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# Influence of Quiescence on the Final Size Distribution of SIQR Model

**Mas'ud Ahmed[a], Usman Sanusi[a*] and Usman Aminu[a]**

*[a]Department of Mathematics, Umaru Musa Yar'adua University, P.M.B 2218, Dutsin-ma Road Katsina, Katsina State, Nigeria.*

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**ABSTRACT**

Mathematical modelling is used to understand the spread of infectious diseases such as malaria, covid-19, HIV etc. in this research, we consider Selke construction which is a probabilistic approach used to study the final size distribution of an epidemic. We added quiescence phase to the standard susceptible – infected – recovered (SIR) model and build susceptible – infected – quiescence – recovered (SIQR) model to examine the effect of quiescence on the final size distribution of an epidemic. We find out analytically and by using simulation that the quiescence phase does not affect the basic reproduction number $R_0$ as well as the final size distribution of the epidemic. However, it does affect the timing, the peak and the duration of the epidemic. We use next generation matrix in computing the basic reproduction number $R_0$. We also perform sensitivity analysis on the parameters of the models using MATLABR2019a. We find that when the rate of entering quiescence stage ($\sigma$) increases while the rate of exiting the quiescence ($\mu$) remains constant, then the peak of the disease curve decreases and the time until the epidemic ends increases. We also find that when keeping the rate of entering quiescence stage ($\sigma$) fixed and increase the rate of exiting the quiescence state ($\mu$), then the peak of the disease curve increases while the time until the epidemic ends decreases.

## 1. Introduction

Mathematics is commonly be used as a tool to model, predict and understand the spread of infectious diseases such as HIV, tuberculosis, measles, malaria, coronavirus etc. However, many diseases have quiescent/dormant stage with malaria as an example (Sanusi *et al*, 2022).

The SIR model, introduced by Kermack and Mckendrick (1927), divides the population in to three compartments: susceptible (S), infectious (I) and recovered (R). The model uses differential equations to describe the flow of individuals between these compartments based on infection and recovery rates (Anderson & May, 1991; Brauer et al., 2008). Despite its simplicity, the SIR model has been instrumental in understanding the basic mechanisms of infectious disease spread (Keeling & Rohani, 2008). Various extensions to the SIR model have been proposed to incorporate additional complexities in real-world epidemics (Lin & Levin, 1998)

Selke construction, introduced by (Selke, 1982) provides a probabilistic approach to modelling epidemics. Unlike deterministic models that describe the time evolution of an epidemic, the Selke construction focuses

* Corresponding author. Tel.: +2347034253094
E-mail address: usman.sanusi@umyu.edu.ng (Usman Sanusi)
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on the final outcome by considering the cumulative infection pressure and individual susceptibilities.

During quiescence, parasites significantly reduce their metabolic activities, including decreased DNA replication, transcription, translation and overall cellular respiration. For instance, the protozoan parasite plasmodium falciparum responsible for malaria, enter a quiescent state known as hypnozoite in liver cells, allowing it to evade host immune responses and remain undetected for extended periods (Culleton & Carter, 2012).

Quiescence refers to a period during which a parasite within an individual is inactive or in a dormant state. This significantly affects disease dynamics by delaying the onset of the epidemics and therefore alters the infectious period in the disease progression (Sanusi & Abdullahi, 2023).

## 2. Model Formulation

To incorporate quiescence, we introduce an additional compartment, Q into the standard SIR model.

The SIQR model divides the population into four compartments:

- Susceptible (S): Individuals who can contact the disease
- Infectious (I): Individuals who have contracted the disease and can transmit it to others.
- Quiescent (Q): Individuals who are in quiescence state.
- Recovered (R): Individuals who have recovered from the disease and are immune

The movement of an individual from one compartment to another compartment is governed by the following sets of ordinary differential equations (ODEs).

$$\frac{dS}{dt} = \frac{-\beta SI}{N} \tag{1}$$

$$\frac{dI}{dt} = \frac{\beta SI}{N} - \delta I - \sigma I + \mu Q \tag{2}$$

$$\frac{dQ}{dt} = \sigma I - \mu Q \tag{3}$$

$$\frac{dR}{dt} = \delta I \tag{4}$$

where:

β is the transmission rate.

$\delta$ is the recovery rate.

$\sigma$ is the rate at which infectious individuals enter the quiescent state.

$\mu$ is the rate at which individuals exist the quiescence state.

Where the total population size $N = S(t) + I(t) + Q(t) + R(t)$ is kept constant. The disease spreads through direct contact between susceptible and infectious individuals and recovered individuals gain permanent immunity.

**2.1 Basic Reproduction Number**

An important parameter in disease modeling, the Basic Reproduction number, denoted by $R_0$.The basic reproduction number is the number of secondary infections produced by the primary infection into the total susceptible population. If $R_0 > 1$ then the disease invades. It increases to reach its maximum and then decreases to zero. If $R_0 < 1$ the disease dies out. It decreases to zero.

**2.2 Basic Reproduction Number of SIQR Model**

To obtain the basic reproduction of SIQR model we use next generation matrix. The basic reproduction number, $R_0$ is given by

$$R_0 = e(FV^{-1}) \tag{5}$$

Thus, $R_0$ is equal to the dominant eigenvalues of $FV^{-1}$

F = F is the terms contain only secondary infection and V= $V$ contains other terms which do not include the secondary infection. From (2) and (3) are the disease class.

$$\frac{d}{dt}\begin{pmatrix} I \\ Q \end{pmatrix} = \begin{pmatrix} \frac{\beta SI}{N} \\ 0 \end{pmatrix} - \begin{pmatrix} \delta I + \sigma I - \mu Q \\ -\sigma I + \mu Q \end{pmatrix} \tag{6}$$

$$\mathrm{F} = \begin{pmatrix} \frac{\beta SI}{N} \\ 0 \end{pmatrix} \tag{7}$$

$$V = \begin{pmatrix} \delta I + \sigma I - \mu Q \\ -\sigma I + \mu Q \end{pmatrix} \quad (8)$$

$$f(I,Q) = \frac{\beta SI}{N} \; and \; g(I,Q) = 0 \quad (9)$$

But

$$F = \begin{pmatrix} \frac{\partial f}{\partial I} & \frac{\partial f}{\partial Q} \\ \frac{\partial g}{\partial I} & \frac{\partial g}{\partial Q} \end{pmatrix} \quad (10)$$

From (9), we have

$$F = \begin{pmatrix} \frac{\beta S}{N} & 0 \\ 0 & 0 \end{pmatrix} \quad (11)$$

At disease free equilibrium $(S^*, I^*) = (1,0)$

$$F = \begin{pmatrix} \frac{\beta}{N} & 0 \\ 0 & 0 \end{pmatrix} \quad (12)$$

$$h(I,Q) = \delta I + \sigma I - \mu Q \; and \; k(I,Q) = -\sigma I + \mu Q \quad (13)$$

But

$$V = \begin{pmatrix} \frac{\partial h}{\partial I} & \frac{\partial h}{\partial Q} \\ \frac{\partial k}{\partial I} & \frac{\partial k}{\partial Q} \end{pmatrix} \quad (14)$$

From (13) we get

$$V = \begin{pmatrix} \delta + \sigma & -\mu \\ -\sigma & \mu \end{pmatrix} \quad (15)$$

$$|V| = \mu\delta + \mu\sigma - \mu\sigma$$

$$|V| = \mu\delta \quad (16)$$

$$V^{-1} = \frac{1}{\mu\delta}\begin{pmatrix}\mu & \mu \\ \sigma & \delta+\sigma\end{pmatrix}$$

$$V^{-1} = \begin{pmatrix}\frac{1}{\delta} & \frac{1}{\delta} \\ \frac{\sigma}{\mu\delta} & \frac{\delta+\sigma}{\mu\delta}\end{pmatrix} \tag{17}$$

$$FV^{-1} = \begin{pmatrix}\frac{\beta}{N} & 0 \\ 0 & 0\end{pmatrix}\begin{pmatrix}\frac{1}{\delta} & \frac{1}{\delta} \\ \frac{\sigma}{\mu\delta} & \frac{\delta+\sigma}{\mu\delta}\end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix}\frac{\beta}{N\delta} & \frac{\beta}{N\delta} \\ 0 & 0\end{pmatrix} \tag{18}$$

Now the eigenvalues of $FV^{-1}$

$$|FV^{-1} - \alpha I| = 0$$

$$\left|\begin{pmatrix}\frac{\beta}{N\delta} & \frac{\beta}{N\delta} \\ 0 & 0\end{pmatrix} - \begin{pmatrix}\alpha & 0 \\ 0 & \alpha\end{pmatrix}\right| = 0$$

$$\left|\begin{pmatrix}\frac{\beta}{N\delta} - \alpha & \frac{\beta}{N\delta} \\ 0 & -\alpha\end{pmatrix}\right| = 0$$

$$\left(\frac{\beta}{N\delta} - \alpha\right)(-\alpha) = 0$$

$$\alpha_1 = 0 \; and \; \alpha_2 = \frac{\beta}{N\delta}.$$

Therefore $\alpha_2 = \frac{\beta}{N\delta}$ is the dominant eigenvalue which is basic reproduction number of SIQR model.

Hence,

$$R_0 = \frac{\beta}{N\delta} \tag{19}$$

Equation (19) is the basic reproduction number of SIQR model which is identical to that of SIR model.

### 2.3 The Final Size of SIQR Model

To obtain the final size distribution of SIQR model we add equation (1), (2) and (3) we have

$$\frac{dS}{dt}+\frac{dI}{dt}+\frac{dQ}{dt}=\frac{-\beta SI}{N}+\frac{\beta SI}{N}-\delta I-\sigma I+\mu Q+\sigma I-\mu Q$$

$$\frac{d}{dt}[S+I+Q]=-\delta I$$

$$d[S+I+Q]=-\delta I dt$$

$$d[S(t)+I(I)+Q(t)]=-\delta I(t)dt \tag{20}$$

Integrating the terms on both sides from 0 to ∞ of (20) we have

$$\lim_{m\to\infty}\int_0^m d[S(t)+I(I)+Q(t)]=\lim_{m\to\infty}\int_0^m -\delta I(t)dt$$

$$\lim_{m\to\infty}[S(t)+I(I)+Q(t)]_0^m=\lim_{m\to\infty}\int_0^m -\delta I(t)dt$$

$$\lim_{m\to\infty}S(m)+\lim_{m\to\infty}I(m)+\lim_{m\to\infty}Q(m)-\lim_{m\to\infty}S(0)-\lim_{m\to\infty}I(0)$$
$$-\lim_{m\to\infty}Q(0)=\lim_{m\to\infty}\int_0^m -\delta I(t)dt \tag{21}$$

But,

$S(0), I(0)\ and\ Q(0)$ are constants the limit of a constant is constant. At end of the epidemic

$$\lim_{m\to\infty}S(m)=S_\infty \tag{22}$$

$$\lim_{m\to\infty}I(m)=0 \tag{23}$$

$$\lim_{m\to\infty}Q(m)=0 \tag{24}$$

$$S_\infty+0+0-S_0-I_0-Q_0=-\delta\lim_{m\to\infty}\int_0^m I(t)dt$$

$$S_\infty-(S_0+I_0+Q_0)=-\delta\lim_{m\to\infty}\int_0^m I(t)dt \tag{25}$$

At start of the epidemic

$$S(t) + I(t) + Q(t) + R(t) = N$$

At $t = 0$,

$$S(0) + I(0) + Q(0) + R(0) = N$$

$$S_0 + I_0 + Q_0 + R_0 = N \tag{26}$$

$$S_\infty - N = -\delta \lim_{m\to\infty} \int_0^m I(t)dt$$

$$\frac{N - S_\infty}{\delta} = \lim_{m\to\infty} \int_0^m I(t)dt \tag{27}$$

From (1)

$$\frac{dS}{dt} = \frac{-\beta SI}{N}$$

Using separating variable, we have

$$\frac{dS}{S} = -\frac{\beta I}{N}dt \tag{28}$$

Integrate from 0 to ∞ (28) we have

$$\lim_{m\to\infty} \int_0^m \frac{dS}{S} = \frac{-\beta}{N} \lim_{m\to\infty} \int_0^m I(t)dt$$

$$\lim_{m\to\infty} |[\ln S(t)]|_0^m = \frac{-\beta}{N} \lim_{m\to\infty} \int_0^m I(t)dt$$

$$\ln S(m) - \ln S(0) = -\frac{-\beta}{N} \lim_{m\to\infty} \int_0^m I(t)dt$$

$$\frac{N(\ln S_\infty - \ln S_0)}{-\beta} = \lim_{m\to\infty} \int_0^m I(t)dt$$

$$\frac{N(\ln S_0 - \ln S_\infty)}{\beta} = \lim_{m\to\infty} \int_0^m I(t)dt \tag{29}$$

Comparing equation (27) and (29) we have

$$\frac{N - S_\infty}{\delta} = \frac{\mathrm{N}(\ln S_0 - \ln S_\infty)}{\beta}$$

$$\frac{\beta}{N\delta}(N - S_\infty) = \ln\left(\frac{S_0}{S_\infty}\right) \tag{30}$$

Substitute (19) in to (30) we have

$$R_0(N - S_\infty) = \ln\left(\frac{S_0}{S_\infty}\right) \tag{31}$$

Equation (31) is the final size of SIQR model, which is the same as the final size of SIR model.

**2.4 The Selke Construction**

We will use Selke construction in this research, which provides a probabilistic framework for simulating stochastic epidemics. It is particularly useful for understanding the randomness inherent in the transmission and progression of infectious diseases. Selke's approach involves assigning each individual a threshold, representing their resistance to infection. When the cumulative force of infection exceeds an individual's threshold, they become infected. This method captures the stochastic nature of disease spread and can be integrated with the SIR model to account for quiescence.

***The Procedure***

We have population of $N$ individuals. At initial time $t = 0$, there are $m$ infected persons $n = N - m$ susceptible no recovered. We label the infected persons by $-(m-1), -(m-2), \dots, -1, 0$ and the susceptible by $1, 2, \dots, n$. Next, we introduce time intervals for the infectious period of the person $i\epsilon\{-(m-1), -(m-2), \dots, -1, 0\}$, where $I_i$ is chosen independently and identically distributed we are relatively free in the choice of the distribution for $I_i$, where $Q_i \sim Exp(\delta)$ to be in line with the infectious period of the standard SIR model.

Last we introduce for all susceptible person $i.i.d.$ value random variable$Q_i$, where$Q_i \sim Exp(1)$. The $Q_i$ could be interpreted as susceptibility of the $i^{th}$ individuals.

We now have all ingredients together to define the dynamics. The initial infected person $i$ stays infected from time $t = 0$ to the time $t = I_i,\ i = -(m-1), -(m-2), \dots, -1, 0$. Next $Y_u$ denote the number of

infected at time $t$ we introduce a kind of cumulative infection pressure

$$A_t = \frac{\beta}{N}\int_0^t Y_u\, du \tag{32}$$

The individual labelled $i \in \{0, 1, 2, \ldots, n\}$ becomes infectious at time $t$ at which $A_t$ exceed $Q_i$ and stays infected for time $I_i$. The epidemic dies out if $A_t$ become constant $A_\infty$ (no infected person any more) and all remaining susceptible have a $Q_i > A_t$

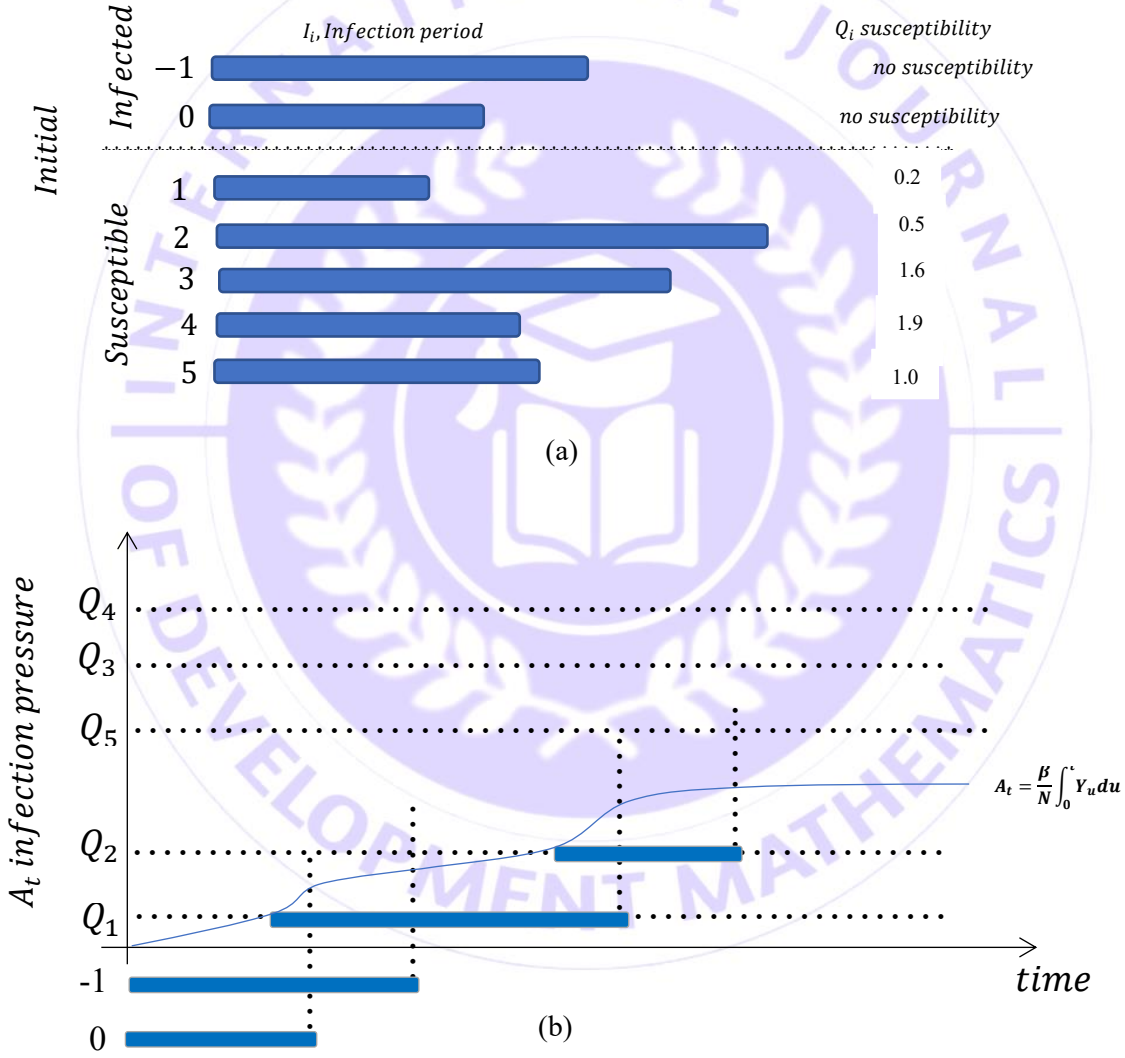



Figure 1: Illustration of Selke Construction

In Figure 1(a), the infection pressure $A_t$ rises proportional to the person and if it reaches the susceptibility $Q_i$ of person $i$, this individual become infected for the time span $I_i$. The epidemic ends if $A_t$ cannot reach any susceptibility levels.

### 2.5 Infectious Duration in SIQR Model

Transition between the states

i. Individuals start in the infectious ($I$) class

ii. From $I$, they can either

Recovered $(I \to R)$ with rate $\delta$ or go to Quiescent $(I \to Q)$ with rate $\sigma$. Then from $Q$ they return to $I$ at rate $\mu$. So the circle looks like this

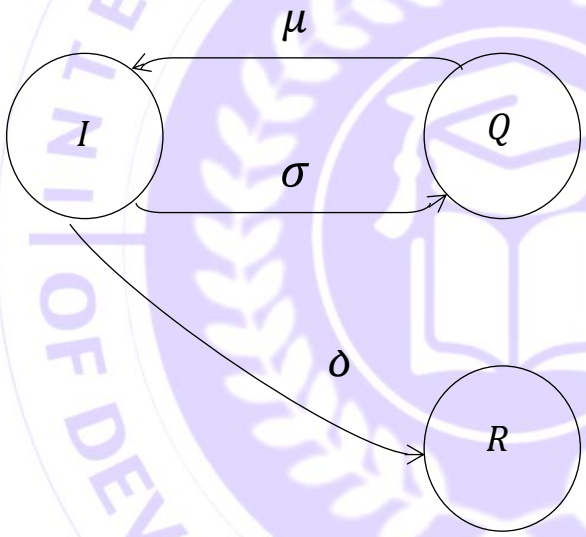



Figure 2: *Shows the transition of the infected person*

They may bounce between $I$ and $Q$ multiple times before finally recovering. To find the expected total time spent before reaching $R$ starting from $I$.

i. $\tau_I = \frac{1}{\delta+\sigma}$ expected time in $I$ per visit (exponential waiting time).

ii. $\tau_Q = \frac{1}{\mu}$ expected time in $Q$ per visit.

iii. $\tau$ is the expected total time starting in $I$ until recovery.

Using recursive expectation. After the first stay in $I$ (which last for $\tau_I$ time), two things happen

Case 1: Recovery directly

i. Probability: $\frac{\delta}{\delta+\sigma}$.

ii. Total time spent in $\tau_I$.

Case 2: Move to $Q$, then return to $I$

i. Probability: $\frac{\sigma}{\delta+\sigma}$.

ii. Time spent: $\tau_I + \tau_Q + we\ are\ back\ to\ I\ compartment\ as\ before\ (so + \tau)$

So the expected total time $\tau$

$$\tau = \tau_I + \left(\frac{\sigma}{\delta+\sigma}\right)(\tau_Q + \tau) \tag{33}$$

$$\tau = \tau_I + \left(\frac{\sigma}{\delta+\sigma}\right)\tau_Q + \left(\frac{\sigma}{\delta+\sigma}\right)\tau$$

$$\tau - \left(\frac{\sigma}{\delta+\sigma}\right)\tau = \tau_I + \left(\frac{\sigma}{\delta+\sigma}\right)\tau_Q$$

$$\tau\left(1 - \left(\frac{\sigma}{\delta+\sigma}\right)\right) = \tau_I + \left(\frac{\sigma}{\delta+\sigma}\right)\tau_Q$$

$$\tau\left(\frac{\delta}{\delta+\sigma}\right) = \tau_I + \left(\frac{\sigma}{\delta+\sigma}\right)\tau_Q \tag{34}$$

Multiply both sides of (34) by $\frac{\delta+\sigma}{\delta}$ we have

$$\tau = \frac{\tau_I}{\delta/(\delta+\sigma)} + \frac{\sigma}{\delta}\tau_Q \tag{35}$$

Since $\tau_I = \frac{1}{\delta+\sigma}$ and $\tau_Q = \frac{1}{\mu}$ equation (35) become

$$\tau = \frac{1}{\delta} + \frac{\sigma}{\delta}.\frac{1}{\mu}$$

$$\tau = \frac{1}{\delta}\left(1 + \frac{\sigma}{\mu}\right) \tag{36}$$

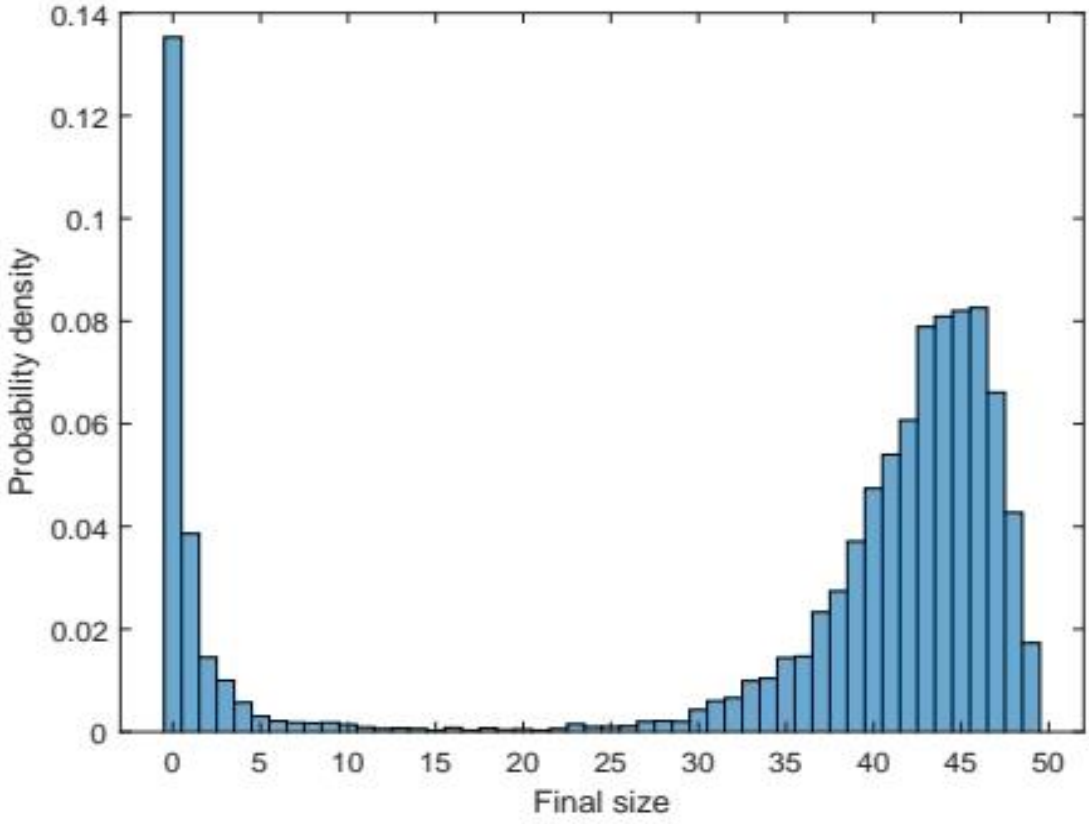
Please observe that in equation (36) the expected infectious period is longer that of the model without

quiescence i.e. SIR model which is equal to $\frac{1}{\delta}$. This tells us that quiescence state increases the time until the epidemic ends.

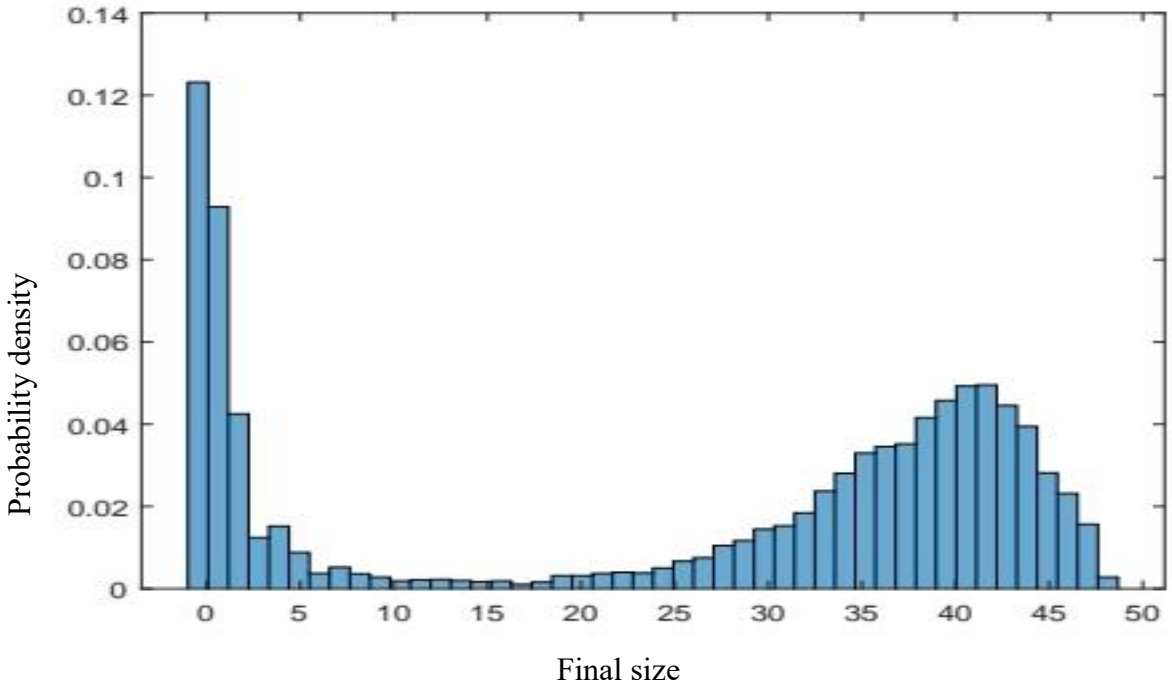
## 3. Result

In this section we will display results that we obtained from stochastic simulation using Selke construction.

### 3.1 Stochastic Simulation of the Models




(3a)




(3b)

Figure 3: Final size distribution of SIR model from 10000 stochastic simulations

Figure 3a show the final size distribution of SIR model from 10000 stochastic simulations with the parameters $\delta = 1, \beta = 2\ such\ that\ R_0 = 2\ and\ the\ \ population\ size\ N = 50$. Figure 3b show the final size distribution of SIQR model from 10000 stochastic simulations with the parameters $\delta = 1,\ \beta = 2,\ \sigma = 0.4\ and\ \mu = 0.2,\ \ such\ that\ R_0 = 2\ and\ \ N = 50$.

### 3.2 Numerical Solution of SIQR Model

We use MATLAB to obtain a numerical Solutions of SIR and SIQR models

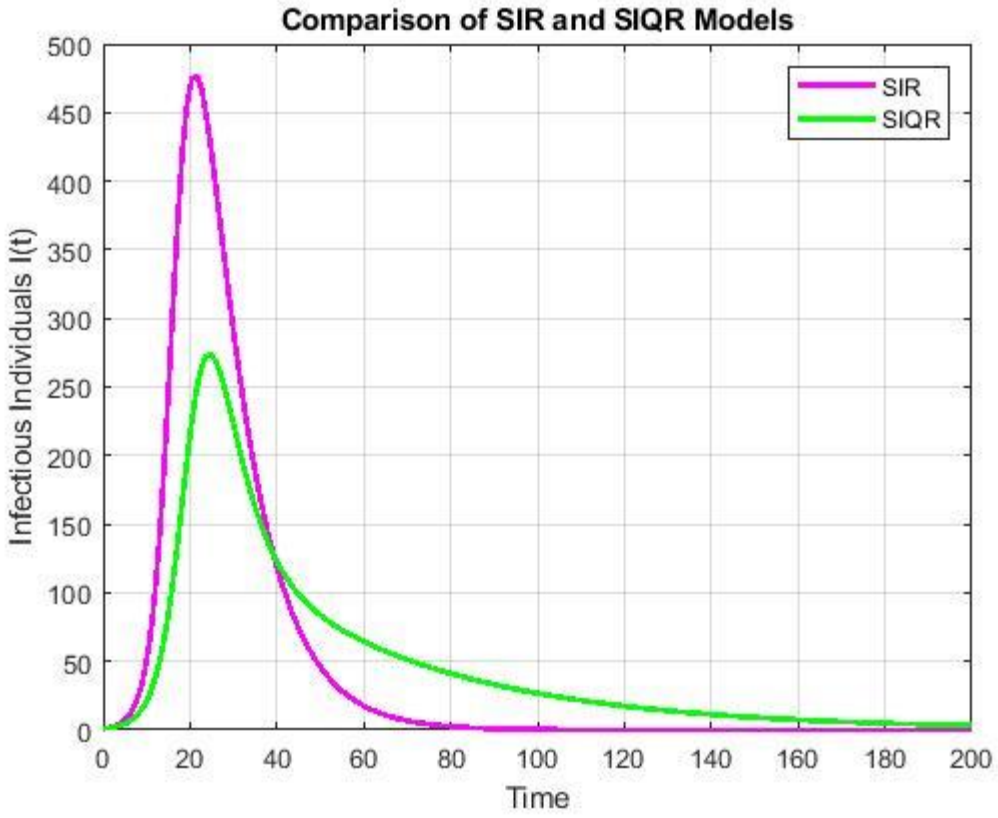



Figure 4

Figure 4: Numerical solution of SIR model with the parameters $\beta = 0.5$ $\delta = 0.1, \sigma = 0, \mu = 0$, $Q_0 = R_0 = 0$, $I_0 = 1$, the initial population size $S_0 = 999$, and total population $N = 1000$ one gets the solution of SIR model while the numerical solution of SIQR model with the parameters $\beta = 0.5$ $\delta = 0.1, \sigma = 0.1$, $\mu = 0.05$, $Q_0 = R_0 = 0$, $I_0 = 1$, the initial population size $S_0 = 999$, and total population $N = 1000$. Please observe due to the additional phase of quiescence, the time until the epidemic ends is longer compared with the model that does not include the quiescence phase (SIR). In addition, noticed that the timing of the epidemic is affected by the quiescence phase, that is, there was a delay.

### 3.3 Sensitivity of sigma and mu SIQR model

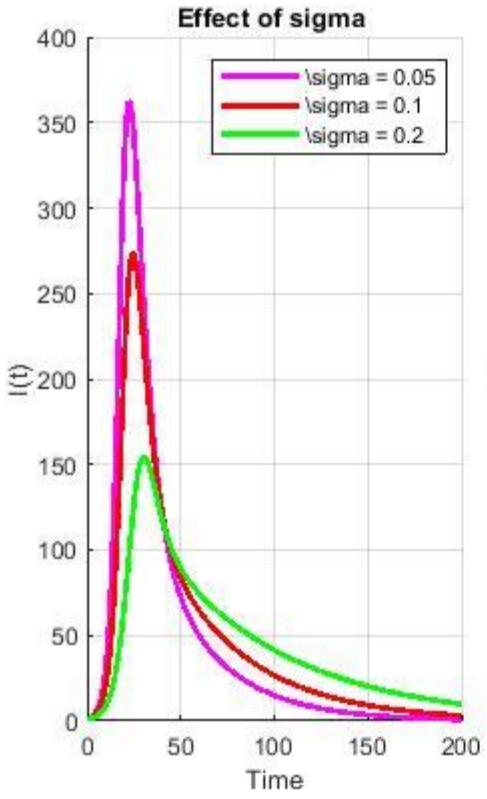



Figure 5 (a)

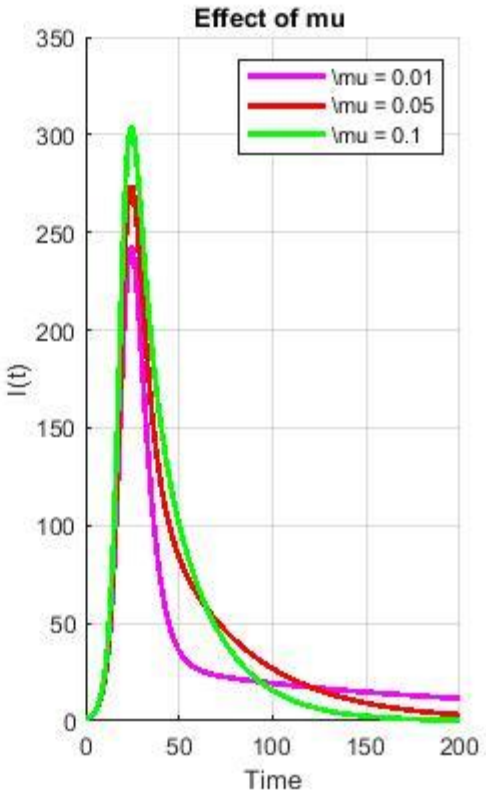



Figure 5 (b)

Figure 5: The numerical solutions of SIQR model

In Figure 5 (a) The numerical solutions of SIQR model with the parameters $\beta = 0.5$ $\delta = 0.1$, where $\mu$ is fix i.e. $\mu = 0.05$, and $\sigma$ varies i.e. $\sigma_1 = 0.05$, $\sigma_2 = 0.1$ $and$ $\sigma_3 = 0.2$ $Q_0 = R_0 = 0$, $I_0 = 1$, the initial population size $S_0 = 999$, and total population $N = 1000$. (b) The numerical solutions of SIQR model with the parameters $\beta = 0.5$ $\delta = 0.1$ while $\sigma$ is kept fixed i.e. $\sigma = 0.1$, and $\mu$ varies i.e. $\mu_1 = 0.01$, $\mu_2 = 0.05$ $and$ $\mu_3 = 0.1$ $Q_0 = R_0 = 0$, $I_0 = 1$, the initial population size $S_0 = 999$, and total population $N = 1000$. Please observe in Figure 5 (a) if $\sigma$ increases, we have shorter peak and longer tail while in (b) if $\mu$ increases, we have longer peak and shorter tail.

## 4. Discussion

We added quiescence to the standard SIR model, the Basic Reproduction number of the SIQR model was calculated and it turned out to be the same to that of SIR model. We also calculated the final size of the epidemic using SIQR model found that the deterministic models (SIR and SIQR) have the same final sizes. We also performed sensitivity analysis on the parameters of the SIQR model in a bid to understand which parameter has the significant effect to the dynamics of SIQR model. We found that when the rate of entering quiescence stage (σ) increase while keeping the rate of exiting the quiescence ($\mu$) constant, then the peak of the disease curve decreases and the time until the epidemic ends increases. We also found that when keeping the rate of entering quiescence stage (σ) fixed and varies the rate of exiting the quiescence state ($\mu$), then the peak of the disease curve increases while the time until the epidemic ends decreases.

The epidemic breakout is increased by the presence of quiescence within the dynamics of an infectious disease. This conclusion emphasises the significance of quiescent phase in disease modelling and emphasises their potential function as important triggers of widespread outbreaks.

## 5. Conclusion

The Selke construction provides a powerful framework for incorporating stochastic elements into the SIR model, allowing for a more realistic representation of disease spread. By extending the SIR model to include quiescence, we gain deeper insights into the dynamics of infectious diseases and the potential impacts of public health interventions. This extended model underscores the importance of considering quiescence state in epidemiological modeling and offers valuable guidance for managing real-world epidemics.

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