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Modelling and Analysing the Spread of Infectious Diseases: The Role of Vaccination and Public Awareness

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ABSTRACT: This paper presents and evaluates a nonlinear mathematical model designed to assess the impact of vaccination programs on the transmission dynamics of infectious diseases. The model categorizes the population into three compartments: susceptible individuals (S), infected individuals (I), and vaccinated individuals (V_c). Employing both analytical approaches and numerical simulations, the study demonstrates that effective media campaigns promoting vaccination, coupled with an adequate vaccine supply, can substantially mitigate the spread of infections. The stability of the model is analyzed using the stability theory of differential equations, and numerical simulations are conducted to validate and reinforce the analytical results.

Key Words: Infectious disease, vaccination, awareness program.

Contents

1. Introduction

Understanding and analysing the spread of infectious diseases are crucial for evaluating and implementing effective control strategies within populations. Traditional models of disease transmission often focus on interactions between susceptible and infected individuals. However, additional factors, such as media influence, vaccination campaigns, and population movement, significantly affect the dynamics of infectious disease spread [3-9]. Vaccination reduces the likelihood of disease transmission between individuals, decreases the probability of outbreaks, and, when sufficient immunity levels are achieved within a population, leads to "community immunity" or "herd immunity." This phenomenon not only protects vaccinated individuals but also provides indirect protection to those more vulnerable to infection.
Mathematical models have become indispensable tools in epidemiology, offering valuable insights into the progression and control of infectious diseases [1, 15, 16]. These models enable the simulation of diverse scenarios, enhancing our understanding of disease dynamics and informing public health strategies. Over the years, numerous models have been developed to explore the transmission dynamics of infectious diseases, often incorporating key elements such as vaccination and public awareness as central to disease mitigation.

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For instance, models show that increasing vaccination coverage can lower the basic reproduction number (R_0) below one, reducing disease spread and potentially eliminating the pathogen. Public awareness also plays a vital role in improving vaccination rates and limiting disease transmission, which is why awareness campaigns are integral to many epidemiological models. These campaigns effectively promote vaccination by countering misinformation and fostering herd immunity. Models that integrate awareness dynamics demonstrate that well-informed populations are more likely to embrace vaccination, leading to improved disease control and, in some cases, eradication of infectious agents.
This study develops and analyses a Susceptible-Infective-Susceptible (SIS) model to investigate the impact of vaccination, driven by media campaigns, on the spread of an infectious disease within a dynamic population influenced by immigration. The structure of this paper is as follows:

- Section 1 provides an introduction and overview of the study.
- Section 2 describes the mathematical model of the problem.
- Sections 3, 4, and 5 focus on identifying the region of attraction, equilibrium points, and their stability analysis.
- Section 6 presents numerical analysis and interprets the findings.
- Section 7 concludes the study by summarizing the results and presenting the key takeaways.
  This research highlights the importance of integrating vaccination and public awareness into mathematical modelling to enhance the understanding and management of infectious disease dynamics in complex, dynamic populations.

2. Mathematical Model

In this study, we assume that the total population in the region at any time t is denoted by $N(t)$, with a constant influx of susceptible individuals through immigration, represented by A. The population is categorized into three groups: susceptible individuals(S), infected individuals (I), and vaccinated individuals (V_c). The spread of the disease is assumed to occur exclusively through direct contact between susceptible and infected individuals. Furthermore, the vaccinated population increases due to awareness campaigns conducted via social and electronic media, with the media influence at any time t indicated by M. As the media engages with the susceptible population, it encourages them to get vaccinated in order to prevent infection. Initially, the media's influence is minimal, but it grows as the infection rate increases. However, the effect of media on the susceptible population is limited and eventually reaches a saturation point due to constraints such as available resources. Media coverage typically doesn't remain focused on one issue for long; it shifts in response to social and political changes. Additionally, media outreach may not cover the entire population due to factors such as time restrictions, literacy barriers, and financial limitations, along with the resources needed to spread information. Therefore, both media influence and its impact on the susceptible population have an upper limit and cannot grow indefinitely, ultimately reaching a saturation point. To model this media influence on the susceptible population, we use a Holling type II functional response, as referenced in [12, 14]. Considering all these aspects, the model's dynamics are governed by the following system of nonlinear ordinary differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= A - \beta SI - \frac{\lambda SM}{1+\gamma M} + vI + \lambda_0 V_c - \delta S,\\
\frac{dI}{dt} &= \beta SI - vI - \alpha I - \delta I,\\
\frac{dV_c}{dt} &= \frac{\lambda SM}{1+\gamma M} - \delta V_c - \lambda_0 V_c,\\
\frac{dM}{dt} &= \mu(S+I) - \mu_0 M. \qquad (1)\\
\text{Where} \quad & S(0) > 0, \quad I(0) > 0, \quad V_c(0) \geq 0, \quad M(0) \geq 0.
\end{aligned}$$

In this model, β represents the rate at which susceptible individuals meet infected individuals, leading to potential disease transmission. The constant λ refers to the rate at which susceptible individuals leave the susceptible group and move into the vaccinated group. The constants v, α and δ correspond to the rate of recovery for infected individuals, the death rate due to the disease, and the natural death rate, respectively. The constant λ_0 indicates the rate at which vaccinated individuals lose their immunity and return to the susceptible group. Additionally, μ measures the speed at which awareness campaigns promoting vaccination are being rolled out, while μ_0 accounts for the decline in these programs, whether due to ineffectiveness or other societal factors. Finally, γ is the half-saturation constant, which helps regulate how certain model dynamics evolve in response to changes in disease progression or intervention strategies.

Using $S+I+V_c = N$, the system of above differential equations can be reduced to the following form:

$$\begin{aligned}
\frac{dI}{dt} &= \beta\left(N - I - V_c\right) I - (v+\alpha+\delta)I, \\
\frac{dV_c}{dt} &= \lambda\frac{(N-I-V_c)}{1+\gamma M}M - \delta V_c - \lambda_0 V_c, \\
\frac{dN}{dt} &= A - \delta N - \alpha I, \\
\frac{dM}{dt} &= \mu\left(N - V_c\right) - \mu_0 M.
\end{aligned} \tag{2}$$

3. Region of Attraction

Lemma 3.1 *The region of attraction for the system (2) is given by the following set:* $\Omega = \{(I, V_c, N, M) : 0 < I \leq I_{\max}, 0 \leq V_c \leq V_{c_{\max}}, 0 < N \leq N_{\max}, 0 < M \leq M_{\max}\}$, *where* $I_{\max} = \frac{\beta A - \delta(v+\alpha+\delta)}{\delta\beta}, V_{c_{max}} = \frac{\lambda\mu A^2}{\delta^2\mu_0(\delta+\lambda_0)}, N_{\max} = \frac{A}{\delta}$ *and* $M_{\max} = \frac{\mu A}{\delta\mu_0}$.

Proof: From the third equation of the system (2), we get

$$\begin{aligned}
\frac{dN}{dt} &= A - \delta N - \alpha I, \\
\frac{dN}{dt} &\leq A - \delta N.
\end{aligned}$$

Which implies that

$$\lim_{t\to\infty} \sup N(t) \leq \frac{A}{\delta} = N_{\max}, (\text{ say })$$

From the last equation of the system (2), we have

$$\begin{aligned}
\frac{dM}{dt} &= \mu N - \mu V_c - \mu_0 M, \\
\frac{dM}{dt} &\leq \mu N_{\max} - \mu_0 M.
\end{aligned}$$

Which implies that

$$\lim_{t\to\infty} \sup M(t) \leq \frac{\mu A}{\mu_0\delta} = M_{\max}, (\text{ say })$$

From the first equation of the system (2), we find the value of $I_{\max}$,

$$\begin{aligned}
\frac{dI}{dt} &= \beta N I - \beta I^2 - \beta V_c I - (v+\alpha+\delta)I, \\
\frac{dI}{dt} &\leq \beta N_{\max} I - \beta I^2 - (v+\alpha+\delta)I \\
\frac{dI}{dt} &\leq \left(\beta N_{\max} - (v+\alpha+\delta)\right) I - \beta I^2.
\end{aligned}$$

Which implies that

$$\lim_{t\to\infty} \sup I(t) \leq \frac{\beta A - \delta(v+\alpha+\delta)}{\delta\beta} = I_{\max}, (\text{ say })$$

From the second equation of the system (2),

$$\frac{dV_c}{dt} \leq \lambda \frac{N_{\max} M_{\max}}{1+\gamma M} - (\delta + \lambda_0) V_c.$$

Which implies that

$$\lim_{t\to\infty} \sup V_c(t) \leq \frac{\lambda\mu A^2}{\delta^2 \mu_0 (\delta+\lambda_0)} = V_{c_{\max}} \quad \text{(say)}.$$

Hence prove the Lemma (3.1).

4. Equilibrium Points

The system (2) has two non-negative equilibrium points, which are:
i) Disease-Free Equilibrium $\boldsymbol{E}_1 \left(0, \bar{V}_c, \bar{N}, \bar{M}\right)$.
ii) Endemic Equilibrium $\boldsymbol{E_2}\left(I^*, V_c^*, N^*, M^*\right)$

Existence of $\boldsymbol{E}_1 \left(\mathbf{0}, \overline{\boldsymbol{V}_{\boldsymbol{c}}}\overline{\boldsymbol{N}}, \overline{\boldsymbol{M}}\right)$

This equilibrium point is given by

$$\bar{V}_c = \frac{\lambda\mu_0}{\mu(\delta+\lambda_0)} \frac{\bar{M}^2}{(1+\gamma\bar{M})}, \bar{N} = \frac{A}{\delta}, \text{ and } \bar{M} \text{ is given by the equation}$$
$$\left(\frac{\lambda}{(\delta+\lambda_0)} + \gamma\right)\bar{M}^2 + \left(1 - \frac{\mu\gamma A}{\mu_0\delta}\right)\bar{M} - \frac{\mu}{\mu_0\delta}A = 0. \tag{4.1}$$

The equation (4.1) has always one positive root. With the value of $\bar{M}$ from above equation we can find the values of $\bar{V}_c$ and $\overline{\mathrm{N}}$.

Existence of $\boldsymbol{E_2}\left(I^*, {V_c}^*, N^*, M^*\right)$

This equilibrium point is given by

$$\begin{aligned}
&\beta(N^* - I^* - V_c^*) - (v+\alpha+\delta) = 0,\\
&\lambda\frac{(N^* - I^* - V_c^*)}{1+\gamma M} M^* - \delta V_c^* - \lambda_0 V_c^* = 0,\\
&A - \delta N^* - \alpha I^* = 0,\\
&\mu(N^* - V_c^*) - \mu_0 M^* = 0.
\end{aligned} \tag{4.2}$$

From system of equations (4.2), we get
$V_c^* = \frac{(v+\alpha+\delta)\delta(R_0-1)-\beta(\alpha+\delta)I^*}{\delta\beta}, N^* = \frac{A-\alpha I^*}{\delta}, M^* = \frac{\mu}{\mu_0\beta}(v+\alpha+\delta+\beta I^*)$.
And I^* is given by following equation
$B_1 I^{*2} + B_2 I^* + B_3 = 0$,
where $B_1 = \beta(\alpha+\delta)\gamma k_1$,
$B_2 = \delta(v+\alpha+\delta)\gamma k_1 + \beta(\alpha+\delta) + \beta(\alpha+\delta)\gamma k_0 + \beta\delta k_2 k_1 - \beta A\gamma k_1$,
$B_3 = \delta(v+\alpha+\delta)\gamma k_0 + \delta(v+\alpha+\delta) - \beta A - \beta A\gamma k_0 + \beta\delta k_2 k_0$,
And k_0, k_1, k_2 are given by
$k_0 = \frac{\mu}{\mu_0\beta}(v+\alpha+\delta), k_1 = \frac{\mu}{\mu_0}, k_2 = \frac{\lambda(v+\alpha+\delta)}{\beta(\delta+\lambda_0)}$.
If $B_3 < 0$ then $\beta\delta k_2 k_0 < \delta(v+\alpha+\delta)(R_0-1)(1+\gamma k_0)$.
Let us define $R_0 = \frac{\beta A}{\delta(v+\alpha+\delta)}$ which is the basic reproduction number for system (1). Thus equilibrium point E_2 exist for $R_0 > 1$.

5. Stability Analysis

In this section, we discuss the local and global stability of the equilibrium points $E_1\left(0,\bar{V}_c,\bar{N},\bar{M}\right)$ and $\boldsymbol{E}_2\left(I^*,V_c^*,N^*,M^*\right)$. By calculating the Jacobian matrix at $E_1\left(0,\bar{V}_c,\bar{N},\bar{M}\right)$, we note that three eigenvalues are always negative and fourth one is negative if $\beta\bar{N} < \beta\bar{V}_c + v + \delta + \alpha$ and positive if $\beta\bar{N} > \beta\bar{V}_c + v + \delta + \alpha$.

Here, two theorems regarding the stability behavior of the equilibrium points.

Theorem 5.1 *The equilibrium point E_1 is stable for $\beta\bar{N} < \beta\bar{V}_c + v + \delta + \alpha$ and unstable for $\beta\bar{N} > \beta\bar{V}_c + v + \delta + \alpha$, and the endemic equilibrium E_2 is locally asymptotically stable if the following conditions are satisfied,*

$$\begin{aligned} a_{12}^2 &< 4a_{11}a_{22}, \\ a_{23}^2 &< 4a_{22}a_{33}, \\ a_{34}^2 &< 4a_{33}a_{44}, \end{aligned} \tag{5.1}$$

where

$$\begin{aligned} &a_{11} = \left(2\beta I^* - \beta N^* + \beta V_c^* + v + \alpha + \delta\right), \quad a_{22} = \left(\frac{\lambda M^*}{1+\gamma M^*} + \delta + \lambda_0\right), a_{33} = \frac{\beta\delta I^*}{\alpha}, \\ &a_{44} = \frac{\mu_0\lambda\left(N^* - I^* - V_c^*\right)}{\mu\left(1+\gamma M^*\right)^2}, \quad a_{12} = -\left(\beta I^* + \frac{\lambda M^*}{1+\gamma M^*}\right), a_{23} = \frac{\lambda M^*}{1+\gamma M^*}, a_{34} = \frac{\lambda\left(N^* - I^* - V_c^*\right)}{\left(1+\gamma M^*\right)^2}. \end{aligned} \tag{5.2}$$

Proof of this theorem is given Appendix A.

Theorem 5.2 *The endemic equilibrium E_2 is globally asymptotically stable in the region Ω if the following conditions are satisfied:*

$$\begin{aligned} &\left(\beta + \lambda\frac{\mu A}{\delta\mu_0}\right)^2 < 4\beta\left(\delta + \lambda_0\right), \\ &\left(\frac{\lambda M^*}{1+\gamma M^*}\right)^2 < 4\frac{\beta\delta}{\alpha}\left(\delta + \lambda_0\right), \\ &\frac{\lambda\left(N_{\max} - I^* - V_c^*\right)}{\mu\left(1+\gamma M^*\right)} < 4\frac{\beta\delta}{\alpha}. \end{aligned} \tag{5.3}$$

Proof of the above theorem is given in Appendix B.

6. Numerical Simulation

To provide a numerical solution for the mathematical model described by the system of equations (2), we choose following values of parameters,

$$\begin{aligned} &A = 250, \beta = 0.0005, \lambda = 0.0022, \gamma = 0.008, v = 0.6\lambda_0 = 0.2, \delta = 0.01, \mu_0 = 0.24 \\ &\alpha = 0.02, \mu = 0.005. \end{aligned} \tag{6.1}$$

The equilibrium values of the endemic state are as follows

$$I^* = 7585.6211, V_c^* = 983.1366, N^* = 9828.7577, M^* = 184.2837.$$

The characteristic equation associated with the Jacobian matrix of the endemic equilibrium is given by

$$x^4 + 4.41666x^3 + 1.91867x^2 + 0.25186x + 0.00582 = 0.$$

The eigenvalues are:

$$-3.94658, \quad -0.22046 + 0.04456i, -0.22046 - 0.04456i, \quad -0.02915.$$

Since all the eigenvalues possess negative real parts, the endemic equilibrium is locally asymptotically stable for the specified set of parameters.

The analysis of the figures provides valuable insights into the dynamics of the system under the influence of vaccination awareness programs and dissemination efforts. Figure 1 illustrates that the system achieves local stability, suggesting that small perturbations or deviations from equilibrium states will eventually return to a stable condition. This stability reflects the resilience of the system to minor changes within the population, such as fluctuations in the number of infected or susceptible individuals. Figure 2 demonstrates the global stability of the system, indicating that regardless of initial conditions, the system converges to a stable equilibrium. This result underscores the robustness of the proposed model, as it ensures that the long-term behaviour of the population remains predictable and manageable under the given parameters. In Figure 3, the infectious population $I(t)$ is shown to decrease over time as the rate of implementation of awareness programs, denoted by μ, increases. This trend highlights the crucial role of awareness initiatives in mitigating the spread of the disease. As more individuals are informed about the benefits of vaccination and preventive measures, the number of infections within the population gradually declines, demonstrating the effectiveness of awareness campaigns in controlling disease transmission. Figure 4 focuses on the vaccinated population $V_c(t)$, which increases over time as the awareness implementation rate μ rises. This positive correlation underscores the impact of media-driven awareness efforts in encouraging vaccine uptake. As public awareness grows, more susceptible individuals are motivated to get vaccinated, contributing to the overall reduction of the disease burden in the population. Figure 5 shows the effect of μ on the media awareness program. In Figure 6 and Figure 7 we can see that the increasing the value of γ, infected population increases and vaccinated population decreases. In Figure 8, the infected population is shown to decrease significantly as the dissemination rate of information, denoted by λ, increases. This observation highlights the importance of rapid and effective communication strategies in curbing the spread of infectious diseases. A higher dissemination rate ensures that critical information about vaccination and preventive measures reaches a larger audience in a timely manner, resulting in fewer infections. Finally, Figure 9 reveals that the vaccinated population continues to grow with increasing dissemination rate λ. This finding reinforces the idea that effective communication strategies are instrumental in promoting vaccination coverage. By enhancing the reach and frequency of awareness campaigns, more individuals are encouraged to take proactive steps in protecting themselves and others, further contributing to the establishment of herd immunity within the population.

Together, these figures demonstrate the critical impact of vaccination awareness programs and information dissemination strategies on the dynamics of infectious disease spread. They underscore the importance of implementing robust public health campaigns to promote vaccination, reduce infections, and achieve long-term disease control.

7. Conclusion

The study concludes that incorporating media-driven vaccination initiatives into the dynamics of infectious disease transmission can significantly alter the course of an epidemic. By analysing a non-linear

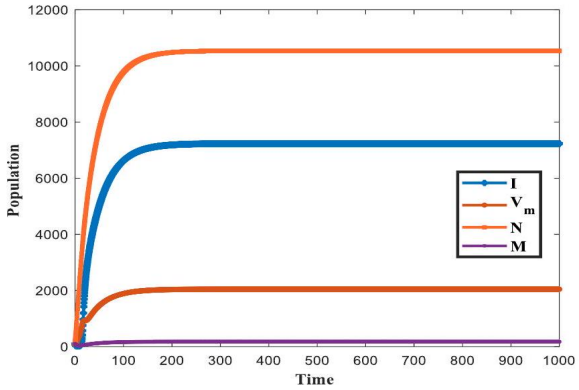

Figure 1: Variation of population with the time.

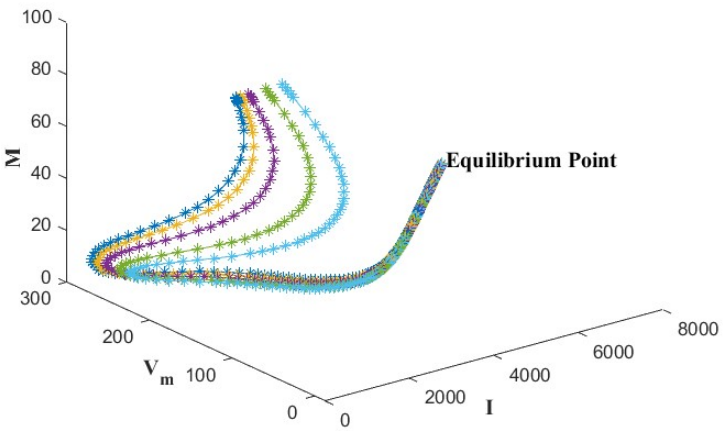

Figure 2: Global behavior of the system

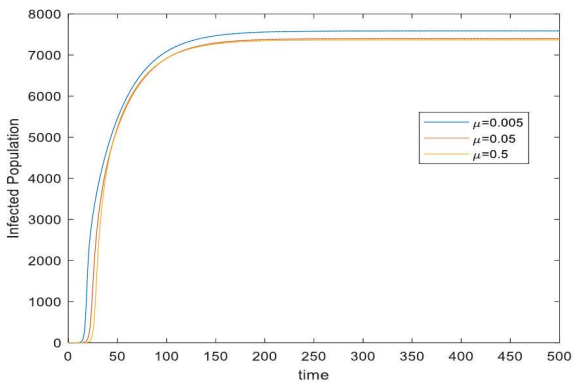

Figure 3: Variation in the infectious population over time for various values of μ.

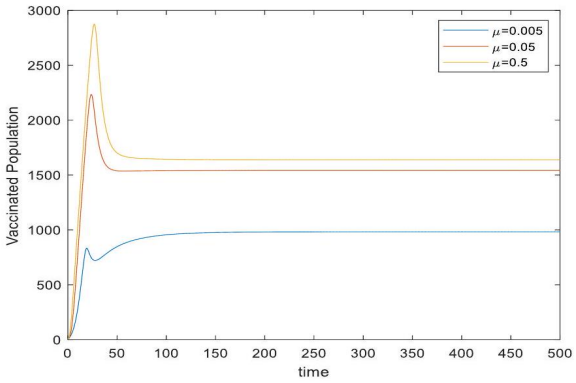

Figure 4: Changes in the vaccinated population over time for different values of μ.

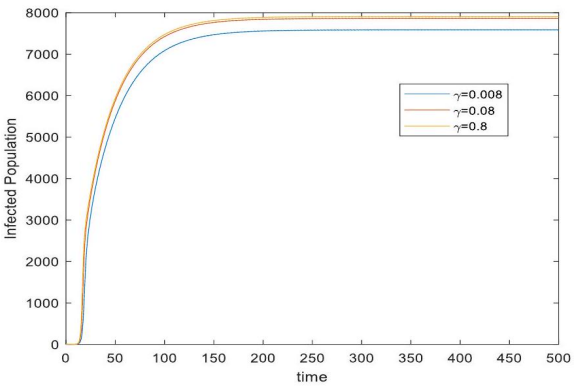

Figure 5: Changes in media awareness programs over time for various values of μ.

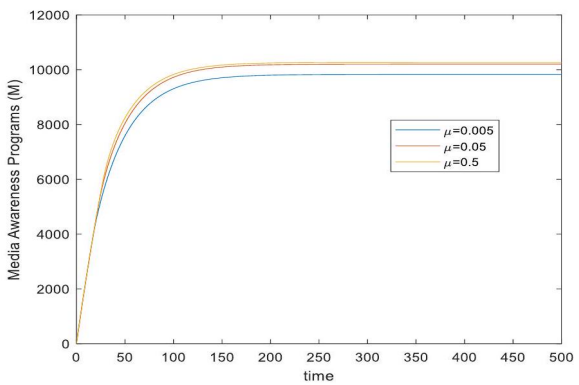

Figure 6: Changes in the infected population over time for different values of γ.

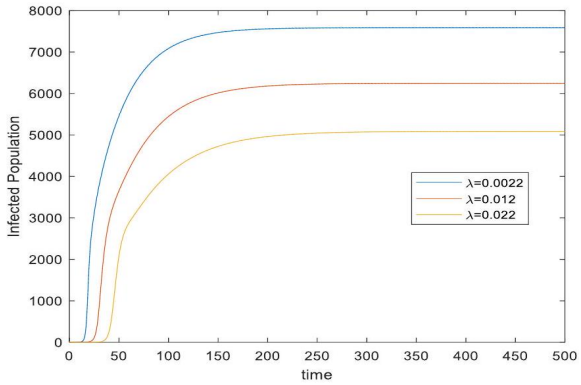

Figure 7: Changes in the vaccinated population over time for various values of γ.

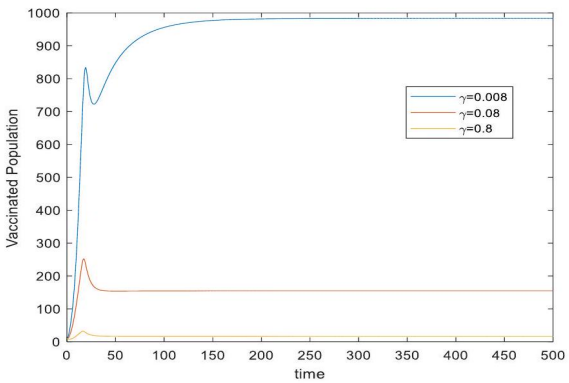

Figure 8: Changes in the infected population over time for different values of λ.

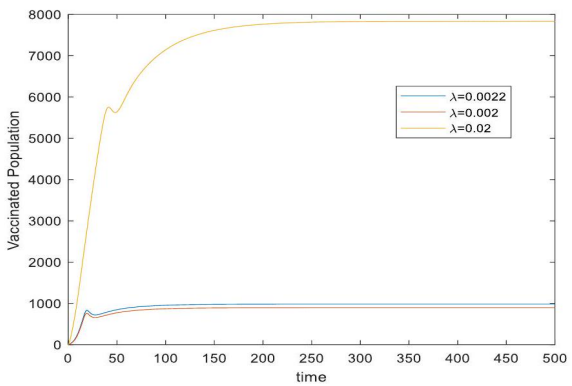

Figure 9: Changes in the vaccinated population over time for various values of λ.

SIS (Susceptible-Infective-Susceptible) model, it is evident that public awareness campaigns play a crucial role in influencing population behaviour. The model demonstrates that as awareness grows, driven by the interaction between susceptible and infected individuals, a portion of the susceptible population adopts preventative measures, effectively reducing their risk of infection. This behavioural shift, fuelled by vaccination programs, contributes to the stability of the endemic equilibrium under certain conditions. Specifically, the results suggest that both local and global stability can be achieved when the rate of vaccination uptake, influenced by media campaigns, surpasses the rate of disease transmission. The findings highlight the importance of sustained and well-targeted media efforts to promote vaccine adoption, particularly in populations experiencing demographic shifts due to immigration. Moreover, the research underscores that while vaccination campaigns are critical, their effectiveness is closely tied to the timely and adequate dissemination of information through media channels. These campaigns not only mitigate the spread of diseases but also foster a more informed population capable of making health-conscious decisions. By addressing the interplay between vaccination efforts, media influence, and population changes, this study provides valuable insights for public health policymakers aiming to curb the spread of infectious diseases in an increasingly globalized world.

8. Appendix A

Proof of Theorem 5.1

Consider the following function, which is positive definite:

$$U_1 = \frac{1}{2}\left(m_0 i^2 + m_1 v_c^2 + m_2 n^2 + m_3 m^2\right).$$

Here, m_i (where $i = 0, 1, 2, 3$) are positive constants. By differentiating the above equation with respect to t and applying the linearized system of model (2) corresponding to E_2

$$\frac{dU_1}{dt} = -m_0\left(2\beta I^* - \beta N^* + \beta V_c^* + v + \alpha + \delta\right) i^2 - m_1\left(\frac{\lambda M^*}{1+\gamma M^*} + \delta + \lambda_0\right) v_c^2 - m_2\delta n^2 - m_3\mu_0 m^2 - \left(m_0\beta I^* + m_1\frac{\lambda M^*}{1+\gamma M^*}\right) i v_c + \left(m_0\beta I^* - m_2\alpha\right) in + m_1\frac{\lambda M^*}{1+\gamma M^*} v_c n + \left(m_1\frac{\lambda\left(N^* - I^* - V_c^*\right)}{\left(1+\gamma M^*\right)^2} - m_3\mu\right) v_c m + m_3\mu nm,$$

we choose,

$$m_0 = 1, m_1 = 1, m_2 = \frac{\beta I^*}{\alpha}, m_3 = \frac{\lambda\left(N^* - I^* - V_c^*\right)}{\mu\left(1+\gamma M^*\right)^2},$$

we get,

$$\frac{dU_1}{dt} = -\left(2\beta I^* - \beta N^* + \beta V_c^* + v + \alpha + \delta\right) i^2 - \left(\frac{\lambda M^*}{1+\gamma M^*} + \delta + \lambda_0\right) v_c^2 - \frac{\beta\delta I^*}{\alpha} n^2 - \frac{\mu_0\lambda\left(N^* - I^* - V_c^*\right)}{\mu\left(1+\gamma M^*\right)^2} m^2 - \left(\beta I^* + \frac{\lambda M^*}{1+\gamma M^*}\right) i v_c + \frac{\lambda M^*}{1+\gamma M^*} v_c n + \frac{\lambda\left(N^* - I^* - V_c^*\right)}{\left(1+\gamma M^*\right)^2} nm,$$

$$\frac{dU_1}{dt} = -a_{11} i^2 - a_{22} v_c^2 - a_{33} n^2 - a_{44} m^2 + a_{12} i v_c + a_{23} v_c n + a_{34} nm,$$

Where $a_{11} = \left(2\beta I^* - \beta N^* + \beta V_c^* + v + \alpha + \delta\right), \quad a_{22} = \left(\frac{\lambda M^*}{1+\gamma M^*} + \delta + \lambda_0\right), \quad a_{33} = \frac{\beta\delta I^*}{\alpha},$

$$a_{44} = \frac{\mu_0\lambda\left(N^* - I^* - V_c^*\right)}{\mu\left(1+\gamma M^*\right)^2}, a_{12} = -\left(\beta I^* + \frac{\lambda M^*}{1+\gamma M^*}\right), a_{23} = \frac{\lambda M^*}{1+\gamma M^*}, a_{34} = \frac{\lambda\left(N^* - I^* - V_c^*\right)}{\left(1+\gamma M^*\right)^2}$$

Sufficient conditions for $\frac{dU_1}{dt}$ to be negative definite are given as follows:

$$a_{12}^2 < 4a_{11}a_{22},$$
$$a_{23}^2 < 4a_{22}a_{33},$$
$$a_{34}^2 < 4a_{33}a_{44}$$

where $a_{11} = (2\beta I^* - \beta N^* + \beta {V_c}^* + v + \alpha + \delta)$, $a_{22} = \left(\frac{\lambda M^*}{1+\gamma M^*} + \delta + \lambda_0\right)$, $a_{33} = \frac{\beta\delta I^*}{\alpha}$, $a_{44} = \frac{\mu_0\lambda(N^*-I^*-{V_c}^*)}{\mu(1+\gamma M^*)^2}$, $a_{12} = -\left(\beta I^* + \frac{\lambda M^*}{1+\gamma M^*}\right)$, $a_{23} = \frac{\lambda M^*}{1+\gamma M^*}$, $a_{34} = \frac{\lambda(N^*-I^*-{V_c}^*)}{(1+\gamma M^*)^2}$

9. Appendix B

Proof of Theorem 5.2

Consider the following positive definite function related to the model system (2) in the context of E_2, $U_2 = k_0\left(I - I^* - I^*\log\frac{I}{I^*}\right) + \frac{k_1}{2}\left(V_c - {V_c}^*\right)^2 + \frac{k_2}{2}\left(N - N^*\right)^2 + \frac{k_3}{2}\left(M - M^*\right)^2$,

Here, $k_i (i = 0, 1, 2, 3)$ are positive constants. By differentiating the above equation with respect to t and utilizing system of equations (2), we obtain

$\frac{dU_2}{dt} = -k_0\beta\left(I - I^*\right)^2 - \left(\frac{\lambda k_1 M}{1+\gamma M} + k_1(\delta + \lambda_0)\right)\left(V_c - {V_c}^*\right)^2 - k_2\delta\left(N - N^*\right)^2 - k_3\mu_0(M-$
$M^*)^2 - \left(k_0\beta + \frac{\lambda k_1 M}{1+\gamma M}\right)\left(I - I^*\right)\left(V_c - V_c^*\right) - \left(k_2\alpha - k_0\beta\right)\left(I - I^*\right)\left(N - N^*\right) + \frac{\lambda k_1 M^*}{1+\gamma M^*}\left(V_c-$
${V_c}^*\right)\left(N - N^*\right) + \left(\frac{\lambda k_1(N-I^*-{V_c}^*)}{(1+\gamma M^*)(1+\gamma M)} - k_3\mu\right)\left(V_c - V_c^*\right)\left(M - M^*\right) + k_3\mu\left(N - N^*\right)\left(M - M^*\right)$,

Choosing $k_0 = 1, k_1 = 1, k_2 = \frac{\beta}{\alpha}, k_3 = \frac{\lambda(N-I^*-{V_c}^*)}{\mu(1+\gamma M^*)(1+\gamma M)}$, we get

$\frac{dU_2}{dt} = -\beta\left(I - I^*\right)^2 - \left(\frac{\lambda M}{1+\gamma M} + (\delta + \lambda_0)\right)\left(V_c - {V_c}^*\right)^2 - \frac{\beta\delta}{\alpha}\left(N - N^*\right)^2 - \frac{\lambda(N-I^*-{V_c}^*)\mu_0}{\mu(1+\gamma M^*)(1+\gamma M)}(M-$
$M^*)^2 - \left(\beta + \frac{\lambda M}{1+\gamma M}\right)\left(I - I^*\right)\left(V_c - {V_c}^*\right) + \frac{\lambda M^*}{1+\gamma M^*}\left(V_c - {V_c}^*\right)\left(N - N^*\right) + \frac{\lambda(N-I^*-{V_c}^*)}{(1+\gamma M^*)(1+\gamma M)}\left(N - N^*\right)\left(M - M^*\right)$,

$\frac{dU_2}{dt} = -b_{11}\left(I - I^*\right)^2 - b_{22}\left(V_c - V_c^*\right)^2 - b_{33}\left(N - N^*\right)^2 - b_{44}\left(M - M^*\right)^2 + b_{12}\left(I - I^*\right)\left(V_c-$
$V_c^*\right) + b_{23}\left(V_c - V_c^*\right)\left(N - N^*\right) + b_{34}\frac{\lambda(N-I^*-V_c^*)}{\mu(1+\gamma M^*)(1+\gamma M)}\left(N - N^*\right)\left(M - M^*\right)$.

Where $b_{11} = \beta, b_{22} = \left(\frac{\lambda M}{1+\gamma M} + (\delta + \lambda_0)\right), b_{33} = \frac{\beta\delta}{\alpha}, b_{44} = \frac{\lambda(N-I^*-{V_c}^*)\mu_0}{\mu(1+\gamma M^*)(1+\gamma M)}$

$b_{12} = -\left(\beta + \frac{\lambda M}{1+\gamma M}\right), b_{23} = \frac{\lambda M^*}{1+\gamma M^*}, b_{34} = \frac{\lambda(N-I^*-{V_c}^*)}{\mu(1+\gamma M^*)(1+\gamma M)}$,

Sufficient conditions for $\frac{dU_2}{dt}$ to be negative definite are given as follows:

$$b_{12}^2 < 4b_{11}b_{22}$$
$$b_{23}^2 < 4b_{22}b_{33}$$
$$b_{34}^2 < 4b_{33}b_{44}$$
$$\left(\beta + \lambda M_{\max}\right)^2 < 4\beta\left(\delta + \lambda_0\right),$$
$$\left(\frac{\lambda M^*}{1+\gamma M^*}\right)^2 < 4\frac{\beta\delta}{\alpha}\left(\delta + \lambda_0\right),$$
$$\frac{\lambda\left(N_{\max} - I^* - V_c\right)^*\right)}{\mu\left(1+\gamma M^*\right)} < 4\frac{\beta\delta}{\alpha}.$$

Hence proved.

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