Stability and Sensitivity Analysis of SS\textsubscript{Q}VEQIHFR Epidemic Model

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Abstract

Objectives: While studies had established standard epidemic models, variants of those standards, which define unique and characteristic behavior of some of disease amidst interventions and population dynamics, are continually developed to represent epidemic dynamics in different ways. Methods: In this study, a compartmental susceptible-susceptible quarantine-vaccinated-exposed-quarantined-infectious-hospitalized-funeral-recovered (SS\textsubscript{Q}VEQIHFR) epidemic model is formulated and analyzed. Model stability was analyzed and found to be both stable for disease-free and endemic equilibrium. Findings: In order to determine its threshold (basic reproduction number), the next generation matrix approach was applied, and was found to represented average secondary transmissions of cases in the community, hospital and at funerals during the entire period of the epidemic. A numerical simulation was used to validate the disease-free and endemic equilibrium stability of the model. Applications: In order to determine influential parameters, a forward sensitivity index analysis was carried out on the model threshold and endemic points. This model has more classes which can be used to investigate infectious diseases outbreak with such characteristic dynamics. Preventive or control measures that averts transmission in the community, hospital and at funeral will stall the growth of epidemic in the population.

Keywords: Epidemic Model, Reproduction Number, Sensitivity Analysis and Stability Model, Stability Analysis

1. Introduction

Epidemic models provide information that can help assess, predict and proffer optimal intervention control measures for stopping outbreaks. Different methods of modeling the spread of diseases have been in practice for different disease characteristics. Stable epidemic model and can be fitted to disease incidence data in order to estimate parameters. If models are stable, their sensitivity to parameter changes will gauge intervention or control implementation strategies in order to achieve expected efficacy of each control measure. Infectious disease model becomes high-dimensional if it includes compartments like latent stage, vector-borne, pre-exposed immunity after recovery, infectious cadavers, asymptomatic infectious, disease-carrier individuals, transmission incidences, interventions and control. However, adding control compartments like vaccine, quarantine, isolation and safe burial, into disease progression dynamics refines the biological process of the disease in the population. Several studies\textsuperscript{1-3} had analyzed infectious disease models with latent asymptomatic compartment in nonlinear susceptible-exposed-infectious-removed (SEIR) epidemic model.

These SEIR variants assessed the impact of newer compartments of control or intervention, disease states or demography on the disease dynamics. In\textsuperscript{4} and\textsuperscript{5} the stability of SS\textsubscript{Q}EIR for SARS epidemic was analyzed. While\textsuperscript{6} analyzed SEQIHR to obtain threshold and equilibria of the model. The threshold of SEIRS was determined for the inclusion of quarantine of susceptible and isolation in the model\textsuperscript{7} subject to quarantine (of asymptomatic cases.

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Similar\textsuperscript{8,10} investigated the SEIIHFR stability in predicting Ebola growth. An SEIIFDR model stability determined the threshold of controlling an outbreak was investigated by\textsuperscript{11}. Considering the vaccine effect to disease transmission in SVEEIJDR model\textsuperscript{12} analyzed the equilibria and stability of this variant, establishing the sufficiency of vaccination to retarding disease spread.

Over the years, intervention and control measures are implemented to effectively fight the spread of emerging and re-emerging diseases. Vaccination is implemented to enhance immunity of the susceptible against the widespread infectious pathogen. The incorporation of early quarantine, susceptible-quarantine (SQ) of those susceptible individuals that are quarantine but later return to susceptible class was implemented in\textsuperscript{13}, to study its impact on controlling of SARS. Later in\textsuperscript{5} it was found to have some impact on disease growth. Similarly\textsuperscript{14}, assessed the risk and benefits of community care centers to the control of Ebola in Sierra Leone. In studies\textsuperscript{15,16,17} it was noted that control measures, like contact tracing, quarantine and isolation, are commonly implemented to control outbreaks. These intervention and control measures have been extensively implemented in epidemics like Measles, cholera, SARS Ebola, MERS, Zika virus etc. Safe burial controls Ebola virus disease and similar infectious diseases that cadavers can transmit virus. The impact of this measure to widespread of the disease had been widely investigated\textsuperscript{18,19}.

Jacobian and next generation matrix approaches\textsuperscript{20} were commonly used to determine thresholds and disease-free or endemic equilibrium, while Lyapunov functions and Routh–Hurwitz\textsuperscript{21} are applied to determine the global stability of the epidemic model. High-dimensioned disease models posed greater challenges in constructing Lyapunov functions hence difficult to analyze the equilibria and stability, however\textsuperscript{22}, suggested matrix-theoretic and graph-theoretic methods of constructing Lyapunov functions that simplify the proof of stability.

The discussion in this paper is divided into four sections. The first section introduced and reviewed the emergence SEIR-variant, the SS\textsubscript{v}VEQIHFR epidemic model. Section two presented the formulation process, including parameter definition, disease dynamics diagram with its system of differential equations. In section three, the analysis of equilibria, the threshold (reproduction number) and stability of the model are carried out using standard procedure and theorems. In section four a numerical simulation to validate the model reality is performed. The study of influential transmission or control parameters, parameter to incidences and prevalence of the disease in the population is carried out using sensitivity analysis of the reproduction number and endemic equilibrium points.

2. Model Formulation

The model considered transmission dynamics of an infectious disease in the presence of susceptible-quarantine, exposed and quarantine individual, vaccine class with chances of immunity waning, isolation of infectious individuals and safe burial of infectious cadavers. The population N is split into subpopulations of susceptible (S), susceptible-quarantine (SQ), vaccine (V), exposed (infected asymptomatic) (E), infected infectious (I), quarantined (Q), isolated infectious (H), dead safely buried (F), recovered and immune or removed class (R) Table 1.

| Parameter | Definition |
|-----------|------------|
| N         | total population size |
| μ         | recruitment rate of susceptible humans |
| α         | rate natural mortality |
| λ         | force of infection |
| β\textsubscript{i} | contact rate in the community |
| β\textsubscript{h} | contact rate in the hospital |
| β\textsubscript{f} | contact rate during traditional unsafe burial |
| b         | fraction of contacts infected that move to quarantine class |
| k         | rate of restriction |
| φ         | safe burial efficiency |
| ρ         | isolation efficiency |
| ϕ         | rate of individuals returning to susceptible from restriction |
The model incorporated interventions or control measures in disease transmission and transition structure. Technically feasible measures, public enlightenment, vaccination, quarantine, isolation and safe burial are defined and incorporated into the model. The flow diagram of the disease transmission is presented Figure 1.

This model is can be used to investigate infectious diseases, the disease is transmitted through contact with body fluids of human or cadavers of victims and vaccines for the disease are readily available alongside intervention measures deployed to effectively control the disease. The corresponding system of ordinary differential equations for the model is,

\[
\begin{align*}
\frac{dS}{dt} &= \Lambda - (1 - \gamma) \beta S - \gamma b S - (1 - b) \lambda S - \xi S - \mu S + \phi S_0 \\
\frac{dS_0}{dt} &= (1 - b) \lambda S - \phi S_0 - \mu S_0 \\
\frac{dV}{dt} &= \xi S - (1 - \eta) \lambda V - \mu V \\
\frac{dE}{dt} &= (1 - \gamma) \beta S + (1 - \eta) \lambda V - (\chi + \alpha_1) E - \mu E \\
\frac{dQ}{dt} &= \chi E + \gamma b S - \alpha_2 Q - \mu Q \\
\frac{dI}{dt} &= \alpha_1 E - (\theta \gamma_{ih} + (1 - \theta) \delta_1 \gamma_{ih} + (1 - \delta_1) \gamma_{ih}) I - \mu I \\
\frac{dH}{dt} &= \alpha_2 Q + \theta \gamma_{ih} I - (\delta_2 \gamma_{ih} + (1 - \delta_2) \gamma_{ih}) H - \mu H \\
\frac{dF}{dt} &= (1 - \theta) \delta_1 \gamma_{ih} I + \delta_2 \gamma_{ih} H - \gamma_{ih} F - \mu F \\
\frac{dR}{dt} &= (1 - \theta)(1 - \delta_1) \gamma_{ih} I + (1 - \delta_2) \gamma_{ih} H + \gamma_{ih} F
\end{align*}
\]

With the force of infection is defined as

\[
\lambda = (1 - \epsilon \omega)(1 - k) \left( \beta_1 I + \beta_2 (1 - \rho) H + \beta_3 (1 - r) F \right) N
\]

Where \( \beta \) is the transmission coefficient, \((1 - \epsilon \omega)\) is the non-impact of media and \((1 - k)\) is the impact of non-restriction on transmission, \(\rho \in [0,1]\) is the fraction of reduction in the transmission rate of isolated with \(\rho = 1, \rho = 0, \text{ and } 0 < \rho < 1\) denotes a completely effective, completely ineffective and partially effective isolation. The fraction of reduction in transmission during safe burial is \(r \in [0,1]\) with \(r = 1, r = 0, \text{ and } 0 < r < 1\) denotes completely effective, completely ineffective and partially effective safe burial.

3. Model Analysis

The model solution exists and remains unique at all times for nonnegative initial values in the region:
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\[ D = \left\{ (S, S_Q, V, E, Q, I, H, F) \in R^8 : S + S_Q \geq \frac{\Lambda}{\mu} \right\} \]

To determine the disease-free equilibrium \( E_0 \) of the model \((SS_Q, VEQIHFR)\) compartment and set the system of equation 1 to zero, and solve. The disease-free equilibrium
\[ E_0 = (S, S_Q, V, E, Q, I, H, F), \]
\[ E_0 = (S = \frac{\Lambda}{(\mu + \xi) - (1-b)\lambda}, S_Q = \frac{(1-b)\lambda\Lambda}{r((\mu + \xi) - (1-b)\lambda)}, \]
\[ V = \frac{\xi\Lambda}{(\mu + (1-\eta)\lambda)((\mu + \xi) - (1-b)\lambda)}, \]
\[ E = 0, Q = 0, I = 0, H = 0, F = 0 \]
Assuming that the population is constant and the epidemic period is so short that birth and death is negligible so that \( \frac{\Lambda}{\mu} \leq 1 \).

In order to determine the endemic equilibrium point \( E^* = (S^*, S_Q^*, V^*, E^*, Q^*, I^*, H^*, F^*) \) set the right-hand side of Equation 1 to zero and solve algebraically the equations to obtain
\[ S^* = \frac{1}{\lambda b(1-\gamma)} \left( \frac{c_i c_i I^*}{\alpha_i} - (1-\eta)\lambda V^* \right) \]
\[ S_Q^* = \frac{(1-b)}{br(1-\gamma)} \left( \frac{c_i c_i I^*}{\alpha_i} - (1-\eta)\lambda V^* \right) \]
\[ V^* = \frac{c_i c_i I^*}{\lambda \alpha_i (1-n)} \]
\[ E^* = \frac{c_i I^*}{\alpha_i} \]
\[ Q^* = \frac{c_i c_i I^*}{\alpha_i} + \frac{\gamma}{\alpha_i (1-\gamma)} \left( \frac{c_i c_i I^*}{\alpha_i} - (1-\eta)\lambda V^* \right) \]
\[ I^* = \frac{(\lambda_2 + \xi) N}{(1-\epsilon_\omega)(1-k)} \left( \frac{\lambda (1-b)}{\lambda_2 + \xi} - 1 \right) \]
\[ H^* = \frac{c_i c_i I^*}{\alpha_i (1-\gamma)} \left( \frac{\gamma}{\alpha_i (1-\gamma)} \left( \frac{c_i c_i I^*}{\alpha_i} - (1-\eta)\lambda V^* \right) + \gamma \theta I^* \right) \]
\[ F^* = \frac{\gamma f (1-\theta) \delta_i I^* + \lambda_{ij} \delta_j H^*}{\gamma_f} \]
\[ R^* = (1-\delta_i)(1-\theta)\gamma_f I^* + (1-\delta_j)\gamma_i H^* + \gamma_f F^* \]

Where
\[ c_i = \gamma_0 \theta + \gamma_i (1-\theta)\delta_i + \gamma_i (1-\theta)(1-\delta_i), c = \alpha_i \]
\[ \lambda_2 = (1-\epsilon_\omega)(1-k) \frac{(1-\rho)\beta_i H + (1-r)\beta_f F}{N}, \]
\[ \lambda = (1-\epsilon_\omega)(1-k) \frac{\beta_i I + \beta_f (1-\rho)H + (1-r)\beta_f F}{N} \]

4. Threshold Analysis

A threshold parameter, the basic reproduction number is defined as the average number of secondary infections caused by an infectious case during its entire epidemic period, is an important concept in the epidemic model analysis. This parameter gauges the spread of infectious diseases in a population. The next generation matrix approach 16,18, is used to determine the reproduction number of the model. It is notable that \( R_0 \) like threshold calculated from differential equation models are not necessarily the reproduction number; however, they share a stability threshold 19,20.

Defining the model system of differential equations as
\[ \frac{dx}{dt} = f_i(x) \]
This can be rewritten as
\[ \frac{dx}{dt} = F_i(x) - V_i(x) \]
\( F_i(x) \) is the rate of new infections entering \( i^{th} \) compartment but does not include terms which describe the transfer of infectious individuals from one infected compartment to another. \( V_i(x) \) is the transition term, in the \( i^{th} \) diseased compartment.
\[ \tilde{F} = \begin{pmatrix}
0 & 0 & \beta_i (1-\rho)\beta_f & 1 - r \beta_f & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
\end{pmatrix} \]
and
\[ \tilde{F} = \begin{pmatrix}
0 & 0 & \beta_i (1-\rho)\beta_f & 1 - r \beta_f & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
\end{pmatrix} \]
For simplification let's mark some entries in $V$

\[
\begin{align*}
V &= \begin{pmatrix}
 a & 0 & 0 & 0 & 0 \\
 -\chi & \alpha_1 & 0 & 0 & 0 \\
 -\alpha_1 & 0 & b & 0 & 0 \\
 0 & -\alpha_2 & -\gamma_w & \phi & 0 \\
 0 & 0 & -\gamma_f(1-\theta)\delta_i & -\gamma_{hf}\delta_2 & \gamma_f
\end{pmatrix},
\end{align*}
\]

The basic reproduction number is the dominant eigenvalue or spectral radius of $(FV^{-1})$ written as $R_0 = \rho(FV^{-1})$ so that

\[
R_0 = \frac{\beta_1\alpha_1}{(\chi + \alpha_i)(\gamma_v\theta + \gamma_i(1-\theta)(1-\delta_i) + \gamma_f(1-\theta)\delta_i)} + \frac{\beta_h(1-\rho)}{\gamma_{hf}\delta_2 + \gamma_h(1-\delta_2)} \left( \frac{\chi}{(\chi + \alpha_i)(\gamma_v\theta + \gamma_i(1-\theta)(1-\delta_i) + \gamma_f(1-\theta)\delta_i)} + \frac{\alpha_1\gamma_w}{\alpha_i\gamma_v(1-\theta)\delta_i} \right) + \frac{\beta_f(1-r)}{\gamma_f} \left( \frac{\gamma_{hf}\delta_2}{\gamma_{hf}\delta_2 + \gamma_h(1-\delta_2)} \left( \frac{\chi}{(\chi + \alpha_i)(\gamma_v\theta + \gamma_i(1-\theta)(1-\delta_i) + \gamma_f(1-\theta)\delta_i)} + \frac{\alpha_1\gamma_w}{(\chi + \alpha_i)(\gamma_v\theta + \gamma_i(1-\theta)(1-\delta_i) + \gamma_f(1-\theta)\delta_i)} \right) \right)
\]

From the relation, it can be deduced that $R_0 = R_{0i} + R_{0H} + R_{0F}$

where $R_{0i}$ is anumber of secondary infections that one non-quarantined and non-isolated infectious individual in the community, $R_{0H}$ the number of secondary infections that quarantined and isolated infectious individual in the hospital and $R_{0F}$ the number of secondary infections one unsafe burial of non-isolated and isolated individual, produces in an entirely susceptible population during its lifespan as infective before it's removed.

5. Stability of Equilibria

**Theorem 1:** If $R_0 < 1$ the disease-free equilibrium $E_0$ is locally asymptotically stable, and is unstable if otherwise.

In order to prove the local stability of the disease-free equilibrium, the Jacobian matrix of the system at the disease-free equilibrium has negative eigenvalues. When evaluated the Jacobian at disease free equilibrium, the characteristic polynomial equation from $|J - \lambda I| = 0$ as,

\[
\begin{pmatrix}
 -\lambda b & \phi & -\xi & 0 & 0 & 0 & 0 & 0 \\
 0 & -\phi + \lambda & -\xi + \lambda & 0 & 0 & 0 & 0 & 0 \\
 0 & 0 & -\xi - \lambda(1-\eta) & 0 & 0 & 0 & 0 & 0 \\
 0 & 0 & 0 & -\lambda(1-b) & -\phi & 0 & 0 & 0 \\
 0 & 0 & 0 & 0 & -\lambda & \xi - \gamma(1-\eta) & 0 & 0 \\
 0 & 0 & 0 & 0 & \lambda b & 0 & (1-\eta) & -\gamma f \\
 0 & 0 & 0 & 0 & \gamma^2b & 0 & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & a_1 & 0 & -1(1) \\
 0 & 0 & 0 & 0 & 0 & 0 & \gamma f(1-\theta)\delta_i & \gamma_{hf}\delta_2 - \gamma_f
\end{pmatrix}
\]

On reducing the above, to row echelon

\[
\begin{pmatrix}
 -\lambda b & \phi & -\xi & 0 & 0 & 0 & 0 & 0 \\
 0 & -\phi + \lambda & -\xi + \lambda & 0 & 0 & 0 & 0 & 0 \\
 0 & 0 & -\xi - \lambda(1-\eta) & 0 & 0 & 0 & 0 & 0 \\
 0 & 0 & 0 & -\lambda(1-b) & -\phi & 0 & 0 & 0 \\
 0 & 0 & 0 & 0 & -\lambda & \xi - \gamma(1-\eta) & 0 & 0 \\
 0 & 0 & 0 & 0 & \lambda b & 0 & (1-\eta) & -\gamma f \\
 0 & 0 & 0 & 0 & \gamma^2b & 0 & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & a_1 & 0 & -1(1) \\
 0 & 0 & 0 & 0 & 0 & 0 & \gamma f(1-\theta)\delta_i & \gamma_{hf}\delta_2 - \gamma_f
\end{pmatrix}
\]

So that the eigenvalues are found to be

\[
\lambda_1 = -\lambda b, \lambda_2 = -(r - \lambda), \lambda_3 = \xi - \lambda(1-\eta), \lambda_4 = -(D), \lambda_5 = a_1, \lambda_6 = -(E), \lambda_7 = -(K), \lambda_8 = -\gamma_f
\]

$D = \chi + \alpha_i, K = (\gamma_{hf}\delta_2 + \gamma_h(1-\delta_2))$

Since the roots of the equation are negative it can be concluded that disease-free equilibrium is locally stable

**Theorem 2:** If $R_0 < 1$, then the disease-free equilibrium point of the system is globally asymptotically stable.
To establish the global stability of the disease-free equilibrium point, the Lyapunov function is constructed as

\[
V(t) = k_1 \left( S - S^* - S^* \ln \frac{S}{S^*} \right) + k_2 \left( S_0 - S_0^* - S_0^* \ln \frac{S_0}{S_0^*} \right) + k_3 (V - V^* - V^* \ln \frac{V}{V^*})
\]

\[
+ \frac{1}{(A)} E + \frac{1}{(A)} Q + \frac{1}{(A)} I + \frac{1}{(A)} (1 + \frac{1}{(A)}) H + \frac{1}{(A)} (1 + \frac{1}{(A)}) F
\]

For \( V(t) > 0 \), taking \( k_1, k_2, k_3 > 0 \) it is obvious that

\[
V(t) = k_1 \left( S - S^* - S^* \ln \frac{S}{S^*} \right)
\]

\[
+ k_2 \left( S_0 - S_0^* - S_0^* \ln \frac{S_0}{S_0^*} \right)
\]

\[
+ k_3 \left( V - V^* - V^* \ln \frac{V}{V^*} \right) > 0
\]

Because the function \( g(x) = x - 1 - \ln x \) achieves a global minimum at \( x = 1 \) and \( g(1) = 0 \) hence \( g(x) > 0 \), for all \( x > 0 \) and \( x \uparrow 1 \), and this is for all the first terms. The remaining terms are also positive, \( V(t) \) is therefore radially unbounded.

The time derivative is

\[
\frac{dV(t)}{dt} = k_1 \left( S - S^* \right) S' + k_2 \left( S_0 - S_0^* \right) S_0' + k_3 (V - V^*) V'
\]

\[
+ \frac{1}{(A)} E' + \frac{1}{(A)} Q' + \frac{1}{(A)} I' + \frac{1}{(A)} \gamma_0 \delta_1 + \frac{1}{(A)} \gamma_0 \delta_2 + \frac{1}{(A)} \gamma_0 \delta_3
\]

Whereby \( k_i, i = 1, 2, 3 \) are positive constant to be determined.

To expand the derivative, let’s assume that \( k_1 = k_2 = k_3 = 1, S = S^* \) and \( S_0 = S_0^* \) and obtained

\[
-\left( \xi + (1 - \eta) \right) \left( V + V' \right) + (1 - \eta) S (1 - \frac{S}{S_0})
\]

\[
-\left( \eta \lambda V - E \right) + \frac{\lambda b S}{(\chi + \alpha)} + \frac{\chi V}{b S}
\]

\[
-\left( \chi \gamma_0 - \alpha \gamma_0 - \chi \gamma_0 \right) Q + \frac{B}{(A)} (R_0 - 1) I
\]

\[
-\left( \chi \gamma_0 - \alpha \gamma_0 - \chi \gamma_0 \right) H + \frac{B}{(A)} (R_0 - 1) I
\]

\[
-\left( \alpha \gamma_0 - \alpha \gamma_0 - \chi \gamma_0 \right) H + \frac{B}{(A)} (R_0 - 1) I
\]

\[
-\left( \gamma_0 + \gamma_0 \right) F
\]

Where \( B = \gamma_0 \theta + \gamma_0 (1 - \theta) \delta_3 + \gamma_0 (1 - \theta) (1 - \delta_3) \) and \( C = \gamma_0 \delta_2 + \gamma_0 (1 - \delta_2) \)

All terms have negative coefficients except the second and sixth. However, \( S_0 < S \) and \( R_0 < 1 \), therefore

\[
\frac{dV(t)}{dt} < 0.
\]

This concludes the proof that the disease-free equilibrium is globally stable.

### 6. Stability of Endemic Equilibrium

To prove the stability of endemic equilibrium for SSQVEQIHFR, we include a lemma that can help in establishing the negativity of time derivative of the Lyapunov function.

**Lemma 1:** Assume \( x_1, x_2, x_3, \ldots, x_n \), are positive numbers. Then their arithmetic mean is greater than or equal to their geometric mean. In particular

\[
\frac{x_1 + x_2 + \cdots + x_n}{n} \geq \sqrt[n]{x_1 x_2 \cdots x_n}
\]

**Theorem 3:** If \( R_0 > 1 \) the endemic equilibrium point \( E^* = (S^*, S_0^*, V^*, I^*, H^*, F^*) \) of the system is globally asymptotically stable.

To prove the global stability of endemic equilibrium of the system, we also construct the Lyapunov function of the endemic equilibrium points as:

\[
V(t) = k_1 \left( S - S^* - S^* \ln \frac{S}{S^*} \right) + k_2 \left( S_0 - S_0^* - S_0^* \ln \frac{S_0}{S_0^*} \right) + k_3 (V - V^* - V^* \ln \frac{V}{V^*})
\]

\[
+ k_4 \left( E - E^* - E^* \ln \frac{E}{E^*} \right) + k_5 \left( Q - Q^* - Q^* \ln \frac{Q}{Q^*} \right) + k_6 \left( I - I^* - I^* \ln \frac{I}{I^*} \right)
\]

\[
+ k_7 \left( H - H^* - H^* \ln \frac{H}{H^*} \right) + k_8 \left( F - F^* - F^* \ln \frac{F}{F^*} \right)
\]

And the derivative of the Lyapunov function is

\[
\frac{dV(t)}{dt} = k_1 \left( S - S^* \right) S' + k_2 \left( S_0 - S_0^* \right) S_0' + k_3 (V - V^*) V'
\]

\[
+ k_4 \left( E - E^* \right) E' + k_5 \left( Q - Q^* \right) Q' + k_6 \left( I - I^* \right) I'
\]

\[
+ k_7 \left( H - H^* \right) H' + k_8 \left( F - F^* \right) F'
\]

Using the result of evaluating first three derivatives, we consider substituting derivatives of subsequent state variables to get
\[
\frac{dV(t)}{dt} = -\lambda \left( S - S^* \right)^2 + k_i \left( 1 - \frac{E}{E^*} \right) \left( (1 - \gamma) b \lambda S + \chi E - \chi Q \right) + \left( 1 - \eta \right) \lambda V - (\chi + \alpha_i)E \\
+ k_i \left( 1 - \frac{Q}{Q^*} \right) \left( (1 - \frac{\alpha_i}{Q}) E - \chi Q \right) + k_i \left( 1 - \frac{I}{I^*} \right) \left( (1 - \frac{\alpha_i}{I}) E - \chi Q \right) + \left( 1 - \delta \right) \delta I - \left( 1 - \delta \right) \delta I' - \left( 1 - \delta \right) \delta I'' \\
+ \left( 1 - \delta \right) \delta I' - \left( 1 - \delta \right) \delta I'' - \left( 1 - \delta \right) \delta I''' - \left( 1 - \delta \right) \delta I'''
\]

To further simplify the Lyapunov function we have to determine the coefficients \( k_i \), \( i = 4, 5, 6, 7, 8 \) by defining their relationships based on the suggestion of as:

- If \( k_i = k_8 \), we have \( \gamma b \lambda S' + \chi E' = \alpha_i Q' \) from the corresponding equation. We need to choose \( k_8 \) such that \( k_i \alpha_i Q' = k_i (\chi + \alpha_i)E' \). Hence \( k_8 = \frac{k_i (\chi + \alpha_i)}{\alpha_i} \).

- If \( k_i = k_9 \), we have
\[
\alpha_i E' = (\theta_{I'} I + (1 - \delta_2) \gamma_{II} + (1 - \delta_2) \gamma_{II} - (1 - \delta_2) \gamma_{II}) I'
\]
from the corresponding equation. We need to choose \( k_9 \) such that
\[
k_i (\theta_{I'} I + (1 - \delta_2) \gamma_{II}) I' = k_i (\chi + \alpha_i)E'
\]
Hence \( k_9 = k_i (\chi + \alpha_i) \).

- If \( k_i = k_9 \), we have
\[
\alpha_i Q' + \theta_{I'} I = (\delta_2 \gamma_{II} + (1 - \delta_2) \gamma_{II}) H'
\]
from the corresponding equation. We need to choose \( k_9 \) such that
\[
k_i (\delta_2 \gamma_{II} + (1 - \delta_2) \gamma_{II}) H' = k_i \alpha_i Q'
\]
Hence \( k_9 = \frac{k_i \alpha_i}{\alpha_i} = k_9 \).

- If \( k_i = k_9 \), we have \( \alpha_i Q' + \theta_{I'} I = (\delta_2 \gamma_{II} + (1 - \delta_2) \gamma_{II}) H' \) from the corresponding equation. We need to choose \( k_9 \) such that
\[
k_i (\delta_2 \gamma_{II} + (1 - \delta_2) \gamma_{II}) H' = k_i \alpha_i Q'
\]
Hence \( k_9 = \frac{k_i \alpha_i}{\alpha_i} = k_9 \).

- If \( k_i = k_9 \), we have \( \gamma b \lambda S' (1 - \frac{Q}{Q^*}) < 0 \), if \( QS < Q^* \). Also \( (1 - \gamma) b \lambda S' \left( \frac{V}{E^*} \left( \frac{S}{S^*} \right) \right) < 0 \) since \( V = V^* \) and \( S^* < S \).

Hence \( k_9 = k_i (\theta_{I'} I + (1 - \delta) \gamma_{II}) I' - \left( 1 - \delta \right) \delta I'' \)

- If \( k_i = k_9 \), we have \( (1 - \delta) \delta I' + \delta_2 \gamma_{II} H' = \gamma F^* \) from the corresponding equation. We need to choose \( k_9 \) such that
\[
k_i (\theta_{I'} I + (1 - \delta) \gamma_{II}) I' = \frac{1}{\theta_{I'} I + (1 - \delta) \gamma_{II}} I'
\]
Hence \( k_9 = \frac{k_i (\theta_{I'} I + (1 - \delta) \gamma_{II}) I'}{(1 - \delta) \delta I''} \)

The first term is negative unless \( S = S^* \), and
\[
(1 - \eta) \lambda (V - V^*) = 0 \text{ because } V = V^*. \]
It is obvious that the remaining terms are negative. Using the lemma, let $x_i, i=1,2,3$ (depending on the elements of epidemic points in brackets), considering $-\theta_{IHF}I'(F'H + HI'H + FI'F - 3)$ so that the fractions within the bracket represent $x_i$, and applying same to other similar expressions. Therefore we can conclude that $\frac{dV(t)}{dt} < 0$, and $\frac{dV(t)}{dt} = 0$, when $(S_0, V_0, E_0, Q_0, I_0, H_0, F_0) = (S^*, V^*, E^*, Q^*, I^*, H^*, F^*)$

7. Numerical Simulation

To validate our model, numerical simulation of $SS_0VEQIHFR$ was done, based on the parameters values in Table 2 by solving the model dynamics using Runge-Kuttaode45 solver implemented in MATLAB. Some parameters values are taken from$^{[12]}$ while some probabilities are assumed. The initial population of each compartment was assumed, Transmission rates in the community, hospital and at the funeral, control parameters or average period of infectives stay in infectious compartments was varied to provide a less than or greater than unity threshold, to prove the global stability of disease-free and endemic equilibrium states. Figure 2, shows disease free equilibrium levels for $R_0 = 0.8576$ and the endemic equilibrium level for $R_0 = 2.6458$

| Parameter | Value |
|-----------|-------|
| $\beta_s$ | 0.22 |
| $\rho$ | 0.05 |
| $\delta_1$ | 0.0025 |
| $\gamma_f$ | 0.05 |
| $\gamma_h$ | 0.23 |
| $\beta_h$ | 0.0022 |
| $\omega$ | 0.02 |
| $\delta_2$ | 0.21 |
| $\gamma_h$ | 0.32 |
| $\beta_F$ | 0.0052 |
| $\epsilon$ | 0.01 |
| $\chi$ | 0.25 |
| $\theta$ | 0.23 |
| $\gamma_f$ | 0.01 |
| $b$ | 0.0025 |
| $\xi$ | 0.13 |
| $\alpha_1$ | 0.23 |
| $\chi$ | 0.25 |
| $\phi$ | 0.00001 |
| $k$ | 0.04 |
| $\eta$ | 0.01 |
| $\alpha_2$ | 0.25 |
| $\gamma_f$ | 0.012 |
| $\gamma$ | 0.5 |

Figure 2, above confirm the results of stability of $SS_0VEQIHFR$. It established that if $R_0 < 1$ the epidemic dies out after a short period of time, but if $R_0 > 1$ the epidemic becomes endemic affecting more people over a long
period of time. Graphically, this explains the fact that the disease-free equilibrium is locally stable for $R_0 < 1$ and the endemic equilibrium is locally stable whenever $R_0 < 1$.

A sample population of 5000 with initially 15 infectious individuals and sample parameters as provided in Table 2 was used to simulate the model within 600 days. The figures showed that the susceptible class declines gradually and remains above 30 through the epidemic period, when the basic reproduction number is less than unity. The rate of exposure, infection, hospitalization and number of funeral delayed to reach their heights until nearly half the epidemic period of 600 days. When the basic reproduction number was greater than unity, $R_0 = 2.6458$ the susceptible population decreases rapidly and reaches the lowest minimum at about 250 days of the outbreak. The equilibrium levels of disease states classes rose rapidly within the early period of the epidemic with the exposed number rising to 850 individuals, while the infected increased to 420 within 120 days of epidemic period, and the total number of hospitalized and funerals remains higher at 550 and 380 respectively. Though recovery rate of victims over time seem to have slight difference in both situations, this is partly due to uniform prevention and control measures that might have been put in place, but the rate reached its peak within shortest period of the outbreak when $R_0 = 2.6458$ unlike when $R_0 = 0.8576$.

8. Sensitivity Analysis

In this section, the sensitivity analysis of reproduction number and endemic equilibrium points are analyzed for disease transmission and prevalence. Sensitivity analysis guides modelers, decision or policy makers to gauge intervention and control measures that can effectively stall the growth of infection in a population. Normalized forward sensitivity index of a variable $R_0$ depending on a parameter $y_j$ is defined as

$$\Gamma_{y_j} R_0 = \frac{\partial R_0}{\partial y_j} \times \frac{y_j}{R_0}$$

The sensitivity indices of the basic reproduction number with respect to its16 parameters in the Table 3.

| Parameter $y_j$ | Sensitivity Index $\Gamma_{y_j} R_0$ |
|-----------------|-----------------------------------------|
| $\beta_1$       | 0.1270                                  |
| $\beta_2$       | 0.0001                                  |
| $\beta_3$       | 0.0047                                  |
| $\rho$          | $-0.0067$                               |
| $r$             | $-0.0000$                               |
| $\chi$          | $-0.0872$                               |
| $\alpha_i$      | 0.0979                                  |
| $\delta_1$      | $-0.0012$                               |
| $\delta_2$      | 0.0120                                  |
| $\theta$        | $-0.1084$                               |
| $\gamma_1$      | $-0.0042$                               |
| $\gamma_2$      | $-0.0240$                               |
| $\gamma_3$      | $-0.0047$                               |
| $\gamma_4$      | $-0.1231$                               |
| $\gamma_5$      | $-0.0005$                               |
| $\gamma_6$      | $-0.0004$                               |

It showed that parameters $\beta_1, \beta_2, \beta_3, \varepsilon, \rho, r, \alpha_i, \delta_2$ have a positive impact on $R_0$. The rest have a negative impact. So that a 10% increase (decrease) in $\beta_1$ will result in a 1.479% increase (decrease) in $R_0$. On the other hand a 10% increase (decrease) in $\theta$, and $\gamma_2$ decreases (increases) $R_0$ by 1.084% and 1.231% respectively. Parameters $\beta_1, \alpha_i, \gamma_2$ and $\theta$ are the most sensitive to $R_0$. This findings concur with results of $\beta_1$ in a study of SEQHRS epidemic model.

Evaluating the normalized forward sensitivity index of endemic equilibrium points involves more analytic matrix operation developed in [9]. At $R_0 = 1$ disease state variables are no threat, however, once an outbreak occurs, the $R_0 > 1$ value indicates an endemic threat. How each
Table 4. Sensitivity indices of endemic equilibrium points with respect to the model parameter values

| Parameter | $\Gamma_y$ | $\Gamma^S_y$ | $\Gamma^V_y$ | $\Gamma^E_y$ | $\Gamma^Q_y$ | $\Gamma^I_y$ | $\Gamma^H_y$ | $\Gamma^F_y$ |
|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|
| $\gamma$ | -0.00298 | -0.00437 | -0.00077 | -0.01624 | 0.00675 | -0.01059 | 0.02024 | 0.03905 |
| $\beta_l$ | 0.58032 | 0.85111 | 0.15023 | 0.07990 | 0.16810 | 0.05211 | -4.63616 | -8.95248 |
| $\beta_H$ | 0.18414 | 0.27006 | 0.04767 | 0.02535 | 0.05334 | 0.01654 | -1.47109 | -2.84069 |
| $\beta_F$ | 0.03341 | 0.04900 | 0.00865 | 0.00460 | 0.00968 | 0.00300 | -0.26694 | -0.51546 |
| $\rho$ | -0.00619 | -0.00907 | -0.00160 | -0.00085 | -0.00179 | -0.00056 | 0.04943 | 0.09545 |
| $\phi$ | -0.00051 | -0.00075 | -0.00013 | -0.00007 | -0.00015 | -0.00005 | 0.00407 | 0.00785 |
| $\epsilon$ | -0.00029 | -0.00043 | -0.00008 | -0.00004 | -0.00008 | -0.00003 | 0.00233 | 0.00449 |
| $\omega$ | -0.00335 | -0.00491 | -0.00087 | -0.00046 | -0.00097 | -0.00030 | 0.02675 | 0.05166 |
| $\kappa$ | -0.14617 | -0.21438 | -0.03784 | -0.02012 | -0.04234 | -0.01313 | 1.16775 | 2.25494 |
| $b$ | -0.28425 | -0.70561 | -0.05877 | -0.01564 | 0.05556 | -0.01020 | -1.08607 | -2.09726 |
| $\xi$ | -1.38072 | -1.24723 | 0.91225 | 0.08314 | -0.03456 | 0.05422 | -0.10358 | -0.19990 |
| $\phi$ | 0.00000 | -0.20000 | 0.00000 | 0.00000 | 0.00000 | 0.00000 | 0.00000 | 0.00000 |
| $\eta$ | 0.00000 | 0.00000 | 0.02002 | 0.00000 | 0.00000 | 0.00000 | 0.00000 | 0.00000 |
| $\chi$ | -0.03766 | -0.05524 | -0.00975 | -0.20515 | 0.08528 | -0.13380 | 0.25559 | 0.49327 |
| $\alpha_l$ | 0.08286 | 0.12153 | 0.02145 | -0.18866 | -0.18761 | 0.29437 | -0.56231 | -1.08520 |
| $\alpha_2$ | 0.00000 | 0.00000 | 0.00000 | 0.00000 | -0.06538 | 0.00000 | 0.00000 | 0.00000 |
| $\gamma_l$ | 0.31821 | 0.46669 | 0.08238 | 0.03762 | 0.08563 | -0.48418 | 5.94945 | 11.48755 |
| $\gamma_h$ | 0.39354 | 0.57718 | 0.10188 | 0.04884 | 0.10834 | 0.03185 | 4.19069 | 8.09243 |
| $\gamma_f$ | -0.00364 | -0.00533 | -0.00094 | -0.00042 | -0.00097 | -0.00027 | 0.02630 | -0.15612 |
| $\gamma_w$ | -0.19585 | -0.28725 | -0.05070 | -0.02679 | -0.05655 | -0.06577 | 1.32909 | 2.56502 |
| $\gamma_q$ | 0.11395 | 0.16712 | 0.02950 | 0.01345 | 0.03064 | -0.16080 | 1.92617 | 4.16744 |
| $\gamma_{hf}$ | 0.13785 | 0.20217 | 0.03569 | 0.01705 | 0.03789 | 0.01112 | 1.34869 | 2.98369 |
| $\theta$ | -0.30747 | -0.45095 | -0.07960 | -0.04182 | -0.08852 | -1.02488 | 1.74869 | 3.37232 |
| $\delta_1$ | -0.00735 | -0.01077 | -0.00190 | -0.00087 | -0.00198 | 0.01130 | -0.13939 | -0.26466 |
| $\delta_2$ | -0.01447 | -0.02123 | -0.00375 | -0.00180 | -0.00399 | -0.00117 | -0.15522 | -0.29625 |
variable is responsive to changes in associated parameters. Table 4 provides sensitivity indices of endemic equilibrium points for the respective 25 parameters of the model.

For the infectious state, $\beta_1, \beta_h, \beta_f, \xi, \alpha_t, \gamma_h, \gamma_{hf}$ and $\theta$ have a positive influence on the number of infected. While effective hospitalization and reduction in mean period asymptomatic patients become infectious have a high influence on infectious cases, tracing infectious individuals have a more negative impact on the infectives, therefore these factors are to be targeted for epidemic control decisions.

9. Conclusion

In this study, the SS$_Q$VEQIHFR epidemic model is formulated which incorporated intervention and control measure, with different classes of disease states. The study established the model stability at both disease-free and endemic equilibrium based on proven theorems of Jacobian and Lyapunov function construction. The model threshold reproduction number is determined using the next generation approach, the threshold accounted for the contribution of new infection from the symptomatic class of the community (I), hospitalized (H) and dead individuals unsafely buried (F). The normalized forward sensitivity analysis of threshold parameter and endemic equilibrium points of the model was analyzed and found that transmission coefficients in the communities, hospital, and funeral, hospitalization rate, tracing the asymptomatic individuals, and vaccine efficiency rate to have a positive influence on the disease transmission. Similarly, the average period of the infected from hospitalization to recovery and death have a positive influence on the endemic equilibrium points. It implies that a percentage increase in the intervention or control measures that target reduction in these rates and proportions, the epidemic prevalence will reduce accordingly. Therefore, these factors should be targeted for controlling the transmission of the disease.

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