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CAN COMMUNITY-BASED CARE AND ENVIRONMENTAL DISINFECTION MITIGATE THE PNEUMONIA BURDEN?

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Pneumonia is one of the major causes of death among children under five years of age and adults over 65, with more than 2 million deaths occurring in developing countries every year. The efforts for early detection, effective treatment, and minimising the transmission of pneumonia are possible if the dynamics of the disease are well understood. In most countries or communities, hospitals have limited capacity to accommodate those who are sick. Therefore, individuals can be treated as outpatients or in community-based care depending on the severity of the infection. In this research, a model for the transmission dynamics of pneumonia is developed to determine the effect of disinfecting the pathogen-contaminated environment, and community-based care on the disease mitigation process. The basic properties of the model including positivity and boundedness, the basic reproduction number ($\mathcal{R}_0$), existence and stability of equilibria were determined as well as the conditions for existence of backward bifurcation, a scenario where reducing $\mathcal{R}_0$ below 1 may not be enough to eradicate the disease. Sensitivity analysis was performed using the Latin Hypercube Sampling Scheme to determine the parameters with the greatest influence on the reproduction number. The results revealed that transmission rate through the contaminated environment and contact rate through person-to-person have the most significant potential of increasing the disease burden if unabated, while effective treatment and increased decay rate of microbes from the environment have the greatest potential of containing the number of infections. Numerical simulations were performed to illustrate the analytical results as well as establish the long-term behaviour of the disease. It was observed that introducing community-based care can accelerate the containment of the disease as well as reducing the number of deaths in the population. In addition, a higher frequency of environmental disinfection is associated with lower infection cases not only around the peaks but also in the long term. We recommend that the community-based care be enhanced in the disease management process to cater for settings where resources are limited. To ensure proper implementation of community-based care services, training of community-based caregivers on the transmission dynamics of the disease, its management and prevention strategies is mandatory. More still, practising proper hygiene, and applying multiple combinations of control measures including vaccination and isolation of the infected is essential if the disease is to be contained in a shorter time.

Keywords: Pneumonia disease; Environmental Contamination; Community-based Care; Sensitivity analysis; numerical simulations.

Mathematics Subject Classification 2020: 00A71, 37N25, 92B05, 92D30

1. Introduction

Pneumonia is a highly inflammatory respiratory disease that predominantly affects the lungs.¹⁻³ The disease has a high incidence and is caused by microbes including bacteria, fungi, parasites, and viruses.² Among these four disease causing microbes, bacteria and viruses are the most potent causes of pneumonia.^{2,3} The lungs are made up of small sacs called alveoli, which fill with air when a healthy person breathes.⁴ It is reported that during infection, the bacteria enters the alveoli and passages of the lungs

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where they grow rapidly and multiply in number.³ When an individual has pneumonia, the area of the lung that is invaded by the bacteria becomes filled with fluid as the body attempts to fight the infection thereby, causing difficulties in breathing, sharp or stabbing chest pain as well as limiting the intake of oxygen.⁴ The most common pathogens are *Streptococcus pneumoniae*, *Haemophilis influenzae* type b (Hib) and Respiratory Syncytial Virus (RSV).^{5,6} The most common cause of bacteria pneumonia in children in the developing world is the *Streptococcus pneumoniae*, which also causes diseases such as meningitis, ear infections, and sinus infections.⁶⁻⁹ Pneumonia infection spreads through micro-aspiration of oropharyngeal organisms and inhalation of aerosols containing bacteria or viruses especially in children who carry the bacteria in their throats without developing the sickness or showing symptoms.¹⁰

Most cases of pneumonia are reported to be as a result of inhaling small droplets (containing the bacteria) that get into the air or surfaces when an infected person coughs or sneezes.¹⁰ In addition, pneumonia may spread through blood, especially during and shortly after birth.¹⁰ The bacteria can remain suspended in air for lengths of time that depend on the prevailing weather conditions and humidity.¹⁰ It can also be carried in the mouth of a healthy person without causing any harm and find its way to invade the lungs causing the infection.¹⁰ The risk factors associated with the spread of pneumonia include smoking history and passive smoking, crowded living conditions, lack of exclusive breastfeeding, indoor air pollution, alcoholism and drug abuse.^{4,10,11} Pneumonia infection can develop at any age, but it is more common in elderly people aged 65 or more and young children under five years.^{6,10} Moreover, those with pre-existing health conditions such as chronic heart, lung or liver diseases or sickle cell anemia, diabetes, antecedent viral infection, HIV/AIDS, people who have had transplants and are taking medications that decrease their immunity to infection are also at high risk of developing the disease when exposed.^{2,11}

Pneumonia is the major cause of mortality in children under five years in most regions of the developing world when compared to AIDS, malaria and measles combined.³ The highest under-five mortality rates are in low- and middle-income countries (LMIC), namely in sub-Saharan Africa and in Southern Asia.³ Children in low-income countries are more likely to die before the age of five due to pneumonia infection compared to children in high-income countries.^{3,12} Children from low socio-economic class, minority ethnic groups or living in isolated geographic areas suffer more from inequity than their counterparts in the developed world.³ They are also at great disadvantage in accessing appropriate and adequate health services.³ The condition is worsened when an individual with pneumonia is not treated or taken to a healthcare facility, hence, increasing the likelihood of dying from the disease.^{3,10} More than 2 million children in the developing countries die from pneumonia every year.^{12,13} Although the under-five mortality rate has been reported to show an average of 30% to 35% reduction since 1990, poor maternal and child health has continued to remain a significant problem in developing countries.³ For example, it is reported that, in Kenya, pneumonia contributes up to 16% of child mortality.³

The treatment for pneumonia depends on its likely cause and the condition of the infected person.^{3,12} It can be treated at home (in the community) or through hospital-based care whereby the usual approach that is mainly applied is to administer antibiotics that are effective against the bacteria causing the infection.^{5,11} Most cases of pneumonia require oral antibiotics, which are often prescribed at a health center.⁵ Supplementary oxygen is given if a patient is having trouble breathing or if the individual is having low oxygen levels.¹¹ The management of the disease is reported to be challenging due to overlap of its symptoms with those for malaria. As a result, there is a possibility of misdiagnosis and mistreatment with antimalarial drugs in malaria endemic countries.¹² Accurate projections on the possibility of the epidemic or the disease becoming endemic, and strategies to put up control measures are required for this to be achieved.⁴ Preventing pneumonia in children is an essential component of the strategy to reduce child mortality whereby immunization against Hib, pneumococcus, measles and whooping cough (pertussis) are the most effective ways.⁹ Moreover, adequate nutrition is key to improving children's natural defenses, starting with exclusive breastfeeding during the first 6 months after birth as well as early diagnosis and treatment.⁹ In addition to being effective in preventing pneumonia, breastfeeding helps to reduce the length of illness in children.⁹ Addressing environmental living conditions such as safe drinking water, sanitation, low household or indoor air pollution, and encouraging proper hygiene also reduces susceptibility to the disease hence, reducing morbidity and mortality rate.^{4,5,10,14}

Mathematical models integrated in epidemiological research provide powerful tools in studying the dynamics of diseases and finding threshold parameters necessary for controlling the disease.^{4,15–18} Several studies to investigate pneumonia transmission dynamics have been carried out. For example, in a study by Kizito *et al.*,⁴ a model of the spread and control of bacterial pneumonia under public health interventions that involve treatment and vaccination was formulated and analyzed. The impact of pneumonia infection control strategies was investigated and the study revealed that, with treatment and vaccination interventions combined, pneumonia can be contained.⁴ However, with treatment intervention alone, pneumonia persists in the population.⁴ Another study was carried out by Otieno *et al.*,¹⁰ with the main aim to provide a mathematical explanation of pneumonia transmission dynamics, taking into consideration the role of carriers and recovery measures in the transmission. The study revealed that, reducing the transfer rates between the carriers and infected class, reduces the prevalence of the disease and this was considered as a control strategy that can be employed for pneumonia dynamics.¹⁰

In most of the previous researches, the contribution of the pathogen infested environment in the transmission of pneumonia was not considered. It is however known that many microbes remain viable on surfaces and the environment for several hours and days. Owing to this observation, we modify previous models by taking into consideration the contaminated environment transmission route. This is also counteracted by considering disinfection as a mitigation strategy to accelerate the decay of the disease causing microbes. More still, the combined significance of community and hospital-based treatment for pneumonia disease has not been considered in previous researches. We note that several factors including religious beliefs, financial constraints and distance from the health centres in most rural communities may restrict people from visiting hospitals, in which case community-based care treatment will be a viable option. In addition, the health facilities have limited capacity to admit the infected individuals. Therefore, a combined disease management approach that involves both community-based and hospital-based care is essential. Prior to making conclusions and informing policy makers about the need for specific disease management approaches, it is necessary to assess and quantify the extent to which the proposed measures can help mitigate the problem. The model developed in this manuscript is earmarked to achieve this target in addition to exploration of other existing disease control measures such as treatment and vaccination.

The rest of the paper is organised as follows: In section 2, a mathematical model capturing pneumonia transmission dynamics as well as several mitigation measures is developed and analysed. Positivity and boundedness of solutions are proved, the model disease threshold and equilibrium points evaluated in the same section. Section 3 contains sensitivity analysis results conducted using the Latin Hypercube Sampling scheme to determine the processes which are likely to over estimate or underestimate the magnitude of the. In the same section, numerical results are presented with different scenarios of the mitigation measures. The manuscript is concluded in section 4 where recommendations to healthcare practitioners are also provided.

2. Model formulation and Analysis

2.1. The model formulation

We present a model for the transmission dynamics of pneumonia disease in the population accounting for community and hospital-based care, the contaminated environment transmission route, as well as mitigation measures such as disinfection and treatment. The population is divided into seven compartments which include Susceptible (S), Carriers (C), Infected (I), Hospital-based care (T_h), Community-based care (T_c), Recovered (R), and free microbes in the Environment (E). The total human population (N), at time t is given by

$$N = S + C + I + T_h + T_c + R. \quad (2.1)$$

We assume that, there is a recruitment rate from new births and immigration, π , into the susceptible population. We note that some individuals in the susceptible class get infected but do not show symptoms in which case they are called carriers and move to the carrier class. Those that show symptoms are put into symptomatic infected class. Taking this into account, ν is the proportion of individuals who move from

the susceptible class to the symptomatic infected class and $(1 - \nu)$ is the proportion of those who move to the carrier class. Therefore, individuals move from the susceptible class to the infected and carrier classes at rate λ , which constitutes the force of infection. In the force of infection λ given by the expression (2.2), the person-to-person transmission follows standard incidence and environmental transmission follows a monod equation such that

$$\lambda = \beta_1 \left(\frac{I + \eta_1 C + \eta_2 T_h + \eta_3 T_c}{N} \right) + \frac{\beta_2 E}{\kappa + E}, \quad (2.2)$$

where β_1 is the transmission rate through person-to-person contact, η_1 is the relative transmission rate from the carrier class, η_2 is the relative transmission rate from the hospital-based care individuals, η_3 is the relative transmission rate from the community-based care individuals, β_2 is the transmission rate from the environment and κ is the pathogen concentration constant in the environment. We assume that $\eta_1 > \eta_2$ and η_3 , because the carriers are not controlled since they are not known, and $\eta_3 \geq \eta_2$ because of too many visitors that may get in contact with the infected individuals being treated in the community. Therefore, $\eta_1 > \eta_3 \geq \eta_2$. Individuals in the carrier class may develop symptoms later on and move to the infected class at a rate φ , or they recover naturally, and move to the recovered class at a rate α_3 . Due to financial challenges and limitations to the number of hospital beds, some infected individuals can be treated at home (i.e. community-based care). In case the condition becomes severe, individuals can be transferred to healthcare facilities for more specialised care. Taking that into consideration, we assume that there is a fraction, ρ , of the infected individuals who are admitted into hospital-based care, and $(1-\rho)$ to community-based care. ε is the treatment rate of individuals sent into the community and hospital-based care. It is noted that, the infected individuals can also die (at a rate δ_1) due to the disease before getting treatment. The hospital-based care individuals can either get treatment and recover at a rate α_1 and move to the recovered class, or die due to the disease at a rate δ_2 . The infected individuals in community-based care can recover from the disease at a rate α_2 and move to the recovered class. We assume that death due to the disease will not occur in the community-based care because when the infection persists or worsens, the patients would be transferred to the hospital at a rate γ to get specialised healthcare services. Individuals may be moved from the hospital and sent to community-based care to continue with their medication at a rate τ . The parameters σ_1 , σ_2 , σ_3 and σ_4 are the shedding rates from the carrier, infected, hospital and community classes to the environment respectively. The virus decays at a rate θ from the environment. We assume that there is natural death rate denoted by μ that can happen to individuals in every compartment. The recovered individuals develop temporary immunity which wanes at a rate ω , and they become susceptible again. We note that σ_1 and σ_2 are greater than σ_3 and σ_4 since the individuals in the hospital- and community-based care are being controlled and monitored.

The aspects described above are all summarized in Fig. 1. The list of state variables and the parameters used in the model and their description are given in Table 1 and 2 respectively.

Table 1. List of state variables used in the model and their description

State variable	Description
S	Susceptible individuals
C	Carrier individuals
I	Infected individuals
T_h	Hospital-based care individuals
T_c	Community-based care individuals
R	Recovered individuals
E	Pathogen in the environment

Following the model assumptions and the schematic illustration in Fig. 1, we present the model system

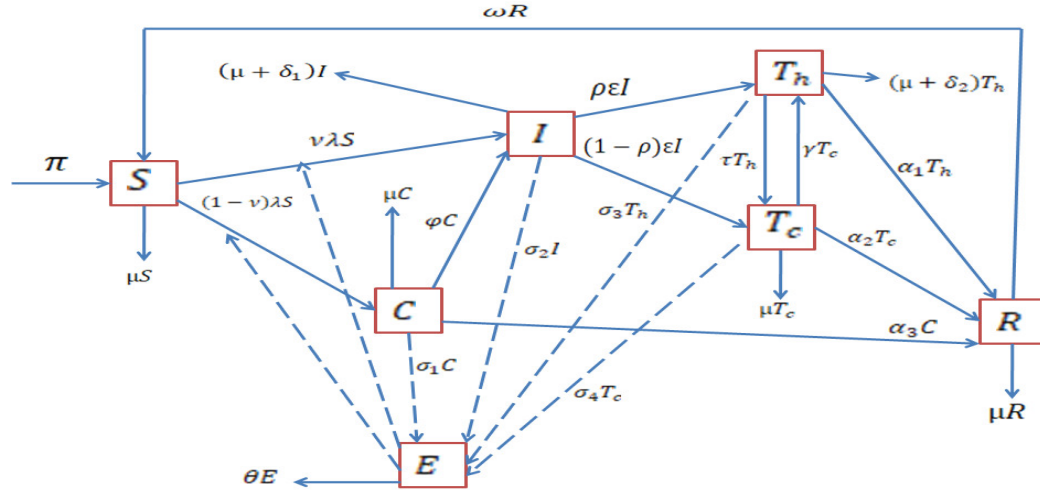


Fig. 1. A compartmental representation of the pneumonia disease model.

Table 2. **Parameters used in the model and their description**

Symbol	Description
π	Recruitment rate into the susceptible class
μ	Natural death rate
ρ	Proportion of individuals admitted in hospital-based care facility
$(1 - \rho)$	Proportion of individuals who are being treated in the community
ε	Treatment rate
η_1, η_2, η_3	Relative transmission rates of the pathogen by individuals in the classes C, H_{bc} and C_{bc} respectively
β_1	Transmission rate through person-to-person contact
β_2	Transmission rate from the environment
κ	Pathogen concentration in the environment
ν	Proportion of infected individuals who move from the susceptible class to the infected class
$(1 - \nu)$	Proportion of infected individuals who move from the susceptible class to the carrier class
$\sigma_1, \sigma_2, \sigma_3, \sigma_4$	Shedding rate from the classes C, I, T_h and T_c respectively into the environment
γ	Rate at which the infected individuals from the community based care get admitted to the hospital due to worsening of the disease
τ	Rate at which the infected individuals move from the hospital to the community based care
$\alpha_1, \alpha_2, \alpha_3$	Recovery rate of infected individuals in classes H_{bc} , C_{bc} and C respectively
φ	Rate at which carrier individuals develop symptoms and send to the infected class
ω	Rate at which recovered individuals become susceptible again
δ_1, δ_2	Virus induced death rate of the infected individuals in the infected and hospital-based classes
θ	Virus decay rate from the environment

of equations for the transmission given in Eqs. (2.3a) - (2.3g).

$$\frac{dS}{dt} = \underbrace{\pi}_{\text{Recruitment}} + \underbrace{\omega R}_{\text{Waned Immunity}} - \underbrace{\lambda S}_{\text{Infections}} - \underbrace{\mu S}_{\text{Natural Death}} \quad (2.3a)$$

$$\frac{dC}{dt} = \overbrace{(1-\nu)\lambda S}^{\text{New infections}} - \overbrace{Q_1 C}^{\text{Depletion of } C \text{ Population}} \quad (2.3b)$$

$$\frac{dI}{dt} = \overbrace{\nu\lambda S + \varphi C}^{\text{New infections \& progression to } I} - \underbrace{Q_2 I}_{\text{Depletion of } I \text{ Population}} \quad (2.3c)$$

$$\frac{dT_h}{dt} = \overbrace{\rho \varepsilon I + \gamma T_c}^{\text{Progression into } T_h \text{ Population}} - \overbrace{Q_3 T_h}^{\text{Depletion of } T_h \text{ Population}} \quad (2.3d)$$

$$\frac{dT_c}{dt} = \overbrace{(1-\rho)\varepsilon I + \tau T_h}^{\text{Progression into } T_c \text{ Population}} - \overbrace{Q_4 T_c}^{\text{Depletion of } T_c \text{ Population}} \quad (2.3e)$$

$$\frac{dR}{dt} = \overbrace{\alpha_1 T_h + \alpha_2 T_c + \alpha_3 C}^{\text{Recovery of infected}} - \overbrace{Q_5 R}^{\text{Depletion of Recovered Population}} \quad (2.3f)$$

$$\frac{dE}{dt} = \overbrace{\sigma_1 C + \sigma_2 I + \sigma_3 T_h + \sigma_4 T_c}^{\text{Pathogen shedding}} - \overbrace{\theta E}^{\text{Pathogen decay}} \quad (2.3g)$$

where $Q_1 = (\mu + \varphi + \alpha_3)$, $Q_2 = (\mu + \delta_1 + \varepsilon)$, $Q_3 = (\mu + \delta_2 + \alpha_1 + \tau)$, $Q_4 = (\mu + \alpha_2 + \gamma)$ and $Q_5 = (\mu + \omega)$.

2.2. Basic Properties of the model

2.2.1. Positivity of Solutions

Since the model describes the infection dynamics of the human population, it is imperative to ensure that all the state variables are non-negative for all $t \geq 0$. We thus state the following lemma.

Lemma 2.1 *Given the initial conditions of the system of Eqs. (2.3) such that*

$$S^0 > 0, \ C^0 \geq 0, \ I^0 \geq 0, \ T_b^0 \geq 0, \ T_c^0 \geq 0, \ R^0 \geq 0 \text{ and } E^0 \geq 0, \quad (2.4)$$

the solutions $S(t)$, $C(t)$, $I(t)$, $T_h(t)$, $T_c(t)$, $R(t)$ and $E(t)$ are non-negative for $t > 0$.

Proof. Assume that

$$\hat{t} = \sup\{t > 0: S(t) > 0, C(t) > 0, I(t) > 0, T_h(t) > 0, T_c(t) > 0, R(t) > 0 \text{ and } E(t) > 0\} \in [0, t].$$

Thus, $\hat{t} > 0$. From the Eq. (2.3a), it follows that

$$\begin{aligned} \frac{dS}{dt} &= \pi + \omega R - \nu \lambda S - (1 - \nu) \lambda S - \mu S, \\ &\geq -\hat{\lambda} S - \mu S, \end{aligned} \quad (2.5)$$

where $\hat{\lambda} = \sup_{t \geq 0} \lambda(t)$. The resulting equation can be solved by separating variables and integrating both sides with respect to correspond variables, and then applying appropriate initial conditions to obtain

$$S(t) \geq S^0 e^{-(\hat{\lambda} + \mu)t} \geq 0. \quad (2.6)$$

From Eq. (2.3b), we have that

$$\begin{aligned} \frac{dC}{dt} &= (1 - \nu)\lambda S - Q_1 C, \\ &\geq -Q_1 C. \end{aligned} \quad (2.7)$$

Using the method of separation of variables on the Eq. (2.7) and applying the necessary initial conditions, we obtain

$$C(t) = C^0 e^{-Q_1 t} > 0. \quad (2.8)$$

Using the same approach for the rest of the equations of the state variables, we obtain

$$\begin{cases} I(t) & \geq I^0 e^{-Q_2 t} > 0, \\ T_h(t) & \geq T_h^0 e^{-Q_3 t} > 0, \\ T_c(t) & \geq T_c^0 e^{-Q_4 t} > 0, \\ R(t) & \geq R^0 e^{-Q_5 t} > 0, \\ E(t) & \geq E^0 e^{-\theta t} > 0. \end{cases} \quad (2.9)$$

Taking the limit as t approaches infinity on each of the obtained state variable expressions, it is clear that $S(t) > 0$, $C(t) > 0$, $I(t) > 0$, $T_h(t) > 0$, $T_c(t) > 0$, $R(t) > 0$ and $E(t) > 0$ for all $t > 0$. This completes the proof. $\square$

2.2.2. Invariant region

Since the model monitors changes in a population, the state variables are shown to be non-negative for all $t > 0$. We ought to show that the total population is bounded above to ensure that the model does not grow without bound and is within a region $\mathcal{D}$ of biological significance. The following lemma presents the region on which the system of Eqs. (2.3) is restricted.

Lemma 2.2 *The feasible region $\mathcal{D}$ for the model (2.3a)-(2.3g) with initial conditions 2.4 defined by*

$$\mathcal{D} = \mathcal{D}_p \times \mathcal{D}_e \in \mathbb{R}_+^6 \times \mathbb{R}_+, \quad (2.10)$$

where

$$\begin{aligned} \mathcal{D}_p &= \left\{ (S(t), E(t), I(t), T_h(t), T_c(t), R(t)) \in \mathbb{R}_+^6 : 0 \leq N(t) \leq \frac{\pi}{\mu} \right\} \quad \text{and} \\ \mathcal{D}_e &= \left\{ E(t) \in \mathbb{R}_+ : 0 \leq E(t) \leq (\sigma_1 + \sigma_2 + \sigma_3 + \sigma_4) \frac{\pi}{\mu} \right\} \end{aligned}$$

are positively invariant and attracting with respect to the system (2.3) for all $t > 0$.

Proof. By considering the system of Eqs. (2.3), the equation for the change in the total population at any time t is given by

$$\begin{aligned} \frac{dN}{dt} &= \pi - \mu N - \delta_1 I - \delta_2 T_h, \\ &\leq \pi - \mu N. \end{aligned} \quad (2.11)$$

The resulting inequality (2.11) can be solved to obtain an explicit solution given by

$$N(t) \leq \frac{\pi}{\mu} + \left(N(0) - \frac{\pi}{\mu} \right) e^{-\mu t}, \quad \text{where } N(0) = N_0. \quad (2.12)$$

From the solution in Eq. (2.12), we observe that as $t \rightarrow \infty$, $N(t) \rightarrow \frac{\pi}{\mu}$. So, if $0 < N_0 \leq \frac{\pi}{\mu}$ then $0 < N \leq \frac{\pi}{\mu}$. This implies that the total population N is bounded from above by $\frac{\pi}{\mu}$. On the other hand, if $N_0 > \frac{\pi}{\mu}$, then N will decrease to $\frac{\pi}{\mu}$ as $t \rightarrow \infty$. This implies that if $N_0 > \frac{\pi}{\mu}$, then the solution $(S(t), C(t), I(t), T_h(t), T_c(t), R(t))$ enters $\mathcal{D}_p$ or approaches $\mathcal{D}_p$ asymptotically.

Similarly, for the concentration of pathogen in the environment, we have

$$\frac{dE}{dt} = \sigma_1 C + \sigma_2 I + \sigma_3 T_h + \sigma_4 T_c - \theta E, \quad (2.13)$$

The Eq. (2.13) can be written as

$$\frac{dE}{dt} \leq (\sigma_1 + \sigma_2 + \sigma_3 + \sigma_4) \frac{\pi}{\mu} - \theta E, \quad E(0) = E_0 \in \mathbb{R}^+, \quad (2.14)$$

since $0 \leq (C^*, I^*, T_h^*, T_c^*) \leq \frac{\pi}{\mu}$ for $t > 0$.

The resulting inequality (2.14) can be solved using the integration factor method and applying the appropriate initial conditions to obtain

$$E(t) \leq (\sigma_1 + \sigma_2 + \sigma_3 + \sigma_4) \frac{\pi}{\mu\theta} + \left(E(0) - (\sigma_1 + \sigma_2 + \sigma_3 + \sigma_4) \frac{\pi}{\mu\theta} \right) e^{-\theta t}. \quad (2.15)$$

We note that as $t \rightarrow \infty$, $E(t) \rightarrow (\sigma_1 + \sigma_2 + \sigma_3 + \sigma_4) \frac{\pi}{\mu\theta}$. In addition, for any initial conditions in the interval $0 < E(0) < (\sigma_1 + \sigma_2 + \sigma_3 + \sigma_4) \frac{\pi}{\mu\theta}$ the resulting solution will in the interval $0 < E(t) < (\sigma_1 + \sigma_2 + \sigma_3 + \sigma_4) \frac{\pi}{\mu\theta}$. Hence, $\mathcal{D}$ is positively invariant under the flow induced by the system (2.3). Therefore, the model (2.3) is well-posed in $\mathcal{D}$ and it is sufficient to study its dynamics in the domain $\mathcal{D}$. $\square$

2.2.3. The disease-free equilibrium and the basic reproduction number

The model has the disease-free equilibrium (DFE) denoted by $\mathcal{E}_0$. At disease-free equilibrium, there are no microbes nor exposed or infected individuals in the population. Therefore, the total population reduces to $N = S$ and the system of Eqs. (2.3a)-(2.3g) reduces to

$$\frac{dS}{dt} = \pi - \mu S. \quad (2.16)$$

The DFE is obtained by equating the right-hand side of the Eq. (2.16) to zero and solving for the steady state values to obtain

$$\mathcal{E}_0 = (S^*, 0, 0, 0, 0, 0, 0) = \left(\frac{\pi}{\mu}, 0, 0, 0, 0, 0, 0 \right).$$

The reproduction number, $\mathcal{R}_0$, is the expected number of secondary cases produced in a completely susceptible population by a typical infective individual.¹⁹ If $\mathcal{R}_0 < 1$, then on average the number of infected individual produced in subsequent generations is less than their predecessors over the course of their infectious period and thus the disease can not establish itself in the population. On the other hand, if $\mathcal{R}_0 > 1$, then each infected individual produces more than one new infectious case which means the disease can remain prevalent in the population. The Disease-free equilibrium, is locally asymptotically stable if all the eigenvalues of the Jacobian matrix evaluated at DFE have negative real parts and unstable if any eigenvalue has a positive real part.¹⁹ In this manuscript, we use the next generation matrix method outlined in¹⁹ to determine the reproduction number. We note that a reproduction number obtained by this method determines the local stability of the disease-free equilibrium point. By considering the infected compartments $\mathcal{Z} = (C, I, T_h, T_c, E)$ and adopting the matrix notations used in,¹⁹ the Jacobian matrices for the new infection terms and the transition terms of the compartments at the disease-free equilibrium are given by

$$F = \begin{pmatrix} (1-\nu)\beta_1\eta_1 & (1-\nu)\beta_1 & (1-\nu)\beta_1\eta_2 & (1-\nu)\beta_1\eta_3 & \frac{(1-\nu)\beta_2\pi}{\kappa\mu} \\ \nu\beta_1\eta_1 & \nu\beta_1 & \nu\beta_1\eta_2 & \nu\beta_1\eta_3 & \frac{\nu\beta_2\pi}{\kappa\mu} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} Q_1 & 0 & 0 & 0 & 0 \\ -\varphi & Q_2 & 0 & 0 & 0 \\ 0 & -\rho\varepsilon & Q_3 & -\gamma & 0 \\ 0 & -(1-\rho)\varepsilon & -\tau & Q_4 & 0 \\ -\sigma_1 & -\sigma_2 & -\sigma_3 & -\sigma_4 & \theta \end{pmatrix}$$

The matrix V^{-1} is given by

$$V^{-1} = \begin{pmatrix} \frac{1}{Q_1} & 0 & 0 & 0 & 0 \\ \frac{\varphi}{Q_1 Q_2} & \frac{1}{Q_2} & 0 & 0 & 0 \\ \frac{\varphi}{Q_1 Q_2} \Phi_2 & \frac{1}{Q_2} \Phi_2 & \frac{Q_4[1 - \Phi_1] + \gamma\tau}{Q_3 Q_4[1 - \Phi_1]} & \frac{\gamma}{Q_3 Q_4[1 - \Phi_1]} & 0 \\ \frac{\varphi}{Q_1 Q_2} \Phi_3 & \frac{1}{Q_2} \Phi_3 & \frac{\tau}{Q_4 \sigma_3 + \tau \sigma_4} & \frac{Q_3 Q_4[1 - \Phi_1]}{Q_3} & 0 \\ \frac{\sigma_1}{Q_1 \theta} + \frac{\varphi}{Q_1 Q_2 \theta} B & \frac{1}{Q_2 \theta} B & \frac{Q_4 \sigma_3 + \tau \sigma_4}{\theta Q_3 Q_4[1 - \Phi_1]} & \frac{\gamma \sigma_3 + Q_3 \sigma_4}{\theta Q_3 Q_4[1 - \Phi_1]} & \frac{1}{\theta} \end{pmatrix},$$

where $\Phi_1 = \frac{\gamma\tau}{Q_3 Q_4}$, $\Phi_2 = \frac{[\rho\varepsilon Q_4 + \gamma(1 - \rho)\varepsilon]}{Q_3 Q_4(1 - \Phi_1)}$, $\Phi_3 = \frac{[\tau\rho\varepsilon + Q_3(1 - \rho)\varepsilon]}{Q_3 Q_4(1 - \Phi_1)}$ and $B = (\sigma_2 + \sigma_3 \Phi_2 + \sigma_4 \Phi_3)$. We note that $\Phi_1 < 1$ since $\frac{\gamma}{Q_4} < 1$ and $\frac{\tau}{Q_3} < 1$. Hence, their product is also less than one. The reproduction number, $\mathcal{R}_0$ is given as the spectral radius (i.e. the largest eigenvalue) of the matrix FV^{-1} such that

$$\mathcal{R}_0 = \mathcal{R}_{0_C} + \mathcal{R}_{0_I},$$

where

$$\begin{cases} \mathcal{R}_{0_C} = \frac{\beta_1(1 - \nu)\varphi}{Q_1 Q_2} + \frac{\beta_1\eta_1(1 - \nu)}{Q_1} + \left(\frac{\beta_1\eta_2(1 - \nu)\varphi}{Q_1 Q_2}\right) \Phi_2 + \left(\frac{\beta_1\eta_3(1 - \nu)\varphi}{Q_1 Q_2}\right) \Phi_3 \\ \quad + \beta_2\pi(1 - \nu) \left(\frac{\sigma_1}{Q_1 \kappa \mu \theta} + \frac{\sigma_2 \varphi}{Q_1 Q_2 \kappa \mu \theta} + \frac{\sigma_3 \varphi}{Q_1 Q_2 \kappa \mu \theta} \Phi_2 + \frac{\sigma_4 \varphi}{Q_1 Q_2 \kappa \mu \theta} \Phi_3\right), \\ \mathcal{R}_{0_I} = \frac{\beta_1 \nu}{Q_2} + \frac{\beta_1 \eta_2 \nu}{Q_2} \Phi_2 + \frac{\beta_1 \eta_3 \nu}{Q_2} \Phi_3 \\ \quad + \beta_2 \pi \nu \left(\frac{\sigma_2}{Q_2 \kappa \mu \theta} + \frac{\sigma_3}{Q_2 \kappa \mu \theta} \Phi_2 + \frac{\sigma_4}{Q_2 \kappa \mu \theta} \Phi_3\right). \end{cases} \quad (2.18)$$

The reproduction number, $\mathcal{R}_0$ is given as the sum of the reproduction numbers associated with new cases generated by the individuals who show symptoms and move to the infected class, and those who are asymptomatic (i.e. carriers). The term $\mathcal{R}_{0_I}$ is given as a sum of five components representing the contributions of individuals in the infected, carrier, hospital-based care, community-based care and microbes infested environment compartments respectively. The term, $\mathcal{R}_{0_C}$ is given as the sum of four components representing the contributions of symptomatic infected class, hospital-based care, community-based care and pathogen infested environment compartments respectively. The term Φ_1 is the proportion of individuals who move back and forth between the hospital and community based care compartments. The term $\frac{\beta_1(1 - \nu)}{Q_1}$ is the fraction of the infected individuals who do not show symptoms and $\frac{\beta_1 \nu}{Q_2}$ is the fraction of individuals who show symptoms after infected by the disease through person to person contact. $\frac{\varphi}{Q_2}$ is the fraction of carrier individuals who show the symptoms later after infected and move to the infected class. The term $\frac{\beta_2(1 - \nu)}{Q_1}$ is the fraction of the infected individuals who become infected by the disease through environment transmission and become carriers whilst $\frac{\beta_2 \nu}{Q_2}$ is the fraction who become symptomatic. Following the major assumptions of the next generation method,¹⁹ we state the following theorem.

Theorem 2.1 *The disease-free equilibrium of the system (2.3) is locally asymptotically stable whenever $\mathcal{R}_0 < 1$ and unstable otherwise.*

2.2.4. The disease-persistent equilibrium point

The model system (2.3) has an endemic equilibrium point

$$\mathcal{E}_1 = (S^*, C^*, I^*, T_h^*, T_c^*, R^*, E^*). \quad (2.19)$$

At the steady state, the system of Eqs. (2.3) reduces to

$$0 = \pi + \omega R^* - \left[\beta_1 \left(\frac{I^* + \eta_1 C^* + \eta_2 T_h^* + \eta_3 T_c^*}{N} \right) + \frac{\beta_2 E^*}{\kappa + E^*} \right] S^* - \mu S^*, \quad (2.20)$$

$$0 = (1 - \nu) \left[\beta_1 \left(\frac{I^* + \eta_1 C^* + \eta_2 T_h^* + \eta_3 T_c^*}{N} \right) + \frac{\beta_2 E^*}{\kappa + E^*} \right] S^* - Q_1 C^*, \quad (2.21)$$

$$0 = \nu \left[\beta_1 \left(\frac{I^* + \eta_1 C^* + \eta_2 T_h^* + \eta_3 T_c^*}{N} \right) + \frac{\beta_2 E^*}{\kappa + E^*} \right] S^* + \varphi C^* - Q_2 I^*, \quad (2.22)$$

$$0 = \rho \varepsilon I^* + \gamma T_c^* - Q_3 T_h^*, \quad (2.23)$$

$$0 = (1 - \rho) \varepsilon I^* + \tau T_h^* - Q_4 T_c^*, \quad (2.24)$$

$$0 = \alpha_1 T_h^* + \alpha_2 T_c^* + \alpha_3 C^* - Q_5 R^*, \quad (2.25)$$

$$0 = \sigma_1 C^* + \sigma_2 I^* + \sigma_3 T_h^* + \sigma_4 T_c^* - \theta E^*. \quad (2.26)$$

We express the steady state values in terms of I^* and obtain

$$\begin{cases} C^* = \Phi_4 I^* & \text{where } \Phi_4 = \frac{(1 - \nu) Q_2}{(\varphi(1 - \nu) + Q_1 \nu)}, \\ T_h^* = \Phi_2 I^* & \text{where } \Phi_2 = \frac{[\rho \varepsilon Q_4 + \gamma(1 - \rho) \varepsilon]}{Q_3 Q_4 (1 - \Phi_1)}, \\ T_c^* = \Phi_3 I^* & \text{where } \Phi_3 = \frac{[\tau \rho \varepsilon + Q_3(1 - \rho) \varepsilon]}{Q_3 Q_4 (1 - \Phi_1)}, \\ R^* = \Phi_5 I^* & \text{where } \Phi_5 = \frac{Q_3 Q_4 (1 - \Phi_1)}{\alpha_1 \Phi_2 + \alpha_2 \Phi_3 + \alpha_3 \Phi_4}, \\ E^* = \Phi_6 I^*, & \text{where } \Phi_6 = \frac{Q_5}{\sigma_1 \Phi_4 + \sigma_2 + \sigma_3 \Phi_2 + \sigma_4 \Phi_3}. \end{cases} \quad (2.27)$$

Adding up Eqs. (2.20), (2.21) and (2.22) and use expressions for R^* and C^* to solve for S^* , we obtain

$$S^* = \frac{\pi}{\mu} - \Phi_7 I^*, \quad \text{where } \Phi_7 = \frac{Q_2 - \omega \Phi_5 + (Q_1 - \varphi) \Phi_4}{\mu} > 0 \quad \text{since } S^* \leq \frac{\pi}{\mu}. \quad (2.28)$$

From the expression for total population, we have

$$N^* = S^* + C^* + I^* + T_h^* + T_c^* + R^*. \quad (2.29)$$

Substituting the relevant terms from (2.27) and (2.28), into Eq. (2.29), we obtain

$$N^* = \frac{\pi}{\mu} - \Phi_8 I^*, \quad \text{where } \Phi_8 = (\Phi_7 - 1 - \Phi_2 - \Phi_3 - \Phi_4 - \Phi_5) > 0 \quad \text{since } N^* < \frac{\pi}{\mu}. \quad (2.30)$$

Using the expression for the force of infection, λ^* and substituting for N^* , C^* , T_h^* , T_c^* and E^* , we obtain

$$\lambda^* = \frac{(\Phi_9 + \Phi_{10} I^*) I^*}{\frac{\pi}{\mu} \kappa + \Phi_{11} I^* - \Phi_{12} I^{*2}}, \quad (2.31)$$

where

$$\begin{cases} \Phi_9 = \beta_1 (1 + \eta_1 \Phi_4 + \eta_2 \Phi_2 + \eta_3 \Phi_3) \kappa + \beta_2 \frac{\pi}{\mu} \Phi_6, \\ \Phi_{10} = [\beta_1 (1 + \eta_1 \Phi_4 + \eta_2 \Phi_2 + \eta_3 \Phi_3) - \beta_2 \Phi_8] \Phi_6, \\ \Phi_{11} = \frac{\pi}{\mu} \Phi_6 - \Phi_8 \kappa, \\ \Phi_{12} = \Phi_6 \Phi_8. \end{cases} \quad (2.32)$$

Substituting the Eqs. (2.31) and (2.28) into the Eq. (2.21), we obtain

$$(1 - \nu) \lambda^* \left(\frac{\pi}{\mu} - \Phi_7 I^* \right) - Q_1 \Phi_4 I^* = 0 \quad (2.33)$$

We note that the expression λ^* is in terms of I^* . Substituting for λ^* in Eq. (2.33) and simplifying the resulting polynomial, we observe that either $I^* = 0$ is one of the roots and the rest of the roots can be obtained from the Eq. (2.34)

$$AI^2 + BI + C_0 = 0, \quad (2.34)$$

where

$$\begin{cases} A &= [(1 - \nu)\Phi_7\Phi_{10} - Q_1\Phi_4\Phi_{12}], \\ B &= [Q_1\Phi_4\Phi_{11} + (1 - \nu)\Phi_7\Phi_9 - (1 - \nu)\Phi_{10}\frac{\pi}{\mu}], \\ C_0 &= \frac{(1 - \nu)Q_1Q_2}{(\varphi(1 - \nu) + Q_1\nu)}\frac{\pi}{\mu}\kappa(1 - \mathcal{R}_0). \end{cases} \quad (2.35)$$

We note that C_0 is positive for $\mathcal{R}_0 < 1$ and is negative for $\mathcal{R}_0 > 1$. From the quadratic Eq. (2.34), we give the following three possible cases for $\mathcal{R}_0 < 1$.

- If $A < 0$, the quadratic Eq. (2.34) has exactly one positive real root.
- If $A > 0, B < 0$ and $B^2 - 4AC_0 > 0$, the quadratic equation (2.34) has two positive real roots.
- In all other cases, the quadratic Eq. (2.34) has no positive real roots.

Therefore, we infer the following statement.

Theorem 2.2 *The model system (2.3) undergoes backward bifurcation at $\mathcal{R}_0 = 1$ if $A > 0$, $B < 0$ and $B^2 - 4AC_0 > 0$.*

In the case when backward bifurcation occurs, the disease persists even if $\mathcal{R}_0 < 1$. This can be seen from Fig. 2 for the model system (2.3) when we take parameter values $\beta_1 = 0.38$ and $\beta_2 = 0.0003$.

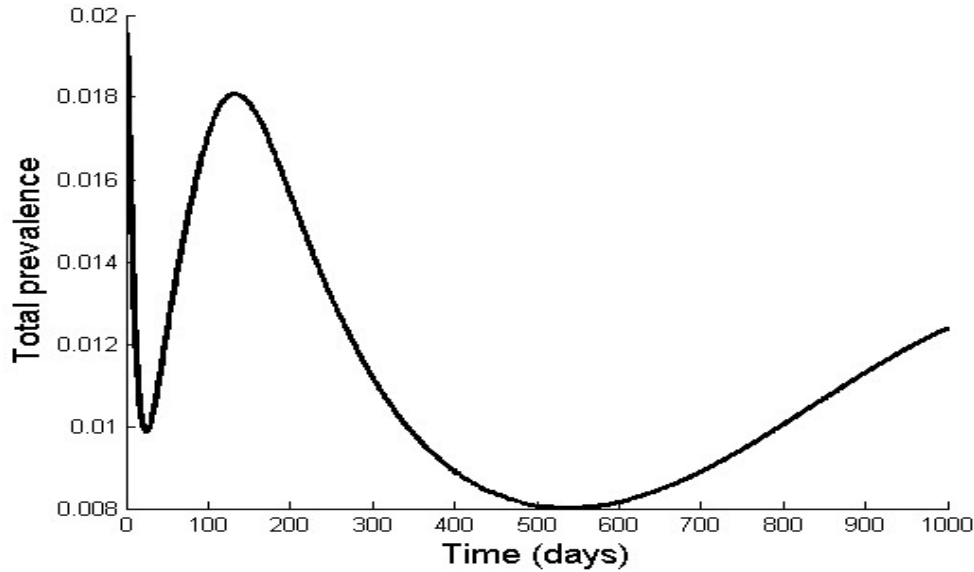


Fig. 2. Change in total prevalence when $\mathcal{R}_0 < 1$. This simulation figure was produced using Matlab 2023a using the parameter values in Table 3.

Following a similar argument as above, for $\mathcal{R}_0 > 1$ since $C_0 < 0$, we have the following three possible conditions for the quadratic Eq. (2.34).

- If $A > 0$, the quadratic Eq. (2.34) has exactly one positive real root.
- If $A < 0, B > 0$, and $B^2 - 4AC_0 > 0$, the quadratic Eq. (2.34) has two positive real roots.
- In all other cases the quadratic equation has no positive real roots.

From the above conditions, we can infer the following statement about the endemic equilibrium of the disease.

3. Numerical simulations

3.1. Parameter estimation

A lot of studies have been done on the dynamics of pneumonia disease, mainly using the data from the literature. In this research, we fit the pneumonia model (2.3) to the data extracted from the Office for National Statistics reports for UK²⁰ and estimated some of the unknown parameter values. We illustrate the analytical results of the pneumonia model by performing numerical simulations using the set of parameter values obtained data fitting as well as published literature. The values assigned to some of parameters have been assumed based on epidemiological information of the disease or demography of the population. The disease induced mortality rates i.e. death due to pneumonia infection was reported to occur after 3 days or within a month if the infected individual does not get the required treatment.^{4, 10, 21, 22} Therefore, we chose the disease induced death rate for the infected individuals (δ_1), and for individuals in hospital-based care, δ_2 , from the range $0.2 - 0.33$ with nominal values 0.33 and 0.125 per day respectively. The total population (N) for UK was approximately 59.12 million in 2001. We consider the recruitment rate $\pi \leq (\mu + r) * N$ where $r = 10^{-6}$ is a small rate of increase to cater for additional number of individuals coming to the population. The initial values considered for the simulation and model fitting are $(S_0, C_0, I_0, T_{h0}, T_{c0}, R_0, E_0) = (0.5 \times N_0, 600\ 000, 400\ 000, 10\ 000, 25\ 000, 200, 5000)$. Time to recovery after an individual with pneumonia infection gets on treatment was stated to be from 7 to 28 days.²³ However, this can be also determined by other health related issues and the type of treatments that are being used. For example, in the community-based care some individuals might not be taking treatments due to lack of financial sources thereby slowing the recovery rate as most cases of pneumonia requires antibiotics. Therefore, we consider the values of general recovery rates from the hospital and community-based care populations i.e. α_1 and α_2 as 0.14 and 0.07 from the range $0.04 - 0.142$ per day respectively.²¹ In a research by Kassa *et al.*,^{24, 25} the concentration of the virus in the environment needed to increase the likelihood of infection by 50% i.e. κ was recorded from a range within $100 - 10^7$ per day, thus we assumed the nominal value as 3000 per day. In some communities, families may lack financial means and some have their societal beliefs that prevent them from visiting the hospitals. In most cases, individuals consider visiting hospitals or contacting a Therefore, we assume 20% of individuals will be hospitalised. The virus that causes the pneumonia infection can stay for 2 up to 7 days on the surfaces depending on the weather conditions.²⁶ We therefore, obtained the virus decay rate from the range 0.14 to 0.5 per day and chose the nominal value of 0.14 per day. The results of the model fit to data are given in Fig. 3.

The summary of the parameter values used in the model numerical simulations are given in Table 3.

3.2. Sensitivity analysis

We examine the sensitivity of $\mathcal{R}_0$ to variations in parameter values and establish the significance of the sensitivity indices. We used the Latin hypercube Sampling (LHS) scheme, which is an efficient stratified Monte Carlo sampling that allows for simultaneous sampling of the multidimensional parameter space and the Partial Rank Correlation Coefficients²⁷ on $\mathcal{R}_0$ for each of the input parameters are obtained. The PRCCs indicate the degree of effect each parameter has on the outcome,^{24, 27} which in this case is the disease reproduction number. The qualitative relationship between the model input and output can be determined by the sign of the PRCC values.¹⁷ A positive PRCC value implies that when the corresponding model input increases, the model output will also increase whilst a negative PRCC value indicates a decrease in the measured model output when the parameters are increased.²⁴ The magnitude of the PRCC measures the importance of the model input in contributing to the model output.^{28, 29} variations of parameters across simultaneously uncertain ranges of individual parameters. The aim of this analysis is to identify the parameters that have the greatest effect on reproduction number as PRCCs illustrate the degree of effect that each parameter has on the outcome.²⁸

The results of sensitivity analysis of input parameters on the reproduction number are indicated in

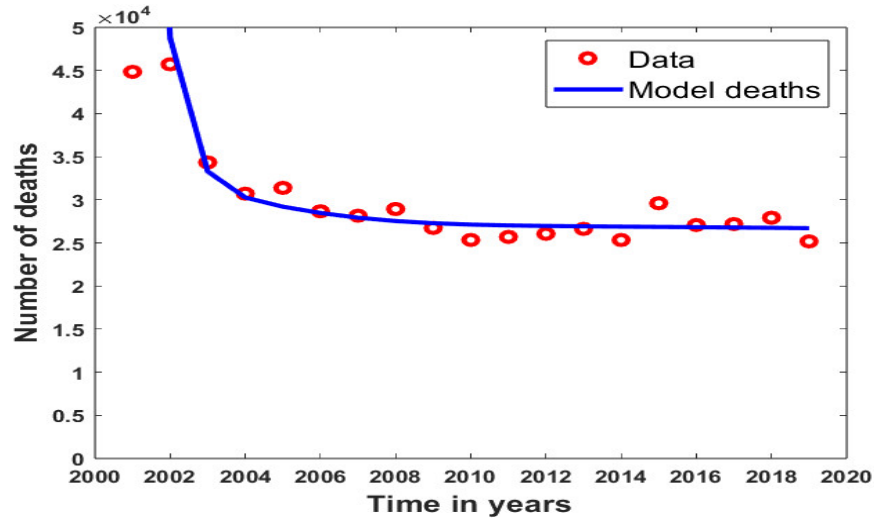


Fig. 3. Model fit to the reported data of deaths due to pneumonia from England for the years 2001 to 2020.

Table 3. Parameter values used in simulations and sensitivity analysis

Parameter	Unit	Range and Nominal Value	Source
π	person day ⁻¹	$\leq (\mu + r) * N_0$	assumed
μ	day ⁻¹	(0.00004-0.00007) 0.000033	30
ρ	day ⁻¹	(0-1) 0.2	fitted
ε	day ⁻¹	(0.01 - 1) 0.46	fitted
η_1	relative value	(0.001-0.05) 0.15	fitted
η_2	relative value	(0.001-0.05) 0.06	fitted
η_3	relative value	(0.001-0.05) 0.09	fitted
β_1	day ⁻¹	(0.0001-1) 0.198	fitted
β_2	day ⁻¹	(0.00001-0.025) 0.003	fitted
κ	number of pathogens per day	(100-10 ⁷) 3000	fitted
ν	day ⁻¹	(0-1) 0.3	fitted
σ_1	number of pathogens person ⁻¹ day ⁻¹	(0.001-0.05) 0.052	fitted
σ_2	number of pathogens person ⁻¹ day ⁻¹	(0.001-0.05) 0.0495	fitted
σ_3	number of pathogens person ⁻¹ day ⁻¹	(0.001-0.05) 0.021	fitted
σ_4	number of pathogens person ⁻¹ day ⁻¹	(0.001-0.05) 0.032	fitted
γ	day ⁻¹	(0.001 -1) 0.25	fitted
τ	day ⁻¹	(0.001 -1) 0.2	fitted
α_1	day ⁻¹	(0.04-0.14) 0.142	4, 21
α_2	day ⁻¹	(0.04-0.14) 0.07	4, 21
α_3	day ⁻¹	(0.001-0.0476) 0.0115	4, 21
φ	day ⁻¹	(0.001 - 0.015) 0.01096	10
ω	day ⁻¹	(0.01-0.5) 0.0241	4
δ_1	day ⁻¹	(0.2-0.33) 0.33	4, 10, 31
δ_2	day ⁻¹	(0.2-0.33) 0.125	4, 10, 31
θ	day ⁻¹	(0.01 - 0.5) 0.14	26

the tornado plot in Fig. 4(a) and the box plot in Fig. 4(b) which gives the summary of values including the minimum, lower quartile, median, upper quartile and maximum value of the computed reproduction number as well as outliers. The process described by the parameters β_1 , β_2 and σ_1 i.e., the transmission rate through person-to-person contact, transmission rate from the environment and shedding rate from the C class, with the greatest positive PRCCs have the greatest potential of worsening the epidemic when increased. Therefore, minimising the associated processes may reduce the transmission of the disease. On the other hand, the processes described by parameters ν , ε , α_3 , φ and θ with negative PRCCs have

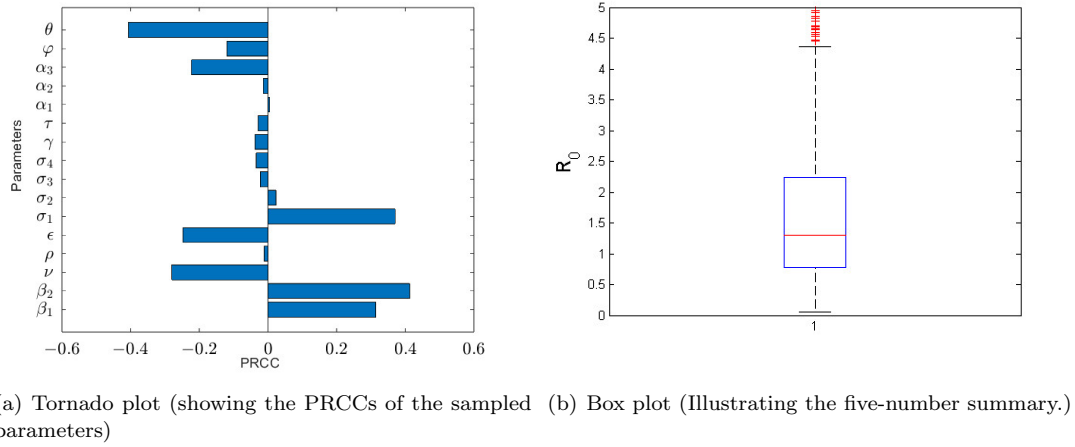


Fig. 4. (a) shows the Tornado plot (with PRCCs in the range $[-1,1]$), (b) The box plot showing the summary of the minimum value, lower quartile, median, upper quartile, the maximum of values of $\mathcal{R}_0$ computed from the sampled parameter space.

the greatest potential in helping contain the infection when maximised. The computed values of the reproduction number, $\mathcal{R}_0$, range within the interval $(0,5)$. From the results in Fig. 4(b), we observed that the lower quartile is below 1, the median is between 1 and 1.5 and the upper quartile is approximately 2.5. Although the computed $\mathcal{R}_0$ has a median above 1, the range of values of $\mathcal{R}_0$ indicate that there are parameter combinations for which $\mathcal{R}_0$ is below 1 potentially containing the disease. Similarly, Different combinations of parameters can lead to a higher value of $\mathcal{R}_0$, potentially worsening the disease.

We note that although some parameters in the model may have very small magnitudes of PRCCs and non-monotonically related to the reproduction number, they may still produce sizable changes in the disease burden when combined with other processes.³² We computed the p -values for the PRCCs of the sampled parameters after Fisher's Transformation (which involved transforming the skew distribution to a normal distribution) as described in^{24,32} to identify the most significant parameters. False discovery rate adjustment was done using the Benjamini-Hochberg procedure to reduce the likelihood of making a Type 1 statistical Error. The parameters PRCCs and their FDR adjusted p -values are given in Table 4.

Table 4. **Parameter PRCC Significance (Adjusted p -value)**

Variable(Parameter)	Parameter Range	Nominal value	PRCCs	p -value	Significant?
β_1	(0.0001,1)	0.198	0.31398	0.0000	Yes
β_2	(0.00001-0.025)	0.003	0.41272	0.0000	Yes
ν	(0,1)	0.03	-0.28036	0.0000	Yes
ρ	(0,1)	0.2	-0.01121	7.737×10^{-01}	No
ε	(0.01,1)	0.46	-0.24805	5.329×10^{-15}	Yes
σ_1	(0.001,0.05)	0.052	0.37041	0.0000	Yes
σ_2	(0.001,0.05)	0.0495	0.02333	6.001×10^{-01}	No
σ_3	(0.001,0.05)	0.021	-0.02215	6.001×10^{-01}	No
σ_4	(0.001,0.05)	0.032	-0.03434	4.507×10^{-01}	No
γ	(0.001,1)	0.25	-0.03788	4.178×10^{-01}	No
τ	(0.001,1)	0.2	-0.02829	5.458×10^{-01}	No
α_1	(0.04,0.14)	0.142	0.00505	8.743×10^{-01}	No
α_2	(0.04,0.14)	0.07	-0.01384	7.594×10^{-01}	No
α_3	(0.001,0.0476)	0.0115	-0.22237	3.138×10^{-12}	Yes
φ	(0.001,0.015)	0.01096	-0.11966	3.294×10^{-04}	Yes
θ	(0.01,0.5)	0.14	-0.40613	0.0000	Yes

The parameter values whose p -values are significant are indicated with a “yes” and those whose p -values are not significant with “No” in the “Significant?” column. From the sensitivity analysis results, we recommend that preventive measures such as increasing physical distancing, practising proper hygiene, and disinfect the infected environment among others should be enhanced to accelerate the containment of the disease. In cases where the health condition is severe, the infected individuals should be admitted to the hospital for more specialised treatment which can accelerate recovery from the disease.

3.3. Simulation results

The reproduction number, $\mathcal{R}_0$, in this manuscript measures the number of secondary infections generated in a purely susceptible population as result of introduction of an infected individual or contact with the microbes in the environment. Therefore, when $\mathcal{R}_0 > 1$ the infection is likely to establish itself in the population and when it is less than one, the disease can potentially be contained. We display the evolution of total prevalence over time considering the reproduction number, $\mathcal{R}_0$ in Fig. 5. The simulations were carried out for selected varying values parameters in Table 3 and the initial conditions $(S_0, C_0, I_0, T_{h_0}, T_{c_0}, R_0, E_0) = (60000, 200, 20, 12, 25, 3, 5000)$. We indicated the comparison of total prevalence for $\mathcal{R}_0$ above one in Fig. 5(a) and $\mathcal{R}_0$ below one in Fig. 5(b). Our model predicted the cases to

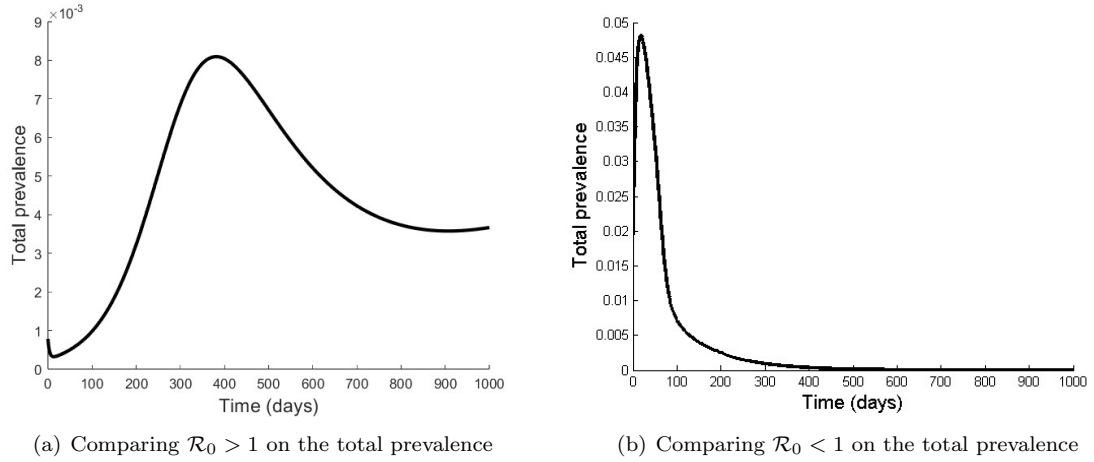


Fig. 5. Comparing total prevalence where in (a) $\mathcal{R}_0 > 1$ and in (b) when $\mathcal{R}_0 < 1$. When $\mathcal{R}_0 > 1$, the disease remains endemic and for $\mathcal{R}_0 < 1$, there is a possibility of containing it.

reach the peak values in about 400 days when $\mathcal{R}_0 > 1$ followed by a decline. However, when $\mathcal{R}_0 < 1$, the results from Fig. 5(a) shows that, in the long term the infection remains in the population. The model also predicted higher prevalence in less 100 days when $\mathcal{R}_0 < 1$. Moreover, we observed that the infection is reduced to negligible values which is likely to show that the disease will not get established in the population when $\mathcal{R}_0$ is below one.

3.3.1. Effect of environmental disinfection

In most cases, pneumonia is reported to be transmitted through inhaling droplets of coughs containing disease causing microbes. Also, sharing utensils, touching used tissues or other item after someone with bacterial or viral pneumonia has used can increase the likelihood of disease transmission. The physical environment considered represents an important source of pathogens that can cause infections. Microbes are often spread within households and/or in crowded places conditions. Therefore, controlling the disease transmission rate from the environment, β_2 for example by wearing personal protective clothes reduces the likelihood of disease transmission. More still, increasing the decay rate θ , of the microbes by disinfecting the environment helps contain the infection in the population. From Fig. 6, we show the effect of increasing

the pathogen decay rate on the prevalence of the diseases. The total prevalence is computed using the expression $\frac{I + T_h + T_c}{N}$. We excluded the carriers from the total prevalence, because they are not known in the population since they do not show symptoms and may have not been screened.

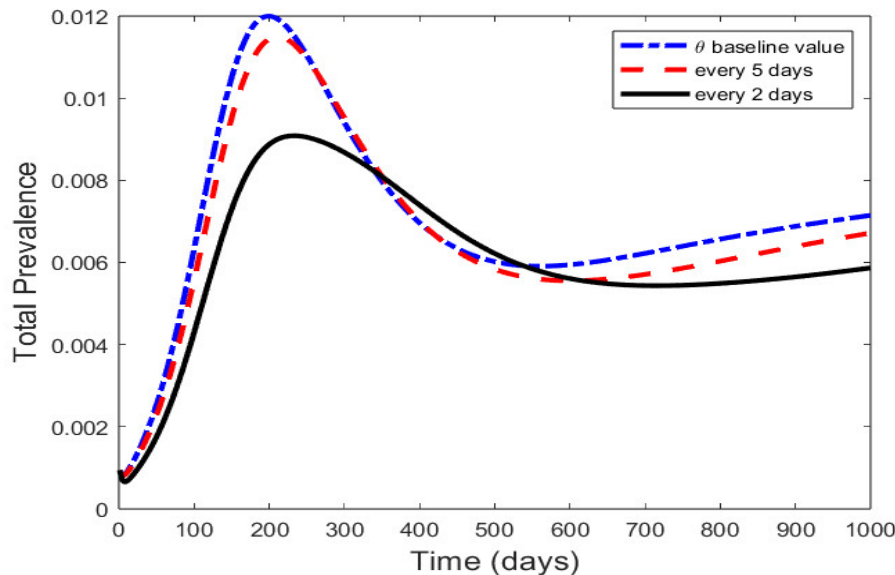


Fig. 6. Effect of increasing pathogen decay rate through environmental disinfection. A higher frequency of environmental disinfection is associated with lower infection cases not only around the peaks but also in the long term.

The baseline value of θ measures the minimal rate of cleaning and disinfection of contaminated surfaces in the environment applied. The baseline value of 0.14 per day is equivalent to 7 days, i.e., disinfecting once every 7 days. In this analysis we consider scenarios where disinfection is done once every after 5 days and after 2 days. From the results, it is observed that, increasing the frequency of disinfection which accelerates the decay rate of microbes can lead to reduced numbers of infected individuals by approximately 5% and 32% for the disinfection frequency of every after 5 days and after 2 days respectively. It is evident that practising proper hygiene and an increased frequency of disinfection reduce the likelihood of new infections and will subsequently mitigate the infection in the affected community.

3.3.2. Benefit of introducing a community-based care

Treatment of pneumonia infection in community-based care may be more affordable than in hospitals. However, there is a challenge of minimising the number of contacts with the infected individuals in the community as well as restricting number of visitors and family members in such settings. Many people in the developing countries and resource limited societies face some constraints hindering them from accessing the hospitals when needed. We note that, people with severe pneumonia are also vulnerable to other infections as a result of weak immunity and the risk could be increased as a result of staying in crowded hospital wards. In most cases, individuals in the community tend to rely on home remedies as the first line of treatment for pneumonia, or other flu like infections. However, in hospitals there are more healthcare professionals to assist and it is advisable to visit the hospital when the infection persists. Severe cases of pneumonia infection should be managed/handled in the hospital since the specialised equipment to provide with extra oxygen may be needed. Moreover, in the hospital there is an advantage of a limited number of visitors hence reducing the rate of transmission. It is also important to limit number of contacts when recovering since the infection remain contagious after for several days even when taking medication. To determine the significance of introducing the community-based care, we conducted

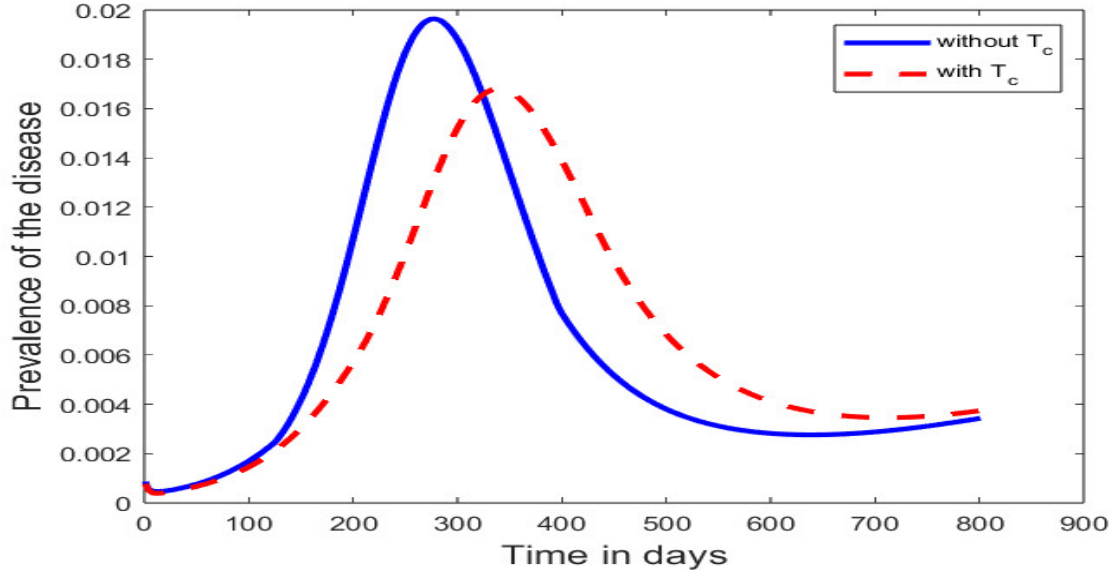


Fig. 7. Effect of treatment uptake on the total prevalence of the disease on the population with and without T_c .

numerical simulations for the model system (2.3) without the community-based care individuals, that is, when $\rho = 1$ and $\tau = 0$. We also conducted the simulations for the model system (2.3) when $\rho = 0.2$, that is, with the community-based care individuals accounting for about 20% of all treated individuals. The results are shown in Fig. 7. From the results we observed that, consideration of treatment in both community and hospital-based care is associated not only with lower peaks of the number of infections but also a delayed time period over which the peaks are reached. With consideration of community-based care the infection peaks are reached at about 300 days compared to the case without community-based care when the peaks are reached after 250 days. The time difference of 100 days may be very essential for countries and healthcare systems to put resources in place to manage the disease and save lives. It is important to note that treatment alone will not suffice in curtailing the disease if effective contacts are not minimised in addition to improved hygiene to prevent new infections.

From the results of the simulations shown on Fig. 7, we observed that introducing the community-based care results in reduced prevalence by 21%. Moreover, the number of deaths is reduced as shown on Fig. 8 since almost every individual would have access to the medication.

Similar to the trends in prevalence for cases of disease management with and without community-based care, lower and delayed peaks are observed. The model also predicts a significant aversion of deaths by about 35%.

3.4. Additional possible interventions

3.4.1. Treatment uptake

Treatment of pneumonia infection can be either in the hospital or community-based. Increasing treatment rate uptake can be done increased screening followed by sending infected individuals to the hospital or treating them in the community as early as the infection is discovered. From Figure 9, we show the effect of increasing uptake into treatment on the total prevalence of pneumonia infection which is obtained by using the formula $\varepsilon(1 + 50\%)$ for 50% increase and $\varepsilon(1 + 90\%)$ for 90% increase. The baseline value of ε is the value given in Table 3. The results show that, with the baseline value, the infection is high in the population and reach the peak value in 100 days. It is observed that, an increase in the rate of treatment uptake by 50% and 90% leads to reduced prevalence rates by approximately 17% and 32% respectively. There was however no observed delay in the time when the peak values are reached.

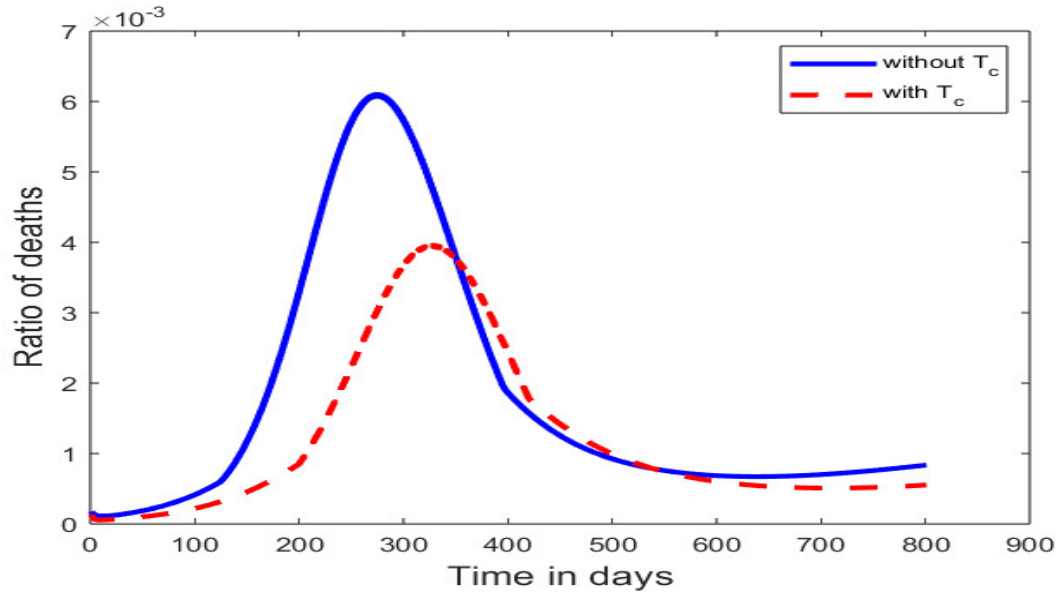


Fig. 8. Total ratio of deaths due to pneumonia in the population.

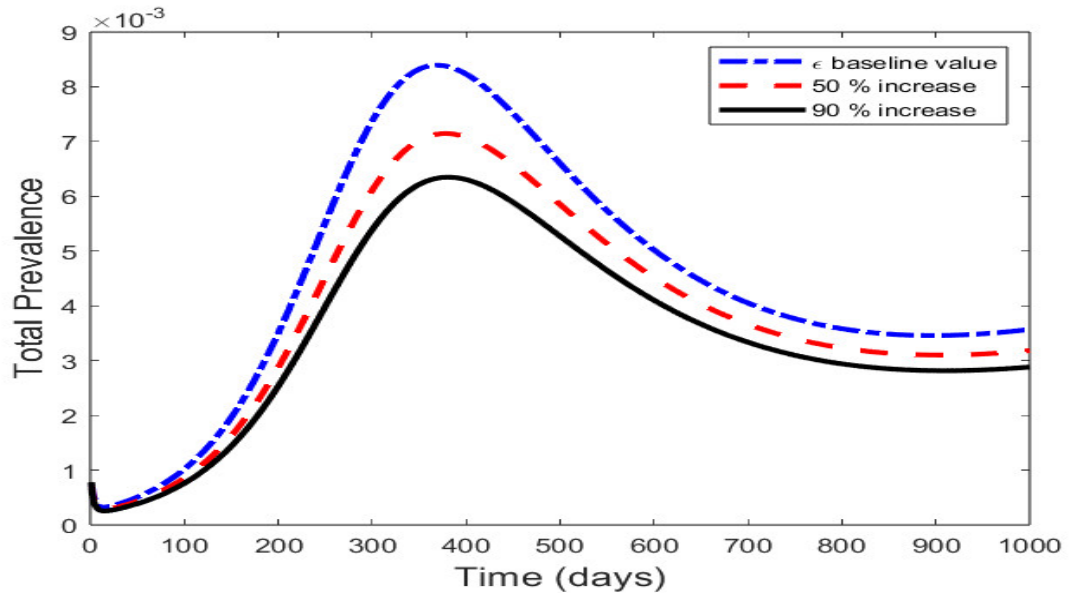


Fig. 9. Effect of increasing the rate of treatment uptake in the total prevalence of the disease.

3.4.2. Effect of reducing person-to-person contact on prevalence

The transmission of pneumonia can be through person-to-person contact as a result of uncovered coughs or sneezes, not washing hands regularly especially after blowing one's nose, coughing, or sneezing. In addition, unhygienic practices by care givers of pneumonia patients increase the likelihood of disease transmission. Reducing person-to-person contact by limiting contact with people who are infected with pneumonia or use of protective clothes when visiting infected individuals and washing hands regularly with soap and water or alcohol based sanitisers help in reducing the number of new cases. Moreover, staying at home or avoiding crowded places reduces the likelihood of disease transmission.

From Fig. 10, we show the effect of reducing person-to-person contact rate on the total prevalence of

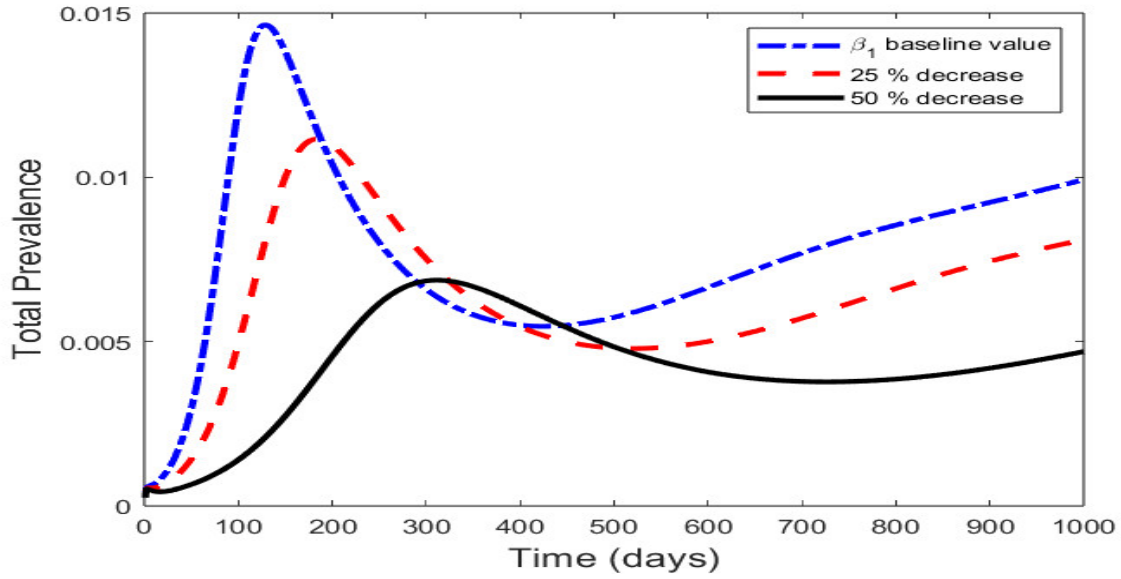


Fig. 10. Effect of reducing the rate of person-to-person contact.

the diseases. The baseline value refers to the nominal value that is shown in Table 3. The baseline value of β_1 is reduced using the formula $\beta_1(1 - 25\%)$ and $\beta_1(1 - 50\%)$ for 25% and 50% decrease respectively. We used a decrease of 25% and 50% to compare the difference from the result of the baseline value.

When the baseline value of β_1 is decreased by 25% and 50%, we observed that the model predicts a reduction in prevalence by approximately 11% and 43% respectively. Moreover, it takes a longer for the prevalence to reach the peak values. This delayed explosion of the disease in the community gives healthcare personnel and policy makers ample time to allocate resources to manage the disease. This scenario also reiterates the importance of behaviour change and focus on preventive measures towards disease management.

3.4.3. Influence of accelerated recovery on prevalence

Most cases of pneumonia require oral antibiotics such as amoxicillin. By increasing the recovery rate we refer to reducing the period remains with the infection. This can be done by increasing the rate of uptake into treatment either in the hospital or community, and using effective medication. There is need for effective treatment to reduce the number of pneumonia cases. Therefore, taking the prescribed course of antibiotics and remedies until full recovery is important because the bacteria can become resistant to antibiotics.

With proper intervention, pneumonia symptoms normally improve within 3 days after starting treatments. Therefore, if being treated at community-based care, it is important to visit the hospital if the infection persists. Figs. 11(a) and 11(b), we show the effect of accelerated recovery rates α_1 and α_2 on the total prevalence respectively. The baseline values of α_1 and α_2 are increased using a similar expression as in section 3.4.1 with a 25% and 50% increase. We observed that, accelerated recovery from both the hospital and community-based care is associated with reduced numbers of infections by an estimate of about 18% and 37% for an increase 25% and 50% respectively in the recovery rate within hospital-based care settings. For community-based care settings the 25% and 50% increase in recovery rate results in reduction in prevalence by 11% and 23% respectively. The model clearly predicts that the used of effective approaches can reduce the severity of the disease. However, the use of treatment alone may not be enough to contain the disease.

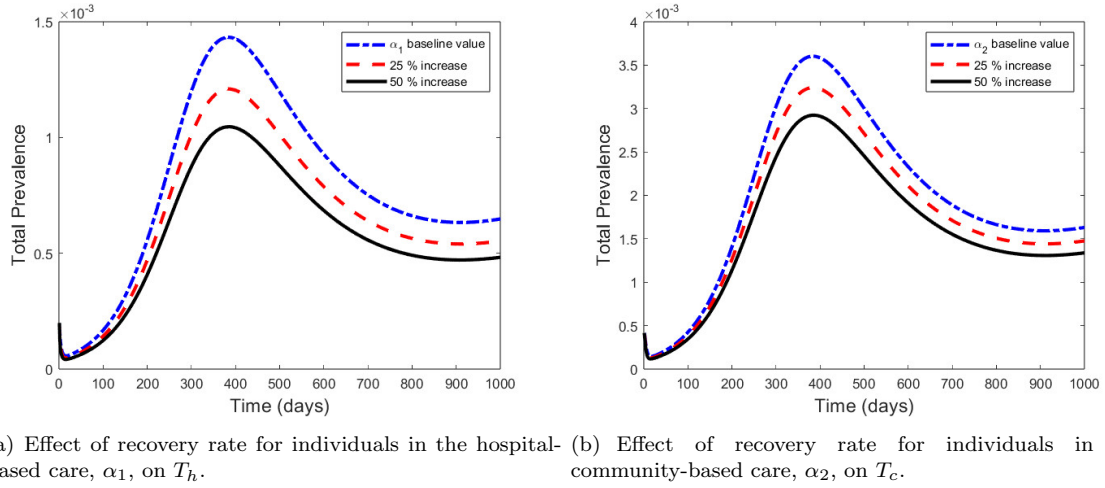


Fig. 11. 11(a) Effect of increasing the recovery rate in the population of hospital-based care, 11(b)Effect of increasing the recovery rate in the population of community-based care.

4. Conclusions

A mathematical model for the dynamics of pneumonia disease transmission was formulated and analyzed. The model considered differential infectivity from population groups (indicated in the force of infection) and disease transmission from pathogen infested environment. The model also considered the disease management processes which included community- and hospital-based care. The developed model was showed to be well-posed, the basic reproduction number was determined and used to identify the conditions for existence and stability of steady states. The model has a disease-free equilibrium which is locally asymptotically stable when the reproduction number is below 1 and an endemic equilibrium when $\mathcal{R}_0 > 1$. Our results showed that there is a possibility of backward bifurcation, a condition of existence of an endemic equilibrium when $\mathcal{R}_0 < 1$. This implies that reducing $\mathcal{R}_0$ just below 1 is not enough to contain the disease. Hence, more disease management and control efforts would be needed to reduce the reproduction number below some critical value if the disease is to be contained.

The model was fitted to data from UK for the time period 2001-2019 and some disease dynamics parameters were estimated from the data while others were determined from published literature. Owing to the uncertainty in the selection of model parameters from literature, sensitivity analysis was performed to identify the parameters that describe the processes which may have a significant effect on the model output. The results indicated that the processes related to transmission rate through a contaminated environment, person-to-person contact rate and microbes shedding rate from the carrier class have the greatest potential of worsening the disease if unabated. On the other hand, the sensitivity analysis results showed that increased uptake of infected individuals into hospital-based care, effective treatment rate that accelerates recovery of infected individuals, and increased decay of the pathogen from the environment (through disinfecting) have a great potential in reducing the reproduction number to below unity. It is therefore important to ensure proper hygiene, sanitary living conditions and reduction of detrimental habits like smoking if the disease is to be contained.

From the numerical simulations results, it was observed that when $\mathcal{R}_0 > 1$, the infection will remain endemic. However, when $\mathcal{R}_0 < 1$ the infection will die out but only when the reproduction number is reduced below a certain critical value. It is evident from the results in Fig. 4(a) and 6 that implementation of mitigation processes (such as disinfection) that result in the decay of the pathogen from the environment increases the likelihood of containing the disease. This further emphasises the need for sensitisation campaigns that enhance proper hygiene practices in both the community and hospital settings. More still, more frequent disinfecting is associated with lower peaks but long-term prevalence of the infection. Our results further show that the disease management strategy that combines both community-based

care and hospital-based care is associated with lower peaks of disease prevalence as well as low deaths.

We recommend that community-based care should be enhanced along with hospital-based care when managing pneumonia cases. We however emphasize that personnel should be trained in managing cases within the community in collaboration with healthcare professionals. This approach is very helpful in resource limited settings and has a three-fold advantage including; (1) training of personnel helps in human capital development of the communities, (2) the approach may be more cost-effective for most families and in the end hospitals are not overwhelmed by non severe cases that could otherwise be managed within the community or home settings, (3) the approach comes with community sensitization on preventive measures, mitigation strategies as well as emphasis on the sanitary standards that should be maintained. In the long-term, this approach may be more sustainable and help in combating other related infections.

The model can be extended to consider optimal control strategies with appropriate admissible controls, cost-effectiveness analysis of the controls strategies can be explored as well as age structuring of the population. The later suggested extension to the model is important since the disease affects specific age groups differently. In addition, the dynamics of multiple strains can also be considered accounting for both community-acquired and hospital-acquired pneumonia.

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Conflict of interest

The authors declare that they have no competing interests related to the work in this manuscript.

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