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ON THE MATHEMATICAL MODEL FOR THE SPREAD AND CONTROL OF MEASLES

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ABSTRACT

Measles is a highly contagious and potentially deadly disease that continues to pose a significant public health challenge, particularly in regions with suboptimal vaccination coverage. Measles remains a major threat to children, leading to severe complications and even death, despite the success of vaccination programs worldwide. This study delves into the dynamics of measles transmission and control, focusing on the development of a mathematical model. The research aims to propose a mathematical model using a system of first-order differential equations, categorizing the population into compartments representing vaccination(V), susceptible(S), exposed(E), infection(I), treatment(T) and recovery(R). The objectives include: obtaining Disease-Free Equilibrium State (DFES), performing stability analysis for DFES and the numerical simulation of the model was presented using maple software. Ultimately, the goal is to inform evidence-based strategies for measles vaccination and prevention, mitigating the disease's impact on children's health.

Keywords: Vaccination, Treatment, Disease-Free Equilibrium, MeV (Measles Virus), Infection

INTRODUCTION

Measles is one of the world's most contagious diseases, spread by contact with infected nasal or throat secretions (coughing or sneezing) or breathing the air that was breathed by someone with measles. The virus remains active and contagious in the air or on infected surfaces for up to two hours. For this reason, it is very infectious, and one person infected by measles can infect nine out of ten of their unvaccinated close contacts. It can be transmitted by an infected person from four days prior to the onset of the rash to four days after the rash erupts. Measles outbreaks can result in severe complications and deaths, especially among young, malnourished children. In countries close to measles elimination, cases imported from other countries remain an important source of infection (Kouadio, Kamigaki, and Oshitani, 2010).

The Measles virus is a paramyxovirus, genus morbillivirus. Measles is an infectious disease which is highly contagious through person-to-person transmission mode, with over 90% attack rates among susceptible persons. It is the first and worst eruptive fever occurring during childhood. It produces also a characteristics-red rash and can lead to serious and fatal complications including pneumonia, diarrhea and encephalitis. Many infected children subsequently suffer blindness, deafness or impaired vision. Measles confer lifelong immunity from further attacks (Momoh et al, 2013).

Measles is a disease of humans; measles virus (MeV) is not spread by any other animal species. The main symptoms of measles are fever, runny nose, cough and a rash all over the body, it also produces characteristics-red rash and can lead to serious and fatal complications including pneumonia, diarrhea, and encephalitis. Many infected children subsequently suffer blindness, deafness or impaired vision. Measles confer lifelong immunity from further attacks (Stephen et al, 2015).

According to world health organization, the two major strategies to eradicate measles are vaccination and treatment. Isolation of infected people is also important in preventing further spread of the disease. However, increasing population immunity through vaccination remains the most effective way to prevent outbreaks of measles in a community. The vaccination is mainly based on MMR (measles, mumps, and rubella) and MMRV (measles, mumps, rubella, and varicella) vaccines. These vaccines are about 95% effective as they globally prevent 4.5 million deaths yearly. There are two doses of MMR vaccine. The first dose produces 90% to 95% immunity to measles while the second dose produces a stronger immunity for those that do not respond to the first dose. Measles outbreak is prevented in a community if 90% to 95% of children are vaccinated (Madubueze et al, 2022).

The World Health Organization (WHO) recommends that children should receive two doses of the MMR vaccine to ensure they are immune. The first dose is usually given at 9 months of age in countries where measles is common and 12-15 months in other countries. A second dose should be given later in childhood, usually at 15–18 months. A second dose is recommended for all children, essential to protect the approximately 15% of children who don't develop protective immunity after their first dose. In developing countries where measles is highly pronounced the WHO Doctors recommended two doses of vaccines to be given at six and nine months of age (WHO, 2024).

Treatment focuses on supportive care to relieve symptoms and prevent complications, which may include rest, plenty of fluids, and over-the-counter medication for fever. Vitamin A supplements can reduce complications in children, especially those under five. Antibiotics are used to treat secondary bacterial infections like ear or eye infections. You should also contact a healthcare provider if your child has been exposed to measles or has symptoms, and keep them home to prevent spreading the virus (WHO, 2025).

Momoh et al. (2013) formulate a deterministic, compartmental, mathematical model to describe the transmission dynamics of measles. The model considered the Susceptible (S), Exposed (E), Infected (I) and Recovered (R) classes. They concluded that, in high measles prevalence countries, testing, diagnosis and exposed individuals at latent period therapy will have a much greater impact on the disease burden. While this conclusion may have practical implications for the control of measles infections, more realistic models that are specific for measles infection and more detailed data need to be employed to further explore its significance in future study.

Christopher et al. (2017) developed a mathematical model for the dynamics of measles under the combined effect of vaccination at the susceptible class, and administering measles drug therapy to screened infected individuals in the exposed class. They compartmentalized the Population into five compartments namely: Susceptible (S), Vaccinated (V), Exposed (E), Infectious (I) and Recovered (R). Analytical study was carried out using linearized stability, the results of the model analysis showed that the model has a unique Disease-Free Equilibrium (DFE) points which are locally asymptotically stable (LAS) whenever the Basic Reproductive Number (R_0) is less than one ($R_0 < 1$). They also carried out numerical experiments using data from momoh et al (2013), the results of the numerical experiments revealed that eradicating measles is possible and more efficient if 50% of exposed individuals receive measles therapy and 95% of the susceptible individuals are vaccinated.
Samuel et al. (2020) developed a mathematical deterministic modeling approach to model measles disease by using the data pertinent to Nigeria. The total human population N(t) is divided into four sub-populations of Susceptible S(t), Exposed E(t), Infective I(t) and Recovered R(t). They introduced control measure into the susceptible and exposed classes to study the prevalence and control of the measles disease. It is recognized that if more people in the susceptible class get immunization and the exposed people at latent period goes for treatment and therapy during this state before they become infective, the disease will be eradicated more quickly with time.
Mahmoud, and Attila, (2023) developed a mathematical model that incorporates a seasonal transmission parameter to examine the measles transmission dynamics. The population is splited into six compartments: Susceptible S(t), individuals who received the first dose of the MMR vaccine $V_1(t)$, individuals who received the second dose of the MMR vaccine $V_2(t)$, Exposed or Asymptomatic E(t), Symptomatic or Infectious I(t), and Recovered Individuals R(t). Their model was applied to the measles outbreak that occurred in Pakistan from 2019 to 2021 and provided a good fit to the observed data. It was found that $R_0 > 1$, indicating that the disease will persist in the population. Their findings highlight the need to increase vaccination coverage and efficacy to mitigate the impact of the epidemic. Their simulations demonstrate that a shorter incubation period accelerates the spread of the disease, while a higher vaccination coverage rate reduces its impact. The importance of the second dose of the measles vaccine is emphasized, and a higher vaccine efficacy rate can also help bring R_0 below one. To prevent future outbreaks, increasing vaccination coverage among the population is the most effective way to reduce the transmission of measles.

MATERIALS AND METHODS

Model Formation

The insights gained from the review of existing literature were utilized to create a mathematical model that captures the spread and control of measles. The approach involved partitioning the population into six distinct compartments: vaccinated (V), susceptible (S), exposed (E), infected (I), treatment (T) and recovered (R).

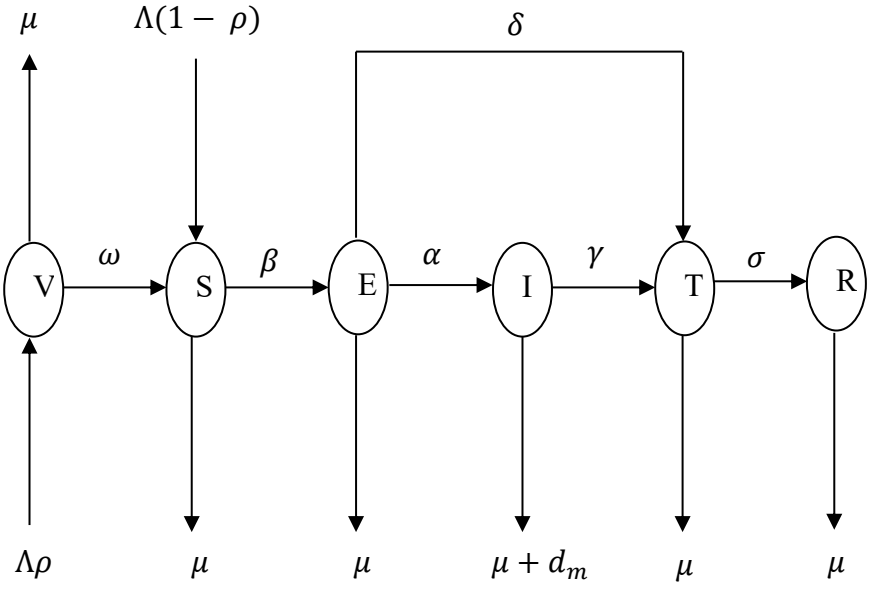

Figure 1: Schematic Diagram for the Spread and Control of Measles

Model Explanation

The model explanation for the VSEITR is as follows:
The population of the Vaccinated Class (V) increases with the incoming proportions of new born that are vaccinated at the rate $\Lambda\rho$. The population of the class (V) decreases due to waning off of immunity after vaccination at the rate ω and also decreases by natural death at the rate μ.
The population of the Susceptible Class (S) increases with the incoming proportion of new born not vaccinated at the rate $\Lambda(1-\rho)$ and also increases when individuals move out of the Vaccinated Class (V) thereby becoming Susceptible due to waning off of immunity after vaccination at the rate ω. The population of the class (S) will decrease when individuals move out of the class (S) via interaction or contact with infected individuals and become exposed at the rate β and also decreases by natural death at the rate μ.
The population of the Exposed Class (E) will increase when individual move out of the class (S) via interaction or contact with infected individuals and become exposed at the rate β, the population of the class (E) decreases when exposed individuals move into the Treatment Class (T) at the rate δ It will also decrease after the maturation of measles infection via

interaction with infected individuals at the rate α and also decrease due to natural death at the rate μ.

The population of the Infected Class (I) increases due to maturation of the measles disease when individual move out of the class (E) via interaction or contact and become infected at the rateα. The population of the class (I) will decrease when infected individuals move into the Treatment Class (T) at the rate γ, natural death at the rate μ and also death due to measles infection at the rate d_m.

The population of the Treatment Class (T) decreases by natural death at rate μ, and increases at rate δ and γ when the exposed and infected individuals are treated, respectively.

The population of the Recovered Class (R) increases when the treated individuals move into the class (R) at rate σ and decreases only by natural death at the rateμ.

The following equations represent the schematic diagram.

$$\frac{dV}{dt} = \Lambda\rho - (\omega + \mu)V \quad (1)$$

$$\frac{dS}{dt} = \Lambda(1-\rho) + \omega V - \beta S - \mu S \quad (2)$$

$$\frac{dE}{dt} = \beta S - (\alpha + \delta + \mu)E \quad (3)$$

$$\frac{dI}{dt} = \alpha E - (\gamma + \mu + d_m)I \quad (4)$$

$$\frac{dT}{dt} = \gamma I + \delta E - (\sigma + \mu)T \quad (5)$$

$$\frac{dR}{dt} = \sigma T - \mu R \quad (6)$$

Where:

V = Vaccinated individuals.

S = Susceptible individuals.

E = Exposed individuals.

I = Infected individuals.

T = Treated individuals.

R = Recovered individuals.

Λ = Recruitment rate.

$\Lambda(1-\rho) =$ The portion of individuals that are not vaccinated.

$\Lambda\rho =$ The portion of the vaccinated individuals.

ω = Rate at which the vaccinated individuals become susceptible.

β = Rate at which the susceptible individuals become exposed.

α = Rate at which the exposed individuals become infected.

γ = Rate at which the infected individuals get treated.

σ = Rate at which the exposed and infected individuals recovered due to treatment.

$\delta =$ Rate at which the exposed individuals get treated.

μ = Natural death rate.

$d_m =$ Death rate due to measles.

Force of infection is given as $\beta = \frac{\eta c}{N} I$ (7)

The total population at time t, N(t) = V(t) + S(t) + E(t) + I(t) + T(t) + R(t).

$\eta =$ Probability of infection upon contact.

c = Per capita contact rate.

Equilibrium State of the Model

At Equilibrium State, each of the Model Equations is set to Zero. i.e

$$\frac{dV^*}{dt} = \frac{dS^*}{dt} = \frac{dE^*}{dt} = \frac{dI^*}{dt} = \frac{dT^*}{dt} = \frac{dR^*}{dt} = 0 \quad (8)$$

Equations (1) to (6) becomes

$$\Lambda\rho - (\omega + \mu)V^* = 0 \quad (9)$$

$$\Lambda(1-\rho) + \omega V^* - \frac{\eta c I^* S^*}{N^*} - \mu S^* = 0 \quad (10)$$

$$\frac{\eta c I^* S^*}{N^*} - (\alpha + \delta + \mu)E^* = 0 \quad (11)$$

$$\alpha E^* - (\gamma + \mu + d_m)I^* = 0 \quad (12)$$

$$\gamma I^* + \delta E^* - (\sigma + \mu)T^* = 0 \quad (13)$$

$$\sigma T^* - \mu R^* = 0 \quad (14)$$

From Equation (9), gives

$$V^* = \frac{\Lambda\rho}{(\omega+\mu)} \quad (15)$$

From Equation (14), gives

$$R^* = \frac{\sigma T^*}{\mu} \quad (16)$$

From Equation (12), gives

$$E^* = \frac{(\gamma+\mu+d_m)I^*}{\alpha} \quad (17)$$

From Equation (13), gives

$$T^* = \frac{\gamma I^* + \delta E^*}{(\sigma+\mu)} \quad (18)$$

Substituting Equation (17) into (18), yields

$$T^* = \left[\frac{\alpha\gamma + \delta(\gamma+\mu+d_m)}{\alpha(\sigma+\mu)}\right] I^* \quad (19)$$

Adding Equations (10) and (11) together, yields

$$\mu S^* = \Lambda(1-\rho) + \omega V^* - (\alpha + \delta + \mu)E^* \quad (20)$$

Substituting Equations (15) and (17) into (20), yields

$$S^* = \frac{\Lambda(\omega+\mu\rho+\mu)}{\mu(\omega+\mu)} - \left[\frac{(\omega+\mu)(\alpha+\delta+\mu)(\gamma+\mu+d_m)}{\alpha(\omega+\mu)\mu}\right] I^* \quad (21)$$

Substituting Equation (19) into (16), yields

$$R^* = \left[\frac{\sigma\alpha\gamma + \sigma\delta(\gamma+\mu+d_m)}{\alpha(\sigma+\mu)\mu}\right] I^* \quad (22)$$

Substituting Equations (17) and (21) into (11), yields

$$\left\{\frac{\eta c}{N^*}\left[\frac{\alpha\Lambda(\omega+\mu\rho+\mu) - [(\omega+\mu)(\alpha+\delta+\mu)(\gamma+\mu+d_m)]I^*}{\alpha(\omega+\mu)\mu}\right] - (\alpha+\delta+\mu)\left(\frac{\gamma+\mu+d_m}{\alpha}\right)\right\} I^* = 0 \quad (23)$$

$$\Longrightarrow \quad I^* = 0 \quad (24)$$

OR

$$I^* = \frac{\left\{\frac{\Lambda\alpha\eta c(\omega+\mu\rho+\mu)}{N^*\mu(\omega+\mu)(\alpha+\delta+\mu)(\gamma+\mu+d_m)}\right\} - 1}{\eta c(\omega+\mu)(\alpha+\delta+\mu)(\gamma+\mu+d_m)} \quad (25)$$

Disease-Free Equilibrium State (DFES)

The equilibrium state with the absence of infection is called the disease-free equilibrium state (DFES). Hence, in the absence of infection, $I = 0$. Therefore, the disease-free equilibrium states of the model are express as follows:

Lemma 1

The DFES of the Model exist at

$$\begin{pmatrix} V^0 \\ S^0 \\ E^0 \\ I^0 \\ T^0 \\ R^0 \end{pmatrix} = \begin{pmatrix} \frac{\Lambda\rho}{(\omega+\mu)} \\ \frac{\Lambda(\omega+\mu\rho+\mu)}{\mu(\omega+\mu)} \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad (26)$$

Proof:

From Equation (15), gives

$$V^0 = \frac{\Lambda\rho}{(\omega+\mu)} \quad (27)$$

From Equation (24), gives

$$I^0 = 0 \quad (28)$$

Substituting Equation (28) into (17), (19), (21) and (22), gives

$$E^0 = T^0 = R^0 = 0 \quad (29)$$

and

$$S^0 = \frac{\Lambda(\omega+\mu\rho+\mu)}{\mu(\omega+\mu)} \quad (30)$$

Hence, the Lemma is proved.

Stability Analysis of Zero Equilibrium State (ZES)

We recall that the system of equations of the model at equilibrium is:

$$\Lambda\rho - (\omega + \mu)V = 0 \quad (31)$$

$$\Lambda(1-\rho) + \omega V - \frac{\eta c I}{N} S - \mu S = 0 \quad (32)$$

$$\frac{\eta c I}{N} S - (\alpha + \delta + \mu)E = 0 \quad (33)$$

$$\alpha E - (\gamma + \mu + d_m)I = 0 \quad (34)$$

$$\gamma I + \delta E - (\sigma + \mu)T = 0 \quad (35)$$

$$\sigma T - \mu R = 0 \quad (36)$$

The Jacobian Matrix of the system of Equations at Disease-Free Equilibrium (DFE) is:

$$\boldsymbol{J}=\begin{bmatrix} -(\omega+\mu) & 0 & 0 & 0 & 0 & 0 \\ \omega & -(\frac{\eta cI}{N}+\mu) & 0 & -\frac{\eta cS}{N} & 0 & 0 \\ 0 & \frac{\eta cI}{N} & -(\alpha+\delta+\mu) & \frac{\eta cS}{N} & 0 & 0 \\ 0 & 0 & \alpha & -(\gamma+\mu+d_m) & 0 & 0 \\ 0 & 0 & \delta & \gamma & -(\sigma+\mu) & 0 \\ 0 & 0 & 0 & 0 & \sigma & -\mu \end{bmatrix} \quad (37)$$

The characteristic equation is obtained from the Jacobian Determinant with the Eigen Values λ i.e

$$\boldsymbol{det(J-\lambda I)}=$$

$$\begin{bmatrix} -(\omega+\mu)-\lambda & 0 & 0 & 0 & 0 & 0 \\ \omega & -(\beta+\mu)-\lambda & 0 & -\frac{\eta cS}{N} & 0 & 0 \\ 0 & \beta & -(\alpha+\delta+\mu)-\lambda & \frac{\eta cS}{N} & 0 & 0 \\ 0 & 0 & \alpha & -(\gamma+\mu+d_m)-\lambda & 0 & 0 \\ 0 & 0 & \delta & \gamma & -(\sigma+\mu)-\lambda & 0 \\ 0 & 0 & 0 & 0 & \sigma & -\mu-\lambda \end{bmatrix} = 0 \quad (38)$$

The characteristic equation is

$$(-(\omega+\mu)-\lambda)(-(\beta+\mu)-\lambda)(-(\alpha+\delta+\mu)-\lambda)(-(\gamma+\mu+d_m)-\lambda)(-(\sigma+\mu)-\lambda)(-\mu-\lambda)=0 \quad (39)$$

Either,

$$(-(\omega+\mu)-\lambda)=0 \text{ or } (-(\beta+\mu)-\lambda)=0 \text{ or } (-(\alpha+\delta+\mu)-\lambda)=0 \text{ or } (-(\gamma+\mu+d_m)-\lambda)=0 \text{ or } (-(\sigma+\mu)-\lambda)=0 \text{ or } (-\mu-\lambda)=0 \quad (40)$$

Therefore,

$$\lambda_1=-(\omega+\mu),\ \lambda_2=-(\beta+\mu), \lambda_3=-(\alpha+\delta+\mu),\ \lambda_4=-(\gamma+\mu+d_m),\ \lambda_5=-(\sigma+\mu),\ \lambda_6=-\mu \quad (41)$$

Hence, the DFE is stable since the Eigen-Values are less than zero.

Analytical Solution of the Model

The analytical solution of the model is carried out using homotopy perturbation method (HPM). The fundamental of homotopy perturbation method (HPM) was first proposed by Ji (2000). The homotopy perturbation method (HPM), which provides analytical approximate solution, is applied to various linear and non-linear equations.

$$\frac{dV}{dt}+(\omega+\mu)V-\Lambda\rho=0 \quad (42)$$

$$\frac{dS}{dt}+\beta S+\mu S-\Lambda(1-\rho)-\omega V=0 \quad (43)$$

$$\frac{dE}{dt}+(\alpha+\delta+\mu)E-\beta S=0 \quad (44)$$

$$\frac{dI}{dt}+(\gamma+\mu+d_m)I-\alpha E=0 \quad (45)$$

$$\frac{dT}{dt}+(\sigma+\mu)T-\gamma I-\delta E=0 \quad (46)$$

$$\frac{dR}{dt}+\mu R-\sigma T=0 \quad (47)$$

With the following initial conditions:

$$V(0)=V_0, S(0)=S_0, E(0)=E_0, I(0)=I_0, T(0)=T_0 \text{ and } R(0)=R_0 \quad (48)$$

The following analytical solution is obtained as follows:

$$V(t)=V_0+(\Lambda\rho-(\omega+\mu)V_0)t-(\omega+\mu)(\Lambda\rho-(\omega+\mu)V_0)\frac{t^2}{2} \quad (49)$$

$$I(t)=I_0+(\alpha E_0-(\gamma+\mu+d_m)I_0)t+[\alpha(\beta S_0 I_0-(\alpha+\delta+\mu)E_0)-(\gamma+\mu+d_m)(\alpha E_0-(\gamma+\mu+d_m)I_0)]\frac{t^2}{2} \quad (50)$$

$$R(t)=R_0+(\sigma T_0-\mu R_0)t+[\sigma(\delta E_0+\gamma I_0-(\sigma+\mu)T_0)-\mu(\sigma T_0-\mu R_0)]\frac{t^2}{2} \quad (51)$$

RESULTS AND DISCUSSION

Table 1: Values for Population-dependent Parameters of the Model

S/N	Parameters	Values (day^{-1})	Source
1	Λ	20,839	Federal Reserve Bank of St. Louis. (2025)
2	μ	3.22 x 10^{-5}	World Bank. (2025)
3	ρ	0.378	World Health Organization, & United Nations Children's Fund. (2023)
4	ω	(0,1)	World Health Organization. (2017)
5	α	0.0909	Centers for Disease Control and Prevention. (2024)
6	δ	0.20	Centers for Disease Control and Prevention. (2025)
7	γ	0.1236	Centers for Disease Control and Prevention. (2024)
8	d_m	0.00138	Portnoy, et al. (2019)
9	σ	0.25	WHO, (2013)
10	c	12	Prem, K., Cook, A. R., & Jit, M. (2017)
11	η	0.126	Guerra, et al. (2017)

Sensitivity Analysis

In order to assess the robustness of the model, a one-way sensitivity analysis is conducted. This involves individually varying the value of each input parameter, while keeping all other parameters constant. The range of variation for each parameter is determined based on the available literature and expert opinions. For instance, in the case of variableα, three different scenarios were tested: a conservative scenario where α is set at its lower bound, an optimistic scenario where α is set at its upper bound, and a base case scenario where $\alpha$is set at its most likely value. The process is repeated for each input parameter, generating a range of results for the outcome variable. To visualize the impact of parameter variation on the results, the sensitivity curves is plotted using maple software, which depict the relationship between each input parameter and the outcome variable. By examining the shape and magnitude of these curves, one is able to identify which parameters had the most significant influence on the results. Furthermore, a diagram analysis is conducted to identify the parameters with the greatest impact on the outcome variable. The diagram presents the sensitivity analysis results in a graphical format, ranking the parameters in descending order of their influence.

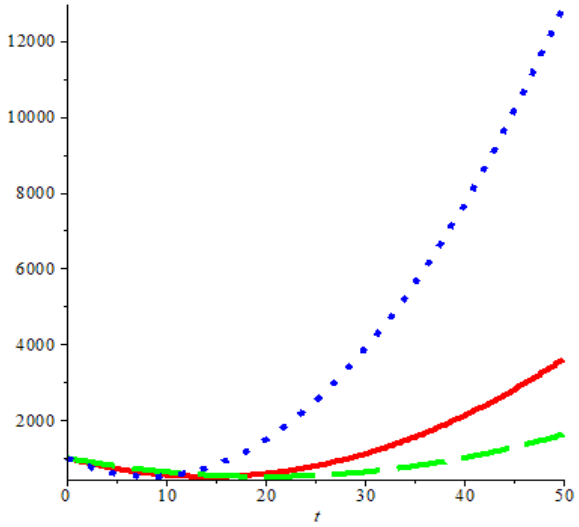

Figure 2: Simulation of Vaccinated Class (V)

Figure 2 shows the effect of varying the rate ω, at which vaccinated individuals become susceptible under three values of $\Lambda = (1.0, 0.8, 1.6)$, $\rho = (0.3, 0.3, 0.25)$, $\beta = (0.35, 0.45, 0.2)$, $\omega = (0.05, 0.3, 0.1)$ per day. The blue dotted curve represents the case where $\omega = 0.3$, meaning vaccinated individuals loses immunity very quickly. This leads to a rapid and steep rise in $V(t)$ after an initial dip, showing that the vaccinated population becomes unstable as many people revert to susceptibility and must be vaccinated again. The red solid curve corresponds to $\omega = 0.1$, a moderate rate of immunity loss. Here, $V(t)$ declines at first but later grow steadily, indicating a balance between vaccination and waning immunity. The green dashed curve shows $\omega = 0.05$, where immunity lasts longer. The vaccinated class remains relatively stable, with only a slow upward trend that reflects sustained vaccine protection and limited re-entry into susceptibility. Across all curves, Λ, β, and ρ jointly shape the dynamics: higher Λ replenishes the vaccinated class, while high β and ω weaken its stability. Thus, the green curve reflects strong and lasting vaccine performance, the red curve shows moderate durability, and the blue curve exposes how rapid immunity loss undermines long-term disease control.

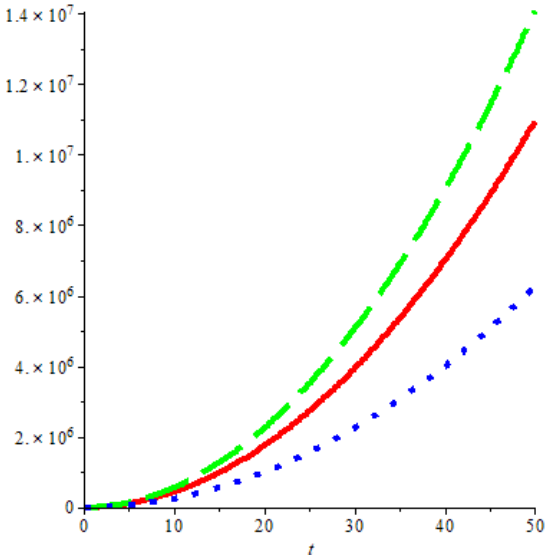

Figure 3: Simulation of Infected Class (I)

Figure 3 illustrates how the infected class $I(t)$ behaves when the waning rate ω varies, using the same associated parameters $\Lambda = (1.0, 0.8, 1.6)$, $\rho = (0.3, 0.3, 0.25)$, and $\beta = (0.35, 0.45, 0.2)$. The green dashed curve corresponds to $\omega = 0.05$, showing the highest growth in infection over time. This happens because slower loss of immunity keeps a large pool of susceptible individuals available, and as recruitment Λ remains high, infections continue to rise sharply. The red solid curve represents $\omega = 0.1$, showing moderate growth of infection as immunity wanes faster than in the first case, allowing some balance between new infections and recoveries. The blue dotted curve, representing $\omega = 0.3$, shows the lowest rise in infections because rapid immunity loss causes quicker transitions between classes, leading to fewer individuals remaining infectious for long. The overall pattern suggests that infections increase with lower ω values, where immunity is more stable, but the susceptible population keeps expanding. Hence, when immunity wanes slowly and recruitment is high, the infected class grows steeply, while faster immunity loss limits infection spread by accelerating the shift between epidemiological states.

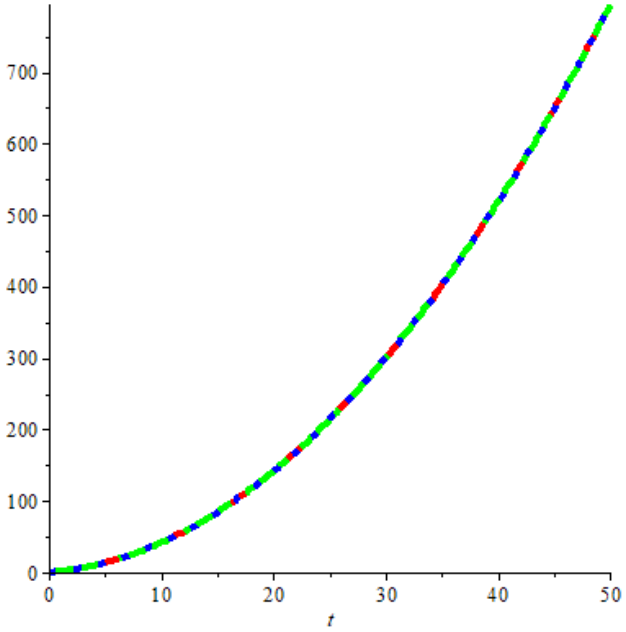

Figure 4: Simulation of Recovered Class (R)

Figure 4 shows how the recovered class $R(t)$ changes under different values of the waning rate ω, while keeping $\Lambda = (1.0, 0.8, 1.6)$, $\rho = (0.3, 0.3, 0.25)$, and $\beta = (0.35, 0.45, 0.2)$ constant. The green dashed ($\omega = 0.05$), red solid ($\omega = 0.1$), and blue dotted ($\omega = 0.3$) curves nearly overlap, which means the waning rate has little influence on recovery within the simulated period. The consistent upward growth of $R(t)$ shows that recovery is mainly governed by the recruitment rate Λ and the recovery rate σ. When σ is high, more individuals enter the population, increasing the number of people who eventually recover from infection after treatment. A slightly lower ρ value (0.25) implies slower recovery, while a higher one (0.3) speeds up the transition from infection to recovery. These small differences in ρ explain the uniform but steady growth in all curves. The behaviour suggests that, although waning immunity affects the susceptible and infected classes more directly, the combined effect of strong recruitment and steady recovery ensures that the recovered population remains stable and increases gradually over time.

CONCLUSION

A deterministic model for the transmission dynamics of measles was developed and analyzed using a compartmental structure with vaccination (V), susceptible (S), exposed (E), infectious (I), treatment (T) and recovered (R) classes. Infection followed a standard-incidence term with β = ηc/N and disease-induced mortality in the infectious class was incorporated. Control parameters considered include recruitment rate (Λ) and vaccination coverage at birth (ρ), waning rate (ω), and recovery rate (σ). The Zero Equilibrium State (ZES) for Disease-Free Equilibrium was analyzed for stability and it reveals that it is stable since the Eigen-values are less than zero. The implication here is that if all Eigen-values are less than zero, then the disease is at its Disease-Free Equilibrium, meaning the disease is not spreading and the population is Disease-Free. Hence, it suggests the illness can be controlled, so the Health Practitioners are encouraged to make sure that children under the age of 5 years are vaccinated both at birth and at school.

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