Stability Analysis of a Mathematical Model of Measles Transmission Dynamics

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Abstract

Measles is an acute viral infectious disease caused by the Measles morbillivirus, a member of the paramyxovirus family. The virus is primarily transmitted through direct contact and airborne droplets. In this study, a mathematical model was developed to examine the transmission dynamics of measles and explore effective control measures. The stability of measles-free equilibrium was analyzed, and the results indicate that the equilibrium is locally asymptotically stable when the basic reproduction number R_0 is less than or equal to unity. Numerical simulations were conducted to validate the analytical findings, demonstrating that measles can be eradicated if a sufficiently high level of treatment is applied to the infected population.

Keywords: Basic reproduction number, Measles Equilibria State, Measles, Stability, Transmission dynamics

Introduction

Measles is a highly contagious viral disease that exclusively affects humans and is transmitted primarily through direct contact and airborne droplets expelled during coughing, sneezing, or even normal breathing. Historically, measles has been regarded as a more formidable threat than smallpox (Gastanaduy *et al.,* 2021). The virus predominantly infects the respiratory tract, facilitating its rapid spread, especially in settings characterized by high population density and inadequate healthcare infrastructure, such as many African and developing countries. In these regions, the absence of effective birth control measures often leads to overpopulation, exacerbating the risk of widespread outbreaks that can have devastating public health consequences.

The clinical presentation of measles is distinct and severe. It typically begins with a high fever, often reaching or exceeding 40°C, which manifests approximately 10 to 12 days after exposure to the virus. This initial phase is followed by symptoms such as a runny nose, persistent cough, conjunctivitis, and the appearance of small white spots (Koplik spots) on the mucous membranes of the mouth. Subsequently, a characteristic rash emerges, initially appearing on the face and subsequently spreading to the hands, feet, and trunk, usually persisting for 5 to 6 days. The incubation period for measles ranges from 7 to 18 days, underscoring the challenges in early detection and containment of the disease.

Measles, also known by names such as morbilli, rubeola, red measles, and English measles, has been recognized for centuries. The first documented descriptions date back to the 7th century, with a more detailed differentiation from smallpox achieved in the 10th century by the Persian physician Rhazes, who noted its particularly alarming nature (Peter *et al.,* 2022). Despite the introduction of

the measles vaccine, the disease continues to pose a significant public health threat. Historically, prior to widespread immunization, measles epidemics were common, particularly among young children aged 2 to 3 years, leading to millions of cases and an estimated 2.6 million deaths annually.

In recent decades, numerous researchers (Adewale *et al.,* 2014; Bauch & Earn, 2014; Fred *et al.,* 2014; Momoh *et al.,* 2013; Peter *et al.,* 2018; Smith *et al.,* 2016) have developed sophisticated mathematical models to study the transmission dynamics of measles. These models have provided valuable insights into the impact of various parameters on disease spread and have been instrumental in guiding public health interventions.

The present study builds on this body of work by developing a comprehensive mathematical model aimed at elucidating the transmission dynamics of measles within a population. By integrating key epidemiological parameters and simulating different intervention scenarios, this model is intended to generate actionable information for government bodies and public health professionals. Ultimately, the insights gained from this study are expected to inform the development of more effective strategies for controlling and preventing measles outbreaks, thereby mitigating the public health risks associated with this highly contagious disease.

Research has demonstrated that measles can be completely prevented through the administration of two doses of a safe and effective vaccine. The World Health Organization (WHO) recommends that children receive their first dose at their first birthday, aiming for a global vaccination coverage of 95% for this initial dose. In 2021, approximately 81% of children received the first dose, while only 67% had received the second dose, indicating room for improvement in vaccination efforts.

To mitigate the risk of measles outbreaks, particularly in developing and underdeveloped countries where large populations remain vulnerable, the WHO, in collaboration with national governments, the Measles & Rubella Partnership, and other international organizations, has implemented a series of preventive measures. These accelerated immunization activities have been highly effective, preventing an estimated 56 million deaths between 2000 and 2021, and reducing global measles mortality from 761,000 deaths in 2000 to 128,000 deaths in 2021 (WHO, 2023).

Mathematical modeling has emerged as a powerful tool in the fight against infectious diseases, including measles. These models allow researchers to propose, test, and refine theories as well as to plan, implement, compare, and evaluate various intervention strategies for detection, prevention, treatment, and control. Historical studies on measles epidemiology date back to 1846, when Danish physician Peter Panum conducted seminal research during a measles epidemic in the Faroe Islands (Berche, 2022). Since then, numerous models have been developed to understand the transmission dynamics of measles.

For example, (Roberts & Tobias, 2000) employed a compartmental SIR (Susceptible-Infectious-Recovered) model to predict and prevent measles epidemics, considering factors such as population size and age structure. These models collectively underscore the importance of vaccination in conferring group immunity and protecting susceptible individuals.

(Momoh *et al.*, 2013) introduced an SEIR (Susceptible-Exposed-Infectious-Recovered) model to assess the role of individuals in the latent period and to explore control strategies. Similarly, (Sowole *et al.,* 2020) enhanced the SEIR framework by incorporating control measures within the susceptible and exposed classes, specifically targeting measles prevalence and control. Recently (Alemneh & Belay, 2023) explored how measles spreads by developing an improved

compartmental model called SVIRP. This model expands on the traditional SEVIR framework by adding a separate category for the pathogen population, allowing them to account for indirect transmission routes, such as environmental contamination. Also (Ahmed *et al.,* 2024), (Bag *et al.,* 2024) and (Peter *et al.,* 2024) studies the transmission dynamics of measles with different compartment and their control strategies.

Historical investigations, such as Peter Panum's work in 1864, revealed that individuals who recover from measles typically develop lifelong immunity. However, outbreaks continue to occur predominantly in communities with low vaccination coverage. This body of research not only highlights the critical role of vaccination in controlling measles but also emphasizes the need for continued and enhanced immunization efforts to achieve and maintain the levels of coverage necessary to prevent outbreaks.

Model Formulation

In this model a population of human in a community is considered. We divide the population into five compartments; Susceptible class, $S(t)$, Vaccinated class, $V(t)$, Exposed class $E(t)$, Infected class, $I(t)$, and Recovered class, $R(t)$. The susceptible human population increase by recruitment rate α. Individuals in the susceptible class receive a vaccination at a rate τ and loss immunity at a vaccine wane rate ρ, and returns to the susceptible class. The susceptible humans get in contact with the release of the infected person at rate β to get expose to measles virus. If the individual has not received the doses of vaccination, the exposed will be infected by the virus at rate θ which make them move to the infected group. And those that were attended to immediately after being i individuals gain immunity to the disease and do not ever get effected again. Natural mortality occurs in all the classes at a rate μ and death due to the disease at rate δ.

The equations of the model are formulated form of a system of ordinary differential equations of the above diagram.

$$\frac{dS}{dt} = \alpha - \beta IS - (\mu + \tau)S + \rho V \quad (1)$$

$$\frac{dV}{dt} = \tau S - (\rho + \mu + \omega)V \quad (2)$$

$$\frac{dE}{dt} = \beta S\mathrm{I} - (\theta + \mu)E \quad (3)$$

$$\frac{dI}{dt} = \theta E - (\sigma + \mu + \delta)I \quad (4)$$

$$\frac{dR}{dt} = \sigma I + \omega V - \mu R \quad (5)$$

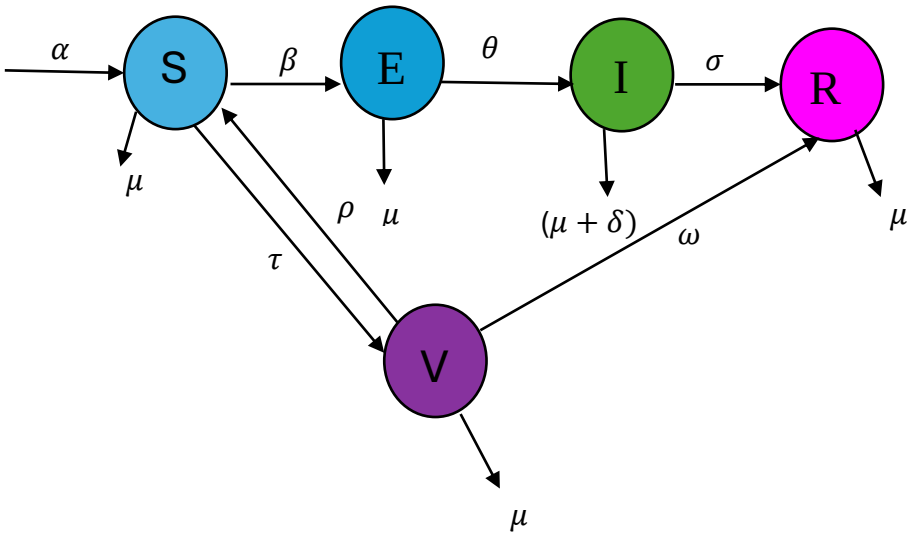

Figure 1: The model flow diagram

Table 1: Table of variables and parameters of the model

Notation	Description of variables and parameters
S(t)	Number of susceptible humans at time t
V(t)	Number of vaccinated humans at time t
E(t)	Number of exposed humans at time t
I(t)	Number of infectious humans at time t
R(t)	Number of recovered humans at time t
α	Recruitment rate of human population
δ	Disease induced death
μ	Natural death rate of human population
θ	The rate at which the exposed get infected by the virus
β	The rate at which susceptible humans get in contact with the release of the infected person
σ	The rate at which the infected get treated and moves to the recovered class
τ	Individuals in the susceptible class receives vaccination
ω	The rate of progression of vaccinated individuals to the recovery class
ρ	The rate at which individuals' losses immunity and returns to the susceptible class

$$N = S + V + E + I + R \tag{6}$$

By adding equations (1) to (5), we have.

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dV}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = \alpha - \mu N \tag{7}$$

The basic dynamic features of the model equations (1) to (5) will be explored.

Theorem 1

$D = \{(S,V,E,I,R)\epsilon\Re_+^5 : S+V+E+I+R \leq N\}$ (8)

Is positively invariant and attracting with respect to the basic model equations (1) to (5)

Proof:

From equations (6) and (7)

$\frac{dN}{dt} \leq \alpha - \mu N$ (9)

It follows that $\frac{dN}{dt} \leq 0$ if $N > \frac{\alpha}{\mu}$, (10)

then a standard comparison theorem can be used to show that

$N(t) \leq N(0)e^{\mu(t)} + \frac{\alpha}{\mu}(1 - e^{-\mu(t)})$ therefore $N(t) \leq \frac{\alpha}{\mu}$ if $N(0) \leq \frac{\alpha}{\mu}$. Thus $D$is positively invariant. Hence if if $N(0) > \frac{\alpha}{\mu}$, then either the solution enters Din finite time or $N(t)\ approches\ \frac{\alpha}{\mu}$, and the infected variables $Eand\ I$ approaches 0. Hence D is attracting, which means all the solutions in $\Re_+^7$ is enters D. Thus in D, the equations (1) to (5) is well posed epidemiologically and mathematically according to (Hethcote, 1978). Hence it is sufficient to study the dynamics of this model.

The model is based on human population; therefore, it is necessary to show that all stated variables are positive at all time (t), making the model well-posed and biologically meaningful.

Theorem 5.1:

Let the initial data be $\{ S(0) > 0, V(0) \geq 0, E(0) \geq 0, I(0) \geq 0, R(0) \geq 0\} \in \Omega$ (11)

Then, the solution set $S(t), V(t), E(t), I(t), R(t)$ (12)

of the system (1) to (5) is positive if all t > 0.

Proof:

From equation (1)

$\frac{dS}{dt} = \alpha - \beta IS - (\tau+\mu)S + \rho V \geq \alpha - (\tau+\mu)S$ (13)

$\frac{dS}{dt} \geq \alpha - (\tau+\mu)S$ (14)

$\frac{dS}{dt} + (\tau+\mu)S \geq \alpha$ (15)

using integrating factor, we have

$\frac{d}{dt}(Se^{(\tau+\mu)t}) \geq \alpha e^{(\tau+\mu)t}$ (16)

$S(t)e^{(\tau+\mu)t} \geq \frac{\alpha}{(\tau+\mu)}e^{(\tau+\mu)t} + C$ (17)

$S(t) \geq \frac{\alpha}{(\tau+\mu)} + Ce^{(\tau+\mu)t}$ (18)

From (16) substituting $t = 0$ we have

$S(0) \geq \frac{\alpha}{(\tau+\mu)} + C => C \leq S(0) - \frac{\alpha}{(\tau+\mu)}$ (19)

Hence

$S(t) \geq \frac{\alpha}{(\tau+\mu)} + (S(0) - \frac{\alpha}{(\tau+\mu)})e^{(\tau+\mu)t} > 0$ (20)

Therefore

$S(t) > 0$ (21)

From equation (2)

$\frac{dV}{dt} = \tau S - (\rho+\mu+\omega)V \geq -(\rho+\mu+\omega)$ (22)

$\int \frac{dV}{dt} \geq \int (\rho+\mu+\omega)\, V$ (23)

$$\int \frac{d}{dV}(v) \geq \int (\rho + \mu + \omega)\, dt \tag{24}$$

$$V(t) \geq V(0)e^{-(\rho+\mu+\omega)t} \geq 0 \tag{25}$$

From equation (3)

$$\frac{dE}{dt} = \beta S\mathrm{I} - (\theta + \mu)E \geq -(\theta + \mu) \tag{26}$$

$$\int \frac{dE}{dt} \geq \int (\theta + \mu)\, E \tag{27}$$

$$\int \frac{d}{dE}(E) \geq \int (\theta + \mu)\, dt \tag{28}$$

$$E(t) \geq E(0)e^{-(\theta+\mu)t} \geq 0 \tag{29}$$

From equation (4)

$$\frac{dI}{dt} = \theta E - (\sigma + \mu + \delta)I \geq -(\sigma + \mu + \delta)I \tag{30}$$

$$\int \frac{dI}{dt} \geq \int (\sigma + \mu + \delta)\, I \tag{31}$$

$$\int \frac{d}{dI}(I) \geq \int (\sigma + \mu + \delta)\, dt \tag{32}$$

$$I(t) \geq I(0)e^{-(\sigma+\mu+\delta)t} \geq 0 \tag{33}$$

From equation (5)

$$\frac{dR}{dt} = \sigma I + \omega V - \mu R \geq -\mu R \tag{34}$$

$$\int \frac{dR}{dt} \geq \int \mu R \tag{35}$$

$$\int \frac{d(R)}{dR} \geq \int \mu dt \tag{36}$$

$$R(t) \geq R(0)e^{-\mu t} \geq 0 \tag{37}$$

Therefore, all the solutions of the system of equations (1) to (5) are positive for all $t > 0$.

Equilibria State of the model

At equilibrium the time derivatives are equal to zero, i.e.

$$\frac{dN}{dt} = \frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0 \tag{38}$$

Measles Free Equilibrium (MFE) State

Let $E^0 = (S, V, E, I, R) = (S^0, V^0, E^0, I^0, R^0)$ (39)

Be the DFE point

Substituting equation (8) into (2) to (6) equates to zero and solve gives

$$E^0 = (S^0, V^0, E^0, I^0, R^0) = \left[\frac{\alpha(\rho+\mu+\omega)}{(\mu+\tau)(\rho+\mu+\omega)-\rho\tau}, \frac{\rho S^0}{(\rho+\mu+\omega)}, 0, 0, \frac{\omega\tau S^0}{\mu(\rho+\mu+\omega)}\right] \tag{40}$$

Equation (13) is Measles-Free Equilibrium MFE point of the model.

Measles Endemic Equilibrium (MEE) State

Let $E^* = (S, V, E, I, R) = (S^*, V^*, E^*, I^*, R^*)$

$$\begin{bmatrix} S^* \\ V^* \\ E^* \\ I^* \\ R^* \end{bmatrix} = \begin{bmatrix} \frac{A_1 A_1}{\beta\theta} \\ \frac{\tau A_3 A_4}{\beta\theta A_2} \\ \frac{\rho\tau A_3 A_4 - A_1 A_2 A_3 A_4 + A_2\alpha\beta\theta}{A_2 A_3 \beta\theta} \\ \frac{\rho\tau A_1 A_1 - A_1 A_2 A_3 A_4 + A_2\beta\theta}{A_2 A_3 A_4 \beta} \\ \frac{\omega\tau A_3^2\, A_4^2 + \theta\sigma\rho A_3 A_4 - \theta\sigma A_1 A_2 A_3 A_4 + \theta^2\sigma\alpha A_2\beta}{A_3 A_4 \beta\theta A_2 \mu} \end{bmatrix} \tag{41}$$

The Basic Reproduction Number (R_0)

In this model, the next generation matrix method as described by (Driessche, 2002) is used to get the basic reproduction number of R_0. The basic reproductive number of an infected person is a threshold that indicates the total number of potential diseases that have been developed into a completely susceptible population during its transmission period. Given by $R_0 = \rho(FV^{-1})$. F and V are the matrices created for the new infection and transmission respectively.

The new infection components are $E(t)\ and\ I(t)$ in equation (3) and (4) above is given by

$$F = \begin{pmatrix} \beta SI \\ 0 \end{pmatrix}, \tag{42}$$

$$V = \begin{pmatrix} (\theta + \mu)E \\ (\sigma + \mu + \delta)I - \theta E \end{pmatrix} \tag{43}$$

F and V are the Jacobian matrices which shall be computed at the DFE for (42) and (43)

$$F = \begin{pmatrix} 0 & \beta S \\ 0 & 0 \end{pmatrix} \tag{44}$$

$$V = \begin{pmatrix} (\theta + \mu) & 0 \\ -\theta & (\sigma + \mu + \delta) \end{pmatrix} \tag{45}$$

$$F = \begin{pmatrix} 0 & \beta S \\ 0 & 0 \end{pmatrix}, \tag{46}$$

The inverse of V is computed using guass Jordan method

$$V^{-1} = \begin{pmatrix} \frac{1}{(\theta+\mu)} & 0 \\ \frac{\theta}{(\theta+\mu)(\sigma+\mu+\delta)} & \frac{1}{(\sigma+\mu+\delta)} \end{pmatrix} \tag{47}$$

Using the next generation matrix, we obtain:

$$FV^{-1} = \begin{pmatrix} \frac{\theta\beta S}{(\theta+\mu)(\sigma+\mu+\delta)} & \frac{\beta S}{(\sigma+\mu+\delta)} \\ 0 & 0 \end{pmatrix} \tag{48}$$

The basic reproduction number R_0 is obtained as the $\rho(FV^{-1})$

$$R_0 = \frac{\theta\beta S}{(\theta+\mu)(\sigma+\mu+\delta)} \tag{49}$$

Substituting $S = \frac{\alpha(\rho+\mu+\omega)}{(\mu+\tau)(\rho+\mu+\omega)-\rho\tau}$ gives

$$R_0 = \frac{\theta\beta\alpha(\rho+\omega+\mu)}{(\theta+\mu)(\sigma+\mu+\delta)[(\tau+\mu)(\rho+\omega+\mu)-\rho\tau]} \tag{50}$$

Which can also be written as:

$$R_0 = \frac{\theta\beta\alpha A_2}{A_3A_4[A_1A_2-\rho\tau]} \tag{51}$$

Where $A_1 = (\tau + \mu),\ A_2 = (\rho + \omega + \mu), A_3 = (\theta + \mu), A_4 = (\sigma + \mu + \delta)$

Local Stability of Measles-Free Equilibrium Point

Theorem 1: Measles-free equilibrium point (ε_0) is locally asymptotically stable if $R_0 < 1$ otherwise it is unstable.

Proof:

$$J(S,V,E,I,R) = \begin{bmatrix} -\beta I - \mu - \tau & \rho & 0 & -\beta S & 0 \\ \rho & -\omega & 0 & 0 & 0 \\ \beta I & 0 & -\theta - \mu & \beta S & 0 \\ 0 & 0 & \theta & -\sigma - \mu - \delta & 0 \\ 0 & \omega & 0 & \sigma & -\mu \end{bmatrix} \quad (52)$$

Reducing equation (24) to upper triangular matrix and the characteristic equation gives,

$$\left|J\left((E^0)\right) - \lambda \mathrm{I}\right| = 0$$

$$\begin{vmatrix} -A_1 - \lambda & \rho & 0 & -\beta S_0 & 0 \\ 0 & -\omega - \lambda & 0 & 0 & 0 \\ 0 & 0 & -A_2 - \lambda & \beta S_0 & 0 \\ 0 & 0 & \theta & -A_3 - \lambda & 0 \\ 0 & 0 & 0 & 0 & -\mu - \lambda \end{vmatrix} = 0 \quad (53)$$

$$[(-A_1 - \lambda)(-\omega - \lambda)(-A_2 - \lambda)(-A_3 - \lambda)(-\mu - \lambda)] = 0 \quad (54)$$

Therefore

$$\lambda_1 = -A_1, \text{ or } \lambda_2 = -\omega, \text{ or } \lambda_3 = -A_2 \text{ or } \lambda_4 = -A_3, \text{ or } \lambda_5 = -\mu \quad (55)$$

Where $A_1 = (\beta I + \mu + \tau)$, $A_2 = (\theta + \mu)$ and $A_3 = (\sigma + \mu + \delta)$

From equation (54) we have

$$\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5 < 0 \quad (56)$$

Hence, we have measles-free equilibrium point to be locally asymptotically stable if (55) holds when $R_0 < 1$ and unstable when $R_0 > 1$.

Results and Discussion

The figure 2 to 5 is the graphical simulation of the basic reproduction number and some selected parameters.

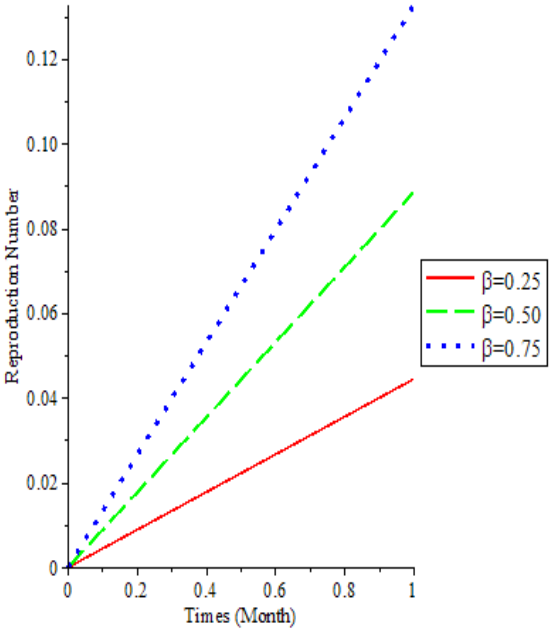

Figure 2: The effect of contact rate on reproduction number

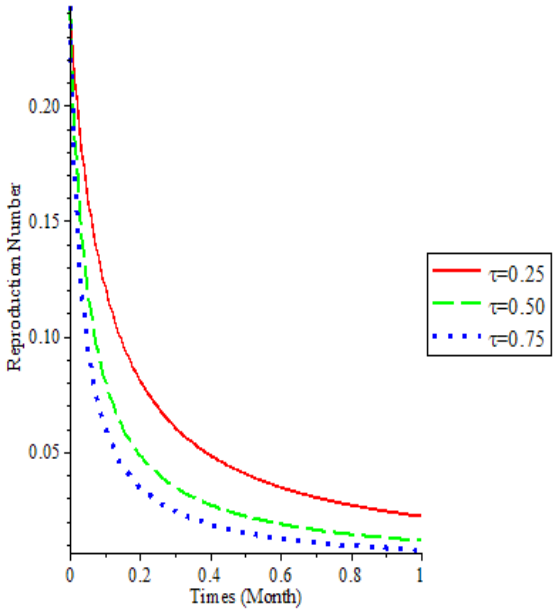

Figure 3: The effect of Vaccination Rate on Reproduction Number

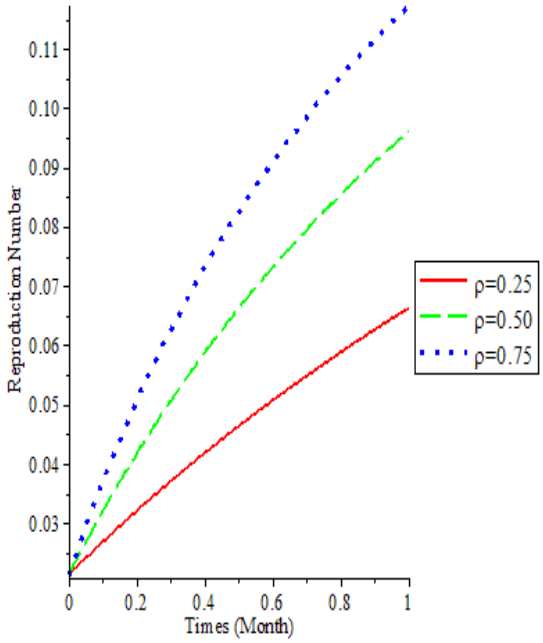

Figure 4: The effect of Immunity loss on Reproduction Number

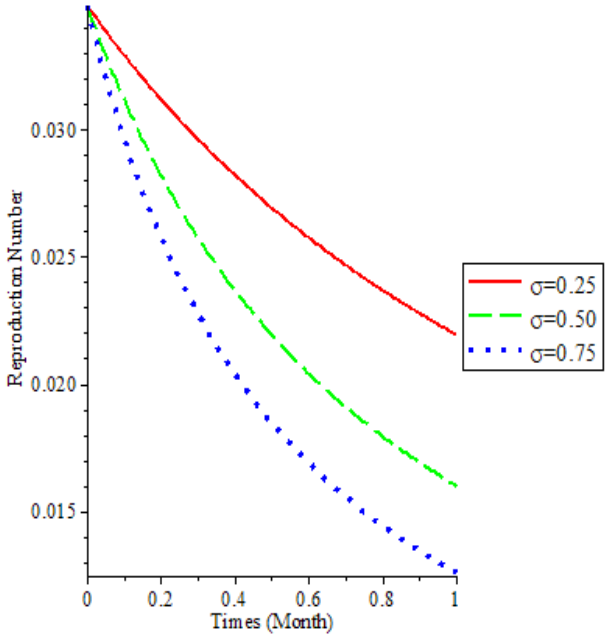

Figure 5: The effect of recovery rate on Reproduction Number

From figure 2 it shows that as the contact rate increases the reproduction number increases. This implies that the more people contact measles the more the reproduction number.

It is observed from figure 3 that as the vaccination rate increases the reproduction number decreases. This implies that the more people are vaccinated the more measles dies down and the less the reproduction number.

we observed from figure 4 that the higher the loss of vaccination, immunity decreases, and the reproduction number also increases. This implies that the more people loss their immunity the more measles persists in the population.

Figure 5 shows that as the recovery rate increases the reproduction number decreases. This implies that the more people recover from measles the less the reproduction number.

Recommendation

Based on our findings, we strongly recommend that governments and health organizations—including international partners like WHO, UNICEF, and those involved in Accelerated Immunization Activities, should collaborate to boost awareness and vaccination efforts. By leveraging local media and social platforms, they can reach a wider audience and ensure that everyone understands the importance of immunization. This unified strategy is essential for eradicating the measles virus soon.

Conclusion

In this study, we developed and analyzed a mathematical model (SVEIR) to understand measles transmission and the role of vaccination. By applying the next-generation matrix method, we derived an expression for the basic reproduction number, R_0. Our findings indicate that if R_0 is less than one, measles will eventually die out, creating a stable, measles-free situation. However, if R_0exceeds one, measles can persist in the population. Numerical simulations were conducted, and it validate the analytical findings. Overall, our analysis strongly supports vaccination as the most effective strategy to control measles outbreaks, paving the way for the measles's eventual eradication.

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