

Mathematical Analysis Of Effect Of Area On The Dynamical Spread Of Measles

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Abstract: - This paper presents a robust compartmental mathematical model of (SVEIR) which incorporated area only. Where this area is the size of the environment where the study is being investigated. It shows that model has a disease free equilibrium which is globally asymptotically stable (GAS). There exists a unique endemic equilibrium point which is locally stable whenever the association threshold quantity (R_0) exceeds unity i.e. $R_0 > 1$. We solved the model numerically using Runge-kutta of order four (4). It is shown that as the area is increasing the total number of infected individual is decreasing. This implies that to reduce the spread of measles, measles patients are to be kept separately for treatment so as to reduce the effective contract rate. The results were presented graphically.

Keywords: - Measles, Reproduction Number, Equilibrium Point, Habitat Area and Stability.

I. INTRODUCTION

Measles is caused by the measles virus. The measles virus is a paramyxovirus of the genus Morbillivirus. It is transmitted by close contact via airborne propagules and spread through droplet transmission from the nose, throat, and mouth of someone who is infected with the virus. These droplets are sprayed out when the infected person coughs or sneezes. Among unimmunized people exposed to the virus, over 90% will contract the disease. Infection leads to the development of a typical rash. The infected person is highly contagious with the rash appearing four days after the person has been infected. The measles virus can remain in the air (and still be able to cause disease) for up to two hours after an infected person has left a room. Individuals infected with measles virus are believed to be immune for life. Also, individual who have received two doses of vaccine after their first birthday has a 98% likelihood of being immune, that is, the probability of having measles even when in contact with those infected with measles is above 0.02, which is very low. Though infants receive some immunity from their mother, this immunity is not complete and they are at increased risk for infection until they receive the vaccinations at 12 to 15 months of age [3, 14].

The infectious period is in the order of a week, after which the hosts recover and develop lifelong immunity. Hosts therefore, are normally infected only once in their lifetime and if the dose of the infectious agent is sufficiently large, this happens at a young age, hence measles is a childhood disease. Although in unvaccinated populations measles is a common disease, infection is not without danger [3, 14].

Individuals at high risk for measles include but not limited to children less than 1 year of age who, though have some immunity passed from their mother, is not 100% effective; individual who have not received the proper vaccination series; individual who received immunoglobulin at the time of measles vaccination and individual immunized from 1963 until 1967 with an older ineffective killed measles vaccine. Mortality rates from measles are often high in tropical Africa because of malnutrition, concurrent infection and inadequate case management. Mortality rates of 5% and 10% are common and rates of 20% have been reported [3, 14].

In Nigeria, measles is an important cause of childhood morbidity and mortality. Failure to deliver at least one dose of measles vaccine to all infants remains the main reason for high measles morbidity and mortality as 95% coverage is required to interrupt measles transmission. The National Program on Immunization aim of reducing measles case fatality to near zero has depended on the adoption and implementation of the WHO four prong strategy; improving routine immunization with at least one dose of measles vaccine at 9 months, providing a second opportunity for measles immunization through supplemental immunization activities, establishing case based surveillance with laboratory confirmation and improving case management (World Health Organization, 2001).

Many physicians and scientists have studied the nature, characteristics, effect and spread of measles in many communities across the globe; these studies are either experimental or theoretical. Similarly, some mathematical modeling studies have been carried out to gain insight into the transmission dynamics and control of measles spread in human population. In this paper, we design a compartmental mathematical model which we use to investigate the effect of habitat area in the control of the spread of measles in the society. The model designed is

an extension of some of the models described by previous researchers. The purpose of the current study is to provide a rigorous mathematical analysis of a model for measles spread, which uses standard incidence function for the infection rate and evaluate the conditions at which epidemics will occur and persist using the basic reproduction number concept.

II. MATHEMATICAL MODEL FORMULATION

Following [3,7,8], the total homogeneously mixing population at time t , denoted by $N(t)$, is sub divided into mutually-exclusive compartments of susceptible ($S(t)$), Vaccinated ($V(t)$), exposed /latent ($E(t)$), intentions ($I(t)$), and recovered ($R(t)$) individuals, so that

$$N(t) = S(t) + V(t) + I(t) + R(t)$$

The susceptible population is increased by the recruitment of people (either by birth or immigration) into the population; all recruited individuals are assumed to be susceptible at a rate, π . Also the susceptible population increases by vaccinated individuals. Anyone who has had measles is believed to be immune for life. This population is decreased by infection, which can be acquired following effective contact with infectious individuals only at a rate λ given by

$$\lambda = \frac{\beta \eta_d I}{N} \tag{1}$$

In (1) β represents the effective contact rate (i.e. contact capable of leading to measles infection). η_d is a modification parameter that compares the transmissibility of the diseases. Here also, we assume that $0 < \eta_d < 1$. Thus the rate of change of the susceptible population is given by

$$\frac{dS}{dt} = (1 - \rho)\pi - \frac{\beta S}{A} - \frac{\lambda S}{A} - \mu S + \omega V \tag{2}$$

The population of the vaccination individual increases by recruiting individual (either by birth or immigration) and vaccinated individuals into the vaccinated class. The population of vaccinated individual is decrease by the progression of the rate at which vaccine wanes (ω) and vaccinated individuals is also reduced by natural death (at the rate μ). Thus

$$\frac{dV}{dt} = \rho\pi - \omega V - \mu V \tag{3}$$

The population of the exposed individual is increased by susceptible individuals whom are infected by those who are infectious per habitat area. The population of the exposed individuals is decreased by exposed individuals whom are infectious and is also reduced by natural death of the exposed individual. Then,

$$\frac{dE}{dt} = \frac{\beta S}{A} + \frac{\lambda S}{A} - \sigma E - \mu E \tag{4}$$

The population of infected individual increased by the exposed individuals whom are infectious. The infected population is decreased by the infected individuals whom are treated and get recovered. The infected individuals are also reduced by those that died of measles and to mention of natural death of measles patient also reduced the population of infected individuals.

Hence,

$$\frac{dI}{dt} = \sigma E - \tau I - \delta I - \mu I \tag{5}$$

Recovery here means recovery from diseases. The population of recovered individuals is increase by recovery of infected individuals after treatment. The population is reduced by natural death of recovered individual.

Therefore

$$\frac{dR}{dt} = \tau I - \mu R \tag{6}$$

Thus, in summary, the measles dynamics transmission model, is given by the following system of non-linear differential equations,

$$\begin{aligned} \frac{dS}{dt} &= (1 - \rho)\pi - \frac{\beta S}{A} - \frac{\lambda S}{A} - \mu S + \omega V \\ \frac{dV}{dt} &= \rho\pi - \omega V - \mu V \end{aligned}$$

$$\frac{dE}{dt} = \frac{\beta S}{A} + \frac{\lambda S}{A} - \sigma E - \mu E \tag{7}$$

$$\frac{dI}{dt} = \sigma E - \tau I - \delta I - \mu I$$

$$\frac{dR}{dt} = \tau I - \mu R$$

Table 1: Description of parameters of the model

Parameters	Description.
ρ	Fraction of recruitment individuals who are vaccinated.
π	The recruitment rate of the individuals (either by birth or immigration).
β	The rate at which susceptible individuals become infected by those who are infectious.
μ	Natural death rate.
ω	The rate at which vaccine wanes (i.e. $\frac{1}{\omega}$ is the duration of the loss of immunity acquired by preventive vaccine or by infectious).
σ	The rate at which exposed individuals becomes infectious.
τ	The rate at which infected individuals are treated and recovered.
A	Area per Square meter.
δ	Measles induced mortality rate.
η_d	Modification parameter.

III. ANALYSIS OF THE MODEL

Theorem 1: The closed set $D = \left\{ (S, V, E, I, R) \in \mathfrak{R}_+^5 : N \leq \frac{\pi}{\mu} \right\}$ is positively invariant and attracting with

respect to the model equation (7) above.

Proof: Consider the biologically-feasible region (1), the rate of change of the total population obtained by adding all the equations of the model (7) above is given by

$$\frac{dN}{dt} = \pi - \mu S - \mu V - \mu E - \mu I - \mu R - \delta I \tag{8}$$

since $N=S+V+E+I+R$

then

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dV}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt}$$

$$\frac{dN}{dt} = \pi - \mu S - \mu V - \mu E - \mu I - \mu R - \delta I$$

$$= \pi - \mu(S + V + E + I + R) - \delta I$$

$$\frac{dN}{dt} = \pi - \mu N - \delta I \tag{9}$$

Therefore, $\frac{dN}{dt} < 0$ whenever the sub total population $N > \frac{\pi}{\mu}$, Hence, for all $t > 0$, all the solutions of the

model with the initial conditions in D will remain in D. Thus, the biologically feasible region D is positively-invariant and attracting.

3. 1 Disease Free Equilibrium (DFE)

The DFE of the modeled equation i.e. (7) can be obtained by setting the right hand side of the model to zero

$$\varepsilon_0 = (S_0, V_0, E_0, I_0, R_0) = \left\{ \frac{A\pi(1-\rho)}{\beta + \mu A}, 0, 0, 0, 0 \right\} \tag{10}$$

The stability of the DFE, ε_0 will be analysed using the next generation method. The non-negative matrix F (of the new infection terms) and the non singular matrix V (of the remaining transfer terms) are given respectively

$$\text{by } F = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta\eta_d}{A} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \quad V = \begin{bmatrix} k_1 & 0 & 0 & 0 \\ 0 & k_2 & 0 & 0 \\ 0 & -\sigma & k_3 & 0 \\ 0 & 0 & -\tau & \mu \end{bmatrix} \quad (11)$$

Where

$$k_1 = (\omega + \mu); \quad k_2 = (\sigma + \mu); \quad k_3 = (\tau + \delta + \mu)$$

The associated reproduction number, denoted by R_0 is given by $R_0 = p(FV^{-1})$ where p denote spectral radius (dominant eigenvalue in magnitude) of the next generation matrix (FV^{-1})

$$\text{It follows that } R_0 = \frac{\beta\eta_d\sigma}{Ak_2k_3} \quad (12)$$

where $k_2 = (\sigma + \mu); \quad k_3 = (\tau + \delta + \mu)$

Hence, the result below follows,

Lemma 1: The DFE of the model equation, given (7), is locally stable if $R_0 < 1$.

The threshold quantity R_0 , is the reproduction number for the model. It measures the average number of new measles infections generated by a single infectious individual in a population where some of the infected individuals have been immunized. The epidemiological implication of this lemma is that measles spread can be effectively controlled in the community (when $R_0 < 1$). If the initial sizes of the sub-population of the model are

in the basin of attraction of the diseases-free equilibrium $\left(\varepsilon_0 = \left(\frac{A\pi(1-\rho)}{\beta + \mu A}, 0, 0, 0, 0 \right) \right)$.

Epidemiologically, if $R_0 < 1$, the disease will dies out in the community and if $R_0 > 1$, the disease spreads in the population. Hence, the basic reproduction number turned out to be an important factor in determining the transmission dynamics of any infectious diseases.

3.2 Stability Analysis of the DFE

Hence, the stability property of the DFE of the model will be explored. At a steady state $S = N^* - V - E - I - R$, hence the stability of ε_0 can be established by considering the following mass action equivalent of the model given as

$$\begin{aligned} \frac{dV}{dt} &= \rho\pi - \omega V - \mu V \\ \frac{dE}{dt} &= \frac{\beta S}{A} + \frac{\lambda S}{A} - \sigma E - \mu E \\ \frac{dI}{dt} &= \sigma E - \tau I - \delta I - \mu I \\ \frac{dR}{dt} &= \tau I - \mu R \end{aligned} \quad (13)$$

$$\text{and } \lambda = \frac{\beta\eta_d I}{N}$$

Here, the invariance region is given by

$$D^* = \left\{ (V, E, I, R) \in \mathfrak{R}_+^4 : V + E + I + R \leq N^* \right\} \quad (14)$$

Theorem 2: The DFE of model (13) given by (11) is global asymptotically stable (GAS) if $R_0 < 1$.

Proof: The equation (13) can be re-written as

$$\begin{bmatrix} \frac{dV}{dt} \\ \frac{dE}{dt} \\ \frac{dI}{dt} \\ \frac{dR}{dt} \end{bmatrix} = (G_1 - G_2) \begin{bmatrix} V \\ E \\ I \\ R \end{bmatrix} \tag{15}$$

where the matrices G_1 , G_2 and G_3 are given as:

$$G_1 = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta\eta_d}{A} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

$$G_2 = \begin{bmatrix} (\omega + \mu) & 0 & 0 & 0 \\ 0 & (\sigma + \mu) & 0 & 0 \\ 0 & -\sigma & (\tau + \delta + \mu) & 0 \\ 0 & 0 & -\tau & \mu \end{bmatrix} \text{ and}$$

$$G_3 = \begin{bmatrix} \lambda & \lambda & \lambda & \lambda \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

Since matrix G_3 is non-negative

$$\begin{bmatrix} \frac{dV}{dt} \\ \frac{dE}{dt} \\ \frac{dI}{dt} \\ \frac{dR}{dt} \end{bmatrix} \leq (G_1 - G_2) \begin{bmatrix} V \\ E \\ I \\ R \end{bmatrix}$$

If $R_0 < 1$ then $(G_1 G_2^{-1}) < 1$ (from the local stability result given in Lemma 1), which is equivalent to $G_1 - G_2$ having all its eigenvalue in the left-half plane [13]. It follows that the linearized differential inequality system (14) is stable whenever $R_0 < 1$

3. 3 Existence of Endemic Equilibrium Point (EEP)

In this section, the possible existence and stability of endemic (positive equilibria of the modeled equation (7). where at least one of the infected components of the model is non-zero) will be considered.

Let $\varepsilon_1 = (S^*, V^*, E^*, I^*, R^*)$ represents any arbitrary endemic equilibrium of the model equation. Solving the equations of the system at the steady-state goes thus:

$$S^* = \frac{A\pi(\omega + \mu - \rho\mu)}{(\omega + \mu)(\mu A + \lambda + \beta)} \tag{16}$$

$$\Rightarrow V^* = \frac{\rho\pi}{\omega + \mu} \tag{17}$$

$$E^* = \frac{\beta S^* + \lambda S^*}{A(\sigma + \mu)} \tag{18}$$

$$\Rightarrow I^* = \frac{\sigma E^*}{(\tau + \delta + \mu)} \tag{19}$$

Substitute for E^* in (18) to (19)

$$= I^* = \frac{\sigma\beta S^* + \sigma\lambda S^*}{A(\sigma + \mu)(\tau + \delta + \mu)} = P_1 S^* + P_2 \lambda^* S^* \tag{20}$$

$$\text{where } P_1 = \frac{\sigma\beta}{A(\sigma + \mu)(\tau + \delta + \mu)} \quad \text{and} \quad P_2 = \frac{\sigma}{A(\sigma + \mu)(\tau + \delta + \mu)} \quad (21)$$

also,

$$R^* = \frac{d^*}{\mu} + \frac{\tau}{\mu}(P_1 S^* + P_2 \lambda^* S^*) \quad (22)$$

$$R^* = \frac{d^* P_1 S^*}{\mu} + \frac{\tau P_2 \lambda^* S^*}{\mu}$$

recall that, $\lambda = \frac{\beta\eta_d I}{N^*} \Rightarrow N^* \lambda = \beta\eta_d I$ (23)

where $N^* = S^* + V^* + E^* + I^* + R^*$

$$\begin{aligned} \lambda\eta S^* + \frac{\rho\pi}{(\omega + \mu)} + \frac{(\beta + \lambda)S^*}{A(\sigma + \mu)} + P_1 S^* + P_2 \lambda^* S^* + \frac{\tau}{\mu} P_1 S^* + \frac{\tau}{\mu} P_2 \lambda^* S^* \\ = \beta\eta_d (P_1 S^* + P_2 \lambda^* S^*) \end{aligned}$$

let

$$(\omega + \mu) = k_1 \quad \text{and} \quad (\sigma + \mu) = k_2 \quad \text{as before}$$

$$\begin{aligned} \lambda\eta S^* + \frac{\rho\pi}{k_1} + \frac{(\beta + \lambda)S^*}{Ak_2} + P_1 S^* + P_2 \lambda^* S^* + \frac{\tau}{\mu} P_1 S^* + \frac{\tau}{\mu} P_2 \lambda^* S^* \\ = \beta\eta_d (P_1 S^* + P_2 \lambda^* S^*) \end{aligned}$$

Subtract $P_1 S^*$ from both sides

$$\begin{aligned} \lambda\eta S^* + \frac{\rho\pi}{k_1} + \frac{(\beta + \lambda)S^*}{Ak_2} + P_2 \lambda^* S^* + \frac{\tau}{\mu} P_1 S^* + \frac{\tau}{\mu} P_2 \lambda^* S^* \\ = \beta\eta_d (P_2 \lambda^* S^*) \end{aligned}$$

Divide both sides by $\lambda^* S^*$

$$\lambda \left\{ 1 + \frac{\rho\pi}{k_1} + \frac{\beta + \lambda}{Ak_2} + P_2 \lambda^* + \frac{\tau}{\mu} P_1 + \frac{\tau}{\mu} P_2 \lambda^* \right\} = \beta\eta_d P_2 \lambda^*$$

$$\text{Let } P_3 = \frac{\rho\pi}{k_1} + \frac{\beta + \lambda}{Ak_2} + P_2 \lambda^* + \frac{\tau}{\mu} P_1 + \frac{\tau}{\mu} P_2 \lambda^*$$

$$1 + P_3 \lambda^* = \beta\eta_d P_2 \lambda^* = R_0$$

$$\lambda^* = \frac{R_0 - 1}{P_3} \quad (24)$$

The component of the unique endemic equilibrium (ε_1) can then be obtained by substituting the unique value of λ^* , given in (24), into the expressions in (16, 18, 20). Thus the following result has been established.

3.4 Local stability of EEP

The local stability of the unique EEP, (ε_1) will now be explored for the special case where the disease induced mortality is negligible (i.e. $\delta = 0$), setting $\delta = 0$ in the model (2-6) above shows that

$$\frac{dN(t)}{dt} = \pi - \mu N(t) \quad (25)$$

Hence, it follows from (23) that $N(t) \rightarrow \frac{\pi}{\mu} = N$ as $t \rightarrow \infty$; further, using the substitution

$S = N^* - V - E - I - R$ (and noting that $\delta = 0$) in the model equation (2-6) gives the following reduced model.

$$\begin{aligned}
 \frac{dV}{dt} &= \rho\pi - (\omega + \mu)V \\
 \frac{dE}{dt} &= \frac{(\beta + \lambda)}{A}(N^* - V - E - I - R) - (\sigma + \mu)E \\
 &= \left[\frac{\beta}{A} + \frac{(\beta\eta_d I)}{NA} \right] (N^* - V - E - I - R) - (\sigma + \mu)E \\
 &= \frac{\beta}{A} + \frac{(1 + \eta_d I)}{N} (N^* - V - E - I - R) - (\sigma + \mu)E \\
 \frac{dI}{dt} &= \sigma E - (\tau + \delta + \mu)I \\
 \frac{dR}{dt} &= \tau I - \mu R
 \end{aligned} \tag{26}$$

So, for the reduced model (26), the associated reproduction number denoted by R_0^* is given by

$$R_0^\infty = \frac{\beta\eta_d\sigma}{A(\sigma + \mu)(\tau + \delta + \mu)} \tag{27}$$

Using the same approach as in Existence of Endemic Equilibrium Point (EEP); it can be shown that the reduced model equation (26) has a unique endemic equilibrium given by $\mathcal{E}_1 | \delta = 0$ whenever $R_0^\infty > 1$. The epidemiological implication of this is that once there is endemic situation of measles in a congested area of a community, the diseases will rapidly spread in that community whenever the associated reproduction number $R_0^\infty > 1$. Hence to avoid the endemic situation we should extend the area where those suffering from measles would be staying. In other words, the associated reproduction number R_0^∞ less than unity.

IV. NUMERICAL SIMULATIONS

Numerical Simulations of the dynamic model were carried out by maple 13, using the Runge-Kutta of order four (4). The set of parameter values in table I were used to investigate the effect of habitat area in the control of the spread of Measles. Five (5) hypothetical cases were considered and in each case, the probability that individuals who are exposed to the diseases will progress fast to infectious class depends on the level of immunity individual has. It is prominent to note here that when measles patient are separated from non-infected (vaccinated or recovered) people and kept in a wider area, it is assumed that they will have herd immunity, (i.e. the level of immunity in a population which prevents epidemics).

We considered five (5) cases as shown in the table below;

Parameters	Case 1	Case 2	Case 3	Case 4	Case 5
β	0.2	0.2	0.2	0.2	0.2
μ	0.02	0.02	0.02	0.02	0.02
π	2000	2000	2000	2000	2000
σ	0.09	0.09	0.09	0.09	0.09
τ	0.8	0.8	0.8	0.8	0.8
δ	0.04	0.04	0.04	0.04	0.04
η_d	0.9	0.9	0.9	0.9	0.9
ω	0.1	0.1	0.1	0.1	0.1
ρ	0.3	0.3	0.3	0.3	0.3
A	1	10	100	200	300

With the following initial conditions:

S [0]: =2885; V [0]: = 4192; E [0]: = 15784

I [0]: =1645; R[0]: = 14215

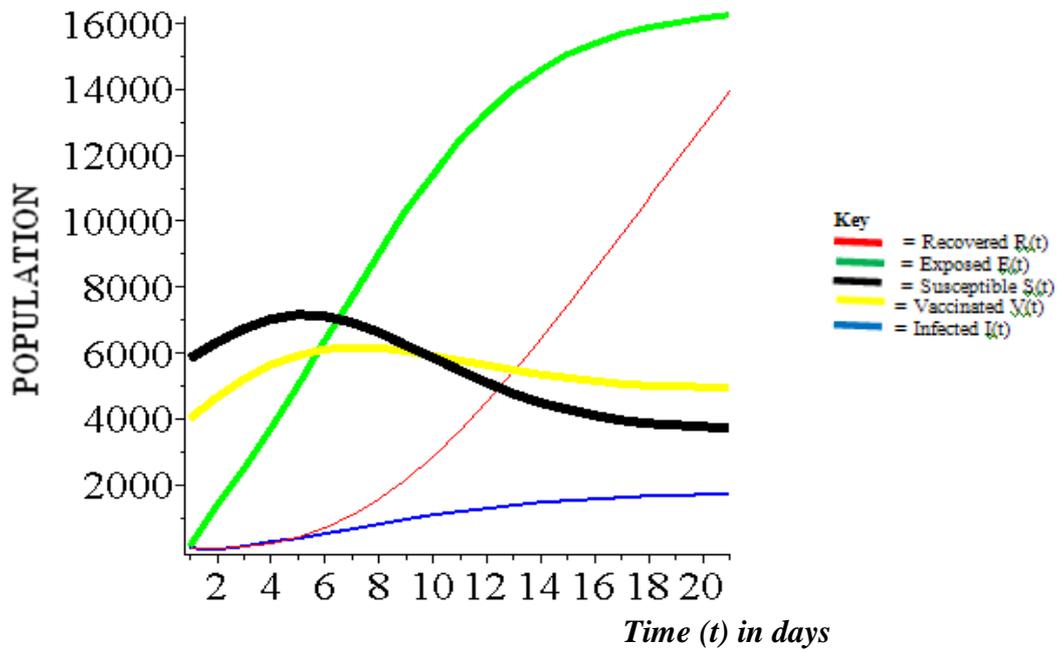


Figure 1: when $A = 1$, $\beta = 0.2$, $N = 0.02$, $\pi = 2000$, $\delta = 0.04$, $\sigma = 0.09$, $\tau = 0.8$, $\eta_d = 0.9$, $\omega = 0.1$, and $\rho = 0.3$

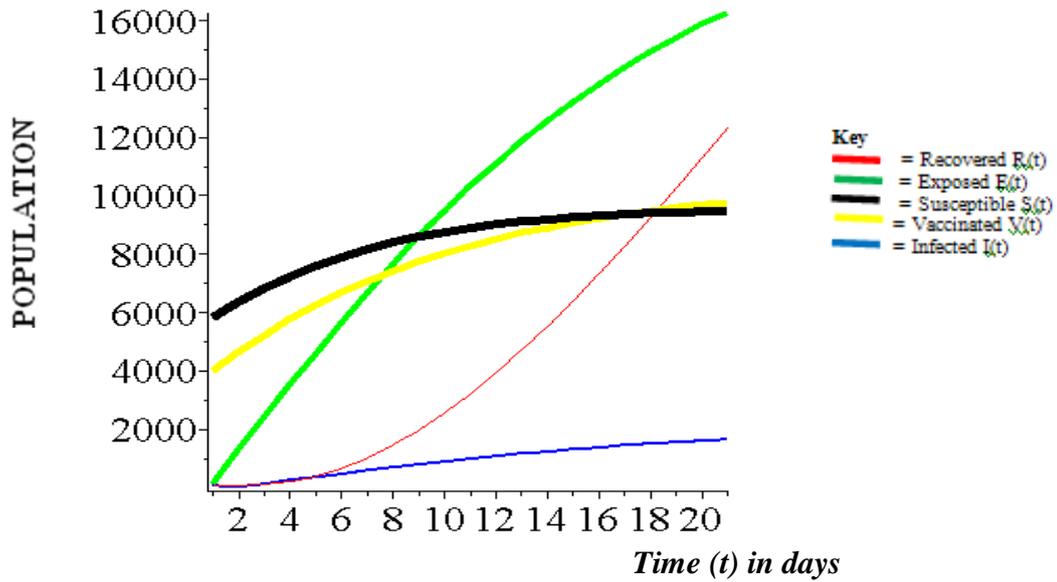


Figure 2: when $A = 10$, $\beta = 0.2$, $N = 0.02$, $\pi = 2000$, $\delta = 0.04$, $\sigma = 0.09$, $\tau = 0.8$, $\eta_d = 0.9$, $\omega = 0.1$ and $\rho = 0.3$

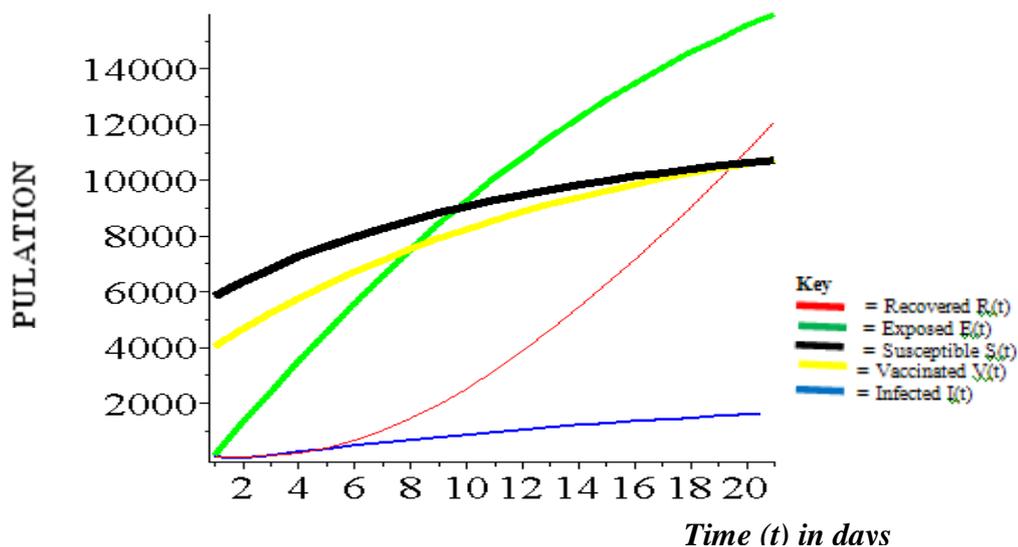


Figure 3: when $A = 100$, $\beta = 0.2$, $N = 0.02$, $\pi = 2000$, $\delta = 0.04$, $\sigma = 0.09$, $\tau = 0.8$, $\eta_d = 0.9$, $\omega = 0.1$ and $\rho = 0.3$

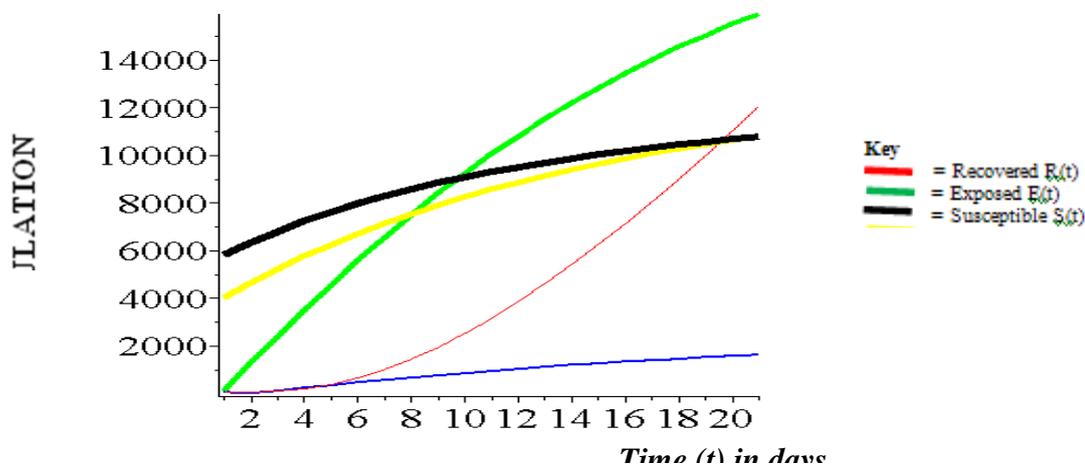


Figure 4: when $A = 200$, $\beta = 0.2$, $N = 0.02$, $\pi = 2000$, $\delta = 0.04$, $\sigma = 0.09$, $\tau = 0.8$, $\eta_d = 0.9$, $\omega = 0.1$ and $\rho = 0.3$

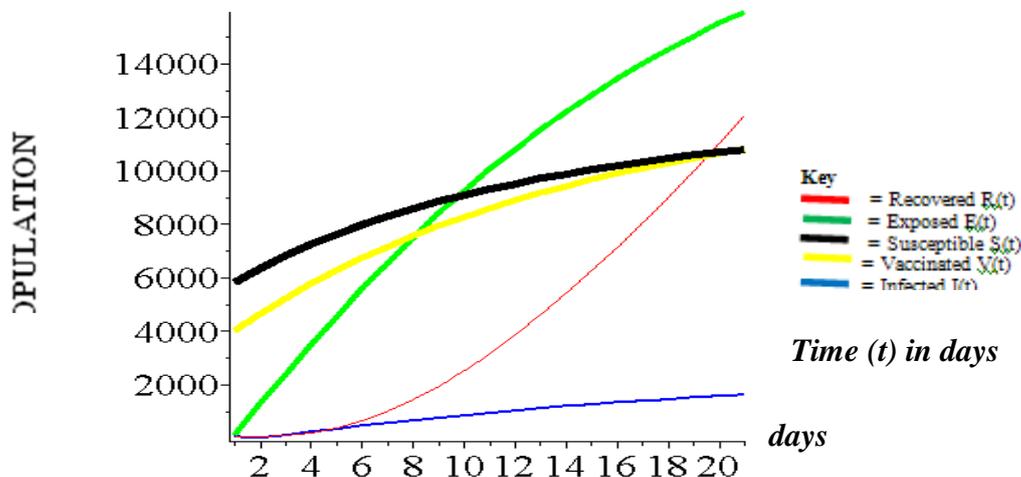


Figure 5: when $A = 300$, $\beta = 0.2$, $N = 0.02$, $\pi = 2000$, $\delta = 0.04$, $\sigma = 0.09$, $\tau = 0.8$, $\eta_d = 0.9$, $\omega = 0.1$ and $\rho = 0.3$

TABLE 1

	Time (t) in days	Susceptible S (t)	Vaccinated V (t)	Exposed E (t)	Infected I (t)	Recovered R (t)
	0	5800	4000	100	50	50
1	0.1	6271.520335	2593.087043	1297.988830	22.66522106	87.78082640
2	0.2	6718.303350	5153.725922	3438.973836	120.9093248	108.054041
3	0.3	700.926740	5592.23625	3604.085427	236.9508916	207.159844
4	0.4	7115.143696	5901.611624	4962.266560	360.3475078	397.445963
5	0.5	7068.738118	6085.713794	8805.425189	497.7408976	685.2611298
6	0.6	6884.478552	6052.1108612	7657.102472	637.26119151	1094.940321
7	0.7	6593.265369	6034.600668	8978.811062	278.2909671	1568.480291
8	0.8	628.928703	6042.293097	10228.64095	916.7278280	2164.613321
9	0.9	5887.494554	5905.484602	11372.01806	1148.404573	2859.28413
10	1.0	5444.258535	5747.831510	12383.33964	1169.598888	3644.89442
11	1.1	5077.989405	5588.557841	13240.1087	1279.500813	4511.13652
12	1.2	4755.168993	5441.114754	13968.30806	1370.51332	5445.91138
13	1.3	4483.182625	5812.839625	14540.973112	1448.295776	6455.33837
14	1.4	4262.240265	5206.668308	15000.3620	1311.604073	7466.77831
15	1.5	4087.897965	5121.961808	15366.67804	1561.942984	8527.6864031
16	1.6	3953.39265	5056.291443	15630.92748	1601.22548	9607.18465
17	1.7	885.370500	5008.483232	15847.08749	1631.396171	10696.1343
18	1.8	3774.938607	4969.329281	16003.18455	1654.327224	11382.1593
19	1.9	3718.162555	4941.060392	16121.45786	1671.676993	12874.509
20	2.0	3096.236098	4921.088885	16208.54162	1684.71183	13953.7985

TABLE 2

	Time (t) in days	Susceptible S (t)	Vaccinated V (t)	Exposed E (t)	Infected I (t)	Recovered R (t)
	0.	5800	4000	100	50	50
1	0.1	6316.86462	4622.771029	1252.922656	22.46549259	87.77514150
2	0.2	6784.613245	5200.202119	2380.0633056	116.9341504	107.738885
3	0.3	7195.685297	5727.3193554	3488.224061	230.6755786	203.585442
4	0.4	7533.718850	6206.270661	4571.985796	346.1474688	388.490820
5	0.5	7863.962834	6640.372251	5625.063966	459.7921259	661.9114736
6	0.6	8131.587253	7033.036420	6642.166903	570.4459598	1020.58139
7	0.7	8361.406588	7387.574983	7619.173067	677.4530241	1460.37104
8	0.8	8557.846721	7707.155916	8553.032763	780.3471722	1976.72960
9	0.9	8724.951695	7994.787177	9441.637381	878.7875120	2564.82650
10	1.0	8866.397017	8253.306918	10283.69473	972.5358023	3219.65933
11	1.1	8985.504869	8485.377041	11078.61487	1061.441768	3936.13877
12	1.2	9085.250963	8693.479793	11826.40590	1145.430717	4709.16009
13	1.3	9168.333053	8879.917268	12527.57881	1224.492412	5533.66347
14	1.4	9237.090954	9046.813575	13183.06093	1298.670946	6404.68414
15	1.5	9293.627705	9196.119333	13794.11739	1368.055527	7317.38363
16	1.6	9339.781455	9329.618074	14362.28063	1432.772132	8267.13266
17	1.7	9377.157591	9448.934137	14889.28757	1492.975978	9249.0562
18	1.8	9407.150674	9555.541647	15377.02415	1548.844819	10260.0562
19	1.9	9430.965792	9650.774132	15827.47702	1600.573012	11294.9682
20	2.0	9449.638994	9735.834654	16242.69198	1648.366342	12350.3863

Table 3

	Time (t) in days	Susceptible S (t)	Vaccinated V (t)	Exposed E (t)	Infected I (t)	Recovered R (t)
	0.	5800	4000	100	50	50
1	0.1	6321.400017	4625.740684	1248.414793	12.44551562	87.7749028
2	0.2	6791.224118	5204.831304	2374.192482	116.5365948	107.707163
3	0.3	7214.715633	5740.575979	3470.982892	230.0503001	203.228075
4	0.4	7597.299301	6236.541303	4533.176076	344.4568101	387.598967
5	0.5	7943.796440	6696.035642	5556.623761	456.0185470	659.606848
6	0.6	8258.376971	7122.050072	6538.513161	563.7260289	1015.18642
7	0.7	8544.636113	7517.281970	7477.125770	667.1638764	1449.57087
8	0.8	8805.680001	7884.168785	8371.614558	766.1146305	1957.76460
9	0.9	9044.200874	8224.919208	9221.817596	860.4699917	5234.70244
10	1.0	9262.540355	8541.540133	10028.10555	950.1992794	3175.33697
11	1.1	9462.742329	8835.859750	10791.25765	1035.330855	3874.69947
12	1.2	9646.597130	9109.547369	11512.36113	1115.938262	4627.94499
13	1.3	9815.678523	9364.130489	12192.72996	1192.129198	5430.38550
14	1.4	9971.374733	9601.009573	12833.83931	1264.036636	6277.51376
15	1.5	10114.91452	9821.470892	13437.27307	1331.811664	7165.01954
16	1.6	10247.38917	10026.69773	14004.68184	1395.617755	8088.80003
17	1.7	10369.77100	10217.78020	14537.74970	1455.626161	9044.96530
18	1.8	10482.92913	10395.72393	15038.16802	1512.012279	10029.8402
19	1.9	10587.64277	10561.45765	15507.61502	1564.952776	11039.9632
20	2.0	10684.61256	10715.84003	15947.74010	1614.623339	12072.0833

Table 4

	Time (t) in days	Susceptible S (t)	Vaccinated V (t)	Exposed E (t)	Infected I (t)	Recovered R (t)
	0	5800	4000	100	50	50
1	0.1	6321.652004	4625.905671	1248.164349	22.44440577	87.7748729
2	0.2	6791.591278	5205.088377	2373.866434	116.5145082	107.705401
3	0.3	7215.770994	5741.311186	3470.026894	230.0155796	203.208222
4	0.4	7599.718735	6238.221806	4531.021357	344.0363045	387.549440
5	0.5	7948.232367	6699.128446	5552.820255	455.8090392	659.478779
6	0.6	8265.428091	7127.000120	6532.747030	563.3525861	1014.88699
7	0.7	8554.838258	7524.503461	7469.212447	666.5915212	1448.97098
8	0.8	8819.503268	7894.040638	8361.485619	765.3218001	1956.71046
9	0.9	9062.049692	8287.782638	9209.505759	859.4474367	2533.02655
10	1.0	9284.755391	8557.697673	10013.72925	948.9405690	3172.86821
11	1.1	9489.603634	8855.575355	10775.00595	1033.862789	3871.27178
12	1.2	9678.328092	9133.047321	11494.47940	1114.271292	4623.40453
13	1.3	9852.450319	9391.604889	12173.50666	1190.287922	5424.59653
14	1.4	10013.31102	9632.614246	12813.59405	1262.050392	6270.36309
15	1.5	10162.09617	9857.32956	13416.34601	1329.713268	7136.41983
16	1.6	10299.85893	10066.90435	13983.42448	1393.442366	8078.69199
17	1.7	10427.53801	10267.40138	14516.51693	1453.410295	9033.31901
18	1.8	10545.97313	10444.80131	15017.31150	1509.792952	10016.6555
19	1.9	10655.91808	10615.01032	15487.47770	1562.766788	11025.2696
20	2.0	10758.05175	10773.86681	15928.65179	1612.506681	12055.939

Table 5

	Time (t) in days	Susceptible S (t)	Vaccinated V (t)	Exposed E (t)	Infected I (t)	Recovered R (t)
	0	5800	4000	100	50	50
1	0.1	6321.735999	4625.960667	1248.080867	22.44403581	87.7748629
2	0.2	6791.713663	52051.17407	2373.757754	116.5071459	107.704813
3	0.3	7216.122737	5741.556228	3469.708274	230.0040064	203.201604
4	0.4	7600.525173	6238.781948	4530.303152	344.3317936	387.532931
5	0.5	7949.711025	6700.159393	5551.552394	455.7392064	659.436092
6	0.6	8267.778627	7128.650248	6530.824819	563.2281029	1014.78712
7	0.7	8558.239480	7526.910975	7466.574190	666.4007200	1448.77102
8	0.8	8824.112242	7897.332100	8358.108195	765.0574772	1956.35907
9	0.9	9068.001808	8242.072194	9205.399602	859.1064762	2532.46787
10	1.0	9292.165021	8563.086747	10008.93322	948.5314482	3172.04515
11	1.1	9498.565185	8862.152690	10769.58230	1033.373045	3870.12889
12	1.2	9688.917319	9140.889279	11488.50907	1113.715000	4621.89041
13	1.3	9864.725785	9400.775942	12167.08489	1189.673190	5422.66573
14	1.4	10027.31563	9643.167596	12806.82648	1261.386910	6267.97761
15	1.5	10177.85873	9869.307841	13409.34516	1329.011880	7153.55024
16	1.6	10317.39565	10080.34038	13976.30671	1392.714698	8075.31813
17	1.7	10446.85398	10277.31895	14509.39988	1452.668436	9029.43043
18	1.8	10567.06369	10461.21598	15010.31186	1509.049167	10012.2516
19	1.9	10678.77012	10632.93010	15480.70943	1562.033290	11020.3597
20	2.0	10782.64486	10793.29303	15922.22465	1611.795426	12050.5418

V. DICUSION OF RESULT

The SVEIR model was considered to gain more insight into the effect of Habitat area on dynamic spread of measles. This Habitat area plays a crucial role in the control of spread of measles virus in the environment. It is observed from the results above that the higher the Habitat area, the lower will be spread of this measles virus and the higher will be the recovery rate. Also considered the possibility of measles outbreak in a community when certain threshold quantity, the basic reproduction number (R_0) exceeded unity (one).

We considered basic reproduction number which is an important tools for public health officials to determined the epidemic state of any deadly disease in a community. Using this threshold, we discovered that when the habitat area of measles patient is highly increased, the lesser would be the number of infected individuals and higher would be the number of recovered people. Conversely, when habitat area was low, for instance at $A = 1$ (Fig. 1), the R_0 is greater than one. This implies that there would be high level of epidemic within few days because of congestion of the area and the population of recovered people rise after six days.

Theoretically, our results are based on the fact that the diseases free equilibrium (DFE) point is locally stable whenever the threshold quantity $R_0 < 1$ and unstable when $R_0 > 1$ (see lemma 1). We also showed that whenever the associated reproduction number $R_0^\infty < 1$ in a community, there would be endemic situation of measles in that community unless there is quick intervention to keep the basic reproduction number less than unity.

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