



## The Analysis and the Solution of Incubation Period in a Disease Model

Niongon R. Anongo<sup>1,a</sup>, Sikari Hickson<sup>2</sup>, Kulari Tanzamado O.<sup>3</sup>, Galaya Tirah<sup>4</sup>, Atimi Atinga<sup>5</sup>, Alvary K. Kwala<sup>6</sup>

<sup>1</sup>Department of Mathematics, American University of Nigeria schools-Charter, Yola, Nigeria

<sup>2</sup>Department of Biological Sciences, Adamawa State College of Education, Hong, Yola, Nigeria

<sup>3</sup>Department of Mathematics Education, Adamawa State Polytechnics, Yola, Nigeria

<sup>4</sup>Department of Integrated Sciences, Adamawa State College of Education, Hong, Yola, Nigeria

<sup>5</sup>Department of Biological Science, American University of Nigeria schools-Charter, Yola, Nigeria

<sup>6</sup>Department of Mathematics, Adamawa State Polytechnics, Yola, Nigeria

<sup>a</sup>ranongo@gmail.com

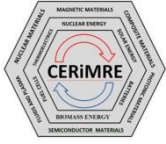
**Abstract.** *This study deals with the analysis and the solution of incubation period in a disease model by adopting the mathematical model with incubation period of diseases and the mathematical model without the incubation period of diseases. In the model equations, we partitioned the population into Susceptible (S), Incubated (I), Infected (D) population. We have compared the model equations without incubation period with the model equation with incubation period by solving and incorporating the system of first order linear equations into fourth order Runge-kutta method which has better error accuracy for solving first order equations. Graphical results for incubation class show that the infectious diseases were fatal if immediate attention is not given to endemic villages and communities.*

**Keywords:** SID Model, Incubation period, Runge-kutta method, numerical simulation, transmission

### Introduction

Most fundamental laws of science and engineering are based on models that explain variation in physical properties described by differential equations. The mathematical study of epidemics has come up with an astonishing number of mathematical models with explanations for spread and causes of epidemic outbreaks [1]. It is a well-established fact that the order of magnitude of deaths due to socio-economic diseases are more than anything else in the world. In recent years, several studies have come up, which have not only explained various diseases due to socio-economic aspect but gained triumphs for developing medicine [1], [2].

Disease can be infectious and non-infectious. An infectious disease can be transmitted from one person to another (COVID-19, SARS, Ebola and tuberculosis etc.), while a non-infectious disease cannot be spread through person-to-person contact (cancer, Alzheimer's disease and epilepsy



etc.) [3]. An infectious disease can be defined as an illness due to a pathogen or its toxic product, which arises through transmission from an infected person, an infected animal, or a contaminated inanimate object to a susceptible host [4]. Infectious diseases are responsible for an immense global burden of disease that impacts public health systems and economies worldwide, disproportionately affecting vulnerable populations [5]. Therefore, the incubation period is defined as the time from exposure to onset of disease [1], and when limited to infectious diseases, corresponds to the time from infection with a microorganism to symptom development.

The incubation period of infectious diseases ranges from the order of a few hours, which is common for toxic food poisoning, to a few decades as seen in the case of tuberculosis, AIDS and variant Creutzfeldt-Jakob disease (vCJD) [6]. Since symptom onset reflects pathogen growth and invasion, excretion of toxins and initiation of host-defense mechanisms, the length of the incubation period varies largely according to the replication rate of the pathogen, the mechanism of disease development, the route of infection and other underlying factors [7]. During the incubation period of acute infectious diseases, which is subsequently followed by a symptomatic period, it should be noted that the infected host can be infectious [1]. Whereas the incubation and symptomatic periods are distinguished by symptom onset, other epidemiologic terms are distinguished by acquisition of infectiousness [1]. That is, the time from infection to acquisition of infectiousness is referred to as the latent period, which is subsequently followed by the infectious period [8]. These two concepts are clearly separated by definition and are not directly related. The incubation period of infectious diseases offers various insights into clinical and public health practices, as well as being important for epidemiologic and ecological studies.

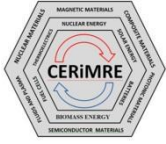
This research studied numerically a mathematical model with incubation period and a mathematical model without incubation period by adopting model with incubation period and model without incubation period developed by [1] and latter modified by [2].

## **Theoretical Background**

Incubation period in a disease model can be traced back to the mid-16th century when Girolamo Fracastoro (Fracastorius) (1478–1553), an Italian physician, documented for the first time the incubation period of rabies in 1546 [3]. Recently, [6], [7], noted that incubation period is frequently used to determine the infecting exposure in foodborne outbreaks and can assist in diagnosis when laboratory resources are unavailable. Criteria were developed and are frequently employed to determine whether an outbreak was caused by norovirus; the incubation period is one of the key elements of these criteria [4]. Also, [8] noted that incubation period is important for accurate surveillance for healthcare associated infections and implementation of effective outbreak control measures (e.g., quarantine and isolation). A summary of the incubation periods for various contagious diseases can be found in **Table 1**. This table highlights the time frame between exposure to the pathogen and the onset of symptoms for different diseases.

## **Materials and Methods**

In epidemiology, the population can be classified into two broad classes viz: Susceptible and infected class. The susceptible populations are prone to infection and infected population can transmit the infection to the susceptible ones [1]. Figure 1 presents a flow diagram illustrating the model with an incubation phase. This diagram outlines the stages and processes involved,



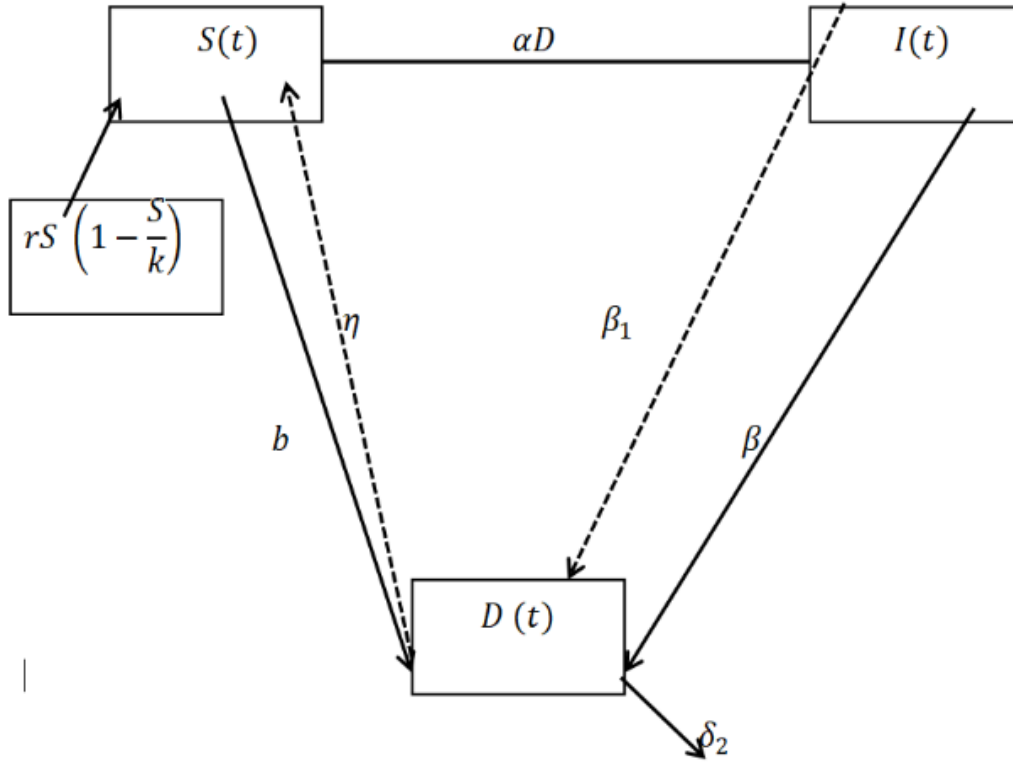
highlighting the incubation period as a key component of the model's progression. The following models are taken from [1].

**Table 1.** Summary of the incubation period of some contagious/communicable diseases

S/N	Diseases	Incubation Period
i	Cellulitis caused by <i>Pasteurella multocida</i>	0 & 1 days, [9].
ii	Chicken pox	9 & 12 days, [10].
iii	Dengue fever	3 & 14 days, [11].
iv	Cholera	0.5 & 4.5 days, [12].
v	Erythema infectiosum	13 & 18 days, [13].
vi	Ebola	1 & 21 days, [14].
vii	Rosela	5 & 15 days, [15].
viii	HIV	2 & 3 weeks to months or longer, [16].
ix	Infectious Mononucleosis (glandular fever)	28 & 42 days, [17].
x	Kuru disease	10.3 & 13.2 years (mean), [18].
xi	Marburg	5 & 10 days, [19].
xii	Measles	9 & 12 days, [20].
xiii	Mumps	14 & 18 days, [21].
xiv	Covid-19	2 to 14 days, [22].

### Model Parameters

- $S(t)$  = Susceptible population at time  $t$   
 $I(t)$  = Incubating population at time  $t$   
 $D(t)$  = Infected population at time  $t$   
 $N(t)$  = Total population at time  $t$   
 $\gamma$  = Intrinsic growth rate  
 $b$  = The disease contact rate  
 $k$  = Carrying capacity  
 $\delta_1 \delta_2$  = The rate of removable population from disease contact rate including death due to disease and natural causes  
 $\eta$  = Fraction of infected population that will rejoin in susceptible class  
 $\beta_1$  = The fraction of incubated class that will go to the disease class  
 $\beta$  = The rate of removable population from incubated class  
 $\alpha$  = Disease contact rate



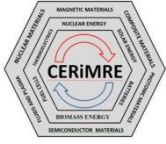
**Figure 1.** Flow Diagram of the Model with Incubation

### *Assumption of the Model*

The assumptions of the model are as follows:

- (i) The population is fixed
- (ii) The only way a person can leave the susceptible group is to become incubated. The only way a person can leave incubated class is to become infected. The only way a person can leave infected class is to rejoin the susceptible class.
- (iii) We also assume that there is no vertical transmission of the disease.
- (iv) Age, sex, social status and race does not affect the probability of being infected.
- (v) There is no inherited immunity within the system.
- (vi) The members of the population mix homogenously (having the same interaction with one another at the same time).

The dotted line indicates some of the incubated class  $\beta_1$  that will be rejoining the disease class. As  $\eta$  dotted line indicates some of the infected members that may later rejoin the susceptible class only.



### *The Model Equations with Incubation*

The equation for the susceptible class at time  $t$  is

$$S'(t) = rs \left(1 - \frac{S}{k}\right) - \alpha SD + \eta D \quad (1)$$

The equation for the incubated class at time  $t$  is

$$I(t) = \alpha SD - \beta I \quad (2)$$

The equation for the infected class at time  $t$  is

$$D'(t) = \beta_1 I - \delta_2 D \quad (3)$$

The equation for population size at time  $t$  is

$$N(t) = S(t) + I(t) + D(t) \quad (4)$$

### *The Model Equations without Incubation*

The equation for the susceptible class at time  $t$  is

$$S'(t) = rs \left(1 - \frac{S}{k}\right) - bSD + \eta D \quad (5)$$

The equation for the infected class at time  $t$  is

$$D'(t) = bSD - \delta D \quad (6)$$

### *The Runge-Kutta Method of Order 4*

Consider the system of autonomous ordinary differential equations

$$\begin{aligned} \frac{dx_1}{dt} &= f_1(x_1, x_2, \dots, x_n) \\ \frac{dx_2}{dt} &= f_2(x_1, x_2, \dots, x_n) \\ \frac{dx_n}{dt} &= f_n(x_1, x_2, \dots, x_n) \end{aligned} \quad (7)$$

Which can simply be expressed as

$$\frac{dx}{dt} = f(x) \quad (8)$$

If (8) is a one-dimensional problem, we can state the fourth order Runge Kutta method for the numerical solution of (8) as follows;

$$y_{i+1} = y_i + \frac{1}{6} (k_1 + 2k_2 + 2k_3 + k_4) h \quad (9)$$

where

$$\begin{aligned} k_1 &= f(x_i, y_i) \\ k_2 &= f\left(x_i + \frac{1}{2}h, y_i + \frac{1}{2}k_1h\right) \\ k_3 &= f\left(x_i + \frac{1}{2}h, y_i + \frac{1}{2}k_2h\right) \\ k_4 &= f(x_i + h, y_i + k_3h) \end{aligned}$$

However, we expressed (1-3) as a system of 3x3-dimensional vector equations gotten from mathematics model with incubation and (1-2) as a system of 2x2-dimensional vector equations gotten from model without incubation period for  $t \geq 0$ . Following [23], [24] and the reference there in, we can approximate these vectors as follows;

*Runge-kutta method of order 4 for a 3x3 system of ordinary differential equations of model with incubation.*

$$\begin{aligned} \begin{pmatrix} k_1S \\ k_1I \\ k_1D \end{pmatrix} &= h \begin{pmatrix} f(t_i, S) \\ f(t_i, I) \\ f(t_i, D) \end{pmatrix} \\ \begin{pmatrix} k_2S \\ k_2I \\ k_2D \end{pmatrix} &= h \begin{pmatrix} f\left(t_i + \frac{1}{2}, S_i + \frac{k_1}{2}\right) \\ f\left(t_i + \frac{1}{2}, I_i + \frac{k_1}{2}\right) \\ f\left(t_i + \frac{1}{2}, D_i + \frac{k_1}{2}\right) \end{pmatrix} \\ \begin{pmatrix} k_3S \\ k_3I \\ k_3D \end{pmatrix} &= h \begin{pmatrix} f\left(t_i + \frac{1}{2}, S_i + \frac{k_2}{2}\right) \\ f\left(t_i + \frac{1}{2}, I_i + \frac{k_2}{2}\right) \\ f\left(t_i + \frac{1}{2}, D_i + \frac{k_2}{2}\right) \end{pmatrix} \\ \begin{pmatrix} k_4S \\ k_4I \\ k_4D \end{pmatrix} &= h \begin{pmatrix} f(t_i + 1, S_i + k_3) \\ f(t_i + 1, I_i + k_3) \\ f(t_i + 1, D_i + k_3) \end{pmatrix} \\ \begin{pmatrix} S_i + 1 \\ I_i + 1 \\ D_i + 1 \end{pmatrix} &= \begin{pmatrix} S_i \\ I_i \\ D_i \end{pmatrix} + \frac{1}{6} \left[ \begin{pmatrix} k_1S \\ k_1I \\ k_1D \end{pmatrix} + 2 \begin{pmatrix} k_2S \\ k_2I \\ k_2D \end{pmatrix} + 2 \begin{pmatrix} k_3S \\ k_3I \\ k_3D \end{pmatrix} + \begin{pmatrix} k_4S \\ k_4I \\ k_4D \end{pmatrix} \right] \end{aligned} \quad (10)$$

**Runge–kutta method of order 4 for a 2x2 system of ordinary differential equations of model without incubation**

$$\begin{aligned} \begin{pmatrix} k_1S \\ k_1D \end{pmatrix} &= h \begin{pmatrix} f(t_i, S) \\ f(t_i, D) \end{pmatrix} \\ \begin{pmatrix} k_2S \\ k_2D \end{pmatrix} &= h \begin{pmatrix} f\left(t_i + \frac{1}{2}, S_i + \frac{k_1}{2}\right) \\ f\left(t_i + \frac{1}{2}, D_i + \frac{k_1}{2}\right) \end{pmatrix} \\ \begin{pmatrix} k_3S \\ k_3D \end{pmatrix} &= h \begin{pmatrix} f\left(t_i + \frac{1}{2}, S_i + \frac{k_2}{2}\right) \\ f\left(t_i + \frac{1}{2}, D_i + \frac{k_2}{2}\right) \end{pmatrix} \\ \begin{pmatrix} k_4S \\ k_4D \end{pmatrix} &= h \begin{pmatrix} f(t_i + 1, S_i + k_3) \\ f(t_i + 1, D_i + k_3) \end{pmatrix} \\ \begin{pmatrix} S_{i+1} \\ D_{i+1} \end{pmatrix} &= \begin{pmatrix} S_i \\ D_i \end{pmatrix} + \frac{1}{6} \left[ \begin{pmatrix} k_1S \\ k_1D \end{pmatrix} + 2 \begin{pmatrix} k_2S \\ k_2D \end{pmatrix} + 2 \begin{pmatrix} k_3S \\ k_3D \end{pmatrix} + \begin{pmatrix} k_4S \\ k_4D \end{pmatrix} \right] \end{aligned} \tag{11}$$

We therefore write a computer program in MATLAB to carry out numerical experiment of equation (10) and equation (11) using ode45 command for the numerical simulations.

*Numerical results*

**Table 2.** Parameters of the model without incubation

S/N	Parameters	Value
1	$\alpha$	0.72
2	$\beta_1$	0.01
3	$\beta$	0.50
4	$b$	0.50
5	$r$	0.8
6	$k$	100
7	$\eta$	0.1
8	$\delta$	0.20
9	$S(0)$	6
10	$D(0)$	3



**Table 3.** Parameters of model with incubation

S/N	Parameters	Value
1	$\alpha$	0.72
2	$\beta_1$	0.01
3	$\beta$	0.50
4	$b$	0.50
5	$r$	0.8
6	$k$	100
7	$\eta$	0.1
8	$\delta$	0.5
9	$S(0)$	6
10	$I(0)$	2
11	$D(0)$	3

We carried out some numerical experiments by assigning specific values to the parameters  $\alpha$ ,  $\beta_1$ ,  $\beta$ ,  $R$ ,  $k$ ,  $\eta$ ,  $\delta$  as defined in the model equation as well as the initial values to  $S(0)$ ,  $I(0)$ ,  $D(0)$  and  $N(0)$  as presented in **Table 2** and **Table 3**. The initial values assigned to **Table 2** are parameter of model without incubation and that of **Table 3** are the parameters of model with incubation.

## Results and Discussion

The curve in **Figure 2** exhibited a short and sharp rise at the initial stage which followed a temporally decline and a continuous rise. When observed critically it was discovered that, at the initial time  $t_0$ , a susceptible individual got contracted with two bacteria approximately. Immediately the bacteria began to multiply to produce five members as the time of exposure increased to day one ( $t_1$ ) in the body of the exposed individual. However, the body immune system began to fight the bacteria naturally and three members of the bacteria died off on day two to the eighth day ( $t_2 - t_8$ ), which caused the sharp decline of the curve. But on the tenth day ( $t_{10}$ ), the graph exhibited a continuous rise which showed that the bacteria were able to defeat the body's natural immune system and then, the pathogens continued to multiply in the body of the exposed individual exponentially. At this point the susceptible individual began to experience unusual body functions such as headaches, neck ache, stomach aches et cetera; measles and chicken pox have this kind of incubation period. In view of the above, serious measures should be put in place in endemic villages by the health workers and the government to help reduce and to curb the spread of such contagious diseases in the affected communities.

This pattern of infection highlights the dynamic interaction between the pathogen and the host's immune response. The initial rise in bacterial population underscores the rapid multiplication of the pathogen during the early stages of exposure. However, the subsequent decline reflects the



body's natural defense mechanisms attempting to control and eliminate the infection. Despite this temporary success, the eventual rise in bacterial levels demonstrates the pathogen's ability to overcome immune defenses, leading to the full onset of symptoms. This pattern of incubation and immune response is crucial in understanding the progression of contagious diseases like measles and chickenpox. The observed trends emphasize the importance of early intervention, as prompt medical and public health measures could help mitigate the pathogen's spread during the critical phases of its incubation and multiplication.

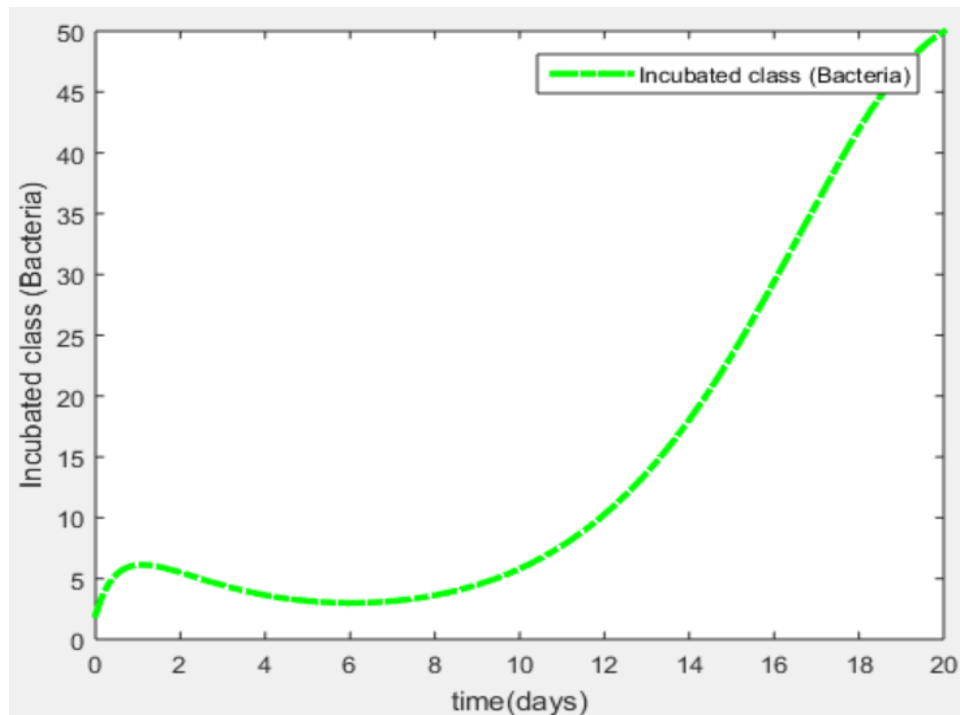


Figure 2. Graph of Incubated population versus time

Figure 3 compared the infected (disease) class with incubation and the infected (disease) class without incubation. In the infected class without incubation compartment, the increase in the infected members were fast and rapid on day one and day 2 as the disease spread from the three members of the population that were initially exposed to the infectious disease to infecting eight out of nine members of the population and followed an oscillatory and continuous declination. It is because most of the infectious diseases when one gets infected and did not die, he/she gain immunity from it which can resist him/her from re-infection even though he/she is expose to it again. As such, the body immunity continues to fight the disease and then the disease may die off from the population naturally. In the graph with incubation period, the graph began to decline from the three members of the population that was exposed to the disease and then got to day 10 where it seems as if no member of the population was infected and then rises to one member of the population at day 23 momentarily. By comparison with the model without incubation period we can say that the population with the model with occupation exhibited a high body resistance to fighting the disease than that of model without incubation period. As observed from the

continued declination of the model with incubation, even the three members that were exposed to the disease actually survived as the graph hit the zero-level population. The sudden rise at day 16 may occurred as a result of people violating the measures put in place to curtailed the infectiousness of the disease.

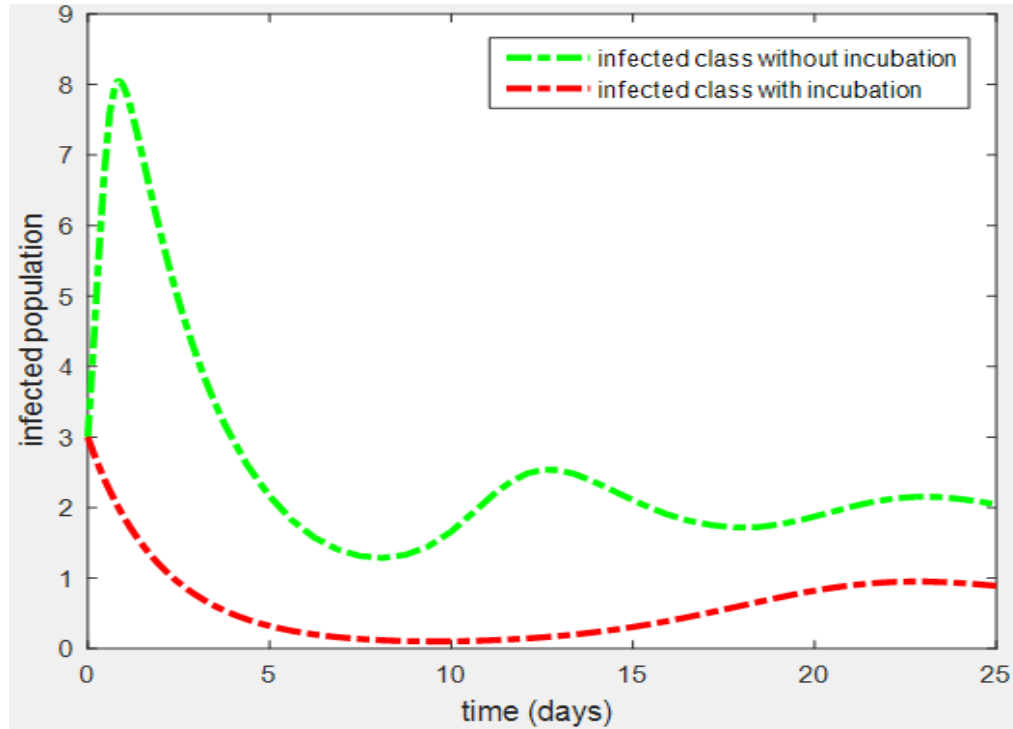


Figure 3. Graph of infected population versus time

## Conclusion

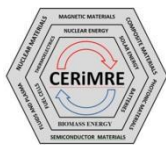
In this research, we have numerically analyzed a mathematical model with three classes of population, namely; susceptible, incubated and infected populations. The model equations were solved using fourth order Runge-Kutta method. Graphical result for incubation class show that the infectious diseases were fatal if immediate attention is not given to endemic villages and communities. Also, we compared the model with incubation period compartment and the model without incubation period.

## ACKNOWLEDGEMENTS

Our profound gratitude goes to Atimi ATinga (PhD) who has taken reasonable time to read and edit this research work.

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