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# A Comparative Study of the Role of Constant and Logistic Recruitment Rates in Epidemiological Models

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## Abstract

Most epidemiological models are formulated by either incorporating constant or logistic recruitment rates into the susceptible class by researchers. The role these recruitment rates play are often not emphasized in most mathematical modelling studies. In this paper, three mathematical models of epidemiology are presented. The next generation operator method is used to compute the reproduction number for each of the models. In each of the models (case I, case II and case III) a scenario where a constant and logistic recruitment rate are incorporated. It was observed in case I that, the reproduction number of the model with logistic recruitment rate is less than the reproduction number from the model with constant recruitment rate. Further, in case II, the reproduction number from the model with both constant and logistic recruitment rates are the same. Finally, in case III, it was observed that the human reproduction number from the model with logistic recruitment rate is higher than the human reproduction number from the model with constant recruitment rate. Consequently, it is recommended that more mathematical models of epidemiology should be deployed as an extension of this study for a reasonable conclusion to be drawn on the roles of these recruitment rates.

**Keywords:** Constant recruitment rate, epidemiological models, logistic recruitment rate, reproduction number

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## Introduction

Over the last three decades, mathematical models have been deployed by several researchers in modelling natural phenomenon or occurrences/events in our environment with the sole aim of analyzing, understanding and taking informed decisions from the results of such studies. Some of these models have been successfully designed and applied to infectious diseases, see for example [1–5].

Mathematical models have over the years provided useful insights into the transmission dynamics, prevention and control of infectious diseases [1, 6]. These models when successfully developed often assume that the recruitment rates of individuals into the susceptible class are either by constant rate, logistics recruitment rate or population dependent.

In this study, however, the emphasis shall be laid on the inclusion of constant and logistic recruitment rates into the susceptible class. It is instructive to note that some mathematical models are formulated by incorporating constant recruitment rates into the susceptible class, (see for example, [1, 6–15] while others are developed by incorporating logistic recruitment rate into the susceptible class of either human or animal sub-population, see for example, [16–18]. Mathematical models developed by incorporating constant recruitment rate into the susceptible class often assume that a population grows proportionately with the populations current size as noted in [4] and of course, those designed and analyzed incorporating the logistic recruitment rate into the susceptible class often assume that the population changes at all time but obviously not more than the maximum population size that a particular environment can support (called the carrying capacity, $K$) [4].

Most developed models of epidemiology often incorporate constant or logistic recruitment rates in their studies, see for example, [1, 3, 5, 6, 11, 14]. In all the aforementioned studies, none to the best of our knowledge have looked at the role of incorporating constant and logistic recruitment rates into the susceptible class as it affects the reproduction number.

The goal of this paper, therefore, is to make a comparative study of the role of constant and logistic recruitment rates in epidemiological models particularly as it affects the reproduction numbers.

### Model Formulation

In this section, a sample of mathematical models are presented to study the role of constant and logistic recruitment rates in some epidemiological models as it affects their reproduction numbers. The variables and parameters of the model equations (1) and (2) are presented in Table 1.

**Case I:** (a) An SIR model with constant recruitment rate (A generalized SIR model)

**Table 1: Variables and parameters of models (1) and (2)**

| Variables/Parameters | Interpretation |
|---|---|
| $S$ | Susceptible individuals |
| $I$ | Infected individuals |
| $R$ | Recovered individuals |
| $\mu$ | Natural death rate |
| $\gamma$ | Progression rate |
| $\delta$ | Disease-induced death rate |
| $\alpha$ | Infection rate |
| $r$ | Birth rate |
| $K$ | Carrying capacity |

$$\begin{aligned}\frac{dS(t)}{dt} &= \Lambda - \alpha SI - \mu R,\\ \frac{dI(t)}{dt} &= \alpha SI - (\mu+\gamma+\delta)I,\\ \frac{dR(t)}{dt} &= \gamma I - \mu R.\end{aligned} \quad (1)$$

(b) An SIR model with logistic recruitment rate (A generalized SIR model)

$$\begin{aligned}\frac{dS(t)}{dt} &= rN\left(1-\frac{N}{K}\right) - \alpha SI - \mu R,\\ \frac{dI(t)}{dt} &= \alpha SI - (\mu+\gamma+\delta)I,\\ \frac{dR(t)}{dt} &= \gamma I - \mu R.\end{aligned} \quad (2)$$

**Case II: (a) A diphtheria model with constant recruitment rate by Kanchanarat *et al.* [4]**

In models (3) and (4), the interaction is between the susceptible ($S$), vaccinated ($V$), exposed ($E$), asymptomatic ($A$), infected ($I$) and recovered ($R$) individuals at time, $t$. The parameters of the model are tabulated in Table 2.

**Table 2: Parameters of models (3) and (4)**

| Parameters | Interpretation |
|---|---|
| $\beta$ | Transmission rate |
| $a$ | Proportion of infectious population |
| $\delta$ | Modification parameter |
| $\phi$ | Rate of vaccination |
| $r$ | Birth rate |
| $\sigma$ | Progression rate |
| $\mu$ | Natural death rate |
| $\alpha$ | Diphtheria mortality rate |
| $\varepsilon$ | Waning rate of vaccine |
| $\theta$ | Progression rate |
| $\gamma$ | Recovery rate of asymptomatic individuals |
| $\tau$ | Recovery rate of infected individuals |
| $K$ | Carrying capacity |

$$\begin{aligned}\frac{dS(t)}{dt} &= \Lambda - \frac{\beta S(\delta A+I)}{N} - (\mu+\phi)S + \varepsilon V,\\ \frac{dV(t)}{dt} &= \phi S + \theta R - (\mu+\varepsilon)V,\\ \frac{dE(t)}{dt} &= \frac{\beta S(\delta A+I)}{N} - (\mu+\sigma)E,\\ \frac{dA(t)}{dt} &= (1-\alpha)\sigma E - (\mu+\gamma)A,\\ \frac{dI(t)}{dt} &= a\sigma E - (\mu+\alpha+\tau)I,\\ \frac{dR(t)}{dt} &= \gamma A + \tau I - (\mu+\theta)R\end{aligned} \quad (3)$$

**(b) A diphtheria model with logistic recruitment rate by Kanchanarat *et al.* [4].**

$$\begin{aligned}\frac{dS(t)}{dt} &= rN\left(1-\frac{N}{K}\right) - \frac{\beta S(\delta A+I)}{N} - (\mu+\phi)S + \varepsilon V,\\ \frac{dV(t)}{dt} &= \phi S + \theta R - (\mu+\varepsilon)V,\\ \frac{dE(t)}{dt} &= \frac{\beta S(\delta A+I)}{N} - (\mu+\sigma)E,\\ \frac{dA(t)}{dt} &= (1-a)\sigma E - (\mu+\gamma)A,\\ \frac{dI(t)}{dt} &= a\sigma E - (\mu+\alpha+\tau)I,\\ \frac{dR(t)}{dt} &= \gamma A + \tau I - (\mu+\theta)R.\end{aligned} \quad (4)$$

**Case III: (a) Monkeypox mathematical model with constant recruitment rate by Ashezua *et al.* [5].**

In models (5) and (9), the interaction is between human and animal population. For the human population, the susceptible ($S_H$), vaccinated ($V_H$), exposed ($E_H$), asymptomatic ($A_H$), symptomatic ($I_H$), isolated ($T_H$) and recovered ($R$) individuals interact at time, $t$ while for the animal population, susceptible ($S_A$), exposed ($E_A$), infected ($I_A$) and recovered ($R_A$) animals interact at time, $t$.

**Table 3: The parameters values of models (5) & (9)**

| Param. | Interpretation |
|---|---|
| $\Lambda_h$ | Recruitment rate for susceptible human |
| $v$ | Vaccination rate of susceptible human |
| $\omega$ | Waning rate of the monkeypox vaccine |
| $\mu_h$ | Natural death rate for the humans |
| $\delta_h$ | Monkeypox induced death rate |
| $\theta$ | Progression rate from $E_H$to $A_H$ |
| $\rho$ | Proportion of the exposed persons moving to class $A_H$ |
| $\phi$ | Progression from $A_H$ to $I_H$ |
| $\sigma$ | Natural recovery rate of the asymptomatic individuals |
| $\tau$ | Treatment rate for the symptomatic individuals |
| $\eta$ | Modification parameter |
| $\varepsilon$ | Rate of public awareness campaign |
| $\psi$ | Efficacy of public awareness campaign |
| $\beta_h$ | Probability of transmitting monkeypox from human-to-human |
| $(\beta_a, \beta_r)$ | Probability of transmitting monkeypox from animal-to-human and animal-to-animal |
| $\Lambda_a$ | Recruitment rate into the susceptible animal compartment |
| $\mu_a$ | Natural death rate for the animal population |
| $\varphi$ | Progression rate from $E_A$ to $I_A$. |
| $\gamma_a$ | Recovery rate for the animal population |
| $\delta_a$ | Disease induced death rate for the animal population |

$$\frac{dS_H(t)}{dt} = \Lambda_h - \lambda_H S_H + \omega V_H - (v + \mu_h)S_H,$$
$$\frac{dV_H(t)}{dt} = vS_H - (\omega + \mu_h)V_H,$$
$$\frac{dE_H(t)}{dt} = \lambda_H S_H - [\theta\rho + \theta(1-\rho) + \mu_h]E_H,$$
$$\frac{dA_H(t)}{dt} = \theta\rho E_H - (\sigma + \phi + \mu_h)A_H,$$
$$\frac{dI_H(t)}{dt} = \phi A_H + \theta(1-\rho)E_H - (\tau + \mu_h + \delta_h)I_H,$$
$$\frac{dT_H(t)}{dt} = \tau I_H - (\gamma_h + \mu_h + \delta_h)T_h \quad (5)$$
$$\frac{dR_H(t)}{dt} = \sigma A_H + \gamma_h T_H - \mu_h R_H,$$
$$\frac{dS_A(t)}{dt} = \Lambda_a - \lambda_A S_A - \mu_a S_A,$$
$$\frac{dE_A(t)}{dt} = \lambda_A S_A - (\varphi + \mu_a)E_A,$$
$$\frac{dI_A(t)}{dt} = \varphi E_A - (\gamma_a + \mu_a + \delta_a)I_A,$$
$$\frac{dR_A(t)}{dt} = \gamma_a I_A - \mu_a R_A.$$

where $\lambda_H$ and $\lambda_A$ are as given in equations (6) and (7), respectively.

$$\lambda_H = (1 - \varepsilon\psi)\left(\frac{\beta_h(I_H + \eta A_H)}{N_H} + \frac{\beta_a I_A}{N_A}\right) \quad (6)$$

Similarly, the susceptible animals acquire the infection following effective contact with an infected animal (i.e., those in the $I_A$ class) at a rate

$$\lambda_A = \frac{\beta_r I_A}{N_A} \quad (7)$$

with,

$$N(t) = N_H(t) + N_A(t),$$
$$N_H(t) = S_H(t) + V_H(t) + E_H(t) + A_H(t) + I_H(t) + T_H(t) + R_H(t), \quad (8)$$
$$S_A(t) + E_A(t) + I_A(t) + R_A(t).$$

**(b) Monkeypox mathematical model with logistic recruitment rate by Ashezua *et al.* [5].**

$$\frac{dS_H(t)}{dt} = rN_H\left(1 - \frac{N_H}{K}\right) - \lambda_H S_H + \omega V_H - (v + \mu_h)S_H,$$
$$\frac{dV_H(t)}{dt} = vS_H - (\omega + \mu_h)V_H,$$
$$\frac{dE_H(t)}{dt} = \lambda_H S_H - (\theta\rho + \theta(1-\rho) + \mu_h)E_H,$$
$$\frac{dA_H(t)}{dt} = \theta\rho E_H - (\sigma + \phi + \mu_h)A_H,$$
$$\frac{dI_H(t)}{dt} = \phi A_H + \theta(1-\rho)E_H - (\tau + \mu_h + \delta_h)I_H,$$
$$\frac{dT_H(t)}{dt} = \tau I_H - (\gamma_h + \mu_h + \delta_h)T_h \quad (9)$$
$$\frac{dR_H(t)}{dt} = \sigma A_H + \gamma_h T_H - \mu_h R_H,$$
$$\frac{dS_A(t)}{dt} = \Lambda_a - \lambda_A S_A - \mu_a S_A,$$
$$\frac{dE_A(t)}{dt} = \lambda_A S_A - (\varphi + \mu_a)E_A,$$
$$\frac{dI_A(t)}{dt} = \varphi E_A - (\gamma_a + \mu_a + \delta_a)I_A,$$
$$\frac{dR_A(t)}{dt} = \gamma_a I_A - \mu_a R_A.$$

where $\lambda_H$ and $\lambda_A$ are as given in equations (10) and (11), respectively.

$$\lambda_H = (1 - \varepsilon\psi)\left(\frac{\beta_h(I_H + \eta A_H)}{N_H} + \frac{\beta_a I_A}{N_A}\right) \quad (10)$$

Similarly, the susceptible animals acquire the infection following effective contact with an infected animal (i.e., those in the $I_A$ class) at a rate

$$\lambda_A = \frac{\beta_r I_A}{N_A} \quad (11)$$

with,

$$N(t) = N_H(t) + N_A(t),$$
$$N_H(t) = S_H(t) + V_H(t) + E_H(t) + A_H(t) + I_H(t) + T_H(t) + R_H(t), \quad (12)$$
$$N_A(t) = S_A(t) + E_A(t) + I_A(t) + R_A(t)$$

**Mathematical Analysis**

In this section, a sample of mathematical models are presented to study the role of constant and logistic recruitment rates in some epidemiological models.

**Case I: (a) An SIR model with constant recruitment rate [Generalized model].**

The disease-free equilibrium of the model (1) is given by $E_0 = (S^0, I^0, R^0) = \left(\frac{\Lambda}{\mu}, 0, 0\right)$.Using the notation in [19], the non-negative matrix $F$, of new infection terms and the M-matrix, $V$, of transition terms associated with the model (1) are

$F = \frac{\alpha\Lambda}{\mu}$ and $V = (\mu + \gamma + \delta)$.

It follows that the basic reproduction number of the model (1), denoted by $R_0 = \rho(FV^{-1})$ (where $\rho$ denotes the spectral radius), is given by

$$R_0 = \frac{\alpha\Lambda}{\mu(\mu + \gamma + \delta)} \quad (13)$$

**(b) An SIR model with logistic recruitment rate [Generalized model].**

Here, the disease-free equilibrium of the model is $E_0 = (S^0, I^0, R^0) = (K, 0, 0)$. The non-negative matrix $F$, of new infection terms and the M-matrix, $V$, of transition terms associated with the model (2) are

$F = \alpha K$ and $V = (\mu + \gamma + \delta)$.

Hence, the basic reproduction number of the model (2), denoted by $R_0$, is given by

$$R_0 = \frac{\alpha K}{\mu(\mu + \gamma + \delta)} \quad (14)$$

**Case II: (a) A diphtheria model with constant recruitment rate by Kanchanarat *et al.* [4].**

The disease-free equilibrium of the model (3) is given by

$E_0 = (S^0, V^0, E^0, A^0, I^0, R^0) = \left(\frac{\mu(\mu+\varepsilon+\phi)}{(\mu+\varepsilon)}, \frac{\phi\mu(\mu+\varepsilon+\phi)}{(\mu+\varepsilon)^2}, 0, 0, 0, 0\right)$.

The non-negative matrix $F$, of new infection terms and the M-matrix, $V$, of transition terms associated with the model (3) are

$$F = \begin{pmatrix} 0 & \frac{k_1\delta\beta}{(k_1+\phi)} & \frac{k_1\beta}{(k_1+\phi)} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} k_3 & 0 & 0 \\ -k_5 & k_4 & 0 \\ -a\sigma & 0 & k_6 \end{pmatrix}$$

where, $k_3 = (\mu+\sigma), k_4 = (\mu+\gamma), k_5 = (1-a)\sigma$ and $k_6 = (\mu+\alpha+\tau)$.

Thus, the basic reproduction number of the model (3), denoted by $R_v$, is given by

$$R_v = \frac{k_1\beta(a\sigma k_4 + \delta k_5 k_6)}{k_3 k_4 k_6 (k_1+\phi)} \qquad (15)$$

**(b) A diphtheria model with logistic recruitment rate by Kanchanarat *et al*. [4].**

The disease-free equilibrium state of the model (4) is $E_0 = (S^0, V^0, E^0, A^0, I^0, R^0) = \left(\frac{(r-\mu)(\mu+\varepsilon)K}{r(\mu+\phi+\varepsilon)}, \frac{(r-\mu)\phi K}{r(\mu+\phi+\varepsilon)}, 0,0,0,0\right)$.

The non-negative matrix $F$, of new infection terms and the M-matrix, $V$, of transition terms associated with the model (4) are

$$F = \begin{pmatrix} 0 & \frac{\beta\delta(\mu+\varepsilon)}{(\mu+\phi+\varepsilon)} & \frac{\beta(\mu+\varepsilon)}{(\mu+\phi+\varepsilon)} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \text{and}$$

$$V = \begin{pmatrix} (\mu+\sigma) & 0 & 0 \\ -(1-a)\sigma & (\mu+\gamma) & 0 \\ -a\sigma & 0 & (\mu+\alpha+\tau) \end{pmatrix}$$

respectively. Hence, the reproduction number of the model (4) is computed to be

$$R_v = \frac{\beta\sigma(\mu+\varepsilon)[a(\mu+\gamma)+\delta(1-a)(\mu+\alpha+\tau)]}{(\mu+\sigma)(\mu+\gamma)(\mu+\alpha+\tau)(\mu+\phi+\varepsilon)} \qquad (16)$$

**Case III: (a) Monkeypox mathematical model (5) with constant recruitment rate by Ashezua *et al.* [5].**

The disease-free equilibrium of the model (5), is given by $E_0 = (S_H^0, V_H^0, E_H^0, A_H^0, I_H^0, T_H^0, R_H^0, S_A^0, E_A^0, I_A^0, R_A^0)$, where

$S_H^0 = \frac{\Lambda_h k_2}{k_1 k_2 - v\omega}, V_H^0 = \frac{v\Lambda_h}{k_1 k_2 - v\omega}, E_H^0 = 0, A_H^0 = 0, I_H^0 = 0, T_H^0 = 0, R_H^0 = 0, S_A^0 = \frac{\Lambda_a}{\mu_a}, E_H^0 = 0, I_A^0 = 0, R_A^0 = 0,$ with $k_1 = (v+\mu_h)$ and $k_2 = (\omega+\mu_h)$.

The local stability of $E_0$ will be determined using the next generation operator method on model (4). Using the notation in van den Driessche and Watmough [19], it follows that matrices $F$ and $V$, for the new infection terms and the remaining transition terms, respectively, are given by

$$F = \begin{pmatrix} 0 & y_1 & y_2 & 0 & y_3 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta_r \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \text{and}$$

$$V = \begin{pmatrix} k_3 & 0 & 0 & 0 & 0 \\ -\theta\rho & k_4 & 0 & 0 & 0 \\ -\theta(1-\rho) & -\phi & k_5 & 0 & 0 \\ 0 & 0 & 0 & k_7 & 0 \\ 0 & 0 & 0 & -\varphi & k_8 \end{pmatrix}$$

where,

$$y_1 = \frac{(1-\varepsilon\psi)\beta_h\eta k_2}{v+k_2}, y_2 = \frac{(1-\varepsilon\psi)\beta_h k_2}{v+k_2}, y_3 = \frac{(1-\varepsilon\psi)\beta_a\mu_a\Lambda_h k_2}{\Lambda_a(k_1k_2 - v\omega)}, \qquad (17)$$
$$k_3 = (\theta\rho + \theta(1-\rho) + \mu_h), k_4 = (\sigma+\phi+\mu_h), k_5 = (\tau+\mu_h+\delta_h), k_7 = (\varphi+\mu_a),$$

It follows that the reproduction numbers of the model (4) are

$$R_0 = \{R_{0h}, R_{0a}\} \qquad (18)$$

with $R_{0h}$ and $R_{0a}$ being the monkeypox induced reproduction numbers for the humans and animals, respectively, which are given by

$$R_{0h} = \frac{(1-\varepsilon\psi)\beta_h k_2[\eta k_5\theta\rho + \phi\theta\rho + k_4\theta(1-\rho)]}{k_3 k_4 k_5 (v+k_2)} \qquad (19)$$

and

$$R_{0a} = \frac{\beta_r\varphi}{k_7 k_8} \qquad (20)$$

The reproduction number (human) for the model (5) was computed to be

$$R_{0h} = \frac{(1-\varepsilon\psi)\beta_h k_2[\eta k_5\theta\rho + \phi\theta\rho + k_4\theta(1-\rho)]}{k_3 k_4 k_5 (v+k_2)} \qquad (21)$$

where,

$$k_2 = (\omega+\mu_h), k_3 = (\theta\rho + \theta(1-\rho) + \mu_h), k_4 = (\sigma+\phi+\mu_h), k_5 = (\tau+\mu_h+\delta_h) \qquad (22)$$

**(b) Monkeypox mathematical model (9) with logistic recruitment rate by Ashezua *et al.* [5].**

The disease-free equilibrium of the model (9), is given by,

$E_0 = (S_H^0, V_H^0, E_H^0, A_H^0, I_H^0, T_H^0, R_H^0, S_A^0, E_A^0, I_A^0, R_A^0)$, where,

$S_H^0 = \frac{K(r-\mu_h)(\omega+\mu_h)}{r(v+\omega+\mu_h)}, V_H^0 = \frac{vK(r-\mu_h)(\omega+\mu_h)}{r(v+\omega+\mu_h)}, E_H^0 = 0, A_H^0 = 0, I_H^0 = 0, T_H^0 = 0, R_H^0 = 0,$

$S_A^0 = \frac{\Lambda_a}{\mu_a}, E_H^0 = 0, I_A^0 = 0, R_A^0 = 0,$

Using the notation in van den Driessche and Watmough [19], it follows that matrices $F$ and $V$, for the new infection terms and the remaining transition terms, respectively, are given by

$$F = \begin{pmatrix} 0 & d_1 & d_2 & 0 & d_3 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta_r \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \text{ and } V = \begin{pmatrix} k_3 & 0 & 0 & 0 & 0 \\ -\theta\rho & k_4 & 0 & 0 & 0 \\ -k_5 & -\phi & k_6 & 0 & 0 \\ 0 & 0 & 0 & k_8 & 0 \\ 0 & 0 & 0 & -\varphi & k_9 \end{pmatrix}$$

where, $q = (1-\varepsilon\psi), d_1 = \frac{\beta_h q\eta}{(1+v)}, d_2 = \frac{\beta_h q}{(1+v)}, d_3 = \frac{q\beta_a\mu_a K(r-\mu_h)(\omega+\mu_h)}{\Lambda_a r(v+\omega+\mu_h)}$.

It follows that the reproduction numbers of the model (9) are

$$R_0 = \{R_{0h}, R_{0a}\} \tag{23}$$

with $R_{0h}$ and $R_{0a}$ being the monkeypox induced reproduction numbers for the humans and animals, respectively, which are given by

$$R_h = \frac{(1-\varepsilon\psi)\beta_h[\theta\rho(\eta k_6+\phi)+k_4k_5]}{k_3k_4k_6(1+v)} \tag{24}$$

and

$$R_{0a} = \frac{\beta_r\varphi}{k_8k_9} \tag{25}$$

The reproduction number (human) for the model (9) was computed to be

$$R_h = \frac{(1-\varepsilon\psi)\beta_h[\theta\rho(\eta k_6+\phi)+k_4k_5]}{k_3k_4k_6(1+v)} \tag{26}$$

where,

$$k_3 = [\theta\rho+\theta(1-\rho)+\mu_h], k_4 = (\sigma+\phi+\mu_h), k_5 = \theta(1-\rho), k_6 = (\tau+\mu_h+\delta_h) \tag{27}$$

## Results and Discussions

Here, we present the results of our findings in the previous section (particularly for equations (13), (14), (15), (16), (21) and (26)) using the parameter values on Table 4 (for case I), Table 5 (for case II) and Table 6 (case III), respectively. The results obtained from the analysis are summarized in Table 7.

**Table 4: The parameters values used for models (1) and (2)**

| Parameter | Nominal value | Reference |
|---|---|---|
| $\Lambda$ | 10 | Assumed |
| $\mu$ | 0.010 | Assumed |
| $\gamma$ | 0.60 | Assumed |
| $\mu_h$ | 0.02 | Assumed |
| $\delta$ | 0.20 | Assumed |
| $\alpha$ | 0.0023 | Assumed |

**Table 5: The parameters values of the diphtheria-vaccine models (3) and (4)**

| Parameter | Value | Reference |
|---|---|---|
| $\beta$ | 18.5 | [4] |
| $a$ | 0.55 | [20] |
| $\delta$ | 0.70 | [20] |
| $\phi$ | 0.0406 | [21] |
| $r$ | 0.0101 | [22] |
| $\sigma$ | 6 | [23] |
| $\mu$ | 0.0011 | [24] |
| $\alpha$ | 0.05 | [23] |
| $\varepsilon$ | 0.0083 | [23] |
| $\theta$ | 0.6667 | [23] |
| $\gamma$ | 2.1429 | [23] |
| $\tau$ | 2.1429 | [23] |
| $K$ | 10,000 | [4] |

**Table 6: The parameters values of models (5) & (9)**

| Parameter | Nominal value ($year^{-1}$) | Reference |
|---|---|---|
| $\Lambda_h$ | 0.029 | [25] |
| $v$ | 0.85 | [26] |
| $\omega$ | 0.60 | Assumed |
| $\mu_h$ | 0.02 | [25] |
| $\delta_h$ | 0.1 | [27] |
| $\theta$ | 0.20 | [25] |
| $\rho$ | 0.6 | Assumed |
| $\phi$ | 0.3 | Assumed |
| $\sigma$ | 0.4 | Assumed |
| $\tau$ | 0.6 | Assumed |
| $\eta$ | 0.75 | Assumed |
| $\varepsilon$ | 0.80 | Assumed |
| $\psi$ | 0.70 | Assumed |
| $\beta_h$ | 0.000063 | [28] |
| $(\beta_a, \beta_r)$ | (0.000252,0.0027) | [28] |
| $\Lambda_a$ | 2 | [28] |
| $\mu_a$ | 1.5 | [28] |
| $\varphi$ | 0.3 | [29] |
| $\gamma_a$ | 0.6 | [28] |
| $\delta_a$ | 0.4 | [28] |

**Table 7: The impact of constant and logistic recruitment rates for models (1), (2), (3), (4), (5) and (9)**

| S/No | Constant recruitment rate (a) | Logistic recruitment rate (b) |
|---|---|---|
| Case I | 2.8395 | 0.1420 |
| Case II | 1.3826 | 1.3826 |
| Case III | 0.00001624 | 0.00002081 |

We observed from Table 7 that reproduction number from the model with constant recruitment rate is higher than the reproduction number from the model with logistic recruitment rate. Note that the models in (1) and (2) are formulated using mass action which is usually used for small population. For case II, the diphtheria modelsin (3) and (4) are formulated using standard incidence and it was observed that the reproduction number for both models with constant and logistic recruitment rates are the same. Finally, in case III, the models in (5) and (9) are zoonotic in nature and formulated using standard incidence functions. We observed here that the human reproduction number from the model with constant recruitment rate is higher than the human reproduction number from the model with logistic recruitment rate.

## Conclusion

In this paper, the role of constant and logistic recruitment rates in three mathematical models of epidemiology is studied. In each of the models, a scenario where constant and logistic recruitment rates are incorporated in order to see their effect on the reproduction number. It was observed in case I that, the reproduction number of the model with logistic recruitment rate is less than the reproduction number from the model with constant recruitment rate. Further, in case II, the reproduction number from the model with both constant and logistic recruitment rates are the same. Finally, in case III, it was observed that the human reproduction number from the model with logistic recruitment rate is higher than the human reproduction number from the model with constant recruitment rate.

So far, three mathematical models have been explored to incorporate constant and logistics recruitment rates into the susceptible class. The essence is to ascertain which recruitment rate will lower the value of the reproduction number better. From the results shown on Table 4, a reasonable conclusion cannot be drawn using the three epidemiological models in this paper. In view of this, further studies should focus on the inclusion of constant and logistics recruitment rates to more mathematical models so as to draw a better conclusion.

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