MATHEMATICAL ANALYSIS OF AN AGE-STRUCTURED VACCINATION MODEL FOR MEASLES

S. M. GARBA[1], A.B. GUMEL [2] AND N. HUSSAINI

Dedicated in memory of Professor J.O.C. Ezeilo (1930-2013).

ABSTRACT. An age-structured vaccination model for the transmission dynamics of measles in a population is designed and rigorously analysed. In the absence of vaccination, the model exhibits the phenomenon of backward bifurcation (where an asymptotically-stable disease-free equilibrium (DFE) co-exists with an asymptotically-stable endemic equilibrium whenever the associated reproduction number is less than unity). This phenomenon is shown to arise due to the imperfect nature of children's natural immunity against infection or measles-induced mortality. For the case when the measles-induced mortality is negligible, it is shown, using a linear Lyapunov function, that the DFE of the model without vaccination is globally-asymptotically stable whenever the associated reproduction number is less than unity. Furthermore, the vaccination-free model has a unique endemic equilibrium whenever the reproduction threshold exceeds unity. This equilibrium is shown, using a non-linear Lyapunov function of Goh-Volterra type, to be globally-asymptotically stable for a special case. Numerical simulations of the vaccination model show that the use of an imperfect anti-measles vaccine can result in the effective control of measles in the community provided the vaccine efficacy and coverage rate are high enough.

1. INTRODUCTION

Measles is a highly contagious respiratory childhood disease caused by a virus of the genus *Morbillivirus*. The virus, which is typically transmitted by coughing and sneezing, causes dangerous (and fatal) complications, such as pneumonia, diarrhea, encephalitis, ear infections and permanent brain damage [25]. It accounts for about 30-40 million infections in children each year, and remains a major killer of children around the world [25]. Over 200 million people

have died of measles over the last 150 years [25]. In 2011, for example, about 158,000 people died of measles (mostly children under the age of five) [30]. The classical signs and symptoms of measles include fever, cough, cold, red eyes and rashes (which begin several days after the onset of fever) [11].

The control of measles is largely based on the use of MMR (measles, mumps and rubella) and MMRV (measles, mumps, rubella and varicella) vaccines [11]. The vaccines are typically administered to children between the ages of 12 to 18 months, with a follow-up booster between the ages of 4 and 6 years [11]. The vaccines are known to be about 95% effective [11, 20, 31], and their coverage rate varies from country-to-country [27]. Although the global measles vaccine coverage reaches 82% in 2009 [27, 29], numerous countries continue to face challenges achieving such high coverage rates [27].

Mathematical models have been used to gain insight into the transmission dynamics of measles in populations (see for instance [1, 2, 5, 6, 7, 8, 11, 17, 20, 22, 23, 25, 27, 32]). These studies are typically based on the use of SEIR (susceptible-exposed-infectious-recovered) compartmental models for assessing the impact of vaccination programs against measles. The SEIR class of models has also been applied in some policy-specific settings, such as in New Zealand where an age-structured measles model was used by the Ministry of Health to predict an epidemic of measles and to design optimal vaccination schedules for the country [23].

The purpose of the current study is to qualitatively assess the role of age-structure and the population-level impact of the widespread use of measles vaccine (i.e., routine anti-measles vaccination program) on the transmission dynamics of measles in a community. The paper is organized as follows. The new age-structured model for measles transmission dynamics (in the presence of an anti-measles vaccine) is formulated in Section 2. The model, in the absence of vaccination, is rigorously analysed in Section 3. The vaccination model is analysed in Section 4. Numerical simulation results are reported.

2. MODEL FORMULATION

The age-structured model to be designed is based on the transmission dynamics of measles in a population, subject to the use of the aforementioned anti-measles vaccine. In this study, "infants" are children under 18 months of age, while "children" are considered to

be those between 18 months to 12 years of age. The total population at time t, denoted by $N(t)$, is sub-divided into twelve mutually-exclusive compartments of unvaccinated susceptible infants ($S_I(t)$), unvaccinated susceptible children ($S_C(t)$), vaccinated susceptible infants (those who received the first MMR dose) ($V_I(t)$), vaccinated susceptible children (those who typically receive the second dose) ($V_C(t)$), exposed infants (infants who have been infected but have not yet shown clinical symptoms of the disease) ($E_I(t)$), exposed children ($E_C(t)$), symptomatic infants ($I_I(t)$), symptomatic children ($I_C(t)$), hospitalized infants ($H_I(t)$), hospitalized children ($H_C(t)$), recovered infants ($R_I(t)$) and recovered children ($R_C(t)$), so that

$$N(t) = S_I(t) + S_C(t) + V_I(t) + V_C(t) + E_I(t) + E_C(t) + I_I(t)$$
$$+ I_C(t) + H_I(t) + H_C(t)R_I(t) + R_C(t).$$

The age-structured vaccination model for measles transmission dynamics is given by the following deterministic system of non-linear differential equations (a flow diagram of the model is depicted in Figure 1; and the associated variables and parameters are described in Tables 1 and 2, respectively):

$$\frac{dS_I}{dt} = \Pi(1-f) - (\lambda_I + \theta_C\lambda_C)S_I - \alpha S_I - \mu S_I,$$

$$\frac{dS_C}{dt} = \alpha S_I + \omega_C V_C - \xi_C S_C - \psi(\theta_I\lambda_I + \lambda_C)S_C - \mu S_C,$$

$$\frac{dV_I}{dt} = \Pi f - (\lambda_I + \theta_C\lambda_C)(1 - \epsilon_I)V_I - \alpha V_I - \mu V_I,$$

$$\frac{dV_C}{dt} = \alpha V_I + \xi_C S_C - \omega_C V_C - \psi(\theta_I\lambda_I + \lambda_C)(1 - \epsilon_C)V_C - \mu V_C,$$

$$\frac{dE_I}{dt} = (\lambda_I + \theta_C\lambda_C)[S_I + V_I(1 - \epsilon_I)] - \alpha E_I - \sigma_I E_I - \mu E_I,$$

$$\frac{dE_C}{dt} = \alpha E_I + \psi(\theta_I\lambda_I + \lambda_C)[S_C + V_C(1 - \epsilon_C)] - \sigma_C E_C - \mu E_C,$$

$$\frac{dI_I}{dt} = \sigma_I E_I - \alpha I_I - \sigma_2 I_I - \gamma_2 I_I - \mu I_I - \delta_I I_I,$$

$$\frac{dI_C}{dt} = \alpha I_I + \sigma_C E_C - \sigma_3 I_C - \gamma_3 I_C - \mu I_C - \delta_C I_C,$$

(1)

$$\frac{dH_I}{dt} = \sigma_2 I_I - \alpha H_I - \gamma_I H_I - \mu H_I - \delta_2 H_I,$$

$$\frac{dH_C}{dt} = \sigma_3 I_C + \alpha H_I - \gamma_C H_C - \mu H_C - \delta_3 H_C,$$

$$\frac{dR_I}{dt} = \gamma_2 I_I + \gamma_I H_I - \alpha R_I - \mu R_I,$$

$$\frac{dR_C}{dt} = \alpha R_I + \gamma_3 I_C + \gamma_C H_C - \mu R_C.$$

In (1), Π represents the birth rate of infants and $0 \leq f \leq 1$ is the fraction of infants who received the first vaccine dose. Susceptible infants acquire measles infection from infected infants (at a rate λ_I), given by

$$\lambda_I = \frac{\beta_I}{N}(\eta_I E_I + I_I), \tag{2}$$

where, β_I is the infection rate of infants and the modification parameter $0 \leq \eta_I \leq 1$ accounts for the assumption that exposed infants transmit at a rate lower than symptomatic infants. Furthermore, susceptible infants acquire infection following effective contacts with infected children (at a rate $\theta_C \lambda_C$), where $0 \leq \theta_C \leq 1$ accounts for the assumed reduced likelihood of infants acquiring infection from older children and

$$\lambda_C = \frac{\beta_C}{N}(\eta_C E_C + I_C), \tag{3}$$

with β_C and η_C similarly defined as β_I and η_I above. Natural death occurs in each compartment at a rate μ, and infants mature (to become children) at a rate α.

Children are vaccinated at a rate ξ_C, and the vaccine is assumed to wane at a rate ω_C. Susceptible children acquire measles infection at a reduced rate $\psi(\theta_I \lambda_I + \lambda_C)$, where $0 \leq \psi \leq 1$ models the natural immunity of susceptible children against measles infection [20, 21] (that is, $\psi = 0$ represents perfect natural immunity against acquisition of infection; and $\psi = 1$ means children do not acquire any immunity against measles infection) and $0 \leq \theta_I \leq 1$ accounts for the assumed reduced likelihood of susceptible children acquiring measles infection from infected infants (because of less likelihood of mixing). The parameters $0 < \epsilon_I, \epsilon_C < 1$ account for the vaccine efficacy in infants and children, respectively, while σ_I and σ_C represent the progression rates of exposed infants and children into the corresponding symptomatic classes, respectively. The parameters σ_2 and σ_3 are hospitalization rates of infants and children, respectively.

Further, γ_2 and γ_3 are recovery rates of symptomatic infants and children, respectively (similarly, γ_I and γ_C are the recovery rates of hospitalized infants and children, respectively). Symptomatic and hospitalized infants and children die due to measles at the respective rates $\delta_I, \delta_C, \delta_2$ and δ_3. The model (1) assumes that recovery induces permanent immunity against re-infection [20]. Furthermore, it is assumed that exposed individuals can transmit infection [2]. It extends numerous measles transmission models (that incorporate a vaccine) in the literature, such as those in [1, 2, 11, 20, 27] by (*inter alia*):

(i): Accounting for age-structure. This is not considered in [2, 27].
(ii): Allowing for the dynamics of exposed infants (E_I) and children (E_C). Exposed classes are not considered in [11].
(iii): Allowing for measles transmission by exposed infants ($\eta_I \neq 0$) and children ($\eta_C \neq 0$). This is not considered in [1, 11, 20, 27].

The model (1) will now be rigorously analysed to gain insight into its dynamical features. Before doing so, it is instructive to consider the model in the absence of vaccination, as below.

3. ANALYSIS OF MODEL WITHOUT VACCINATION

Consider the model (1) in the absence of routine anti-measles vaccination (i.e., $f = V_I = V_C = \epsilon_I = \epsilon_C = \omega_C = \xi_C = 0$), given by (denoted as "vaccination-free model"):

$$\frac{dS_I}{dt} = \Pi - (\lambda_I + \theta_C \lambda_C)S_I - \alpha S_I - \mu S_I,$$

$$\frac{dS_C}{dt} = \alpha S_I - \psi(\theta_I \lambda_I + \lambda_C)S_C - \mu S_C,$$

$$\frac{dE_I}{dt} = (\lambda_I + \theta_C \lambda_C)S_I - \alpha E_I - \sigma_I E_I - \mu E_I,$$

$$\frac{dE_C}{dt} = \alpha E_I + \psi(\theta_I \lambda_I + \lambda_C)S_C - \sigma_C E_C - \mu E_C, \qquad (4)$$

$$\frac{dI_I}{dt} = \sigma_I E_I - \alpha I_I - \sigma_2 I_I - \gamma_2 I_I - \mu I_I - \delta_I I_I,$$

$$\frac{dI_C}{dt} = \alpha I_I + \sigma_C E_C - \sigma_3 I_C - \gamma_3 I_C - \mu I_C - \delta_C I_C,$$

$$\frac{dH_I}{dt} = \sigma_2 I_I - \alpha H_I - \gamma_I H_I - \mu H_I - \delta_2 H_I,$$

$$\frac{dH_C}{dt} = \sigma_3 I_C + \alpha H_I - \gamma_C H_C - \mu H_C - \delta_3 H_C,$$

$$\frac{dR_I}{dt} = \gamma_2 I_I + \gamma_I H_I - \alpha R_I - \mu R_I,$$

$$\frac{dR_C}{dt} = \alpha R_I + \gamma_3 I_C + \gamma_C H_C - \mu R_C.$$

For mathematical convenience, the modification parameters θ_I and θ_C are, from now on, set to unity.

3.1. Invariant Region.

Lemma 1. *The following biologically-feasible region of the vaccination-free model* (4)

$$\Omega = \left\{ (S_I, S_C, E_I, E_C, I_I, I_C, H_I, H_C, R_I, R_C) \in \mathbb{R}_+^{10} : \right.$$

$$\left. S_I + S_C + E_I + E_C + I_I + I_C + H_I + H_C + R_I + R_C \leq \frac{\Pi}{\mu} \right\}$$

is positively-invariant and attracting.

Proof. Adding the equations in the model system (4) gives

$$\frac{dN(t)}{dt} = \Pi - \mu N(t) - (\delta_I I_I(t) + \delta_C I_C(t) + \delta_2 H_I(t) + \delta_3 H_C(t)), \quad (5)$$

so that,

$$\frac{dN(t)}{dt} \leq \Pi - \mu N(t). \quad (6)$$

It follows from (6), and the Gronwall inequality, that

$$N(t) \leq N(0)e^{-\mu(t)} + \frac{\Pi}{\mu}[1 - e^{-\mu(t)}].$$

In particular, $N(t) \leq \Pi/\mu$ if $N(0) \leq \Pi/\mu$. Thus, Ω is positively-invariant. Hence, it is sufficient to consider the dynamics of the model (4) in Ω. In this region, the model can be considered as been epidemiologically and mathematically well-posed [18].   □

3.2. Positivity of Solutions.

Theorem 3.1. *Let the initial data $S_I(0) > 0$, $S_C(0) > 0$, $E_I(0) > 0$, $E_C(0) > 0$, $I_I(0) > 0$, $I_C(0) > 0$, $H_I(0) > 0$, $H_C(0) > 0$, $R_I(0) > 0$, $R_C(0) > 0$, then the solutions $S_I(t)$, $S_C(t)$, $E_I(t)$, $E_C(t)$, $I_I(t)$, $I_C(t)$, $H_I(t)$, $H_C(t)$, $R_I(t)$, $R_C(t)$ of the vaccination-free model* (4) *are positive for all $t \geq 0$.*

Proof. It is clear from the first equation of (4) that

$$\frac{dS_I}{dt} \geq -(\lambda_I + \lambda_C + \alpha + \mu)S_I,$$

so that,

$$S_I(t) \geq S_I(0) \exp\left[-\int_0^t (\lambda_I + \lambda_C + \alpha + \mu)du\right] > 0, \text{ for all } t > 0.$$

It can be shown, using similar approach, that $S_C(t) > 0$, $E_I(t) > 0$, $E_C(t) > 0$, $I_I(t) > 0$, $I_C(t) > 0$, $H_I(t) > 0$, $H_C(t) > 0$ and $R_I(t) > 0$, $R_C(t) > 0$ for all $t \geq 0$. $\qquad\square$

3.3. **Stability of Disease-Free Equilibria (DFE).**

3.3.1. *Local stability.* The DFE of the vaccination-free model (4) is given by

$$\mathcal{E}_0 = (S_I^*, S_C^*, E_I^*, E_C^*, I_I^*, I_C^*, H_I^*, H_C^*, R_I^*, R_C^*)$$

$$= \left(\frac{\Pi}{\alpha + \mu}, \frac{\alpha}{\mu} S_I^*, 0, 0, 0, 0, 0, 0, 0, 0\right). \tag{7}$$

Since the population of recovered children (R_C) does not feature in any of the other equations of the vaccination-free model (4), the equation for dR_C/dt is temporarily removed from the analysis.

The linear stability of $\mathcal{E}_0$ can be established using the next generation operator method on the system (4) [28]. The matrices F (for the new infection terms) and V (of the transition terms) are given, respectively, by

$$F = \frac{1}{K_1}\begin{bmatrix} \beta_1\eta_1\mu & \beta_2\eta_2\mu & \beta_1\mu & \beta_2\mu & 0 & 0 \\ \psi\beta_1\eta_1\alpha & \psi\beta_2\eta_2\alpha & \psi\beta_1\alpha & \psi\beta_2\alpha & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix},$$

$$V = \begin{bmatrix} K_2 & 0 & 0 & 0 & 0 & 0 \\ -\alpha & K_3 & 0 & 0 & 0 & 0 \\ -\sigma_I & 0 & K_4 & 0 & 0 & 0 \\ 0 & -\sigma_C & -\alpha & K_5 & 0 & 0 \\ 0 & 0 & -\sigma_2 & 0 & K_6 & 0 \\ 0 & 0 & 0 & -\sigma_3 & -\alpha & K_7 \end{bmatrix},$$

where, $K_1 = \alpha + \mu$, $K_2 = \alpha + \sigma_I + \mu$, $K_3 = \sigma_C + \mu$, $K_4 = \alpha + \sigma_2 + \gamma_2 + \mu + \delta_I$, $K_5 = \sigma_3 + \gamma_3 + \mu + \delta_C$, $K_6 = \alpha + \gamma_I + \mu + \delta_I$

and $K_7 = \gamma_C + \mu + \delta_C$. It follows then that the *basic reproduction number* of the model (4), denoted by $\mathcal{R}_0$, is given by

$$\mathcal{R}_0 = \frac{\psi\alpha\beta_C(\eta_C K_5 + \sigma_C)}{K_1 K_3 K_5} + \frac{\mu\beta_I(\eta_I K_4 + \sigma_I)}{K_1 K_2 K_4}$$
$$+ \frac{\beta_C\mu\alpha[K_4(\eta_C K_5 + \sigma_C) + \sigma_I K_3]}{\displaystyle\prod_{i=1}^{5} K_i}.$$

Hence, using Theorem 2 of [28], the following result is established.

Lemma 2. *The DFE, $\mathcal{E}_0$, of the measles model* (4) *is locally-asymptotically stable (LAS) in Ω if $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$.*

The threshold quantity, $\mathcal{R}_0$, represents the average number of secondary cases that one infected case can generate if introduced into a completely-susceptible population [3, 4, 18]. The epidemiological implication of Lemma 2 is that the spread of measles can be effectively controlled in the population (when $\mathcal{R}_0 < 1$ if the initial sizes of the state variables of the model are in the basin of attraction of the DFE ($\mathcal{E}_0$).

3.4. **Endemic Equilibrium and Backward Bifurcation.** Let $\mathcal{E}_1 = (S_I^{**}, S_C^{**}, E_I^{**}, E_C^{**}, I_I^{**}, I_C^{**}, H_I^{**}, H_C^{**}, R_I^{**})$ represents any arbitrary endemic equilibrium of the model (4). Furthermore, let

$$\lambda^{**} = \lambda_I^{**} + \lambda_C^{**} \;\; = \frac{\beta_I}{N^{**}}(\eta_I E_I^{**} + I_I^{**}) + \frac{\beta_C}{N^{**}}(\eta_C E_C^{**} + I_C^{**}) \quad (8)$$

be the force of infection at steady-state. Solving the equations in (4) at steady-state gives

$$S_I^{**} = \frac{\Pi}{\lambda^{**} + K_1}, \ S_C^{**} = \frac{\alpha\Pi}{(\psi\lambda^{**} + \mu)(\lambda^{**} + K_1)},$$

$$E_I^{**} = \frac{\Pi\lambda^{**}}{K_2(\lambda^{**} + K_1)}, \ E_C^{**} = \frac{\alpha\Pi\lambda^{**}[\psi(\lambda^{**} + K_2) + \mu]}{K_2 K_3(\psi\lambda^{**} + \mu)(\lambda^{**} + K_1)},$$

$$I_I^{**} = \frac{\sigma_I\Pi\lambda^{**}}{K_2 K_4(\lambda^{**} + K_1)},$$

$$I_C^{**} = \frac{\alpha\Pi\lambda^{**}[\psi\sigma_2 K_4(\lambda^{**} + K_2) + \sigma_I K_3(\psi\lambda^{**} + \mu) + K_4\sigma_2\mu]}{(\psi\lambda^{**} + \mu)(\lambda^{**} + K_1)\prod\limits_{i=2}^{5} K_i}, \quad (9)$$

$$R_I^{**} = \frac{\sigma_I\Pi\lambda^{**}(\gamma_I\sigma_3 + \gamma_3 K_6)}{K_1 K_2 K_4 K_6(\lambda^{**} + K_1)}, \ H_I^{**} = \frac{\sigma_I\sigma_2\Pi\lambda^{**}}{K_2 K_4 K_6(\lambda^{**} + K_1)},$$

$$H_C^{**} = \frac{\alpha\Pi\lambda^{**}[\sigma_C\sigma_2 K_4 K_6[\psi(\lambda^{**} + K_2) + \mu] + \sigma_I K_3(\sigma_I K_5 + \sigma_2 K_6)(\psi\lambda^{**} + \mu)]}{(\psi\lambda^{**} + \mu)(\lambda^{**} + K_1)\prod\limits_{i=2}^{7} K_i}.$$

It can be shown, by substituting (9) into (8), that the non-zero equilibria of the vaccination-free model (4) satisfy the following quadratic (in terms of λ^{**})

$$a_1(\lambda^{**})^2 + b_1\lambda^{**} + c_1 = 0, \quad (10)$$

where,

$$\begin{aligned}
a_1 &= \psi(K_1\{K_3 K_5 K_6 K_7(\sigma_I + K_4) + \alpha\sigma_I K_3[K_6(K_7 + \sigma_2) + \sigma_I K_5] \\
&\quad + \alpha K_4 K_6[K_7(K_5 + \sigma_C) + \sigma_C\sigma_3]\} + \sigma_I K_3 K_5 K_7(\sigma_2 K_1 + \gamma_2 K_6)),
\end{aligned}$$

$$\begin{aligned}
b_1 &= K_3 K_7\{\mu\sigma_I K_5(\gamma_2 K_6 + \gamma_I\sigma_3) + K_1[K_5 K_6(\mu K_4 + \psi K_2 K_4 + \mu\sigma_I) \\
&\quad + \mu\sigma_I(\alpha K_6 + \sigma_3 K_5)]\} + \alpha K_1\{\mu\sigma_I K_3(\sigma_2 K_4 + \sigma_I K_5) \\
&\quad + K_4 K_6 K_7[\mu\sigma_C + \mu K_5 + \psi K_2(\sigma_C + K_5)] + \sigma_C\sigma_2 K_4 K_6(\mu + \psi K_2)\} \\
&\quad - \psi K_1 K_6 K_7[\beta_2\alpha\sigma_I K_3 + \beta_1 K_3 K_5(\eta_1 K_4 + \sigma_I) + \beta_2\alpha K_4(\eta_2 K_5 + \sigma_2)],
\end{aligned}$$

$$c_1 = K_1(1 - \mathcal{R}_0)\prod\limits_{i=1}^{7} K_i.$$

Thus, the positive endemic equilibria of the vaccination-free model (4) can be obtained by solving for λ^{**} from (10) and substituting the results (positive values of λ^{**}) into the expressions in (9). Once the components for I_C^{**}, H_C^{**}, R_I^{**} are obtained, they can then be substituted into the equation for dR_C/dt in (4) to obtain R_C^{**}. It should be mentioned that the coefficient a_1, of (10), is always positive, and c_1 is positive (negative) if $\mathcal{R}_0$ is less than (greater than) unity, respectively. Thus, the following result is established.

Theorem 3.2. *The vaccination-free model* (4) *has:*

(i): *a unique endemic equilibrium if $c_1 < 0 \Leftrightarrow \mathcal{R}_0 > 1$;*

(ii): *a unique endemic equilibrium if $b_1 < 0$, and $c_1 = 0$ or $b_1^2 - 4a_1c_1 = 0$;*

(iii): *two endemic equilibria if $c_1 > 0$, $b_1 < 0$ and $b_1^2 - 4a_1c_1 > 0$;*

(iv): *no endemic equilibrium otherwise.*

It is clear from Theorem 2 (Case (i)) that the model has a unique endemic equilibrium whenever $\mathcal{R}_0 > 1$. Furthermore, Case (iii) indicates the possibility of backward bifurcation (where the stable DFE co-exists with a stable endemic equilibrium when $\mathcal{R}_0 < 1$; see, for instance, [13, 15, 16]). It is instructive, therefore, to explore the possibility of backward bifurcation in the model (4).

Theorem 3.3. *The vaccination-free model* (4) *undergoes backward bifurcation at $\mathcal{R}_0 = 1$ whenever the bifurcation coefficient a, given by* (A.4) *(in Appendix A), is positive.*

The proof of Theorem 3.3, based on using the centre manifold theory [9, 10, 28], is given in Appendix A. The epidemiological significance of the phenomenon of backward bifurcation is that the classical requirement of $\mathcal{R}_0 < 1$ is, although necessary, no longer sufficient for the effective control or elimination of measles in the community. That is, the presence of backward bifurcation in the vaccination-free model (4) suggests that the feasibility of controlling measles when $\mathcal{R}_0 < 1$ would be dependent on the initial sizes of the sub-population of the model (4). Hence, backward bifurcation makes the ability to effectively control the spread of the disease difficult. To the authors' knowledge, this is the first time such a phenomenon has been established in the transmission dynamics of measles. The phenomenon of backward bifurcation is illustrated in Figure 2. The possible causes of this phenomenon are now explored.

<div align="center">Non-existence of Backward Bifurcation</div>

Case I: Effect of natural infection-acquired immunity

It is convenient to define

$$\mathcal{R}_0^* = \mathcal{R}_0|_{\psi=0} = \frac{\mu\beta_I(\eta_I K_4 + \sigma_I)}{K_1 K_2 K_4} + \frac{\beta_C \mu\alpha[K_4(\eta_C K_5 + \sigma_C) + \sigma_I K_3]}{\displaystyle\prod_{i=1}^{5} K_i}.$$

We claim the following result:

Theorem 3.4. *The vaccination-free model* (4) *with* $\psi = 0$ *has no endemic equilibrium when* $\mathcal{R}_0^* \leq 1$, *and has a unique endemic equilibrium if* $\mathcal{R}_0^* > 1$.

Proof. Setting $\psi = 0$ (that is, children have acquired perfect natural immunity against measles infection) in equation (10) reduces the quadratic to the following linear equation:

$$b_2 \lambda^{**} + c_2 = 0, \tag{11}$$

with,

$$
\begin{aligned}
b_2 &= K_3 K_7 \{\mu \sigma_I K_5 (\gamma_2 K_6 + \gamma_I \sigma_3) + K_1 [K_5 K_6 (\mu K_4 + \psi K_2 K_4 \\
&\quad + \mu \sigma_I) + \mu \sigma_I (\alpha K_6 + \sigma_3 K_5)]\} + \alpha K_1 \{\mu \sigma_I K_3 (\sigma_2 K_4 + \sigma_I K_5) \\
&\quad + K_4 K_6 K_7 [\mu \sigma_C + \mu K_5 + \psi K_2 (\sigma_C + K_5)] + \sigma_C \sigma_2 K_4 K_6 (\mu \\
&\quad + \psi K_2)\},
\end{aligned}
$$

$$c_2 = K_1 (1 - \mathcal{R}_0^*) \prod_{i=1}^{7} K_i.$$

Clearly, $b_2 > 0$ and $c_2 \geq 0$ whenever $\mathcal{R}_0^* \leq 1$ (so that $\lambda^{**} = \frac{-c_2}{b_2} \leq 0$). Therefore, the vaccination-free model (4), with $\psi = 0$, has no positive (endemic) equilibrium whenever $\mathcal{R}_0^* \leq 1$. Furthermore, $\lambda^{**} = \frac{-c_2}{b_2} > 0$ (i.e., the model (4) has a unique endemic equilibrium) if $\mathcal{R}_0^* > 1$.

$\square$

The above result suggests the impossibility of backward bifurcation in the vaccination-free model (4) when $\psi = 0$ (since no endemic equilibrium exists when $\mathcal{R}_0^* \leq 1$; and the phenomenon of backward bifurcation requires the presence of at least two endemic equilibria when $\mathcal{R}_0^* \leq 1$). Setting $\psi = 0$ in the expression for the backward bifurcation coefficients, a and b in equation (A.4) of Appendix A, gives, respectively,

$$
\begin{aligned}
a &= \frac{2\mu}{\Pi K_1} [\beta_I^*(w_3 \eta_I + w_5) + \beta_C (w_4 \eta_C + w_6)][(w_1 \alpha - \mu w_2) v_3 \\
&\quad - \mu v_3 (w_3 + w_4 + w_5 + w_6 + w_7 + w_8 + w_9)], \\
&= -\frac{\mu}{4 C_1 \sigma_I K_1 K_5 K_7} \Big(K_1 K_7 C_0 + 2\sigma_I K_5 [K_1 \alpha \sigma_2 \\
&\quad + 2 K_7 (\sigma_2 \gamma_I + \gamma_3)] \Big),
\end{aligned}
\tag{12}
$$

and,

$$b = \frac{K_5^2(\eta_1 K_4 + \sigma_I)}{2C_1\beta_C\sigma_C\sigma_I\mu},$$

where,

$$
\begin{aligned}
C_0 &= \{2K_5(K_4 + \sigma_I\sigma_C) + \sigma_I[\sigma_C C_1 + 2\alpha + C_1 K_5 \\
&+ \sigma_3(\sigma_C C_1 + 2\alpha)]\},
\end{aligned}
$$

$$
C_1 = \frac{\sigma_I K_5^2(K_1 K_4 + \mu\beta_I^*) + \mu\alpha\beta_C K_1 K_5(K_4 + K_5)}{\beta_C\sigma_C\sigma_I K_4},
$$

with $\beta_I^* > 0$ as defined in Appendix A.

Since $C_0 > 0$ and $C_1 > 0$, it follows that the bifurcation coefficient, a, is automatically negative (see Appendix A, where it is evident that $w_1\alpha - \mu w_2 = 0$ for $\psi = 0$). Furthermore, since the bifurcation coefficient b is positive, it follows from Theorem 4.1 of [10] that the vaccination-free model (4) with $\psi = 0$ will not undergo backward bifurcation at $\mathcal{R}_0 = 1$ (it should be noted that the model (4) has a varying total population size). These analyses show that the assumed natural immunity of children against measles infection ($\psi \neq 0$) can induce the phenomenon of backward bifurcation in measles transmission dynamics.

3.4.1. *Case II: Effect of disease-induced mortality.* It is convenient to define $\delta_I = \delta_C = \delta_2 = \delta_3 = \delta$. Consider the vaccination-free model (4) with the associated disease-induced mortality (δ) set to zero. Thus,

$$\frac{dN(t)}{dt} = \Pi - \mu N(t),$$

so that, $N(t) \to \Pi/\mu$ as $t \to \infty$. In other words, this special case considers the model (4) with constant total population size, $N(t) = \frac{\Pi}{\mu}$ (unlike in Case I above). Using $N = \frac{\Pi}{\mu}$ in (2) and (3) show, respectively, that

$$\lambda_I = \frac{\mu\beta_I}{\Pi}(\eta_I E_I + I_I) \text{ and } \lambda_C = \frac{\mu\beta_C}{\Pi}(\eta_C E_C + I_C). \qquad (13)$$

Let,

$$\lambda_m^{**} = \frac{\mu}{\Pi}[\beta_I(\eta_I E_I^{**} + I_I^{**}) + \beta_C(\eta_C E_C^{**} + I_C^{**})]. \qquad (14)$$

Consequently, the resulting (mass action) vaccination-free model, obtained by using (13) in (4), has the same DFE, given by (7), as

the model (4). Furthermore, the associated reproduction number of the mass action vaccination-free model, denoted by $\mathcal{R}_0^m$, is given by:

$$\mathcal{R}_0^m = \frac{\psi\alpha\beta_C(\eta_C K_5 + \sigma_C)}{K_1 K_3 \tilde{K}_5} + \frac{\mu\beta_I(\eta_I \tilde{K}_4 + \sigma_I)}{K_1 K_2 \tilde{K}_4}$$
$$+ \frac{\beta_C\mu\alpha[\tilde{K}_4(\eta_C \tilde{K}_5 + \sigma_C) + \sigma_I K_3]}{K_1 K_2 K_3 \tilde{K}_4 \tilde{K}_5},$$

where, $\tilde{K}_4 = \alpha + \sigma_2 + \gamma_2 + \mu$, $\tilde{K}_5 = \sigma_3 + \gamma_3 + \mu$, $\tilde{K}_6 = \alpha + \gamma_I + \mu$ and $\tilde{K}_7 = \gamma_C + \mu$. It can be shown that the non-zero equilibria of the mass action model satisfy the following quadratic (in terms of λ_m^{**})

$$a_3(\lambda_m^{**})^2 + b_3\lambda_m^{**} + c_3 = 0, \tag{15}$$

where,

$$a_3 = \psi, \ b_3 = \frac{\psi\alpha\beta_C(\eta_C \tilde{K}_5 + \sigma_C)}{K_3 \tilde{K}_5} + \mu\psi K_1(1 - \mathcal{R}_0^m),$$
$$c_3 = \mu K_1(1 - \mathcal{R}_0^m).$$

It is clear from (15) that $a_3 > 0$, $b_3 > 0$ and $c_3 > 0$ whenever $\mathcal{R}_0^m < 1$. Thus, by the Routh-Hurwith criterion, the quadratic (15) has no positive root in this case. Hence, the mass action model has no endemic equilibrium when $\mathcal{R}_0^m < 1$. Furthermore, the case when $\mathcal{R}_0^m = 1$ makes $c_3 = 0$. Thus, the quadratic in (15) reduces to $\lambda_m^{**}(a_3\lambda_m^{**} + b_3)$, with solutions $\lambda_m^{**} = 0$ (corresponding to the DFE, $\mathcal{E}_0$), and the linear equation $a_3\lambda_m^{**} + b_3$ (so that $\lambda_m^{**} = -b_3/a_3 < 0$). Thus, no endemic equilibrium exists whenever $\mathcal{R}_0^m \leq 1$. The above result shows that measles-induced mortality causes backward bifurcation in measles transmission dynamics.

It is worth mentioning that, for this case (with $\delta = 0$), the global asymptotic stability property of the DFE ($\mathcal{E}_0$) can be established, as below. Define, first of all, the invariant region

$$\tilde{\Omega} = \{(S_I, S_C, E_I, E_C, I_I, I_C, H_I, H_C, R_I) \in \Omega : S_I \leq S_I^*, \ S_C \leq S_C^*\}.$$

Theorem 3.5. *The DFE, $\mathcal{E}_0$, of the vaccination-free model* (4) *with $\delta = 0$, is globally-asymptotically stable (GAS) in $\tilde{\Omega}$ if $\mathcal{R}_0^m \leq 1$.*

Proof. Consider the model (4) with $\delta = 0$ and $\mathcal{R}_0^m \leq 1$. Furthermore, consider the linear Lyapunov function $\mathcal{F} = d_1 E_I + d_2 E_C + d_3 I_I + d_4 I_C$, where

$$d_1 = \frac{\mu\beta_1(\eta_1\tilde{K}_4 + \sigma_I)}{K_1 K_2 \tilde{K}_4} + \frac{\mu\beta_2\alpha[\tilde{K}_4(\eta_2\tilde{K}_5 + \sigma_2) + \sigma_I K_3]}{K_1 K_2 K_3 \tilde{K}_4 \tilde{K}_5},$$

$$d_2 = \frac{\mu\beta_2(\eta_2\tilde{K}_5 + \sigma_2)}{K_1 K_3 \tilde{K}_5}, \ d_3 = \frac{\mu(\beta_1\tilde{K}_5 + \beta_2\alpha)}{K_1 \tilde{K}_4 \tilde{K}_5}, \ d_4 = \frac{\mu\beta_2}{K_1 \tilde{K}_5},$$

with Lyapunov derivative given by (where a dot represents differentiation with respect to time t)

$$\dot{\mathcal{F}} = d_1 \dot{E}_I + d_2 \dot{E}_C + d_3 \dot{I}_I + d_4 \dot{I}_C,$$

$$= d_1\left\{\left[\frac{\mu\beta_I}{\Pi}(\eta_I E_I + I_I) + \frac{\mu\beta_C}{\Pi}(\eta_C E_C + I_C)\right]S_I - K_2 E_I\right\}$$

$$+ d_2\left\{\alpha E_I + \psi\left[\frac{\mu\beta_I}{\Pi}(\eta_I E_I + I_I) + \frac{\mu\beta_C}{\Pi}(\eta_C E_C + I_C)\right]S_C\right.$$

$$-K_3 E_C\}$$

$$+ d_3(\sigma_I E_I - \tilde{K}_4 I_I) + d_4(\alpha I_I + \sigma_C E_C - \tilde{K}_5 I_C),$$

$$= \left[\frac{\mu\beta_I\eta_I}{\Pi}(d_1 S_I + d_2\psi S_C) - d_1 K_2 + d_2\alpha + d_3\sigma_I\right]E_I$$

$$+ \left[\frac{\mu\beta_C\eta_C}{\Pi}(d_1 S_I + d_2\psi S_C) - d_2 K_3 + d_4\sigma_2\right]E_C$$

$$+ \left[\frac{\mu\beta_I}{\Pi}(d_1 S_I + d_2\psi S_C) - d_3\tilde{K}_4 + d_4\alpha\right]I_I$$

$$+ \left[\frac{\mu\beta_C}{\Pi}(d_1 S_I + d_2\psi S_C) - d_4\tilde{K}_5\right]I_C,$$

$$\leq \left[\frac{\beta_I\eta_I\mu}{K_1}(\mathcal{R}_0^m - 1)\right]E_I + \left[\frac{\beta_C\eta_C\mu}{K_1}(\mathcal{R}_0^m - 1)\right]E_C + \left[\frac{\beta_I\mu}{K_1}(\mathcal{R}_0^m - 1)\right]I_I$$

$$+ \left[\frac{\beta_C\mu}{K_1}(\mathcal{R}_0^m - 1)\right]I_C, \text{ since } S_I \leq S_I^* \text{ and } S_C \leq S_C^* \text{ in } \tilde{\Omega},$$

$$= \frac{\mu}{K_1}[\beta_I(\eta_I E_I + I_I) + \beta_C(\eta_C E_C + I_C)](\mathcal{R}_0^m - 1).$$

Since all the parameters and variables of the model (4), with (13), are non-negative (Theorem 1), it follows that $\dot{\mathcal{F}} \leq 0$ for $\mathcal{R}_0^m \leq 1$ with $\dot{\mathcal{F}} = 0$ if and only if $E_I = E_C = I_I = I_C = 0$. Hence, $\dot{\mathcal{F}}$ is a Lyapunov function on $\tilde{\Omega}$. Thus, it follows, by LaSalle's Invariance Principle [19], that

$$(E_I(t),\, E_C(t),\, I_I(t),\, I_C(t)) \rightarrow (0,\, 0,\, 0,\, 0) \text{ as } t \rightarrow \infty. \qquad (16)$$

Since $\lim\limits_{t\to\infty} \sup E_I(t) = 0$, $\lim\limits_{t\to\infty} \sup E_C(t) = 0$, $\lim\limits_{t\to\infty} \sup I_I(t) = 0$ and $\lim\limits_{t\to\infty} \sup I_C(t) = 0$ (from (2)), it follows that, for sufficiently small $\tau^* > 0$, there exist constants $N_1 > 0$, $N_2 > 0$, $N_3 > 0$, $N_4 > 0$, such that, $\lim\limits_{t\to\infty} \sup E_I(t) \leq \tau^*$ for all $t > N_1$, $\lim\limits_{t\to\infty} \sup E_C(t) \leq \tau^*$ for all $t > N_2$, $\lim\limits_{t\to\infty} \sup I_I(t) \leq \tau^*$ for all $t > N_3$, and $\lim\limits_{t\to\infty} \sup I_C(t) \leq \tau^*$ for all $t > N_4$. Hence, it follows from the seventh equation of the vaccination-free model (4) that, for $t > \max\{N_1, N_2, N_3, N_4\}$, $\dot{H}_I = \sigma_2 \tau^* - K_6 H_I$. Thus, by comparison theorem [26],

$$H_I^\infty = \lim_{t\to\infty} \sup H_I(t) \leq \frac{\sigma_2 \tau^*}{K_6}, \qquad (17)$$

so that, by letting, $\tau^* \to 0$

$$H_I^\infty = \lim_{t\to\infty} \sup H_I(t) \leq 0. \qquad (18)$$

Similarly, it can be shown that

$$H_{I\infty} = \lim_{t\to\infty} \inf H_I(t) \geq 0. \qquad (19)$$

Thus, it follows from (18) and (19) that

$$H_{I\infty} \geq 0 \geq H_I^\infty. \qquad (20)$$

Hence,

$$\lim_{t\to\infty} H_I(t) = 0. \qquad (21)$$

Similarly, it can be shown that

$$\lim_{t\to\infty} S_I(t) = S_I{}^*, \ \lim_{t\to\infty} S_C(t) = S_C{}^*, \ \lim_{t\to\infty} H_C(t) = H_C{}^* \qquad (22)$$

and $\lim\limits_{t\to\infty} R_I(t) = R_I{}^*$.

Substituting $(R_I(t), I_C(t), H_C(t)) = (0, 0, 0)$ in the equation for $\frac{dR_C}{dt}$ in (4) shows that $\lim\limits_{t\to\infty} R_C(t) = 0$. Thus, by combining (16), (21) and (22) and noting that $\lim\limits_{t\to\infty} R_C(t) = 0$, it follows that every solution of the equations of the model (4), with (13) and initial conditions in $\tilde{\Omega}$, approaches $\mathcal{E}_0$ as $t \to \infty$ (whenever $\mathcal{R}_0^m \leq 1$). $\qquad \square$

The epidemiological implication of Theorem 3.5 is that measles will be effectively-controlled or eliminated from the community if $\mathcal{R}_0^m \leq 1$ (regardless of the initial sizes of the sub-populations of the vaccination-free model (4) with (13)). Figure 3A depicts the simulation results of the model (4), with (13), for the case when $\mathcal{R}_0^m < 1$ (showing convergence of the solutions to the DFE, $\mathcal{E}_0$ (in line with Theorem 3.5)).

3.5. **Global Stability of Endemic Equilibrium: Special Case.** In this section, the global asymptotic stability of the unique endemic equilibrium of model (4) is given for the special case where the associated disease-induced mortality is negligible (i.e., $\delta = 0$) and children do not acquire natural immunity against infection (i.e., $\psi = 1$). For this scenario, the following change of variables can be made: $S = S_I + S_C$, $E = E_I + E_C$, $I = I_I + I_C$, $H = H_I + H_C$, $R = R_I + R_C$. Using these transformations in (4) gives the following reduced vaccination-free model (without age-structure).

$$
\begin{aligned}
\frac{dS}{dt} &= \Pi - \lambda S - \mu S, \\
\frac{dE}{dt} &= \lambda S - \sigma_I E - \mu E, \\
\frac{dI}{dt} &= \sigma_I E - \sigma_2 I - \gamma_2 I - \mu I, \\
\frac{dH}{dt} &= \sigma_2 I - \gamma_I H - \mu H, \\
\frac{dR}{dt} &= \gamma_2 I + \gamma_I H - \mu R,
\end{aligned}
\tag{23}
$$

where, now, $\lambda = \dfrac{\mu \beta}{\Pi}(\eta E + I)$. It can be shown that the associated reproduction number of the reduced model (23) is given by

$$
\mathcal{R}_{0s}^m = \frac{\tilde{\beta}(\eta P_1 + \sigma_I)}{P_1 P_2},
\tag{24}
$$

where, $\tilde{\beta} = \dfrac{\mu \beta}{\Pi}$, $P_1 = \sigma_I + \mu$, $P_2 = \sigma_2 + \gamma_2 + \mu$ and $P_3 = \gamma_I + \mu$. Furthermore, the following result can be shown, using the technique in Section 3.4.

Lemma 3. *The reduced model,* (23), *has a unique positive endemic equilibrium, of the form* $\mathcal{E}_2 = (S^{***}, E^{***}, I^{***}, H^{***}, R^{***})$, *whenever* $\mathcal{R}_{0s}^m > 1$.

Define the invariant region

$$\Omega_r = \left\{ (S, E, I, H, R) \in \mathbb{R}_+^5 : S + E + I + H + R \le \frac{\Pi}{\mu} \right\}$$

and (the stable manifold of the DFE of the reduced model (23))

$$\Omega_1 = \{(S, E, I, H, R) \in \Omega_r : E = I = H = R = 0\}.$$

Theorem 3.6. *The unique endemic equilibrium of the reduced model (23), given by $\mathcal{E}_2$, is GAS in $\Omega_r \backslash \Omega_1$ if $\mathcal{R}_{0s}^m > 1$.*

Proof. Consider the reduced model (23). Let $\mathcal{R}_{0s}^m > 1$, so that the associated unique endemic equilibrium of the reduced model (23) exists (Lemma 3). Furthermore, consider the following non-linear Lyapunov function (of Goh-Volterra type) for the sub-system of the reduced model (23) involving the state variables S, E and I:

$$\mathcal{F} = S - S^{***} - S^{***} \ln\left(\frac{S}{S^{***}}\right) + E - E^{***} - E^{***} \ln\left(\frac{E}{E^{***}}\right)$$
$$+ \frac{P_1}{\sigma_I}\left[I - I^{***} - I^{***} \ln\left(\frac{I}{I^{***}}\right)\right],$$

with Lyapunov derivative,

$$\begin{aligned}
\dot{\mathcal{F}} &= \dot{S} - \frac{S^{***}}{S}\dot{S} + \dot{E} - \frac{E^{***}}{E}\dot{E} + \frac{P_1}{\sigma_I}\left(\dot{I} - \frac{I^{***}}{I}\dot{I}\right), \\
&= \Pi - \tilde{\beta}(\eta E + I)S - \mu S - \frac{S^{***}}{S}\left[\Pi - \tilde{\beta}(\eta E + I)S - \mu S\right] \\
&\quad + \tilde{\beta}(\eta E + I)S - P_1 E - \frac{E^{***}}{E}\left[\tilde{\beta}(\eta E + I)S - P_1 E\right] \\
&\quad + \frac{P_1}{\sigma_I}\left[\sigma_I E - P_2 I - \frac{I^{***}}{I}(\sigma_I E - P_2 I)\right].
\end{aligned} \tag{25}$$

It can be shown, from (23), that the following relations hold at the endemic steady-state $\mathcal{E}_2$:

$$\begin{aligned}
\Pi &= \tilde{\beta}(\eta E^{***} + I^{***})S^{***} + \mu S^{***}, \quad \tilde{\beta}(\eta E^{***} + I^{***})S^{***} \\
&= P_1 E^{***}, \\
\sigma_I E^{***} &= P_2 I^{***}, \ \sigma_2 I^{***} = P_3 H^{***}, \ \gamma_2 I^{***} + \gamma_I H^{***} = \mu R^{***}.
\end{aligned} \tag{26}$$

Using the first three relations of (26) in (25), and simplifying, gives

$$\dot{\mathcal{F}} = \tilde{\beta}(\eta E^{***} + I^{***})S^{***} + \mu S^{***} - \mu S$$
$$- \frac{S^{***}}{S}\left[\tilde{\beta}(\eta E^{***} + I^{***})S^{***} - \tilde{\beta}(\eta E + I)S + \mu S^{***} - \mu S\right]$$
$$- \frac{E^{***}}{E}\tilde{\beta}(\eta E + I)S + P_1 E^{***} - \frac{P_1 P_2 I}{\sigma_I} - P_1 E\frac{I^{***}}{I} + \frac{P_1 P_2 I^{***}}{\sigma_I},$$
$$= \mu S^{***}\left(2 - \frac{S^{***}}{S} - \frac{S}{S^{***}}\right)$$
$$+ \tilde{\beta}\eta E^{***}S^{***}\left(3 - \frac{S}{S^{***}} - \frac{S^{***}}{S} - \frac{I}{I^{***}} - \frac{I^{***}E}{IE^{***}}\right)$$
$$+ \tilde{\beta}I^{***}S^{***}\left(3 - \frac{S^{***}}{S} - \frac{I}{I^{***}} - \frac{I^{***}E}{IE^{***}} - \frac{E^{***}IS}{EI^{***}S^{***}}\right).$$

Finally, since the arithmetic mean exceeds the geometric mean, it follows then that

$$\mu S^{***}\left(2 - \frac{S^{***}}{S} - \frac{S}{S^{***}}\right) \leq 0,$$
$$\tilde{\beta}\eta E^{***}S^{***}\left(3 - \frac{S}{S^{***}} - \frac{S^{***}}{S} - \frac{I}{I^{***}} - \frac{I^{***}E}{IE^{***}}\right) \leq 0,$$
$$\text{and } \tilde{\beta}I^{***}S^{***}\left(3 - \frac{S^{***}}{S} - \frac{I}{I^{***}} - \frac{I^{***}E}{IE^{***}} - \frac{E^{***}IS}{EI^{***}S^{***}}\right) \leq 0.$$

Furthermore, since all the model parameters are non-negative, it follows that $\dot{\mathcal{F}} \leq 0$ for $\mathcal{R}_{0s}^m > 1$. Thus, $\dot{\mathcal{F}}$ is a Lyapunov function for the sub-system of the model (23) on $\Omega_r \backslash \Omega_1$. Therefore, it follows, by LaSalle's Invariance Principle [19], that

$$\lim_{t \to \infty} S(t) = S^{***}, \ \lim_{t \to \infty} E(t) = E^{***}, \ \lim_{t \to \infty} I(t) = I^{***}.$$

Since $I(t) \to I^{***}$ as $t \to \infty$, it follows from the equation for dH/dt in (23) that, $H(t) \to \frac{\sigma_2 I^{***}}{P_3} = H^{***}$ as $t \to \infty$. Similarly, $R(t) \to \left(\frac{\gamma_2}{\mu} + \frac{\gamma_I \sigma_2}{\mu P_3}\right)I^{***} = R^{***}$ as $t \to \infty$. The proof is concluded using similar arguments as in the proof of Theorem 3.5. □

The epidemiological implication of Theorem 3.6 is that measles will establish itself (be endemic) in the community whenever $\mathcal{R}_{0s}^m > 1$ and the disease-induced mortality (δ) is negligible and children do not acquire natural immunity against infection ($\psi = 1$). Figure 3B depicts the numerical results obtained by simulating the reduced

model (23) with $\delta = 0$ and $\psi = 1$, using various initial conditions, for the case when $\mathcal{R}_{0s}^m > 1$ (showing convergence of the solutions to an endemic equilibrium in line with Theorem 3.6).

4. Analysis of the Vaccination Model

It can be shown, using the approach in Section 3, that the following biologically-feasible region

$$
\begin{aligned}
\Gamma \ &= \ (S_I, S_C, V_I, V_C, E_I, E_C, I_I, I_C, H_I, H_C, R_I, R_C) \in \mathbb{R}_+^{12} : \\
& \quad S_I + S_C + V_I + V_C + E_I + E_C + I_I + I_C + H_I + H_C + R_I \\
& + \ R_C \leq \frac{\Pi}{\mu}
\end{aligned}
$$

is positively-invariant and attracting for the vaccination model (1).

4.1. **Local Stability of DFE.** The DFE of the vaccination model (1) is given by

$$
\begin{aligned}
\mathcal{E}_v &= (S_I^*, S_C^*, V_I^*, V_C^*, E_I^*, E_C^*, I_I^*, I_C^*, H_I^*, H_C^*, R_I^*,, R_C^*) \\
&= \left(S_I^*, S_C^*, V_I^*, V_C^*, 0, 0, 0, 0, 0, 0, 0, 0 \right),
\end{aligned}
\tag{27}
$$

where,

$$
S_I^* = \frac{(1-f)\Pi}{K_1}, \ S_C^* = \frac{\alpha\Pi[K_9(1-f)+fw]}{\mu K_1(K_8+\omega_C)}, \ V_I^* = \frac{f\Pi}{K_1},
$$

$$
V_C^* = \frac{\alpha\Pi[\xi(1-f)+fK_8]}{\mu K_1(K_8+\omega_C)},
$$

with $K_8 = \xi_C + \mu$ and $K_9 = \omega_C + \mu$. The associated *vaccination reproduction number*, denoted by $\mathcal{R}_v$, of the model is given by

$$
\begin{aligned}
\mathcal{R}_v &= \frac{q_2\psi\alpha\beta_C(\eta_C K_5 + \sigma_C)}{K_1 K_3 K_5} + \frac{q_1\mu\beta_I(\eta_I K_4 + \sigma_I)}{K_1 K_2 K_4} \\
& + \frac{q_1\beta_C\mu\alpha[K_4(\eta_C K_5 + \sigma_C) + \sigma_I K_3]}{\displaystyle\prod_{i=1}^{5} K_i},
\end{aligned}
$$

where,

$$
q_1 = \frac{K_1}{\mu N^*}[S_I^* + V_I^*(1-\epsilon_I)] = 1 - f\epsilon_I,
$$

and,

$$q_2 = \frac{K_1}{\alpha N^*}[S_C^* + V_C^*(1 - \epsilon_C)] = 1 - \frac{[(1-f)\xi + fK_8]\epsilon_C}{K_8 + \omega_C}.$$

The following result can be established for the model (1), using the approach in Section 3.

Lemma 4. *The DFE, $\mathcal{E}_v$, of the model* (1) *is LAS if $\mathcal{R}_v < 1$, and unstable if $\mathcal{R}_v > 1$.*

It should be mentioned that, like for the case of the vaccination-free model (4), the vaccination model (1) also undergoes backward bifurcation. This phenomenon occurs even if the natural immunity acquired by children is permanent ($\psi = 0$), provided the vaccine efficacy ($\epsilon_I = \epsilon_C = \epsilon$) is not perfect (i.e., $0 < \epsilon < 1$). Thus, the models (1) and (4) have essentially the same qualitative dynamics with respect to the local stability of the DFE and the backward bifurcation property observed in measles transmission dynamics.

4.2. **Global Asymptotic Stability of DFE.** In this section, the global asymptotic stability property of the DFE of the vaccination model (1) is explored for the special case where the vaccine efficacy is perfect ($\epsilon_I = \epsilon_C = \epsilon = 1$) and the disease-induced mortality is negligible ($\delta = 0$). Let

$$\tilde{\Gamma} = \{(S_I, S_C, V_I, V_C, E_I, E_C, I_I, I_C, H_I, H_C, R_I, R_C) \in \Gamma : S_I \leq S_I^*,$$
$$S_C \leq S_C^*, \ V_I \leq V_I^*, \ V_C \leq V_C^*\}.$$

It can be shown that the associated reproduction number of the model (1), with $\delta = 0$ and $\epsilon = 1$, is given by

$$\mathcal{R}_v^m = \mathcal{R}_v|_{\delta=0,\,\epsilon=1} = \left\{1 - \frac{[(1-f)\xi + fK_8]}{K_8 + \omega_C}\right\} \frac{\psi\alpha\beta_C(\eta_C\tilde{K}_5 + \sigma_C)}{K_1 K_3 \tilde{K}_5}$$
$$+ \mu\beta_I \frac{(1-f)(\eta_I\tilde{K}_4 + \sigma_I)}{K_1 K_2 \tilde{K}_4} + \frac{\beta_C\mu\alpha(1-f)[\tilde{K}_4(\eta_C\tilde{K}_5 + \sigma_C) + \sigma_I K_3]}{K_1 K_2 K_3 \tilde{K}_4 \tilde{K}_5}.$$

Theorem 4.1. *The DFE, $\mathcal{E}_v$, of the vaccination model* (1) *with $\delta = 0$ and $\epsilon = 1$, is GAS in $\tilde{\Gamma}$ if $\mathcal{R}_v^m \leq 1$.*

Proof. Consider the model (1) with $\delta = 0$ and $\epsilon = 1$. Further, let $\mathcal{R}_v^m \leq 1$. Consider the following Lyapunov function $\mathcal{F} = g_1 E_I + g_2 E_C + g_3 I_I + g_4 I_C$, where,

$$g_1 = \frac{\mu(1-f)}{K_1 K_2 \tilde{K}_4} \left\{ \beta_1(\eta_1 \tilde{K}_4 + \sigma_I) + \frac{\beta_2 \alpha[\tilde{K}_4(\eta_2 \tilde{K}_5 + \sigma_2) + \sigma_I K_3]}{K_3 \tilde{K}_5} \right\},$$

$$g_2 = \frac{\mu \beta_2(1-f)(\eta_2 \tilde{K}_5 + \sigma_2)}{K_1 K_3 \tilde{K}_5}, \ g_3 = \frac{\mu(1-f)(\beta_1 \tilde{K}_5 + \beta_2 \alpha)}{K_1 \tilde{K}_4 \tilde{K}_5},$$

$$g_4 = \frac{\mu \beta_2(1-f)}{K_1 \tilde{K}_5},$$

with Lyapunov derivative given by

$$\dot{\mathcal{F}} = g_1 \dot{E}_I + g_2 \dot{E}_C + g_3 \dot{I}_I + g_4 \dot{I}_C,$$

$$= g_1 \left\{ \left[\frac{\mu \beta_I(1-f)}{\Pi}(\eta_I E_I + I_I) + \frac{\mu \beta_C(1-f)}{\Pi}(\eta_C E_C + I_C) \right] S_I - K_2 E_I \right\}$$

$$+ g_2 \left\{ \alpha E_I + \psi \left[\frac{\mu \beta_I(1-f)}{\Pi}(\eta_I E_I + I_I) + \frac{\mu \beta_C(1-f)}{\Pi}(\eta_C E_C + I_C) \right] S_C$$

$$- K_3 E_C \right\} + g_3(\sigma_I E_I - \tilde{K}_4 I_I) + g_4(\alpha I_I + \sigma_C E_C - \tilde{K}_5 I_C),$$

$$= \left[\frac{(1-f)\mu \beta_I \eta_I}{\Pi}(g_1 S_I + g_2 \psi S_C) - g_1 K_2 + g_2 \alpha + g_3 \sigma_I \right] E_I$$

$$+ \left[\frac{(1-f)\mu \beta_C \eta_C}{\Pi}(g_1 S_I + g_2 \psi S_C) - g_2 K_3 + g_4 \sigma_2 \right] E_C$$

$$+ \left[\frac{(1-f)\mu \beta_I}{\Pi}(g_1 S_I + g_2 \psi S_C) - g_3 \tilde{K}_4 + g_4 \alpha \right] I_I$$

$$+ \left[\frac{(1-f)\mu \beta_C}{\Pi}(g_1 S_I + g_2 \psi S_C) - g_4 \tilde{K}_5 \right] I_C,$$

$$\leq \left[\frac{(1-f)\beta_I \eta_I \mu}{K_1}(\mathcal{R}_v^m - 1) \right] E_I + \left[\frac{(1-f)\beta_C \eta_C \mu}{K_1}(\mathcal{R}_v^m - 1) \right] E_C$$

$$+ \left[\frac{(1-f)\beta_I \mu}{K_1}(\mathcal{R}_v^m - 1) \right] I_I + \left[\frac{(1-f)\beta_C \mu}{K_1}(\mathcal{R}_v^m - 1) \right] I_C,$$

since $S_I \leq S_I^*$ and $S_C \leq S_C^*$ in $\tilde{\Gamma}$,

$$= \frac{(1-f)\mu}{K_1}[\beta_I(\eta_I E_I + I_I) + \beta_C(\eta_C E_C + I_C)](\mathcal{R}_v^m - 1) \leq 0 \text{ for } \mathcal{R}_v^m \leq 1.$$

The proof is concluded using the same approach as in the proof of Theorem 3.5. □

Theorem 4.1 shows that, for the case of the model (1) with $\delta = 0$ and $\epsilon = 1$, the use of an anti-measles vaccine could lead to the effective control of the disease in the community whenever the vaccine could reduce (and maintain) the associated reproduction threshold $(\mathcal{R}_v^m)$ to a value less than unity. Figure 4 depicts the solutions profiles of the model (1) for the case when $\delta = 0$, $\epsilon = 1$ and $\mathcal{R}_v^m < 1$

(showing convergence of the solutions to the DFE, $\mathcal{E}_0$, in line with Theorem 4.1).

4.3. **Threshold Analysis and Vaccine Impact.** The population-level impact of the widespread use of the anti-measles vaccines will be assessed by re-writing the vaccination reproduction threshold, $\mathcal{R}_v$, as follows:

$$\mathcal{R}_v = \mathcal{R}_0 \left[1 - \frac{V_I^* + V_C^*}{N^*} \left(1 - \frac{\mathcal{R}_{0v}}{\mathcal{R}_0} \right) \right], \tag{28}$$

where,

$$\begin{aligned} \mathcal{R}_{0v} &= \frac{\psi \alpha \beta_C (1 - \epsilon_C)(\eta_C K_5 + \sigma_C)}{K_1 K_3 K_5} + \mu(1 - \epsilon_I)Q_1, \\ &= \mathcal{R}_0 - \left[\frac{\epsilon_C \psi \alpha \beta_C (\eta_C K_5 + \sigma_C)}{K_1 K_3 K_5} + \epsilon_I \mu Q_1 \right], \end{aligned} \tag{29}$$

where, $Q_1 = \dfrac{\beta_I(\eta_I K_4 + \sigma_I)}{K_1 K_2 K_4} + \dfrac{\beta_C \alpha [K_4(\eta_C K_5 + \sigma_C) + \sigma_I K_3]}{\displaystyle\prod_{i=1}^{5} K_i}.$

The quantity, $\mathcal{R}_{0v}$, is the reproduction number of the vaccination model (1) for the case when every individual in the community is vaccinated [13, 24]. Following [12, 13], define the *vaccine impact* (Λ) by:

$$\Lambda = \frac{V_I^* + V_C^*}{N^*} \left(1 - \frac{\mathcal{R}_{0v}}{\mathcal{R}_0} \right). \tag{30}$$

We claim the following result.

Theorem 4.2. (i): *positive impact in the community if $\Lambda > 0$*
    ($\mathcal{R}_{0v} < \mathcal{R}_0$),
(ii): *no impact in the community if $\Lambda = 0$*
    ($\mathcal{R}_{0v} = \mathcal{R}_0$),
(iii): *negative (detrimental) impact in the community if $\Lambda < 0$*
    ($\mathcal{R}_{0v} > \mathcal{R}_0$).

Proof. Substituting (30) into (28) gives $\mathcal{R}_v = \mathcal{R}_0(1 - \Lambda)$. Thus, $1 - \Lambda = \frac{\mathcal{R}_v}{\mathcal{R}_0}$. Hence, $1 - \Lambda < 1$ whenever $\mathcal{R}_v < \mathcal{R}_0$ (so that, $\Lambda > 0$, and the vaccine has positive impact). Similarly, $1 - \Lambda > 1$ whenever $\mathcal{R}_v > \mathcal{R}_0$ (so that, $\Lambda < 0$, and, in this case, the vaccine has negative population-level impact). Finally, $1 - \Lambda = 1$ for $\mathcal{R}_v = \mathcal{R}_0$ (so that, $\Lambda = 0$, and the vaccine has no population-level impact). $\square$

It is worth noting from Equation (29) that the quantity $\mathcal{R}_0$ is always greater or equal to $\mathcal{R}_{0v}$. Thus, the vaccine will never have a detrimental impact (as suggested by Case (iii) of Theorem 4.2). The above result (Theorem 4.2) is numerically illustrated in Figure 5. Furthermore, it is worth noting from (28) that

$$\frac{\partial \mathcal{R}_v}{\partial f} = - \left[\frac{\mu \alpha \beta_C \epsilon_C \psi (\eta_C K_5 + \sigma_C)}{(K_8 + \omega_C) K_1 K_3 K_5} + \mu \epsilon_I Q_1 \right] < 0,$$

and,

$$\frac{\partial \mathcal{R}_v}{\partial \xi_C} = - \frac{\psi \alpha \beta_C (\eta_C K_5 + \sigma_C)[\omega + \mu(1 - f)]}{(K_8 + \omega_C) K_1 K_3 K_5} < 0.$$

Thus, the vaccination reproduction number ($\mathcal{R}_v$) decreases with increasing fraction of infants (f) or children (ξ_C) vaccinated. A decrease in $\mathcal{R}_v$ implies a decrease in disease burden (as measured in terms of new cases of measles and measles-induced mortality). Thus, these analyses show that the use of the anti-measles vaccine will always have positive population-level impact.

A contour plot of the reproduction threshold ($\mathcal{R}_v$), as a function of vaccine efficacy ($\epsilon = \epsilon_I = \epsilon_C$) and the fraction of individuals vaccinated at steady-state ($p = \frac{V_I^* + V_C^*}{N^*}$), is depicted in Figure 6. For the set of parameter values used in these simulations, the contours show a decrease in *vaccination reproduction number* ($\mathcal{R}_v$) with increasing vaccine efficacy (ϵ) and the fraction of susceptible infants and children vaccinated (p) at steady-state. It is clear from this plot that significantly high vaccine coverage would be needed to effectively control or eliminate the disease from the community (i.e., achieve $\mathcal{R}_v < 1$). In particular, with the assumed 95% vaccine efficacy [11, 20], about 90% of susceptible infants and children need to be vaccinated (at steady-state) to have a realistic chance for the effective control (or elimination) of measles in the community.

5. CONCLUDING REMARKS

A new age-structured deterministic model for the transmission dynamics of measles in a community is designed and rigorously analyzed. Some of the main findings of the study are:

(i): In the absence of vaccination, the resulting vaccination-free model undergoes the phenomenon of backward bifurcation. Two causes of this phenomenon (namely, children's natural immunity against measles infection and measles-induced

       mortality) have been identified. In the absence of disease-induced mortality, the disease-free equilibrium of the vaccination-free model is shown to be globally-asymptotically stable when the associated reproduction number of the vaccination-free model is less than unity.

(ii): The model without vaccination has a unique endemic equilibrium when its reproduction number exceeds unity. This equilibrium is shown to be globally-asymptotically stable for the special case where the disease-induced mortality is negligible ($\delta = 0$) and children do not acquire natural immunity against measles infection ($\psi = 1$).

(iii): The vaccination model also undergoes backward bifurcation, and, unlike in the vaccination-free model, this phenomenon persists even when children acquire permanent immunity against measles infection ($\psi = 0$). It is shown that this model does not undergo backward bifurcation if the vaccine efficacy is perfect (i.e., $\epsilon = 1$) and the measles-induced mortality is negligible ($\delta = 0$). For this case (with $\epsilon = 1$ and $\delta = 0$), the DFE of the model is shown to be globally-asymptotically stable when the associated reproduction threshold is less than unity.

(iv): The widespread use of the anti-measles vaccine always induces positive community-wide impact.

(v): With the assumed 95% efficacy of the currently-available anti-measles vaccines, at least 90% of the susceptible infants and children need to be vaccinated to have a realistic chance for the effective control or elimination of the disease in the community.

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NOMENCLATURE

Table 1: Description of parameters of the models (1) *and* (4)

Parameter	Interpretation
Π	Birth rate of infants
μ	Natural death rate
f	Fraction of infants vaccinated
β_I	Effective contact rate for infants
β_C	Effective contact rate for children
ξ_C	Continuous vaccination rate for children
ω_C	Vaccine waning rate
α	Maturation rate of infants
ψ	Modification parameter for the infection rate of children due to natural immunity
θ_C	Modification parameter for the reduced likelihood of infant acquiring infection from older child
θ_I	Modification parameter for the reduction of transmission rate of infants to children
$\eta_I,\ \eta_C$	Modification parameters for the reduction in infectiousness of exposed infants and children in comparison to symptomatic infant and children
$\sigma_I,\ \sigma_C$	Rate of development of clinical symptoms of measles for exposed infants and children
$\sigma_2,\ \sigma_3$	Hospitalization rate for infected infants and children
$\delta_I,\ \delta_2$	Disease-induced death rate for infants
$\delta_C,\ \delta_3$	Disease-induced death rate for children
$\epsilon_I,\ \epsilon_C$	Vaccine efficacy for infants and children
$\gamma_I,\ \gamma_C,\ \gamma_2,\ \gamma_3$	Recovery rate for infants and children

APPENDIX A: PROOF OF THEOREM 3.3

Proof. The proof is based on using centre manifold theory [9, 10]. Consider the vaccination-free model (4). Let $S_I = x_1$, $S_C = x_2$, $E_I = x_3$, $E_C = x_4$, $I_I = x_5$, $I_C = x_6$, $H_I = x_7$, $H_C = x_8$ and $R_I = x_9$. Thus, $N = \sum_{i=1}^{9} x_i$. Further, by using the vector notation $X = (x_1, x_2, x_3, x_4, x_5, x_6, x_7, x_8, x_9)^T$, the age-structured model (4) can be written in the form $\frac{dX}{dt} = (f_1, f_2, f_3, f_4, f_5, f_6, f_7, f_8, f_9)^T$ as follows (where K_i, $i = 1, ..., 7$ are as defined in Section 3).

$$\frac{dx_1}{dt} = \Pi - (\lambda_I + \theta_C\lambda_C)x_1 - K_1x_1,$$

$$\frac{dx_2}{dt} = \alpha x_1 - \psi(\theta_I\lambda_I + \lambda_C)x_2 - \mu x_2,$$

$$\frac{dx_3}{dt} = (\lambda_I + \theta_C\lambda_C)x_1 - K_2x_3,$$

$$\frac{dx_4}{dt} = \alpha x_3 + \psi(\theta_I\lambda_I + \lambda_C)x_2 - K_3x_4,$$

$$\frac{dx_5}{dt} = \sigma_I x_3 - K_4x_5, \qquad\qquad\qquad \text{(A.1)}$$

$$\frac{dx_6}{dt} = \alpha x_5 + \sigma_C x_4 - K_5x_6,$$

$$\frac{dx_7}{dt} = \sigma_2 x_5 - K_6x_7,$$

$$\frac{dx_8}{dt} = \sigma_3 x_6 + \alpha x_7 - K_7x_8,$$

$$\frac{dx_9}{dt} = \gamma_2 x_5 + \gamma_I x_7 - K_1x_9,$$

with the associated forces of infection given by

$$\lambda_I = \frac{\beta_I(\eta_I x_3 + x_5)}{\displaystyle\sum_{i=1}^{9} x_i} \text{ and } \lambda_C = \frac{\beta_C(\eta_C x_4 + x_6)}{\displaystyle\sum_{i=1}^{9} x_i}.$$

Consider the case with $\mathcal{R}_0 = 1$. Suppose, further, that $\beta_I = \beta_I^*$ is chosen as a bifurcation parameter. Solving for $\beta_I = \beta_I^*$ from $\mathcal{R}_0 = 1$ gives

$$\beta_I = \beta_I^* = \frac{K_1K_2K_4}{\mu(\eta_I K_4 + \sigma_I)} \times$$

$$\left(1 - \alpha\beta_C \left\{ \frac{\psi(\eta_C K_5 + \sigma_C)}{K_1K_3K_5} + \frac{\mu[K_4(\eta_C K_5 + \sigma_C) + \sigma_I K_3]}{\displaystyle\prod_{i=1}^{5} K_i} \right\} \right).$$

The Jacobian of the system (A.1), evaluated at the DFE ($\mathcal{E}_0$) with $\beta_I = \beta_I^*$ (denoted by J^*), is given by

$$
J^* = \begin{bmatrix}
-K_1 & 0 & -P_1 & -P_2 & -\dfrac{\beta_I^*\mu}{K_1} & -\dfrac{\beta_C\mu}{K_1} & 0 & 0 & 0 \\[2mm]
\alpha & -\mu & -P_3 & -P_4 & -\dfrac{\alpha\psi\beta_I^*}{K_1} & -\dfrac{\alpha\psi\beta_C}{K_1} & 0 & 0 & 0 \\[2mm]
0 & 0 & P_1 - K_2 & P_2 & \dfrac{\beta_I^*\mu}{K_1} & \dfrac{\beta_C\mu}{K_1} & 0 & 0 & 0 \\[2mm]
0 & 0 & P_3 + \alpha & P_4 - K_3 & \dfrac{\alpha\psi\beta_I^*}{K_1} & \dfrac{\alpha\psi\beta_C}{K_1} & 0 & 0 & 0 \\[2mm]
0 & 0 & \sigma_I & 0 & -K_4 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \sigma_C & \alpha & -K_5 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & \sigma_I & 0 & -K_6 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & \sigma_2 & \alpha & -K_7 & 0 \\
0 & 0 & 0 & 0 & \gamma_3 & 0 & \gamma_I & 0 & -K_1
\end{bmatrix},
$$

where $P_1 = \dfrac{\beta_I^*\eta_I\mu}{K_1}$, $P_2 = \dfrac{\beta_C\eta_C\mu}{K_1}$, $P_3 = \dfrac{\alpha\psi\beta_I^*\eta_I}{K_1}$ and $P_4 = \dfrac{\alpha\psi\beta_C\eta_C}{K_1}$.

The Jacobian (J^*) of the linearized system has a simple zero eigenvalue (with all other eigenvalues having negative real part). Hence, the centre manifold theory [9, 10, 28] can be used to analyse the dynamics of the system (A.1) around $\beta_I = \beta_I^*$.

Eigenvectors of $J^*\Big|_{\beta_I=\beta_I^*}$

For the case when $\mathcal{R}_0 = 1$, it can be shown that the J^* has a right eigenvector (corresponding to the zero eigenvalue), given by $\mathbf{w} = [w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8, w_9]^T$, where,

$$
w_1 = \frac{-\mu}{K_1^2}[\beta_I^*(\eta_I w_3 + w_5) + \beta_C(\eta_C w_4 + w_6)], \quad w_2 = \frac{\alpha}{\mu}\left(1 + \frac{\psi K_1}{\mu}\right)w_1,
$$

$$
w_3 = \frac{K_4}{\sigma_I}w_5, \quad w_4 = w_4, \quad w_5 = w_5, \quad w_6 = \frac{\sigma_C w_4 + \alpha w_5}{K_5}, \quad w_7 = \frac{\sigma_2 w_5}{K_6},
$$

$$
w_8 = \frac{\alpha w_7 + \sigma_3 w_6}{K_7}, \quad w_9 = \frac{\gamma_I w_7 + \gamma_3 w_5}{K_1}.
$$

$$\text{(A.2)}$$

Similarly, the components of the left eigenvector of J^* (corresponding to the zero eigenvalue), denoted by $\mathbf{v} = [v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8, v_9]$, are given by,

$$
v_3 = v_3, \quad v_4 = v_4, \quad v_5 = \frac{1}{K_1 K_4}\left(\beta_I^* + \frac{\alpha\beta_C}{K_5}\right)(\mu v_3 + v_4 \alpha\psi),
$$

$$
v_6 = \frac{\beta_C}{K_1 K_5}(\mu v_3 + v_4 \alpha\psi), \quad v_1 = v_2 = v_7 = v_8 = v_9 = 0.
$$

$$\text{(A.3)}$$

It is worth mentioning that the free eigenvectors w_4, w_5, v_3 and v_4 are chosen, respectively, to be

$$w_4 = 1/4,\ w_5 = \frac{1}{2A_1},\ v_3 = \frac{K_1 K_5^2}{\beta_C \sigma_C \mu}\ \text{and}\ v_4 = \frac{K_1 K_5^2}{K_1 K_5^2 + \beta_C \sigma_C \mu \alpha \psi},$$

where,

$$A_1 = \frac{\sigma_I K_5^2 (K_1 K_4 + \mu \beta_I^*) + \mu \alpha \beta_C K_1 K_5 (K_4 + K_5)}{\beta_C \sigma_C \sigma_I K_4} +$$
$$\frac{\alpha \psi [\beta_I^* K_5^2 + \alpha \beta_C (K_4 + K_5)]}{K_4 (K_1 K_5^2 + \beta_C \alpha \psi)},\ \text{so that}\ \mathbf{v.w} = 1\ \text{(in line with [10])}.$$

It can be shown, by computing the non-zero partial derivatives of the right-hand side functions, $f_i (i = 1, ..., 9)$, that the associated backward bifurcation coefficients, a and b, are given, respectively, by (see Theorem 4.1 in [10]):

$$a = \sum_{k,i,j=1}^{8} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0),$$

$$= \frac{2\mu}{\Pi K_1} [\beta_I^*(w_3 \eta_I + w_5) + \beta_C (w_4 \eta_C + w_6)][(w_1 \alpha - \mu w_2)(v_3 - \psi v_4)$$
$$- (\mu v_3 + \psi \alpha v_4)(w_3 + w_4 + w_5 + w_6 + w_7 + w_8 + w_9)],$$

$$= \frac{2\mu A_2}{\Pi K_1} \left\{ \frac{A_2 \psi}{K_1} \left(\frac{K_1 K_5^2}{\beta_C \sigma_C \mu} - \frac{K_1 K_5^2 \psi}{K_1 K_5^2 + \beta_C \sigma_C \mu \alpha \psi} \right) \right.$$
$$- \frac{(\mu + \psi \alpha)}{4 A_1 \sigma_I K_1 K_5 K_7} \left(K_1 K_7 \{ 2K_5 (K_4 + \sigma_I \sigma_C) + \sigma_I [\sigma_C A_1 + 2\alpha \right.$$
$$\left. + A_1 K_5 + \sigma_3 (\sigma_C A_1 + 2\alpha)] \} + 2\sigma_I K_5 [K_1 \alpha \sigma_2 + 2K_7 (\sigma_2 \gamma_I + \gamma_3)] \right) \bigg\},$$
$$\text{(A.4)}$$

where, $A_2 = \dfrac{\beta_I^*}{2 A_1 \sigma_I}(\eta_I K_4 + \sigma_I) + \dfrac{\beta_C}{4 A_1 K_5}[A_1 (\eta_C K_5 + \sigma_C) + \alpha]$, and,

$$b = \sum_{k,i=1}^{9} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta_I^*}(0,0) = \frac{1}{K_1}(v_3 \mu + v_4 \psi \alpha)(w_3 \eta_1 + w_5),$$

$$= \frac{K_5^2 (\eta_1 K_4 + \sigma_I)}{2 A_1 \sigma_I} \left(\frac{1}{\beta_C \sigma_C \mu} + \frac{\psi \alpha}{K_1 K_5^2 + \beta_C \sigma_C \mu \alpha \psi} \right).$$

Since the bifurcation coefficient, b, is automatically positive, it follows from Theorem 4.1 in [10] that the vaccination-free model (4)

(or its transformed equivalent (A.1)) will undergo backward bifurcation if the bifurcation coefficient, a, given by (A.4), is positive. $\square$

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DEPARTMENT OF MATHEMATICS AND APPLIED MATHEMATICS, UNIVERSITY OF PRETORIA, PRETORIA 0002, SOUTH AFRICA

E-mail address: `Salisu.Garba@up.ac.za`

DEPARTMENT OF MATHEMATICS, UNIVERSITY OF MANITOBA, WINNIPEG, MANITOBA, R3T 2N2, CANADA

E-mail address: `gumelab@cc.umanitoba.ca`

DEPARTMENT OF MATHEMATICS, BAYERO UNIVERSITY KANO, P.M.B. 3011, KANO, NIGERIA

E-mail address: `nhussaini.mth@buk.edu.ng`

Table 2: Parameter Values

Parameter	Nominal Value (*per* year)	References
Π	Variable	Assumed
$\dfrac{1}{\mu}$	78 years	[27]
f	0.85	[7]
ϵ_I	0.85	[7]
ϵ_C	0.95	[7]
β_I	0.09091	[27]
β_C	0.09091	[27]
ξ_C	70%	[7]
ω_C	1.5% *per* year	[7]
ρ	0.6	[27]
$\sigma_I,\ \sigma_2$	0.125	[27]
$\sigma_C,\ \sigma_3$	0.125	[27]
$\gamma_I,\ \gamma_2$	0.14286	[27]
$\gamma_C,\ \gamma_3$	0.14286	[27]
$\gamma_I,\ \gamma_C$	0.14286	[27]
$\delta_I,\ \delta_C,\ \delta_2,\ \delta_3$	0.0000351	[27]
θ_I	1	[27]
θ_C	1	[27]
η_I, η_C	$[0, 1]$	
$\tau_1,\ \tau_v$	0.09	Assumed
ψ	[0,1]	Assumed
α	0.025	Assumed

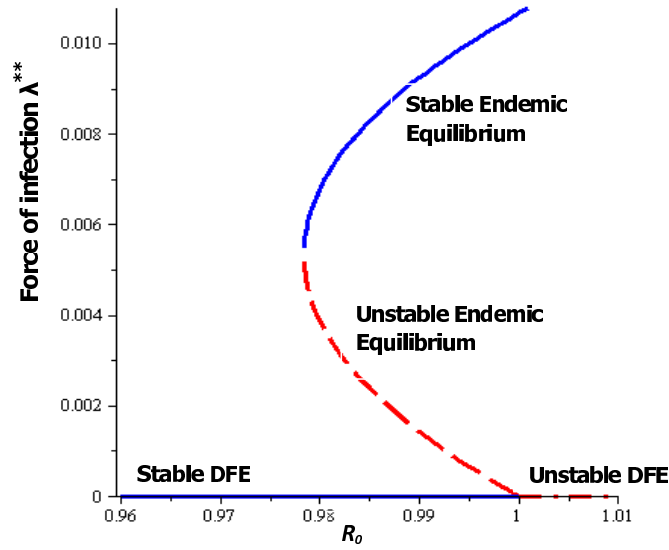FIGURE 1. Schematic diagram of the vaccination model (1).

FIGURE 2. Bifurcation diagram for the vaccination-free model (4). Parameter values used are: $\Pi = 1000$, $\mu = 0.00004$, $\psi = 0.54$, $\alpha = 0.025$, $\eta_I = 0.65$, $\eta_C = 0.5$, $\beta_I = 0.9$, $\beta_C = 0.9$, $\delta_I = 0.0599$, $\delta_C = 0.45$, $\gamma_I = 0.5$, $\gamma_C = 0.5$, $\gamma_2 = 0.25$, $\gamma_3 = 0.25$, $\sigma_I = 0.853$, $\sigma_C = 0.5$, $\sigma2 = 0.853$, $\sigma_3 = 0.52$ (so that, $\mathcal{R}_0 = 0.9832984689 < 1$, and $a = 0.000002779040026 > 0$).
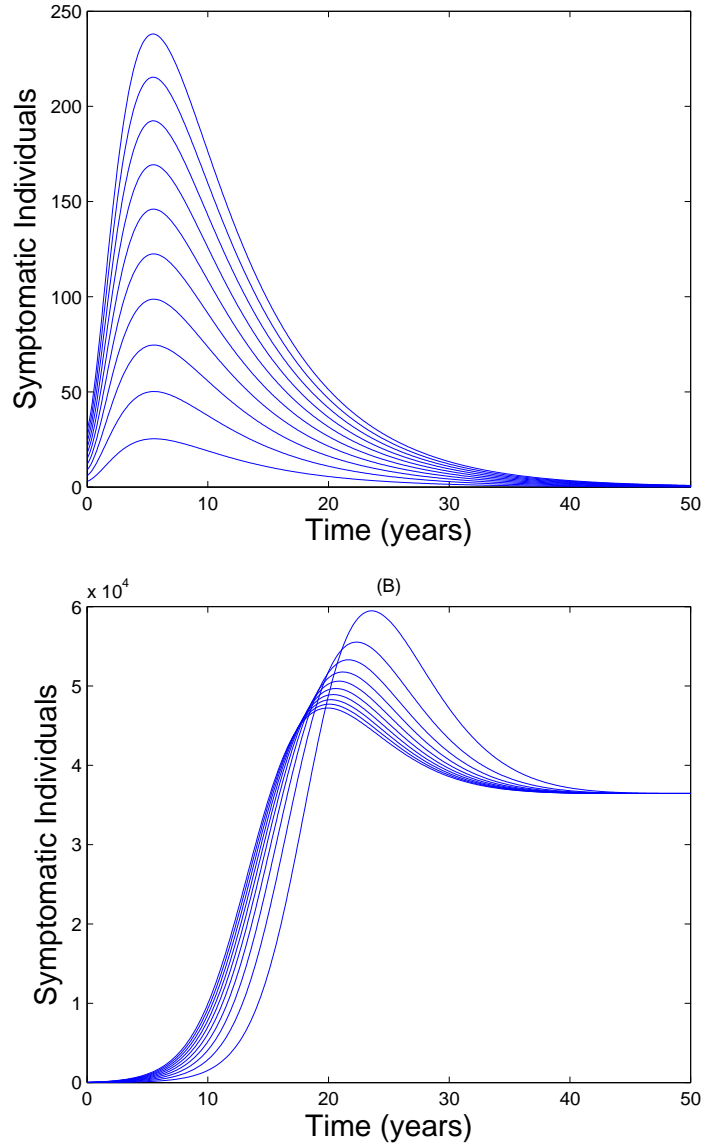
FIGURE 3. Simulations of the vaccination-free models (A) (4) and (B) (23), showing the total number of symptomatic individuals $(I_I(t) + I_C(t))$ as a function of time. Parameter values used are as in Table 2, with (A) $\beta_I = 0.7$ and $\beta_C = 0.085$ (so that, $\mathcal{R}_0 = 0.4150 < 1$) and (B) $\beta = 0.7$ (so that, $\mathcal{R}_{0s}^m = 3.4182 > 1$).
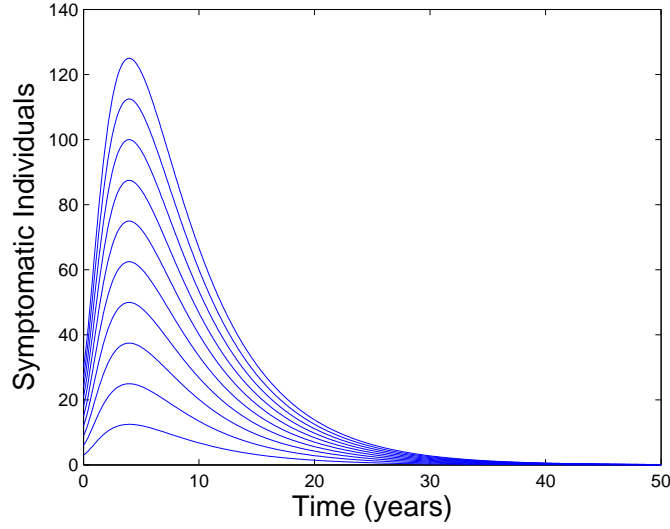
FIGURE 4. Simulations of the vaccination model (1), showing the total number of symptomatic individuals $(I_I(t) + I_C(t))$ as a function of time. Parameter values used are as in Table 2, with $\delta = 0$, $\epsilon = 1$, $\beta_I = 0.07$ and $\beta_C = 0.7$ (so that, $\mathcal{R}_v^m = 0.3418 < 1$).
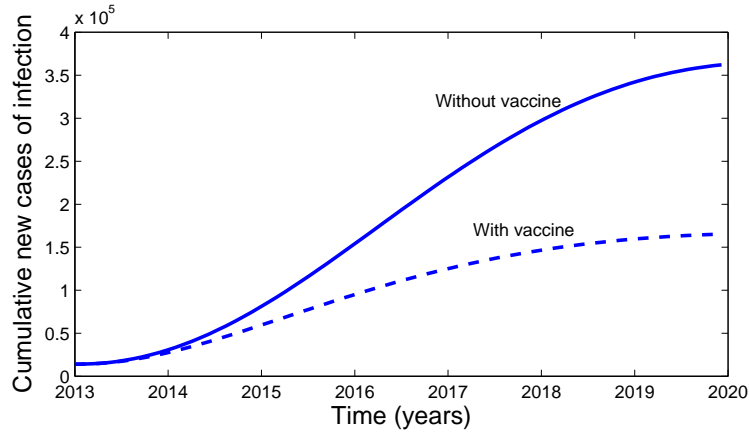

FIGURE 5. Simulations of the vaccination model (1), showing the cumulative number of symptomatic cases of measles infection as a function of time in the presence or absence of routine measles vaccination. Parameter values used are as given in Table 2, with $\epsilon_I = 0.9$, $\epsilon_C = 0.9$, $\omega_C = 0.25$, $\xi = 0.80$ and $f = 0.75$ (so that, $\Lambda = 0.7572 > 0$, and the vaccine has a positive impact).
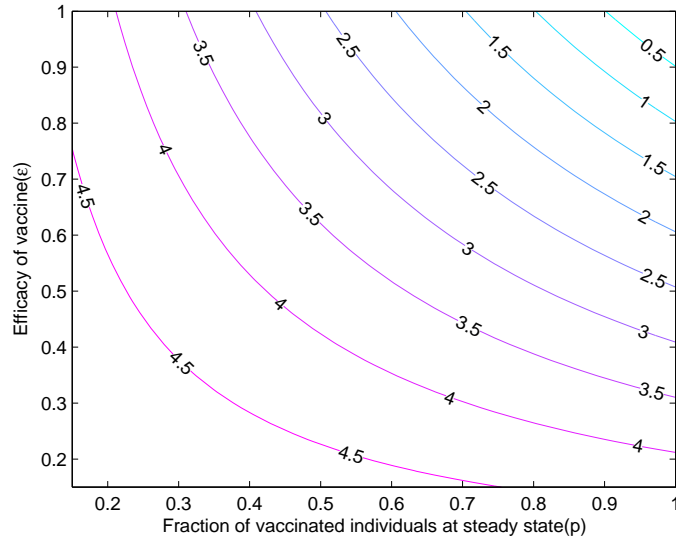
FIGURE 6. Simulations of the vaccination model (1), showing a contour plot of $\mathcal{R}_v$ as a function of the fraction of vaccinated individuals at steady-state (p) and vaccine efficacy (ϵ). Parameter values used are as given in Table 2, with $\delta = 0$.