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A Model of Measles Dynamics in the Presence of Weak and Strong Vitamin A

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Abstract

In this paper, a non linear (M-S-V-E-I₁-I₂-R) compartmental model for measles has been proposed and analyzed by incorporating maternal immunity, taking into consideration the effect of vaccination and the effect of Vitamin A in the body. The system of the equations describing the phenomena is express as a system of ordinary differential equations. The two infectiousness phases was captured in the model as a result of strong and weak vitamin A in the body. From the model equations we obtained the effective reproduction number R_c using the next generation approach and hence confirmed the criteria for local and global stability of disease free equilibrium, we showed that the disease free equilibrium is locally asymptotically stable (LAS) when $R_c < 1$ and globally asymptotically stable for $R_c \leq 1$.

Keywords: Measles, Effective Reproduction Number, Disease-Free Equilibrium, Maternal Immunity, Stability.

.0 Introduction

asles is one of the most contagious but vaccine-preventable disease which is caused by a morbilli virus of the amyxovirus family. Paramyxovirus normally grows in the cell that lines the back of the throat and lungs. It resides in the ucus in the nose and throat of an infected person, so transmission typically occurs through coughing and sneezing. Measles est known for causing rash and fever in childhood, but can lead to severe health complication in adults [1].

ere are two types of measles each caused by different virus. Although both produce rash and fever, they are really different ase. The first type is rubella which causes –"German measles" known as the "three day, measles" and other, rubeola ich causes "red measles" also known as "hard measles" or just "measles". When most people use the term measles, they referring to the later. The biggest difference between them (the two) is that rubella is considered to be a milder disease only last around three days, Rubeola can become a serious illness that lasts several days and can caused other serious manent complications. Measles can result in higher risk of premature labor, low birth weight infants, miscarriage, or ificant birth defects if a pregnant woman is infected or passes the virus to her unborn child. It can also lead to pneumonia nflammation of the brain (encephalitis) [2].

ildren who are deficient in vitamin A seem to be more likely to have severe measles (and are more likely to die from the ection). therefore the world health organization and UNICEF recommend giving 1 to 3 doses of vitamin A to children der than 6 month who have measles and are hospitalized because of measles or its complication or who are malnourished, ve

mune system problems, or who are proven to have a vitamin A deficiency [3].

amin A is a group of unsaturated nutritional organic compounds that includes retinol, retinal, retinoic acid, and several vitamin A carotenoids. Vitamin A is important for growth and development, maintenance of immune system and good ion. Vitamin A deficiency (VAD) or hypovitaminosis A is a lack of vitamin A in blood and tissues and the leading couse preventable childhood blindness and diminishes the ability to fight infections [3]. It is most common in poorer countries rarely seen in more developed countries [4]

rldwide, Measles vaccination has been very effective, preventing an over 85 million cases and more than 5 million deaths ually [4,5]. Although global incidence has been significantly reduced through vaccination, measles remains an important blic health problem. Since vaccination coverage is not uniformly high worldwide, measles stands as the leading vaccine-eventable killer of children worldwide. In 2002 alone, more than 1,700 deaths were recorded daily due to measles with over % of these deaths dominant in sub-Saharan Africa [6,7]. There was a record of over 450 daily deaths from measles in 2011, ich shows an increase of about 12% when compared with the case of 2010 [7].

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In as much as understanding dynamics of diseases is important, preventing, controlling and eradicating such disease are of greater importance. One disease whose mortality and morbidity burden has been dramatically reduced following large scale vaccination is measles. New borns are afforded protection to measles through maternal antibodies, which may be effective for up to one year after birth. Vaccination against is not recommended until these anti bodies have waned out. It has been demonstrated that vaccine efficacy is substantially higher in older infants with no maternal anti bodies [8]. Measles outbreak is still prominent even in countries with high vaccination coverage. Vaccine for measles has been available for the past 40 years, yet measles is still the leading cause of the death among young children with not less than 85% secondary infection rate within unvaccinated population [2].

Mathematical models have become important tools in analyzing the spread, control and eradication of infectious diseases. Also, mathematical modeling is increasingly becoming a key method in determining effective control policy for a range of epidemic.

Siabouh and Adetunde [1] developed a deterministic model of four (4) compartments with Susceptible (S), Exposed (E), Infected (I) and Recovered (R). They obtained the Basic Reproduction number R_0 and concluded that the disease can be eliminated if the level of immunity can be exceeded not only by means of mass vaccination but also early detection and education. In a similar pattern, a mathematical modeling of the effect of vaccination on the transmission dynamics of measles was developed in [8] with five (5) compartments of Passively Immune Infants (M), Susceptible (S), Exposed (E), Infected (I) and Recovered (R). They established the conditions for the stability of the disease-free equilibrium and found out that in high measles prevalence countries, effective vaccination will have a greater impact on the transmission dynamics of the disease. Bolarin [2] also developed a compartmental model of five (5) classes, including the Vaccinated class (V). He obtained the Basic Reproduction number, R_0 and found out that the R_0 under vaccination approaches zero as the proportion of successfully vaccinated individuals increases.

2.0 The Model

2.1 Model Formulation

In this work, we complement and extend the works of the aforementioned authors by having seven (7) compartments of Passively Immune (P), Susceptible (S), Vaccinated (V), Latent Class (L), Infected Class (I_1), Infected Class (I_2) and Recovered Class (R). We also studied the effect of Vaccine and Vitamin A deficiency in the body.

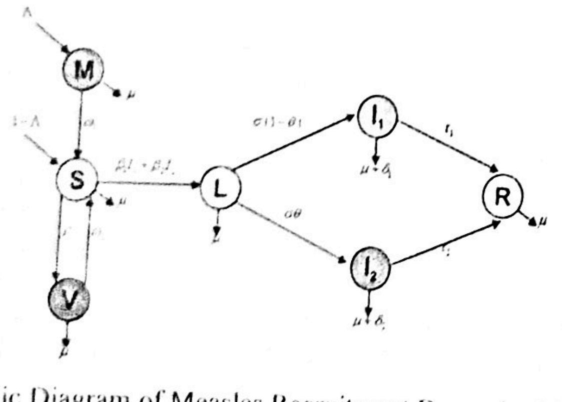

Figure 1: Schematic Diagram of Measles Recruitment Dynamics Model

Since infants are passively immune at birth, we assumed that all offspring are not susceptible. The passively immune infant's M population is generated from daily recruitment of uninfected individuals through births given by Λ and diminished by natural death μ. The class S of susceptible individuals is generated through waning out of the maternal antibodies at rate ω_1 and remaining individuals $(1-\Lambda)$ who are not passively immune at birth or migrated into the population and also increases due to waning out of efficacy of vaccines at rate ω_2. The susceptible class is decreased by infection due to effective contacts with the infected individuals I_1 and I_2 at rates β_1 and β_2 respectively. The class S diminished by vaccination at rate ρ and natural death at rate μ. Individuals in this class then move to the class L of the exposed individuals through interaction with infected class at rate β_1, β_2. L is decreased by progression to infection at rates $\sigma(1-\theta), \sigma\theta$ and diminished by natural death at rate μ. I_1 and I_2 are generated by progression from class L at rates $\sigma(1-\theta), \sigma\theta$ respectively. These class are decrease by recoveries of infected class I_1, I_2 at rates τ_1, τ_2 and natural death at rate μ as well as those that die due to the

...se at rates δ_1, δ_2 respectively. The model assumes that recovered infected individuals become permanently immune to ...disease. This generates a class R of individuals who have complete protection against the disease. the class R of ...ered individuals diminished by natural death at rate μ.

...orresponding mathematical equations of the schematic diagram can be described by a system of Ordinary Differential ...ations (ODEs) given below:

$$= \Lambda - (\omega_1 + \mu) M \quad (1)$$

$$\omega_1 M + (1-\Lambda) + \omega_2 V - (\beta_1 I_1 + \beta_2 I_2) S - (\mu + \rho) S \quad (2)$$

$$= \rho S - (\omega_2 + \mu) V \quad (3)$$

$$= (\beta_1 I_1 + \beta_2 I_2) S - (\sigma + \mu) L \quad (4)$$

$$= \sigma (1-\theta) L - (\tau_1 + \delta_1 + \mu) I_1 \quad (5)$$

$$= \sigma \theta L - (\tau_1 + \delta_1 + \mu) I_2 \quad (6)$$

$$= \tau_1 I_1 + \tau_2 I_2 - \mu R \quad (7)$$

...biological-feasible region:

$$\left.\begin{array}{l}(M, S, V, L, I_1, I_2, R) \in \Re^7_+ : M \geq 0, S \geq 0, V \geq 0, \\ \qquad L \geq 0, I_1 \geq 0, I_2 \geq 0 R \geq 0;\end{array}\right\} \quad (8)$$

...can be shown to be positively invariant with respect to the system (1) – (7).

...symbols used in the model are as follows:

...Passively immune infants	S Susceptible individuals
...accinated individuals	L Individuals who are infected but not yet infectious
...fected individuals with strong ...une system and rich vitamin A	I_2 Infected individuals with weak immune system and weak vitamin A
...ndividuals who have recovered from ...ction	Λ Per capita recruitment rate
...ffective contact rate between I_1 and S	β_2 Effective contact rate between I_2 and S
...ate of progression from L to I_1 and I_2	θ Proportion of individuals with weak immune system and vitamin A deficiency
...eath due to infection I_1	$1-\theta$ Proportion of individuals with strong immune system and rich vitamin A
...eath due to infection I_2	τ_1 Recovery rate of infected class I_1
...aning rate of the maternal antibodies	τ_2 Recovery rate of infection class I_2
	μ Per capita natural death rate
	ω_2 Waning rate of efficacy of vaccine

Model Analysis

...tions (9) – (16) are substituted into the system of equation (1)-(7) in order to efficiently simplify the equations.

$$\omega_1 + \mu \quad (9)$$

$$\mu + \rho \quad (10)$$

$$k_3 = \omega_2 + \mu \tag{11}$$

$$k_4 = \sigma + \mu \tag{12}$$

$$k_5 = \tau_1 + \delta_1 + \mu \tag{13}$$

$$k_6 = \tau_2 + \delta_2 + \mu \tag{14}$$

$$\eta = 1 - \Lambda \tag{15}$$

$$\vartheta = 1 - \theta \tag{16}$$

Hence, (1) to (7) becomes

$$\frac{dM}{dt} = \Lambda - k_1 M \tag{17}$$

$$\frac{dS}{dt} = \omega_1 M + \omega_2 V + \eta - (\beta_1 I_1 + \beta_2 I_2) - k_2 S \tag{18}$$

$$\frac{dV}{dt} = \rho S - k_3 V \tag{19}$$

$$\frac{dL}{dt} = (\beta_1 I_1 + \beta_2 I_2) S - k_4 L \tag{20}$$

$$\frac{dI_1}{dt} = \sigma \vartheta L - k_5 I_1 \tag{21}$$

$$\frac{dI_2}{dt} = \sigma \theta L - k_6 I_2 \tag{22}$$

$$\frac{dR}{dt} = \tau_1 I_1 + \tau_2 I - \mu R \tag{23}$$

3.1 Existence of disease-free equilibrium state, E_f

At the disease-free equilibrium state, we have absence of disease. Thus, all the infected classes will be zero and the ent population will comprise of only susceptible individuals.

Theorem 1: A disease-free equilibrium state of the model exist at the point

$$E_f = \begin{pmatrix} M^* \\ S^* \\ V^* \\ L^* \\ I_1^* \\ I_2^* \\ R^* \end{pmatrix} = \begin{pmatrix} \frac{\Lambda}{k_1} \\ \frac{k_3(\Lambda\omega_1 + \eta k_1)}{k_1(k_2 k_3 - \rho\omega_2)} \\ \frac{\rho(\Lambda\omega_1 + \eta k_1)}{k_1(k_2 k_3 - \rho\omega_2)} \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \tag{24}$$

Proof: At equilibrium state, the rate of change of each variable is equal to zero. i.e.

$$\frac{dM}{dt} = \frac{dS}{dt} = \frac{dV}{dt} = \frac{dL}{dt} = \frac{dI_1}{dt} = \frac{dI_2}{dt} = \frac{dR}{dt} = 0 \tag{25}$$

Let

$$(M, S, V, L, I_1, I_2, R) = (M^*, S^*, V^*, L^*, I_1^*, I_2^*, R^*) \tag{26}$$

From (17)

$$M^* = \frac{\Lambda}{k_1} \tag{27}$$

From (19)

$$I^* = \frac{\rho S^*}{k_3} \tag{28}$$

From (21)

$$I_1^* = \frac{\sigma \vartheta L^*}{k_5} \tag{29}$$

From (22)

$$I_2^* = \frac{\sigma \theta L^*}{k_6} \tag{30}$$

Substituting (29) and (30) into (23) gives

$$R^* = \frac{\left(k_6 \tau_1 \sigma \vartheta + k_5 \tau_2 \sigma \theta\right) L^*}{\mu k_5 k_6} \tag{31}$$

Substituting (29) and (30) into (20) gives

$$\left(\frac{k_6 \beta_1 \sigma \vartheta S^* + k_5 \beta_2 \sigma \theta S^* - k_4 k_5 k_6}{k_5 k_6}\right) L^* = 0$$

Now, either

$$L^* = 0 \tag{32}$$

Or

$$\frac{\left(\sigma \vartheta \beta_1 k_6 + \sigma \theta \beta_2 k_5\right) S^* - k_4 k_5 k_6}{k_5 k_6} = 0 \tag{33}$$

and (33) will be greater than zero if

$$\frac{\left(\sigma \vartheta \beta_1 k_6 + \sigma \theta \beta_2 k_5\right) S^*}{k_4 k_5 k_6} > 1 \tag{34}$$

$$R_c > 1 \tag{35}$$

As seen from (31b), L can never be less than zero. Either $L = 0$ as seen from (32) or $L > 0$ whenever $R_c > 1$ as seen from (35), which resulted into equilibrium state where each of the sub-population is greater than zero. Therefore, the system (1) (7) has two different equilibrium states, namely: the disease-free equilibrium in which all the infected compartments are zero and the endemic equilibrium in which all the compartments are greater than zero.

Substituting (32) into (29), (30) and (31) gives

$$I_1^* = I_2^* = R^* = 0 \tag{36}$$

Now, substituting (27), (28) and (36) into (18) and simplifying gives

$$S^* = \frac{k_3\left(\Lambda \omega_1 + \eta k_1\right)}{k_1\left(k_2 k_3 - \rho \omega_2\right)} \tag{37}$$

Substituting (37) into (28) gives

$$V^* = \frac{\rho\left(\Lambda \omega_1 + \eta k_1\right)}{k_1\left(k_2 k_3 - \rho \omega_2\right)} \tag{38}$$

Hence, a disease-free equilibrium of the model exists at:

$$E_1 = \begin{pmatrix} M^* \\ S^* \\ V^* \\ E^* \\ I_1^* \\ I_2^* \\ R^* \end{pmatrix} = \begin{pmatrix} \frac{\Lambda}{k_1} \\ \frac{k_3(\Lambda\omega_1 + \eta k_1)}{k_1(k_2k_3 - \rho\omega_2)} \\ \frac{\rho(\Lambda\omega_1 + \eta k_1)}{k_1(k_2k_3 - \rho\omega_2)} \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \tag{39}$$

4.0 Effective Reproduction Number, R_c

One of the highly essential worry about any infectious disease is its ability to invade a population. The basic reproduction number R_0 is 'one of the foremost and most valuable ideas that mathematical thinking has brought to epidemic theory' [9]. It is a measure of the number of infections produced, on average, by an infected individual in the early stages of an epidemic when virtually all contacts are susceptible. If $R_0 < 1$, then on average, an infected individual produces less than one new infected individual over the course of its infection period, in this case, the infection may die out in the long run. On the other hand, if $R_0 > 1$, each infected individual produces, on average more than one new infection, the infection will be able to spread in a population, thus becoming an epidemic. A large value of R_0 may indicate the possibility of a major epidemic. Similarly, the effective reproduction number R_C represents the average number of secondary cases generated by an infected individual if introduced into a susceptible population where control strategies are employed.

Using the next generation operator technique described in [10] and subsequently analyzed in [11], we obtained the effective reproduction number, R_c of the model (1) – (7), which is the spectral radius (ρ) of the next generation matrix, G.

i.e

$$R_c = \rho\left(FV^{-1}\right)$$

Now, (40)

$$F = \begin{pmatrix} 0 & \beta_1 S & \beta_2 S \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \text{ and } V = \begin{pmatrix} k_4 & 0 & 0 \\ -\sigma\vartheta & k_5 & 0 \\ -\sigma\theta & 0 & k_6 \end{pmatrix} \tag{41}$$

In order to compute the matrix, V^{-1}, we used the Gauss-Jordan elimination method as explained in [12,13].

i.e

$$\left(\begin{array}{ccc|ccc} k_4 & 0 & 0 & 1 & 0 & 0 \\ \sigma\vartheta & k_5 & 0 & 0 & 1 & 0 \\ \sigma\vartheta & 0 & k_6 & 0 & 0 & 1 \end{array}\right) \tag{42}$$

Simplifying gives

$$FV^{-1} = \begin{pmatrix} M_1 & \frac{S^*\beta_1}{k_5} & \frac{S^*\beta_2}{k_6} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \tag{43}$$

where

$$M_1 = \frac{\sigma\left(\vartheta k_6\beta_1 + \theta k_5\beta_2\right)S^*}{k_4k_5k_6} \tag{44}$$

We now evaluate $\left|FV^{-1}-\lambda I\right|=0$ to find the eigenvalues

$$\left|FV^{-1}-\lambda I\right|=\begin{vmatrix} M_1-\lambda & M_2 & \frac{S^*\beta_2}{k_6} \\ 0 & -\lambda & 0 \\ 0 & 0 & -\lambda \end{vmatrix}=0$$

$$R_c=(\rho)K=(\rho)FV^{-1}=M_1 \tag{45}$$

Hence

$$R_c=\frac{k_2\sigma(\vartheta\beta_1k_6+\theta\beta_2k_5)(\Lambda\omega_1+\eta k_1)}{k_1k_4k_5k_6(k_2k_3-\rho\omega_2)} \tag{46}$$

5.0 Local Stability of Disease-free Equilibrium, E_f

We used the Jacobian stability approach to prove the stability of the disease-free equilibrium state. Linearization of (17) – (23) at E_f gives the Jacobian matrix

$$J(E_f)=\begin{pmatrix} -k_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ \omega_1 & -k_2 & \omega_2 & 0 & -\beta_1S & -\beta_2S & 0 \\ 0 & \rho & -k_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -k_4 & \beta_1S & \beta_2S & 0 \\ 0 & 0 & 0 & \sigma\vartheta & -k_5 & 0 & 0 \\ 0 & 0 & 0 & \sigma\theta & 0 & -k_6 & 0 \\ 0 & 0 & 0 & 0 & \tau_1 & \tau_2 & -\mu \end{pmatrix} \tag{47}$$

Using elementary row-transformation, we have

$$J(E^0)=\begin{pmatrix} -k_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -k_2 & \omega_2 & 0 & -\beta_1S & -\beta_2S & 0 \\ 0 & 0 & -M_1 & 0 & \frac{-\rho S\beta_1}{k_2} & \frac{-\rho S\beta_2}{k_2} & 0 \\ 0 & 0 & 0 & -k_4 & \beta_1S & \beta_2S & 0 \\ 0 & 0 & 0 & 0 & -M_2 & \frac{\sigma\vartheta\beta_2S}{k_4} & 0 \\ 0 & 0 & 0 & 0 & 0 & -M_3 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\mu \end{pmatrix} \tag{48}$$

Where

$$M_1=\frac{k_2k_3-\rho\omega_2}{k_2}$$

$$M_2=\frac{k_4k_5-\sigma\vartheta\beta_1S}{k_4}$$

$$M_3=\frac{\sigma(\vartheta k_6\beta_1+\theta k_5\beta_2)S}{k_4k_5k_6} \tag{49}$$

Thus, the characteristics equation of the row-transformed Jacobian matrix, (48) is given by $\left|J(E^0)-\lambda I\right|=0$

$$\begin{vmatrix} -(k_1+\lambda) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -(k_2+\lambda) & \omega_2 & 0 & -\beta_1 S & -\beta_2 S & 0 \\ 0 & 0 & -(M_1+\lambda) & 0 & \frac{-\rho S\beta_1}{k_2} & \frac{-\rho S\beta_2}{k_2} & 0 \\ 0 & 0 & 0 & -(k_4+\lambda) & \beta_1 S & \beta_2 S & 0 \\ 0 & 0 & 0 & 0 & -(M_2+\lambda) & \frac{\sigma\vartheta\beta_2 S}{k_4} & 0 \\ 0 & 0 & 0 & 0 & 0 & -(M_3+\lambda) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -(\mu+\lambda) \end{vmatrix} = 0 \quad (50)$$

And therefore, the eigenvalues are

$$\lambda_1 = -(\omega_1 + \mu) < 0 \quad (51)$$

Since from (18), $k_2 > \frac{\omega_1 V^*}{S^*}$ then

$$\lambda_2 = -\left(k_2 - \frac{\rho\omega_2}{k_3}\right) < 0 \quad (52)$$

$$\lambda_3 = -M_1$$

$$\lambda_3 = -\frac{k_2 k_3 - \rho\omega_2}{k_2} < 0 \quad (53)$$

$$\lambda_4 = -(\sigma + \mu) < 0 \quad (54)$$

$$\lambda_5 = -\frac{k_4 k_5 - \sigma\vartheta\beta_1 S^*}{k_4} < 0 \quad (55)$$

Since from (20) and (21) $k_4 k_5 > \sigma\vartheta\beta_1 S^*$

$$\lambda_6 = -M_3$$

$$\lambda_7 = -\mu < 0 \quad (56)$$

For λ_6 to be negative, then (57)

$$-\left(\frac{\sigma\vartheta\beta_1 k_6 S^* + \sigma\theta\beta_2 k_5 S^* - k_4 k_5 k_6}{\sigma\vartheta\beta_1 S^* - k_4 k_5}\right) < 0$$

$$\frac{\sigma\vartheta\beta_1 k_6 S^* + \sigma\theta\beta_2 k_5 S^* - k_4 k_5 k_6}{k_4 k_5 - \sigma\vartheta\beta_1 S^*} < 0$$

This is true only if

$$\sigma\vartheta\beta_1 k_6 S^* + \sigma\theta\beta_2 k_5 S^* - k_4 k_5 k_5 < 0$$

$$\sigma\vartheta\beta_1 k_6 S^* + \sigma\theta\beta_2 k_5 S^* < k_4 k_5 k_5$$

$$\frac{\sigma S^*(\vartheta\beta_1 k_6 + \theta\beta_2 k_5)}{k_4 k_5 k_6} < 1$$

Substituting the value S^* and Simplifying gives

$$\frac{\sigma k_3(\Lambda\omega_1 + \eta k_1)(\vartheta\beta_1 k_6 + \theta\beta_2 k_5)}{k_1 k_4 k_5 k_6 (k_2 k_3 - \rho\omega_2)} < 1 \quad (58)$$

i.e

$R_c < 1$ (59)

Thus, $\lambda_5 < 0$ if $R_c < 1$, implying that all the eigenvalues have negative real parts, we therefore, established the following result.

Theorem 2: The disease-free equilibrium E_f of the model is locally asymptotically stable (LAS) if $R_c < 1$.

The epidemiological implication of this theorem is that disease can be eliminated (control) from the population when $R_c < 1$, if the initial size of the sub-populations of the model are in the basin of attraction of the DFE.

6.0 Global Stability of Disease-free Equilibrium, E_f

In order to ensure that the disease-free equilibrium (DFE) is independent of the initial size of the sub-populations of the model, it is necessary to show that the DFE is globally asymptotically stable (GAS). There are many ways of proving the global stability of disease-free equilibrium which include among others the Lyapunov theorem and the Castillo-Chavez global stability theorem [14]. We used the latter in this paper.

Theorem 3: The disease-free equilibrium, E_f of (1) – (7) is globally asymptotically stable (GAS) if $R_c < 1$.

Proof: To establish the global stability of the disease-free equilibrium, the two conditions (H1) and (H2) as in [14,15] must be satisfied for $R_c < 1$. The model system (17)-(23) can be written in the form

$$X_1'(t) = F(X_1, X_2) \tag{60}$$

$$X_2'(t) = G(X_1, X_2);\ G(X_1, 0) = 0 \tag{61}$$

where $X_1 = (M^*, S^*, V^*, R^*)$ and $X_2 = (L^*, I_1^*, I_2^*)$ with the components of $X_1 \in R^4$ denoting the uninfected individuals and the components of $X_2 \in R^3$ denoting the infected individuals.

The disease-free equilibrium is now denoted as

$$E = (X_1^*, 0) \tag{62}$$

where

$$X_1^* = (M^*, S^*, V^*, R^*) \tag{63}$$

Now, to proof that the first condition, $(H1)$ for $X_1'(t) = F(X_1^*, 0)$ is true, i.e X_1^* is a globally asymptotically stable. We have linear differential equations as thus

$$X_1'(t) = F(X_1, 0) = \begin{pmatrix} \Lambda - k_1 M^* \\ \omega_1 M^* + \omega_2 V^* + \eta - k_2 S^* \\ \rho S^* - k_3 V^* \\ -\mu R^* \end{pmatrix} \tag{64}$$

Solving gives

$$M^*(t) = \frac{\Lambda}{k_1}\left(1 - e^{-k_1 t}\right) + M^*(0) e^{-k_1 t} \tag{65}$$

$$S^*(t) = \left(\frac{\omega_1 M^*(t) + \omega_2 V^*(t) + \eta}{k_2}\right)\left(1 - e^{-k_2 t}\right) + S^*(0) e^{-k_2 t} \tag{66}$$

$$V^*(t) = \frac{\rho S^*}{k_3}\left(1 - e^{-k_3 t}\right) + V^*(0) e^{-k_3 t} \tag{67}$$

$$R^*(t) = R^*(0)e^{-\mu t} \tag{68}$$

Now, clearly from (24), we have that $M^*(t)+S^*(t)+V^*(t)+R^*(t) \rightarrow N^*(t)$ as $t \rightarrow \infty$ regardless of the value of $M^0(0)$, $S^0(0)$, V^0, and $R^0(0)$. Thus, $X_1^* = (N^0, 0)$ is globally asymptotically stable.

Next, to prove that the second condition $(H2)$ is true, that is $\hat{G}(X_1, X_2) = AX_2 - G(X_1, X_2)$ gives

$$A = \begin{pmatrix} -k_4 & \beta_1 S & \beta_2 S \\ \sigma\vartheta & -k_5 & 0 \\ \sigma\theta & 0 & -k_6 \end{pmatrix} \tag{69}$$

Since from (20) and (21), $k_4 k_5 > \sigma\vartheta\beta_1 S^*$ then $(\sigma\vartheta\beta_1 S^* - k_4 k_5) < 0$

Thus, it is clear that matrix A is an M-matrix (the off-diagonal elements of A are non-negative).

$$G(X_1, X_2) = \begin{pmatrix} (\beta_1 I_1 + \beta_2 I_2) S^* - k_4 L^* \\ \sigma\vartheta L^* - k_5 I_1^* \\ \sigma\theta L^* - k_6 I_2^* \end{pmatrix} \tag{70}$$

then,

$$\hat{G}(X_1, X_2) = \begin{pmatrix} -k_4 & \beta_1 S^* & \beta_2 S^* \\ \sigma\vartheta & -k_5 & 0 \\ \sigma\theta & 0 & -k_6 \end{pmatrix} \begin{pmatrix} L^* \\ I_1^* \\ I_2^* \end{pmatrix} - \begin{pmatrix} (\beta_1 I_1^* + \beta_2 I_2^*) S^* - k_4 L^* \\ \sigma\vartheta L^* - k_5 I_1^* \\ \sigma\theta L^* - k_6 I_2^* \end{pmatrix}$$

Recall that at disease-free equilibrium, $L^* = I_1^* = I_2^* = 0$. Thus

$$\hat{G}(X_1, X_2) = AX_2 - G(X_1, X_2) = \begin{pmatrix} 0 \\ 0 \\ 0 \end{pmatrix} \tag{71}$$

i.e.

$$\hat{G}(X_1, X_2) = (0 \quad 0 \quad 0)^T \tag{72}$$

It is thus obvious that $\hat{G}(X_1, X_2) = 0$. Hence, the proof is complete.

7.0 Conclusion

Model formulation with inclusion of some vital factors that plays significant role in the recruitment dynamics and control of measles infection can lead to elimination of measles in the population. These factors considered include maternal immunity, vaccine effects and presence of strong and weak vitamin A in the body. We found the local and global stability of the disease free equilibrium and showed that the disease free equilibrium are both locally and globally asymptotically stable for $R_c < 1$ and $R_c \leq 1$. The effective reproductive number was computed and we demonstrated that it is one of the most effective tool in gaining an insight into the transmission dynamics of measles infection under different situations. The model strongly indicated that the spread of measles infection largely depend on the contact rates with infected individuals within the population. Our analysis reveals that measles infection can be controlled if the effective reproduction number R_c is less than unity regardless of the initial population profile. Thus, every effort must be put in place by all agencies concerned to prevent disease by reducing R_c strictly less than unity.

8.0 References

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