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A Mathematical Model Analysis on the Spread and Control of Diarrhea in the Case of Bibugn Woreda

Bayenes Ayenew Ewnetu[1*] Dr.Mohammed Yiha[2]

[1]Raya University, Ethiopia

[2]Mathematics department head in Hawasa university, Ethiopia

Corresponding Author:** ***Bayenes Ayenew Ewnetu, *Raya University, Ethiopia*

Abstract: In this work we present a nonlinear mathematical model to analyze the spread and control of diarrhea in the case of Bibugn Woreda. We found the basic reproduction number of the mathematical model system, $R_0 = \frac{\lambda\beta\sigma((1-\rho)\mu+w)[\nu^2(\mu+\delta+\tau)+(1-\nu)((1-\nu)\mu+\gamma)]}{\mu(\mu+w)(\mu+\sigma)(\mu+\gamma)(\mu+\delta+\tau)}$ which depends on ten parameters. We also found the numerical value of the reproduction number of the dynamical system based on real data collected from Bibugn Woreda is $R_0 = 3.943295539 > 1$. This in principle implies that the disease spreads in the community of Bibugn Woreda. We found two equilibrium points, unstable disease-free equilibrium point and stable endemic equilibrium point. To control the spread of the disease, we identified the control parameters. One of such control parameters is the contact rate $\beta = 0.1287$. To make the basic reproduction number less than one the contact rate β not to exceed the numerical value 0.1287. The effect of the remaining parameters on the basic reproduction number discussed detail in their sub section.

Keywords: Dynamical system, Routh-Hurwitz stability criterion, reproduction number,symptomatic infected, asymptomatic infected.

ACRONYMS: AIDS(Acquired Immuno Deficiency Syndrome), DE(Differential Equation), DALY(Disability Adjusted Life Year), DFE(Disease Free Equilibrium Point), GAPPD(Global Action Plan for the Prevention and Control of Pneumonia and Diarrhoea), HIV(Human Immunodeficiency Virus), ORS(Oral Rehydration Salts), ORS(Oral Rehydration Solution), ORT(Oral Rehydration Therapy), UNICEF(United Nations Children's Fund), WHO(World Health Organization)

1. INTRODUCTION

"Diarrhea is stools of decreased consistency and increased volume due to imbalance of secretion and absorption of water and salts in the intestine'' [4]. In diarrhea, stools contain more water than normal they are often called loose or watery stools. They may also contain blood; in which case the diarrhea is called dysentery [16]. "Diarrheal diseases are amongst the most frequent childhood illnesses and leading cause of preventable death, especially among children under five in developing countries" [11]. Diarrhea is the second leading cause of death among children under five. Nearly one in five child deaths (about1.5 billion each year) is due to diarrhea [5].

As [3] proposed that most enteric infections are asymptomatic, and the proportion that is asymptomatic increases beyond 2 years of age owing to the development of active immunity. During asymptomatic infections, which may last for several days or weeks, stools containinfectious viruses, bacteria, or protozoa1 cysts. People with asymptomatic infections play an important role in the spread of many enteric pathogens, especially as they are unaware of their infection, take no special hygienic precautions and move normally from place to place.

Rotavirus vaccines have been recommended by the WHO for use in national immunization programs worldwide and have been listed as necessary components of WHO and UNICEF's Integrated Global Action Plan for the Prevention and Control of Pneumonia and Diarrhea(GAPPD). They are also key

elements of the WHO Global Vaccine Action Plan, which is endorsed by 194-member states, including India. In India, the Indian Academy of Pediatrics has recommended the use of these vaccines for all Indian Children Rotavirus vaccines are currently being used in more than 70 countries. Reductions in Diarrhea hospitalizations have been demonstrated in more than 10 countries, including low – and middle-income countries [8]. Measles immunization: Measles immunization can substantially reduce the incidence and severity of diarrheal diseases. Every infant should be immunized against measles at the recommended age [15].

An epidemic is defined as a widespread occurrence of an infectious disease in a community at a particular time [9]. Models describe our beliefs about how the world functions. In mathematical modeling, we translate those beliefs into the language of mathematics [10]. Mathematical epidemiology is concerned with quantitative aspects of the subjects and usually consists ofmodel building, estimation of parameters, investigation of the sensitivity of the model to changesin the parameters, simulations.

In a compartmental model, we assume that the population is split into a number of different compartments, each individual belonging to exactly one compartment at any point in time. An individual can change state by moving from one compartment to another [9].
In this thesis, we proposed an improvement of the model [1] that a nonlinear mathematical modelstudied using SVEIS model, the analysis of Treatment Model of diarrhea Epidemic by considered four (2) compartmental models to gain insight into the effect of vaccine on the dynamical spread of diarrhea disease in a community. But in this study the researcher wanted to extend the SVEIS model [1] by considering the infected groups are divided in to two compartments, those are symptomatic infected and asymptomatic infected group. Theasymptomatic infected are decreased by natural death rate and symptom gain rate and symptomatic infected are decreased by natural death rate and diseases induced death rate. the model [1] is the motivation of our study. The conclusion of the study is determined by the result of the basic reproduction number. This research is concerned with the analysis of a mathematicalmodel on the spread and control of diarrhea under the screening of asymptomatic infective in the case of Bibugn Woreda, East Gojam, Amhara Region, Ethiopia .The dynamics of mathematical model are setting on the background of mathematical, biological and epidemiological knowledge .the combination of those mathematics, biology and epidemiology provides the source of interesting result and give the powerful application for the people living symptomatic andasymptomatic with diarrhea and those policy makers who involved the spread and control of diarrheal disease under the screening of asymptomatic infective.

2. METHODOLOGY

In this work, diarrheal disease between the compartments shall be described by a system of differential equation which shall be solved to obtain both the disease-free equilibrium point and the endemic equilibrium point. The stability analysis of those equilibrium points shall be carried out by using Routh-Hurwitz stability criteria. In this study our models are analyzed in the numerical simulation by collecting the real data to classifying their steady states and to determinethe numerical value of the reproduction number.

2.1. Basic Reproduction Number

The basic reproductive number, R_0 is defined as the expected number of new infections from one infected individual in a fully susceptible population through the entire duration of the infectious period. R_0 forms a threshold quantity for most models of infectious diseases since we have for $R_0 < 1$, the disease-freeequilibrium point is locally asymptotically stable while for $R_0 > 1$, the disease-free equilibrium points are unstable. In many models, there is a trans critical bifurcation at $R_0 = 1$ and a locally asymptotically stable endemic equilibrium appears in the positive orthant for $R_0 > 1$. As [6] proposed that it is important to note that R_0 is a dimensionless number and not a rate, which would have units of time^{-1}.

2.2. The Mathematical Model

"Mathematical models can be defined as a method of emulating real-life situations with mathematical equations to expect their future behavior. In epidemiology, mathematical models play role as a tool in analyzing the spread and control of infectious diseases'' [12].

Mathematical models have been used to study the dynamics of infectious diseases for more than a century. In recent years, applications of mathematics in infectious disease have shown remarkably growing trends. As a result, separate branches like mathematical epidemiology have emerged. Rapid diagnostic test, available clinical data and electronic surveillance can facilitate the applications ofmathematical models to testing scientific hypotheses and to design practical strategies. The emerging and reemerging diseases have stimulated the interest in mathematical modelling. Models can provide estimates of underlying parameters of a real-world problem which are difficult or expensive to obtain through experiment or otherwise. By estimating transmissionrate, reproduction number and other variables and parameters a model can predict whether the associated disease will spread through the population or die out. It can also estimate the impactof a control measure and provide useful guidelines to public health for further efforts required fordisease elimination [13]. A major assumption of many mathematical models of epidemics is that the population can be divided into a set of distinct compartments. These compartments are defined with respect to disease status [7].

2.3. The model

In this study we analyze the non-linear system of ODE of diarrhea according to the following assumption by dividing the population in to five compartments susceptible, exposed, vaccinated, symptomatic infected and asymptomatic infected. Hence the total population N is

$N = S + V + E + I_A + I_S$.

Model assumption

- Susceptible(S): This represents the individuals of the whole human population that can catch the disease, The susceptible population increased by the recruitment of individuals into the population by the rate λ, the population decreased by a fraction of recruitment forvaccinated individuals at the rate ρ and by susceptible individuals who acquire diarrhea infection with effective contact with people both symptomatic and asymptomatic infectedwith diarrhea, where β is the infective contact rate and ν is the rate of the probability of asymptomatic infected. The population is increased by recovered individuals that has been treated and vaccinated individuals who lost vaccine due to vaccines wanes off at therate τ and w respectively. The susceptible individual further reduced by natural death at the rate μ.

Hence, we have: -

$$\frac{dS}{dt} = (1-\rho)\lambda - \beta S(\nu I_A + (1-\nu)I_S) + wV + \tau I_S - \mu S$$

- Vaccinated (V): This represents an individual which are take vaccination. The vaccinated individuals are increased by the fraction of vaccination from susceptible individuals at therate ρλ. This population is decreased by natural death rate and vaccine wanes off of vaccinated individuals at the rate μ and w respectively

Hence, we have

$$\frac{dV}{dt} = \rho\lambda - \mu V - wV$$

- Exposed individuals (E): Are those that carry the bacterial but not capable of infecting susceptible individuals. The population increased by new infection from susceptibleindividuals who acquire diarrhea infection with effective contact with people both symptomatic and asymptomatic infected with diarrhea, where β is the effective contact rate. The population decreased by natural death rate μ and progression from exposed class to asymptomatic and symptomatic infected class at the rate $\sigma\nu$ and $(1-\nu)\sigma$ respectively.

Hence, we have

$$\frac{dE}{dt} = \beta S(\nu I_A + (1-\nu)I_S) - (\mu + \sigma)E$$

- ✓ Asymptomatic Infected (I_A): This are an individual who have the pathogen within them and can transmit the disease but so not show any symptoms of the disease, yet they continue to contribute to the pathogen population. This population increased by the exposed individuals who develop disease symptoms at the rate $\sigma\nu$ and decreased by natural death rate and symptom gain rate μ and γ respectively.

Hence, we have

$$\frac{dI_A}{dt} = \sigma\nu E - (\mu + \gamma)I_A$$

- ✓ Symptomatic Infected (I_S) – An individual who have the pathogen and show symptoms of the disease. This group of population is fed in by the exposed population that becomes symptomatic at an infectivity rate $\sigma(1-\nu)$. And this population is increased by symptom gain rate γ and decreased by natural death rate, death induced death rate and treatment rate μ, δ and τ respectively.

Hence, we have

$$\frac{dI_S}{dt} = \sigma(1-\nu)E + \gamma I_A - (\mu + \delta + \tau)I_S$$

And the corresponding flow chart of this assumption is

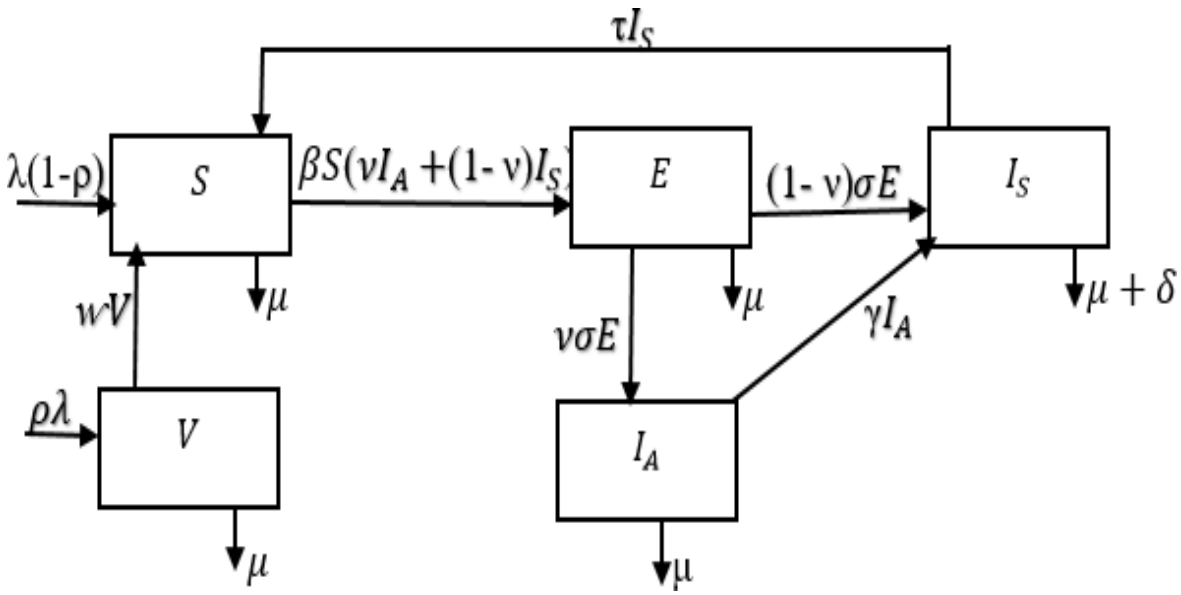

Fig1. *Flow diagram of extended model*

The dynamical system of the above flow diagram has the form:

$$\frac{dS}{dt} = (1-\rho)\lambda - \beta S(\nu I_A + (1-\nu)I_S) + wV + \tau I_S - \mu S,$$

$$\frac{dE}{dt} = \beta S(\nu I_A + (1-\nu)I_S) - (\mu+\sigma)E,$$

$$\frac{dV}{dt} = \rho\lambda - \mu V - wV,$$

$$\frac{dI_A}{dt} = \sigma\nu E - (\mu+\gamma)I_A,$$

$$\frac{dI_S}{dt} = \sigma(1-\nu)E + \gamma I_A - (\mu+\delta+\tau)I_S,$$

with initial conditions $S(0) = S_0 > 0, V(0) = V_0 \geq 0, E(0) = E_0 \geq 0, I_A(0) \geq 0, I_S(0) \geq 0.$

Table1. *descriptions of parameter and compartments of the extended model.*

Parameter andvariables	Description
S	Susceptible
V	Vaccinated
E	Exposed
I_A	Asymptomatic Infected
I_S	Symptomatic infected
ρ	Vaccine rate
λ	Recruitment rate
β	Contact rate
r	Rate at which infected individual are treated
w	Rate at which vaccine wanes off
σ	Rate at which exposed individuals become infected
μ	Natural death rate
δ	Induced disease death rate
ν	The rate of probability of asymptomatic infected
γ	Symptom gain rate

2.4. Model Analysis

Positivity of the solution

In the dynamical system a model to be epidemiologically meaningful, it is important to show that all the solutions with nonnegative initial conditions will remain nonnegative. We have thedynamical system with initial condition given as follow

$$\frac{dS}{dt} = (1-\rho)\lambda - \beta S(\nu I_A + (1-\nu)I_S) + wV + \tau I_S - \mu S,$$

$$\frac{dE}{dt} = \beta S(\nu I_A + (1-\nu)I_S) - (\mu+\sigma)E,$$

$$\frac{dV}{dt} = \rho\lambda - \mu V - wV,$$

$$\frac{dI_A}{dt} = \sigma\nu E - (\mu+\gamma)I_A,$$

$$\frac{dI_S}{dt} = \sigma(1-\nu)E + \gamma I_A - (\mu+\delta+\tau)I_S,$$

with initial condition $S(0) = S_0 > 0, V(0) = V_0 \geq 0, E(0) = E_0 \geq 0, I_A(0) \geq 0, I_S(0) \geq 0$ and all parameters are positive.

From the first differential equation we have:

$$\frac{dS}{dt} = (1-\rho)\lambda - \beta S(\nu I_A + (1-\nu)I_S) + wV + \tau I_S - \mu S$$

$\Leftrightarrow \frac{dS}{dt}+\beta S(\nu I_A + (1-\nu)I_S + \mu) = wV + \tau I_S - (1-\rho)\lambda$

$\Leftrightarrow \frac{dS}{dt}+SB(t) = wV + \tau I_S - (1-\rho)\lambda$, where $B(t) = \beta(\nu I_A + (1-\nu)I_S + \mu)$ which is first order linear ordinary differential equation with integrating factor $e^{\int_0^t B(r)dr}$ and its solution is given by: $S(t) = S_0 e^{-\int_0^t B(r)dr} \int_0^t (wV + \tau I_S + (1-\rho)\lambda) e^{\int_0^t B(r)dr} dr$ Since $S_0 > 0$ and $0 < \rho < 1 \Rightarrow 0 < (1-\rho) < 1$ and also the range of all exponential function is positive.

Therefore, $S(t) \geq 0$.

From the second differential equation we have

$\frac{dE}{dt} = \beta S(\nu I_A + (1-\nu)I_S) - (\mu+\sigma)E$ and this implies that

$\frac{dE}{dt} + (\mu+\sigma)E = \beta S(\nu I_A + (1-\nu)I_S)$ which is a first order linear differential equation with integrating factor $e^{\int_0^t (\mu+\sigma)dr} = e^{(\mu+\sigma)t}$ and the solution is given by

$E(t) = E_0 e^{-(\mu+\sigma)t} \int_0^t \beta S(\nu I_A + (1-\nu)I_S) e^{(\mu+\sigma)t} dr \geq 0$ as $E_0 \geq 0$ and the range of all exponential function is positive.

From the third differential equation we have

$$\frac{dV}{dt} = \rho\lambda - \mu V - wV$$

$\frac{dV}{dt} + (\mu+w)V = \rho\lambda$ which is first order linear differential equation with integrating factor $e^{\int_0^t (\mu+w)dr} = e^{(\mu+w)t}$ and the solution is given by $V(t) = V_0 e^{-(\mu+w)t} \int_0^t \rho\lambda e^{(\mu+w)t} dr \geq 0$ since $V_0 \geq 0$ and the range of exponential function are positive.

From the fourth differential equation we have $\frac{dI_A}{dt} = \sigma\nu E - (\mu+\gamma)I_A$ this becomes

$\frac{dI_A}{dt} + (\mu+\gamma)I_A = \sigma\nu E$ which is also first order linear differential equation with an integrating factor $e^{\int_0^t (\mu+\gamma)dr} = e^{(\mu+\gamma)t}$ and the solution is given by

$I_A(t) = I_A(0)e^{-(\mu+\gamma)t} \int_0^t \sigma\nu E\, e^{(\mu+\gamma)t} dr \geq 0$ since $I_A(0) \geq 0$ and all parameters are positive.

From the 5th differential equation is

$\frac{dI_S}{dt} = \sigma(1-\nu)E + \gamma I_A - (\mu+\delta+\tau)I_S$ and we can write this as

$\frac{dI_S}{dt} + (\mu+\delta+\tau)I_S = \sigma(1-\nu)E + \gamma I_A$ which is a first order linear differential equation with integrating factor $e^{\int_0^t (\mu+\delta+\tau)dr} = e^{(\mu+\delta+\tau)t}$. The solution of this differential equation is given by

$I_S = I_S(0)e^{-(\mu+\delta+\tau)t} \int_0^t (\sigma(1-\nu)E + \gamma I_A)\, e^{(\mu+\delta+\tau)t} dr \geq 0$ since $I_S(0) \geq 0$ and $\sigma(1-\nu) > 0$ as $0 < \nu < 1$ and the range of all exponential function is positive.

Hence, all state variables of the model system $S(t), V(t), E(t), I_S(t)$ and $I_A(t)$ are nonnegative.

Boundedness of the solution

From the previous assumption the total population $N(t) = S(t) + V(t) + E(t) + I_S(t) + I_A(t)$.

Differentiating this both sides with respect to time t we have

$$\frac{dN}{dt} = \frac{dS(t)}{dt} + \frac{dV(t)}{dt} + \frac{dE(t)}{dt} + \frac{dI_S(t)}{dt} + \frac{dI_A(t)}{dt}.$$

Substituting those values in the right-hand side of the above from the dynamical system we get: -

$$\frac{dN}{dt} = (1-\rho)\lambda - \beta S(\nu I_A + (1-\nu)I_S) + wV + \tau I_S - \mu S + \beta S(\nu I_A + (1-\nu)I_S) - (\mu+\sigma)E + \rho\lambda - \mu V - wV + \sigma\nu E - (\mu+\gamma)I_A + \sigma(1-\nu)E + \gamma I_A - (\mu+\delta+\tau)I_S$$

$$= \lambda - \mu S - \mu V - \mu E - \mu I_A - \mu I_S - \delta I_S = \lambda - \mu(S+V+E+I_A+I_S) - \delta I_S$$

But we have $N(t) = S(t) + V(t) + E(t) + I_S(t) + I_A(t)$.

Thus, $\frac{dN}{dt} = \lambda - \mu(S+V+E+I_A+I_S) - \delta I_S = \lambda - \mu N - \delta I_S$ ..... (1)

$\frac{dN}{dt} = \lambda - \mu N - \delta I_S \leq \lambda - \mu N$ because $N(t) = S(t) + V(t) + E(t) + I_S(t) + I_A(t)$.

Now, $\frac{dN}{dt} \leq \lambda - \mu N$, multiply both side by $\frac{dt}{\lambda - \mu N}$,

$\frac{dN}{\lambda - \mu N} \leq dt$. Integrating both sides we

$\int \frac{dN}{\lambda - \mu N} \leq \int dt + c$ where c is arbitrary constant. If we let $x = \lambda - \mu N$ we have

$$dx = -\mu dN$$

which implies $\frac{-dx}{\mu} = dN$.

Thus, $\int \frac{dN}{\lambda - \mu N} = \int \frac{-dx}{\mu x} \leq \int dt + c$

$$\frac{-lnx}{\mu} \leq t + c$$

$$x \geq e^{-\mu(t+c)}$$

$\lambda - \mu N \geq e^{-\mu(t+c)}$ because $x = \lambda - \mu N$.

$$N \leq \frac{\lambda - e^{-\mu(t+C)}}{\mu}$$

$N \leq \frac{\lambda}{\mu}$ since as $t \to \infty$, $\frac{\lambda - e^{-\mu(t+c)}}{\mu} \to \frac{\lambda}{\mu}$ (2)

Hence N is bounded above by $\frac{\lambda}{\mu}$.

From (1) we have $\frac{dN}{dt} = \lambda - \mu N - \delta I_S$ and we know that $N \geq I_S$ thus we have

$\frac{dN}{dt} = \lambda - \mu N - \delta I_S \geq \lambda - \mu N - \delta N = \lambda - (\mu+\delta)N$

$\frac{dN}{dt} \geq \lambda - (\mu+\delta)N$ multiply both side by $\frac{dt}{\lambda - (\mu+\delta)N}$

$\frac{dN}{\lambda - (\mu+\delta)N} \geq dt$ let $y = \lambda - (\mu+\delta)N \Rightarrow dN = -\frac{dy}{\mu+\delta}$ and taking both side integration

$\int \frac{dN}{\lambda - (\mu+\delta)N} \geq \int dt + c = t + c$ where c is an arbitrary constant.

$$\int -\frac{dy}{y(\mu+\delta)} \geq t + c$$

$$-lny \geq (\mu + \delta)(t + c)$$

$$y \leq e^{-(\mu+\delta)(t+c)}$$

$$\lambda - (\mu + \delta)N \leq e^{-(\mu+\delta)(t+c)}$$

$$\frac{\lambda - e^{-(\mu+\delta)t}}{\mu + \delta} \leq N$$

$N \geq \frac{\lambda}{\mu+\delta}$since as $t \to \infty$,then $\frac{-e^{-(\mu+\delta)(t+c)}}{\mu} \to 0$ (3)

Hence, N is bounded below.

Therefore, the solution is bounded in $\mathcal{R} = \left\{(S, V, E, I_A, I_S): N(t) = S(t) + V(t) + E(t) + I_S(t) + I_A(t), \frac{\lambda}{\mu+\delta} \leq N \leq \frac{\lambda}{\mu}\right\}$.

Equilibrium points and reproduction number of the model

The dynamical system we have two equilibrium points: Disease free equilibrium $(S^0, V^0, E^0, I_A{}^0, I_S{}^o) = \left(\frac{((1-\rho)\mu+ w)\lambda}{\mu(\mu+w)}, \frac{\rho\lambda}{\mu+w}, 0, 0, 0\right)$

and endemic equilibrium point

$$S^* = \frac{(\mu+\gamma)(\mu+\delta+\tau)(\mu+\sigma)}{\sigma\beta[\nu^2(\mu+\delta+\tau)+(1-\nu)((1-\nu)\mu+\gamma)]}$$

$$V^* = \frac{\rho\lambda}{\mu+w}$$

$$E^* = \frac{[\mu[(1-\rho)\lambda\sigma\beta[\nu^2(\mu+\delta+\tau)+(1-\nu)((1-\nu)\mu+\gamma)]-(\mu+\gamma)(\mu+\delta+\tau)(\mu+\sigma)(\mu+w)](\mu+\gamma)}{\sigma\beta[\nu^2(\mu+\delta+\tau)+(1-\nu)((1-\nu)\mu+\gamma)][(\mu+w)[(\mu+\sigma)(\mu+\gamma)(\mu+\delta+\tau)-\tau\sigma((1-\nu)\mu}$$

$$I_A{}^* = \frac{\nu\mu[(1-\rho)\lambda\sigma\beta[\nu^2(\mu+\delta+\tau)+(1-\nu)((1-\nu)\mu+\gamma)]-(\mu+\gamma)(\mu+\delta+\tau)(\mu+\sigma)(\mu+w)](\mu+\delta+\tau)}{\beta[\nu^2(\mu+\delta+\tau)+(1-\nu)((1-\nu)\mu+\gamma)][(\mu+w)[(\mu+\sigma)(\mu+\gamma)(\mu+\delta+\tau)-\tau\sigma((1-\nu)\mu+\gamma)]]}$$

$$I_S{}^* = \frac{\mu[((1-\nu)\mu+\gamma)[(1-\rho)\lambda\sigma\beta[\nu^2(\mu+\delta+\tau)+(1-\nu)((1-\nu)\mu+\gamma)]-(\mu+\gamma)(\mu+\delta+\tau)(\mu+\sigma)(\mu+w)]}{\beta[\nu^2(\mu+\delta+\tau)+(1-\nu)((1-\nu)\mu+\gamma)][(\mu+w)[(\mu+\sigma)(\mu+\gamma)(\mu+\delta+\tau)-\tau\sigma((1-\nu)\mu+\gamma)]]}$$

The reproduction number of the model is

$$R_o = \frac{\lambda\beta\sigma((1-\rho)\mu + w)[\nu^2(\mu+\delta+\tau)+(1-\nu)((1-\nu)\mu+\gamma)]}{\mu(\mu+w)(\mu+\sigma)(\mu+\gamma)(\mu+\delta+\tau)}.$$

Parameter estimation

We estimate the parameters of the model based on the collected data from Bibugn Woreda and other sources which can help as to study our model. Most parameters are identified from the collected data of Bibugn Woreda, but there exist few parameters which can be estimated based on different research findings as follow.

Table2. *Parameter values*

Parameter description	Symbol	Values
Contact rate	β	0.507538601
Natural death rate	μ	0.004166541
Death induced death rate	δ	0
Recruitment rate	λ	0.042633899
Vaccination rate	ρ	0.032969139
Treatment rate	τ	1
Progression rate	σ	0.06666667 per day
Symptom gain rate	γ	1
Vaccine wanes off rate	w	0.001369863 per day
The rate of probability of asymptomatic infected	υ	0.214653679

From [14] we do have an incubation period of diarrhea is from 1 day to 4 weeks from the time a person is exposed until symptoms start. Thus, the average incubation period of diarrhea is 15 days. Therefore,

$$\sigma = \frac{1}{\text{incubation period}} = \frac{1}{15}\ per\ day = 0.06666667\ per\ day.$$

Hence, we have

$$\upsilon\sigma = \frac{intial\ number\ of Asymptomatic\ infected\ people}{Initial\ number\ of\ exposed\ people} = \frac{I_{A(0)}}{E_0} = 0.014310246.$$

$$v = \frac{0.014310246}{0.06666667} = 0.214653679.$$

According to the reference [2] rotavirus vaccine is effective against severe rotavirus gastroenteritis in the first 2 years of life in African countries with high mortality in infants younger than 5 years. Therefore, the vaccine wanes off rate is given by:

$$w = \frac{1}{2\ \times 365\ day} = 0.001369863\ per\ day$$

Now according to the parameter value of the above table we do have The disease free equilibrium point is: $(S^0, V^0, E^0, I_A{}^0, I_S{}^o)$= (9.978560633, 0.253883748, 0, 0, 0)

and the endemic equilibrium point is:

$$(S^*, V^*, E^*, I_A{}^*, I_S{}^*) = (2.530513002, 0.253883748, 4.549633703, 0.064836235, 0.301781401)$$

Finally, we obtained the reproduction number given by R_0 = 3.943295539 > 1, this shows that the disease spread in the community.

Stability analysis of equilibrium points

We do have unstable disease-free equilibrium point and stable equilibrium point, so as the physical meaning of stability of the equilibrium points shows that; unstable equilibrium point (disease dies out) and unstable equilibrium point (the disease spreads through the community).

3. NUMERICAL SIMULATION

In this section, we consider the reproduction number of a nonlinear $SVEI_AI_SS$ mathematical model given by $R_0 = \frac{\lambda\beta\sigma((1-\rho)\mu+w)[v^2(\mu+\delta+\tau)+(1-v)((1-v)\mu+\gamma)]}{\mu(\mu+w)(\mu+\sigma)(\mu+\gamma)(\mu+\delta+\tau)}$, which depends on ten parameters and the effect of each parameter change on the reproduction number graphically using winplot software, where the parameter values are taken from table 2.

1. The graphical representation of the reproduction number versus contact rate(β) is given by:

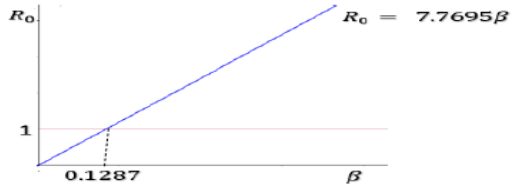

Fig 2 The graph of basic reproduction number R_0 verses contact rate β

Now the intersection point of $R_0(\beta) = 7.7695\beta$ and $R_0 = 1$ is $(\beta, R_0) = (0.1287, 1)$. from this we observe that if $\beta < 0.1287$, then $R_0 < 1$ and if $\beta > 0.1287$, then $R_0 > 1$.

2. The graphical representation of the reproduction number versus progression rate (σ) is given by:

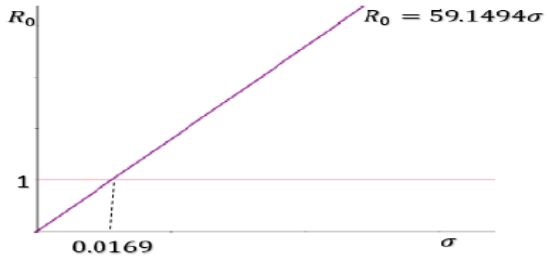

Fig 3 The graph of basic reproduction number R_0 verses progression rate σ

Now the intersection point of $R_0(\sigma) = 59.1494\sigma$ and $R_0 = 1$ is $(\sigma, R_0) = (0.0169,1)$ and we observe that if $\sigma > 0.0169$, then $R_0 > 1$ and if $\sigma < 0.0169$, then $R_0 < 1$.

3. The graphical representation of the reproduction number versus *vaccine* rate (ρ) is given by

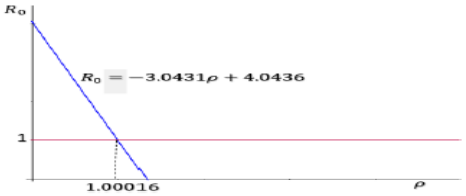

Fig 4 The graph of basic reproduction number R_0 verses vaccine rate ρ

Now the intersection point of $R_0(\rho) = -3.0431\rho + 4.0436$ and $R_0 = 1$ is $(\rho, R_0) = (1.00016,1)$ and we observe that if $\rho > 1.00016$, then $R_0 < 1$ and if $\rho < 1.00016$, then $R_0 > 1$.

4. The graphical representation of the reproduction number versus treatment τ is given by

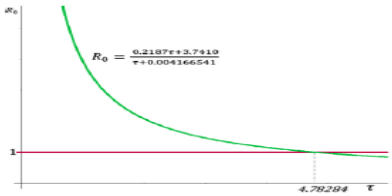

Fig 5 The graph of basic reproduction number R_0verses treatment rate τ

The intersection point of $R_0(\tau)$and $R_0 = 1$ is $(\tau, R_0) = (4.78284,1)$ And from the graph we observe that, if $\tau > 4.78284$, then $R_0 < 1$ and if $\tau < 4.78284$, then $R_0 > 1$.

4. Discussions and Results

The SVEIAISS mathematical model of diarrhea is considered to predict the effect of asymptomatic infected of diarrhea on the spread and control of diarrhea. This model was formulated to investigate the transition dynamics of diarrhea, to identifying the spread and control parameters in the model. The mathematical model of diarrhea is formulating as five- dimensional system of ordinary differential equations.

A complete qualitative and quantitative analysis was done. It is showed that the positivity of the solution as the population is not counted in the negative real number and boundedness of the solution and mathematically well-posed solution in the region. The model basic reproduction number is calculated using the next generation matrix and it was formulated as

$R_0 = \frac{\lambda\beta\sigma((1-\rho)\mu+w)[\nu^2(\mu+\delta+\tau)+(1-\nu)((1-\nu)\mu+\gamma)]}{\mu(\mu+w)(\mu+\sigma)(\mu+\gamma)(\mu+\delta+\tau)}$ which depends on ten parameters. The model has two equilibrium points which are disease free and endemic equilibrium points obtained in terms of parameter. The stability analysis of those equilibrium points was done using the Routh-Hurwitz stability criteria.

Based on the collected data from Bibugn Woreda we obtained the reproduction number given by $R_0 = 3.943295539 > 1$, this shows that the disease spread in the community and we obtained also the disease free equilibrium point $(S^0, V^0, E^0, I_A{}^0, I_S{}^0) = (9.978560633, 0.253883748, 0, 0, 0)$ which is unstable equilibrium point and the endemic equilibrium point given by

$(S^*, V^*, E^*, I_A{}^*, I_S{}^*)$ = (2.530513002,0.253883748 , 4.549633703,0.064836235,0.301781401)

which is stable equilibrium point.

From numerical simulation part we were discussed the reproduction number verses those ten parameters which are involved on the expression of reproduction number and we understood the control parameters for the spread and control of diarrhea by plotting the graph of the reproduction number verses those each parameter by fixing the remaining from the collected databy calculating their values with a preferable formula.

From fig-2: the graph of reproduction number verses contact rate β, we understood that, if the contact rate is increased($\beta > 0.1287$), then the reproduction number also increased($R_0 > 1$) meaning that the disease spread in Bibugn Woreda. And also, if the contact rate is decreased($\beta < 0.1287$), then the reproduction number also increased($R_0 < 1$) meaning that the spread of the disease will be decreased in Bibugn Woreda.

From fig-3: the graph of reproduction number verses progression rate σwe understood that, if the progression rate ($\sigma > 0.0169$) then the reproduction number also increased($R_0 > 1$) meaning that the infected population is increased and the disease spread in Bibugn Woreda and if the progression rate is decreased($\sigma < 0.0169$) then the reproduction number also decreased($R_0 < 1$) meaning that the infected group will decreased in Bibugn Woreda.

From Fig-4 the graph of reproduction number verses vaccine rateρwe understood that, if the vaccine rate is decreased($\rho < 1.00016$), then the reproduction number also increased($R_0 > 1$) meaning that if vaccination rate is low, then diarrheal disease is spread in the community and if vaccine rate ($\rho >$ 1.00016), then the reproduction number is decreased($R_0 < 1$) meaning that if vaccination rate is very high, then the spread of the disease will be reduced.

From Fig-5 the graph of reproduction number versus treatment rate$r$ we understood that, if the treatment rate is increased $r > 4.7828$, then the reproduction number is decreased($R_0 < 1$), which means that high treatment activity should be done in order to decrease the spread of diarrhea. And also, if the treatment rate is decreased $r < 4.7828$, then the reproduction number is increased($R_0 > 1$): which means that if there is no treatment or if the treatment activity is low then the disease spread in the society indefinitely.

5. Conclusion

From real parameter estimation and numerical simulation result, the researcher conclude that the disease spread in the community of Bibugn Woreda without any control measure, because of as we understood from the stability of the equilibrium point the endemic equilibrium point was stabile means the disease spread in the society and also the reproduction number is obtained as

R_0 = 3.943295539 > 1, which means that the disease is spreading in the society without any control measure. And also, as we discussed in the discussion part the asymptomatic infected of diarrhea in the Bibugn Woreda Population Is Very High.

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