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# MATHEMATICAL MODEL OF THE TRANSMISSION DYNAMICS OF MUMPS VIRUS DISEASES

<sup>1</sup>Baba Y. B.<sup>2</sup>Adamu, M.M.<sup>3</sup>Kwami, A.M.<sup>4</sup>Waziri I.M.

<sup>1,2,3,4</sup>Department of Mathematical Science, Faculty of Science & Technology Abubakar Tafawa Balewa University Bauchi

#### Abstract

In this paper we develop a deterministic compartmental mathematical model for the spread of the Mumps virus disease in the community. We develop our model by modifying the model designed by Yong *et al.* (2017) by incorporating the isolation compartment as control strategies to control the transmission rate. An appropriate system of ordinary differential equations (ODE) was formulated for the transmission and the method of linearized stability approach was used to solve the equations. The Existence and uniqueness, Disease free equilibrium (DFE), positivity of the solution, Reproduction number and stability analysis were carried out. The equilibria state showed that the disease can easily by trigged off or reduced, so the need to be constantly alert and effective prevention measures put in place against its spread, in addition, numerical analyses were carried out with the model parameter assigned specific hypothetical values and graphs were plotted to investigate the effect of these parameters on the transmission of the disease. The result showed that, with the nature of the virus, uncontrolled transmittable contact between infected individuals and the susceptible can lead to a very serious outbreak with effective isolation structure put in place such situation can be better managed and outbreak controlled.

#### Keywords: Mathematical model, Mumps Virus, Transmission Dynamics, Reproductive number

#### 1. Introduction

Mumps is a contagious viral illness caused by the paramyxovirus. It is a human systemic disease that occurs worldwide but is highly preventable via the mumps vaccine. The mumps vaccine was introduced and licensed in United State in 1967 [4], and the disease became nationally reportable in 1968. The incidence has decreased substantially with vaccination, but periods of resurgence have occurred in recent years. Humans are the only known hosts for the virus that causes mumps and the causative agent of mumps infection, is an enveloped RNA virus [16]. Since the disease is generally benign and self- resolving, its mortality is rare, but aseptic meningitis can affect 10% of case-patients [7]. Although mumps incidence is highest in the winter and spring months, in warm climates it is present throughout the year, [3]. Initial signs and symptoms often include fever, muscles pain and headache, then usually followed by painful swelling of one or both parotid salivary glands. The virus is a significant cause of paediatric deafness, and up to 37% of post-pubertal males develop orchitis, 13% of whom have impaired fertility, [3].

The transmission of the virus is by direct physical contact, droplet spread, or contaminated fomites [6,8,9]. The incubation period is about 15 to 24 days (median is 19 days) [13]. Infected patients become the most contagious in 1-2 days before onset of clinical symptoms and continue so for a few days afterwards. Generally speaking, the infectious period is about eight days [5], and the patients will recover between 10 to 14 days [1,14]. It is often associated with some pain on swallowing, fever, which is usually mild and loss of appetite. Most of the symptoms are treatable with simple pain killers, bed rest and fluids. Oral fluids should be warm, which makes it easier to swallow, but sometimes, the symptoms are severe enough to make it practical to institute medical attention such as intravenous fluid therapy. [1].

Unfortunately, the disease itself has no direct treatment in the same way as you might use a specific medication to achieve a cure of something like malaria; antibiotic prescriptions are often written out to deter any bacteria from seizing an opportunistic way of invade the person's tissues. This is called a prophylactic drug treatment. In the end, the resolution of the infection has a lot more to do with the former range of therapies rather than the antibiotic measures taken, no matter how potent the drugs are.When mumps is suspected, a genuine effort needs to be made to confirm if indeed it is the disease being suspected. Many times, mumps does not really appear to be typical in the same way that conjunctivitis causes redness of the eyes. Swelling of the salivary glands occurs in just over a third of all cases. However, even this feature is seen in a variety of bacterial infectious mononucleosis and similar viruses. There are, thus, a fair number of copycat diseases and they can be frankly deceptive. It is important to note that other conditions may also look like mumps, as seen above. In nearly half of all patients, they would have symptoms as though they had a simple cold. Many cases of this disease therefore escape notice, but it is one condition which spreads like wildfire in hostels and dormitories. This is where educators, healthcare professionals and parents should exercise maximum vigilance. [3].

The spread of mumps through the saliva includes shared plates, cups and glasses. It is also spread through coughing, sneezing and kissing. It is thus able to spread rapidly among children and also from children to adults, thus demonstrating that no age group is immune except those who have had prior immunisation. It is therefore important to ensure that anyone in contact with a mumps patient should be up to date with their vaccines. Commonly, two doses are recommended for children at 12 to 15 months of age and again at between four and six years of life. [7]. The MMR vaccine which protects against mumps, measles and rubella is given in a wide spectrum of temperate countries but it is not incorporated in the National Programme on Immunisation in Nigeria where only the measles vaccine is given. However, a number of private hospitals avail their patients of this service. These two doses do not provide 100 per cent protection against mumps, but its routine use in the United States for one has reduced the incidence of mumps to less than one percentage point. That is the major reason why the debate over whether to add a third dose has not been widely received. [7].

All these facts put together motivated us to study the dynamics of these diseases and contribute to the body of existing knowledge on it. Infectious disease are diseases caused by pathogenic microorganism such as bacteria, viruses, parasites, or fungi, the diseases can be spread, directly or indirectly from one person to another. And some of them are deadly diseases.Mumps is also known as common childhood viral disease and is highly contagious to human beings. It is a human system disease that occurs worldwide but is highly preventable via the mumps vaccine.

The principal aim of modelling infectious diseases is to be able to make judicious decisions in the application of control interventions of the infection to eliminate and ideally to eradicate it from the population. Simulations and modelling can optimize control efforts such that limited resources are targeted to achieve the highest impact[2].

Yong *et al.* (2017). Designed and developed a dynamic transmission model of Mumps Virus in which they used SVEILR model (susceptible-vaccinated-exposed-severely infectious-mild infectious-recovered). They consider the influence of vaccination but didn't incorporate the isolation class.

In this paper we aim to modify the model due to (Yong *et al.* 2017) by incorporating the isolation compartment considering the nature of the virus, isolation is of great important in controlling

the spread of the virus and the objectives is to Establish the disease free equilibrium state of the model, obtain reproduction number of the model and Analyse the global stability of the disease free equilibrium state of the model.

The rest of our paper is organized as follows, second section will be best on formulation of the model, third section will be on model Analysis, the fourth on simulation and discussion and lastly we will wind up our work by conclusion and recommendations.

# 2.0. Model Formulation

In this paper we designed a deterministic compartmental mathematical model for Mumps Virus disease that captures isolation compartment as control strategies base on the following assumptions: (1). The total population is constant in a short period of time.

(2). They assume that the birth rate equals to the natural death rate, denoted by  $\mu$ .

(3). The population associated with Mumps is divided into seven epidemiological Sub-classes.

(4). The proportion Susceptible to total population (s)

(5). The proportion Mildly infectious to total population (L, Mild infections, including both asymptomatic and those with Mild symptoms and self-care).

(6). The proportion recovered to the total population (R), subject to the restriction.

(7). The proportion severely infectious to the total population (I, severely infectious requiring medical attention)

(8). The proportion of vaccinated to the total population (V)

(9). The proportion Exposed to the total population (E, infected but not infectious)

(10). We assume the possibility of getting recovered from severe condition without been isolation.

(11). The proportion isolation to the total population

Based on the above mentioned assumptions and motivated by the work of [12,10] we designed a new deterministic model as follows

Our model is designed base on the characteristic of mumps transmission; therefore, the model composed of seven sub – classes namely S(t), E(t), I(t), L(t), J(t), R(t) and V(t).S(t) is the number of susceptible individuals at a time t, E(t) is the number of latent individuals at a time t, who are infected but not infectious yet, or individuals with symptoms but misdiagnosed by a doctor, I(t) is the number of severe – infected individuals at a time whose require medical attention, L(t) is the number of Mild – infected individuals at a time including both asymptomatic and those with mild symptoms and self - care, J(t) is the number of isolated individuals at a time, V(t) is the number of vaccine individuals at a time, and R(t) is the number of recovered individuals at a time subject to the restriction S + V + E + I + L + J + R = 1. The transmission dynamics associated with these sub-classes are illustrated in Figure 1.



#### Figure 1: Flowchart of mumps transmission in a population

When susceptible people S(t) are infected, they progress to the exposed, but not infectious state (E) from there, they become infectious to the susceptible population. A mild – infectious peoples progress to recover class after self- medication. While severely – infectious progress to isolation class for medical attention as well as reducing the spread to the people than progress to recover class.

Variables/	Description	Assumed
Parameters		value for
		simulation
S(t)	The number of Susceptible individuals at a time t	800
E(t)	The number of Exposed individuals at a time t,	200
I(t)	The number of severe - infected individuals at time t,	50
L(t)	The number of Mild – infectious individuals at a time t,	100
J(t)	The number of Isolated individuals at a time t,	40
R(t)	The number of Recovered individuals at a time t	200
V(t)	The number of vaccinated individuals at a time t	500
β	The transmission rate	0.2
μ	Present the birth and natural mortality rate	500
γ	Proportion of severe infectious seeking medical advice	0.6
λ	Loss of vaccination rate	0.4
α	Rate moving from Exposed to Severe or mild Infectious population	0.4
κ	Invalid vaccination rate	0.6
ε	Vaccine coverage of the Susceptible	0.5
δ1	Rate moving from severe Infectious to Isolation population	0.8
δ <sub>2</sub>	Rate moving from Mild Infectious to Recovered population	1.0
δ <sub>3</sub>	Rate moving from isolation to Recovered population	1.0
ρ	Fraction of the offspring from Exposed parent birth into Exposed population	0.4
ε <sub>1</sub>	Vaccine coverage of the Exposed population	0.3

Table 1: Detailed description of variables/parameters.

#### 2.1 Model Equations

From the description of the dynamics of Mumps virus and with the aid of the compartmental diagram in Figure 1, the following set of non-linear ordinary differential equations can be derived:

$$\frac{dS}{dt} = \mu - \rho \mu E - \beta S(I+L) + \lambda V - (\varepsilon + \mu)S \qquad \dots (1)$$

$$\frac{dv}{dt} = \varepsilon S + \varepsilon_1 E - \lambda V - \kappa \beta V (I+L) - \mu V \qquad \dots (2)$$

$$\frac{dE}{dt} = \beta S(I+L) + \rho \mu E + \kappa \beta V(I+L) - (\alpha + \varepsilon_1 + \mu)E \qquad \dots (3)$$

$$\frac{dI}{dt} = \alpha \gamma E - (\delta_1 + \mu)I \qquad \dots (4)$$

$$\frac{dL}{dt} = \alpha (1 - \gamma)E - (\delta_2 + \mu)L \qquad \dots (5)$$

$$\frac{dJ}{dt} = \delta_1 I - (\mu + \delta_3) J \qquad \dots (6)$$

$$\frac{dR}{dt} = \delta_2 L + \delta_3 J - \mu R \qquad \dots (7)$$

The validity and authenticity of any mathematical model depends on whether the given system of equation has a solution or not. And if model given monitor human population, it is significant to show that all the state variables in the model is non negative for all time. To achieve that we used the existences and uniqueness of the solution and positivity of solution of the model.

#### 2.2. The Existence and Uniqueness of the Solution

We use the theorem of existence and uniqueness of ode by Derrick and Grossman (1776), the existence and uniqueness of the model equation given byEqs(1 - 7).

# **Theorem 1**

Consider the system of equation below

$$x_{1}^{1} = f_{1}(t, x_{1}, x_{2}, \dots, x_{n}), x_{1,}(t_{0}) = x_{10}$$

$$x_{2}^{1} = f_{1}(t, x_{1}, x_{2}, \dots, x_{n}), x_{2,}(t_{0}) = x_{20}$$

$$\vdots$$

$$x_{n}^{1} = f_{n}(t, x_{1}, x_{2}, \dots, x_{n}), x_{n,}(t_{0}) = x_{n0}$$

$$\dots (8)$$

We may write Eq (8) in compact form as

$$x^{1} = f_{1}(t, x), x_{1}(t_{0}) = x_{0} \qquad \dots (9)$$
**Theorem 2**

$$Let D \text{ denote the region}$$

$$|t - t_{0}| \le a, ||x - x_{0}|| \le b, x = (x_{1}, x_{2}, \dots, x_{n}), x_{0} = (x_{10}, x_{20}, \dots, x_{n0}) \qquad \dots (10)$$
And suppose that  $F(t, x)$  satisfies the Lipchitz condition
$$||f(t, x_{1}) - f(t, x_{2})|| \le k ||x_{1} - x_{2}|| \qquad \dots (*)$$

Whenever the pairs  $(t, x_1)$  and  $(t, x_2)$  belong to  $D^l$ . Where K is a positive constant. Then there is a constant  $\delta > 0$  such that there exist a unique continues vector solution of X(t) of Eq (9) in the interval  $|t - t_0| \le \delta$ . It is important to note that the condition (\*) is satisfied by the requirement that  $\frac{\delta f_i}{\delta x_j}$ , i, j = 1, 2, ... be continues and bounded in  $D^1$ 

#### **Theorem 3**

Let D denote the region defined in  $1 \le \varepsilon \le R$ , and  $0 < R < \infty$ , hold then these exist a unique solution of Eqs (1 - 7) of the model equation which is bounded in the region D.

#### Proof: Let

$$f_1 = \mu - \rho \mu E - \beta S(I+L) + \lambda V - (\varepsilon + \mu)S \qquad \dots (12)$$

$$f_2 = \varepsilon S + \varepsilon_1 E - \lambda V - \kappa \beta V (I + L) - \mu V \qquad \dots (13)$$

$$f_3 = \beta S(I+L) + \rho \mu E + \kappa \beta V(I+L) - (\alpha + \varepsilon_1 + \mu)E \qquad \dots (14)$$

$$f_4 = \alpha \gamma E - (\delta_1 + \mu)I \qquad \dots (15)$$

$$f_5 = \alpha (1 - \gamma) E - (\delta_2 + \mu) L \qquad ... (16)$$

$$f_6 = \delta_1 I - (\mu + \delta_3) H \qquad \dots (17)$$
  

$$f_7 = \delta_2 L + \delta_3 H - \mu R \qquad \dots (18)$$

It is sufficient to show that  $\frac{\partial f_i}{\partial x_i}$ , i, j = 1, 2, ..., 7 are continuous.

Let  $S = x_1, V = x_2, E = x_3, I = x_4, L = x_5, J = x_6$ , and  $R = x_7$ ,

Taken the partial derivative of Eqs (12-18) separately, we have

$$\begin{split} f_1 \Rightarrow \left| \frac{\partial f_1}{\partial x_6} \right| &= \left| \frac{\partial f_1}{\partial x_7} \right| = 0 < \infty, \qquad f_2 \Rightarrow \left| \frac{\partial f_1}{\partial x_6} \right| &= \left| \frac{\partial f_1}{\partial x_7} \right| = 0 < \infty, \\ f_3 \Rightarrow \left| \frac{\partial f_3}{\partial x_6} \right| &= \left| \frac{\partial f_3}{\partial x_7} \right| = 0 < \infty, \qquad f_4 \Rightarrow \left| \frac{\partial f_4}{\partial x_1} \right| &= \left| \frac{\partial f_4}{\partial x_2} \right| = \left| \frac{\partial f_4}{\partial x_6} \right| = \left| \frac{\partial f_4}{\partial x_7} \right| = 0 < \infty, \\ f_5 \Rightarrow \left| \frac{\partial f_5}{\partial x_1} \right| &= \left| \frac{\partial f_5}{\partial x_2} \right| = \left| \frac{\partial f_5}{\partial x_4} \right| = \left| \frac{\partial f_5}{\partial x_6} \right| = \left| \frac{\partial f_5}{\partial x_7} \right| = 0 < \infty, \\ f_6 \Rightarrow \left| \frac{\partial f_6}{\partial x_1} \right| &= \left| \frac{\partial f_6}{\partial x_2} \right| = \left| \frac{\partial f_6}{\partial x_3} \right| = \left| \frac{\partial f_6}{\partial x_5} \right| = \left| \frac{\partial f_6}{\partial x_5} \right| = \left| \frac{\partial f_7}{\partial x_4} \right| = 0 < \infty, \\ f_7 \Rightarrow \left| \frac{\partial f_7}{\partial x_1} \right| = \left| \frac{\partial f_7}{\partial x_2} \right| = \left| \frac{\partial f_7}{\partial x_3} \right| = \left| \frac{\partial f_7}{\partial x_4} \right| = 0 < \infty \end{split}$$

Clearly all these partial derivatives are continuous and bounded. Hence by Theorem 2, these exist a unique solution of Eq (12 - 18) in the region D.

#### 2.3 **Positivity of Solution of the Model**

Since the model equations given by Eqs(1 - 7) monitors human population, it is significant to show that all the state variables in the model is non negative for all time.

#### **Theorem 4**

For non-negative initial conditions of the model equations given by Eqs (12 – 18), the solution (S,V,E,I,L,J,R) of the model Eqs (12 – 18) are all non-negative for all time  $t \ge 0$ .

#### **Proof:**

Eq (12-18) can be rewritten as

$\frac{dS}{dt} \geq -(\varpi + k_1)S$	(19)
$\frac{dV}{dt} \ge -(k_2 + \varphi)V$	(20)
$\frac{dE}{dt} \ge (\zeta - k_3)E$	(21)
$\frac{dI}{dt} \ge -k_4$	(22)
$\frac{dL}{dt} \ge -k_5$	(23)
$\frac{dJ}{dt} \ge -k_6$	(24)
$\frac{dR}{dt} \geq -k_7$	(25)

Integrating both side of Eq (19-25) separately and set t = 0. We have

 $S(0) \ge 0$ ,  $V(0) \ge 0$ ,  $E(0) \ge 0$ ,  $I(0) \ge 0$ ,  $L(0) \ge 0$ ,  $J(0) \ge 0$ , and  $R(0) \ge 0$ .

Which proof that all the variables in the model are non-negative for all time.

# 3. Model Analysis

The model Eqs (1-7) is analyzed qualitatively to get insights into its dynamical features which give better understanding of the impact control strategies on the transmission dynamics of Mumps virus

# 3.1 Disease free equilibrium state

Consider the system of Eqs (1 - 7). In order to obtain the equilibrium, point we set the right hand sides of the Eqs (1 - 7) to zero (0) that is

i.e. 
$$\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dJ}{dt} = \frac{dR}{dt} = \frac{dL}{dt} = 0$$

At disease free, all the compartments are zero except that of Susceptible and Vaccination.

Eqs (1-7) reduced to

$$\mu + \lambda V^* - (\varepsilon + \mu)S^* = 0 \qquad \dots (26)$$

$$\varepsilon S^* - \lambda V^* - \mu V^* = 0 \qquad \dots (27)$$

From Eq (27)

$$V^* = \frac{\varepsilon S^*}{(\lambda + \mu)} \qquad \dots (28)$$

Putting Eq(28) in Eq(26)

$$\implies S^* = \frac{\mu(\mu+\lambda)}{((\lambda+\mu)(\varepsilon+\mu)-\lambda\varepsilon)} \qquad \dots (29)$$

Putting Eq(29) in Eq(28)

$$\Longrightarrow V^* = \frac{\varepsilon \mu}{(\lambda + \mu)(\varepsilon + \mu) - \lambda \varepsilon}$$

Therefore the disease free equilibrium state is  $E_0 = (S, V, E, I, L, J, R)$ 

$$\Longrightarrow E_0 = \left[\frac{\mu(\mu+\lambda)}{((\lambda+\mu)(\varepsilon+\mu)-\lambda\varepsilon)}, \frac{\varepsilon\mu}{((\lambda+\mu)(\varepsilon+\mu)-\lambda\varepsilon)}, 0, 0, 0, 0, 0\right]$$

# **3.2.** Basic reproduction number $R_0$

The basic reproduction number denoted by  $R_0$  is the average number of secondary infections caused by an infectious individual during his or her entire period of infectiousness [16]. The basic reproduction number is an important non-dimensional quantity in epidemiology as it sets the threshold in the study of a disease both for predicting its outbreak and for evaluating its control strategies. Thus, whether a disease becomes persistent or dies out in a community depends on the value of the reproduction number, R0. Furthermore, stability of equilibria can be analyzed using $R_0$ ; if  $R_0 < 1$  it means that every infectious individual will cause less than one secondary infection and hence the disease will die out and when  $R_0>1$ , every infectious individual will cause more than one secondary infection and hence the disease will invade the population. A large number of R0 may indicate the possibility of a major epidemic. For the case of a model with a single infected class,  $R_0$  is simply the product of the infection rate and the mean duration of the infection.

Due to complicated epidemics in our model, we compute the reproduction number,  $R_0$  using the next generation operator approach by [11].

Firstly, we arrange the system to get group of infections classes only that is (E, I, L, J). Let  $f_i(x)$  be the rate of appearance of new infections (transmission) in compartment  $i, V_i^+(x)$  be the rate of transmission after new infections (transmission rate by all other means) and  $V_i^-(x)$  be the rate of transfer of individuals out of compartment *i*.

... (30)

Consider the system of equation for the infected population given by below

$$\frac{\partial E}{\partial t} = \beta S(I+L) + \rho \mu E + \kappa \beta V(I+L) - (\alpha + \varepsilon_1 + \mu)E 
\frac{dI}{dt} = \alpha \gamma E - (\delta_1 + \mu)I 
\frac{\partial L}{\partial t} = \alpha (1-\gamma)E - (\delta_2 + \mu)L 
\frac{\partial J}{\partial t} = \delta_1 I - (\mu + \delta_3)J$$
... (31)

From Eq (31) we have

$$F_{i} \begin{bmatrix} F_{1} \\ F_{2} \\ F_{3} \\ F_{4} \end{bmatrix} = \begin{bmatrix} \beta S(I+L) + \rho \mu E + \kappa \beta V(I+L) \\ 0 \\ 0 \\ 0 \end{bmatrix} \dots (32)$$

From Eq (31) we consider those terms that does not have new infection and multiply by (-1) That is, we have

$$V_{i} = \begin{bmatrix} V_{1} \\ V_{2} \\ V_{3} \\ V_{4} \end{bmatrix} = \begin{bmatrix} (\alpha + \varepsilon_{1} + \mu)E \\ (\delta_{1} + \mu)I - \alpha\gamma E \\ (\delta_{2} + \mu)L - \alpha(1 - \gamma)E \\ (\mu + \delta_{3})J - \delta_{1}J \end{bmatrix} \dots (33)$$

Now, taken  $\frac{\delta f_i}{\delta x_i}$  of Eq (32) and evaluate at disease free state. We have

$$F = \begin{bmatrix} A_1 & A_2 & A_3 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \dots (34)$$

Similarly, taken  $\frac{\delta V_i}{\delta x_i}$  of Eq (33). We have

$$\Rightarrow V = \begin{bmatrix} B_1 & 0 & 0 & 0 \\ B_2 & B_3 & 0 & 0 \\ B_4 & 0 & B_5 & 0 \\ 0 & B_6 & 0 & B_7 \end{bmatrix} \dots (35)$$

Taken inverse of Eq (35) and multiply by Eq (35) gives.

Finding  $|FV^{-1} - \lambda| = 0$ . Of Eq (39). We have

$$\lambda_1 = D_1$$
,  $\lambda_2 = 0$ ,  $\lambda_3 = +0$  and  $\lambda_4 = -0$ 

 $\Rightarrow$  Our  $R_0 = D_1$  Because  $D_1$  is the largest eigenvalue.

$$\Rightarrow R_0 = R_1 + R_2 R_3. \text{ Where } R_1 = \frac{\rho \mu}{(\alpha + \varepsilon_1 + \mu)} \qquad R_2 = \frac{\beta \mu (\lambda + \mu)}{((\lambda + \mu)(\varepsilon + \mu) - \lambda \varepsilon)} R_3 = \frac{\alpha \gamma}{(\alpha + \varepsilon_1 + \mu)(\delta_1 + \mu)}$$

#### 3.3. Global stability of disease free equilibrium (DFE)

To establish the global stability of the disease free equilibrium of the model using the theorem by (Castillo – Chavez *et al., 2002).* the conditions  $H_1$  and  $H_2$  must be satisfied.

 $H_1: \frac{dx}{dy} = H(x, 0), x^0$  is globally asymptotically stable (GAS)  $H_2: \hat{G}(x,z) = pz - \hat{G}(x,z), \hat{G}(x,z) \ge 0$  for  $(x,z) \in \Omega$ , where  $P = \Delta_z G(x^0,0)$  is an M- matrix (the off diagonal elements of p are non-negative) and is also jacobain of G(x, z)

We write the model equation given by (1 - 7) as  $\frac{dx}{dt} = H(X, Z),$ 

$$\frac{dz}{dt} = G(X, Z), G(X, 0) = 0$$
$$E_0(X^0, 0) = \left[\frac{\mu(\mu + \lambda)}{((\lambda + \mu)(\varepsilon + \mu) - \lambda\varepsilon)}, \frac{\varepsilon\mu}{((\lambda + \mu)(\varepsilon + \mu) - \lambda\varepsilon)}, \right]$$

Where  $X = (S, V, R) \in \mathbb{R}^3$  denotes the number of un-infected individuals and Where  $X = (S, V, K) \in \mathbb{R}^{4}$  denotes the number of un-infected individuals.  $E_{0} = (K^{0}, 0)$  denotes the DFE of the system. Take (E, I, L, J) and evaluated at  $E_{0}(S, V) = \left[\frac{\mu(\mu + \lambda)}{((\lambda + \mu)(\varepsilon + \mu) - \lambda\varepsilon)}, \frac{\varepsilon\mu}{((\lambda + \mu)(\varepsilon + \mu) - \lambda\varepsilon)}, \right].$ 

If the system satisfies the condition  $H_1$  and  $H_2$  above, then according to Castillo – Chavez *et.al* (2002), the following theorems holds.

#### Theorem 4.5

The fixed point  $E_0(x^0, 0)$  is a globally asymptotic stable (GAS) provided that  $R_0 < 1$  (*Locally asymptotically stable* (*L.A.S*)) and that assumptions  $H_1$  and  $H_2$  are statisfied.

**Proof:** from, the two functions H(x, z) and G(x, z) are given by

$$H(x,z) = \begin{bmatrix} \mu - \rho\mu E - \beta S(I+L) + \lambda V - (\varepsilon + \mu)S \\ \varepsilon S + \varepsilon_1 E - \lambda V - \kappa \beta V(I+L) - \mu V \\ \delta_2 L + \delta_3 J - \mu R \end{bmatrix} \dots (40)$$

$$G(x,z) = \begin{bmatrix} \beta S(I+L) + \rho \mu E + \kappa \beta V(I+L) - (\alpha + \varepsilon_1 + \mu)E \\ \alpha \gamma E - (\delta_1 + \mu)I \\ \alpha (1-\gamma)E - (\delta_2 + \mu)L \\ \delta_1 I - (\mu + \delta_3)J \end{bmatrix} \dots (41)$$

Consider the reduced system  $\frac{dx}{dt} = H(x, 0)$  from condition (1)

$$H(x,0) = \begin{bmatrix} \mu + \lambda V - (\varepsilon + \mu)S\\ \varepsilon S - \lambda V - \mu V\\ 0 \end{bmatrix} \dots (42)$$

Integrate the first equation of Eq (42), we have

$$S(t) = \frac{(\mu + \lambda V)}{(\varepsilon + \mu)} + C e^{-(\varepsilon + \mu)t} \qquad \dots (43)$$

At  $t \rightarrow 0$ , Eq (43) becomes

$$C = S(0) - \frac{(\mu + \lambda V)}{(\varepsilon + \mu)} \qquad \dots (44)$$

Putting Eq (44) in Eq(43) and evaluate at  $t \to \infty$ , we have

$$S(t) = \frac{(\mu + \lambda V)}{(\varepsilon + \mu)} \qquad \dots (45)$$

Putting the value of  $V^*$  in Eq (30) in Eq (45). We have

$$S(t) = \frac{\mu[((\lambda+\mu)(\varepsilon+\mu)-\lambda\varepsilon)] + \lambda\varepsilon\mu}{(\mu+\lambda)(\varepsilon+\mu)^2 - (\varepsilon+\mu)\lambda\varepsilon} \qquad \dots (46)$$

Apply the same process for the second equation of Eq (42), we have

$$V(t) = \frac{\varepsilon \mu (\lambda + \mu)}{(\lambda + \mu)^2 (\varepsilon + \mu) - (\lambda + \mu) \lambda \varepsilon}$$

Convergence of  $X^0$  is therefore globally in  $\Omega$ 

$$X^{0} = \left[\frac{\mu[((\lambda + \mu)(\varepsilon + \mu) - \lambda\varepsilon)] + \lambda\varepsilon\mu}{(\mu + \lambda)(\varepsilon + \mu)^{2} - (\varepsilon + \mu)\lambda\varepsilon}, \frac{\varepsilon\mu(\lambda + \mu)}{(\lambda + \mu)^{2}(\varepsilon + \mu) - (\lambda + \mu)\lambda\varepsilon}\right]$$

is globally asymptotically stable equilibrium of  $\frac{dx}{dt} = H(x, 0)$ .

Next we compute H(x, z) = pz - G(x, z) and show that  $G(x, z) \ge 0$ 

$$\therefore J \ [G(X,Z)] = p \qquad \dots (47)$$

Where j is the jacobain of G(x, z) taken in (E, I, L, J) and evaluate at

$$E_{0} = (S, V, E, I, L, J, R)$$

$$E_{0} = \begin{bmatrix} \mu(\mu + \lambda) & \epsilon \mu \\ \overline{((\lambda + \mu)(\epsilon + \mu) - \lambda \epsilon)}, \frac{\epsilon \mu}{((\lambda + \mu)(\epsilon + \mu) - \lambda \epsilon)}, 0, 0, 0, 0, 0 \end{bmatrix}$$
Therefore, Eq (47) gives
$$E & I & L & J \\ P = \begin{bmatrix} \rho \mu - (\alpha + \epsilon_{1} + \mu) & \beta S + \kappa \beta V & \beta S + \kappa \beta V & 0 \\ \alpha \gamma & -(\delta_{1} + \mu) & 0 & 0 \\ \alpha (1 - \gamma) & 0 & -(\delta_{2} + \mu) & 0 \\ 0 & \delta_{1} & 0 & -(\mu + \delta_{3}) \end{bmatrix}$$

$$PZ = \begin{bmatrix} \rho \mu E - (\alpha + \epsilon_{1} + \mu) & \beta S + \kappa \beta V & \beta S + \kappa \beta V & 0 \\ \alpha \gamma & -(\delta_{1} + \mu) & 0 & 0 \\ \alpha (1 - \gamma) & 0 & -(\delta_{2} + \mu) & 0 \\ 0 & \delta_{1} & 0 & -(\mu + \delta_{3}) \end{bmatrix} \begin{bmatrix} E \\ I \\ L \\ J \end{bmatrix}$$

$$PZ = \begin{bmatrix} \rho \mu E - (\alpha + \epsilon_{1} + \mu)E + \beta SI + \kappa \beta VI + \beta SL + \kappa \beta VL + 0 \\ \alpha \gamma E - (\delta_{1} + \mu)I + 0 + 0 \\ \alpha (1 - \gamma)E + 0 - (\delta_{2} + \mu)L + 0 \\ 0 + \delta_{1}I - (\mu + \delta_{3})H \end{bmatrix}$$

Evaluating PZ - G(X, Z), we have

 $PZ - G(X, Z) = [0 \ 0 \ 0 \ 0]^T.$  $\therefore \hat{G}(X, Z) = PZ - G(X, Z) = [0 \ 0 \ 0 \ 0]^T.$ i.e.  $G(X, Z) = [0 \ 0 \ 0 \ 0]^T.$ 

This shows that G(X, Z) = 0. Hence, the model is globally asymptotically stable.

#### 4. Simulation and Discussion

The main objective of this paper was to model the transmission dynamic of Mumps Virus disease with isolation as control strategies. In order to support the analytical results, numerical result was presented with the aid of MATLAB programming language, we present graphical representation showing the variation in parameters with respect to effective reproduction number.

#### 4.1.1 Effect of vaccine on infected population



Figure 2: The simulation result of infected population without vaccination rate ( $\varepsilon = 0.0$ )



**Figure 3:** The simulation result of infected population with vaccination rate of ( $\varepsilon = 0.2$ )



Figure 4: The simulation result of infected population with vaccination rate of ( $\varepsilon = 0.6$ )



Figure 5: The simulation result of infected population with vaccination rate of ( $\varepsilon = 0.9$ )

4.1.2 Effect of Isolation on infected population



**Figure 6:** The simulation result of infected population without isolation rate of ( $\delta_1 = 0.0$ )



**Figure 7:** The simulation result of infected population with isolation rate of ( $\delta_1 = 0.2$ )



**Figure 8:** The simulation result of infected population with isolation rate of ( $\delta_1 = 0.6$ )



**Figure 9:** The simulation result of infected population with isolation rate of ( $\delta_1 = 0.9$ )

4.2 Discussion of analytical Results

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We established the existence and uniqueness of the solution. Positivity of solution and disease free equilibrium for Mumps disease dynamics model. We also obtained the model's basic reproduction using the next generation matrix technique. Lastly, we obtained the global stability of the disease free equilibrium using the method of Castillo-Chavez *et*, *al.*, and is found to be globally asymptotically stable. This means the disease can be eradicated in a stable equilibrium.

# 4.3. Discussion of Simulation Results

Figures (2 - 5) show the effects of vaccination rate on infected population. The population of the infected individual is reducing when the vaccination rate is increase, in figure (2) the vaccination rate is 0 and the population of the infected individual is 650, in figure (3) the vaccination rate is 0.2 and the population of the infected individual is 250, in figure (4) the vaccination rate is 0.6 and the population of the infected individual is 86 and in figure (5) the vaccination rate is 0.9 and the population of the infected individual is 60. Figures (6– 9) show the effects of isolation on infected population. The infected population grows rapidly with no treatment. But as a result of increment of isolation/treatment you can see that the infected class is reducing. in figure (6) the isolation rate is 0 and the population of the infected individual is 1200, in figure (7) the isolation rate is 0.2 and the population of the infected individual is 1100, in figure (8) the isolation rate is 0.6 and the population of the infected individual is 550.

The simulation result indicated that the both vaccine and isolation are inversely proportional to the infected population.

# 5.1 Conclusion and Recommendation

This research work is a modification of the spread and control of Mumps virus disease models. The model subdivides the population into seven (7) compartments namely; Susceptible classS(t), Vaccination classV(t), Exposed classE(t), severely-infectiousclass I(t), mild-infectious class L(t), Isolation J(t), and Recovered R(t). The analytical studies were carried out which revealed that the disease free equilibrium of the model, model is locally asymptotically stable if  $R_0 < 0$ . We obtained Endemic equilibrium and Global stability of disease free equilibrium. The numerical simulation carried out shows that treatment of Mumps virus diseases increases the population.

# 5.2 Recommendations

In view of the findings of this study, we recommend that Authorities concern should shade more light on the control strategies that will help to reduce the effect of Mumps virus diseases especially on the importance of vaccine and isolation.

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