sciendo

DOI: 10.2478/bile-2023-0006

Biometrical Letters
Vol. 60 (2023), No. 1, 77-95

Mathematical model for the transmission of mumps and its optimal control

Emmanuel Chidiebere Duru, Michael Chimezie Anyanwu

Department of Mathematics, Michael Okpara University of Agriculture, Umudike, Pmb 7267, Umuahia, Abia State, Nigeria, duru.emmanuel@mouau.edu.ng, manyanwu71@yahoo.com

SUMMARY

Mumps is a viral contagious disease associated with puffy cheeks and tender and swollen jaw. It spreads through direct contact with saliva or respiratory droplets from the mouth, nose or throat of infected persons. In this work, we present a mathematical model which describes the dynamics of the disease in a human population. The model incorporates isolation and treatment of infected individuals as a control measure. It is shown that the disease-free equilibrium (DFE) is locally and globally asymptotically stable when the control reproduction number R_c is less than one. It is also shown that the model has a unique endemic equilibrium which exists when $R_c > 1$. The existence of a unique endemic equilibrium confirms the global stability of the DFE, and the absence of backward bifurcation in the model. Optimal control analysis is performed on the model to obtain the proportion of infected humans to be isolated for optimal control of the disease. Plots are presented to show the dynamics of the disease in the presence of the control measures.

Key words: Mumps, Mathematical model, Control reproduction number, Optimal control

1. Introduction

Mumps is a viral contagious disease (CDC, 2021; NHS, 2021) and spreads through direct contact with saliva or respiratory droplets from the mouth, nose or throat. Infected persons can spread the virus by coughing, sneezing or talking, sharing items that are contaminated with saliva such as water bottles or cups, and close-contact activities with others such as playing sports, dancing or kissing (Mayo, 2023; Cleveland, 2023; Ikhisemojie, 2016). Mumps is associated with puffy cheeks and tender and swollen jaw, which are caused by the swelling of the salivary glands under the ears on one or both sides, often referred to as parotitis

(CDC, 2021; Mayo, 2023). Other symptoms which may appear before the swelling of the parotid glands include fever, muscle aches, headaches, tiredness, and loss of appetite (NHS, 2021; Kalen and Mcleod, 1977). The symptoms usually appear within 16–18 days of infection (CDC, 2021; Cleveland, 2023). Persons with mumps can be asymptomatic and may not know they have the disease, while some experience mild symptoms such as cold (Kalen and Mcleod, 1977). Very rarely mumps leads to complications, which may include swelling of the ovaries or testicles in persons after puberty, meningitis when the virus moves to the outer layer of the brain, febrile convulsions, arthritis, and orchitis (Cleveland, 2023; Walker et al., 1980; Mortimer, 1978).

A person with mumps should limit their contact with others during the period of infectiousness. Mumps can spread from infected persons to others from a few days before the swelling of their salivary glands to about five days after the start of the swelling (CDC, 2021; Cleveland, 2023). The spread of mumps can be prevented by regular washing of the hands with soap, proper disposal of used tissues, and isolation for at least five days after the appearance of the first symptoms (NHS, 2021; Mayo, 2023). Mumps has no cure, but the infection usually passes within 1 or 2 weeks. Treatment is usually given to relieve symptoms, and may include having sufficient bed rest and fluids, use of painkillers such as ibuprofen and paracetamol, and application of warm or cool compresses to the swollen glands to relieve pain (Ikhisemojie, 2016; RCGP, 1974). Mumps can be prevented through vaccination using the MMR vaccine during childhood (NHS, 2021; Cleveland, 2023; Ikhisemojie, 2016). Patients who have suffered from mumps usually develop life-long immunity against further infection (Mayo, 2023; Galbraith et al., 1984). Vaccination against mumps was first introduced in the United States in 1967, and has resulted in a decrease of about 97% in the rate of infection (Anderson et al., 1987).

Beard et al. (1977) carried out a population study of mumps orchitis over a 40-year period in Rochester, Minnesota, and discovered that there was no increase in genitourinary malformations in male offspring conceived after the occurrence of mumps orchitis in their fathers, while a few had testicular neoplasms and some atrophic testis after the occurrence of orchitis. Wagenvoort

et al. (1980) worked on the epidemiology of mumps in the Netherlands, where a neutralizing antibody against the mumps virus in a Dutch population group was discovered by a plaque reduction technique that proved reproducible, sensitive and specific. Anderson et al. (1987) worked on a preliminary analysis of the predicted impact of the introduction of a mumps vaccine to children based on epidemiological data in the UK. Donaghy and Friederichs (2006) showed in their work that mumps was primarily a risk for the 13–25 year age group, because they had not been offered two routine doses of the MMR vaccine, and transmission was heightened by increased social interactions. Fanoy et al. (2011) investigated the potential of vaccinated people to spread the disease caused by a genotype D mumps virus strain to close contacts in the Netherlands. They compared mumps viral titers of oral fluid specimens obtained by quantitative PCR from vaccinated (n = 60) and unvaccinated (n = 111) mumps patients. They also investigated the occurrence of mumps infection among the household contacts of vaccinated mumps patients. They discovered that viral titers are higher for unvaccinated patients than for vaccinated patients during the first three days after onset of the disease. They also discovered that while no symptomatic cases were reported among the household contacts (n = 164) of vaccinated mumps patients (n = 36), there were cases with serological evidence of asymptomatic infection among vaccinated household contacts (9 of 66 vaccinated siblings). For two of these siblings, the vaccinated index patient was the most probable source of infection. This work showed that there is a risk of a close contact becoming infected by vaccinated patients.

Li et al. (2017) designed and analyzed long-term mumps surveillance data in an SVEILR (susceptible–vaccinated–exposed–severely infectious–mildly infectious–recovered) dynamic transmission model with optimized parameter values to describe the dynamics of mumps infections in China. They carried out sensitivity analysis of the basic reproduction number and the endemic equilibrium to evaluate the effectiveness of mumps control measures. They proposed an increase in the vaccine coverage and two doses of MMR (measles, mumps and rubella) vaccines to be freely given in China. Kitano (2018) looked at the dynamic transmission model of routine mumps vaccination in Japan. They calculated the

incremental cost-effectiveness ratio compared with a current programme over a projected 50-year period. They created a Japanese population model and performed dynamic simulation to estimate the number of patients enrolled in the current programme, the routine one-dose programme, and the routine two-dose programme over the next 50 years using the Berkeley Madonna program. Their work confirmed that the routine two-dose vaccination programme was more cost-effective and QALY-saving than either the one-dose programme or the current programme. Bai et al. (2021) worked on a model of mumps transmission with quarantine measures. Their work showed that the disease-free equilibrium point is globally asymptotically stable (GAS) if the reproduction number is less than 1 and the endemic equilibrium point is GAS if the reproduction number is greater than 1. Their results revealed that mumps can be eliminated from the community if the reproduction number is less than or equal to one and will be persistent if the reproduction number is greater than 1, and quarantine measures can also effectively control the transmission of mumps.

In this work, we looked at the dynamics of transmission of mumps infection, incorporating the effect of effective isolation and treatment of infectious humans in a population. We performed control optimization on the model to obtain the proportion of infected humans to be isolated for optimal control of the disease.

Mathematical modelling of infectious diseases has helped to study the mechanisms through which diseases spread, to make predictions on the future course of an outbreak and to evaluate methods of controlling them. It can project how infectious diseases progress, showing the likely outcome and helping inform public health interventions. It makes use of assumptions in defining the mathematical model of the system and then follows various analytical and simulation methods to obtain a solution which will be useful in making predictions about the system. Mathematical modelling plays a major role in public health research in epidemiology; it has been said that mathematical modelling helps in interpreting findings in epidemiology and clearly identifying the various different elements that could cause a severe epidemic or serious pandemic (Song et al., 2019; Campos et al., 2021). The goal of this paper is to present a mathematical model for the disease transmission dynamics of mumps,

incorporating effects of isolation and treatment for effective disease control in Nigeria.

2. Mathematical model

The mathematical model was derived considering the disease compartment in Fig. 1. It is an SEIR-like model where $S(t)$ represents the susceptible human population, $E(t)$ is the exposed human population, $I_s(t)$ represents symptomatic infectious humans, $I_A(t)$ represents asymptomatic infectious humans, $I_{IS}(t)$ represents isolated and treated symptomatic humans, and $R(t)$ represents recovered humans.

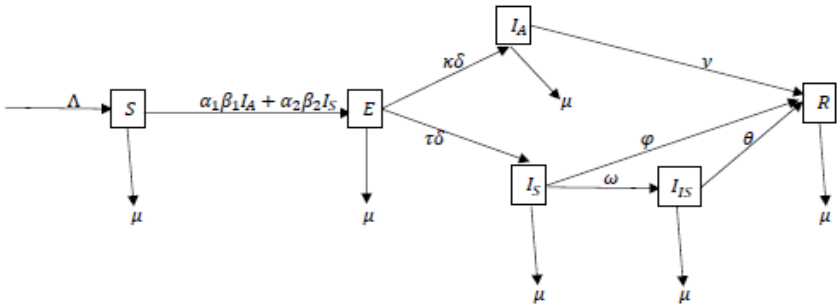


Figure 1. Flow diagram for the transmission of mumps

The susceptible human population S is increased by the level of recruitment Λ and reduced by the natural death rate μ and the number of persons $\left(\frac{\alpha_1\beta_1 I_A+\alpha_2\beta_2 I_S}{N}\right)S$ infected at time t, where α_1 is the rate at which S have contact with I_A, α_2 is the rate at which S have contact with I_S, β_1 is the probability of transmission of the virus from I_A to S, and β_2 is the probability of transmission of the virus from I_S to S. The exposed human population is increased by the number of persons $\left(\frac{\alpha_1\beta_1 I_A+\alpha_2\beta_2 I_S}{N}\right)S$ infected at time t. It is reduced by natural death and $((\kappa+\tau)\delta+\mu)E$, where κ is the proportion of exposed humans who are asymptomatically infected, τ is the proportion of exposed humans who are symptomatically infected, and δ is the incubation rate of the disease. We assume that the proportion ω of I_S is isolated and

treated at time t, while φ is the proportion that is not isolated. I_A, I_S and I_{IS} recover from the disease at the rates ν, φ and θ respectively.

Using the flow diagram in Fig. 1 and the description of the disease dynamics, we arrive at the following system of nonlinear ordinary differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \frac{\alpha_1\beta_1 I_A}{N}S - \frac{\alpha_2\beta_2 I_S}{N}S - \mu S,\\
\frac{dE}{dt} &= \frac{\alpha_1\beta_1 I_A}{N}S + \frac{\alpha_2\beta_2 I_S}{N}S - ((\kappa+\tau)\delta + \mu)E,\\
\frac{dI_A}{dt} &= \kappa\delta E - (\nu+\mu)I_A,\\
\frac{dI_S}{dt} &= \tau\delta E - (\omega+\varphi+\mu)I_S,\\
\frac{dI_{IS}}{dt} &= \omega I_S - (\theta+\mu)I_{IS},\\
\frac{dR}{dt} &= \theta I_{IS} + \varphi I_S + \nu I_A - \mu R. \qquad (1)
\end{aligned}$$

where $S(0) = S^0$, $E(0) = E^0$, $I_S(0) = I_S^0$, $I_A(0) = I_A^0$, $I_{IS}(0) = I_{IS}^0$ $R(0) = R^0$, $\varphi = 1-\omega, \tau = 1-\kappa$, $N = S + E + I_S + I_A + I_{IS} + R$.

We assume that the solution to (1) exists in the region Ω described by

$$\{S, E, I_S,\ I_A,\ I_{IS},\ R\} \in R^6 \,|S + E + I_S + I_A + I_{IS} + R \leq \frac{\Lambda}{\mu}.$$

Table 1. Description of parameters

Parameters	Description
Λ	Level of recruitment
μ	Natural death
α_1	Contact rate with asymptomatic humans
α_2	Contact rate with symptomatic humans
β_1	Rate of transmission from contact with asymptomatic humans
β_2	Rate of transmission from contact with symptomatic humans
δ	Incubation rate
κ	Level of movement into asymptomatic humans
τ	Level of movement into symptomatic humans
ω	Rate of isolation
φ	Proportion of un-isolated symptomatic humans that recover with time
θ	Rate of recovery of isolated and treated humans
ν	Rate of recovery of asymptomatic humans

2.1. Positivity and boundedness of solutions

Lemma 1: Let the initial data set for the model be $\{S^0 > 0, E^0 > 0, I_S^0 > 0,\ I_A^0 > 0,\ I_{IS}^0 > 0,\ R^0 > 0\}$. Then the solution set $\{S(t), E(t), I_S(t), I_A(t), I_{IS}(t), R(t)\}$ of the model with the given initial data will remain positive and bounded for all times $t > 0$.

Proof: It can be shown from eqn. (1) that

$$\frac{dS}{dt} \geq -\left(\frac{\alpha_1\beta_1 I_A}{N} + \frac{\alpha_2\beta_2 I_S}{N} + \mu\right) S,$$

$$\frac{dE}{dt} \geq -((\kappa + \tau)\delta + \mu)E,$$

$$\vdots$$

$$\frac{dR}{dt} \geq -\mu R\,. \tag{2}$$

Hence, solving the differential inequality (2) with the given initial solutions gives

$$S \geq S^0 e^{-\left(\frac{\alpha_1\beta_1 I_A}{N} + \frac{\alpha_2\beta_2 I_S}{N} - \mu\right)t} > 0\,,$$

$$E \geq E^0 e^{-((\kappa+\tau)\delta+\mu)t}\,,$$

$$\vdots$$

$$R \geq R^0 e^{-\mu t}.$$

Hence, given initial solutions that are individually positive, the solution to the model system remains positive $\forall\ t > 0$. Further, we have that the total human population N satisfies the equation

$$\frac{dN}{dt} = \Lambda - \mu N.$$

Hence, as $t \to \infty$, $0 < N(t) \leq \frac{\Lambda}{\mu}$, showing that the human population is always bounded. Therefore, the region Ω is positively invariant and is an attractor of all positive solutions of the system.

2.2. Local stability of the disease-free equilibrium

The disease-free equilibrium (DFE), $E^0 = \left(\frac{\Lambda}{\mu}, 0,0,0,0,0\right)$ of (1) is locally asymptotically stable if the eigenvalues of the Jacobian matrix of the model of

the system at E^o are negative or have negative real parts. Let $J(E^0)$ denote the Jacobian matrix evaluated at E^0; then

$$J(E^0) = \begin{bmatrix} -\mu & 0 & -\alpha_1\beta_1 & -\alpha_2\beta_2 & 0 & 0 \\ 0 & -(\kappa+\tau)\delta+\mu) & \alpha_1\beta_1 & \alpha_2\beta_2 & 0 & 0 \\ 0 & \kappa\delta & -(\nu+\mu) & 0 & 0 & 0 \\ 0 & \tau\delta & 0 & -(\omega+\varphi+\mu) & 0 & 0 \\ 0 & 0 & 0 & \omega & -(\theta+\mu) & 0 \\ 0 & 0 & \nu & \varphi & \theta & -\mu \end{bmatrix}$$

The eigenvalues of $J(E^0)$ are $-\mu, -\mu, -(\theta+\mu)$, and we consider the eigenvalues of the submatrix J_1 obtained by deleting the rows and columns containing these eigenvalues:

$$J_1(E^0) = \begin{bmatrix} -\big((\kappa+\tau)\delta+\mu\big) & \alpha_1\beta_1 & \alpha_2\beta_2 \\ \kappa\delta & -(\nu+\mu) & 0 \\ \tau\delta & 0 & -(\omega+\varphi+\mu) \end{bmatrix}$$

An eigenvalue λ of J_1 satisfies the cubic equation

$$Q_3\lambda^3 + Q_2\lambda^2 + Q_1\lambda + Q_0 = 0,$$

where

$$Q_3 = 1,$$

$$Q_2 = ((\kappa+\tau)\delta+\mu) + (\nu+\mu) + (\omega+\varphi+\mu),$$

$$Q_1 = \big((\kappa+\tau)\delta+\mu\big)(\nu+\mu)[1-R_c] + \big((\kappa+\tau)\delta+\mu\big)(\omega+\varphi+\mu)\times [1-R_c] + (\nu+\mu)(\omega+\varphi+\mu) + \frac{\alpha_1\beta_1\kappa\delta(\omega+\varphi+\mu)}{(\nu+\mu)} + \frac{\alpha_2\beta_2\tau\delta(\nu+\mu)}{(\omega+\varphi+\mu)},$$

$$Q_0 = ((\kappa+\tau)\delta+\mu)(\nu+\mu)(\omega+\varphi+\mu)[1-R_c]$$

where $R_c = \frac{\alpha_1\beta_1\kappa\delta(\omega+\varphi+\mu)+\alpha_2\beta_2\tau\delta(\nu+\mu)}{(\nu+\mu)\big((\kappa+\tau)\delta+\mu\big)(\omega+\varphi+\mu)}$.

Using the Routh–Hurwitz criterion, we require that $Q_2 > 0, Q_1 > 0, Q_0 > 0 \; and \; Q_2Q_1 > Q_0$ to have all the eigenvalues negative and guarantee local stability. Since $Q_3 > 0$ and $Q_2 > 0$, $Q_1 > 0$ and $Q_0 > 0$ if $R_c < 1$. Also, $Q_2Q_1 > Q_0$ as required. This shows that the Routh–Hurwitz criterion is satisfied if $R_c < 1$ and guarantees that all eigenvalues of the polynomial will be negative if $R_c < 1$. Hence, the disease-free equilibrium will be locally asymptotically stable

if $R_c < 1$. The quantity $R_c = \frac{\alpha_1\beta_1\kappa\delta(\omega+\varphi+\mu)+\alpha_2\beta_2\tau\delta(\nu+\mu)}{(\nu+\mu)((\kappa+\tau)\delta+\mu)(\omega+\varphi+\mu)}$ is the control reproduction number of the disease. It gives the average number of secondary infectious cases caused by a single infection in the total susceptible population in the presence of the control measure (Mahato and Mishra, 2018). If $R_c < 1$, then the control measure is effective; otherwise it is ineffective.

2.3. Existence of endemic equilibrium point

Theorem 1. The positive endemic equilibrium exists and is unique if and only if $R_c > 1$.

The endemic equilibrium point (EEP) is a steady-state solution where the disease persists in the population (i.e. all state variables are positive) with $EEP = (S^*, E^*, I_S^*, I_A^*, I_{IS}^*, R^*)$. The steady-state solutions are given by

$$S^* = \frac{\Lambda}{\left(\frac{\alpha_1\beta_1 I_A^*}{N^*}+\frac{\alpha_2\beta_2 I_S^*}{N^*}+\mu\right)}, \quad E^* = \frac{(\alpha_1\beta_1 I_A^*+\alpha_2\beta_2 I_S^*)S_h^*}{N^*(\kappa+\tau)\delta+\mu)}, \quad I_A^* = \frac{\kappa\delta E^*}{(\nu+\mu)}, \quad I_S^* = \frac{\tau\delta E^*}{(\omega+\varphi+\mu)},$$
$$I_{IS}^* = \frac{\omega I_S^*}{(\theta+\mu)}, \quad R_h^* = \frac{\theta I_{IS}^*+\varphi I_S^*+\nu I_A^*}{\mu}.$$

Substituting these variables into E^* where applicable and simplifying gives $((\kappa+\tau)\delta+\mu)[\alpha_1\beta_1\kappa\delta(\omega+\varphi+\mu)+\alpha_2\beta_2\tau\delta(\nu+\mu)]E^{*2}+[\Lambda((\kappa+\tau)\delta+\mu)(\nu+\mu)(\omega+\varphi+\mu)-\Lambda\alpha_1\beta_1\kappa\delta(\omega+\varphi+\mu)-\Lambda\alpha_2\beta_2\tau\delta(\nu+\mu)]E^*=0,$ with $E^*=0$ or $((\kappa+\tau)\delta+\mu)[\alpha\beta_1\kappa\delta(\omega+\varphi+\mu)+\alpha\beta_2\tau\delta(\nu+\mu)]E^*+[\Lambda((\kappa+\tau)\delta+\mu)(\nu+\mu)(\omega+\varphi+\mu)-\Lambda\alpha\beta_1\kappa\delta(\omega+\varphi+\mu)-\Lambda\alpha\beta_2\tau\delta(\nu+\mu)]=0.$

Then we will have $((\kappa+\tau)\delta+\mu)R_cE^*+\Lambda(1-R_c)=0$ with

$$E^* = \frac{\Lambda}{((\kappa+\tau)\delta+\mu)R_c}[R_c-1].$$

Hence, $E^* > 0$ if $R_c > 1$. Thus, the EEP exists and is unique.

2.4. Global stability of the disease-free equilibrium

Using the method of Lyapunov given by Yusuf (2021) to test for global stability, we construct a Lyapunov function V to be

$$V(t, S, E, I_A, I_S, I_{IS}, R) = C_1E + C_2I_A + C_3I_S.$$

Then,

$$\frac{dV}{dt} = C_1\left(\frac{dE}{dt}\right) + C_2\left(\frac{dI_A}{dt}\right) + C_3\left(\frac{dI_S}{dt}\right) = C_1\left[\frac{\alpha_1\beta_1 I_A}{N}S + \frac{\alpha_2\beta_2 I_S}{N}S - \big((\kappa + \tau)\delta + \mu\big)E\right] + C_2[\kappa\delta E - (\nu + \mu)I_A] + C_3[\tau\delta E - (\omega + \varphi + \mu)I_S] =$$

$$[C_2\kappa\delta - C_1((\kappa + \tau)\delta + \mu) + C_3\tau\delta]E + [C_1\alpha_1\beta_1 - C_2(\nu + \mu)]I_A +$$

$$[C_1\alpha_2\beta_2 - C_3(\omega + \varphi + \mu)]I_S$$

Taking $C_1 = 1$, $C_2 = \frac{\alpha\beta_1(\omega+\varphi+\mu)}{(\omega+\varphi+\mu)(\nu+\mu)}$ and $C_3 = \frac{\alpha\beta_2(\nu+\mu)}{(\nu+\mu)(\omega+\varphi+\mu)}$, we have $\frac{dV}{dt} = ((\kappa + \tau)\delta + \mu)[R_c - 1]E$. It should be noted that $\frac{dV}{dt} = 0$ only when $E = 0$. Moreover, the substitution of $E = 0$ into the model system of equations gives $S = \frac{\Lambda}{\mu}$ as $t \to \infty$. We see that $\frac{dV}{dt} < 0$ if $R_c < 1$. Thus, by Lassalle's invariance principle, the disease-free equilibrium is globally asymptotically stable when $R_c < 1$ (Lassalle and Lefschetz, 1976).

3. Optimal control of mumps

In this case, we assume that the rate of isolation and treatment $\omega(t)$ is not constant but is a function of time: $\omega = \omega(t)$. Therefore, we consider an optimal control problem with objective function given by

$$J(x;\omega) = \int_0^T A_1E(t) + A_2I_A(t) + A_3I_S(t) + \frac{A_4}{2}\omega^2(t)dt$$

where the constants A_i; $i = 1; 2; 3; 4$ are positive weights which help to balance each term in the integrand. The quadratic term $\frac{A_4}{2}\omega^2(t)$ represents the cost involved in isolating and treating those that are symptomatically infectious at any time *t*. In the above optimal control problem, we assume that the control parameter $\omega^*(t)$ is Lebesgue measurable with $a < \omega^*(t) \leq b, \forall t \in [0, t_f]$. Therefore, we seek an optimal control $\omega^*(t)$ such that

$$J(\omega^*(t)) = min\{J(\omega(t)) : \omega(t) \in \Omega\}$$

where the control set Ω is given by

$$\Omega = \{\omega(t) : \omega(t) \textit{ is piecewise continuous on } [0, T], a \leq \omega(t) \leq b\}$$

with a and b being nonnegative constants. As shown in Fleming and Rishel (1975), the existence of the optimal control $\omega^*(t)$ and the associated optimal solutions$(S^*, E^*, I_A^*, I_S^*, I_{IS}^*, R^*)$ is guaranteed since the state solutions are bounded as proved earlier, and the state system satisfies the Lipschitz property with respect to the state variables, since the solutions exist and are unique. It remains to show the convexity of the integrand, $L(t, E, I_A, I_S, \omega) = A_1E + A_2\, I_A + A_3I_S + \frac{1}{2}A_4\omega^2$, with respect to the control parameter ω in the set Ω. Therefore, we need to show that for $\lambda \in (0,1)$

$L(t, E, I_A, I_S, \lambda\omega + (1-\lambda)\omega') \leq \lambda\, L(t, E, I_A, I_S, \omega) + (1-\lambda)L(t, E, I_A, I_S, \omega').$

Note that

$$\mathrm{L(t, E, I_A, I_S, \lambda\omega\ + (1-\lambda)\omega') = A_1E + A_2I_A + A_3I_S + \tfrac{1}{2}A_4(\lambda\omega + (1-\lambda)\omega')^2} \qquad (3)$$

and

$$\lambda\, L(t, E, I_A, I_S, \omega) + (1-\lambda)L(t, E, I_A, I_S, \omega') = A_1E + A_2I_A + A_3I_S + \tfrac{1}{2}A_4\lambda\omega^2 + \tfrac{1}{2}A_4(1-\lambda)\omega'^2) \qquad (4)$$

This means that from (3) and (4), we must prove that

$$(\lambda\omega + (1-\lambda)\omega')^2 \leq \lambda\omega^2 + (1-\lambda)\omega'^2.$$

Expanding $(\lambda\omega + (1-\lambda)\omega')^2$ gives

$$\begin{aligned}(\lambda\omega + (1-\lambda)\omega')^2 &= \lambda^2\omega^2 + 2\lambda(1-\lambda)\omega\omega' + (1-\lambda)^2\omega'^2\\ &= \lambda(\lambda - 1 + 1)\omega^2 + 2\lambda(1-\lambda)\omega\omega' + (1-\lambda)(1-\lambda)\omega'^2\\ &= \lambda\omega^2 + (1-\lambda)\omega'^2 - \lambda(1-\lambda)[\omega^2 - 2\omega\omega' + \omega'^2]\\ &= \lambda\omega^2 + (1-\lambda)\omega'^2 - \lambda(1-\lambda)[\omega - \omega']^2\\ &\leq \lambda\omega^2 + (1-\lambda)\omega'^2.\end{aligned}$$

This proves the convexity of the integrand on the singleton Ω.

3.1. The Pontryagin maximum principle

The Pontryagin maximum principle converts a constrained control problem to an unconstrained one by introducing an additional variable, called an adjoint

variable, to the original problem. Generally, given a state x and a control u, the Pontryagin maximum principle stated below provides the condition under which the pair (x^*, u^*) is optimal.

Theorem 1: (Pontryagin Maximum Principle): Suppose u^* and x^* are optimal for the control problem

$$\min \int_{t_0}^{t_f} f(x(t), u(t))dt$$

subject to

$$\frac{dx(t)}{dt} = g(x(t), u(t)), \quad x(t_0) = x_0.$$

Then there exists a piecewise differentiable function ϕ(t) called the adjoint variable or costate such that the Hamiltonian defined by

$$H(t, x, u, \phi) = f(t, x(t), u(t)) + \phi g(t, x(t), u(t)) \tag{5}$$

satisfies

$$H(t, x, u, \phi) \leq H(t, x^*, u^*, \phi)$$

$$\frac{dH(t, x, u, \phi)}{du} = 0$$

$$\frac{d\phi(t)}{dt} = \frac{dH(t, x^*, u^*, \phi)}{dx}$$

$$\frac{dx(t)}{dt} = \frac{dH(t, x^*, u^*, \phi)}{d\phi} \tag{6}$$

for all controls u at each time t. The adjoint variable φ satisfies the transversality condition φ(tf) = 0.

From (5), the Hamiltonian is given by

$$H = A_1E + A_2I_A + A_3I_S + \frac{1}{2}A_4\omega^2(t) + \phi_1(t)(\Lambda - \rho(t)S(t) - \mu S(t))$$

$$+\phi_2(t)(\rho(t)S(t) - (\delta + \mu)E(t)) + \phi_3(t)(\kappa\delta E - (\nu + \mu)I_A)$$

$$+\phi_4(t)((1-\kappa)\delta E-(1+\mu)I_S)+\phi_5(t)(\omega I_S-(\theta+\mu)I_{IS})$$
$$+\phi_6(t)(\theta I_{IS}+(1-\omega)I_S+\nu I_A-\mu R)$$

where the adjoint variables $\phi i(t), i = 1,2,\cdots,6$, satisfy the system of ordinary differential equations

$$\frac{d\phi_1}{dt}=\rho(t)\left(\frac{N-S}{N}\right)(\phi_1-\phi_2)+\mu\phi_1$$
$$\frac{d\phi_2}{dt}=\frac{\rho(t)S}{N}(\phi_1-\phi_2)+(\delta+\mu)\phi_2-\kappa\delta\phi_3-(1-\kappa)\delta\phi_4-A_1$$
$$\frac{d\phi_3}{dt}=\alpha\beta_1 S\left(\frac{N-I_A}{N^2}\right)(\phi_1-\phi_2)+(\nu+\mu)\phi_3-\nu\phi_6-A_2$$
$$\frac{d\phi_4}{dt}=\alpha\beta_2 S\left(\frac{N-I_S}{N^2}\right)(\phi_1-\phi_2)+(1+\mu)\phi_4-\omega\phi_5-(1-\omega)\phi_6-A_3$$
$$\frac{d\phi_5}{dt}=\frac{\rho(t)S}{N}(\phi_1-\phi_2)+(\theta+\mu)\phi_5-\theta\phi_6$$
$$\frac{d\phi_6}{dt}=\frac{\rho(t)S}{N}(\phi_1-\phi_2)+\mu\phi_6.$$

with the transversality condition $\phi_i(T) = 0, i = 1,2,\cdots,10$. Using the optimality condition (6), we obtain that $\omega^*(t) = \frac{(\phi_6(t)-\phi_5(t))I_S(t)}{A_4}$. Therefore, the optimal control parameter can be characterized as

$$\omega^*(t) = max\left(a, min\left(b, \frac{(\phi_6(t)-\phi_5(t))I_S(t)}{A_4}\right)\right) \quad (7)$$

Hence, (7) gives the proportion of symptomatically infectious humans that must be isolated and treated for optimal control of the disease.

4. Numerical experiments and discussion

Here, we obtain a numerical solution to the optimal control problem using a forward-backward sweep technique. This method involves solving the optimal

state equation using the forward Runge–Kutta order 4 method with an initial guess for the optimal control parameter. This is followed by solving the adjoint system of equations with the backward Runge–Kutta order 4 method, starting with the transversality conditions $\phi_i(T) = 0$. The optimal control is updated with the current state and adjoint solutions at the end of each iteration. The iteration is repeated until a convergence criterion is reached. The initial solutions used are $S_0 = 500, E_0 = 50, I_A^0 = 8, I_S^0 = 27, I_{IS}^0 = 13, R^0 = 20$. We also used $A_1 = 10, A_2 = 100, A_3 = 5, A_4 = -10000, a = 0.1, b = 1$. The parameter values used in the numerical experiment were assumed from the disease dynamics as stated in the introduction. For example, the probability of disease transmission β_2 from symptomatic humans to susceptible humans is expected to be higher than the corresponding probability for asymptomatic humans, β_1. It is also expected that those who undergo treatment will recover faster than those who do not; hence the choice of values for θ and ν. Since the infection is not generally severe, we assumed that some of the un-isolated individuals φ will recover with time. Furthermore, the disease dynamics show that the number of symptomatic cases τ is larger than the number of asymptomatic cases κ.

Table 2. Description of parameters and values

Parameters	Values	Source
Λ	50	Assumed
μ	0.0002042	Anyanwu et al. (2020)
α_1	0.75	Assumed
α_2	0.75	Assumed
β_1	0.15	Assumed
β_2	0.75	Assumed
δ	0.0833	CDC, 2021
κ	0.250	Assumed
τ	0.745	Assumed
ω	0.12	Assumed
φ	0.0608	Assumed
θ	0.0714	Ikhisemojie, 2016 and RCGP, 1974
ν	0.0212	Assumed

The results of the numerical experiments are shown in Figures 2–10 below. We observe that the application of optimal control drastically reduced the rate of spread of the disease. Figures 2, 4, 6 and 8 show the graphs of exposed humans,

asymptomatic humans, symptomatic humans, and isolated and treated humans without optimal control. The various populations increase with time before experiencing a decline. The number of exposed humans reached over 500, asymptomatic humans reached 190, symptomatic humans reached over 150, while the isolated and treated humans reached over 180, before these various populations experienced a decline.

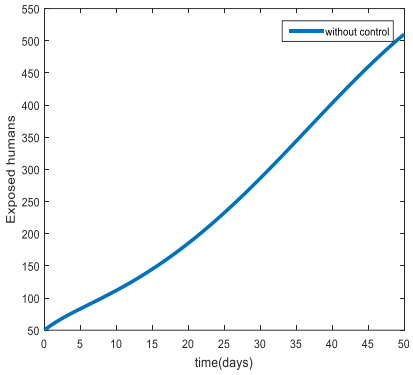


Figure 2. Graph of exposed humans without control

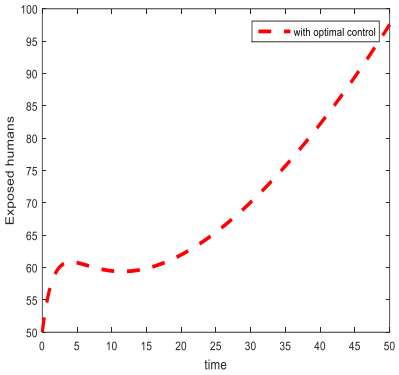


Figure 3. Graph of exposed humans with control

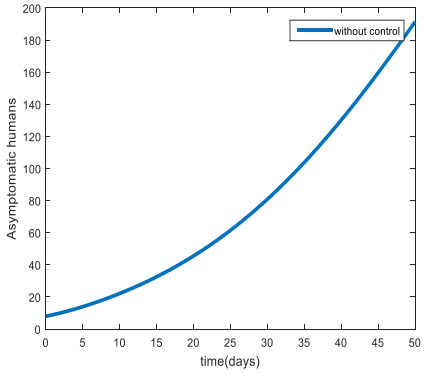


Figure 4. Graph of asymptomatic humans without control

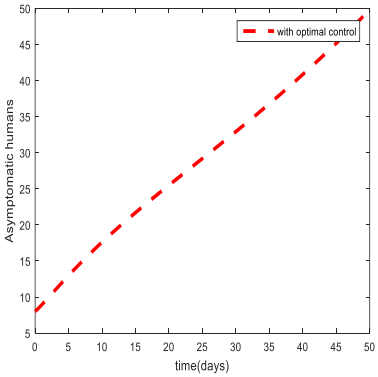


Figure 5. Graph of asymptomatic humans with control

Figures 3, 5, 7 and 9 show the results of the simulation with optimal control. We observe that with effective optimal isolation of infectious individuals, the populations of exposed, asymptomatically infectious and symptomatically infectious humans decrease over time. This is because optimal isolation of

symptomatically infectious humans reduces the rate of contact of infectious individuals with the susceptible population. This will cause the disease to be controlled with time. Fig. 9 gives the proportion of symptomatically infectious humans that need to be isolated and treated at any time t in order to optimally control the disease in a given human population. At the initial time, $t = 0$, no persons have been isolated since this is the point of commencement of the disease.

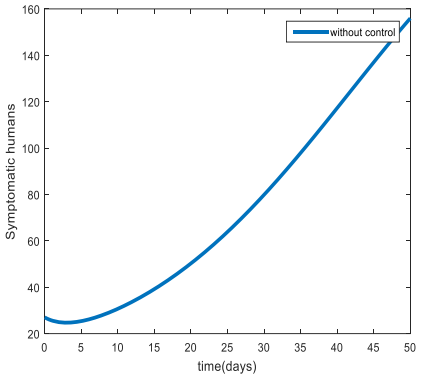


Figure 6. Graph of symptomatic humans without control

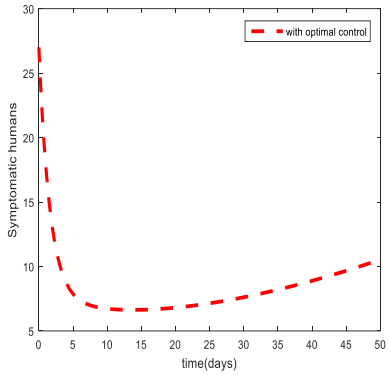


Figure 7. Graph of symptomatic humans with control

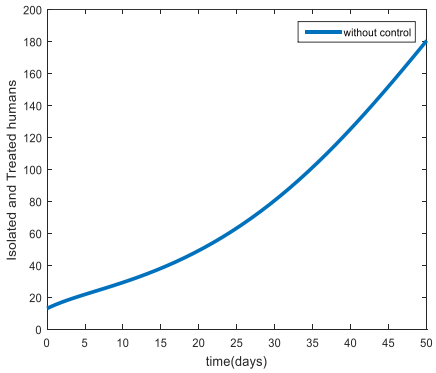


Figure 8. Graph of isolated humans without control

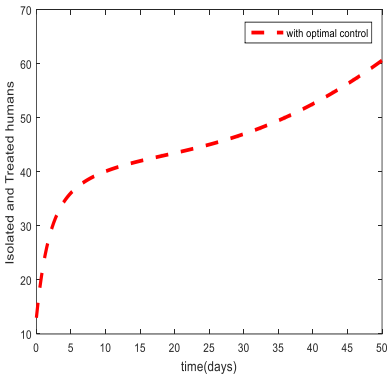


Figure 9. Graph of isolated humans without control

Hence, $\omega(0) = 0$. The proportion ω increases afterwards as t increases, but begins to decrease after reaching its peak. The increase occurs in the time when the number of infected persons is increasing, and hence a greater proportion of infectious humans needs to be isolated and treated for optimal control of the

disease. The point where the graph begins to decrease shows that the optimal control has effectively reduced the number of infectious humans, and hence those that should be isolated. With these reductions in the populations of infected and infectious individuals, mumps will not spread in the population; thus the disease will be effectively controlled.

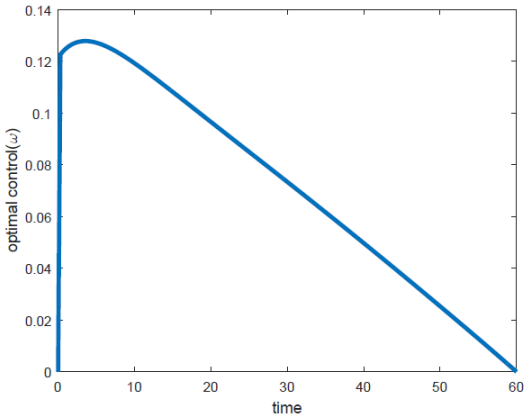


Figure 10. Graph of optimal control

5. Summary and conclusion

We have presented a mathematical model for the transmission dynamics of the disease of mumps and its optimal control. It was shown that the disease-free equilibrium is both locally and globally asymptotically stable when the control reproduction number $R_c < 1$. We also showed that a unique endemic equilibrium point of the model exists when $R_c > 1$. This confirms the global stability of the disease-free equilibrium, and the absence of backward bifurcation in the model. The application of optimal control methods in isolating infectious humans drastically reduced the spread of the disease.

We conclude that isolating infectious individuals effectively and treating them will help to control the spread of the disease and possibly eliminate it. All mathematical calculations and simulations were performed using Maple 14 and MATLAB 2015a.

REFERENCES

Anderson R.M., Crombie J.A., Grenfell, B.T. (1987): The epidemiology of mumps in the UK: a preliminary study of virus transmission, herd immunity and the potential impact of immunization. Epidemiology Infectious Disease, 99: 65–84.

Anyanwu M.C., Mbah G.C., Duru, E.C. (2020): On Mathematical Model for Zika Virus Disease Control with Wolbachia-Infected Mosquitoes. Journal of the Mathematical Association of Nigeria, 47(1): 35–54.

Bai Y., Wang X., Guo S. (2021): Global Stability of a Mumps Transmission Model with Quarantine Measure. Acta Mathematicae Applicatae Sinica, English Series, 37(4): 665–672.

Beard C.M., Benson R.C. Jr., Kelalis P.P., Elveback L.R., Kurland L.T. (1977): The incidence and outcome of mumps orchitis in Rochester, Minnesota, 1935–1974. Mayo Clinic Proceedings 52: 3–7.

Campos E.L., Cysne R.P., Madureira A.L., Mendes G.L. (2021): Multi-generational SIR Modeling: Determination of Parameters, Epidemiological Forecasting and Age-dependent Vaccination Policies. Infectious Disease Modelling, 6(1): 751–765.

Centre for Disease Control and Prevention (2021): Mumps: Cases and Outbreak. https://www.cdc.gov/mumps/index.html, retrieved January 12, 2023.

Donaghy M., Friederichs V. (2006): Increasing incidence of mumps in Scotland: Options for reducing transmission. Journal of clinical virology, 35(2): 121–129.

Fanoy E.B., Cremer J., Ferreira J.A., Dittrich S., Van Lier A. et al. (2011): Transmission of mumps virus from mumps-vaccinated individuals to close contacts. Vaccine-Elsevier 29: 9551–9556.

Fleming W.H., Rishel R.W. (1975): Deterministic and Stochastic Optimal Control. Springer Verlag.

Galbraith N.S., Youno S.E.J., Pusky J.J. Crombik D.L., Sparks I.P. (1984): Mumps surveillance in England and Wales 1962–1981. Lancet 91–94.

https://www. mayoclinic.org/diseases-conditions/mumps/diagnosis-treatment/drc-20375366, retrieved January 12, 2023.

https://www.clevelandclinic.org/health/diseases/15007-mumps. Retrieved January 12, 2023.

Ikhisemojie S. (2016): Dealing with mumps. punchng.com

Kalen A.E., McLeod D.A. (1977): Paramyxoviruses: comparative diagnosis of parainfluenza, mumps, measles and respiratory syncytial virus infections. In Comparative Diagnosis of Viral Diseases. I. Human and Related Viruses Part A. (ed. Kurstak, E. and Kurstak, C.), 503–607. New York, Academic Press.

Kitano T. (2018): Dynamic transmission model of routine mumps vaccination in Japan. Epidemiology and Infection 1–8. https://doi.org/10.1017/S0950268818003230

LaSalle J., Lefschetz S. (1976): The stability of dynamical systems. SIAM Philadelphia.

Li Y., Liu X., Wang L. (2017): Modelling the Transmission Dynamics and Control of Mumps in Mainland China. International Journal of Environmental Research and Public Health,15(33): 1–17.

Mahato B., Mishra B.K. (2018): Global Stability Analysis on the Transmission Dynamics of Zika Virus. International Journal of Applied Engineering Research, 13(15):12296–12303.

Mortimer P.P. (1978): Mumps prophylaxis in the light of a new test for antibody, British Medical Journal 2: 1523–1524.

National Health Service (2021): Mumps https://www.nhs.uk/conditions/mumps/, retrieved January 12, 2023.

RCGP (1974): A retrospective survey of the complications of mumps. Journal of the Royal College of General Practitioners 24: 552–556.

Song X.H., Xiao-Nan H.E., Nie Y.T. (2019): Epidemiological Analysis on Public Health Emergencies of Infectious Diseases at Schools in Luoyang between 2008 and 2017. Modern Preventive Medicine.

Wagenvoort J.H.T., Harmsen M., Boutahar-Troun B.J.K., Kraaijeveld C.A., Winkler K.C. (1980): Epidemiology of mumps in the Netherlands. Journal of Hygiene 85: 313–326.

Walker O., Carter H., Jones I.G. (1980): Measles, mumps and rubella: the need for a change in immunization policy, British Medical Journal 22: 1501–1502.

Yusuf T.T. (2021): On Global Stability of Disease-free Equilibrium in Epidemiological Models. https://www.researchgate.net/publication/353891583.