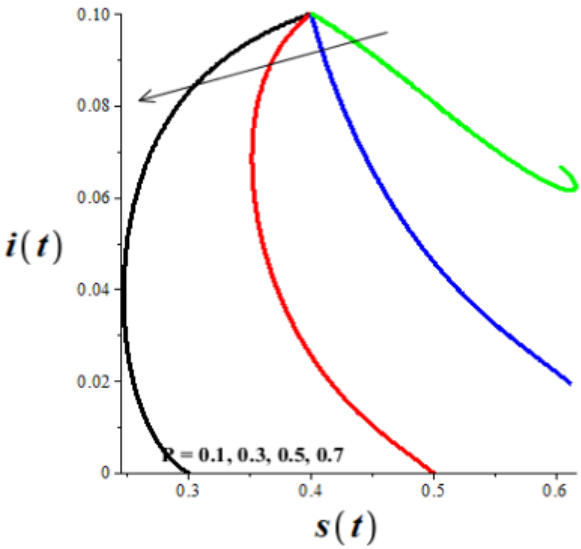

Figure 4.7: Phase portrait of infected and susceptible population with increasing vaccination (P)

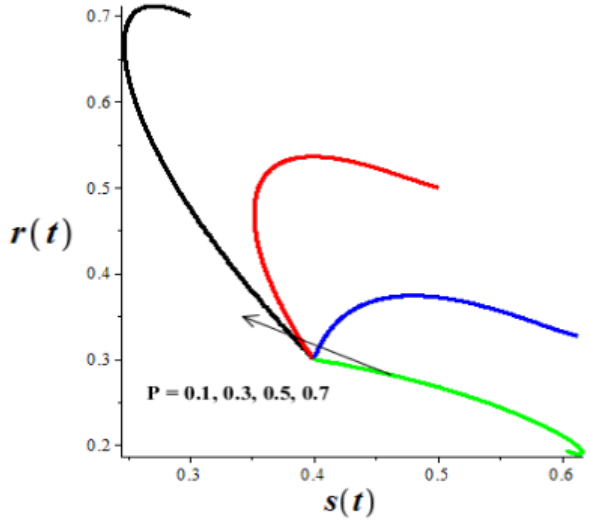

Figure 4.8: Phase portrait of removed and susceptible population with increasing vaccination (P)

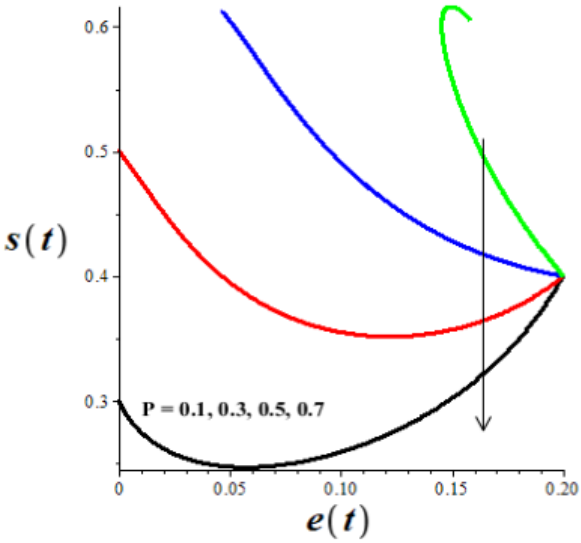

Figure 4.9: Phase portrait of susceptible and exposed population with increasing vaccination (P)

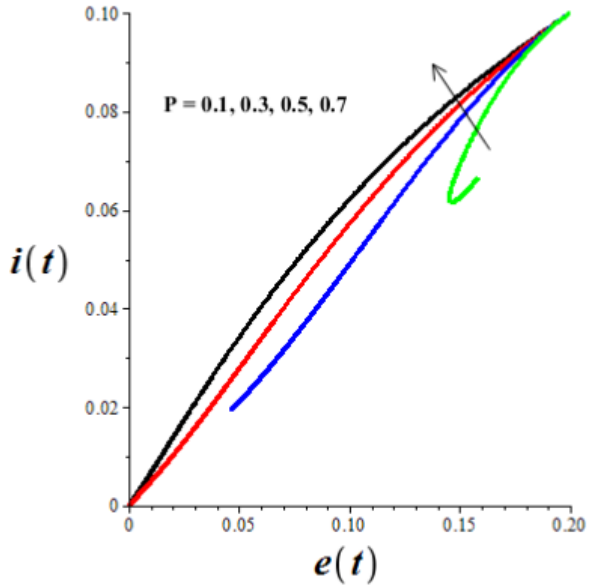

Figure 4.10: Phase portrait of infected and exposed population with increasing vaccination (P)

The phase portraits in **Figures 4.6-Figure 4.10** Depicts Case $1-4$ and illustrates the trajectories of susceptible, exposed, infective and recovered population from their initial states with increasing vaccination proportion. We observe that the trajectories starting from their initial states approach the disease-free state when the vaccination proportion is large enough i.e. $P > P_c$ and move away from the disease-free state when $P < P_c$. Therefore, we conclude that in order to successfully prevent disease, the vaccination coverage needs to be large enough ($P > P_c$).

The results achieved in this study are in agreement with the findings obtained by Makinde (2007, 2009) when solving a SIR childhood disease model using Adomain Decomposition approach and an SIV model solved using ADM approach coupled with He's Variational Iteration Method and Padé Approximation Technique, while Arafa et al. (2012) solved a fractional order childhood disease model using Homotopy Analysis Method. The studies confirmed that there is a certain critical threshold, P_c above which the childhood diseases will be successfully eradicated from the community. In addition, their results confirmed that the childhood diseases persists within the community if the vaccination coverage, P, is below the critical vaccination threshold value, P_c.

The next chapter was devoted to conclusions and recommendations for the study.

Chapter 5

Conclusions and Recommendations

5.1 Conclusions

A SEIR deterministic model that monitors the temporal dynamics of childhood diseases in the presence of preventive vaccine was analyzed. The model incorporates the fact that exposed individuals are infectious to the community. We prove the existence of a feasible region that is mathematically and biologically feasible. The existence and uniqueness of the disease free and endemic equilibrium were determined.

A qualitative analysis revealed that the disease-free equilibrium was globally asymptotically stable provided that the critical vaccination threshlod value is exceeded. The global stability of endemic equilibrium was achieved via Lyapunov method under certain conditions.

A forward bifurcation diagram reveals a stable disease free state if vaccination reproduction number is less than unity and unstable if vaccination reproduction number is greater than unity, while the endemic equilibrium attains a unique stable state if vaccination reproduction number is greater than unity. A sensitivity analysis reveals that the proportion of susceptible that are vaccinated at birth is the most sensitive parameter and death rate due to disease infection is the least sensitive.

Adomain Decomposition Method and fourth order Runge-Kutta Method are employed to compute the approximate solution of the model problem. The computations revealed that the Adomain Decomposition series only converge and agree with fourth order Runge-Kutta Method for very small values of time and does not agree as time increases. The fourth order Runge-Kutta scheme gave results that

were in agreement with the qualitative analysis and converged for all time. The results confirm the high level of accuracy, reliabilty and efficiency of fourth order Runge-Kutta Method when combined with a small step-size in computing the approximate solution of non-linear system of Equations.

5.2 Recommendations

- Public health authorities can use the formulated model to understand the spread and prevention/control of childhood diseases. The SEIR model can help the policy makers or public health agencies offer travel advisories since they will have a better understanding of how to optimize the allocation of vaccines to prevent an outbreak of an epidemic and be aware of how to respond to a changing dynamics of childhood diseases.

- A successful eradication of childhood diseases demands that the vaccinated proportion of the population must be large enough. The threshold value will be significant in decision making and policy analysis in under-resourced communities.

- There is need for more mathematical studies to optimize the use of vaccines that is crucial in ensuring successful global eradication of childhood diseases such as measles, mumps, polio etc.

- Researchers and students can apply the governing model equations formulated and results achieved in this study to further advance studies on childhood disease re-emergence burden.

- In order to improve the ADM series radius of convergence, we recommend the application of enhancement methods such as Padé Approximation Method which has proven to give a better and converging approximation of polynomials.

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Appendix

According to the Estimated parameter values in Table 4.1, the following Adomain Decomposition Method approximate series solutions were obtained:

Case1:

$$s(t) = \frac{2}{5} - \frac{91}{2500}t + \frac{5141}{2500000}t^2 - \frac{419219}{3750000000}t^3 + \frac{65906323}{7500000000000}t^4 + O(t^5)$$

$$e(t) = \frac{1}{5} + \frac{18}{625}t - \frac{737}{2500000}t^2 + \frac{228073}{3750000000}t^3 - \frac{53761001}{7500000000000}t^4 + O(t^5)$$

$$i(t) = \frac{1}{10} - \frac{9}{2500}t + \frac{1161}{625000}t^2 - \frac{287453}{1875000000}t^3 + \frac{38544821}{3750000000000}t^4 + O(t^5)$$

$$r(t) = \frac{3}{10} + \frac{27}{500}t - \frac{369}{125000}t^2 + \frac{723}{3906250}t^3 - \frac{3747371}{375000000000}t^4 + O(t^5)$$

Case2:

$$s(t) = \frac{2}{5} - \frac{41}{2500}t + \frac{991}{2500000}t^2 - \frac{265369}{3750000000}t^3 + \frac{50195873}{7500000000000}t^4 + O(t^5)$$

$$e(t) = \frac{1}{5} + \frac{18}{625}t + \frac{1013}{2500000}t^2 + \frac{273323}{3750000000}t^3 - \frac{41249251}{7500000000000}t^4 + O(t^5)$$

$$i(t) = \frac{1}{10} - \frac{9}{2500}t + \frac{1161}{625000}t^2 - \frac{243703}{1875000000}t^3 + \frac{34601071}{3750000000000}t^4 + O(t^5)$$

$$r(t) = \frac{3}{10} + \frac{17}{500}t - \frac{61}{31250}t^2 + \frac{14227}{93750000}t^3 - \frac{3128621}{375000000000}t^4 + O(t^5)$$

Case3:

$$s(t) = \frac{2}{5} + \frac{9}{2500}t - \frac{3159}{2500000}t^2 - \frac{37173}{1250000000}t^3 + \frac{7995141}{2500000000000}t^4 + O(t^5)$$

$$e(t) = \frac{1}{5} + \frac{18}{625}t + \frac{2763}{2500000}t^2 + \frac{106191}{1250000000}t^3 - \frac{6079167}{2500000000000}t^4 + O(t^5)$$

$$i(t) = \frac{1}{10} - \frac{9}{2500}t + \frac{1161}{625000}t^2 - \frac{66651}{625000000}t^3 + \frac{10219107}{1250000000000}t^4 + O(t^5)$$

$$r(t) = \frac{3}{10} + \frac{7}{500}t - \frac{119}{125000}t^2 + \frac{5551}{46875000}t^3 - \frac{2509871}{375000000000}t^4 + O(t^5)$$

Case4:

$$s(t) = \frac{2}{5} + \frac{59}{2500}t - \frac{7309}{2500000}t^2 + \frac{42331}{3750000000}t^3 - \frac{12725027}{7500000000000}t^4 + O(t^5)$$

$$e(t) = \frac{1}{5} + \frac{18}{625}t + \frac{4513}{2500000}t^2 + \frac{363823}{3750000000}t^3 + \frac{15274249}{7500000000000}t^4 + O(t^5)$$

$$i(t) = \frac{1}{10} - \frac{9}{2500}t + \frac{1161}{625000}t^2 - \frac{156203}{1875000000}t^3 + \frac{26713571}{3750000000000}t^4 + O(t^5)$$

$$r(t) = \frac{3}{10} - \frac{3}{500}t + \frac{3}{62500}t^2 + \frac{2659}{31250000}t^3 - \frac{1891121}{375000000000}t^4 + O(t^5)$$