



Global Stability Analysis to Measles Transmission Dynamic with Vaccination

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ABSTRACT

Measles remains a significant public health concern globally, despite the availability of vaccines. Understanding the dynamics of measles transmission through mathematical modeling is crucial for designing effective control strategies. In this study, we present a comprehensive mathematical model that incorporates susceptible, exposed, infectious, recovered and vaccinated compartments to capture the complexity of measles dynamics. We conducted a global stability analysis of the proposed model to explore the long-term behavior of measles transmission dynamics. By analyzing the model's equilibrium points and their stability properties, we elucidate the conditions under which measles can persist or be eradicated within a population. Our analysis accounts for the impact of vaccination coverage and vaccine efficacy on the dynamics of measles transmission. Furthermore, we perform sensitivity analysis to identify key parameters driving the persistence or elimination of measles. We assess the effectiveness of vaccination strategies in reducing measles incidence and our findings provide valuable insights into the dynamics of measles transmission and the potential impact of vaccination programs on disease control which shows a 19.5% vaccination rate effectively prevents transmission, while a 1.25 percent decreases transmission but cannot completely eliminate it, and a 0.57% rate suggests significant spread necessitates further actions or higher vaccination rates.

Keyword: Measles, Transmission, Global stability, Vaccination

INTRODUCTION

Measles is a highly contagious disease, primarily affecting children, and vaccination is the most effective method to prevent its spread. Measles mortality decreased from 761,000 in 2000 to 130,000 in 2022 due to vaccination. The disease remains active for up to two hours, affecting nine out of ten unvaccinated contacts. Community-wide vaccination is the most effective method for measles prevention, with routine and mass immunization campaigns reducing global measles deaths (WHO, 2023). Measles epidemiology is a crucial field of study for scientists, utilizing mathematical,

theoretical, and experimental methods to determine effective control and prevention methods globally. Momoh *et al.* (2013) looked at the effects of asymptomatic people throughout the latent period on measles dynamics using an SEIR deterministic epidemic model. Kuddus *et.al* (2021) developed a modified measles compartmental model in Bangladesh, revealing two equilibrium points and a significant impact of transmission rate, progression rate, and double dose vaccination rate. Jaharuddin and Bakhtiar's 2020 mathematical model uses the Pontryagin maximum principle to identify optimal controls for minimizing measles

exposure and infected individuals, demonstrating effective reduction in cases. Abadi *et al*, (2022) applied a model for measles virus transmission in Jakarta, Indonesia, revealing that hospitalization for measles patients improved vaccination effectiveness, urging city policymakers. Abboubakar *et al*, (2022) utilized the Caputo derivative to model measles transmission dynamics, demonstrating its unique solution and global stability, and validating its results through simulations. Sowole *et al*, (2023) utilized a mathematical deterministic modeling method to study measles disease prevalence and control in Nigeria, highlighting the significant impact of control measures. Dipo Aldila and Dinda Asrianti developed a modified SVIQR model for measles infection control, establishing a measles-free and endemic equilibrium using R_0 as an indicator. Alemneh and Belay (2023) developed a SVIRP model for measles, revealing indirect contact rate maximizes transmission dynamics, and prevention and treatment strategies significantly reduce disease effects. A mathematical model by James Peter *et al*, (2022), revealed that combined control strategies reduce measles infection rates faster than single strategies, emphasizing the importance of vaccination rates and hospitalization in controlling the disease in Nigeria. Motivated by the above literature, we extend the work of Alemneh & Belay, 2023 by including exposed class in the developed model for measles to help with understanding the illness's course, spreading, and assessing prevention actions. To comprehend the long-term behavior of a measles model incorporating vaccination and bacterium class, global stability analysis is essential. It aids in figuring out equilibrium, evaluating control strategies, and determining the dynamics of disease. It helps with public health actions and strategies by identifying critical elements

influencing the transmission and control of disease.

Model Formulation

$N(t)$ represents the total population of the model at time (t), which is split up into six (6) compartments. Viz: The susceptible population $S(t)$, describes those who have not had the measles but who could get it if they come into contact with the virus. The measles vaccination population $V(t)$, or those who have got the two dose of vaccination, is essential in lowering vulnerability and halting the disease's spread. The exposed population $E(t)$, describes a category of people who have contracted the measles but are not yet contagious; it is a stage in between infection and sickness. Those who are presently infected with the measles and have the potential to transfer the virus to others are considered members of the infected class $I(t)$. The recovered populations $R(t)$, describe people who have overcome their measles infection, become immune to other infections, and are no longer at risk of contracting the illness. The bacteria class $P(t)$ adds complexity to disease transmission, indicating the presence of other infectious agents or environmental factors. Therefore $N(t)$ equal to $S(t) + E(t) + I(t) + V(t) + R(t) + P(t)$. Where φ_h , is the vaccination rate from susceptible individuals and Π is the recruitment rate of humans into the susceptible class, and q is the fraction of newly recruited community members who receive vaccinations. Following a measles infection, the susceptible class shrank; the forces of infection are depicted by $\lambda = \beta_h I + \beta_p P$. The class of persons who have received

vaccinations declines at a rate of ρ_h , when the rate of waning vaccination for susceptible individuals rises. The rate at which an exposed individual is moved from the susceptible class to the infected class is represented by λ . The rate at which an exposed individual recovers from the infection is represented by ϕ_h , and the rate at which an induced death occurs is represented by δ_h . The rate of environmental contamination caused by diseased individuals is θ_h . The number of people who recover

from infection at the rate of ϕ_h and those who receive complete vaccinations at the rate of γ_h increase the recovered class. A rate μ_h , is used to represent the natural death rate of the human population. The rate θ_h , which is caused by infected individuals coughing and sneezing into the air and measles virus droplets landing on different things, creates the pathogen population. The pathogen decay rate is considered to be μ_p .

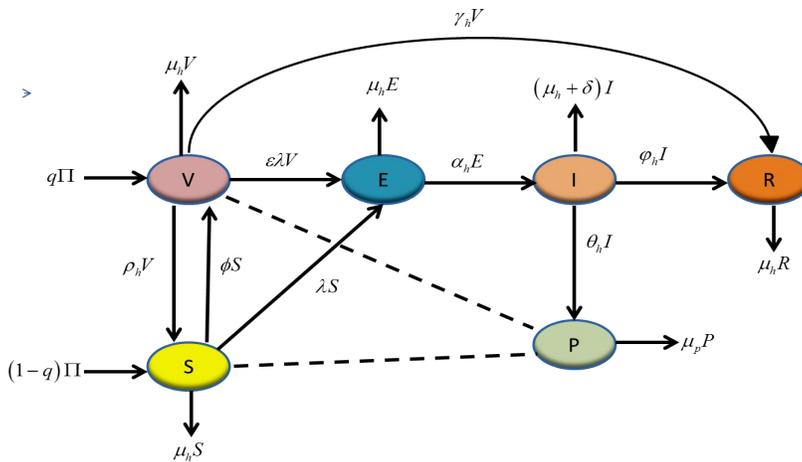


Figure1: Schematic Diagram depicting the measles model (1)

$$\left. \begin{aligned}
 \frac{dS}{dt} &= (1-q)\Pi + \rho_h V - (\mu_h + \lambda + \phi) S \\
 \frac{dE}{dt} &= (S + \epsilon_h V) \lambda - (\mu_h + \alpha_h) E \\
 \frac{dI}{dt} &= \alpha_h E - (\mu_h + \delta_h + \phi_h + \theta_h) I \\
 \frac{dR}{dt} &= \gamma_h V + \phi_h I - \mu_h R \\
 \frac{dV}{dt} &= q\Pi + \phi S - (\mu_h + \epsilon_h \lambda + \rho_h + \gamma_h) V \\
 \frac{dP}{dt} &= \theta_h I - \mu_p P
 \end{aligned} \right\} \quad (1)$$

The measles model's (1) positive invariant region is a state space where some epidemiological quantities stay constant or

non-decreasing across time, and where solutions to differential equations remain non-negative.

Positivity of the Solution

Theorem 1: Suppose that the initial data for the model (1) be $S(0) > 0, E(0) \geq 0, I(0) \geq 0, R(0) \geq 0, V(0) > 0, P(0) \geq 0$, then the solutions $S(t), E(t), I(t), R(t), V(t)$ and $P(t)$ of the model with positive initial data will remain positive for all $t \geq 0$.

Proof

Let $t_1 = \sup\{t > 0 : S(0) > 0, E(0) \geq 0, I(0) \geq 0, R(0) \geq 0, V(0) > 0, P(0) \geq 0\} > 0$ and from the first equation of the model 1, we have

$$\frac{dS}{dt} \geq -(\mu_h + \lambda + \phi)S. \quad (2)$$

So that,

$$S(t_1) = S(0) \exp\left(-\exp\left(\int_0^{t_1} (\mu_h + \lambda + \phi) dx\right)\right) > 0.$$

Hence, it can be shown that $E(t_1) > 0, I(t_1) > 0, R(t_1) > 0, V(t_1) > 0, P(t_1) > 0$. Hence all the solutions of the model remain positive for all $t \geq 0$.

The Invariant Region

Theorem 2: The closed set

$$\Omega = \left\{ (S, E, I, R, V, P) \in \mathbb{R}_+^6; N \leq \frac{\Pi}{\mu_h} \right\}. \quad (3)$$

is positively- invariant and attract all the positive solutions of the model

Proof

Adding equations in system (1), yield

$$\frac{dN}{dt} \leq \Pi - \mu_h N. \quad (4)$$

It follows from (4), and the Gronwall inequality, that

$$N \leq N_0 e^{-\mu_h t} + \frac{\Pi}{\mu_h} (1 - e^{-\mu_h t}).$$

At $t \rightarrow \infty$ the inequality becomes $N \leq \frac{\Pi}{\mu_h}$, which shows that the feasible solution of the

system as $N \rightarrow \frac{\Pi}{\mu_h}$ is the system consist of six possible solutions and the solution model for this

system is uniformly bounded in the subset of \mathbb{R}_+^6 the feasible solution of the region Ω is positively invariant and attracting with respect to system Ω , the invariant region is Ω .

Stability Analysis

Analyzing a system's behavior close to equilibrium points like endemic or disease-free equilibria, while assuming small

disturbances is known as local stability analysis. By evaluating the stability of the disease-free equilibrium and the endemic equilibrium, it assists in the prediction of

short-term trends and outbreaks in certain populations. The behavior of the system over its whole state space is taken into account by global stability analysis, however. Depending

on the original conditions, the system will eventually settle into the equilibrium, which is either endemic or disease-free, if the equilibrium is globally asymptotically stable.

Local and Global Stability of the Disease-free Equilibrium

The measles model (4)'s DFE E^* , is provided by

$$E^* = \{S^*, 0, 0, R^*, V^*, 0\} \tag{4}$$

Where,

$$S^* = \frac{(1-q)\Pi(\mu_h + \rho_h + \gamma_h) + \rho_h q \Pi}{(\mu_h + \alpha_h)(\mu_h + \rho_h + \gamma_h) - \rho_h \phi}, \quad R^* = \frac{\gamma_h q \Pi(\mu_h + \phi) + \gamma_h(1-q)\phi \Pi}{(\mu_h + \alpha_h)(\mu_h + \rho_h + \gamma_h) - \rho_h \phi}$$

$$V^* = \frac{q \Pi(\mu_h + \phi) + (1-q)\phi \Pi}{(\mu_h + \alpha_h)(\mu_h + \rho_h + \gamma_h) - \rho_h \phi}$$

The method of next generation operator on system (4) can be used to establish the linear stability of E^* , (Van den Driessche & Watmough, 2002). The transition terms' matrix v and the new infection terms' matrix F are provided by

$$F = \begin{pmatrix} 0 & \beta_h(S^* + \varepsilon_h V^*) & \beta_p(S^* + \varepsilon_h V^*) \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad \text{and}$$

$$v = \begin{pmatrix} (\mu_h + \alpha_h) & 0 & 0 \\ -\alpha_h & (\mu_h + \delta_h + \varphi_h + \theta_h) & 0 \\ 0 & -\theta_h & \mu_p \end{pmatrix}.$$

Hence, the basic number for reproduction (R_0) is provided as

$$R_0 = \rho(F v^{-1}) = R_1 + R_2,$$

where, $R_1 = \frac{\beta_h \alpha (S^* + \varepsilon_h V^*)}{(\mu_h + \alpha_h)(\mu_h + \delta_h + \varphi_h + \theta_h)}$ and $R_2 = \frac{\beta_p \alpha_h \theta_h (S^* + \varepsilon_h V^*)}{\mu_p (\mu_h + \alpha_h)(\mu_h + \delta_h + \varphi_h + \theta_h)}$.

Hence,

$$R_0 = \frac{\Pi \alpha_h (\beta_h \mu_p + \beta_h \theta_h) \{ (1-q)(\mu_h + \rho_h + \gamma_h + \varepsilon_h \phi) + q(\varepsilon_h(\mu_h + \phi) + \rho_h) \}}{\mu_p (\mu_h + \alpha_h)(\mu_h + \delta_h + \varphi_h + \theta_h) \{ (\mu_h + \alpha_h)(\mu_h + \rho_h + \gamma_h) - \rho_h \phi \}}.$$

The number of infected individuals are generated by the infected individuals which is the product of the infected rate $\beta_h(S^* + \varepsilon_h V^*)$, while the number of infected individuals generated by the pathogen is the product of the infected rate $\beta_p(S^* + \varepsilon_h V^*)$. The probability that an exposed

survives the exposed class and moves to infected class is $\frac{\alpha_h}{(\mu_h + \alpha_h)}$, the average duration in P is

$\frac{1}{\mu_p}$ and the probability that an individual survives the infected class and moves to recovered class

is $\frac{\theta_h}{(\mu_h + \delta_h + \varphi_h + \theta_h)}$, while is the contribution of I to P , is θ_h .

Lemma 1: The measles system (1) exhibits a locally asymptotically stable disease-free equilibrium E^* , when $R_0 < 1$ and an unstable equilibrium whenever $R_0 > 1$.

Proof

The measles system's Jacobian matrix at DFE is provided by

$$J(E^*) = \begin{bmatrix} -(\mu_h + \phi) & 0 & -\beta_h S^* & 0 & \rho_h & -\beta_p S^* \\ 0 & -(\mu_h + \alpha_h) & (S^* + \varepsilon_h V^*)\beta_h & 0 & 0 & (S^* + \varepsilon_h V^*)\beta_p \\ 0 & \alpha_h & -(\mu_h + \delta_h + \varphi_h + \theta_h) & 0 & 0 & 0 \\ 0 & 0 & \varphi_h & -\mu_h & \gamma_h & 0 \\ \phi & 0 & -\beta_h \varepsilon_h V^* & 0 & -(\mu_h + \rho_h + \gamma_h) & -\beta_p \varepsilon_h V^* \\ 0 & 0 & \theta_h & 0 & 0 & -\mu_p \end{bmatrix}$$

Clearly we observed that $-\mu_h$ is one of the eigenvalue of the matrix, while the remaining are obtained from the polynomial

$$P(\varpi) = (\varpi^2 + (m_2 + m_4)\varpi + m_1 m_4 - \rho_h \phi) \begin{pmatrix} (\alpha_h + m_2)\varpi^2 + (m_2 m_3 (1 - R_1) + \alpha_h (m_2 + 1))\varpi \\ + m_2 m_3 (1 - R_0) \end{pmatrix} \text{ as,}$$

$$\varpi = \frac{1}{2} \left(-(m_1 + m_4) \pm \sqrt{(m_1 - m_4)^2 + 4\rho_h \phi} \right)$$

$$\varpi = \frac{- (m_2 m_3 (1 - R_1) + \alpha_h (m_2 + 1)) \pm \sqrt{(m_2 m_3 (1 - R_1) + \alpha_h (m_2 + 1))^2 - 4m_2 m_3 (\alpha_h + m_2) (1 - R_0)}}{2(\alpha_h + m_2)}$$

where,

$$m_1 = (\mu_h + \phi), m_2 = (\mu_h + \alpha_h), m_3 = (\mu_h + \delta_h + \varphi_h + \theta_h), m_4 = (\mu_h + \rho_h + \gamma_h).$$

Hence the DFE is locally asymptotically stable when, $R_0 < 1$.

Theorem 3: The disease-free equilibrium, E^* , of the measles model is globally asymptotically stable within the feasible interval if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof.

Consider the Lyapunov function denoted by

$$L = \frac{\alpha(\beta_h \mu_p + \beta_p \theta_h)}{\mu_p m_1 m_2} E + \frac{(\beta_h \mu_p + \beta_p \theta_h)}{\mu_p m_2} I + \frac{\beta_p \theta_h}{\mu_p} P.$$

whose derivative is

$$\begin{aligned} \dot{L} &= \frac{\alpha(\beta_h \mu_p + \beta_p \theta_h)}{\mu_p m_1 m_2} \dot{E} + \frac{(\beta_h \mu_p + \beta_p \theta_h)}{\mu_p m_2} \dot{I} + \frac{\beta_p \theta_h}{\mu_p} \dot{P} \\ \dot{L} &= \frac{\alpha(\beta_h \mu_p + \beta_p \theta_h)}{\mu_p m_1 m_2} ((S + \varepsilon_h V) \lambda - m_1 E) + \frac{(\beta_h \mu_p + \beta_p \theta_h)}{\mu_p m_2} (\alpha_h E - m_2 I) + \frac{\beta_p \theta_h}{\mu_p} (\theta_h I - \mu_p P) \\ \dot{L} &= \frac{\alpha(\beta_h \mu_p + \beta_p \theta_h)(S + \varepsilon_h V)(\beta_h I + \beta_p P)}{\mu_p m_1 m_2} - \frac{(\beta_h \mu_p + \beta_p \theta_h) I}{\mu_p} + \frac{\beta_p \theta_h}{\mu_p} (\theta_h I - \mu_p P) \\ \dot{L} &= (\beta_h I + \beta_p P) R_0 - \beta_h I - \beta_p P \\ \dot{L} &= (\beta_h I + \beta_p P)(R_0 - 1). \end{aligned}$$

Hence $\dot{L} \leq 0$ if and only if $R_0 < 1$ and $\dot{L} = 0$ if and only if $P = I = 0$. Given that every parameter is non-negative, it can be inferred that L is a Lyapunov function on Ω . Moreover, the singleton set (E^*) is the biggest compact invariant set in

$\{(S^*(t), E^*(t), I^*(t), R^*(t), V^*(t), P^*(t)) \in \Omega : \dot{L} = 0\}$. Consequently, every solution to the model with a starting condition in Ω , approaches E^* as $t \rightarrow \infty$ whenever $R_0 < 1$ so that E^* is GAS in Ω if $R_0 < 1$, according to LaSalle's Invariant Principles (LaSalle, 1968).

Existence of the Endemic Equilibrium Point EEP and Global Stability

Let $E^{**} = \{S^{**}, E^{**}, I^{**}, R^{**}, V^{**}, P^{**}\}$ be the arbitrary endemic equilibrium point of the model 1, and let $\lambda^{**} = \beta_h I^{**} + \beta_p P^{**}$ be the force of infection at steady states, solving the equations in system 1 at steady states yield,

$$\begin{aligned} S^{**} &= \frac{(1-q)\Pi(\mu_h + \varepsilon_h \lambda + \rho_h + \gamma_h) + \rho_h q \Pi}{(\mu_h + \lambda + \phi)(\mu_h + \varepsilon_h \lambda + \rho_h + \gamma_h) - \rho_h \phi}, \quad V^{**} = \frac{q \Pi (\mu_h + \lambda + \phi) + (1-q) \Pi \phi}{(\mu_h + \lambda + \phi)(\mu_h + \varepsilon_h \lambda + \rho_h + \gamma_h) - \rho_h \phi}, \\ E^{**} &= \lambda \left\{ \frac{(1-q)\Pi((\mu_h + \varepsilon_h \lambda + \rho_h + \gamma_h)) + q \Pi(\rho_h + \varepsilon_h((\mu_h + \lambda + \phi)))}{\mu_p(\mu_h + \alpha_h)(\mu_h + \delta_h + \varphi_h + \theta_h) \{(\mu_h + \lambda + \phi)(\mu_h + \varepsilon_h \lambda + \rho_h + \gamma_h) - \rho_h \phi\}} \right\}, \\ I^{**} &= \lambda \alpha_h \left\{ \frac{(1-q)\Pi((\mu_h + \varepsilon_h \lambda + \rho_h + \gamma_h)) + q \Pi(\rho_h + \varepsilon_h((\mu_h + \lambda + \phi)))}{\mu_p(\mu_h + \alpha_h)(\mu_h + \delta_h + \varphi_h + \theta_h) \{(\mu_h + \lambda + \phi)(\mu_h + \varepsilon_h \lambda + \rho_h + \gamma_h) - \rho_h \phi\}} \right\}, \end{aligned}$$

$$P^{**} = \lambda \alpha_h \theta_h \left\{ \frac{(1-q)\Pi((\mu_h + \varepsilon_h \lambda + \rho_h + \gamma_h)) + q\Pi(\rho_h + \varepsilon_h((\mu_h + \lambda + \phi)))}{\mu_p(\mu_h + \alpha_h)(\mu_h + \delta_h + \varphi_h + \theta_h)\{(\mu_h + \lambda + \phi)(\mu_h + \varepsilon_h \lambda + \rho_h + \gamma_h) - \rho_h \phi\}} \right\},$$

$$R^{**} = \frac{\gamma_h V^{**} + \varphi_h I^{**}}{\mu_h}$$

Evaluating $\lambda^{**} = \beta_h I^{**} + \beta_p P^{**}$, we obtained the quadratic equation (in terms λ^{**}) as

$$\varepsilon_h (\lambda^{**})^2 + \Upsilon_1 \lambda^{**} + \Upsilon_2 = 0.$$

where,

$$\Upsilon_1 = 1 - \left\{ \frac{(\beta_h \alpha \mu_p + \beta_h \alpha_h \theta_h)((1-q)\Pi \varepsilon_h + \Pi q \varepsilon_h)}{\mu_p(\mu_h + \alpha_h)(\mu_h + \delta_h + \varphi_h + \theta_h)(\mu_h + \alpha_h + \mu_h + \phi)} \right\}.$$

$$\Upsilon_2 = \{ \mu_h \gamma_h + (\mu_h + \phi)(\mu_h + \gamma_h) \} (1 - R_0).$$

The measles model (1) has a unique endemic equilibrium since $\Upsilon_2 < 0$, when $R_0 > 1$, by Descartes rule of sign.

Theorem 4: The measles model (1) endemic equilibrium, E^{**} is globally asymptotically stable within the feasible interval if $R_0 > 1$

Proof

Consider the function

$$M = \left(S - S^{**} - S \ln \frac{S^{**}}{S} \right) + \left(V - V^{**} - V^{**} \ln \frac{V^{**}}{V} \right) + \left(E - E^{**} - E^{**} \ln \frac{E^{**}}{E} \right) + \frac{(\varepsilon_h V^{**} + S^{**})(\beta_h I^{**} + \beta_p P^{**})}{\alpha_h E^{**}} \left(I - I^{**} - I^{**} \ln \frac{I^{**}}{I} \right) + \frac{(\varepsilon_h V^{**} + S^{**})(\beta_h I^{**} + \beta_p P^{**})}{\theta_h I^{**}} \left(P - P^{**} - P^{**} \ln \frac{P^{**}}{P} \right).$$

whose derivative with respect to t, is

$$M' = \left(1 - \frac{S^{**}}{S} \right) \dot{S} + \left(1 - \frac{V^{**}}{V} \right) \dot{V} + \left(1 - \frac{E^{**}}{E} \right) \dot{E} + \frac{(\varepsilon_h V^{**} + S^{**})(\beta_h I^{**} + \beta_p P^{**})}{\alpha_h E^{**}} \left(1 - \frac{I^{**}}{I} \right) \dot{I} + \frac{(\varepsilon_h V^{**} + S^{**})(\beta_h I^{**} + \beta_p P^{**})}{\theta_h I^{**}} \left(1 - \frac{P^{**}}{P} \right) \dot{P}.$$

$$M' = \left(1 - \frac{S^{**}}{S} \right) \left((1-q)\Pi + \rho_h V - (\mu_h + \lambda + \phi)S \right) + \left(1 - \frac{V^{**}}{V} \right) \left(q\Pi + \phi S - (\mu_h + \varepsilon_h \lambda + \rho_h + \gamma_h)V \right) + \left(1 - \frac{E^{**}}{E} \right) \left((S + \varepsilon_h V)\lambda - (\mu_h + \alpha_h)E \right) + \frac{(\varepsilon_h V^{**} + S^{**})(\beta_h I^{**} + \beta_p P^{**})}{\alpha_h E^{**}} \left(1 - \frac{I^{**}}{I} \right) + \frac{(\varepsilon_h V^{**} + S^{**})(\beta_h I^{**} + \beta_p P^{**})}{\theta_h I^{**}} \left(1 - \frac{P^{**}}{P} \right) (\theta_h I - \mu_p P).$$

$$(1-q)\Pi = (\mu_h + \beta_h I^{**} + \beta_p P^{**} + \phi)S^{**} - \rho_h V^{**}, \quad q\Pi = (\mu_h + \varepsilon_h \lambda + \rho_h + \gamma_h)V^{**} - \phi S^{**}. \quad (6)$$

Substituting the (6) in (5) and further simplification

gives

$$\begin{aligned} \dot{M} \equiv & \mu S^{**} \left(2 - \frac{S}{S^{**}} - \frac{S^{**}}{S} \right) + \phi S^{**} \left(2 - \frac{S}{S^{**}} - \frac{S^{**}}{S} \right) + \beta_h I^{**} S^{**} \left(3 - \frac{S^{**}}{S} - \frac{ISE^{**}}{I^{**} S^{**} E} - \frac{EI^{**}}{IE^{**}} \right) \\ & + (\phi S + \rho_h V) \left(1 - \frac{S^{**}}{S} \right) \left(1 - \frac{V^{**}}{V} \right) + \left(1 - \frac{P^{**}}{P} \right) \left(1 - \frac{I^{**} P}{IP^{**}} \right) + (\mu_h + \rho_h + \gamma_h) \left(2 - \frac{V}{V^{**}} - \frac{V^{**}}{V} \right) \\ & + \varepsilon_h \beta_p I^{**} V^{**} \left(3 - \frac{V^{**}}{V} - \frac{IVE^{**}}{I^{**} V^{**} E} - \frac{EI^{**}}{IE^{**}} \right) + \beta_h I^{**} S^{**} \left(4 - \frac{S^{**}}{S} - \frac{PSE^{**}}{P^{**} S^{**} E} - \frac{EI^{**}}{IE^{**}} - \frac{P^{**} I}{I^{**} P} \right) \\ & + \varepsilon_h \beta_p I^{**} V^{**} \left(4 - \frac{V^{**}}{V} - \frac{PVE^{**}}{P^{**} V^{**} E} - \frac{EI^{**}}{IE^{**}} - \frac{P^{**} I}{I^{**} P} \right) - \beta_h I^{**} S^{**} \frac{P}{P^{**}}. \end{aligned}$$

$$\begin{aligned} \dot{M} \equiv & \mu S^{**} \left(2 - \frac{S}{S^{**}} - \frac{S^{**}}{S} \right) + \phi S^{**} \left(2 - \frac{S}{S^{**}} - \frac{S^{**}}{S} \right) + \beta_h I^{**} S^{**} \left(3 - \frac{S^{**}}{S} - \frac{ISE^{**}}{I^{**} S^{**} E} - \frac{EI^{**}}{IE^{**}} \right) \\ & + (\phi S + \rho_h V) \left(1 - \frac{S^{**}}{S} \right) \left(1 - \frac{V^{**}}{V} \right) + \left(1 - \frac{P^{**}}{P} \right) \left(1 - \frac{I^{**} P}{IP^{**}} \right) + (\mu_h + \rho_h + \gamma_h) \left(2 - \frac{V}{V^{**}} - \frac{V^{**}}{V} \right) \\ & + \varepsilon_h \beta_p I^{**} V^{**} \left(3 - \frac{V^{**}}{V} - \frac{IVE^{**}}{I^{**} V^{**} E} - \frac{EI^{**}}{IE^{**}} \right) + \beta_h I^{**} S^{**} \left(4 - \frac{S^{**}}{S} - \frac{PSE^{**}}{P^{**} S^{**} E} - \frac{EI^{**}}{IE^{**}} - \frac{P^{**} I}{I^{**} P} \right) \\ & + \varepsilon_h \beta_p I^{**} V^{**} \left(4 - \frac{V^{**}}{V} - \frac{PVE^{**}}{P^{**} V^{**} E} - \frac{EI^{**}}{IE^{**}} - \frac{P^{**} I}{I^{**} P} \right). \end{aligned}$$

Since the arithmetic mean exceeds the geometric mean the following inequalities hold;

$$\begin{aligned} 2 - \frac{S}{S^{**}} - \frac{S^{**}}{S} \leq 0, \quad 3 - \frac{S^{**}}{S} - \frac{ISE^{**}}{I^{**} S^{**} E} - \frac{EI^{**}}{IE^{**}} \leq 0, \quad 3 - \frac{V^{**}}{V} - \frac{IVE^{**}}{I^{**} V^{**} E} - \frac{EI^{**}}{IE^{**}} \leq 0, \\ 4 - \frac{S^{**}}{S} - \frac{PSE^{**}}{P^{**} S^{**} E} - \frac{EI^{**}}{IE^{**}} - \frac{P^{**} I}{I^{**} P} \leq 0, \quad 4 - \frac{V^{**}}{V} - \frac{PVE^{**}}{P^{**} V^{**} E} - \frac{EI^{**}}{IE^{**}} - \frac{P^{**} I}{I^{**} P} \leq 0, \quad 2 - \frac{V}{V^{**}} - \frac{V^{**}}{V} \leq 0. \end{aligned}$$

Furthermore, since all the model are non-negative, thus $\dot{M} \leq 0$, and $\frac{I^{**} P}{IP^{**}} \geq 1$ if $S^{**} = S$,

$E^{**} = E$, $V^{**} = V$, $I^{**} = I$, $R^{**} = R$, $P^{**} = P$ then the largest compact invariant set in Ω such that $\dot{M} \leq 0$ is the singleton set (E^{**}) then by LaSalle Invariant Principle (LaSalle, 1968) it implies E^{**} , is globally asymptotically stable (GAS) in the interior of Ω .

Sensitivity Analysis

Sensitivity analysis is a technique for figuring out how certain parameters affect the spread of illness. Because of their considerable influence, it aids in identifying areas that

require intervention efforts. Sensitivity indices evaluate the proportionate change in a variable that results from a change in a parameter. Raising one parameter $(\Pi, \beta_h, \beta_p, \theta_h, \rho_h, \alpha)$ is associated with a

greater chance of an epidemic (positive indexes), whereas increasing another parameter ($\mu_h, \mu_p, \gamma_h, \delta_h, \epsilon_h, \phi$) is associated with a lower burden of measles among human populations (negative indexes).

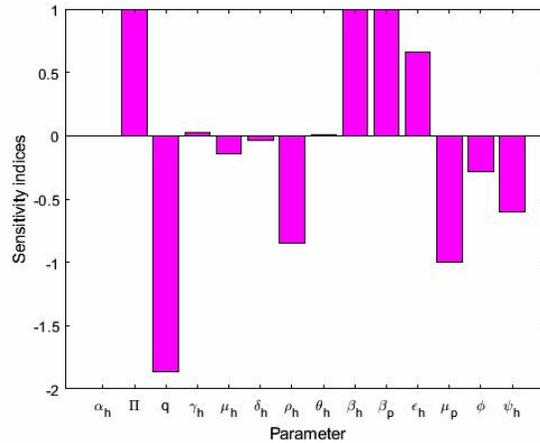


Figure 2: Sensitivity indices of the basic reproduction number in relation to the basic reproduction number's parameter value

Table 1: Parameter Values

Parameter	Value	Sources
Π	9875.8775/day	James Peter <i>et al</i> , (2022)
β_h	0.00000000142/day	James Peter <i>et al</i> , (2022)
β_p	0.0000000016/day	Assumed
μ_h	0.000045/day	James Peter <i>et al</i> , (2022)
δ_h	0.03372/day	James Peter <i>et al</i> , (2022)
μ_p	0.071/day	Alemneh & Belay, 2023
ϵ_h	0.2	Raimundo <i>et al</i> , (2007)
ρ_h	0.0004694/day	Abboubakar <i>et al</i> , (2022)
θ_h	0.042/day	Alemneh & Belay, (2023)
α_h	0.5/day	James Peter <i>et al</i> , (2022)
ϕ	0.125/day	Sowole <i>et al</i> , (2023)
φ_h	0.07143/day	Nwankwo, (2021)
γ_h	0.08333/day	Nwankwo, (2021)
q	0.85 (Dimensionless)	Assumed



DISCUSSION

Measles mathematical modeling simulation offers important insights into the dynamic behavior of the illness within a community. The disease-free equilibrium point becomes globally asymptotically stable when R_0 is less than 1, indicating that measles transmission will eventually cease over time. The simulation shows a rapid decline in infected individuals towards zero, ultimately leading to the eradication of measles from the population due to high vaccination coverage (as shown in figure 3). Conversely, the endemic equilibrium point is globally stable when R_0 greater than 1 is, indicating measles transmission persists within the population (figure 4). This occurs when vaccination coverage is insufficient to prevent sustained transmission. When R_0 is less than 1, the number of infected individuals declines, accompanied by an increase in vaccination coverage. When $R_0 < 1$ it means that, on average, each sick person is infecting fewer people than one other, which slows down the disease's transmission. When $\phi = 195$ in this case, that means $R_0 = 0.4565$ which is less than 1. This shows that the disease cannot persist in the population and that, with a 19.5% vaccination rate, the disease's spread is

effectively contained. Going on to $R_0 = 1.3870$, this indicates that, on average, each sick individual is infecting roughly 1.387 more individuals. R_0 stays greater than 1 when $\phi = 0.0125$, signifying a vaccination rate of 1.25%, is observed. The disease can persist in this situation, although it can do so more slowly than it does when R_0 is higher. The 1.25% vaccination rate may not be enough to completely stop the spread, but it does help to lower the transmission rate. Lastly, when $R_0 = 4.2753$, it suggests that the illness spreads quickly and that an infected person often infects more than four other people. Even with a vaccination rate of $\phi = 0.0057$ or 0.57%, (R_0) stays well above 1, suggesting that the disease may still spread widely. In such a case, reducing the reproductive population to a reasonable level may require further steps or a greater vaccination rate as shown in figure 6. Global stability of the measles transmission model (1) is essential for sustainable control measures, predictability, intervention methods, and global health security in the context of epidemiological control. Global health security is eventually aided by this stability, which forecasts vaccine affects, optimizes intervention measures, and lowers outbreak risks.

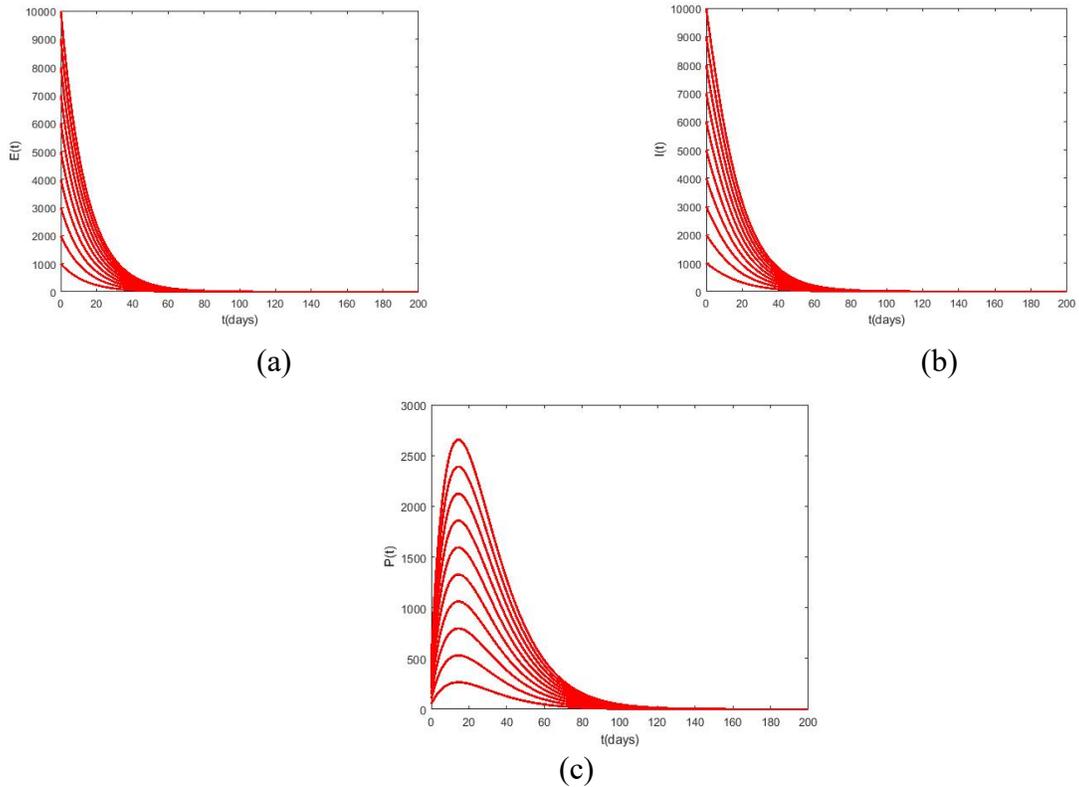


Figure 3: Convergence of solution to the disease free equilibrium point of model system (1), for the exposed (a), infected (b) and pathogen (c) populations with different initial values. Parameter values used are as provided in Table 1. So that $R_0 = 0.0722 < 1$

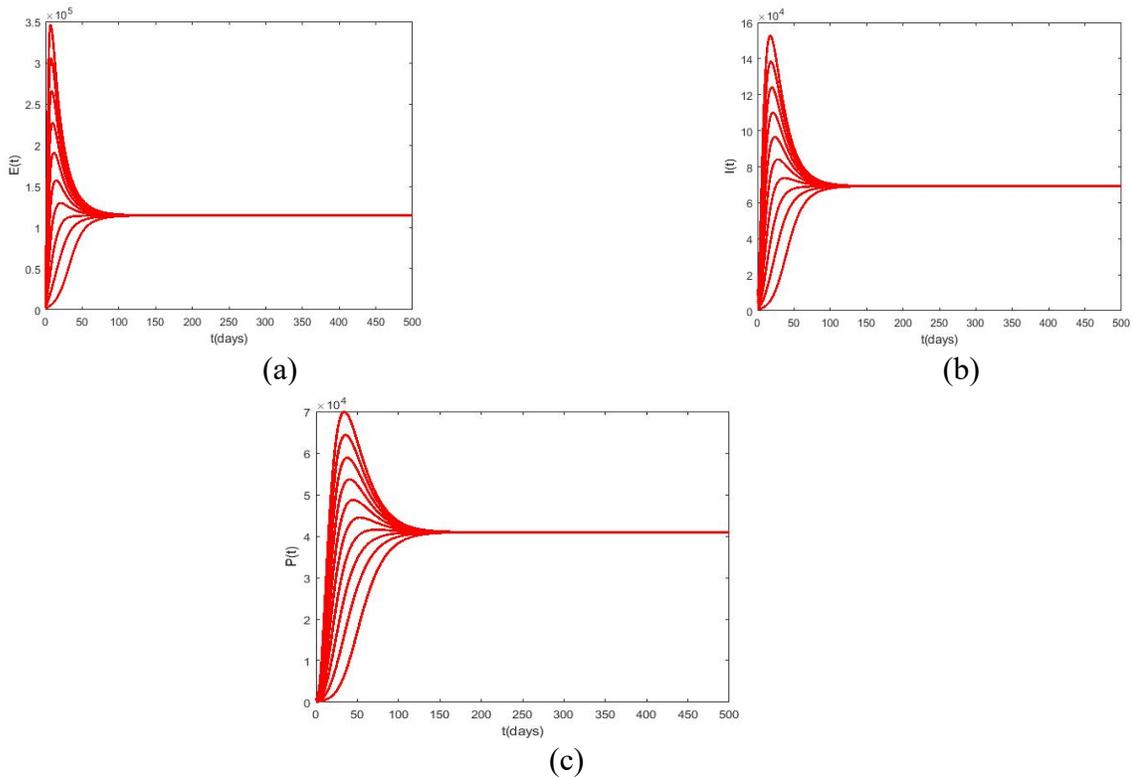


Figure 4: Convergence of solution to the endemic equilibrium points of model system (1), for the exposed (a), infected (b) and pathogen (c) population with different initial values. Parameter values used are as provided in Table 1, except $\beta_p = 0.0000016$ and $\beta_h = 0.00000142$ So that $R_0 = 721.51 > 1$

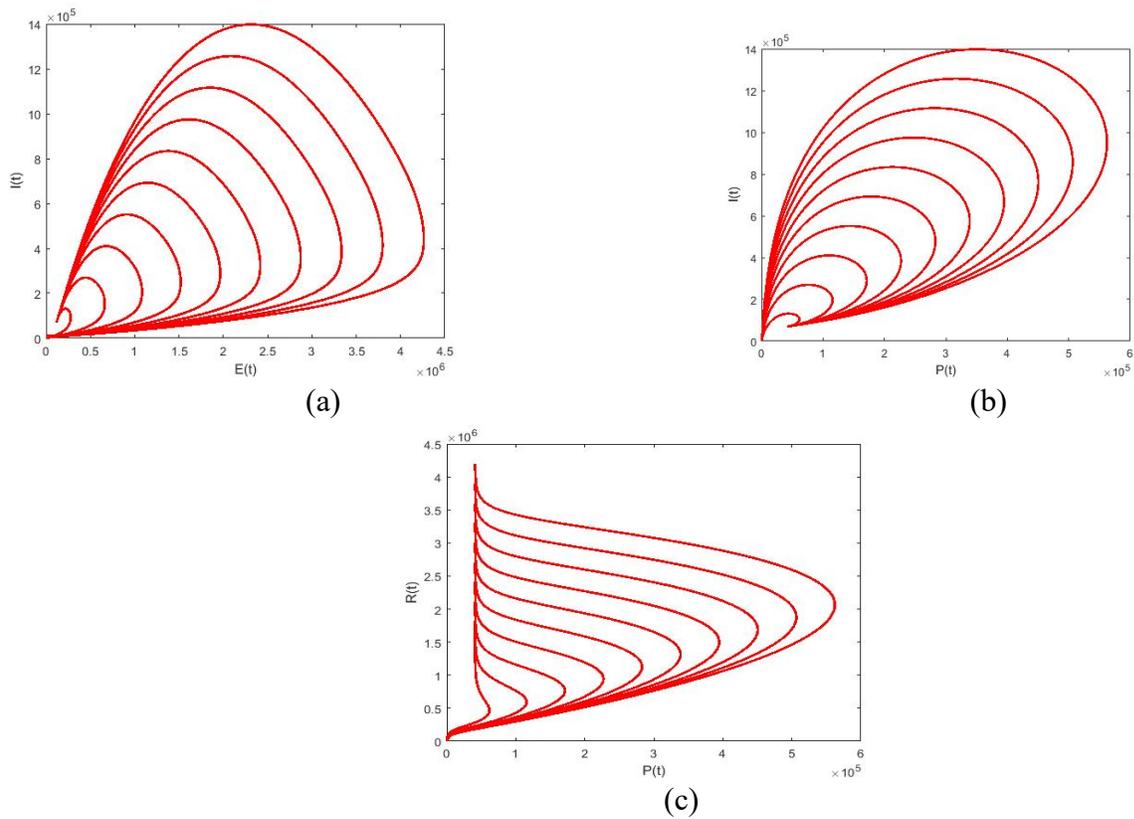


Figure 5: A numerical simulation depicting the measles model's phase portrait

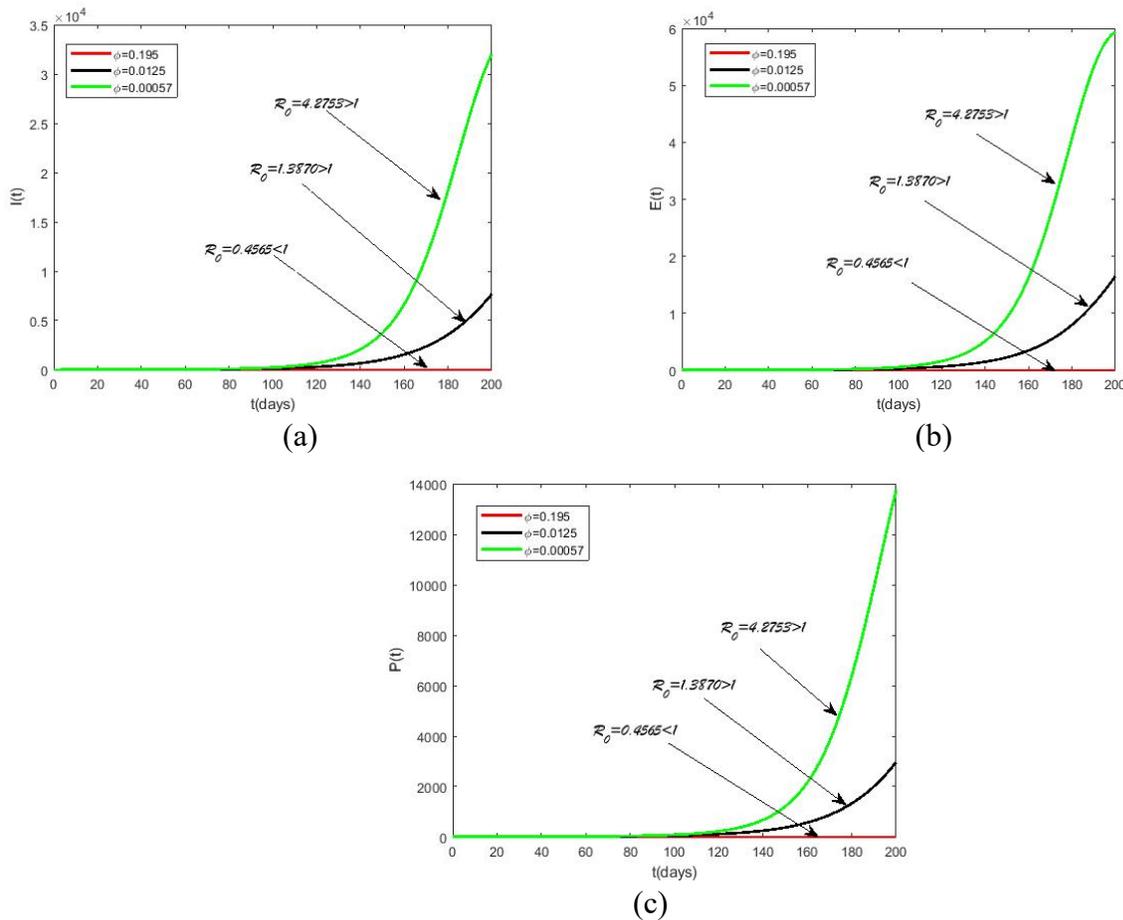


Figure 6: A numerical simulation demonstrating the impact of vaccination on $E(t)$, $I(t)$, $P(t)$.

CONCLUSION

In conclusion, our global stability analysis of a measles model that includes exposed and vaccination classes sheds light on the dynamics of the disease's spread and the effectiveness of vaccination programs. We have determined the main variables impacting the long-term patterns of measles within a community by means of an extensive mathematical investigation. Our results highlight the critical role that both high vaccination rates and vaccine effectiveness play in preventing the spread of the measles. We have shown that maintaining and attaining measles elimination requires consistent immunization campaigns. The sensitivity analysis we conducted also emphasizes how

crucial it is to comprehend how different characteristics influence the dynamics of measles transmission in order to help policymakers develop effective intervention strategies. Overall, this study adds to the expanding corpus of research that aims to guide public health strategies and policies for the control and elimination of measles. Through our analysis, we have been able to establish a framework for the optimization of vaccination campaigns and the advancement of worldwide initiatives aimed at eliminating or persistently containing measles. But in order to handle new issues and guarantee the long-term viability of measles elimination programs, ongoing research, vaccine outreach, and surveillance are crucial. This study



contributes to the ongoing efforts to optimize vaccination strategies and achieve sustained measles elimination on a global scale.

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