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On The Stability Analysis Of Infantile Paralysis

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Abstract

The infantile paralysis model formulated by* Agarwal and Bhadauria (2011) *was modified and painstakingly examined. In the model, we considered the role of vaccine and the effectiveness of reproduction number was established. We stated and proved that both the disease-free equilibrium point and endemic equilibrium point exist and they are locally asymptotically stable when-ever the effective reproduction number, R_c , is less than unity and greater than unity respectively. Numerical simulations was carried out to confirm the analytic results and explored the possible behavior of the formulated model.

Keywords: Infantile paralysis, epidemic, polio virus, DFE, EE, Equilibrium point

1.0 Introduction

Treatments are focused on increasing comfort, managing symptoms and preventing complications. This my include providing bed rest, antibiotics for additional infections, pain killers, ventilators to help breathing, physiotherapy and moderate exercise, and a proper diet. Although polio essentially has been eradicated in the US since 1979 and in the Western Hemisphere since 1991, children and adults in Afghanistan, India, Nigeria, and Pakistan are still contending with the disease. There are two vaccines available to fight polio-inactivated polio virus (IPV) and oral polio vaccine (OPV).

IPV, which consist of a series of injections beginning two months after birth and continuing until a child is 4 to 6 years old. The vaccine is created from inactive polio virus, but it is very safe and effective and cannot cause polio. OPV is created from a weakened or attenuated form of polio virus, and it is the vaccine of choice in many countries because of its low cost, ease of administration, and ability to provide excellent immunity in the intestine.

More than 100 different types of viruses can be found in human waste and are potentially transmitted through contaminated water [1]. These viruses are more resistant to environmental conditions and sewage treatment processes, including chlorination and ultra violet radiation than many of the sewage associated bacteria. Poliovirus is categorized into three different species of the enterovirus genus (Poliovirus 1, 2 and 3) and is the causative agent of poliomyelitis [2]. The campaign for global eradication of poliomyelitis and the mass vaccination of children with the OPV vaccine had dramatically reduced the number of poliomyelitis cases caused by wild polioviruses.

Future work may intend to study the effect of infective immigration; drug resistance, imperfect prophylactic vaccines, as well as bifurcation analysis and optimal control of the model can also be investigated.

1.1 The Global Infantile Paralysis Eradication Initiative

❖ Launch

In 1988, the forty-first World Health Assembly adopted a resolution for the worldwide eradication of polio. It marked the launch of the Global Polio Eradication Initiative (GPEI), spearheaded by national governments, WHO, Rotary International, the US Centers for Disease Control and Prevention (CDC), UNICEF, and supported by key partners including the Bill and Melinda Gates Foundation. This followed the certification of the eradication of smallpox in 1980, progress during the 1980s towards elimination of poliovirus in America, and Rotary International's commitment to raise funds to protect all children from the disease.

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❖ **Progress**

Overall, since the GPEI was launched, the number of cases has fallen by over 99%. In 2014, only 3 countries in the world remain polio-endemic: Nigeria, Pakistan and Afghanistan.

In 1994, the WHO Region of the Americas was certified polio-free, followed by the WHO Western Pacific Region in 2000 and the WHO European Region in June 2002. On 27 March 2014, the WHO South-East Asia Region was certified polio-free, meaning that transmission of wild poliovirus has been interrupted in this bloc of 11 countries stretching from Indonesia to India. This achievement marks a significant leap forward in global eradication, with 80% of the world's population now living in certified polio-free regions.

Of the 3 types of wild poliovirus (type 1, type 2 and type 3), type 2 wild poliovirus transmission has been successfully stopped (since 1999).

More than 10 million people are today walking, who would otherwise have been paralyzed. An estimated more than 1.5 million childhood deaths have been prevented, through the systematic administration of Vitamin A during polio immunization activities.

❖ **Opportunity and risks: an emergency approach**

The strategies for polio eradication work when they are fully implemented. This is clearly demonstrated by India's success in stopping polio in January 2011, in arguably the most technically-challenging place, and polio-free certification of the entire South-East Asia Region of the World Health Organization occurred in March 2014.

However, failure to implement strategic approaches leads to ongoing transmission of the virus. Endemic transmission is continuing in Nigeria, Pakistan and Afghanistan. Failure to stop polio in these last remaining areas could result in as many as 200 000 new cases every year, within 10 years, all over the world.

Recognizing both the epidemiological opportunity and the significant risks of potential failure, the new Polio Eradication and Endgame Strategic Plan 2013-2018 has been developed, in consultation with polio-affected countries, stakeholders, donors, partners and national and international advisory bodies. The new Plan was presented at a Global Vaccine Summit in Abu Dhabi, United Arab Emirates, at the end of April 2013. It is the first plan to eradicate all types of polio disease simultaneously – both due to wild poliovirus and due to vaccine-derived polioviruses.

Global leaders and individual philanthropists signaled their confidence in the Plan by pledging three-quarters of the Plan's projected US$5.5 billion cost over the 6 years. They also called upon additional donors to commit upfront the additional US$1.5 billion needed to secure a lasting polio-free world.

❖ **Future benefits of Infantile Paralysis eradication**

Once polio is eradicated, the world can celebrate the delivery of a major global public good that will benefit all people equally, no matter where they live. Economic modeling has found that the eradication of polio would save at least US$ 40–50 billion over the next 20 years, mostly in low-income countries. Most importantly, success will mean that no child will ever again suffer the terrible effects of lifelong polio-paralysis.

1.1 Infantile Paralysis in Nigeria

The 9th meeting of the Expert Review Committee (ERC) on polio Eradication in Nigeria, 2006, revealed that, poliovirus can travel from state to state and village to village and country to country through un-immunize children. Rapid progress continues to be made towards polio eradication in Nigeria. The number of polio cases has reduced tremendously. As of 6th May, 2005, Nigeria has 78 confirmed cases of wild poliovirus, in 18 states. This compares to 125 cases in 25 states in the same period in 2004. The number of Nigerian children crippled by polio increased from 355(total) in 2003 to 792 cases in 2004. Nigeria accounted for 63% of the total number of poliovirus cases reported globally in 2004. Currently 70% of Nigeria's poliovirus cases are children below 3 years of age and 67% have received less than 3 doses of OPV. 6 states in the northwest – Kano, Kaduna, Sokoto, Zamfara, Kebbi, and Jigawa account for 73% of the total number of children paralyzed by polio.

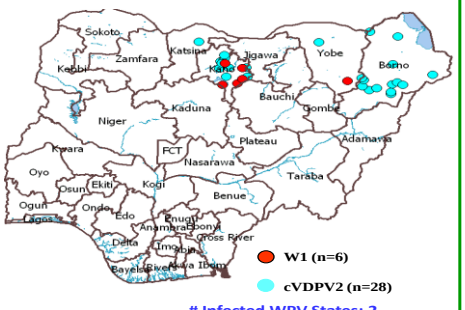
There has been a downward trend in the number of cases since May 2004. This indicates a degree of control of the infection in the southern states, along with the impact of resumed immunization in the north. Polio anywhere is a threat to children everywhere. It is easy to forget that polio once crippled over 1000 children every day. We are now on the brink of eradicating this crippling disease globally. But if we stop our efforts now, we will again see a resurgence of polio across Nigeria. Health groups are working towards wiping out polio throughout the world, and much progress has been made. But several countries still have polio circulating, which means that the virus could occur in others. If the polio reaches a country where not enough people have been immunized, it could spread from person to person, just as it happened in some countries in Africa and Asia. So until it has been eliminated worldwide, it is important to continue vaccinating kids against poliovirus. Two fresh infections of wild polio virus in early June, 2016 have emerged in Gwarzo and Jere local Government Areas of Borno State, Nigeria, threatening Nigeria's progress towards a polio free status. It was due to be declared polio free by the World Health Organization [3] if it made it to July 24th 2017 without a new case. The last known infection was July, 2014. There had been concern about children on camps for displaced people in Borno missing immunization as Nigeria struggled to prevent a flare up. Vaccines are live attenuated and can replicate in the gastrointestinal tract, inducing local intestinal as well as long lived systemic immunity [4]. One of the disadvantages of the mass vaccination is the dissemination of live attenuated individuals (such as immune deficient ones) and cause poliomyelitis. The fecal contamination by

aerosols from waste water treatment plants could potentially be another source of virus contamination for humans. Information was retrieved from [3] and [7].

Many authors had worked on body paralysis, a non-linear mathematical model for the spread of polio in a population with variable size structure including the role of vaccination as proposed and analyzed by [5]. Similarly, a model of S-I-R type for the spread of polio virus disease was formulated by [6]. We used the Derrick and Grossman Theorem that satisfies the Lipschitz condition such that the existence and uniqueness solution holds. This paper extend the work of [5] by considering a class population of recovered but deformed individuals by reflection on the Oral polio vaccine induce immunity and study its effect on the entire population using all the model analysis tools described by the aforementioned authors.

Table 1: Polio Cases in Nigeria

Nigeria: Polio cases as at Week 1, 2015



As at January 2, 2015 Nigeria has:

- 6 confirmed WPV1 in 2 States in 2014 compared to 50 cases in 9 States for the same period in 2013.
  - Date of onset of latest WPV case is 24 July, 2014
- There have been no confirmed WPV3 since November 2012.
- 28 confirmed vaccine derived poliovirus (cVDPV2) in 5 States compared to 4 cases in 2 States for the same period in 2013.
  - Date of onset of latest cVDPV2 case is 02 November, 2014

Table 2: Polio Cases by Zone and States

Nigeria: 2013/2014 (Jan – Dec) as at week 1, 2015 WPV & cVDPV2 cases by Zone and States

S/No	Zone	State	2014 New Results Received this week				2014				2013			
			WPV1	WPV3	cVDPV2	Total	WPV1	WPV3	cVDPV 2	Cum Total	WPV1	WPV3	cVDPV2	Cum Total
1	NC	FCT, Abuja	-	-	-	-	0	0	0	0	1	0	0	1
2		Nasarawa	-	-	-	-	0	0	0	0	1	0	0	1
3		Niger	-	-	-	-	0	0	0	0	1	0	0	1
4	NW	Jigawa	-	-	-	-	0	0	2	2	0	0	0	0
5		Kaduna	-	-	-	-	0	0	0	0	0	0	0	0
6		Kano	-	-	-	-	5	0	10	15	15	0	0	15
7		Katsina	-	-	-	-	0	0	1	1	0	0	0	0
8		Kebbi	-	-	-	-	0	0	0	0	0	0	0	0
9		Sokoto	-	-	-	-	0	0	0	0	0	0	0	0
10		Zamfara	-	-	-	-	0	0	0	0	0	0	0	0
11	NE	Adamawa	-	-	-	-	0	0	0	0	0	0	1	1
12		Bauchi	-	-	-	-	0	0	0	0	6	0	0	6
13		Borno	-	-	-	-	0	0	14	14	17	0	4	21
14		Gombe	-	-	-	-	0	0	0	0	1	0	0	1
15		Taraba	-	-	-	-	0	0	0	0	3	0	0	3
16		Yobe	-	-	-	-	1	0	1	2	8	0	0	8
TOTAL			-	-	-	-	6	0	28	34	53	0	5	58

Latest onset of confirmed WPV was 24-Jul-2014 from Sumaila LGA, Kano State.

2.0 Formulation of the Model

The mathematical model is formulated to describe the transmission dynamics of Poliovirus. The progression of polio within the total population can be simplified to six differential equations. These six equations represent six different groups of people.

The unvaccinated susceptible individuals S(t), vaccinated individuals V(t); asymptomatic individuals E(t), symptomatic individuals I(t); recovered but deformed individuals R_D (t) and recovered individuals R(t).

The total population N(t) is given as

$$N(t) = S(t) + V(t) + E(t) + I(t) + R_D(t) + R(t) \quad (1)$$

2.1 Assumptions of the model

In order to construct a detailed realistic model on polio virus, the following assumptions were made;

(1) The force of infection Γ is defined as

$$\Gamma = \beta[rE + I]$$

(2) Treatment is administered to individuals in I compartment, since polio associated symptom is detectable.

(3) Individuals in R compartment are assumed to posses permanent immunity against polio infection.

(4) Individuals in R_D Compartment are assumed to be recovered but deformed.

2.2 Model equations

The model takes the form of the following deterministic system of non- linear equations.

$$\frac{dS}{dt} = h_1 + \theta_2 V - \Gamma S - b_1 S \quad (2)$$

$$\frac{dV}{dt} = h_2 + \theta_1 S - (1-\epsilon)\Gamma V - b_2 V \quad (3)$$

$$\frac{dE}{dt} = \Gamma S + (1-\epsilon)\Gamma V - b_3 E \quad (4)$$

$$\frac{dI}{dt} = \gamma_1 E - b_4 I \quad (5)$$

$$\frac{dR_D}{dt} = gI - b_5 R_D \quad (6)$$

$$\frac{dR}{dt} = \gamma_2 I + \eta V - \mu R \quad (7)$$

Where

$$\left.\begin{array}{l} b_1 = \mu + \theta_1, \quad b_2 = \theta_2 + \eta + \mu, \quad b_3 = \mu + \gamma_1, \quad b_4 = \mu + \delta_1 + g + \gamma_2, \quad b_5 = \mu + \delta_2 \\ h_1 = (1-\rho)\Lambda + (1-\phi)\pi, \quad h_2 = \rho\Lambda + \phi\pi, \end{array}\right\} (8)$$

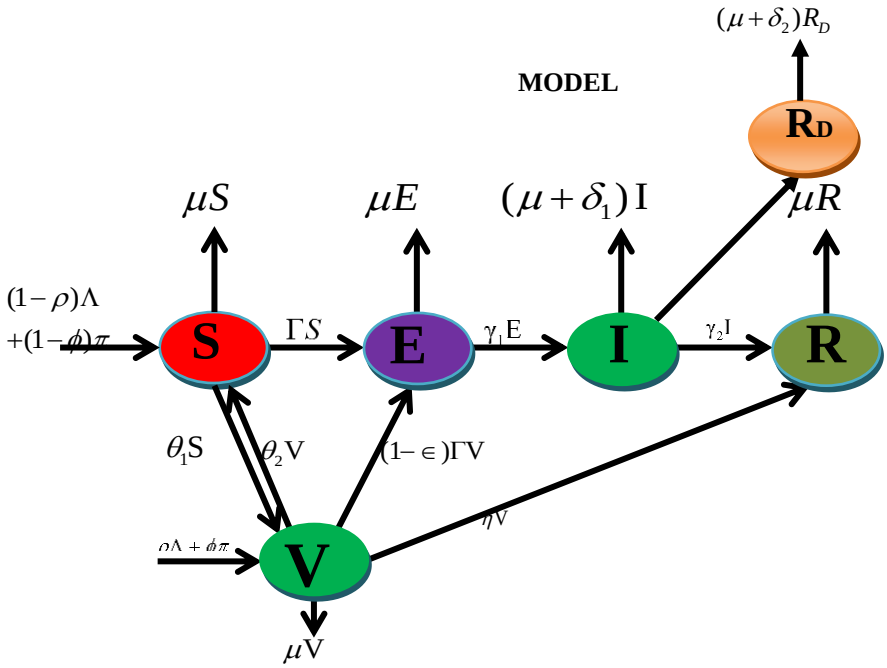


Fig. 1: Schematic diagram of the

Table 3: Variable Description

Variable	Interpretation
S	Population of unvaccinated susceptible individuals
V	Population of vaccinated individuals
E	Population of asymptomatic infected individuals
I	Population of symptomatic infected individuals
R_D	Population of recovered but deformed individuals
R	Population of recovered individuals.

Table 4: Parameter Definition

Parameter	Interpretation
π	Immigration rate
ϕ	Rate of vaccinated migrant
Λ	Recruitment rate
ρ	Vaccination rate of new born
θ_1	Vaccination rate of susceptible individuals
θ_2	Waning rate of Oral polio Vaccine
$\in$	Efficacy of Oral Polio Vaccine
β	Effective contact rate
δ_1	Polio induced death rate of I
δ_2	Polio induced death rate despite recovery, as a result of deformation
γ_1	Progression rate from E to I
γ_2	Recovery rate of I individuals due to treatment
μ	Natural death rate
η	Oral Polio vaccine induce immunity
M	Deformation rate from I to R_D
r	Disease transmission coefficient

3.0 Existence and uniqueness solution of the model

The validity and usability of any mathematical model depends on whether the given set of equations has a solution, if it has, is the solution Unique?

This section is concern with finding if the system of equations has a solution and if the solution to the system is unique. We shall use the lipchtiz condition to verify the existence and uniqueness of equations. Let

$$\left.\begin{aligned} f_1 &= h_1 + \theta_2 V - \Gamma S - b_1 S \\ f_2 &= h_2 + \theta_1 S - (1- \in)\Gamma V - b_2 V \\ f_3 &= \Gamma S + (1- \in)\Gamma V - b_3 E \\ f_4 &= \gamma_1 E - b_4 I \\ f_5 &= gI - b_5 R_D \\ f_6 &= \gamma_2 I + \eta V - \mu R \end{aligned}\right\} \quad (9)$$

Using Derrick and Grossman, let D denotes the region $|t - t_0| \le a, \quad \|x - x_0\| \le b,\ x = (x_1, x_2, ...x_n), x_0 = (x_{10}, x_{20}, ...x_n)$ and suppose that $f(t,x)$ satisfies the Lipschitz condition $\|f(t,x) - f(t,x_2)\| \le k\|x_1 - x_2\|$. Whenever the pairs (t, x_1) and (t, x_2) belong to D, where k is a positive constant, then there is a constant $\delta > 0$ such that there exist a unique continuous vector solution $x(t)$ of the system in the interval $t - t_0 < \delta$.

It is important to note that the condition is satisfied by requirement that $\frac{\partial f_i}{\partial x_i}, i = 1, 2, ...,$ be continuous and bounded in D.

We now return to our model equations. We are interested in the region
$0 \le \alpha \le m$.

We look for a bounded solution in this region and whose partial derivatives satisfy $0 \le \alpha < \infty$, where α and δ are positive constants.

Let D denote the region $0 \le \alpha \le m$, then the model equations have a unique solution. We show that $\frac{\partial f_i}{\partial x_i}, i = 1, 2, 3, 4, 5$ are continuous and bounded in D.

Recall that

$$f_1 = h_1 + \theta_2 V - \Gamma S - b_1 S$$
$$f_2 = h_2 + \theta_1 S - (1 - \in)\Gamma V - b_2 V$$
$$f_3 = \Gamma S + (1 - \in)\Gamma V - b_3 E$$
$$f_4 = \gamma_1 E - b_4 I$$
$$f_5 = gI - b_5 R_D$$
$$f_6 = \gamma_2 I + \eta V - \mu R$$

Taking the partial derivative of the above equations, we have

$$\left|\frac{\partial f_1}{\partial S}\right| = \left|-(\Gamma + b_1)\right| < \infty; \quad \left|\frac{\partial f_1}{\partial V}\right| = |\theta_2| < \infty; \quad \left|\frac{\partial f_1}{\partial E}\right| = |-\beta r S| < \infty$$

$$\left|\frac{\partial f_1}{\partial I}\right| = |-\beta S| < \infty; \quad \left|\frac{\partial f_1}{\partial R_D}\right| = 0 < \infty; \quad \left|\frac{\partial f_1}{\partial R}\right| = 0 < \infty$$

$$\left|\frac{\partial f_6}{\partial S}\right| = \left|\frac{\partial f_6}{\partial E}\right| = \left|\frac{\partial f_1}{\partial R_D}\right| = 0 < \infty; \left|\frac{\partial f_6}{\partial I}\right| = |\gamma_2| < \infty; \quad \left|\frac{\partial f_6}{\partial V}\right| = |\eta| < \infty; \quad \left|\frac{\partial f_6}{\partial R}\right| = |-\mu| < \infty$$

As clear shown above, the partial derivatives of the whole system of equation exist, they are finite and bounded. Hence, by the model system equations, it shows that, it has a unique solution.

4.0 Equilibrium State and Stability Analysis of the Model

Since the Uniqueness solution exists, we now find the existence of Equilibrium point and DFE and EE points and the local stability of DFE and EE point.

4.1 Existence of equilibrium point of the model

At equilibrium, the left hand side of the equations are equated to zero, i.e

$$\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR_D}{dt} = \frac{dR}{dt} = 0$$

Thus it becomes

$$h_1 + \theta_2 V - \Gamma S - b_1 S = 0 \qquad (10)$$
$$h_2 + \theta_1 S - (1 - \in)\Gamma V - b_2 V = 0 \qquad (11)$$
$$\Gamma S + (1 - \in)\Gamma V - b_3 E = 0 \qquad (12)$$
$$\gamma_1 E - b_4 I = 0 \qquad (13)$$
$$gI - b_5 R_D = 0 \qquad (14)$$
$$\gamma_2 I + \eta V - \mu R = 0 \qquad (15)$$

From (10), we have

$$S = \frac{h_1 + \theta_2 V}{\Gamma + b_1} \qquad (16)$$

Similarly from (11) we get

$$S = \frac{\left[\left(1-\in\right)\Gamma + b_2\right]V - h_2}{\theta_1} \qquad (17)$$

Substituting (17) into (16) to obtain

$$S = \frac{\left[\left(1-\in\right)\Gamma + b_2\right]h_1 + \theta_2 h_2}{\left[\left(1-\in\right)\Gamma + b_2\right]\left[\Gamma + b_1\right] - \theta_1\theta_2} \qquad (18)$$

For convenience, (17) and (18) are re written as

$$V = \frac{h_1\theta_1 + h_2\left[\Gamma + b_1\right]}{T_0} \qquad (19)$$

$$S = \frac{\left[\left(1-\in\right)\Gamma + b_2\right]h_1 + \theta_2 h_2}{T_o} \qquad (20)$$

Where

$T_0 = \left[\left(1-\in\right)\Gamma + b_2\right]\left[\Gamma + b_1\right] - \theta_1\theta_2$, it is important to note that

$T_0 > 0$ since $b_1 b_2 - \theta_1\theta_2 = \left(\mu + \theta_1\right)\left(\eta + \mu\right) + \theta_2\mu > 0 \; and \quad \in < 1$

From (13), we have

$$I = \frac{\gamma_1 E}{b_4} \qquad (21)$$

Substituting (21) into Γ, recalling that

$$\Gamma = \beta\left(I + rE\right)$$

Thus

$$\Gamma = \beta\left(\frac{\gamma_1 E}{b_4} + rE\right)$$

$$\Gamma = \frac{\beta\left(\gamma_1 + rb_4\right)E}{b_4}$$

$$\therefore E = \frac{\Gamma b_4}{\beta\left(\gamma_1 + rb_4\right)} \qquad (22)$$

From(14) $R_D = \frac{gI}{b_5}$ (23)

From (15)

$$R = \frac{\gamma_2 I + \eta V}{\mu} \qquad (24)$$

With the above equations, we have,

$$S + \left(1-\in\right)V = \frac{\left[\left(1-\in\right)\Gamma + b_2\right]h_1 + \theta_2 h_2}{T_o} + \frac{\left(1-\in\right)\left\{h_1\theta_1 + h_2\left[\Gamma + b_2\right]\right\}}{T_0}$$

$$S + \left(1-\in\right)V = \frac{\left(1-\in\right)\left(h_1 + h_2\right)\Gamma + h_1\left[\theta_1\left(1-\in\right) + b_2\right] + h_2\left[b_1\left(1-\in\right) + \theta_2\right]}{T_o} \qquad (25)$$

From (12) we obtain

$$b_3 E = \Gamma\left[S + \left(1-\in\right)V\right] \qquad (26)$$

Substitute (22) into (26) to get

$$\frac{b_3 b_4 \Gamma}{\beta\left(\gamma_1 + rb_4\right)} = \Gamma\left[S + \left(1-\in\right)V\right]$$

$$\Gamma\left\{b_3 b_4 - \beta\left(\gamma_1 + rb_4\right)\left[S + \left(1-\in\right)V\right]\right\} = 0$$

Thus, either

$$\Gamma = 0 \ \ or \ \ S + (1-\in)V = \frac{b_3 b_4}{\beta(\gamma_1 + rb_4)} \tag{27}$$

From (23)

$$R_D = \frac{g\gamma_1}{b_4 b_5}\left\{\frac{\Gamma b_4}{\beta(\gamma_1 + rb_4)}\right\} \tag{28}$$

4.2 Existence Disease Free Equilibrium of the model

In the absence of infection, i.e $\Gamma^* = 0$, we have $E^* = I^* = 0$

Let ε_0 denote the disease free equilibrium, such that

$$\varepsilon_0 = \left(S^* V^* E^* I^* I_c^* R^*\right) = \left(\frac{h_1 b_2 + h_2\theta_2}{b_1 b_2 - \theta_1\theta_2}, \frac{h_1\theta_1 + h_2 b_1}{b_1 b_2 - \theta_1\theta_2}, 0, 0, 0, \frac{\eta\left[h_1\theta_1 + h_2 b_1\right]}{\mu\left[b_1 b_2 - \theta_1\theta_2\right]}\right)$$

5.0 Computation of Reproductive Number of the model

Effective reproduction number is the parameter that estimates the average number of secondary cases per infectious case in a population made up of both susceptible and non- susceptible hosts [11]. It is obtained by taking the largest (dominant) eigen-value (spectral radius) of the next generation matrix [10].

$$R_0 = \left[\frac{\partial f_i(x_0)}{\partial(x_j)}\right]\left[\frac{\partial V_i(x_0)}{\partial x_i}\right]^{-1}$$

$$\therefore R_0 = \frac{\beta\left\{h_1\left[\theta_1 + (1-\in) + b_2\right] + h_2\left[b_1(1-\in) + \theta_2\right]\right\}(rb_4 + \gamma_1)}{\left[b_1 b_2 - \theta_1\theta_2\right] b_3 b_4} \tag{29}$$

6.0 Local stability of DFE of the model

The Disease free equilibrium ε_0 is locally asymptotically stable whenever $R_c < 1$ and unstable whenever $R_c > 1$

The evaluated Jacobian matrix at ε_0 is given as

$$J(\varepsilon_0) = \begin{bmatrix} -b_1 & \theta_2 & -\beta r S^* & -\beta S^* \\ \theta_1 & -b_2 & -(1-\in)\beta V^* & -(1-\in)\beta V^* \\ 0 & 0 & \beta r\left[S^* + (1-\in)V^*\right] - b_3 & \beta\left[S^* + (1-\in)V^*\right] \\ 0 & 0 & \gamma_1 & -b_4 \end{bmatrix}$$

Thus, the characteristics equation of $J(\varepsilon_0)$ is obtain as

$$\left[a_4\lambda^4 + a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0\right] = 0$$

Where the coefficient of the Eigen value (i.e λ) in the above equation are expressed in a simplified form as

$$a_4 = 1$$

$$a_3 = \frac{b_4\left[b_4 + b_2 + b_1\right] + Q\gamma_1 + b_3 b_4(1 - R_0)}{b_4}$$

$$a_2 = \frac{b_3 b_4\left[\gamma_1 + r(b_4 + b_2 + b_1)\right](1 - R_0) + (b_1 b_2 - \theta_1\theta_2)(\gamma_1 + rb_4) + (b_2 + b_1)\left[\gamma_1 b_3 + b_4(\gamma_1 + rb_4)\right]}{(\gamma_1 + rb_4)}$$

$$a_1 = \frac{\left[b_4(\gamma_1 + rb_4) + b_3\gamma_1\right]\left[b_1 b_2 - \theta_1\theta_2\right] + b_3 b_4\left[(b_2 + b_1)(\gamma_1 + rb_4) + r(b_1 b_2 - \theta_1\theta_2)\right](1 - R_0)}{(\gamma_1 + rb_4)}$$

$a_0 = b_3 b_4 \left(b_1 b_2 - \theta_1 \theta_2\right)\left(1 - R_0\right)$

Hence, the Disease Free Equilibrium of the model is locally asymptotically Stable.

7.0 Existence of Endemic Equilibrium Point of the model

In the presence of infection, i.e $I^{**} \neq 0,\ E^{**} \neq 0,$ thus $\Gamma^{**} \neq 0$ .We have

$$S^{**} + (1-\in)V^{**} = \frac{b_3 b_4}{\beta(\gamma_1 + rb_4)} \tag{30}$$

Substituting into the equation, we have

$$\frac{(1-\in)(h_1+h_2)\Gamma^{**} + h_1\left[\theta_1(1-\in)+b_2\right] + h_2\left[b_1(1-\in)+\theta_2\right]}{T_o} = \frac{b_3 b_4}{\beta(\gamma_1 + rb_4)}$$

$$\beta(\gamma_1 + rb_4)(1-\in)(h_1+h_2)\Gamma^{**} + \beta(\gamma_1 + rb_4)\left\{h_1\left[\theta_1(1-\in)+b_2\right] + h_2\left[b_1(1-\in)+\theta_2\right]\right\} = b_3 b_4 T_0 \tag{31}$$

$$b_3 b_4 T_0 = b_3 b_4\left[\left[(1-\in)\Gamma^{**} + b_2\right]\left[\Gamma^{**} + b_1\right] - \theta_1\theta_2\right]$$

$$b_3 b_4 T_0 = b_3 b_4\left[(1-\in)\Gamma^{**2} + \left(b_1(1-\in)+b_2\right)\Gamma^{**} + b_1 b_2 - \theta_1\theta_2\right]$$

$$b_3 b_4 T_0 = b_3 b_4(1-\in)\Gamma^{**2} + b_3 b_4\left[b_1(1-\in)+b_2\right]\Gamma^{**} + b_3 b_4\left(b_1 b_2 - \theta_1\theta_2\right) \tag{32}$$

Substitute (32) into (31) we have

$$b_3 b_4(1-\in)\Gamma^{**2} + b_3 b_4\left[b_1(1-\in)+b_2\right]\Gamma^{**} + b_3 b_4\left(b_1 b_2 - \theta_1\theta_2\right) = \beta(\gamma_1 + rb_4)(1-\in)(h_1+h_2)\Gamma^{**}$$
$$+ \beta(\gamma_1 + rb_4)\left\{h_1\left[\theta_1(1-\in)+b_2\right] + h_2\left(b_1(1-\in)+\theta_2\right)\right\}$$

$$b_3 b_4(1-\in)\Gamma^{**2} + b_3 b_4\left[b_1(1-\in)+b_2\right]\Gamma^{**} - \beta(\gamma_1 + rb_4)(1-\in)(h_1+h_2)\Gamma^{**} + b_3 b_4\left(b_1 b_2 - \theta_1\theta_2\right)$$
$$- \beta(\gamma_1 + rb_4)\left\{h_1\left[\theta_1(1-\in)+b_2\right] + h_2\left(b_1(1-\in)+\theta_2\right)\right\} = 0$$

$$b_3 b_4(1-\in)\Gamma^{**2} + \left\{b_3 b_4\left[(1-\in)+b_2\right] - \beta(\gamma_1 + rb_4)(1-\in)(h_1+h_2)\right\}\Gamma^{**}$$
$$+ b_3 b_4\left(b_1 b_2 - \theta_1\theta_2\right) - \beta(\gamma_1 + rb_4)\left\{h_1\left[\theta_1(1-\in)+b_2\right] + h_2\left(b_1(1-\in)+\theta_2\right)\right\} = 0 \tag{33}$$

Equation (33) is rewritten in this form

$$A\Gamma^{**2} + B\Gamma^{**} + C = 0$$

Where

$$A = b_3 b_4(1-\in)$$

$$B = b_3 b_4\left[b_1(1-\in)+b_2\right] - \beta\left[\gamma_1 + rb_4\right](1-\in)\left[h_1+h_2\right]$$

$$C = b_3 b_4\left[b_1 b_2 - \theta_1\theta_2\right]\left[1 - R_0\right]$$

Thus, $A > 0$ since $\in < 1$ and $C < 0$, if and only if $R_{c1} > 1$. Hence the theorem below is established.

The Model equations has a unique positive (endemic) equilibrium if and only if $R_{c1} > 1$

8.0 Local Stability of Endemic Equilibrium Point of the model: Special Case

The local stability of the unique endemic equilibrium ε_1 is investigated for the special case, where the imperfectness of the prophylactic vaccine (i.e OPV) is negligible or assumed to be absence i.e $\varepsilon = 1$ thus the associated reproductive number denoted by R_{c1} is given as

$$R_{c1} = \frac{\beta\left\{h_1 b_2 + h_2\theta_2\right\}\left(rb_4 + \gamma_1\right)}{\left(b_1 b_2 - \theta_1\theta_2\right)b_3 b_4} \tag{34}$$

The endemic equilibrium for a special case when $\varepsilon = 1,$ denoted by $\varepsilon_2 = \varepsilon_1 / \varepsilon = 1$ is locally asymptotically stable whenever $R_{c1} > 1$ and whenever $R_{c1} < 1$.

The variational matrix of the model evaluated at $\varepsilon_2 = \varepsilon_1 / \varepsilon = 1$ is gotten as

$$J(\varepsilon_2) = \begin{bmatrix} \dfrac{-(\theta_2 V^{**} + h_1)}{S} & \theta_2 & -\beta r S^{**} & -\beta S^{**} \\ \theta_1 & -b_2 & 0 & 0 \\ \Gamma & 0 & -\dfrac{\beta S^{**} I^{**}}{E^{**}} & \beta^{**} S^{**} \\ 0 & 0 & \gamma_1 & -b_4 \end{bmatrix}$$

Where

$$\frac{\theta_2 V^{**} + h_1}{S^{**}} = \Gamma^{**} + b_1$$

$$-\frac{\beta S^{**} I^{**}}{E^{**}} = \beta r S^{**} - b_3 \qquad where \ \in = 1$$

Thus, the characteristic equation is obtained as

$$\left[\lambda^4 + a_0\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3\right] = 0$$

Where

$$a_0 = \frac{(b_4 + b_2)E^{**}S^{**} + \beta S^{**2} I^{**} + E^{**}(\theta_2 V^{**} + h_1)}{E^{**}S^{**}}$$

$$a_1 = \frac{(b_4 E^{**} + \beta S^{**} I^{**})(b_2 S^{**} + \theta_2 V^{**} + h_1) + E^{**}(h_2\theta_2 + h_1 b_2) + \Gamma^{**}\beta r S^{**2} E^{**}}{E^{***} S^{**}}$$

$$a_2 = \frac{\beta S^{**2}\Gamma^{**}E^{**}(\gamma_1 + r b_4) + b_4 E^{**}(h_2\theta_2 + b_2 h_1) + \beta S^{**2}\Gamma^{**} r E^{**} b_2}{E^{**}S^{**}}$$

$$a_3 = \beta\Gamma^{**} b_2 S^{**}\left[\gamma_1 + r b_4\right]$$

It is clearly seen that $a_i > 0, \ \forall i = 0,....3$ only if $R_0 > 1$, hence we concluded the proof since the Routh Hurtwiz Criterion is satisfied, that the system is locally asymptotically stable.

8.1 Numerical Simulation of the Model

We simulated numerically according to the value of the parameters in the table below. The parameters were estimated accordingly and we discussed their stability as follows:

Table 5: Numerical Simulation of the model

θ_1	θ_2	ε	η	G	R_0	$E^{**} + I^{**}$	Remark
0.8	0	0.9	1	0	0.77	0	Unstable
0.8	0.2	0.9	0.8	0.2	0.95	0	Unstable
0.8	0.2	0.8	0.8	0.4	0.91	0	Unstable
0.8	0.2	0.8	0.6	0.4	1.07	30.58	Stable
0.8	0.4	0.8	0.6	0.4	1.44	143.87	Stable
0.8	0.4	0.6	0.6	0.4	1.74	229.00	Stable
0.8	0.4	0.6	0.4	0.6	2.07	265.30	Stable
0.8	0.6	0.6	0.4	0.6	2.52	309.13	Stable

Discussion of Result of the Model

In our model, people are susceptible, infected or recovered. Susceptible individuals may become infected by drinking or eating contaminated water or food. We proposed and analyzed a deterministic mathematical model in an attempt to understanding the role of vaccine as a means of reducing the spread of the disease in the society.

After comparing the model effective reproductive number, the parameters used, their estimated values with its appropriate source, we obtained values for R_0 and comparing it to the exposed and infected class, the result shows that, it is unstable whenever the sum of the two classes is less than unity otherwise stable when it is greater than unity.

The system of equations were tested for existence and uniqueness of solution, analyzed for criteria under which it is stable and the reproduction number for both the endemic and disease free equilibrium were obtained.

This section simulates the system of equation using hypothetical data and presented the table as indicated. The hypothetical values were used to simulate the value of reproduction number at both the disease free and the endemic equilibrium with the aid of Maple 18 software and the result was tabulated.

In this paper, we discussed the dynamic of Poliovirus transmission with the usage of immunization vaccine. We discovered that:

i. The model is positively invariant.
ii. The model has both disease free and endemic equilibrium.
iii. The model has a local asymptotic stability at disease free equilibrium when the effective reproduction number was proved to be less than unity.
iv. The model has a local asymptotic stability at the endemic equilibrium point when the effective reproduction number was proved to be greater than unity.
v. The numerical simulation result obviously revealed that the immunization vaccine (under the basic assumptions) will work perfectly in the community.

9.0 Conclusion

Until the 1950s, polio has crippled thousands of children every year in industrialized countries. Soon after the introduction of effective vaccines in the late 1950s (IPV) and early 1960s (OPV), polio was brought under control, and practically eliminated as a public health problem in industrialized countries. It took somewhat longer for polio to be recognized as a major problem in developing countries, but in the early 1970s during the routine immunization with OPV as part of national of national immunization programmes (Expanded Programme on Immunization, or EPI programme) was introduced worldwide helping, to control the disease in many developing countries. Today, the disease has been eliminated from most of the world, and only four countries worldwide remain polio-endemic (India, Pakistan, Afghanistan and Nigeria).

However, in this paper, we have shown the transmission dynamics of poliovirus, describing the rate of change of susceptible unvaccinated, vaccinated, asymptomatic, symptomatic, recovered and recovered but deformed individuals, in a population with respect to time. We formulate a mathematical model of the dynamics of poliovirus epidemic with the six compartments and assume some hypothetical values of parameters, where the compartments interact with each other. It has been shown that the proposed model has both disease free and endemic equilibrium points. The disease free equilibrium is shown to be locally asymptotically stable whenever $R_c < 1$ and unstable whenever $R_c > 1$. The simulated result depicts the efficacy of the model as a tool for measuring the dynamics of poliovirus transmission in a population.

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