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MODELLING THE TRANSMISSION DYNAMICS OF MUMPS WITH CONTROL MEASURES

ASINA KIBONGE*, STEPHEN EDWARD, MONICA KUNG'ARO

Department of Mathematics and Statistics, University of Dodoma, P.O.Box 338, Dodoma, Tanzania

Abstract. This paper proposes a nonlinear deterministic model of mumps transmission dynamics with double-dose vaccination, public health education campaigns, and quarantine control strategies. The stability analysis of the equilibria is established via Routh-Hurwitz criteria and the Lyapunov function method. Also, sensitivity analysis is conducted to assess the parameters that significantly influence the dynamics of mumps transmission. The numerical results of the model show that the number of mumps infections decreases when at least a single control measure is implemented effectively. Furthermore, the findings show that the most effective method of reducing mumps transmission from the community is a combination of three control measures. Therefore, this study recommends that there is a necessity to increase double-dose vaccination, public health education campaigns, and quarantine so as to reduce mumps transmission.

Keywords: mumps; quarantine; vaccination; carrier; control measures.

2020 AMS Subject Classification: 92C60.

1. INTRODUCTION

Mumps is a viral disease caused by the mumps virus. It is an envelope, single-stranded RNA Virus of the paramyxovirus family and mainly affects children less than 5 years [1]. It

*Corresponding author

E-mail address: kibongeasina@gmail.com

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is commonly known as childhood viral disease [2]. Mumps virus is spread through respiratory droplets, close contact with sick individuals, and contaminated fomites [3, 4]. The most susceptible individuals live close to schools, colleges, and the most populated areas.

Human being is the only natural host, where infection can be localized to the mucosa of the respiratory tract through the nose, and mouth [4]. The unusual swelling of a person's parotid glands takes between 15 and 24 days to manifest. The most contagious people eventually are those with the infection; this starts one to two days before parotitis symptoms appear and lasts for several days afterward [2, 5].

According to [6], the infectious period is 8 days, and the afflicted person will recover from the infection in 10 to 14 days. Bilateral enlargement of the parotid glands, a flu-like illness, headache, body aches, loss of appetite, fatigue, low-grade fever, anorexia, and malaise, are among the most typical signs of infection [7, 8]. But, symptoms in adults, women and men are always more severe than in children, such as meningitis, orchitis, oophoritis, encephalitis, and aseptic are common complications of mumps to those adults [9].

In addition, mumps caused many outbreak cases, mainly in China and the United States [1]. It was reported that more than 300,000 young people contract mumps yearly in China; through these cases, children aged 18-24 months in China received their first dose of the Measles-Mumps-Rubella (MMR1) vaccine free of charge [5]. On the other hand, in 2006, the United States experienced a multi-state outbreak involving 6584 cases, with the highest attack rate among 18 to 24 years of age. Most of the affected people had received a second MMR vaccine, which protects against measles, mumps, and rubella [10].

In Africa, it was reported that one month to six years of children aged was found to be 9% that affected with mumps in South Africa [11]. Doshi [12] reported that children aged one month to six years were found to be 22% affected in the Democratic Republic of Congo (DRC). In Tanzania, Rakiru [13] showed that 21.4% among children aged 6 to 12 years were found to be affected in the Mwanza region. According to Minja [14] 16.7% among children in Buguruni, Dar es salaam was found to be deaf due to mumps complication.

According to [15], multiple factors contribute to the persistence of mumps outbreaks, including inadequate vaccination rates, inherent restrictions in mumps protective immunity, incorrect

diagnosis of infection, failure of the primary or secondary vaccine, and elevated risk of transmission associated with college campuses. The vaccine is considered the best control for mumps, providing two complete doses for mumps [5]. Mumps virus vaccine was approved in the United States in 1967 [16], whereas, the approval of the measles, mumps, and rubella vaccine was in 1969 [7].

Several mathematical models have been developed to investigate the dynamics of mumps transmission such as [5, 17, 18, 19, 20]. Most of these models target minimizing or eradicating disease transmission among the populace.

Li et al 2018 [5] formulated a non-autonomous SVEILR (Susceptible-Vaccinated-Exposed-severely Infectious-mild Infectious-Recovered) model with a seasonal varying transmission rate to describe the mumps epidemic and suggested government health departments and hospitals encourage teenagers of the appropriate age to continue receiving the mumps vaccination.

[17] proposed a non-autonomous SVEILHR (susceptible-vaccinated-exposed-mild infectious-severe infectious-hospitalized-recovered) model with varying seasonal transmission rates and suggested that the Chinese government increase vaccination rates and offer two-doses of the MMR2 (Measles, Mumps, and Rubella) vaccine for free. Their finding showed that vaccination was the best measure to reduce the transmission of mumps in China.

Bai et al 2021 [18] formulated a SEIAQR (susceptible-exposed-symptomatically infected-asymptomatically infected-quarantined-recovered) model of mumps transmission with quarantine measures to control mumps. It was shown that quarantine was the best measure to reduce the transmission of mumps to the community.

Liu et al. (2017) [19] conducted a study on modeling and analysis of the global resurgence of mumps. A novel multi-group SVEIAR (Susceptible- Vaccinated-Exposed- Symptomatic Infected- Asymptomatic Infected- Recovered) epidemic model with infinite distributed vaccination delays and latency, asymptomatic infection, and nonlinear incidence was formulated.

The study conducted by Peng et al. (2021)[20] formulated the SEIAR (Susceptible- Exposed-Infectious- Asymptomatically Infectious- Recovered) model for estimating the transmissibility of mumps in Wuhan city, China. The study considered that the prevention and control measures of vaccination for children aged 5 to 10 years old should be taken before the peak transmission

capacity each year, 2 months before the outbreak's peak occurs, to reduce the spread of the mumps virus. The study recommended improving mumps vaccine coverage in Wuhan, China, especially two vaccination doses for 5 to 10 years old, to reduce the transmission of mumps in the population.

Most previous studies have not considered public health education campaigns either singly or in a combination with other strategy as a control measure to reduce the risk of mumps transmission among people. Moreover, double vaccination has not been presented by previous studies to control mumps. It is in this ground, therefore, that this study should be carried out to investigate the impacts of control measures that incorporate double-dose vaccination, public health education campaign, and quarantine as control strategies to reduce the transmission dynamics of mumps in the community.

The remaining sections of the study are structured as follows: Section 2 focuses on model formulation, while Section 3 is based on model analysis. Numerical results are presented in Section 4, while the concluding remarks are presented in Section 5.

2. Model Formulation

A deterministic mathematical model is formulated by extending the basic SEIR model (susceptible- exposed- infected- recovered). An improvement of the work by [5, 18, 19] is made by considering second-dose vaccination and public health education campaigns. Thus, the current work will capture double-dose vaccination, public health education campaigns, carriers, and quarantine. The total population is divided into eight classes which are susceptible humans (S), the first-dose of vaccinated humans (V_1), the double-dose vaccinated humans (V_2), exposed humans (E), carriers (C), symptomatic infectious humans (I), quarantine humans (Q) and recovered humans (R).

It is assumed that individuals are recruited into the population by immigration and birth at the rate τ and Λ respectively. Moreover, it is assumed that the recruits can be either vaccinated or non-vaccinated. If individuals are vaccinated, they can either be first-dose or second-dose vaccinated.

It is also assumed that the proportion τ of the recruits are vaccinated with the first dose, while the compliment $(1-\tau)$ joins the susceptible class. Susceptible individuals who missed the

first-dose vaccination can receive the first-dose vaccination at the rate ε. According to [5], it is known that the first-dose vaccination is inefficient, thus calling for the second dose. Therefore, it is assumed that individuals who received the efficient first-dose vaccine at some desirable interval can complete the second dose at the rate θ. It is also hypothesized that individuals who received a double dose of vaccine acquire some permanent immunity at a rate of ϕ_3.

Following effective direct contact with symptomatic infectious individuals or carriers, susceptible individuals could contact the mumps at the time-dependent infection rate $\lambda(t)$ modelled by the standard mass action principle. It is given by

$$\lambda(t) = \beta_1 I + \beta_2 C, \tag{2.1}$$

where, β_1, stands the transmission rate for symptomatic infectious humans whereas, β_2 represents the transmission rate for carrier individuals.

Also, it is assumed that public health education campaigns have some effects for both susceptible and first-dose vaccinated individuals to reduce the number of mumps infections. So, the direct transmission will be reduced by the rate $(1-\psi)$, and thus, the force of infection will be given by $(1-\psi)\lambda(t)$, where $\psi \in [0,1]$ measures the efficacy of the education program. If $\psi = 0$, it suggests that public health education campaigns have been disregarded as an intervention strategy, whereas $\psi = 1$ indicates that education campaigns are 100% effective in preventing the spread of the mumps.

It is also assumed that the first-dose vaccine could reduce but not eliminate the possibility for individuals to be infected with the mumps virus. So, the direct transmission will be reduced by the rate $(1-\omega)\lambda(t)$, where $\omega \in [0,1]$ measures the efficacy of the first-dose vaccine. If $\omega = 0$, it indicates that the first-dose vaccine is the inefficiency to prevents mumps spread; as a result, individuals are exposed and move to the exposed class (see also [5]), while $\omega = 1$, indicates that the first-dose vaccine is 100% efficiency in reducing the spread of the mumps.

Humans exposed could either become carriers or become symptomatic infectious at the rate α. A fraction p of the exposed humans may progress to the symptomatic infectious class at the rate α while the complement (1-p) of the exposed becomes carrier at the same rate α. Also, [21] have shown that carriers who have natural body immunity recover naturally. Thus, it is assumed that the carriers join the recovered class at the rate ϕ_2.

To reduce the risk of more mumps transmission to the population, the symptomatic infectious individuals are quarantined at the rate σ. It is assumed that quarantined individuals recover due to health care, medical treatment (especially antibiotics to reduce symptoms and pains), and natural body immunity at the rate of ϕ_1 see also in [18]. It is also assumed that each individual in any of the compartment experience a natural mortality rate denoted by μ.

Table 1, and Table 2 contain a description of the variables and parameters, respectively, that were used in the model. Also, Figure 1 presents the flow chart for the model.

Following the model description, and the flow chart 1, then, system of differential equations is obtained as:

$$
\begin{aligned}
\frac{dS}{dt} &= (1-\tau)\Lambda - \big(\mu+\varepsilon+(1-\psi)(\beta_1 I+\beta_2 C)\big)S,\\
\frac{dV_1}{dt} &= \varepsilon S+\tau\Lambda - \big(\theta+\mu+(1-\psi)(1-\omega)(\beta_1 I+\beta_2 C)\big)V_1,\\
\frac{dV_2}{dt} &= \theta V_1-(\phi_3+\mu)V_2,\\
\frac{dE}{dt} &= (1-\psi)(\beta_1 I+\beta_2 C)S+(1-\psi)(1-\omega)(\beta_1 I+\beta_2 C)V_1-(\alpha+\mu)E,\\
\frac{dC}{dt} &= \alpha(1-p)E-(\phi_2+\mu)C,\\
\frac{dI}{dt} &= \alpha pE-(\sigma+\mu)I,\\
\frac{dQ}{dt} &= \sigma I-(\phi_1+\mu)Q,\\
\frac{dR}{dt} &= \phi_3 V_2+\phi_2 C+\phi_1 Q-\mu R.
\end{aligned}
\tag{2.2}
$$

The initial conditions for the model system (2.2) are $S(0)>0$, $V_1(0)>0$, $V_2(0)>0$, $E(0)\geq 0$, $C(0)\geq 0$, $I(0)\geq 0$, $Q(0)\geq 0$, $R(0)\geq 0$.

TABLE 1. State variables and their description.

Variable	Description
S	Number of the susceptible humans at time t.
V_1	Number of the first-dose vaccinated humans at time t.
V_2	Number of the second-dose vaccinated humans at time t.
E	Number of the exposed human population at time t.
C	Number of carrier human population at time t.
I	Number of symptomatic infectious humans at time t.
Q	Number of the quarantined human population at time t.
R	Number of the recovered human population at time t.

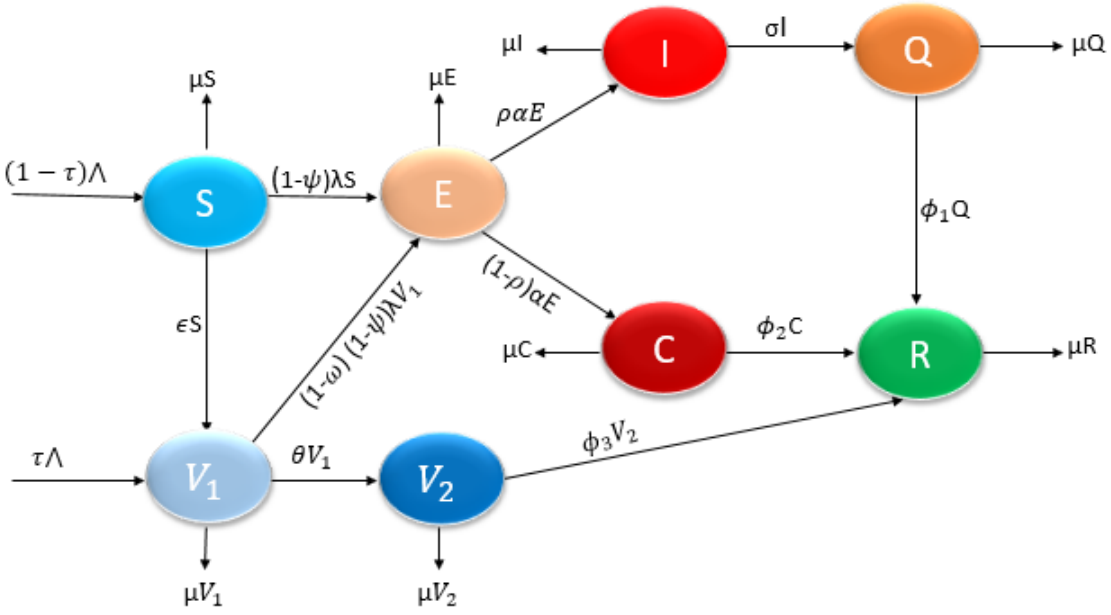

FIGURE 1. A flow diagram for mumps transmission dynamics with control measures.

TABLE 2. Parameters and their description.

Parameter	Description	Value	Source
Λ	Per capita birth rate.	0.02755 Humans/ year	[22]
τ	A proportion of immigrants who are vaccinated with first-dose.	0.6	[23]
ε	Rate at which first-dose vaccine coverage of the susceptible.	0.7/year	[24]
ω	Efficacy of first-dose vaccine.	0.9	Assumed
μ	Per capita natural mortality rate.	0.01428/year	[25]
β_1	Transmission rate for symptomatic infectious individuals.	0.4580/year	[5]
β_2	Transmission rate for carriers.	0.1250/year	[20]
ψ	Efficacy of the education program.	0.2	Assumed
θ	Second-dose of vaccine rate.	0.8/year	[23]
ϕ_3	Rate of acquiring permanent immunity by the second-dose vaccinated humans.	0.8/year	[26]
α	Rate at which exposed individuals move to either symptomatic infectious class or carrier class.	19.2105/year	[5, 27]
p	A fraction of exposed individuals who progress to symptomatic infectious class.	0.7	Assumed
σ	Rate of quarantine for symptomatic infected individuals.	0.3/year	Assumed
ϕ_2	Recovery rate of carrier humans.	30.4166/year	[5]
ϕ_1	Recovery rate of quarantine humans.	0.5/year	Assumed

3. ANALYSIS OF THE MODEL

3.1. Boundedness of model. The dynamical system of the model in equation (2.2) is considered to be well-posed and have meaning in its implication if its global solution is contained within a positive invariant region with all non-negative variables and parameters for all t ≥ 0 [28]. Thus, the following theorem is stated:

Theorem 1. *The solution set* $S,V_1,V_2,E,C,I,Q,R \in \mathbb{R}^8_+$ *of the model system* (2.2) *is confined in the positive invariant region* Ω*, for all* $t \geq 0$.

Proof. Let the feasible invariant region be

$\Omega = S,V_1,V_2,E,C,I,Q,R \in \mathbb{R}^8_+$: for all t ≥ 0.

From a model system (2.2) total human population $N(t)$ at any time t is given by

$$N(t) = S(t)+V_1(t)+V_2(t)+E(t)+C(t)+I(t)+Q(t)+R(t) \tag{3.1}$$

Differentiating equation (3.1) with respect to time t obtains

$$\frac{dN(t)}{dt} = \frac{dS}{dt}+\frac{dV_1}{dt}+\frac{dV_2}{dt}+\frac{dE}{dt}+\frac{dC}{dt}+\frac{dI}{dt}+\frac{dQ}{dt}+\frac{dR}{dt}, \tag{3.2}$$

Substituting equation (2.2) into the equation (3.2) and simplifying gives

$$\frac{dN(t)}{dt} = \Lambda - \mu N(t). \tag{3.3}$$

On solving equation (3.3) gives

$$N(t) = \frac{\Lambda}{\mu} + Ae^{-\mu t}, \tag{3.4}$$

where A is a constant. At $t = 0$, $N(t) = N(0) = N_0$, thus equation (3.4) becomes

$$N(t) = \frac{\Lambda}{\mu} + (N_0 - \frac{\Lambda}{\mu})e^{-\mu t}, \tag{3.5}$$

Now as $t \to \infty$ equation (3.5) gives

$$N(t) \leq \frac{\Lambda}{\mu}. \tag{3.6}$$

Therefore, the region Ω contains all solutions of model system equation in (2.2) in $\mathbb{R}^8_+$ such that $\Omega = \left\{(S(t),V_1(t),V_2(t),E(t),C(t),I(t),Q(t),R(t)) \in \mathbb{R}^8_+ : 0 \leq N(t) \leq \frac{\Lambda}{\mu}\right\}$. □

3.2. Positivity of model solutions. Biologically, the population is said to be meaningful and well-behaved if its model equation solutions are positive for all $t \geq 0$. Using the approach as applied by [29] and [30], we test the positivity solution of the model by using the following theorem.

Theorem 2. *Let the initial condition of model system* (2.2) *be*
$S(0) > 0$, $V_1(0) > 0$,$V_2(0) > 0$, $E(0) \geq 0$, $C(0) \geq 0$, $I(0) \geq 0$, $Q(0) \geq 0$, $R(0) \geq 0 \in \Omega$, *then the solution for* $S(t);V_1(t);V_2(t);E(t);C(t);I(t);Q(t);R(t)$ *of the system in equation* (2.2) *are positive for all* $t \geq 0$.

Proof. To prove theorem 2, all systems of equation (2.2) must be taken into consideration, hence by starting with the equation of the susceptible human population:

$$\frac{dS}{dt} = (1-\tau)\Lambda - \big(\mu + \varepsilon + (1-\psi)(\beta_1 I + \beta_2 C)\big)S, \tag{3.7}$$

Equation (3.7) can be written as,

$$\frac{dS}{dt} > -\big(\mu + \varepsilon + (1-\psi)(\beta_1 I + \beta_2 C)\big)S, \tag{3.8}$$

Solving equation (3.8) we get:

$$S(t) > S(0)e^{-\left(\mu+\varepsilon+(1-\psi)(\beta_1 I+\beta_2 C)\right)t}, \tag{3.9}$$

Therefore, as $t \to \infty$, equation (3.9) becomes:

$$S(t) > 0,$$

Thus, for all $t \geq 0$, $S(t) > 0$.
In the same way, the remaining variables of the model system in equation (2.2) can be positive for all $t \geq 0$. □

3.3. Disease-free Equilibrium Point (DFE). The Disease-Free Equilibrium (DFE) is the point at which no disease is present in the population. Let E_0 be a disease-free equilibrium point, and then from the system (2.2), we solve for variables by setting the right-hand side equal to zero. After solving the following DFE is obtained,

$$E_0 = \left(S^0, V_1^0, V_2^0, E^0, C^0, I^0, Q^0, R^0\right),$$

is given by

(3.10)
$$E_0 = \Big(\frac{(1-\tau)\Lambda}{\varepsilon+\mu}, \frac{\Lambda(\varepsilon+\tau\mu)}{(\varepsilon+\mu)(\theta+\mu)}, \frac{\theta\Lambda(\varepsilon+\tau\mu)}{(\varepsilon+\mu)(\theta+\mu)(\phi_3+\mu)} 0,0,0,0, \frac{\phi_3\theta\Lambda(\varepsilon+\tau\mu)}{\mu(\phi_3+\mu)(\varepsilon+\mu)(\theta+\mu)}\Big).$$

3.4. Effective Reproduction Number (R_e). Effective reproduction number (R_e) refers to an expected average number of secondarily infected individuals produced from a single primarily infected individual in its lifetime duration when all population is entirely susceptible at the time of its infectious period [31].

The reproduction number is significant to determine whether the disease exists or clears out in the population. To obtain the effective reproduction number, the next-generation matrix method is employed [32]. Hence, the spectral radius of the next-generation matrix is the effective reproduction number [33]. From the system (2.2), the effective reproduction number R_e is computed by re-arranging the first four infective classes, giving:

(3.11)
$$\begin{aligned}
\frac{dE}{dt} &= (1-\psi)(\beta_1 I+\beta_2 C)S+(1-\psi)(1-\omega)(\beta_1 I+\beta_2 C)V_1-(\alpha+\mu)E,\\
\frac{dC}{dt} &= \alpha(1-p)E-(\phi_2+\mu)C,\\
\frac{dI}{dt} &= \alpha pE-(\sigma+\mu)I,\\
\frac{dQ}{dt} &= \sigma I-(\phi_1+\mu)Q.
\end{aligned}$$

Then, from the system (3.11), we obtain:

(3.12)
$$F_i = \begin{bmatrix} (1-\psi)(\beta_1 I+\beta_2 C)S+(1-\psi)(1-\omega)(\beta_1 I+\beta_2 C)V_1 \\ 0 \\ 0 \\ 0 \end{bmatrix},$$

and

$$V_i = \begin{bmatrix} (\alpha+\mu)E \\ (\phi_2+\mu)C-\alpha(1-p)E \\ (\sigma+\mu)I-\alpha pE \\ (\phi_1+\mu)Q-\sigma I \end{bmatrix}. \tag{3.13}$$

From equation equations in (3.12) and (3.13), by taking partial derivatives with respect to E, C, I, and Q at DFE gives:

(3.14)

$$F = \begin{bmatrix} 0 & (1-\psi)\beta_2 S^0+(1-\psi)(1-\omega)\beta_2 V_1^0 & (1-\psi)\beta_1 S^0+(1-\psi)(1-\omega)\beta_1 V_1^0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},$$

and

$$V = \begin{bmatrix} m_{11} & 0 & 0 & 0 \\ -m_{31} & m_{33} & 0 & 0 \\ -m_{21} & 0 & m_{22} & 0 \\ 0 & 0 & -\sigma & \phi_1+\mu \end{bmatrix}. \tag{3.15}$$

Taking the inverse matrix in equation (3.15) gives

$$V^{-1} = \begin{bmatrix} {m_{11}}^{-1} & 0 & 0 & 0 \\ \frac{m_{31}}{m_{11}m_{33}} & {m_{33}}^{-1} & 0 & 0 \\ \frac{m_{21}}{m_{11}m_{22}} & 0 & {m_{22}}^{-1} & 0 \\ -\frac{\sigma m_{31}}{m_{11}m_{33}(\phi_1+\mu)} & -\frac{\sigma}{m_{33}(\phi_1+\mu)} & 0 & -(\phi_1+\mu)^{-1} \end{bmatrix}. \tag{3.16}$$

The product of the matrices F and V^{-1} can be calculated as:

$$FV^{-1} = \begin{bmatrix} \frac{m_{13}m_{31}}{m_{11}m_{33}} + \frac{m_{12}m_{21}}{m_{11}m_{22}} & \frac{m_{13}}{m_{33}} & \frac{m_{12}}{m_{22}} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}. \tag{3.17}$$

where

$$\begin{aligned} &m_{11} = \mu + \alpha, m_{12} = (1-\psi)\beta_2 S^0 + (1-\psi)(1-\omega)\beta_2 V_1^0, \\ &m_{13} = (1-\psi)\beta_1 S^0 + (1-\psi)(1-\omega)\beta_1 V_1^0, \\ &m_{21} = \alpha(1-p), m_{22} = \mu + \phi_2, m_{31} = \alpha p, m_{33} = \mu + \sigma, \end{aligned} \tag{3.18}$$

The effective reproduction number is therefore given by

$$R_e = \rho(FV^{-1}) = \frac{m_{13}m_{31}}{m_{11}m_{33}} + \frac{m_{12}m_{21}}{m_{11}m_{22}} \tag{3.19}$$

which can also be written as

$$R_e = R_{01} + R_{02}, \tag{3.20}$$

where

$$\begin{aligned} R_{01} &= \frac{m_{13}m_{31}}{m_{11}m_{33}} = \frac{\alpha p\big((1-\psi)\beta_1 S^0 + (1-\psi)(1-\omega)\beta_1 V_1^0\big)}{(\mu+\alpha)(\mu+\sigma)}, \\ R_{02} &= \frac{m_{12}m_{21}}{m_{11}m_{22}} = \frac{\alpha(1-p)\big((1-\psi)\beta_2 S^0 + (1-\psi)(1-\omega)\beta_2 V_1^0\big)}{(\mu+\alpha)(\mu+\phi_2)}. \end{aligned} \tag{3.21}$$

Additionally, R_{0i} $(i = 1,2)$ are partial effective reproduction number induced by susceptible-to-symptomatic infectious, susceptible-to-carriers transmission respectively. In a more compact form, the effective reproduction is written as

$$R_e = \frac{\alpha p(1-\psi)\big(\beta_1 S^0 + (1-\omega)\beta_1 V_1^0\big)}{(\mu+\alpha)(\mu+\sigma)} + \frac{\alpha(1-p)(1-\psi)\big(\beta_2 S^0 + (1-\omega)\beta_2 V_1^0\big)}{(\mu+\alpha)(\mu+\phi_2)}, \tag{3.22}$$

where S^0 and V_1^0 have been defined in equation (3.10). Epidemiologically, if $R_e > 1$, it implies that mumps-infected individuals can transmit the disease to more than one individual, thus spreading the disease in the entire community. However, if $R_e < 1$, then it implies that the single infected individual, on average, can transmit the disease to less than one individual in the human population; hence the disease dies out.

3.4.1. *Sensitivity analysis of R_e.* Sensitivity analysis is carried out to identify parameters that highly impact the disease's transmission dynamics. The normalized forward sensitivity method, which employs the effective reproduction number (R_e), is used to attain this goal. The same approach was also carried out by [28, 34, 35]. If R_e is differentiated with respect to parameter k, then the sensitivity index of k is given by

$$Y_k^{R_e} = \frac{\partial R_e}{\partial k} \times \frac{k}{R_e}. \tag{3.23}$$

For example, the sensitivity index of R_e with respect to parameter Λ is given by

$$Y_\Lambda^{R_e} = \frac{\partial R_e}{\partial \Lambda} \times \frac{\Lambda}{R_e} = +1. \tag{3.24}$$

The rest of the sensitivity indices for all parameters used in R_e can be computed similarly and given in Table 3. From Table 3, a positive index value implies that an increase in the particular parameter will increase R_e and hence mumps transmission. In contrast, the negative index indicates that an increase in the specific parameter value will increase will decrease R_e and therefore reduce mumps transmission.

TABLE 3. Sensitivity of Numerical Scale for R_e

Parameter Symbol	Sensitivity Index value
Λ	$+1.0000$
τ	-1.2298
ε	-0.8035
μ	-0.0664
ψ	-0.2500
α	$+0.0007$
p	$+0.9468$
σ	-0.9393
ϕ_2	-0.0160
β_1	$+0.9840$
β_2	$+0.0160$
θ	-0.1755
ω	-1.6081

Similarly, Figure 2 shows the parameters with their respective sensitivity index values. It can be observed that the most sensitive parameters are: $\omega, \tau, \Lambda, p, \sigma, \beta_1$, and ε since they have large values of sensitivity index (> 0.5); this implies that they have very high contribution to the transmission of mumps. Conversely, the parameters $\psi, \theta, \beta_2, \phi_2, \mu$, and α contribute less to the mumps transmission as their sensitivity indices are less than 0.5.

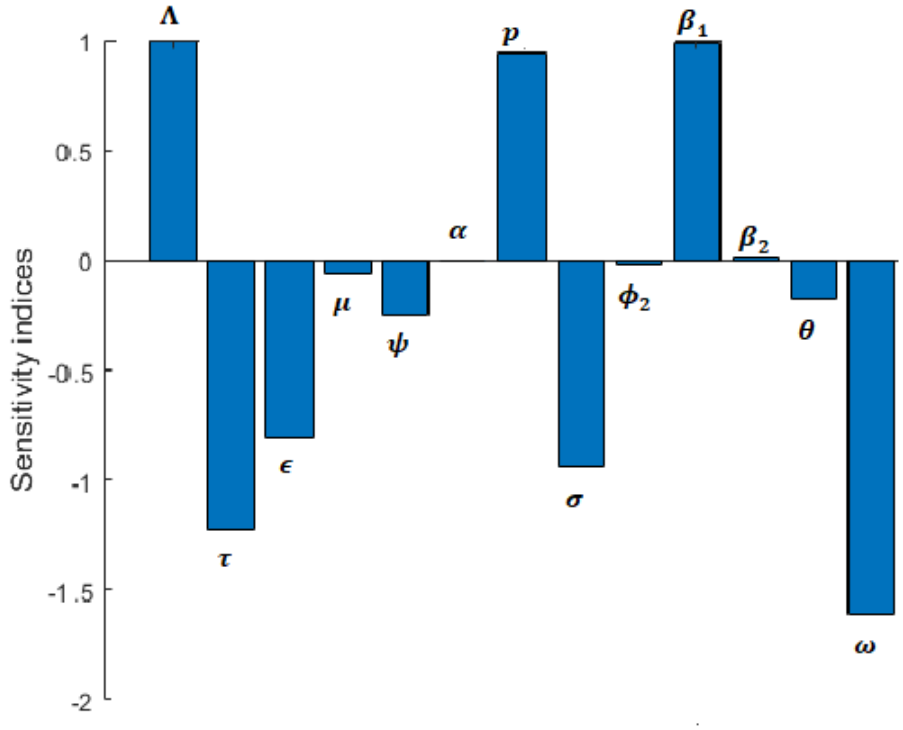

FIGURE 2. Parameters with their sensitivity indices.

3.5. Stability Analysis of the Model.

3.5.1. *Local Stability of the Disease-Free Equilibrium Point.* The local stability of the disease-free equilibrium point is determined by linearizing the model system (2.2) by using a technique of Jacobian matrix [28, 29].

Theorem 3. *The disease-free equilibrium point (DFE) of the model system* (2.2) *is locally asymptotically stable if* $R_e < 1$ *and is unstable if* $R_e > 1$.

Proof. The partial differentiation of system (2.2) with respect to (S,V_1,V_2,E,C,I,Q,R) at the DFE gives the Jacobian Matrix J as

(3.25)
$$J(E_0)=\begin{bmatrix} -\mu-\varepsilon & 0 & 0 & 0 & -(1-\psi)\beta_2 S^0 & -(1-\psi)\beta_1 S^0 & 0 & 0 \\ \varepsilon & -\mu-\theta & 0 & 0 & (1-\psi)a\beta_2 V_1^0 & (1-\psi)a\beta_1 V_1^0 & 0 & 0 \\ 0 & \theta & -\mu-\phi_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\mu-\alpha & m_{12} & m_{13} & 0 & 0 \\ 0 & 0 & 0 & \alpha(1-p) & -\mu-\phi_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & \alpha p & 0 & -\mu-\sigma & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma & -\mu-\phi_1 & 0 \\ 0 & 0 & \phi_3 & 0 & \phi_2 & 0 & \phi_1 & -\mu \end{bmatrix}$$

where $a=(1-\omega)$ from matrix (3.25).

The matrix (3.25) has the following eigenvalues

(3.26)
$$\begin{aligned} \lambda_1 &= -(\varepsilon+\mu), \\ \lambda_2 &= -(\theta+\mu), \\ \lambda_3 &= -(\phi_3+\mu), \\ \lambda_7 &= -(\phi_1+\mu), \\ \lambda_8 &= -\mu, \end{aligned}$$

The rest eigenvalues can be found in the sub-matrix

(3.27)
$$J_1(E_0)=\begin{bmatrix} -m_{11} & m_{12} & m_{13} \\ m_{21} & -m_{22} & 0 \\ m_{31} & 0 & -m_{33} \end{bmatrix}$$

where $m_{11},m_{12},m_{13},m_{21},m_{22},m_{31}$, and m_{33} have been defined in equation (3.18). The remaining eigenvalues are the polynomial's roots: $|J_1(E_0)-\lambda|=0$, which is given by

$$\lambda^3 + c_2\lambda^2 + c_1\lambda + c_0 = 0, \tag{3.28}$$

where

$$\begin{aligned} c_2 &= m_{33} + m_{22} + m_{11}, \\ c_1 &= m_{33}m_{22} + m_{22}m_{11}(1 - R_{01}) + m_{33}m_{11}(1 - R_{02}), \\ c_0 &= m_{33}m_{22}m_{11}(1 - R_e), \end{aligned}$$

Equivalently, R_e can be split into parts

$$R_e = R_{01} + R_{02}, \tag{3.29}$$

where R_{01} and R_{02} have been defined in equation (3.21).

To ensure that all roots of equation (3.28) have negative real parts, the Routh-Hurwitz stability criterion requires that

$$c_2 > 0, c_1 > 0, c_0 > 0, \tag{3.30}$$

and

$$\begin{aligned} D_1 &= c_2 > 0, \\ D_2 &= \begin{vmatrix} c_2 & 1 \\ c_0 & c_1 \end{vmatrix} = c_2c_1 - c_0 > 0, \end{aligned} \tag{3.31}$$

It is clear that $D_1 = c_2 > 0$. In addition, if $R_e < 1$, it implies that $R_{01}, R_{02} < 1$ and hence $c_0, c_1, c_2 > 0$.

D_2 can also be demonstrated to hold as follows:

$$\begin{aligned} D_2 = c_2c_1 - c_0 = m_{33}\big(&m_{22}m_{33} + m_{22}m_{11} + m_{33}m_{11}(1 - R_{02}) \\ &+ m_{22}m_{22} + m_{11}m_{22} + m_{11}m_{11}(1 - R_{02})\big) + m_{22}m_{11}m_{22}(2 - R_{01}), \end{aligned}$$

hence D_2 is positive. Therefore, whenever $R_e < 1$, the disease-free equilibrium E_0 is locally asymptotically stable if all Routh-Hurwitz requirements are met. □

3.5.2. *Global Stability of the Disease-Free Equilibrium Point.*

Theorem 4. *The disease-free equilibrium E_0 of system* (2.2)*is globally asymptotically stable on* Ω*, if* $R_e \leq 1$*, and is unstable if* $R_e > 1$.

Proof. To prove theorem (4), consider the system (2.2), the Lypunov function $L(t)$ with non-negative coefficients M_1, M_2, M_3 and M_4 in the trivial equilibrium points then

$$\begin{aligned} L = (S - S^0 - S^0 ln\frac{S}{S^0}) + (V_1 - V_1^0 - V_1^0 ln\frac{V_1}{V_1^0}) + (V_2 - V_2^0 - V_2^0 ln\frac{V_2}{V_2^0}) + \\ M_1 E + M_2 C + M_3 I + M_4 Q + (R - R^0 - R^0 ln\frac{R}{R^0}) \end{aligned} \tag{3.32}$$

Differentiating equation (3.32) with respect to time t gives:

$$\begin{aligned} \frac{dL}{dt} = (1 - \frac{S^0}{S})\frac{dS}{dt} + (1 - \frac{V_1^0}{V_1})\frac{dV_1}{dt} + (1 - \frac{V_2^0}{V_2})\frac{dV_2}{dt} + \\ M_1\frac{dE}{dt} + M_2\frac{dC}{dt} + M_3\frac{dI}{dt} + M_4\frac{dQ}{dt} + (1 - \frac{R^0}{R})\frac{dR}{dt}. \end{aligned} \tag{3.33}$$

Now we substitute respective equations from the model system equation (2.2) into equation (3.33) results give:

$$\begin{aligned} \frac{dL}{dt} &= (1 - \frac{S^0}{S})\Big((1-\tau)\Lambda - \big(\mu + \varepsilon + (1-\psi)(\beta_1 I + \beta_2 C)\big)S\Big) \\ &+ (1 - \frac{V_1^0}{V_1})\Big(\varepsilon S + \tau\Lambda - \big(\theta + \mu + (1-\psi)(1-\omega)(\beta_1 I + \beta_2 C)\big)V_1\Big) \\ &+ (1 - \frac{V_2^0}{V_2})\Big(\theta V_1 - (\phi_3 + \mu)V_2\Big) \\ &+ M_1\Big((1-\psi)(\beta_1 I + \beta_2 C)S + (1-\psi)(1-\omega)(\beta_1 I + \beta_2 C)V_1 - (\alpha + \mu)E\Big) \\ &+ M_2\Big(\alpha(1-p)E - (\phi_2 + \mu)C\Big) \\ &+ M_3\Big(\alpha pE - (\sigma + \mu)I\Big) \\ &+ M_4\Big(\sigma I - (\phi_1 + \mu)Q\Big) + (1 - \frac{R^0}{R})\Big(\phi_3 V_2 + \phi_2 C + \phi_1 Q - \mu R\Big) \end{aligned} \tag{3.34}$$

Suppose $S \leq S^0$, $V_1 \leq V_1^0$, $V_2 \leq V_2^0$, and $R \leq R^0$ then by substituting these expressions in equation (3.34) and simplifying gives:

$$\begin{aligned}\frac{dL}{dt} \leq\ & M_1\Big((1-\psi)(\beta_1 I+\beta_2 C)S+(1-\psi)(1-\omega)(\beta_1 I+\beta_2 C)V_1-(\alpha+\mu)E\Big)\\ & +M_2\Big(\alpha(1-p)E-(\phi_2+\mu)C\Big)\\ & +M_3\Big(\alpha pE-(\sigma+\mu)I\Big)\\ & +M_4\Big(\sigma I-(\phi_1+\mu)Q\Big),\end{aligned} \tag{3.35}$$

where S^0, V_1^0 are defined in (3.10), by collecting similar terms E, C, I and Q and keeping in mind that at disease-free equilibrium point (E_0): E^0=C^0= I^0= Q^0= 0, so by solving for coefficients M_1, M_2, M_3, and M_4 and further simplification (see also [28]) gives $\frac{dL}{dt} \leq 0$. Hence, the largest compact invariant set in $\{(S(t),V_1(t),V_2(t),E(t),C(t),I(t),Q(t),R(t)) \in \Omega : \frac{dV}{dt} < 0\}$ is the singleton set E_0. Therefore, using Lasalle's invariant principle [36], it can be concluded that E_0 is globally asymptotically stable in Ω whenever $R_e < 1$ and unstable for $R_e > 1$ (see also [37, 38]). □

3.5.3. *Endemic Equilibrium Point.* Endemic equilibrium point (E_1^*) is a situation whereby the disease exists in the population. For this particular case, the endemic is obtained when

$$S^*, V_1^*, V_2^*, E^*, C^*, I^*, Q^*, R^* > 0.$$

Setting the right side of each equation in the model system (2.2) to zero and solving the resulting system yields the endemic equilibrium point in terms of symptomatic humans I^*; thus, we have

$$E_1^* = (S^*, V_1^*, V_2^*, E^*, C^*, I^*, Q^*, R^*), \tag{3.36}$$

where

$$\begin{aligned}S^* &= \frac{(1-\tau)\Lambda}{\mu+\varepsilon+(1-\psi)(\beta_1+\beta_2 h_2)I^*},\\ V_1^* &= \frac{\varepsilon(1-\tau)\Lambda+\tau\Lambda\big(\mu+\varepsilon+(1-\psi)(\beta_1+\beta_2 h_2)I^*\big)}{\big(\mu+\theta+(1-\psi)(1-\omega)(\beta_1+\beta_2 h_2)I^*\big)\big(\mu+\varepsilon+(1-\psi)(\beta_1+\beta_2 h_2)I^*\big)},\\ V_2^* &= \frac{\theta\Big(\varepsilon(1-\tau)\Lambda+\tau\Lambda\big(\mu+\varepsilon+(1-\psi)(\beta_1+\beta_2 h_2)I^*\big)\Big)}{(\phi_3+\mu)\big(\mu+\theta+(1-\psi)(1-\omega)(\beta_1+\beta_2 h_2)I^*\big)\big(\mu+\varepsilon+(1-\psi)(\beta_1+\beta_2 h_2)I^*\big)},\end{aligned}$$

$$E^* = \frac{(\sigma+\mu)}{\alpha p} I^*,$$
$$C^* = \frac{(1-p)(\sigma+\mu)}{p(\phi_2+\mu)} I^*,$$
$$Q^* = \frac{\sigma}{\mu+\phi_1} I^*,$$
$$R^* = \frac{\phi_3 V_2^* + \phi_2 C^* + \phi_1 Q^*}{\mu}.$$

with I^* being the positive root of the polynomial (3.37).

$$c_2 (I^*)^2 + c_1 (I^*) + c_0 = 0, \tag{3.37}$$

where

(3.38)

$$c_2 = -(1-\omega) h_1 (\psi - 1)^2 (\alpha+\mu) (\beta_1 + \beta_2 h_2)^2,$$
$$c_1 = (\psi-1)(\beta_1+\beta_2 h_2)\left(h_1(\alpha+\mu)((1-\omega)(\mu+\varepsilon)+\theta+\mu)+(1-\omega)\Lambda(\psi-1)(\beta_1+\beta_2 h_2)\right),$$
$$c_0 = h_1(-(\alpha+\mu))(\theta+\mu)(\mu+\varepsilon) - (\psi-1)(\beta_1+\beta_2 h_2)((1-\omega)\Lambda(\mu\tau+\varepsilon)+(\theta+\mu)(\Lambda-\tau\Lambda)),$$

Additionally,

$$h_1 = \frac{(\sigma+\mu)}{\alpha p}, h_2 = \frac{(1-p)(\sigma+\mu)}{p(\phi_2+\mu)}. \tag{3.39}$$

3.5.4. *Global Stability Analysis of Endemic Equilibrium Point.*

Theorem 5. *The endemic equilibrium point E_1^* of system* (2.2) *is globally asymptotically stable if $R_e > 1$.*

Proof. Suppose $R_e > 1$; then E_1^* exists. As in Vargas [39], to determine the global stability, we define and derive the Lyapunov function V as follows.

$$V(x_1, x_2, x_3, .., x_8) = \sum_{i=1}^{8} \frac{c_i}{2} (x_i - x_i^*)^2, \tag{3.40}$$

where

x_i= Human population classes $(S, V_1, V_2, E, C, I, Q, R)$,

x_i^*= Human population at endemic equilibrium point $(S^*, V_1^*, V_2^*, E^*, C^*, I^*, Q^*, R^*)$.

Thus from equation (3.40): Choosing $c_i = 1$ we have

$$V(S,V_1,V_2,E,C,I,Q,R) = \frac{1}{2}\Big((S-S^*)+(V_1-V_1^*)+(V_2-V_2^*)+(E-E^*) + (C-C^*)+(I-I^*)+(Q-Q^*)+(R-R^*)\Big)^2. \tag{3.41}$$

Differentiating equation (3.41) with respect to t gives;

$$\frac{dV}{dt} = \Big((S-S^*)+(V_1-V_1^*)+(V_2-V_2^*)+(E-E^*)+(C-C^*) + (I-I^*)+(Q-Q^*)+(R-R^*)\Big)\frac{d}{dt}\Big(S+V_1+V_2+E+C+I+Q+R\Big) \tag{3.42}$$

Using equation (3.2), it follows that,

$$\frac{d}{dt}\Big(S+V_1+V_2+E+C+I+Q+R\Big) = \frac{dN(t)}{dt} \tag{3.43}$$

From equation (3.3)

$$\frac{dN(t)}{dt} = \Lambda - \mu N(t) \tag{3.44}$$

From (3.6), we obtain;

$$S^*+V_1^*+V_2^*+E^*+C^*+I^*+Q^*+R^* \leq \frac{\Lambda}{\mu} \tag{3.45}$$

Then, substituting equation (3.44) and (3.45) into equation (3.42) gives

$$\frac{dV}{dt} = \Big(N(t)-\frac{\Lambda}{\mu}\Big)\Big(\Lambda-\mu N(t)\Big) \tag{3.46}$$

Simplifying equation (3.46) becomes

$$\frac{dV}{dt} = -\frac{1}{\mu}\Big(\Lambda-\mu N(t)\Big)^2 \tag{3.47}$$

From equation (3.47), it is clear that $\frac{dV}{dt}$ is always negative, and is zero if and only if $S = S^*, V_1 = V_1^*, V_2 = V_2^*, E = E^*, C = C^*, I = I^*, Q = Q^*, R = R^*$. Moreover, every solution of system (2.2) with the initial conditions approaches E_1^* as $t \to \infty$ (see [37, 38]); hence, the largest compact invariant set in $\{(S(t),V_1(t),V_2(t),E(t),C(t),I(t),Q(t),R(t)) \in \Omega : \frac{dV}{dt} < 0\}$ is the singleton set E_1^*. Therefore, from Lasalle's invariant principle [36], it implies that the endemic equilibrium E_1^* is globally asymptotically stable in Ω whenever $R_e > 1$. □

4. NUMERICAL RESULTS

In this section, a model system (2.2) is solved numerically via the Rungekutta fourth-order method and is implemented in MATLAB software. The goal is to showcase the analytical results presented in the previous sections. The solutions are presented in the form of graphs. Parameters used for numerical simulations are presented in Table 2 together with the following initial conditions: $S(0) = 0.25, V_1(0) = 0.15, V_2(0) = 0.1, E(0) = 0.1, C(0) = 0.15, I(0) = 0.1, Q(0) = 0.1, R(0) = 0.05$.

4.1. Effects of first-dose vaccination only. From Figure 3 (a), it can be seen that with an increase of first-dose vaccination coverage from 0.3 to 0.9, there is a reduction in the proportion of susceptible from 25% to 0.2% respectively in 50 years' time. This is because as more people are vaccinated, they become immune to the diseases leaving a small proportion of susceptible individuals. Similarly, Figure 3 (b) shows that as first-dose vaccination increases, the number of exposed individuals decreases sharply, implying as more people become immune, they are less exposed to infection. Also, Figure 3 (c) shows that the number of symptomatic infectious people has decreased with an increase in first-dose vaccination coverage. However, such a decrease can not approach a disease-free situation because of the inefficiency of this vaccine [5]. First-dose vaccinated individuals might lose immunity in the long run and be easily re-exposed to this disease. On the other hand, Figure 3(d) shows that as the first-dose vaccine coverage increases, the recovered population initially indicates a sharp rise but later decreases. The fall in the recovered population might have been caused by the fall of symptomatic infectious individuals experienced Figure 3 (c) or carriers who are the influx of the recovered population.

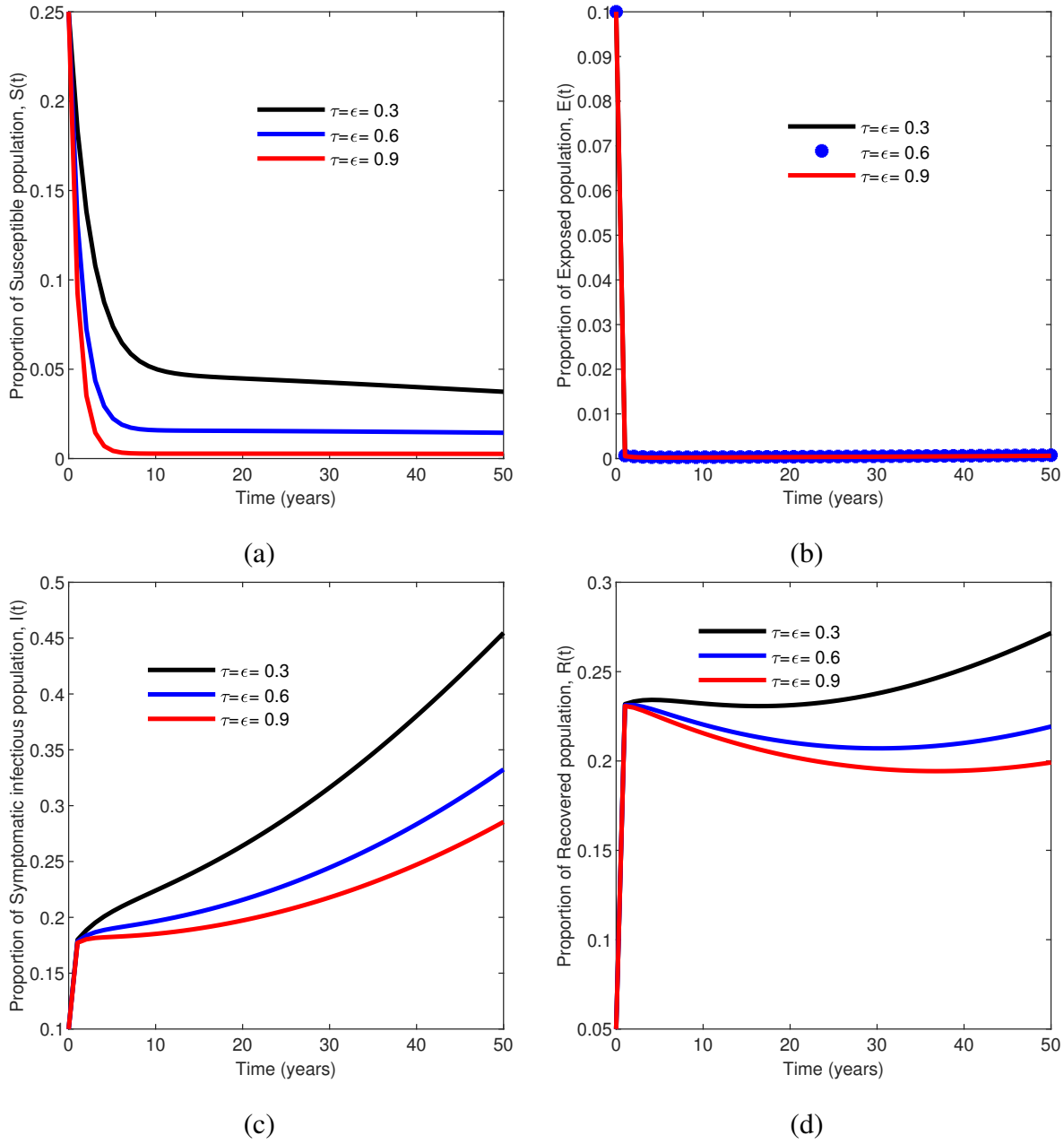

FIGURE 3. (a)-(d) show the impacts of first-dose vaccination on susceptible, exposed, symptomatic infectious, and recovered populations.

4.2. Effects of double-vaccination only. Figure 4(a) depicts that the proportion of susceptible population decreases from 0.25 to 0.002 in 50 years as a result of double-vaccination coverage variation from 0.3 to 0.9; this is because more susceptibles move into the first-dose vaccination population (V_1) and the second-dose vaccination (V_2). Also, Figure 4(b) demonstrates that with an increase in double-vaccination coverage, there is a significant drop in the

proportion of symptomatic infectious individuals. On the other hand, it is illustrated in Figure 4(c) that as double-vaccination coverage increases, the proportion of the recovered population increases. Our findings align with the results by Qu [17].

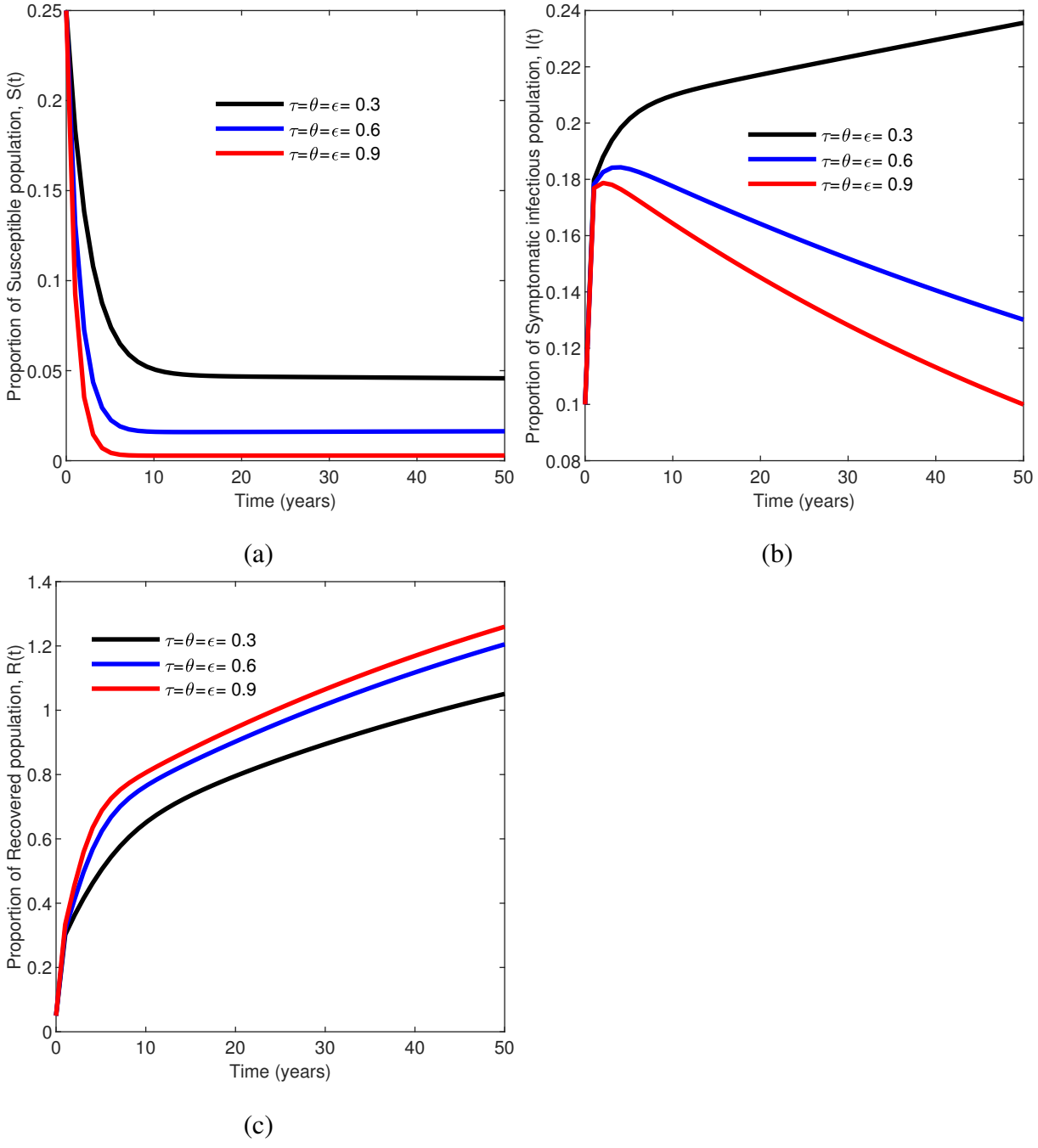

FIGURE 4. (a)-(c) show the impacts of second-dose vaccination on susceptible, symptomatic infectious, and recovered populations.

4.3. Effects of education campaign only. Figure 5(a) shows that with an increase in public health education campaigns, more people continue to be susceptible since they tend to avoid getting new infections or infecting others. Also, from Figures 5(b,c), an increased education campaign leads to a decrease in the number of carriers and symptomatic infectious individuals, respectively. Further, Figure 5(d) shows that with an increase in education campaigns, there is a drop in the proportion of the recovered individuals since fewer and fewer individuals become infectious or carriers, thus affecting influx into the recovered compartment.

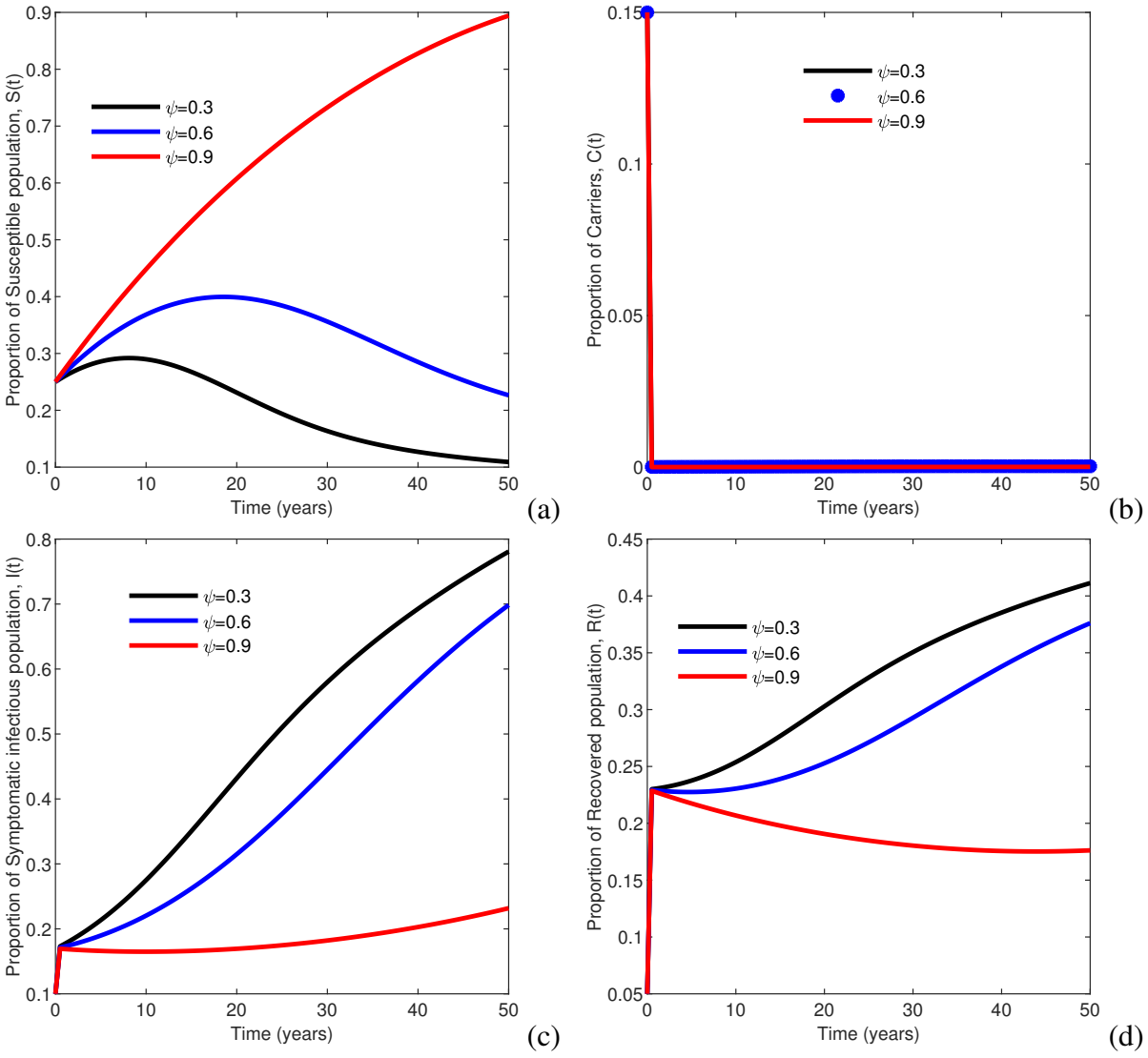

FIGURE 5. (a)-(d) show the impacts of public health education campaigns on the susceptible, carrier, symptomatic infectious, and recovered populations.

4.4. Effects of quarantine only. Figure 6 reveals that, as the quarantine rate (σ) increase, the number of symptomatic infectious population decrease. This is because the contagious individuals are being bounded where they receive medical care, and quarantined individuals are not allowed to interact with healthy people until they are cured. In this way, the transmission of mumps is easily controlled, as suggested by Bai [18].

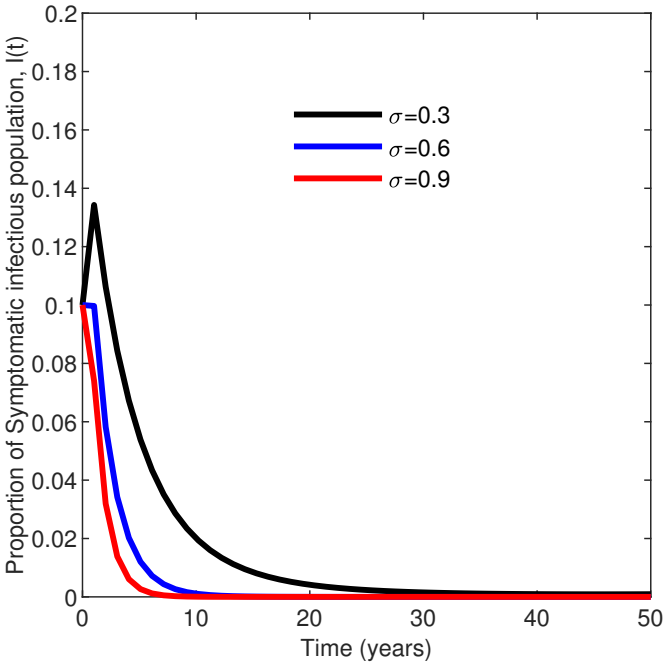

FIGURE 6. Show the impacts of quarantine on symptomatic infectious population.

4.5. Effects of a combination of two control strategies. Figure 7(a) reveals that a strategy that combines double-vaccination and education campaigns has similar effects as the one that combines double-vaccination and quarantine on decreasing the proportion of the susceptible population, and this is because more susceptible individuals move into the first-dose vaccination population (V_1) and the second-dose vaccination (V_2). However, combining education campaigns and quarantine has no direct effect on decreasing the number of susceptible individuals. This is because as more people obtain education about mumps disease, it reduces the risk of disease transmission, so more people continue to be susceptible since they tend to avoid

getting new infections or infecting others. Also, Figure 7(b) shows that the strategy that combines education and quarantine has the same effect as the one combining double vaccination and quarantine, whereas the strategy that combines double vaccination and education has less impact on minimizing the proportion of the symptomatic infectious population. From Figure 7(c), one can see that to attain a maximum number of recovered proportions. The strategy combining double-vaccination and quarantine should be adopted, else one can adopt a strategy combining double-vaccination and education campaigns.

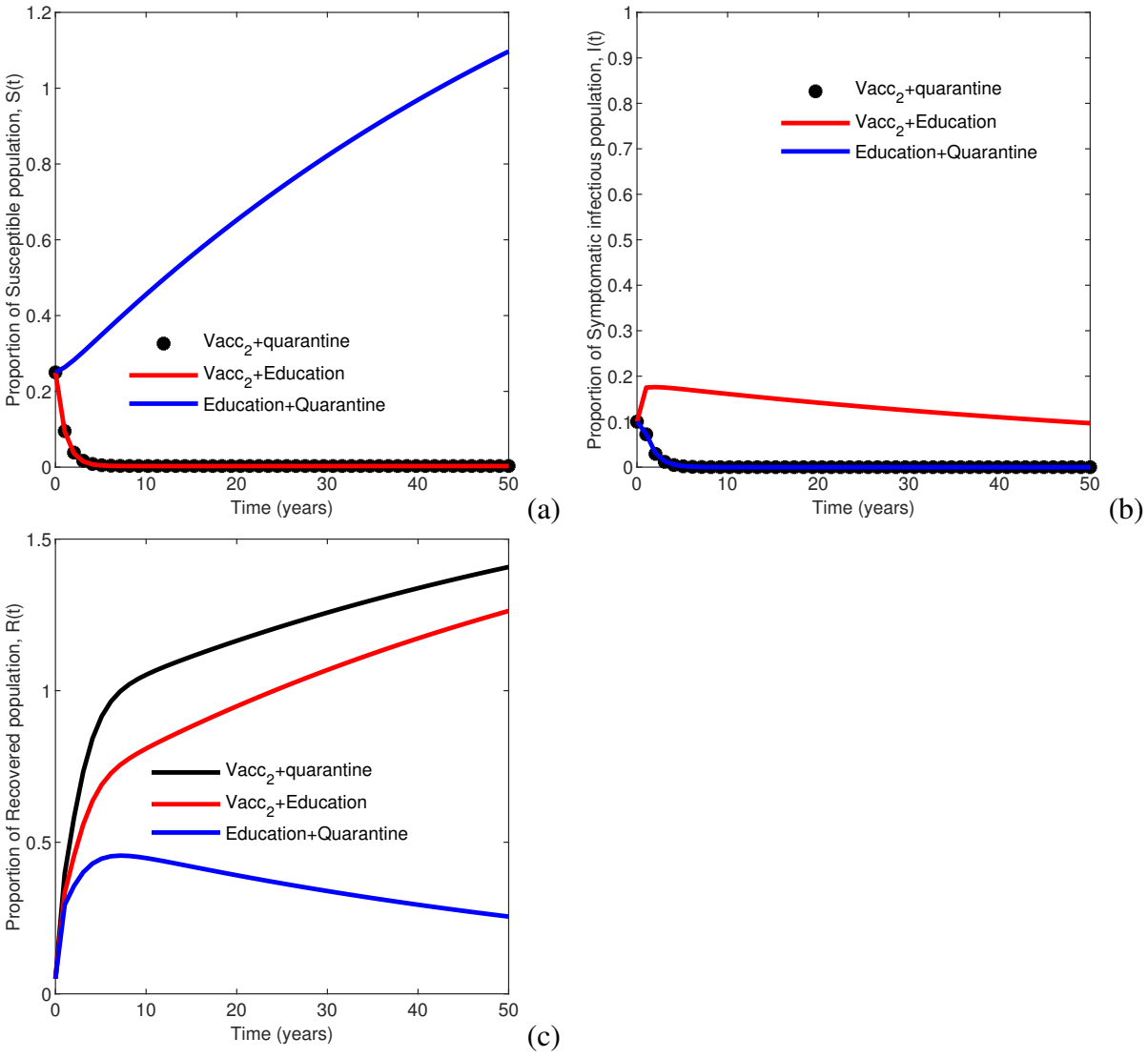

FIGURE 7. (a)-(c) show the impacts of two control strategies on susceptible, symptomatic infectious, and recovered populations.

4.6. Effects of a combination of three versus two control strategies. Figure 8(a) reveals that combining three strategies: double vaccination, education campaigns, and quarantine, has similar effects as the one that combines double vaccination and quarantine to decrease the proportion of the susceptible population. However, combining education campaigns and quarantine has no direct effect on decreasing the number of susceptible individuals. Figure 8(b) shows that as double-vaccination, education campaigns, and quarantine coverage increase, the proportion of exposed individuals decreases, which has the same impact as a combination of education and quarantine, so one can adopt one among these two combination control strategies to reduce the same number of exposed individuals. Also, Figure 8(c) demonstrates that the approach that three controls have the same effect as the one combining education and quarantine on minimizing the number of symptomatic infectious. In contrast, the strategy that combines double vaccination and education has less impact on minimizing the proportion of the symptomatic infectious population. Figure 8(d)shows that a maximum number of recovered proportions is attained when the strategy combines double-vaccination, education campaigns, quarantine, or the one that combines double-vaccination and quarantine is opted for.

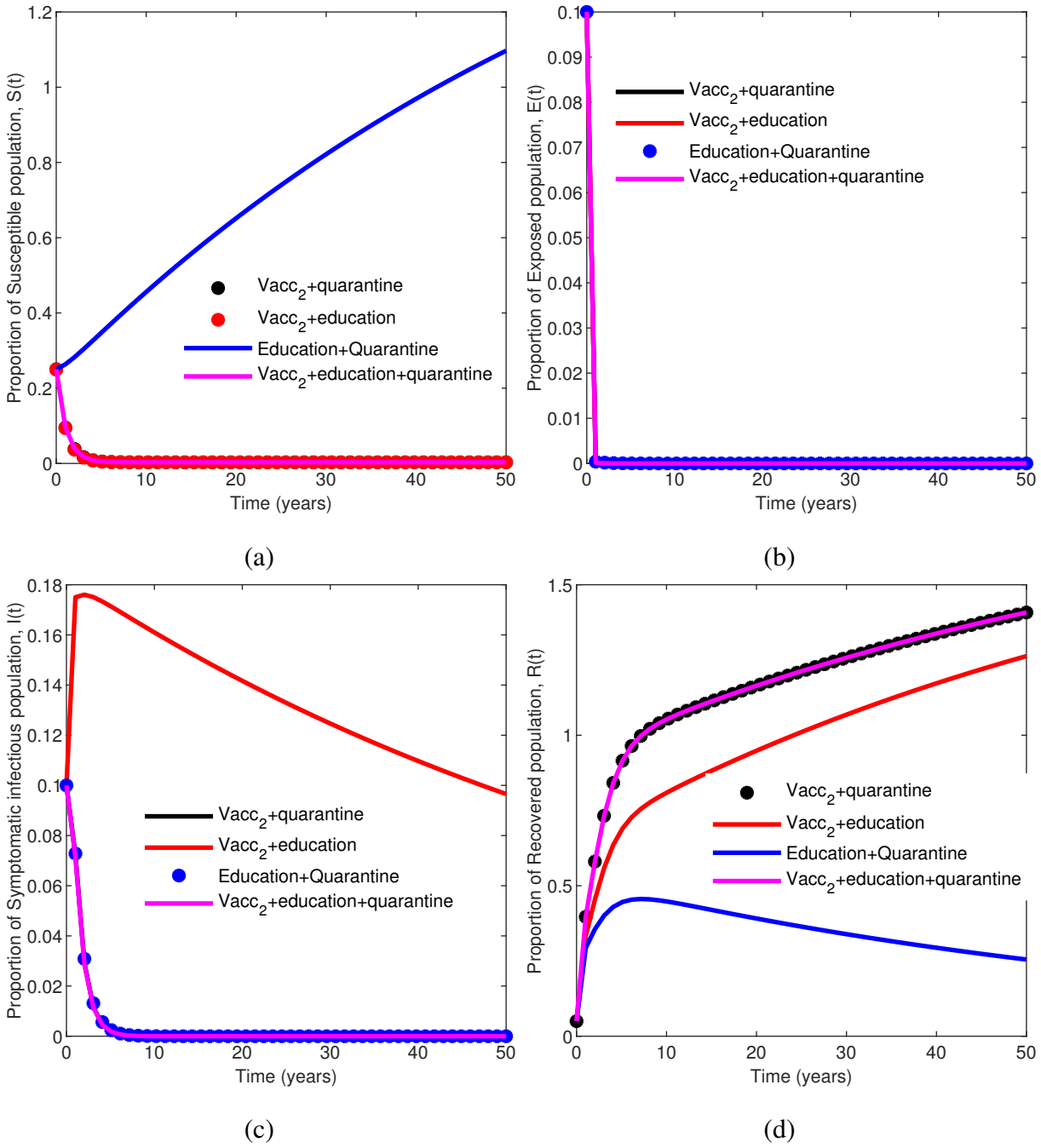

FIGURE 8. (a)-(d) show the impacts of three control strategies on susceptible, exposed, symptomatic infectious, and recovered populations.

5. CONCLUSIONS

This paper formulated a deterministic mathematical model for the transmission dynamics of mumps that incorporates three control measures: double vaccination, public health education campaign, and quarantine. The effective reproduction number was computed through the next-generation matrix method and was used to establish the stability of the equilibrium points. It was found that the model system has two equilibrium points, namely, the disease-free equilibrium and endemic equilibrium points. It was also proved that the disease-free equilibrium is locally asymptotically stable when $R_e < 1$ and unstable otherwise. The endemic equilibrium point was globally asymptotically stable when $R_e > 1$.

The findings from sensitivity analysis imply that to minimize mumps infection: vaccine efficacy for the first dose (ω) should be kept high, ensure that a large proportion of immigrants are vaccinated (τ), the general public should avoid traveling to regions where the mumps is endemic (Λ), symptomatic infectious individuals should be quarantined (σ) to limit further spread of infection, individuals should determine unnecessarily close contact with symptomatic infectious or carriers to lower the risk of the mumps transmission rate (β_1, β_2), education campaign (ψ) about mumps disease should be increased among the population, this will help people to understand details about the disease and thus avoid its risks. Also, individuals who missed the first-dose vaccine (ε) should be encouraged to receive the first-dose vaccine (MMR1) at any time. This will help to reduce risks associated with mumps. Furthermore, it has been reported by some scholars that the first dose of vaccine alone is not sufficient to eliminate mumps [5]. Thus, there is a need to receive a double-dose vaccine (θ), which guarantees permanent body immunity.

On the other hand, numerical simulations of the model indicate that whenever the single control strategies are implemented, then double-vaccination is the best strategy to reduce mumps transmission for the community (see Figure 4(c)). Also, when consideration is for the combinations of two controls, then the best combination is double-vaccination and quarantine (see Figure 7(c)). However, when consideration is for the combinations of three controls, then the best combination is double-vaccination, education campaign, and quarantine (see Figure 8(d)). Therefore, it can be concluded that vaccination is an essential option for controlling mumps

transmission, whether implemented singly, in two combinations, or in three combinations with other controls. The study might be helpful for policymakers and public health practitioners since it has proposed double-dose vaccination, public health awareness, and quarantine as possible measures to control mumps in the community.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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