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Controllability of Infections in SIR Models with Waned Childhood Vaccination-Induced Immunity and Booster Vaccination

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ABSTRACT

The aim of this paper is to study the controllability of infections represented by SIR endemic models with interventions based on vaccination and the impact of disease-induced reduction in contact-activity on the efforts required to eliminate the infection. Two kinds of vaccination-interventions are considered. The first is the routine-immunization, where a proportion p of newborns gets vaccinated immediately after birth and their immunity wanes overtime, while the other is to vaccinate those whose immunity acquired by routine-immunization waned and became susceptible again. The model analysis shows that the earlier the admission of booster vaccination is, the better the chance to eliminate the infection is. The analysis shows further that the higher the reduction in the contact activity of infected individuals is, the lower the booster vaccination rate needed to ensure an effective control of the infection is and, consequently, the less the minimum effort required to eliminate the infection is.

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1 Introduction

Due to the significant impact of infectious diseases on population health and economy, there is a big public health interest in controlling, eliminating, or even eradicating them. Among the various strategies used to control infectious diseases is vaccine administration (WHO, 2012). Globally, vaccines are routinely given to newborns as a way to implement a herd immunity that would prevent the occurrence of certain infections outbreak (e.g., diphtheria, tetanus, pertussis, poliomyelitis, measles, mumps, rubella). If available and totally effective, vaccines are also used to eradicate certain infections (e.g., smallpox) (Henderson, 2014). However, vaccines are rarely fully protective and the immunity acquired by vaccination does not always last for life, but it sometimes wanes and individuals become susceptible again, especially for inactivated vaccines (e.g., Tetanus and also the case of killed measles vaccine). Therefore, booster shots are recommended to restore waning immunity (WHO, 2012). As we don't live in ideal communities and (sometimes) due to the limited supply of vaccines, not every individual in the population receives vaccines. Therefore, assessing the minimum vaccination coverage needed to effectively protect the population from infection's outbreak is of great concern. To this end, mathematical models are used to imitate and have better understanding of the dynamics and to gain informative deductions (Brauer and Castillo-Chavez, 2012; Hadeler et al., 2016; Safan et al., 2006).

Mathematical models have been used to study the possible elimination of infectious diseases with strategies based on vaccination. For example, Anderson and May (1982) used an SEIR model with age-dependent parameters to study the controllability of directly transmitted infections with vaccination. Castillo-Chavez and Feng (1996) considered an age-structure population model to study optimal vaccination strategies for tuberculosis, with two vaccination programs aimed at determining the optimal age or ages at which an individual should be vaccinated. Makinde (2007) considered an age-independent SIR model for a varying population size with constant vaccination strategy. These papers and many others (e.g., Anderson and May, 1985; Hadeler and Müller, 2007; Makinde, 2007; Safan, 2020) consider totally perfect vaccines, where they confer life-long immunity. However, as the vaccine-acquired-immunity declines/wanes to below protection levels (or even disappears) over time, vaccinated individuals may become susceptible again and get reinfected, but with different susceptibility (Cai et al., 2018; Hadeler and Castillo-Chavez, 1995; Hadeler and van den Driessche, 1997; Liu et al., 2008; Mossong et al., 1999; Safan and Dietz, 2009; Safan and Rihan, 2014;

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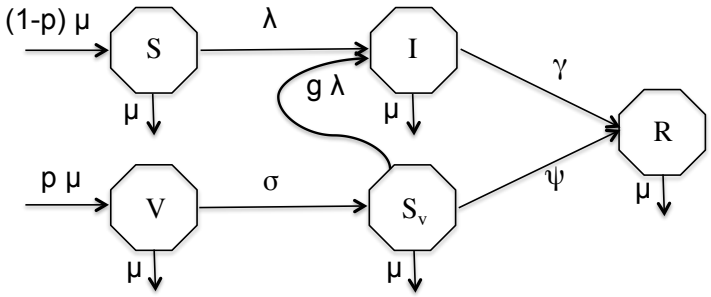

Figure 1: Schematic diagram for the transition between states of the model with waning vaccine-induced immunity and booster vaccination.

Safan et al., 2013; Safan, 2019). Therefore, booster vaccination strategies are used to help contain the infection (Glass and Grenfell, 2003).

Individual's immunity is boosted either due to exposure to natural infections (Barbarossa and Röst, 2015; Lavine et al., 2011; Leung et al., 2018) or due to booster vaccination dosage(s). This work focuses on boosting individual's immunity by vaccination, where an SIR endemic model for a demographically stationary population is considered. It is assumed in this work that the immunity acquired by childhood vaccination (i.e., vaccination given at birth for a proportion p of newborns) wanes over time, while the immunity acquired after recovery from the infection is permanent (e.g., the case of measles) (Christenson and Böttiger, 1994). Moreover, individuals whose childhood-vaccine-acquired immunity declined and became susceptible again will be vaccinated at some rate (say, ψ) and that booster vaccination confers immunity for their rest of life. It is aimed here to study the possibility to eliminate the infection with vaccination solely and find the critical booster vaccination rate above which the infection washes out. We further extend the model to study the impact of including reduction in contact activity on the models dynamics and on reducing the minimum effort needed in the infection elimination process.

The manuscript is organized as follows. Section 2 includes the model formulation and its equilibrium and stability analyses. Extended model to include disease-induced reduction in contact activity is shown in Section 3. The possibility to control the infection with strategies based solely on vaccination has been discussed in Section 4. A summary of the main results and conclusion is given in Section 5.

2 Model Formulation

Consider a demographically stationary population, in the sense that its size remains fixed with respect to time t. This population is stratified into five mutually exclusive independent categories according to the individuals epidemiological status, namely, naive susceptible, vaccinated, vaccinated susceptible, infected and recovered. It is assumed that newborns are all (naive) susceptible and the birth rate as well as the natural death rate for all classes is μ. Moreover, a proportion p of the newborns is assumed to get vaccinated immediately after birth. This childhood vaccine induces temporary immunity.

In its duration, the immunity acquired by vaccination varies from vaccine/disease to another. Not always do vaccines induce lifelong immunity, but it may last for a specific period of time after which it wanes. In this work, we assume that the immunity acquired by childhood vaccination wanes, with a duration of immunity $1/\sigma$ time units, while the natural immunity (i.e., immunity acquired by recovery after experiencing the infection) is assumed to last for life. Consequently, we differentiate between vaccinated (denoted by V) and recovered individuals (denoted by R). We assume that individuals whose immunity wanes become susceptible again, denoted by S_v. Therefore, we differentiate between naive susceptible individuals S (i.e., individuals who have neither acquired the infection nor received vaccine) and vaccinated-susceptible individuals S_v (i.e., those who lost their childhood vaccine-induced immunity and became susceptible again), where it is assumed that the relative susceptibility of S_v-individuals with respect to S-individuals is $g \in [0, 1]$. It is assumed further that naive susceptible individuals acquire the infection due to successful contacts with infected ones (denoted by I) at rate $\lambda(t)$, while S_v individuals acquire the infection at rate $g\lambda(t)$. Moreover, S_v individuals may boost their immunity by getting vaccinated at rate ψ and acquire immunity for the rest of their life, while infected individuals recover naturally from the infection at rate γ. The transition diagram between model states is shown in figure 1.

It is worth noting that the state variables S, V, S_v, I and R represent proportions of subpopulations so that $S+V+S_v+I+R = 1$. In the standard incidence setting and for a homogeneously mixing population, the force of infection $\lambda(t) = \beta I(t)$, where β is

the successful contact rate between naive susceptible and infected individuals. Therefore, the model reads

$$\begin{aligned}
\frac{dS}{dt} &= (1-p)\mu - \beta SI - \mu S,\\
\frac{dV}{dt} &= p\mu - (\sigma+\mu)V,\\
\frac{dS_v}{dt} &= \sigma V - g\beta S_v I - (\psi+\mu)S_v,\\
\frac{dI}{dt} &= \beta SI + g\beta S_v I - (\gamma+\mu)I,\\
\frac{dR}{dt} &= \gamma I + \psi S_v - \mu R,
\end{aligned} \tag{1}$$

with initial conditions $S(0), V(0), S_v(0), I(0), R(0)$ and $S(t)+V(t)+S_v(t)+I(t)+R(t)=1$. Model (1) is defined on the set

$$\Omega = \left\{(S,V,S_v,I,R)\in\mathbb{R}^5_+ : 0\le S,V,S_v,I,R\le 1,\ S(t)+V(t)+S_v(t)+I(t)+R(t)=1\right\}. \tag{2}$$

The following proposition results on the basic properties of model (1). Its proof is deferred to Appendix A.

Proposition 1. *The set Ω is positively invariant and attracts all solutions in $\mathbb{R}^5_+$. Moreover, for any nonnegative initial conditions $\big(S(0),V(0),S_v(0),I(0),R(0)\big)\in\Omega$, the solution set $\big(S(t),V(t),S_v(t),I(t),R(t)\big)$ of the system (1) remains positive for all $t>0$. Also, Model (1) has a unique solution.*

2.1 Equilibrium and stability analyses

2.1.1 Infection-free equilibrium and its local stability

The equilibrium analysis of model (1) shows that it has an infection-free equilibrium $E_0 = (S_0, V_0, S_{v0}, I_0, R_0)'$, where the prime ($'$) means vector transpose, whose components are given by

$$S_0 = 1-p, \qquad V_0 = \frac{p\mu}{\sigma+\mu}, \qquad S_{v0} = \frac{p\mu\sigma}{(\mu+\psi)(\sigma+\mu)}, \qquad I_0 = 0, \qquad R_0 = \frac{p\psi\sigma}{(\mu+\psi)(\sigma+\mu)}. \tag{3}$$

Moreover, the Jacobian matrix of the right hand side of model (1), evaluated at the infection-free equilibrium E_0, reads

$$J_{E_0} = \begin{pmatrix}
-\mu & 0 & 0 & -(1-p)\beta & 0\\
0 & -(\sigma+\mu) & 0 & 0 & 0\\
0 & \sigma & -(\mu+\psi) & -g\beta S_{v0} & 0\\
0 & 0 & 0 & (1-p)\beta + g\beta S_{v0} - (\gamma+\mu) & 0\\
0 & 0 & \psi & \gamma & -\mu
\end{pmatrix}. \tag{4}$$

It is clear that J_{E_0} has the four negative eigenvalues $-\mu, -\mu, -(\sigma+\mu), -(\psi+\mu)$ in addition to the fifth eigenvalue

$$\beta\left(1-p+gp\frac{\sigma}{\sigma+\mu}\times\frac{\mu}{\mu+\psi}\right)-(\gamma+\mu)$$

which is negative if and only if the vaccine-control reproduction number $\mathcal{R}_\psi<1$, where

$$\mathcal{R}_\psi = \frac{\beta}{\gamma+\mu}\left(1-p+gp\frac{\sigma}{\sigma+\mu}\times\frac{\mu}{\mu+\psi}\right). \tag{5}$$

In summary, we show the following proposition.

Proposition 2. *Model (1) has an infection-free equilibrium $E_0 = (S_0, V_0, S_{v0}, I_0, R_0)'$ whose components are defined in (3). This equilibrium is locally asymptotically stable if and only if the vaccine-control reproduction number $\mathcal{R}_\psi<1$.*

2.1.2 Endemic equilibrium and its local stability analysis

In model (1), the S-, V-, and S_v-equations at equilibrium imply, respectively, that

$$\bar{S} = \frac{(1-p)\mu}{\mu+\beta\bar{I}}, \qquad \bar{V} = \frac{p\mu}{\sigma+\mu}, \qquad \bar{S}_v = \frac{\sigma\bar{V}}{\psi+\mu+g\beta\bar{I}}. \tag{6}$$

However, the I-equation at equilibrium implies, for $\bar{I} \neq 0$, that

$$\frac{\gamma+\mu}{\beta} = \bar{S} + g\bar{S}_v. \tag{7}$$

Now, we use (6) in (7) to get the characteristic epidemiological equation

$$\frac{(1-p)\mu}{\mu+\beta\bar{I}} = \frac{\gamma+\mu}{\beta} - \frac{gp\sigma\mu}{(\sigma+\mu)(\psi+\mu+g\beta\bar{I})}. \tag{8}$$

The left hand side of equation (8) is monotonically decreasing in the endemic prevalence of infection $\bar{I}$, while its right hand side is monotonically increasing in $\bar{I}$. Therefore, equation (8) has a unique solution if and only if

$$1-p > \frac{\gamma+\mu}{\beta} - \frac{gp\sigma\mu}{(\sigma+\mu)(\psi+\mu)}$$

which is equivalent to having $\mathcal{R}_\psi > 1$. Once we obtain a solution $\bar{I}$ of (8), we substitute in (6) to get the proportions $\bar{S}, \bar{S}_v$ and $\bar{R}$ in the endemic situation. Now, to find the endemic prevalence of infection, we rewrite (8) in the form

$$A_1(\beta\bar{I})^2 + B_1(\beta\bar{I}) + C_1 = 0 \tag{9}$$

where

$$\begin{aligned} A_1 &= g(\gamma+\mu)(\sigma+\mu), \\ B_1 &= (\gamma+\mu)(\sigma+\mu)(\psi+\mu+g\mu) - g\mu\beta\Big(p\sigma + (1-p)(\sigma+\mu)\Big), \\ C_1 &= \mu(\gamma+\mu)(\sigma+\mu)(\psi+\mu) - \mu\beta\Big(gp\sigma\mu + (1-p)(\psi+\mu)(\sigma+\mu)\Big). \end{aligned} \tag{10}$$

Therefore,

$$\bar{I} = \frac{-B_1 + \sqrt{B_1^2 - 4A_1C_1}}{2\beta A_1}$$

where the discriminant $B_1^2 - 4A_1C_1$ (see Appendix B for detailed derivation) is given by

$$B_1^2 - 4A_1C_1 = \Big((\gamma+\mu)(\sigma+\mu)(\psi+(1-g)\mu) + g\mu\beta((1-p)(\sigma+\mu) - p\sigma)\Big)^2 + 4p(1-p)g^2\mu^2\beta^2\sigma(\sigma+\mu). \tag{11}$$

Therefore, we state the following proposition.

Proposition 3. *Model* (1) *has a unique endemic equilibrium* $E_e = (\bar{S}, \bar{V}, \bar{S}_v, \bar{I}, \bar{R})'$ *if and only if* $\mathcal{R}_\psi > 1$.

To establish the local stability of the endemic equilibrium E_e, we compute the Jacobian matrix J_e of the system (1) at E_e. The computations show that J_e has the two negative eigenvalues $-\mu$ and $-(\sigma+\mu)$ in addition to the eigenvalues of the sub-matrix

$$J_{sub} = \begin{pmatrix} -(\mu+\beta\bar{I}) & 0 & -\beta\bar{S} \\ 0 & -(\mu+\psi+g\beta\bar{I}) & -g\beta\bar{S}_v \\ \beta\bar{I} & g\beta\bar{I} & 0 \end{pmatrix}. \tag{12}$$

The matrix J_{sub} has the characteristic polynomial

$$P(\rho) = \rho^3 + a_2\rho^2 + a_1\rho + a_0 \tag{13}$$

where ρ denotes the eigenvalue and

$$\begin{aligned} a_2 &= (\mu+\beta\bar{I}) + (\mu+\psi+g\beta\bar{I}) > 0, \\ a_1 &= (g\beta\bar{I})(g\beta\bar{S}_v) + (\mu+\beta\bar{I})(\mu+\psi+g\beta\bar{I}) + (\beta\bar{I})(\beta\bar{S}) > 0, \\ a_0 &= (g\beta\bar{I})(g\beta\bar{S}_v)(\mu+\beta\bar{I}) + (\beta\bar{I})(\beta\bar{S})(\mu+\psi+g\beta\bar{I}) > 0. \end{aligned}$$

It is clear that the characteristic polynomial (13) is cubic with positive coefficients. Therefore, the Routh-Hurwitz criterion (see Theorem 1.4 of Hadeler, 2017) implies that $P(\rho) = 0$ has negative roots if and only if the Hurwitz determinants, given explicitly

by $\Delta_1 = a_2$, $\Delta_2 = a_2a_1 - a_0$ and $\Delta_3 = a_0\Delta_2$, are all positive. Since the coefficients a_0, a_1 and a_2 are all positive, then the Hurwitz determinants are positive if and only if $\Delta_2 > 0$. To this end, we compute

$$\begin{aligned}\Delta_2 &= \Big((\mu+\beta\bar{I})+(\mu+\psi+g\beta\bar{I})\Big)\Big((g\beta\bar{I})(g\beta\bar{S}_v)+(\mu+\beta\bar{I})(\mu+\psi+g\beta\bar{I})+(\beta\bar{I})(\beta\bar{S})\Big)\\ &\quad-(g\beta\bar{I})(g\beta\bar{S}_v)(\mu+\beta\bar{I})-(\beta\bar{I})(\beta\bar{S})(\mu+\psi+g\beta\bar{I})\\ &= (\mu+\beta\bar{I})(\mu+\psi+g\beta\bar{I})\Big((\mu+\beta\bar{I})+(\mu+\psi+g\beta\bar{I})\Big)+(\beta\bar{I})(\beta\bar{S})(\mu+\beta\bar{I})\\ &\quad+(g\beta\bar{I})(g\beta\bar{S}_v)(\mu+\psi+g\beta\bar{I})\\ &> 0.\end{aligned}$$

Hence, the endemic equilibrium E_e is locally asymptotically stable whenever exists. Thus, we show the following proposition.

Proposition 4. *The unique endemic equilibrium* $E_e = (\bar{S}, \bar{V}, \bar{S}_v, \bar{I}, \bar{R})'$ *is locally asymptotically stable whenever exists.*

3 Effect of Disease-Induced Reduction in Contact Activity

Assume now that infected individuals reduce their contact activity with a proportion $P_r \in [0,1]$, see Feng and Thieme (1983); Safan (2020). Then the number of individuals available to mingle together at time t is $N(t) - P_rI(t)N(t)$. Also, the probability that a non-infected individual has a contact with an infected one is $(1-P_r)I\,/\,(1-P_rI(t))$. Therefore, the rate at which susceptible individuals S acquire the infection is

$$\lambda_S = \frac{(1-P_r)\beta I}{1-P_rI(t)} \tag{14}$$

while that of susceptible vaccinated individuals is $g\lambda_S$. Therefore, model (1) modifies to

$$\begin{aligned}\frac{dS}{dt} &= (1-p)\mu - \frac{(1-P_r)\,\beta\,SI}{1-P_r\,I} - \mu S,\\ \frac{dV}{dt} &= p\mu - (\sigma+\mu)V,\\ \frac{dS_v}{dt} &= \sigma V - \frac{g(1-P_r)\,\beta\,S_vI}{1-P_r\,I} - \psi S_v - \mu S_v,\\ \frac{dI}{dt} &= \frac{(1-P_r)\,\beta\,SI}{1-P_r\,I} + \frac{g(1-P_r)\,\beta\,S_vI}{1-P_r\,I} - (\gamma+\mu)I,\\ \frac{dR}{dt} &= \gamma I + \psi S_v - \mu R,\end{aligned} \tag{15}$$

with initial conditions $S(0)$, $V(0)$, $S_v(0)$, $I(0)$, $R(0)$ and $S + V + S_v + I + R = 1$. The model is still defined on the positively invariant set Ω:

$$\Omega = \Big\{(S,V,S_v,I,R) \in \mathbb{R}^5_+ : 0 \le S,V,S_v,I,R \le 1,\ S(t)+V(t)+S_v(t)+I(t)+R(t) = 1\Big\}. \tag{16}$$

Since $P_rI < 1$, then the right hand side of (15) is continuous and differentiable in the state variables, hence, it is locally Lipschitz. Thus, model (15) has a unique time-dependent solution.

The equilibrium analysis of model (15) shows that it has the infection-free equilibrium

$$E_{0,P_r} = \left(1-p, \frac{p\mu}{\sigma+\mu}, \frac{p\sigma}{\sigma+\mu}\times\frac{\mu}{\psi+\mu}, 0, \frac{p\sigma}{\sigma+\mu}\times\frac{\psi}{\psi+\mu}\right)' \tag{17}$$

which exists under no constraint. Linearizing model (15) about the infection-free equilibrium E_{0,P_r} and computing the eigenvalues of the Jacobian matrix J at E_{0,P_r} reveal that J has the four negative eigenvalues $-\mu$, $-\mu$, $-(\sigma+\mu)$, $-(\psi+\mu)$ in addition to the fifth eigenvalue

$$(1-P_r)\beta\left(1-p+g\frac{p\sigma}{\sigma+\mu}\times\frac{\mu}{\psi+\mu}\right) - (\gamma+\mu)$$

which is negative if and only if the control reproduction number in the presence of self-contactivity-reduction satisfies $\mathcal{R}_{P_r} < 1$, where

$$\mathcal{R}_{P_r} = (1-P_r)\times\frac{\beta}{\gamma+\mu}\times\left(1-p+gp\frac{\sigma}{\sigma+\mu}\times\frac{\mu}{\psi+\mu}\right) = (1-P_r)\mathcal{R}_\psi. \tag{18}$$

Hence, we show the following proposition.

Proposition 5. *Model* (15) *has an infection-free equilibrium* E_{0,P_r}*, given by* (17)*. It is locally asymptotically stable if and only if the control reproduction number in the presence of self-contactivity-reduction* $\mathcal{R}_{P_r}$ *is less than one.*

The equilibrium analysis shows further that model (15) has a unique endemic equilibrium, whose existence is constrained by the satisfaction of the inequality $\mathcal{R}_{P_r} > 1$. This is proven by assuming that at equilibrium $I \neq 0$. Hence, the equilibrium force of infection $\bar{\lambda}_S \neq 0$. Now, we put the derivatives in the left hand side of (15) equal zero and solve with respect to the state variables at equilibrium to get

$$\widetilde{S} = \frac{(1-p)\mu}{\bar{\lambda}_S + \mu}, \qquad \widetilde{V} = \frac{p\mu}{\sigma+\mu}, \qquad \widetilde{S}_V = \frac{p\mu}{\sigma+\mu} \times \frac{\sigma}{g\bar{\lambda}_S + \psi + \mu}, \qquad \widetilde{R} = 1 - (\widetilde{S} + \widetilde{V} + \widetilde{S}_v + \widetilde{I}) \tag{19}$$

and

$$\widetilde{I} = \frac{\bar{\lambda}_S}{\gamma+\mu}\left(\frac{(1-p)\mu}{\bar{\lambda}_S+\mu} + \frac{p\mu}{\sigma+\mu} \times \frac{g\sigma}{g\bar{\lambda}_S + \psi + \mu}\right) \tag{20}$$

where

$$\bar{\lambda}_S = \frac{(1-P_r)\beta\widetilde{I}}{1-P_r\widetilde{I}}. \tag{21}$$

Now, we substitute from (20) into (21) to get (for $\bar{\lambda}_S \neq 0$)

$$1 - \frac{P_r\mu}{\gamma+\mu}\left(\frac{(1-p)\bar{\lambda}_S}{\bar{\lambda}_S+\mu} + \frac{p\sigma}{\sigma+\mu} \times \frac{g\bar{\lambda}_S}{g\bar{\lambda}_S + \psi + \mu}\right) = \frac{(1-P_r)\mu\,\beta}{\gamma+\mu}\left(\frac{1-p}{\bar{\lambda}_S+\mu} + \frac{p\sigma}{\sigma+\mu} \times \frac{g}{g\bar{\lambda}_S + \psi + \mu}\right). \tag{22}$$

Once a solution $\bar{\lambda}_S \in [0, \infty)$ for equation (22) is obtained, we substitute in (19) and (20) to get the equilibrium components. It is clear that both sides of (22) are monotonically decreasing in $\bar{\lambda}_S$, given that all model parameters are kept fixed. However, the left hand side of (22) tends to

$$1 - \frac{P_r\mu}{\gamma+\mu}\left(1 - p + \frac{p\sigma}{\sigma+\mu}\right)$$

when $\bar{\lambda}_S$ tends to ∞, while its right hand side approaches zero as $\bar{\lambda}_S$ approaches ∞. Hence, a unique solution for (22) exists if and only if the value of the left hand side of (22) at $\bar{\lambda}_S = 0$ is less than the value of its right hand side at $\bar{\lambda}_S = 0$, while otherwise equation (22) has no feasible solution. Thus, model (15) has a unique endemic equilibrium if and only if

$$1 < \frac{(1-P_r)\,\beta}{\gamma+\mu}\left(1 - p + \frac{p\sigma}{\sigma+\mu} \times \frac{g\mu}{\psi+\mu}\right).$$

which is equivalent to having $\mathcal{R}_{P_r} > 1$. In summary, we show the following proposition.

Proposition 6. *Model* (15) *has a unique endemic equilibrium whose components are given by* (19) *and* (20)*. It does exist if and only if the control reproduction number in the presence of contact-activity-reduction* $\mathcal{R}_{P_r}$ *is bigger than one.*

In order to investigate the possible existence of endemic equilibria for $\mathcal{R}_{P_r} < 1$, we rewrite equation (22) in the polynomial form

$$G(\beta, \bar{\lambda}_S) := A_2\bar{\lambda}_S^2 + B_2\bar{\lambda}_S + C_2 = 0 \tag{23}$$

with

$$\begin{aligned}
A_2 &= g\Big((\gamma+\mu)(\sigma+\mu) - P_r\mu(p\sigma + (1-p)(\sigma+\mu))\Big),\\
B_2 &= (\gamma+\mu)(\sigma+\mu)(\psi+\mu+g\mu) - P_r\mu\Big(gp\sigma\mu + (1-p)(\sigma+\mu)(\psi+\mu)\Big) - g(1-P_r)\mu\beta(p\sigma + (1-p)(\sigma+\mu)),\\
C_2 &= \mu(\gamma+\mu)(\sigma+\mu)(\psi+\mu) - (1-P_r)\mu\beta\Big(gp\sigma\mu + (1-p)(\sigma+\mu)(\psi+\mu)\Big).
\end{aligned}$$

Equation (23) could be seen as a bifurcation equation in β and $\bar{\lambda}_S$. It has a bifurcation point $P_0 = (\beta^c_{P_r}, 0)$ in the plane $(\beta, \bar{\lambda}_S)$, where

$$\beta^c_{P_r} = \frac{(\gamma+\mu)(\sigma+\mu)(\psi+\mu)}{(1-P_r)\Big((1-p)(\sigma+\mu)(\psi+\mu) + gp\sigma\mu\Big)}. \tag{24}$$

Assuming that all model parameters, except β, are fixed, then

$$\frac{d\bar{\lambda}_S}{d\beta} = -\frac{\partial G/\partial\beta}{\partial G/\partial\bar{\lambda}_S}. \tag{25}$$

At the point $P_0 = (\beta^c_{P_r}, 0)$, we have (see Appendix C for a complete derivation)

$$\begin{aligned}
&\left.\frac{\partial G}{\partial\beta}\right|_{(\beta^c_{P_r},0)} = -(1-P_r)\mu\big(gp\sigma\mu + (1-p)(\sigma+\mu)(\psi+\mu)\big) < 0,\\
&\left.\frac{\partial G}{\partial\bar{\lambda}_S}\right|_{(\beta^c_{P_r},0)} \geq \frac{p\sigma(g\mu)^2((\gamma+\mu)(\sigma+\mu)-p\sigma\mu)}{gp\sigma\mu + (1-p)(\sigma+\mu)(\psi+\mu)}\\
&\qquad + \frac{gp(1-p)\sigma\mu^2(\sigma+\mu)(\psi+\mu)}{gp\sigma\mu + (1-p)(\sigma+\mu)(\psi+\mu)}\left(\left(\frac{\gamma}{gp\mu}\times\frac{\sigma+\mu}{\sigma}\times\frac{\psi+\mu}{\mu}-1\right)+\left(\frac{\sigma+\mu}{g\sigma}\times\frac{\psi+\mu}{\mu}-1\right)\right) > 0.
\end{aligned} \tag{26}$$

Hence,

$$\left.\frac{d\bar{\lambda}_S}{d\beta}\right|_{(\beta^c_{P_r},0)} > 0.$$

Therefore, the bifurcation direction is forward (i.e., supercritical) at $(\beta^c_{P_r}, 0)$. Thus, model (15) doesn't have endemic equilibria for $\mathcal{R}_{\mathcal{B}_r} < 1$, but has a unique endemic equilibrium for $\mathcal{R}_{\mathcal{B}_r} > 1$. This unique endemic equilibrium corresponds to the unique feasible solution of (23), which is given by

$$\bar{\lambda}_S = \frac{-B_2 + \sqrt{B_2^2 - 4A_2C_2}}{2A_2}. \tag{27}$$

The following proposition summarizes the above results.

Proposition 7. *In the plane $(\beta, \bar{\lambda}_S)$ there is a bifurcation point $P_0 = (\beta^c_{P_r}, 0)$, at which the bifurcation is forward (supercritical). The model doesn't exhibit backward bifurcation and no endemic equilibrium exists for $\mathcal{R}_{\mathcal{B}_r} < 1$.*

4 Controllability of the Infection

In the absence of any type of vaccination (i.e., $p = 0$ and $\psi = 0$), model (1) reduces to the standard SIR model and the infection dies out if the basic reproduction number $\mathcal{R}_0 = \beta/(\gamma+\mu)$ is reduced to slightly below one, a condition which is equivalent to reducing the successful contact rate β to slightly below a critical level, say, $\beta_0 = \gamma + \mu$. However, if herd immunity only (i.e., vaccinating a proportion $p > 0$ of newborns) is applied, where the vaccine is assumed imperfect, then model (1) reduces to the case where booster vaccination is neglected (i.e., $\psi = 0$). In this case, the infection washes out if and only if the control reproduction number

$$\mathcal{R}_{\mathcal{B}} = \frac{\beta}{\gamma+\mu}\left(1 - p + gp\frac{\sigma}{\sigma+\mu}\right) \tag{28}$$

is reduced to slightly below one, which is equivalent to reducing the contact rate to slightly below the critical level

$$\beta^c_p = \frac{(\gamma+\mu)(\sigma+\mu)}{(1-p)(\sigma+\mu) + gp\sigma}. \tag{29}$$

It is clear that β^c_p tends to $\beta^r_p =: (\gamma+\mu)(\sigma+\mu)/(g\sigma)$ as p tends to one, which means that if the contact rate β is higher than the reinfection contact rate β^r_p, then the infection can not be eliminated from the population by vaccination solely, even if every newborn is vaccinated, immediately after birth, with an imperfect vaccine.

4.1 Effect of booster mass vaccination ($\psi \geq 0$)

If a booster vaccination dose is applied, as considered in model (1), then the control reproduction number is $\mathcal{R}_\psi$ and the infection washes out if and only if $\mathcal{R}_\psi < 1$. In this case, the critical contact rate below which the infection disappears and doesn't persist in the population is

$$\beta^c_\psi = \frac{(\gamma+\mu)(\sigma+\mu)(\psi+\mu)}{(1-p)(\sigma+\mu)(\psi+\mu) + gp\sigma\mu} \geq \beta^c_p. \tag{30}$$

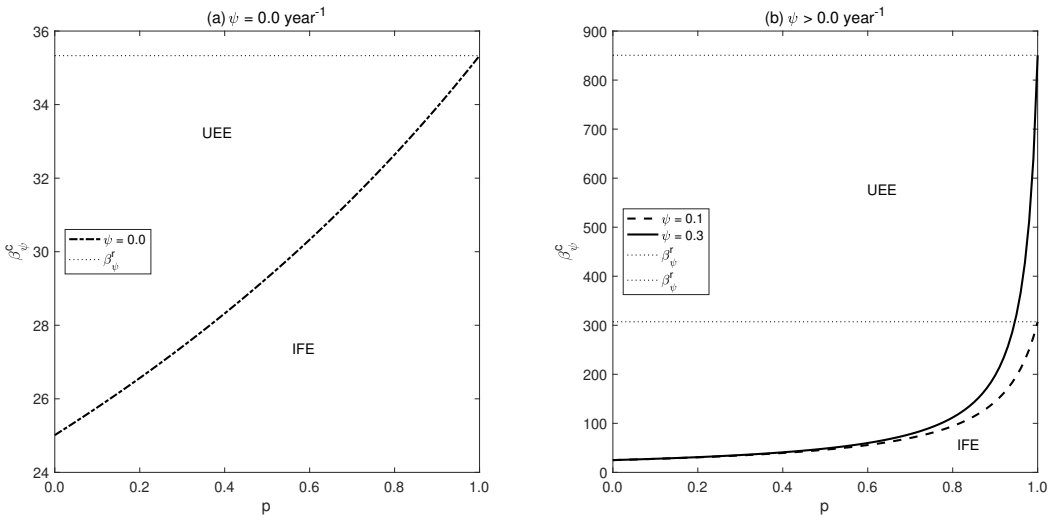

Figure 2: The critical contact rate β^c_ψ as a function of the vaccination coverage p for different levels of booster vaccination rate ψ, while keeping other model parameters fixed. Simulations have been produced with parameter values $\mu = 0.013$ year^{-1}, $\gamma = 25$ year^{-1}, $\sigma = 0.1$ year^{-1}, $g = 0.8$. Part (a) has been produced with $\psi = 0$ year^{-1}, while part (b) is produced with $\psi = 0.1$ year^{-1} (dashed curve) and $\psi = 0.3$ year^{-1} (solid curve). Horizontal dotted lines represent the reinfection contact rate. Here IFE and UEE stand for infection-free equilibrium and unique endemic equilibrium, respectively.

When p approaches one, then the critical contact rate β^c_ψ approaches a reinfection contact rate threshold β^r_ψ, where

$$\beta^r_\psi = \frac{(\gamma+\mu)(\sigma+\mu)}{g\sigma} \times \frac{\psi+\mu}{\mu} \geq \beta^r_p. \tag{31}$$

Formula (30) says that applying a booster vaccination program on susceptible vaccinated individuals increases the critical contact rate below which the infection dies out and, therefore, extends the region of attraction of the infection-free equilibrium (see figure 2), which in turn increases the possibility to eliminate the infection. However, formula (31) says that the reinfection contact rate threshold increases in the presence of mass vaccination and consequently increases the possibility to eliminate the infection with vaccination solely. In fact, $\beta^r_\psi \to \infty$ as $\psi \to \infty$.

The condition $\mathcal{R}_\psi < 1$ could be implemented if and only if

$$\frac{\mu}{\psi+\mu} < \frac{\sigma+\mu}{gp\sigma}\left(\frac{\gamma+\mu}{\beta} - (1-p)\right) \tag{32}$$

or equivalently if

$$\psi > \mu\left(\frac{\sigma}{\sigma+\mu} \times \frac{gp}{\frac{\gamma+\mu}{\beta} - (1-p)} - 1\right) := \psi^c_0 \tag{33}$$

which is well defined if and only if

$$\frac{1}{1-p} < \frac{\beta}{\gamma+\mu} < \frac{1}{1-p+\frac{gp\sigma}{\sigma+\mu}}.$$

Formula (33) defines the critical booster vaccination rate above which the infection is possibly eliminated.

4.2 Effect of reduction in contact-availability $0 \leq P_r \leq 1$

If infected individuals reduce their availability to mingle in the population (either voluntary or due to governmental constraints, like in case of covid-19) with proportion P_r, then the control reproduction number for the modified model (15) is $\mathcal{R}_{P_r}$, and the infection dies out from the population if control measures have been applied to reduce $\mathcal{R}_{P_r}$ to slightly below one or equivalently

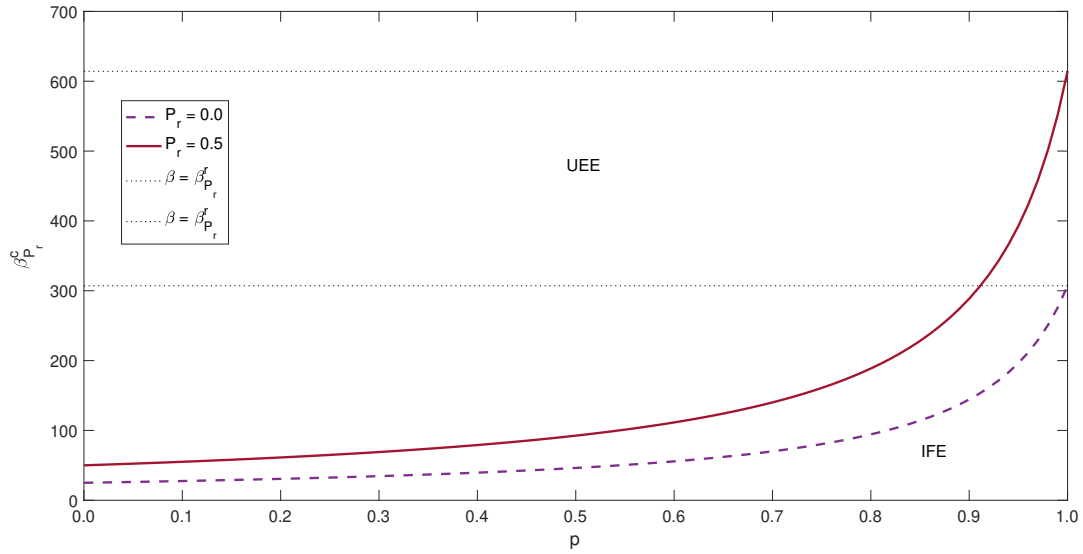

Figure 3: The critical contact rate $\beta^c_{P_r}$ as a function of the vaccination coverage p for different levels of contact activity reduction proportion P_r, while keeping other model parameters fixed. Simulations have been produced with parameter values $\mu = 0.013$ year^{-1}, $\gamma = 25$ year^{-1}, $\sigma = 0.1$ year^{-1}, $\psi = 0.1$ year^{-1}, $g = 0.8$ and ($P_r = 0.0$ for the dashed curve, $P_r = 0.5$ for the solid curve). The dotted horizontal lines show the reinfection contact rate corresponding to the various values of P_r. Below the curve, only the infection-free equilibrium (IFE) is the attractor, while above it a unique endemic equilibrium (UEE) does exist.

to reduce the effective contact rate β to slightly below a critical level $\beta^c_{P_r}$, where

$$\beta^c_{P_r} = \frac{(\gamma+\mu)(\sigma+\mu)(\psi+\mu)}{(1-P_r)\Big((1-p)(\sigma+\mu)(\psi+\mu)+gp\sigma\mu\Big)} = \frac{\beta^c_{\psi}}{1-P_r} > \beta^c_{\psi}. \tag{34}$$

It is clear that the critical contact rate $\beta^c_{P_r}$ increases with the increase of the reduction proportion P_r, see figure 3. Moreover, formula (34) says that a 50% reduction in the contact activity level of infected individuals enlarges the critical contact rate β^c_{ψ} by two folds, which in turn duplicates the region of attraction of the infection-free equilibrium and the possibility to eliminate the infection, see figure 3. Also, the reinfection contact rate reads

$$\beta^r_{P_r} = \frac{(\gamma+\mu)(\sigma+\mu)(\psi+\mu)}{(1-P_r)g\sigma\mu} \tag{35}$$

which tends to ∞ as $P_r \to 1$. If we solve the inequality $\mathcal{R}_{P_r} < 1$ in terms of the booster vaccination rate ψ, we get

$$\psi > \mu\left(\frac{\sigma}{\sigma+\mu} \times \frac{gp}{\dfrac{\gamma+\mu}{(1-P_r)\beta} - (1-p)} - 1\right) := \psi^c_{P_r} \tag{36}$$

which is well defined if and only if

$$\frac{1}{1-p} < \frac{(1-P_r)\beta}{\gamma+\mu} < \frac{1}{1-p+\dfrac{gp\sigma}{\sigma+\mu}}.$$

Formula (36) defines the critical booster vaccination rate ($\psi^c_{P_r}$) above which the infection is possibly eliminated in the presence of contact-activity reduction (i.e., $0 < P_r \leq 1$). The formula says that the higher the reduction in contact activity P_r is, the lower the critical booster vaccination rate $\psi^c_{P_r}$ is, which induces a reduction in the effort needed to eliminate the infection.

5 Summary and Conclusion

Vaccination is a safe and highly effective method used to prevent the spread of various infectious diseases. For example, most children are globally protected from many infectious diseases through routine immunization programs. However, the immunity

acquired by vaccination could last for a limited period of time after which it wanes. Consequently, waned-immunity individuals become subject to acquire the infection. Therefore, booster vaccination could be applied to raise their immunity, which in turn prevents them from acquiring the infection. In this work, the extent to which these infections are eliminated from the population with strategies based solely on vaccination has been studied. To this end, a mathematical deterministic SIR endemic model has been introduced and analyzed. The model takes into account routine immunization for a proportion p of the newborns. Vaccinated newborns are assumed to lose their vaccine-induced immunity and become susceptible-vaccinated after a period of $1/\sigma$ unit time. Therefore, the model differentiates between two types of susceptible individuals, namely, naive susceptible and susceptible-vaccinated, who were vaccinated and lost their acquired immunity. Susceptible-vaccinated individuals either acquire the infection, die naturally or get vaccinated again (booster vaccination) at rate ψ.

The analysis showed that the model has an infection-free equilibrium that is locally asymptotically stable if and only if a control reproduction number, denoted by $\mathcal{R}_\psi$, is less than one. In addition, it has a unique endemic equilibrium that is shown to exist and be stable if and only if $\mathcal{R}_\psi > 1$. The analysis shows further that the critical contact rate separating between non-persistence and persistence of the infection is β^c_ψ, given by (30), that increases with the increase of the booster vaccination rate ψ, which in turn increases the possibility (and decreases the effort required) to eliminate the infection. However, it has a finite (in p) reinfection contact rate β^r_ψ, given by (31). This reinfection level increases with the increase of the booster vaccination rate ψ, which means that the earlier the admission of booster vaccination is, the better the chance to eliminate the infection is.

The model has been extended to include disease-induced reduction in contact-activity of infected individuals at a proportion P_r and study its impact on the overall dynamics and the controllability of the infection. The analysis shows that the critical contact rate below which the infection dies out $\beta^c_{P_r}$, given in (34), increases with the increase of P_r and therefore enhances the possibility to eliminate the infection. Moreover, the higher the reduction in the contact activity of infected individuals is, the lower the booster vaccination rate ψ needed to ensure an effective control of the infection is and, consequently, the less the minimum effort required to eliminate the infection is.

Our model could possibly be applied to the case of measles if disease-induced mortality has been taken into account. It can further be extended to consider the loss of infection-induced immunity which models pertussis dynamics.

Appendix A Properties of Model (1) – Proof of Proposition 1

The S-equation implies that $dS/dt \geq -(\mu + \beta I)S$. Hence, we obtain

$$S(t) \geq S(0) \exp\left(-\int_0^t (\beta I(u) + \mu) du\right) \geq 0 \quad \forall \quad S(0) \geq 0.$$

Similarly, it is possible to obtain

$$\begin{aligned}
V(t) &\geq V(0) \exp\left(-(\sigma + \mu)t\right) \geq 0 \quad \forall \quad S(0) \geq 0,\\
S_v(t) &\geq S_v(0) \exp\left(-\int_0^t (q\beta I(u) + \mu + \psi) du\right) \geq 0 \quad \forall \quad S_v(0) \geq 0,\\
I(t) &\geq \exp\left(-(\gamma + \mu)t\right) \geq 0 \quad \forall \quad I(0) \geq 0,\\
R(t) &\geq R(0) \exp\left(-\mu t\right) \geq 0 \quad \forall \quad R(0) \geq 0.
\end{aligned}$$

Therefore, for any non-negative initial values $S(0)$, $V(0)$, $S_v(0)$, $I(0)$, $R(0)$, all solutions $S(t)$, $V(t)$, $S_v(t)$, $I(t)$, $R(t)$ remain non-negative. Moreover, since $S(t) + V(t) + S_v(t) + I(t) + R(t) = 1$, then the solutions are bounded from above. Thus, given $\big(S(0), V(0), S_v(0), I(0), R(0)\big) \in \Omega$, the solution set $\big(S(t), V(t), S_v(t), I(t), R(t)\big)$ remains in Ω for all $t > 0$. Consequently, Ω is positively invariant.

On the other hand, as the right hand side of the system (1) is continuous and differentiable in the state variables S, V, S_v, I, R, then it is locally Lipschitz and therefore the system has a unique time-dependent solution on a maximum forward interval of existence.

Appendix B Derivation of Formula (11)

From (10) we get

$$\begin{aligned}
B_1^2 - 4A_1C_1 &= \big(g\mu\beta(p\sigma + (1-p)(\sigma+\mu))\big)^2 + (\gamma+\mu)^2(\sigma+\mu)^2(\psi+\mu+g\mu)^2 \\
&\quad - 2g\mu\beta(\gamma+\mu)(\sigma+\mu)(\psi+\mu+g\mu)(p\sigma+(1-p)(\sigma+\mu)) \\
&\quad + 4g\mu\beta(\gamma+\mu)(\sigma+\mu)(gp\sigma\mu+(1-p)(\sigma+\mu)(\psi+\mu)) - 4g\mu(\gamma+\mu)^2(\sigma+\mu)^2(\psi+\mu) \\
&= \big(g\mu\beta(p\sigma + (1-p)(\sigma+\mu))\big)^2 + (\gamma+\mu)^2(\sigma+\mu)^2(\psi+\mu-g\mu)^2 \\
&\quad + 2g\mu\beta(\gamma+\mu)(\sigma+\mu)\Big(gp\sigma\mu+(1-p)(\sigma+\mu)(\psi+\mu) - p\sigma(\psi+\mu) - (1-p)g\mu(\sigma+\mu)\Big) \\
&= \big(g\mu\beta(p\sigma + (1-p)(\sigma+\mu))\big)^2 + (\gamma+\mu)^2(\sigma+\mu)^2(\psi+\mu-g\mu)^2 \\
&\quad + 2g\mu\beta(\gamma+\mu)(\sigma+\mu)(\psi+\mu-g\mu)((1-p)(\sigma+\mu)-p\sigma) \\
&= \Big((\gamma+\mu)(\sigma+\mu)(\psi+\mu-g\mu) + g\mu\beta((1-p)(\sigma+\mu)-p\sigma)\Big)^2 \\
&\quad + 4p^2g^2\sigma^2\mu^2\beta^2 + 4pg^2\sigma\mu^2\beta^2((1-p)(\sigma+\mu)-p\sigma) \\
&= \Big((\gamma+\mu)(\sigma+\mu)(\psi+\mu-g\mu) + g\mu\beta((1-p)(\sigma+\mu)-p\sigma)\Big)^2 + 4p(1-p)g^2\mu^2\beta^2\sigma(\sigma+\mu).
\end{aligned}$$

Appendix C Derivation of Formula (26)

$$\left.\frac{\partial G}{\partial \bar{\lambda}_S}\right|_{(\beta^c_{P_r},0)} = B_2\big|_{\beta=\beta^c_{P_r}} = (\gamma+\mu)(\sigma+\mu)(\psi+\mu+g\mu) - P_r\mu\big(gp\sigma\mu + (1-p)(\sigma+\mu)(\psi+\mu)\big) - g(1-P_r)\mu\big(p\sigma+(1-p)(\sigma+\mu)\big)\beta^c_{P_r} \quad (37)$$

Now, we use (24) in (37) to get

$$\begin{aligned}
(gp\sigma\mu + (1-p)(\sigma+\mu)(\psi+\mu))\left.\frac{\partial G}{\partial \lambda_S}\right|_{(\beta^c_{P_r},0)} &= (\gamma+\mu)(\sigma+\mu)(\psi+\mu+g\mu)\big(gp\sigma\mu+(1-p)(\sigma+\mu)(\psi+\mu)\big) \\
&\quad - P_r\mu(gp\sigma\mu+(1-p)(\sigma+\mu)(\psi+\mu))^2 \\
&\quad - g\mu(\gamma+\mu)(\sigma+\mu)(\psi+\mu)(p\sigma+(1-p)(\sigma+\mu)) \\
&= (\gamma+\mu)(\sigma+\mu)\Big(p\sigma(g\mu)^2 + (1-p)(\sigma+\mu)(\psi+\mu)^2\Big) \\
&\quad - P_r\mu\Big(gp\sigma\mu+(1-p)(\sigma+\mu)(\psi+\mu)\Big)^2 \\
&\geq (\gamma+\mu)(\sigma+\mu)\Big(p\sigma(g\mu)^2 + (1-p)(\sigma+\mu)(\psi+\mu)^2\Big) \\
&\quad - \mu\Big(gp\sigma\mu+(1-p)(\sigma+\mu)(\psi+\mu)\Big)^2 \\
&= (\gamma+\mu)(\sigma+\mu)(p\sigma)(g\mu)^2 + (1-p)(\gamma+\mu)(\sigma+\mu)^2(\psi+\mu)^2 \\
&\quad - \mu\Big((g\mu)^2(p\sigma)^2 + 2gp(1-p)\sigma\mu(\sigma+\mu)(\psi+\mu) + (1-p)^2(\sigma+\mu)^2(\psi+\mu)^2\Big) \\
&= (p\sigma)(g\mu)^2\Big((\gamma+\mu)(\sigma+\mu) - p\sigma\mu\Big) \\
&\quad + (1-p)(\sigma+\mu)(\psi+\mu) \\
&\quad\quad \times \Big(\big(\gamma(\sigma+\mu)(\psi+\mu) - gp\sigma\mu^2\big) + p\mu\big((\sigma+\mu)(\psi+\mu) - g\sigma\mu\big)\Big) \\
&= (p\sigma)(g\mu)^2\Big((\gamma+\mu)(\sigma+\mu) - p\sigma\mu\Big) \\
&\quad + gp(1-p)\sigma\mu^2(\sigma+\mu)(\psi+\mu) \\
&\quad\quad \times \left(\left(\frac{\gamma}{gp\mu}\times\frac{\sigma+\mu}{\sigma}\times\frac{\psi+\mu}{\mu} - 1\right) + \left(\frac{\sigma+\mu}{g\sigma}\times\frac{\psi+\mu}{\mu} - 1\right)\right) > 0.
\end{aligned}$$

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