Modelling the impact of air pollution on the spread of viral respiratory diseases in heterogeneous populations

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Abstract

Despite the fact that the impact of air pollution on respiratory diseases is receiving more and more attention and that the level of pollution will undoubtedly have an impact, there aren't enough studies on how air pollution affects the spread of viral respiratory diseases in heterogeneous populations. For different air pollution levels, including patients who visited the clinic, asymptomatic patients who did not visit the clinic, and symptomatic patients who did not visit the clinic, respectively, the respiratory disease models $SI_hI_sI_aS$ and $SI_pI_hI_sI_aSP$ are constructed in this study. Theoretical analysis demonstrates that when the air pollution level is high, the thresholds that determine the existence and stability of the equilibria of the system are closely related to the daily emissions of air pollutants and the inhalation of pollutants by humans, and the system will undergo fold bifurcation at disease-free equilibrium under certain condition. When the air pollution level is low, the basic reproduction number of the system and the global stability of the equilibria are obtained. Air pollution causes complex dynamical behavior in the spread of viral respiratory diseases, as it can be shown by comparing the two models. Finally, the sensitivity analysis and numerical simulation results show that regardless of the level of air pollution, the change in the proportion of symptomatic infected patients can significantly impact the peak number of patients with viral respiratory diseases. This effect is more pronounced when the level of air pollution is high, and the total number of patients is strongly correlated with daily air pollution emissions, pollutant inhalation, and the proportion of asymptomatic infected patients. Hence, reducing daily emissions of air pollutants and human pollutant inhalation, raising visitor awareness, lowering infection rates, improving cure rates, and boosting immunity, can successfully prevent and control the spread of disease.

Keywords: Air pollution;Asymptomatic infection;Clinic visit;Viral respiratory disease;Fold bifurcation

1 Introduction

Under the current global trend of increasing air pollution, there are widespread epidemics of emerging infectious diseases and even the reappearance of once-controlled infectious diseases. Numerous studies in medicine and public health have shown how air pollution has a significant impact on the occurrence of respiratory diseases[17, 18, 19, 20]. Carugno[1] used Poisson regression models and Bayesian random effects meta-analysis to confirm the relationship between airborne

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PM_{10} and NO_2 concentrations and respiratory illness in haze-polluted areas in Italy. In [2], by using time series analysis, Tolbert et al. demonstrated that the presence of patients with respiratory tract infections was substantially linked with CO, NO_2 and O_3. Using the Cox proportional hazards model, Dong et al.[3] discovered a strong association between PM_{10}, NO_2 concentrations and death from respiratory illnesses.

The effect of air pollution on the infection transmission of respiratory diseases is a topic that many academics are dedicated to researching [29]-[34]. Air pollution, in particular pollutants like NO_2 and $PM_{2.5}$, can erode the respiratory system's defenses. As a result, people may become more susceptible to respiratory illnesses, including the flu, pneumonia, and bronchitis. In addition, air pollution can cause inflammatory responses in the respiratory system, increasing infection susceptibility. Inflammation can impair the immune system's performance, making it more difficult for the body to fight against infection. Air pollution can also make some infections that spread through the air worse, like tuberculosis. Infections can travel through the air for extended lengths of time when carried by pollutants, increasing the risk of transmission. According to the study findings of Chowdhury et al [34], aqueous extract of $PM_{2.5}$ contains elements that have an impact on cell viability. The invasion of inhaled xenobiotics, such as allergens, may worsen several respiratory diseases due to the decrease in cell viability induced by these components.

In recent years, many researchers have been examining the effects of $PM_{2.5}$ on the spread of respiratory diseases and human health using the modeling concepts of infectious disease models. Chen et al.[5] presented a system with an air pollution state-dependent control approach described by the air quality index (AQI), and the results of this model highlighted the significance of proper threshold values of air pollution concentrations to initiate interventions. In [6], Tang et al. developed a mathematical model of AQI trends and respiratory infection dynamics. Meanwhile, some academics have included viral populations or contaminated compartments to examine the dynamics of these models' transmission. Cai et al.[7] constructed a model of tuberculosis in which the transmission rate is a continuous periodic function, and the results showed that a lower level of environmental pollution can effectively inhibit the transmission of tuberculosis. In a recent study, Shi et al.[8] treated air pollutant concentrations as a separate compartment and gave thresholds for $PM_{2.5}$ emissions and pathogenicity. However, the heterogeneity of the population is not taken into account in the above literature.

In biological populations, heterogeneity is a common phenomenon in which different things react differently depending on particular features, such as an individual's own physical qualities, way of life, frequency of social interaction, etc. When people contract diseases, these heterogeneities may cause them to exhibit a variety of characteristics[4]. When patients seek medical assistance, they can be isolated as soon as possible to lessen the likelihood of the disease spreading. Hsu et al.[12] modeled the patients who visited the clinic by dividing them into those with asymptomatic infections, and those with symptomatic infections, and the results elucidated the effect of asymptomatic infections on disease transmission. However, due to their robust immunity, some individuals with respiratory diseases, such as those with acute upper respiratory infections and bronchitis with moderate symptoms, will recover on their own[10, 11]. Despite the fact that these patients may decide not to seek medical assistance, they are still contagious and could spread the illness to healthy individuals[21]. Bao et al.[13] considered the impact of non-visiting patients on the spread of respiratory diseases based on Hsu's study[12] and showed that the number of non-visiting patients had a substantial impact on the initial spread of the epidemic. To examine the effect of individual heterogeneity in the transmission of viral respiratory diseases, mathematical models must thus be developed.

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Air pollution, non-visiting patients and the presence or absence of patient symptoms are the factors considered in the aforementioned literature that affect disease transmission, but few models have integrated the effect of air pollution on the transmission kinetics of viral respiratory diseases in heterogeneous populations. It should be emphasized that susceptible individuals have heightened airway reactivity while breathing in air pollutants, which causes enhanced airway reactivity to breathed-in aeroallergens[15]. This, however, does not always result in the development of allergic respiratory disease. Allergic respiratory disease can occur only when the concentration of air pollutants inhaled by humans is above the critical threshold for making susceptible people sick[14].

Taking into account the sensitivity of different people to air pollutants, awareness of consultation and presence of symptoms, we divide the patients into three groups: those with allergic respiratory diseases brought on by inhaling air pollutants, those with respiratory viral infections brought on by the effects of air pollution and those with respiratory viral infections without the effects of air pollution(specifically, patients with consultation, symptomatic patients without consultation, and asymptomatic patients without consultation). According to the level of air pollution, the transmission dynamics of two different viral respiratory diseases are modeled. On the one hand, by ignoring the effects of air pollution and only taking into account patients who were present at the clinic, asymptomatic infected patients who were not present at the clinic, and symptomatic infected patients, a four-dimensional model is developed to study the transmission dynamics of viral respiratory diseases at low air pollution levels, and on the other hand, a six-dimensional model is built to describe the transmission dynamics of viral respiratory diseases at high air pollution levels by considering the air pollution concentration as a separate compartment. The impact of air pollution on the transmission of viral respiratory infections in heterogeneous populations can be determined by comparing the dynamics results of these two models.

This article has the following structure. In Section 2.1, the $SI_hI_sI_aS$ viral respiratory disease transmission dynamics are modeled, and the boundedness of solutions is given. In Section 2.2, the basic reproduction number and the existence of equilibria of the model are obtained. In Sections 2.3 and 2.4, the local stability and global stability of the equilibria of the model are studied. In Section 3.1, the $SI_pI_hI_sI_aSP$ respiratory disease transmission model is developed, and the boundedness of solutions is given. In Section 3.2, the existence of disease-free equilibrium, boundary equilibria, and endemic equilibrium of the model is investigated, and the local stability and global stability of disease-free equilibrium and boundary equilibria are given. In Section 3.3, the case in which the system may undergo fold bifurcation at disease-free equilibrium is analyzed. In Section 4, the sensitivity analysis of the number of patients, threshold conditions, and the basic reproduction number on the parameters is presented. In Section 5, numerical simulations are carried out. Finally, the results and discussion of this paper are given.

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2 Model (1)

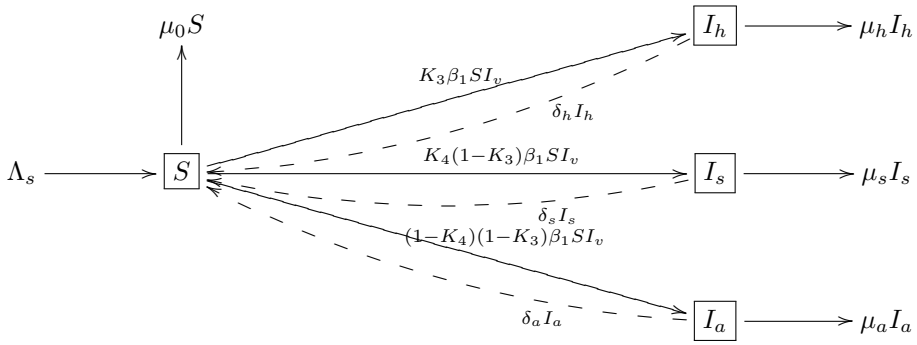

Figure 1: the schematic diagram to the $SI_hI_sI_aS$ model

This section focuses solely on viral respiratory infections, individual heterogeneity, and the impacts of air pollution at low levels. It ignores the effects of air pollution and does not take into account the allergic respiratory diseases brought on by exposure to air pollution. Susceptible individuals (S) become infectious with viral respiratory disease through contact with patients with viral respiratory disease (I_v) (specifically, patients with consultation (I_h); symptomatic patients without consultation (I_s); and asymptomatic patients without consultation (I_a)). The $SI_hI_sI_aS$ multi-cluster infectious disease model shown below is created based on the schematic diagram in Figure 1.

$$\begin{cases} \dfrac{dS}{dt} = \Lambda_s - \mu_0 S - \beta_1 S(I_h + I_s + I_a) + \delta_h I_h + \delta_s I_s + \delta_a I_a, \\ \dfrac{dI_h}{dt} = K_3\beta_1 S(I_h + I_s + I_a) - \delta_h I_h - \mu_h I_h, \\ \dfrac{dI_s}{dt} = K_4(1-K_3)\beta_1 S(I_h + I_s + I_a) - \delta_s I_s - \mu_s I_s, \\ \dfrac{dI_a}{dt} = (1-K_4)(1-K_3)\beta_1 S(I_h + I_s + I_a) - \delta_a I_a - \mu_a I_a, \end{cases} \tag{1}$$

where Λ_s is the recruitment rate of susceptible persons, μ_0 is the natural mortality rate of susceptible persons,μ_h, μ_s, μ_a are the total morality rate of I_h ,I_s and I_a respectively,β_1 is the infection rate of viral patients to persons who are not affected by air pollution, $\delta_h, \delta_s, \delta_a$ are the cure rate of I_h ,I_s and I_a respectively, the combination of K_i(i=3,4) represents the proportion of susceptible persons transformed into different types of patients. It's reasonable to assume that $\mu_0 < \min\{\mu_h, \mu_s, \mu_a\}$. All parameters are nonnegative constants.

It is evident that the right hand of system (1) is continuous with respect to the variables, satisfying the existence of the solutions. It is easy to get that the solutions of system (1) with respect to the initial value $S(0) > 0, I_h \geq 0, I_s \geq 0, I_a \geq 0$ are positive for all $t > 0$, and they are all uniformly bounded on

$$D = \{(S, I_h, I_s, I_a) \in R_+^4 : 0 \leq S + I_h + I_s + I_a = N \leq \tfrac{\Lambda_s}{\mu_0}\}.$$

2.1 The basic reproduction number and equilibria of the system (1)

By a straightforward calculation, we can obtain the disease-free equilibrium of system (1), which is given by $E_0^* = (\frac{\Lambda_s}{\mu_0}, 0, 0, 0)$.

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System (1) has three infected compartments I_h, I_s and I_a. According to the definition and calculation method of Van Den Driessche and Watmough[16], we get the basic reproduction number of system (1) is given by

$$R_0 = \frac{\beta_1 \Lambda_s}{\mu_0}[\frac{K_3}{\mu_h+\delta_h} + \frac{K_4(1-K_3)}{\mu_s+\delta_s} + \frac{(1-K_4)(1-K_3)}{\mu_a+\delta_a}].$$

Denote

$$R_{0h} = \frac{\beta_1 \Lambda_s K_3}{\mu_0(\mu_h+\delta_h)}, R_{0s} = \frac{\beta_1 \Lambda_s K_4(1-K_3)}{\mu_0(\mu_s+\delta_s)}, R_{0a} = \frac{\beta_1 \Lambda_s (1-K_4)(1-K_3)}{\mu_0(\mu_a+\delta_a)}.$$

Here, each element has its own biological significance. The threshold R_0 indicates the average number of second-generation infections caused by a patient in a susceptible population during its infectious period. R_{0h} shows the number of second-generation infections in the susceptible population caused by the patient attending the clinic during its period of illness. R_{0s} represents the transmission from a symptomatic patient who was not seen during its period of illness. R_{0a} represents the transmission from an asymptomatic patient who was not seen during its period of illness.

Next, the existence of equilibria of system (1) is given.

Theorem 2.1 *For system* (1)*, we have:*

(1)When $R_0 \leq 1$*, system* (1) *has only one disease-free equilibrium* $E_0^* = (\frac{\Lambda_s}{\mu_0}, 0, 0, 0)$.

(2)When $R_0 > 1$*, system* (1) *has a unique endemic equilibrium* $E_1^* = (S^*, I_h^*, I_s^*, I_a^*)$ *except for* E_0^**, here*

$$S^* = \frac{\Lambda_s}{\mu_0 R_0}, I_h^* = \frac{\Lambda_s(R_0-1)R_{0h}}{R_0(\mu_h R_{0h}+\mu_s R_{0s}+\mu_a R_{0a})}, I_s^* = \frac{\Lambda_s(R_0-1)R_{0s}}{R_0(\mu_h R_{0h}+\mu_s R_{0s}+\mu_a R_{0a})}, I_a^* = \frac{\Lambda_s(R_0-1)R_{0a}}{R_0(\mu_h R_{0h}+\mu_s R_{0s}+\mu_a R_{0a})}.$$

Proof *By adding the second, third and fourth equations of system* (1)*, we can obtain* $S = \frac{(\mu_h+\delta_h)I_h+(\mu_s+\delta_s)I_s+(\mu_a+\delta_a)I_a}{\beta_1(I_h+I_s+I_a)}$.

Substituting the above equation into system (1) *yields*

$$C_1 I_h^2 + C_2 I_h = 0,$$

where $C_1 = -\frac{\beta_1 R_0}{R_{0h}^2}(\mu_h R_{0h} + \mu_s R_{0s} + \mu_a R_{0a})$, $C_2 = \frac{\Lambda_s \beta_1 (R_0-1)}{R_{0h}}$.

In addition to the disease-free equilibrium $E_0^* = (\frac{\Lambda_s}{\mu_0}, 0, 0, 0)$*, when the aforementioned equation is solved, there are* $S^* = \frac{\Lambda_s}{\mu_0 R_0}, I_h^* = \frac{\Lambda_s(R_0-1)R_{0h}}{R_0(\mu_h R_{0h}+\mu_s R_{0s}+\mu_a R_{0a})}, I_s^* = \frac{\Lambda_s(R_0-1)R_{0s}}{R_0(\mu_h R_{0h}+\mu_s R_{0s}+\mu_a R_{0a})}, I_a^* = \frac{\Lambda_s(R_0-1)R_{0a}}{R_0(\mu_h R_{0h}+\mu_s R_{0s}+\mu_a R_{0a})}$.

Denote $E_1^* = (S^*, I_h^*, I_s^*, I_a^*)$*, then if and only if* $R_0 > 1$*, there are* $S^* > 0, I_h^* > 0, I_s^* > 0, I_a^* > 0$*, that is, the endemic equilibrium* E_1^* *exists.*

2.2 Stability of disease-free equilibrium E_0^*

2.2.1 Local stability

First, we prove the local stability of the disease-free equilibrium. As a result, the following is the outcome.

Theorem 2.2 *For system* (1)*, if* $R_0 < 1$*, the disease-free equilibrium* E_0^* *is locally asymptotically stable; it is unstable when* $R_0 > 1$.

Proof *The corresponding characteristic equation is*

$$det(\lambda I - J(E_0^*)) = (\lambda + \mu_0)(\lambda^3 + b_1\lambda^2 + b_2\lambda + b_3),$$

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where

$b_1 = (\mu_h + \delta_h) + (\mu_s + \delta_s) + (\mu_a + \delta_a) - \frac{\beta_1 \Lambda_s}{\mu_0}$,

$b_2 = (\mu_h + \delta_h)(\mu_s + \delta_s) + (\mu_h + \delta_h)(\mu_a + \delta_a) + (\mu_s + \delta_s)(\mu_a + \delta_a) - \frac{\beta_1 \Lambda_s}{\mu_0}[(\mu_h + \delta_h)(1 - K_3) + (\mu_s + \delta_s)(1 - K_4(1 - K_3)) + (\mu_a + \delta_a)(1 - (1 - K_4)(1 - K_3))]$,

$b_3 = (\mu_h + \delta_h)(\mu_s + \delta_s)(\mu_a + \delta_a) - \frac{\beta_1 \Lambda_s}{\mu_0}[K_3(\mu_s + \delta_s)(\mu_a + \delta_a) + K_4(1 - K_3)(\mu_h + \delta_h)(\mu_a + \delta_a) + (1 - K_4)(1 - K_3)(\mu_h + \delta_h)(\mu_s + \delta_s)]$.

And $b_3 > 0, (\mu_h + \delta_h)(\mu_s + \delta_s)(\mu_a + \delta_a)(1 - R_0) > 0, R_0 < 1$. $b_1 > 0$ *is equal to* $(\mu_h + \delta_h)(1 - R_{0h}) + (\mu_s + \delta_s)(1 - R_{0s}) + (\mu_a + \delta_a)(1 - R_{0a}) > 0$. *If* $R_0 < 1$, *then obviously there are* $R_{0h} < 1, R_{0s} < 1, R_{0a} < 1$. *When* $b_3 > 0$, *i.e.,* $R_0 < 1$, *the computation reveals that* $b_2 > 0, b_1 b_2 - b_3 > 0$.

According to the Routh-Hurwitz criterion, if $R_0 < 1$, *then the real part of all eigenvalues of* E_0^* *are negative. As a result,* E_0^* *is locally asymptotically stable.*

2.2.2 Global stability

The global stability of the disease-free equilibrium is given below.

Theorem 2.3 *If* $R_0 < 1$, *then the disease-free equilibrium* E_0^* *is globally asymptotically stable.*

Proof *We construct a Lyapunov function as follows*

$$V(t) = \frac{I_h(t)}{\mu_h + \delta_h} + \frac{I_s(t)}{\mu_s + \delta_s} + \frac{I_a(t)}{\mu_a + \delta_a}.$$

Calculating the derivative of V(t) along the solutions of system (1) *yields*

$$\begin{aligned}
\frac{dV}{dt} &= \frac{1}{\mu_h + \delta_h}\frac{dI_h}{dt} + \frac{1}{\mu_s + \delta_s}\frac{dI_s}{dt} + \frac{1}{\mu_a + \delta_a}\frac{dI_a}{dt} \\
&= \frac{1}{\mu_h + \delta_h}[K_3\beta_1 S(I_h + I_s + I_a) - (\delta_h + \mu_h)I_h] + \frac{1}{\mu_s + \delta_s}[K_4(1 - K_3)\beta_1 S(I_h + I_s + I_a) - (\delta_s + \mu_s)I_s] \\
&\quad + \frac{1}{\mu_a + \delta_a}[(1 - K_4)(1 - K_3)\beta_1 S(I_h + I_s + I_a) - (\delta_a + \mu_a)I_a] \\
&\leq I_v(R_0 - 1).
\end{aligned}$$

Therefore,when $R_0 < 1$, *we have* $\frac{dV}{dt} < 0$. *And* $\frac{dV}{dt} = 0$ *if and only if* $I_h = 0, I_s = 0, I_a = 0$. *The disease-free equilibrium* E_0^* *is globally asymptotically stable according to the LaSalle invariant set principle.*

2.3 Stability of endemic equilibrium E_1^*

2.3.1 Local stability

Theorem 2.4 *For system* (1), *if* $R_0 > 1$, *the endemic equilibrium* E_1^* *is locally asymptotically stable.*

Proof *The characteristic equation of Jacobian matrix of* E_1^* *is*

$$\lambda^4 + c_1\lambda^3 + c_2\lambda^2 + c_3\lambda + c_4 = 0,$$

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where

$$
\begin{aligned}
c_1 &= \mu_0+\beta_1 I_v^*+\frac{(\mu_h+\delta_h)(R_{0s}+R_{0a})}{R_0}+\frac{(\mu_s+\delta_s)(R_{0h}+R_{0a})}{R_0}+\frac{(\mu_a+\delta_a)(R_{0h}+R_{0s})}{R_0}>0,\\
c_2 &= \frac{\mu_0}{R_0}[(\mu_h+\delta_h)(R_{0s}+R_{0a})+(\mu_s+\delta_s)(R_{0h}+R_{0a})+(\mu_a+\delta_a)(R_{0h}+R_{0s})]+\beta_1 I_v^*[\mu_h+\mu_s+\mu_a+\delta_h(1-K_3)\\
&\quad +\delta_s(1-K_4(1-K_3))+\delta_a(1-(1-K_4)(1-K_3))]+(\mu_h+\delta_h)(\mu_s+\delta_s)+(\mu_h+\delta_h)(\mu_a+\delta_a)+(\mu_s+\delta_s)(\mu_a+\delta_a)\\
&\quad -\frac{\beta_1\Lambda_s}{\mu_0 R_0}[(\mu_h+\delta_h)(1-K_3)+(\mu_s+\delta_s)(1-K_4(1-K_3))+(\mu_a+\delta_a)(1-(1-K_4)(1-K_3))],\\
c_3 &= \mu_0\{(\mu_h+\delta_h)(\mu_s+\delta_s)+(\mu_h+\delta_h)(\mu_a+\delta_a)+(\mu_s+\delta_s)(\mu_a+\delta_a)-\frac{\beta_1\Lambda_s}{\mu_0 R_0}[(\mu_h+\delta_h)(1-K_3)+(\mu_s+\delta_s)\\
&\quad (1-K_4(1-K_3))+(\mu_a+\delta_a)(1-(1-K_4)(1-K_3))]\}+\beta_1 I_v^*[K_3(\mu_s+\delta_s)(\mu_a+\delta_a)+K_4(1-K_3)\mu_s(\mu_a+\delta_a)\\
&\quad +(1-K_4)(1-K_3)\mu_a(\mu_s+\delta_s)+K_3\mu_h(\mu_a+\delta_a)+K_4(1-K_3)(\mu_h+\delta_h)(\mu_a+\delta_a)+(1-K_4)(1-K_3)\mu_a(\mu_h+\delta_h)\\
&\quad +K_3\mu_h(\mu_s+\delta_s)+K_4(1-K_3)\mu_s(\mu_h+\delta_h)+(1-K_4)(1-K_3)(\mu_h+\delta_h)(\mu_s+\delta_s)],\\
c_4 &= \beta_1\Lambda_s(\mu_h+\delta_h)(\mu_s+\delta_s)(\mu_a+\delta_a)(R_0-1).
\end{aligned}
$$

When $R_0>1$, there is naturally $c_4>0$. To prove that $c_2>0, c_3>0$, we simply prove that $(\mu_h+\delta_h)(\mu_s+\delta_s)+(\mu_h+\delta_h)(\mu_a+\delta_a)+(\mu_s+\delta_s)(\mu_a+\delta_a)-\frac{\beta_1\Lambda_s}{\mu_0 R_0}[(\mu_h+\delta_h)(1-K_3)+(\mu_s+\delta_s)(1-K_4(1-K_3))+(\mu_a+\delta_a)(1-(1-K_4)(1-K_3))]>0$.

Let

$$
H_1=c_1, H_2=\begin{vmatrix} c_1 & c_3\\ 1 & c_2\end{vmatrix}, H_3=\begin{vmatrix} c_1 & c_3 & 0\\ 1 & c_2 & c_4\\ 0 & c_1 & c_3\end{vmatrix}, H_4=\begin{vmatrix} c_1 & c_3 & 0 & 0\\ 1 & c_2 & c_4 & 0\\ 0 & c_1 & c_3 & 0\\ 0 & 1 & c_2 & c_4\end{vmatrix}.
$$

When $R_0>1$, it is easy to get that $H_1=c_1>0, H_2=c_1c_2-c_3>0, H_3=c_3H_2-c_1^2c_4>0, H_4=H_3c_4>0$. According to the Routh-Hurwitz criterion, if $R_0>1$, then the real part of all eigenvalues of E_1^* are negative. Therefore, E_1^* is locally asymptotically stable.

2.3.2 Global stability

By building a Lyapunov function, we analyze the global stability of the endemic equilibrium E_1^* in this section under a particular set of circumstances. The conclusion is given below.

Theorem 2.5 *Assume that $\delta=\delta_h=\delta_s=\delta_a$. If $R_0>1$, then the endemic equilibrium E_1^* is globally asymptotically stable.*

Proof *For convenience, we note $I_h=I_1, I_s=I_2, I_a=I_3, K_3=p_1, K_4(1-K_3)=p_2, (1-K_4)(1-K_3)=p_3$.*

Substituting E_1^* into system (1) yields

$$
\begin{cases}
\Lambda_s-\mu_0 S^*-\beta_1 S^* I_v^*+\sum_{i=1}^{3}\delta I_i^*=0,\\
p_i\beta_1 S^* I_v^*=(\mu_i+\delta)I_i^*(i=1,2,3),
\end{cases}
\quad \text{then } \sum_{i=1}^{3}\frac{p_i\beta_1}{\mu_i+\delta}=\frac{1}{S^*}.
$$

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And for $i = 1, 2, 3$, there is

$$\begin{aligned}
\frac{dI_i}{dt} &= p_i\beta_1 SI_v - (\mu_i + \delta)I_i \\
&= (\mu_i + \delta)(\frac{p_i\beta_1}{\mu_i + \delta}SI_v - I_i) \\
&= (\mu_i + \delta)[(\frac{1}{S^*} - \sum_{j\neq i}\frac{p_i\beta_1}{\mu_i + \delta})S(I_i + \sum_{j\neq i} I_j) - I_i] \\
&= (\mu_i + \delta)[(\frac{S}{S^*} - 1)I_i + S(\frac{1}{S^*}\sum_{j\neq i} I_j - \sum_{j\neq i}\frac{p_i\beta_1}{\mu_i + \delta}I_v)] \\
&= (\mu_i + \delta)(\frac{S}{S^*} - 1)I_i + S(\mu_i + \delta)\sum_{j\neq i}[\frac{p_j\beta_1 I_i^*}{\mu_j + \delta}\frac{I_i - I_i^*}{I_i}(\frac{I_j}{I_j^*} - \frac{I_i}{I_i^*})].
\end{aligned}$$

Firstly, let $V_1(t) = \frac{(S-S^*)^2}{2}$, then the derivative of $V_1(t)$ along system (1) is

$$\begin{aligned}
\frac{dV_1}{dt} &= (S - S^*)(\Lambda_s - \mu_0 S - \beta_1 SI_v + \sum_{i=1}^{3}\delta I_i) \\
&= -(\mu_0 + \beta_1 I_v)(S - S^*)^2 - (\beta_1 S^* - \delta)(S - S^*)(I_v - I_v^*).
\end{aligned}$$

Secondly, let $V_2(t) = S^*(\beta_1 S^* - \delta)\sum\limits_{i=1}^{3}\frac{1}{\mu_i+\delta}(I_i - I_i^* - I_i^*\ln\frac{I_i}{I_i^*})$, we have

$$\beta_1 S^* - \delta = \frac{\beta_1 S_0}{R_0} - \delta = \frac{1 - \delta[\frac{K_3}{\mu_h+\delta} + \frac{K_4(1-K_3)}{\mu_s+\delta} + \frac{(1-K_4)(1-K_3)}{\mu_a+\delta}]}{\frac{K_3}{\mu_h+\delta} + \frac{K_4(1-K_3)}{\mu_s+\delta} + \frac{(1-K_4)(1-K_3)}{\mu_a+\delta}} > 0,$$

so $V_2(t)$ is a positive definite function on D.

The derivative of $V_2(t)$ along system (1) is

$$\begin{aligned}
\frac{dV_2}{dt} &= S^*(\beta_1 S^* - \delta)\sum_{i=1}^{3}(1 - \frac{I_i^*}{I_i})\{(\frac{S}{S^*} - 1)I_i + S\sum_{j\neq i}[\frac{p_j\beta_1 I_i^*}{\mu_j + \delta}\frac{I_i - I_i^*}{I_i}(\frac{I_j}{I_j^*} - \frac{I_i}{I_i^*})]\} \\
&= (\beta_1 S^* - \delta)(S - S^*)(I_v - I_v^*) + SS^*(\beta_1 S^* - \delta)\sum_{i=1}^{3}\sum_{j\neq i} w_{ij},
\end{aligned}$$

where $w_{ij} = \frac{p_j\beta_1 I_i^*}{\mu_j+\delta}\frac{I_i-I_i^*}{I_i}(\frac{I_j}{I_j^*} - \frac{I_i}{I_i^*})$. We have

$$w_{ij} + w_{ji} = (\frac{p_j\beta_1 I_i^*}{\mu_j + \delta}\frac{I_i - I_i^*}{I_i} - \frac{p_i\beta_1 I_j^*}{\mu_i + \delta}\frac{I_j - I_j^*}{I_j})(\frac{I_j}{I_j^*} - \frac{I_i}{I_i^*}) = -\frac{p_j\beta_1 I_j^*(I_i^*)^2}{(\mu_j + \delta)I_i I_j}(\frac{I_j}{I_j^*} - \frac{I_i}{I_i^*})^2 < 0.$$

Finally, we construct a Lyapunov function $V(t) = V_1(t) + V_2(t)$, from the above analysis, we know that $V(t)$ is a positive definite function on D. And the total derivative of $V(t)$ along system (1) is

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$$\begin{aligned}\frac{dV}{dt} &= -(\mu_0+\beta_1 I_v)(S-S^*)^2-(\beta_1 S^*-\delta)(S-S^*)(I_v-I_v^*)+(\beta_1 S^*-\delta)(S-S^*)(I_v-I_v^*)+SS^*(\beta_1 S^*-\delta)\sum_{i=1}^{3}\sum_{j\neq i} w_{ij}\\ &= -(\mu_0+\beta_1 I_v)(S-S^*)^2-SS^*(\beta_1 S^*-\delta)\sum_{i,j=1,i<j}\frac{p_j\beta_1 I_j^*(I_i^*)^2}{(\mu_j+\delta)I_iI_j}(\frac{I_j}{I_j^*}-\frac{I_i}{I_i^*})^2.\end{aligned}$$

If $R_0>1$, we get $\frac{dV}{dt}<0$, and $\frac{dV}{dt}=0$ if and only if $S=S^*$, $\frac{I_j}{I_j^*}=\frac{I_i}{I_i^*}$, the maximum invariant set is $\{E_1^*\}$. According to LaSalle invariant set principle, the endemic equilibrium E_1^* is globally asymptotically stable.

3 Model (2)

3.1 Model Formulation and boundedness of solutions

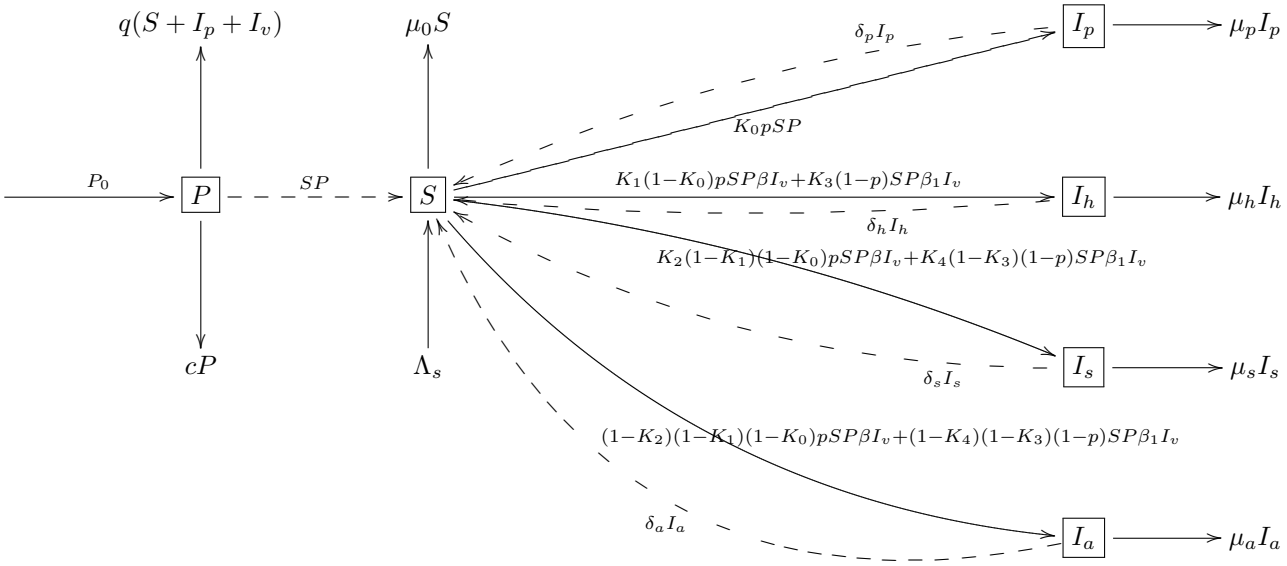

Figure 2: the schematic diagram of the $SI_pI_hI_sI_aSP$ model

When the level of air pollution is high, on the one hand, some susceptible individuals (K_0pSP) in S become allergic respiratory disease patients (I_p) by inhaling air pollutants; on the other hand, susceptible persons ($(1-K_0)pSP$) in S who inhaled air pollutants but did not experience allergic reactions may become infected with viral respiratory disease patients by interacting with viral respiratory disease patients (I_v) (especially divided into (I_h)for patients who have visited, symptomatic patients without consultation(I_s) and the asymptomatic patients without consultation (I_a)), while susceptible people ($(1-p)SP$) in S who are not affected by air pollution may also contract a viral respiratory illness through contact with sufferers I_v. Based on the schematic diagram depicted in Figure 2, the following $SI_pI_hI_sI_aSP$ infectious disease model is established.

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$$
\begin{cases}
\frac{dS}{dt} = \Lambda_s - \mu_0 S - K_0 pSP - (1-K_0)pSP\beta(I_h+I_s+I_a) - (1-p)SP\beta_1(I_h+I_s+I_a) + \delta_p I_p + \delta_h I_h + \delta_s I_s + \delta_a I_a, \\
\frac{dI_p}{dt} = K_0 pSP - \delta_p I_p - \mu_p I_p, \\
\frac{dI_h}{dt} = K_1(1-K_0)pSP\beta(I_h+I_s+I_a) + K_3(1-p)SP\beta_1(I_h+I_s+I_a) - \delta_h I_h - \mu_h I_h, \\
\frac{dI_s}{dt} = K_2(1-K_1)(1-K_0)pSP\beta(I_h+I_s+I_a) + K_4(1-K_3)(1-p)SP\beta_1(I_h+I_s+I_a) - \delta_s I_s - \mu_s I_s, \\
\frac{dI_a}{dt} = (1-K_2)(1-K_1)(1-K_0)pSP\beta(I_h+I_s+I_a) + (1-K_4)(1-K_3)(1-p)SP\beta_1(I_h+I_s+I_a) - \delta_a I_a - \mu_a I_a, \\
\frac{dP}{dt} = P_0 - cP - q(S+I_p+I_h+I_s+I_a),
\end{cases} \tag{2}
$$

where P represents air pollutant concentration, Λ_s is the recruitment rate of susceptible persons, μ_0 is the natural mortality rate of susceptible persons, p is the conversion rate of susceptible persons become individuals affected by air pollution, $\mu_p, \mu_h, \mu_s, \mu_a$ are the total morality rate of I_p, I_h , I_s and I_a respectively, β is the infection rate of viral patients to individuals affected by air pollution, β_1 is the infection rate of viral patients to persons who are not affected by air pollution, $\delta_p, \delta_h, \delta_s, \delta_a$ are the cure rate of I_p, I_h, I_s and I_a respectively, the combination of K_i(i=0,1,2,3,4) represents the proportion of susceptible persons transformed into different types of patients, P_0 is the daily emission of air pollutants, c is the clearance rate of air pollutants, q is the inhalation rate for air pollutants per person. It's reasonable to assume that $\mu_0 < \min\{\mu_p, \mu_h, \mu_s, \mu_a\}$. All parameters are nonnegative constants. It is evident that the right hand of system (2) is continuous with respect to the variables, satisfying the existence of the solutions.

Theorem 3.1 *The solutions of system* (2) *with respect to the initial value* $S(0) > 0, I_p \geq 0, I_h \geq 0, I_s \geq 0, I_a \geq 0, P(0) > 0$ *are positive for all* $t > 0$. *All solutions of system* (2) *are uniformly bounded on*

$$\Omega = \{(S, I_p, I_h, I_s, I_a, P) \in R_+^6 : 0 \leq S + I_p + I_h + I_s + I_a = N \leq \tfrac{\Lambda_s}{\mu_0}, 0 \leq P \leq \tfrac{P_0}{c}\}.$$

Proof *Let* $N(t) = S(t) + I_p(t) + I_h(t) + I_s(t) + I_a(t)$, *the derivative of* $N(t)$ *along the solution of system* (2) *is*

$$
\begin{aligned}
\frac{dN(t)}{dt} &= \frac{dS(t)}{dt} + \frac{dI_p(t)}{dt} + \frac{dI_h(t)}{dt} + \frac{dI_s(t)}{dt} + \frac{dI_a(t)}{dt} \\
&= \Lambda_s - \mu_0 S(t) - \mu_p I_p(t) - \mu_h I_h(t) - \mu_s I_s(t) - \mu_a I_a(t) \\
&\leq \Lambda_s - \mu_0 (S(t) + I_p(t) + I_h(t) + I_s(t) + I_a(t)) \\
&= \Lambda_s - \mu_0 N(t).
\end{aligned}
$$

Thus, we get $N(t) \leq \frac{\Lambda_s}{\mu_0} - (\frac{\Lambda_s}{\mu_0} - N(0))e^{-\mu_0 t}$ *for all* $t \geq 0$. *Therefore,* $lim_{t\to\infty} supN(t) \leq \frac{\Lambda_s}{\mu_0}$.

From the sixth equation of system (2)

$$\tfrac{dP(t)}{dt} = P_0 - cP(t) - q(S(t) + I_p(t) + I_h(t) + I_s(t) + I_a(t)) \leq P_0 - cP(t),$$

there is $P(t) \leq \frac{P_0}{c} - (\frac{P_0}{c} - P(0))e^{-ct}$ *for all* $t \geq 0$. *So,* $lim_{t\to\infty} supP(t) \leq \frac{P_0}{c}$.

To sum up, the positive invariant set of system (2) *is*

$$\Omega = \{(S, I_p, I_h, I_s, I_a, P) \in R_+^6 : 0 \leq S + I_p + I_h + I_s + I_a = N \leq \tfrac{\Lambda_s}{\mu_0}, 0 \leq P \leq \tfrac{P_0}{c}\}.$$

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3.2 Existence and stability of equilibria

3.2.1 Existence and stability of disease-free equilibrium

Theorem 3.2 *For system (2), there exists a disease-free equilibrium* $E_0 = (S_0, 0, 0, 0, 0, 0)$ *if* $q = q_1$, *here* $S_0 = \frac{\Lambda_s}{\mu_0} = \frac{P_0}{q}$, $q_1 = \frac{\mu_0 P_0}{\Lambda_s}$.

Theorem 3.3 *For system (2), if* $q = q_1$, $R_1 = \frac{P_0 K_0 p}{c(\mu_p+\delta_p)}(\frac{\mu_p}{\mu_0} - 1) < 1$, *the disease-free equilibrium* E_0 *is locally asymptotically stable.*

Proof *The corresponding characteristic equation is*

$$det(\lambda I - J(E_0)) = (\lambda + \mu_h + \delta_h)(\lambda + \mu_s + \delta_s)(\lambda + \mu_a + \delta_a)(\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3),$$

where

$a_1 = c + \mu_0 + \mu_p + \delta_p > 0$, $a_2 = c\mu_0 + (c + \mu_0)(\mu_p + \delta_p) > 0$, $a_3 = c\mu_0(\mu_p + \delta_p) + \Lambda_s K_0 pq(1 - \frac{\mu_p}{\mu_0})$.

Obviously, $\lambda_1 = -(\mu_h + \delta_h) < 0, \lambda_2 = -(\mu_s + \delta_s) < 0, \lambda_3 = -(\mu_a + \delta_a) < 0$, *the remaining characteristic roots are given by* $\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$. *When* $c\mu_0(\mu_p + \delta_p) + \Lambda_s K_0 pq(1 - \frac{\mu_p}{\mu_0}) > 0$, *that* $\frac{\Lambda_s K_0 pq}{c\mu_0(\mu_p+\delta_p)}(\frac{\mu_p}{\mu_0} - 1) < 1$, *we have* $a_3 > 0$.

Denote $R_1 = \frac{P_0 K_0 p}{c(\mu_p+\delta_p)}(\frac{\mu_p}{\mu_0} - 1)$. *Thus,* $R_1 = \frac{\Lambda_s K_0 pq}{c\mu_0(\mu_p+\delta_p)} \frac{\mu_p - \mu_0}{\mu_0}$ *when* $q = q_1$. *Here,* $\frac{1}{\mu_0}$ *represents the average life span of the population,* $\frac{1}{\mu_p+\delta_p}$ *represents the average period of infection of allergic respiratory disease patients,* $\mu_p - \mu_0$ *represents the case fatality rate for allergic respiratory disease. In fact,* R_1 *represents the number of disease-related fatalities in the new generation of patients with allergic respiratory diseases.*

And we have $a_1 a_2 - a_3 = c\mu_0(c + \mu_0) + (c + \mu_0)(\mu_p + \delta_p)(c + \mu_0 + \mu_p + \delta_p) + \Lambda_s K_0 pq(\frac{\mu_p}{\mu_0} - 1) > 0$.

According to Routh-Hurwitz criterion, the real part of all eigenvalues of E_0 *are negative when* $R_1 < 1$. *Therefore,* E_0 *is locally asymptotically stable.*

Lemma 3.1 *There is no periodic solution for system (2).*

Proof *Let* $X = (S, I_p, I_h, I_s, I_a, P)$. *By constructing a Dulac function* $G = \frac{1}{S(I_h+I_s+I_a)} = \frac{1}{SI_v}$, *we have*

$G\frac{dS}{dt} = \frac{\Lambda_s}{SI_v} - \frac{\mu_0}{I_v} - \frac{K_0 pP}{I_v} - (1 - K_0)p\beta P - (1 - p)\beta_1 P + \frac{\delta_p I_p + \delta_h I_h + \delta_s I_s + \delta_a I_a}{SI_v}$,

$G\frac{dI_p}{dt} = \frac{K_0 pP}{I_v} - \frac{(\delta_p+\mu_p)I_p}{SI_v}$,

$G\frac{dI_h}{dt} = K_1(1 - K_0)pP\beta + K_3(1 - p)P\beta_1 - \frac{(\delta_h+\mu_h)I_h}{SI_v}$,

$G\frac{dI_s}{dt} = K_2(1 - K_1)(1 - K_0)pP\beta + K_4(1 - K_3)(1 - p)P\beta_1 - \frac{(\delta_s+\mu_s)I_s}{SI_v}$,

$G\frac{dI_a}{dt} = (1 - K_2)(1 - K_1)(1 - K_0)pP\beta + (1 - K_4)(1 - K_3)(1 - p)P\beta_1 - \frac{(\delta_a+\mu_a)I_a}{SI_v}$,

$G\frac{dP}{dt} = \frac{P_0}{SI_v} - \frac{cP}{SI_v} - \frac{q(S+I_p+I_v)}{SI_v}$.

Further, there are

$$\begin{aligned}
\frac{dGX}{dt} &= \frac{\partial}{\partial S}(G\frac{dS}{dt}) + \frac{\partial}{\partial I_p}(G\frac{dI_p}{dt}) + \frac{\partial}{\partial I_h}(G\frac{dI_h}{dt}) + \frac{\partial}{\partial I_s}(G\frac{dI_s}{dt}) + \frac{\partial}{\partial I_a}(G\frac{dI_a}{dt}) + \frac{\partial}{\partial P}(G\frac{dP}{dt}) \\
&= -\frac{1}{SI_v}[\frac{1}{S} + \frac{\delta_p I_p + \delta_h I_h + \delta_s I_s + \delta_a I_a}{S} + (\delta_p + \mu_p) + \frac{1}{I_v}((\delta_h + \mu_h)(I_s + I_a) + (\delta_s + \mu_s)(I_h + I_a) \\
&\quad +(\delta_a + \mu_a)(I_h + I_s)) + c] < 0.
\end{aligned}$$

Thus, there is no periodic solution for system (2).

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Meanwhile, Ω is the positive invariant set of system (2), the following theorem can be deduced from *Poincarè – Bendixson* theorem [28].

Theorem 3.4 *For system* (2)*, if* $q = q_1$*,* $R_1 = \frac{P_0 K_0 p}{c(\mu_p+\delta_p)}(\frac{\mu_p}{\mu_0} - 1) < 1$*, the disease-free equilibrium* E_0 *is globally asymptotically stable.*

Proof *According to Theorem 3.3, when* $q = q_1$ *and* $R_1 = \frac{P_0 K_0 p}{c(\mu_p+\delta_p)}(\frac{\mu_p}{\mu_0} - 1) < 1$*, disease-free equilibrium* E_0 *is locally asymptotically stable. Further, we know that there is no periodic solution for system* (2) *from Lemma 3.1. Hence, all trajectories in region* Ω *approach* E_0*, as* $t \to \infty$*. That is,* E_0 *is globally asymptotically stable.*

3.2.2 Existence and stability of boundary equilibria

Theorem 3.5 *For system* (2)*, we have:*

(1)There is a unique boundary equilibrium $E_1 = (S_1^*, I_{p1}^*, 0, 0, 0, P_1^*)$ *when* $0 < q < q_1$*,where*

$$S_1^* = \frac{\Lambda_s - \mu_p I_{p1}^*}{\mu_0}, P_1^* = \frac{\mu_0 P_0 - q\Lambda_s + q(\mu_p - \mu_0) I_{p1}^*}{c\mu_0}, I_{p1}^* = \frac{-A_2 - \sqrt{\Delta_1}}{2A_1} > 0.$$

(2)There is a unique boundary equilibrium $E_2 = (S_2^*, I_{p2}^*, 0, 0, 0, P_2^*)$ *if* $q = q_1$ *and* $R_1 = \frac{P_0 K_0 p}{c(\mu_p+\delta_p)}(\frac{\mu_p}{\mu_0} - 1) > 1$*,where*

$$S_2^* = \frac{\Lambda_s - \mu_p I_{p2}^*}{\mu_0} = \frac{\Lambda_s}{\mu_0 R_1}, P_2^* = \frac{\mu_0 P_0 - q\Lambda_s + q(\mu_p - \mu_0) I_{p2}^*}{c\mu_0} = \frac{P_0(\mu_p - \mu_0)(R_1 - 1)}{c\mu_p R_1}, I_{p2}^* = \frac{-A_2}{A_1} = \frac{\Lambda_s}{\mu_p}(1 - \frac{1}{R_1}) > 0.$$

(3)There is a unique boundary equilibrium $E_3 = (S_3^*, I_{p3}^*, 0, 0, 0, P_3^*)$ *if* $q = q_3$ *and* $R_1 = \frac{P_0 K_0 p}{c(\mu_p+\delta_p)}(\frac{\mu_p}{\mu_0} - 1) > 1$*,where*

$$S_3^* = \frac{\Lambda_s - \mu_p I_{p3}^*}{\mu_0}, P_3^* = \frac{\mu_0 P_0 - q\Lambda_s + q(\mu_p - \mu_0) I_{p3}^*}{c\mu_0}, I_{p3}^* = \frac{-A_2}{2A_1} > 0.$$

(4)There are two boundary equilibria $E_{31} = (S_{31}^*, I_{p31}^*, 0, 0, 0, P_{31}^*)$*, and* $E_{32} = (S_{32}^*, I_{p32}^*, 0, 0, 0, P_{32}^*)$*, if* $q_1 < q < q_3$ *and* $R_1 = \frac{P_0 K_0 p}{c(\mu_p+\delta_p)}(\frac{\mu_p}{\mu_0} - 1) > 1$*,where*

$$S_{31}^* = \frac{\Lambda_s - \mu_p I_{p31}^*}{\mu_0}, P_{31}^* = \frac{\mu_0 P_0 - q\Lambda_s + q(\mu_p - \mu_0) I_{p31}^*}{c\mu_0}, S_{32}^* = \frac{\Lambda_s - \mu_p I_{p32}^*}{\mu_0}, P_{32}^* = \frac{\mu_0 P_0 - q\Lambda_s + q(\mu_p - \mu_0) I_{p32}^*}{c\mu_0}$$

$$I_{p31}^* = \frac{-A_2 - \sqrt{\Delta_1}}{2A_1} > I_{p32}^* = \frac{-A_2 + \sqrt{\Delta_1}}{2A_1} > 0.$$

Here, $q_3 = \frac{1}{\Lambda_s K_0 p}\{c(\mu_p+\delta_p)(2\mu_p - \mu_0) + K_0 p \mu_p P_0 - 2\sqrt{c\mu_p(\mu_p+\delta_p)(\mu_p - \mu_0)[c(\mu_p+\delta_p) + K_0 p P_0]}\} > 0$*,* $A_1 = -q\mu_p(\frac{\mu_p}{\mu_0} - 1) < 0$*,* $A_2 = q\Lambda_s(\frac{2\mu_p}{\mu_0} - 1) - \mu_p P_0 - \frac{c\mu_0(\mu_p+\delta_p)}{K_0 p}$*,* $A_3 = \Lambda_s(P_0 - \frac{q\Lambda_s}{\mu_0})$*,* $\Delta_1 = A_2^2 - 4A_1 A_3$*.*

Proof *Let the right side of system* (2) *be zero, we can obtain that*

(1)If $I_p = 0$*,then* $P = 0, I_h + I_s + I_a = 0$*, and* $S = \frac{\Lambda_s}{\mu_0} = \frac{P_0}{q}$*, that is the disease-free equilibrium* E_0 *of system* (2)*.*

(2)$I_h + I_s + I_a = 0$ *or* $P = 0$ *if at least one of* I_h, I_s*, and* I_a *is zero. The disease-free equilibrium is now reached, assuming* $I_p = 0$*.*

Therefore, we consider the case $I_h + I_s + I_a = 0$ *and* $I_p \neq 0, P \neq 0$ *in the following.*

We have $S = \frac{\Lambda_s - \mu_p I_p}{\mu_0}, P = \frac{\mu_0 P_0 - q\Lambda_s + q(\mu_p - \mu_0) I_p}{c\mu_0}$*, these two expressions are substituted into the first equation of the system* (2) *to produce*

$$f(I_p) = A_1 I_p^2 + A_2 I_p + A_3 = 0, \tag{3}$$

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where

$A_1 = -q\mu_p(\frac{\mu_p}{\mu_0} - 1) < 0$, $A_2 = q\Lambda_s(\frac{2\mu_p}{\mu_0} - 1) - \mu_p P_0 - \frac{c\mu_0(\mu_p+\delta_p)}{K_0 p}$, $A_3 = \Lambda_s(P_0 - \frac{q\Lambda_s}{\mu_0})$, $\Delta_1 = A_2^2 - 4A_1A_3$.

To take the existence of the boundary equilibrium $E^* = (S^*, I_p^*, 0, 0, 0, P^*)$, *then* S^*, P^* *must be positive, and the roots* I_p^* *of the quadratic equation* (3) *must also be positive. Therefore, the quadratic equation* (3) *has no real roots if* $\Delta_1 < 0$. *The quadratic equation* (3) *has one real root* $I_{p3}^* = \frac{-A_2}{2A_1}$ *when* $\Delta_1 = 0$. *The quadratic equation* (3) *has two different real roots* $I_p^* = \frac{-A_2 \pm \sqrt{\Delta_1}}{2A_1}$ *if* $\Delta_1 > 0$.

From what has been discussed above,we can draw the following conclusion:

(i)When $A_3 > 0$, *that is* $0 < q < q_1$, *from the relationship between roots and coefficients of a quadratic equation,* $I_{p1}^* I_{p2}^* = \frac{A_3}{A_1} < 0$, *we have* $I_{p1}^* = \frac{-A_2 - \sqrt{\Delta_1}}{2A_1} > 0 > I_{p2}^* = \frac{-A_2 + \sqrt{\Delta_1}}{2A_1}$, *hence, there is a unique boundary equilibrium* $E_1 = (S_1^*, I_{p1}^*, 0, 0, 0, P_1^*)$, *if* $\Delta_1 > 0, \Lambda_s - \mu_p I_{p1}^* > 0, \mu_0 P_0 - q\Lambda_s + q(\mu_p - \mu_0) I_{p1}^* > 0$, *here* $I_{p1}^* = \frac{-A_2 - \sqrt{\Delta_1}}{2A_1}$. *And we know* $\Delta_1 > 0, \Lambda_s - \mu_p I_{p1}^* > 0, \mu_0 P_0 - q\Lambda_s + q(\mu_p - \mu_0) I_{p1}^* > 0$ *are always true from* $0 < q < q_1$. *As a result, system* (2) *has a single boundary equilibrium* E_1 *when* $0 < q < q_1$.

(ii)When $A_3 = 0$, *that is* $q = q_1$, *we have*

$A_2 = q\Lambda_s(\frac{2\mu_p}{\mu_0} - 1) - \mu_p P_0 - \frac{c\mu_0(\mu_p+\delta_p)}{K_0 p} = (\mu_p - \mu_0)P_0 - \frac{c\mu_0(\mu_p+\delta_p)}{K_0 p} = (\mu_p - \mu_0)P_0[1 - \frac{c\mu_0(\mu_p+\delta_p)}{(\mu_p-\mu_0)P_0 K_0 p}] = (\mu_p - \mu_0)P_0(1 - \frac{1}{R_1})$.

The two scenarios below are discussed:

(a)If $A_2 < 0$, *that is* $R_1 < 1$, *equation* (3) *has no positive roots and there is only a disease-free equilibrium* E_0.

(b)If $A_2 > 0$, *that is* $R_1 > 1$, *there is a unique positive root* $I_{p2}^* = \frac{-A_2}{A_1} = \frac{\Lambda_s}{\mu_p}(1 - \frac{1}{R_1})$ *for equation* (3). *At this time,* $S_2^* = \frac{\Lambda_s - \mu_p I_{p2}^*}{\mu_0} = \frac{\Lambda_s}{\mu_0 R_1}, P_2^* = \frac{\mu_0 P_0 - q\Lambda_s + q(\mu_p - \mu_0) I_{p2}^*}{c\mu_0} = \frac{P_0(\mu_p - \mu_0)(R_1 - 1)}{c\mu_p R_1}$. *We get* $\Lambda_s - \mu_p I_{p2}^* > 0, P_0(\mu_p - \mu_0)(R_1 - 1) > 0$ *from* $q = q_1$ *and* $R_1 > 1$, *hence, system* (2) *has one boundary equilibrium* E_2.

(iii)When $A_3 < 0$, *that is* $q > q_1$, *there are two positive roots* $I_p^* = \frac{-A_2 \pm \sqrt{\Delta_1}}{2A_1}$ *for equation* (3) *if* $A_2 > 0, \Delta_1 \geq 0$. *From* $A_2 > 0$, *one has* $q > \frac{\mu_0 P_0}{\Lambda_s}[\frac{\mu_p}{2\mu_p - \mu_0} + \frac{c\mu_0(\mu_p+\delta_p)}{K_0 p P_0(2\mu_p - \mu_0)}] = q_2$; *we have*

$\Delta_1 = A_2^2 - 4A_1A_3 = \Lambda_s^2 q^2 - 2\Lambda_s[\mu_p P_0 + \frac{c(\mu_p+\delta_p)(2\mu_p-\mu_0)}{K_0 p}]q + [\mu_p P_0 + \frac{c\mu_0(\mu_p+\delta_p)}{K_0 p}]^2$. *Due to* $\Lambda_s^2 > 0, 2\Lambda_s[\mu_p P_0 + \frac{c(\mu_p+\delta_p)(2\mu_p-\mu_0)}{K_0 p}] > 0, [\mu_p P_0 + \frac{c\mu_0(\mu_p+\delta_p)}{K_0 p}]^2 > 0$, *equation* $\Delta_1 \geq 0$ *has two positive roots* q_3 *and* q_4,

$$\begin{cases} 0 < q \leq \dfrac{1}{\Lambda_s K_0 p}\{c(\mu_p+\delta_p)(2\mu_p-\mu_0) + K_0 p\mu_p P_0 - 2\sqrt{c\mu_p(\mu_p+\delta_p)(\mu_p-\mu_0)[c(\mu_p+\delta_p) + K_0 p P_0]}\} = q_3, \\ q \geq \dfrac{1}{\Lambda_s K_0 p}\{c(\mu_p+\delta_p)(2\mu_p-\mu_0) + K_0 p\mu_p P_0 + 2\sqrt{c\mu_p(\mu_p+\delta_p)(\mu_p-\mu_0)[c(\mu_p+\delta_p) + K_0 p P_0]}\} = q_4. \end{cases}$$

We calculate the size of q_1, q_2, q_3, *and* q_4 *in the following.*

Assume $q_1 > q_3$, *that is*

$\frac{\mu_0 P_0}{\Lambda_s} < \frac{1}{\Lambda_s K_0 p}\{c(\mu_p+\delta_p)(2\mu_p-\mu_0) + K_0 p\mu_p P_0 - 2\sqrt{c\mu_p(\mu_p+\delta_p)(\mu_p-\mu_0)[c(\mu_p+\delta_p) + K_0 p P_0]}\}$,

$[K_0 p P_0(\mu_p - \mu_0) - c\mu_0(\mu_p+\delta_p)]^2 < 0$, *this contradicts the fact that the squared term is non-negative, so* $q_1 < q_3$.

Assume $q_2 > q_4$, *that is*

$2K_0 p\mu_p P_0(\mu_p - \mu_0) + 4c\mu_p(\mu_p+\delta_p)(\mu_p-\mu_0) + 2(2\mu_p - \mu_0)\sqrt{c\mu_p(\mu_p+\delta_p)(\mu_p-\mu_0)[c(\mu_p+\delta_p) + K_0 p P_0]} < 0$. *By* $\mu_0 < \min\{\mu_p, \mu_h, \mu_s, \mu_a\}$, *we know that this is a contradiction. so* $q_2 < q_4$.

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Suppose $q_2 < q_1$, *that is* $\frac{P_0K_0p}{c(\mu_p+\delta_p)}(\frac{\mu_p}{\mu_0}-1) > 1$, $R_1 > 1$. *Therefore,* $q_2 < q_1$ *if and only if* $R_1 > 1$.

Suppose $q_2 < q_3$, *that is* $(R_1-1)(R_1-(\frac{\mu_0}{\mu_p}-1)) > 0$, $R_1 > 1$ *or* $R_1 < \frac{\mu_0}{\mu_p}-1 < 0$. *So,* $q_2 < q_3$ *if and only if* $R_1 > 1$.

From the above analysis, we can know that $q_1 < q_3, q_2 < q_4$, $q_2 < q_1$ *if and only if* $R_1 > 1$, *and when* $q_2 > q_1$, *one has* $q_3 < q_2 < q_4$. *The existence of the boundary equilibria is discussed below in two cases.*

(c)If $q > q_1, A_2 > 0, \Delta_1 = 0$, *then equation* (3) *has only one positive root* $I^*_{p3} = \frac{-A_2}{2A_1}$. *According to* $q = q_3, R_1 > 1$, *we get* $\Lambda_s - \mu_p I^*_{p3} > 0, \mu_0 P_0 - q\Lambda_s + q(\mu_p - \mu_0)I^*_{p3} > 0$, *hence, there is only one boundary equilibrium* E_3 *for system* (2).

(d)If $q > q_1, A_2 > 0, \Delta_1 > 0$, *there are two positive roots* $I^*_{p31} = \frac{-A_2-\sqrt{\Delta_1}}{2A_1}$, *and* $I^*_{p32} = \frac{-A_2+\sqrt{\Delta_1}}{2A_1}, (I^*_{p31} > I^*_{p32})$. *We can acquire* $\Lambda_s - \mu_p I^*_{p31} > 0, \mu_0 P_0 - q\Lambda_s + q(\mu_p - \mu_0)I^*_{p31} > 0, \Lambda_s - \mu_p I^*_{p32} > 0, \mu_0 P_0 - q\Lambda_s + q(\mu_p - \mu_0)I^*_{p32} > 0$ *from* $q_1 < q < q_3$, *and* $R_1 > 1$, *so system* (2) *has two coexisting boundary equilibria* E_{31} *and* E_{32}.

In the following, the conditions for determining the local asymptotic stability of the boundary equilibria of system (2) are given.

For convenience, let the arbitrary boundary equilibrium be $E^* = (S^*, I^*_p, 0, 0, 0, P^*)$, accordingly, the Jacobian matrix of system (2) at boundary equilibrium E^* is

$$J(E^*) = \begin{pmatrix} -\mu_0-K_0pP^* & \delta_p & \delta_h-(d+e) & \delta_s-(d+e) & \delta_a-(d+e) & -K_0pS^* \\ K_0pP^* & -\mu_p-\delta_p & 0 & 0 & 0 & K_0pS^* \\ 0 & 0 & -\mu_h-\delta_h+(d_1+e_1) & d_1+e_1 & d_1+e_1 & 0 \\ 0 & 0 & d_2+e_2 & -\mu_s-\delta_s+(d_2+e_2) & d_2+e_2 & 0 \\ 0 & 0 & d_3+e_3 & d_3+e_3 & -\mu_a-\delta_a+(d_3+e_3) & 0 \\ -q & -q & -q & -q & -q & -c \end{pmatrix},$$

where

$d = (1-K_0)p\beta S^*P^*, d_1 = K_1 d, d_2 = K_2(1-K_1)d, d_3 = (1-K_2)(1-K_1)d, d = d_1+d_2+d_3,$

$e = (1-p)\beta_1 S^*P^*, e_1 = K_3 e, e_2 = K_4(1-K_3)e, e_3 = (1-K_4)(1-K_3)e, e = e_1+e_2+e_3.$

The corresponding characteristic equation is

$$det(\lambda I - J(E^*)) = (\lambda^3 + M_1\lambda^2 + M_2\lambda + M_3)(\lambda^3 + D_1\lambda^2 + D_2\lambda + D_3),$$

where

$M_1 = c+\mu_0+\mu_p+\delta_p+K_0pP^*$, $M_2 = c\mu_0+(c+\mu_0)(\mu_p+\delta_p)+cK_0pP^*+K_0p\mu_pP^*$, $M_3 = c\mu_0(\mu_p+\delta_p)+cK_0p\mu_pP^*-K_0pqS^*(\mu_p-\mu_0)$,

$D_1 = (\mu_h+\delta_h)+(\mu_s+\delta_s)+(\mu_a+\delta_a)-(d+e)$,

$D_2 = (\mu_h+\delta_h)(\mu_s+\delta_s)+(\mu_h+\delta_h)(\mu_a+\delta_a)+(\mu_s+\delta_s)(\mu_a+\delta_a)-[(\mu_h+\delta_h)(d_2+d_3+e_2+e_3)+(\mu_s+\delta_s)(d_1+d_3+e_1+e_3)+(\mu_a+\delta_a)(d_1+d_2+e_1+e_2)]$,

$D_3 = (\mu_h+\delta_h)(\mu_s+\delta_s)(\mu_a+\delta_a)[1-(\frac{d_1+e_1}{\mu_h+\delta_h}+\frac{d_2+e_2}{\mu_s+\delta_s}+\frac{d_3+e_3}{\mu_a+\delta_a})]$.

It is clear that $M_1 > 0, M_2 > 0, M_1M_2 - M_3 > 0$. If $M_3 > 0$, we get $f'(I^*_p) < 0$. Hence, when $M_3 > 0$, that is $f'(I^*_p) < 0$, the real parts of the eigenvalues of the equation $\lambda^3 + M_1\lambda^2 + M_2\lambda + M_3 = 0$ are all negative according to the Routh-Hurwitz criterion.

$D_1 > 0, D_2 > 0$, and $D_1D_2 - D_3 > 0$ can be calculated directly by $D_3 > 0$. Denote $R_2 = \frac{d_1+e_1}{\mu_h+\delta_h}+\frac{d_2+e_2}{\mu_s+\delta_s}+\frac{d_3+e_3}{\mu_a+\delta_a}$. Thus, $D_3 > 0$ if and only if $R_2 < 1$. According to the Routh-Hurwitz criterion, the real parts of the eigenvalues of the equation $\lambda^3 + D_1\lambda^2 + D_2\lambda + D_3 = 0$ are all negative when $R_2 < 1$.

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In summary, the local stability theorem for boundary equilibria is given below.

Theorem 3.6 *The boundary equilibrium E^* is locally asymptotically stable if and only if $R_2 < 1, f'(I_p^*) < 0$.*

Note: It is obvious that the boundary equilibria E_3 and E_{32} are not locally asymptotically stable based on the boundary equilibria' existence and stability requirements $f'(I_p^*) < 0$. On the contrary, the boundary equilibria E_1, E_2 and E_{31} are locally asymptotically stable when the existence and stability conditions are satisfied.

Theorem 3.7 *The boundary equilibrium E^* is globally asymptotically stable if and only if $R_2 < 1, f'(I_p^*) < 0$, subject to the corresponding existence conditions of Theorem 3.5.*

Proof *We construct a Lyapunov function*

$$V(t) = \frac{I_h(t)}{\mu_h+\delta_h} + \frac{I_s(t)}{\mu_s+\delta_s} + \frac{I_a(t)}{\mu_a+\delta_a}.$$

Calculating the total derivative of the solution of $V(t)$ along system (2) yields

$$\begin{aligned}
\frac{dV}{dt} &= \frac{1}{\mu_h+\delta_h}\frac{dI_h}{dt} + \frac{1}{\mu_s+\delta_s}\frac{dI_s}{dt} + \frac{1}{\mu_a+\delta_a}\frac{dI_a}{dt} \\
&= \frac{1}{\mu_h+\delta_h}[K_1(1-K_0)pSP\beta(I_h+I_s+I_a) + K_3(1-p)SP\beta_1(I_h+I_s+I_a) - (\delta_h+\mu_h)I_h] + \frac{1}{\mu_s+\delta_s}[K_2(1-K_1)(1 \\
&\quad -K_0)pSP\beta(I_h+I_s+I_a) + K_4(1-K_3)(1-p)SP\beta_1(I_h+I_s+I_a) - (\delta_s+\mu_s)I_s] + \frac{1}{\mu_a+\delta_a}[(1-K_2)(1-K_1)(1 \\
&\quad -K_0)pSP\beta(I_h+I_s+I_a) + (1-K_4)(1-K_3)(1-p)SP\beta_1(I_h+I_s+I_a) - (\delta_a+\mu_a)I_a] \\
&\leq I_v(\frac{d_1+e_1}{\mu_h+\delta_h} + \frac{d_2+e_2}{\mu_s+\delta_s} + \frac{d_3+e_3}{\mu_a+\delta_a} - 1) \\
&= I_v(R_2 - 1).
\end{aligned}$$

Hence, $\frac{dV}{dt} < 0$ if $R_2 < 1$. Meanwhile, $\frac{dV}{dt} = 0$ if and only if $I_h = 0, I_s = 0, I_a = 0$, the maximum invariant set is $\{E^*\}$. If the boundary equilibrium E^* at this time satisfies the conditions specified for existence in Theorem 3.5, then , according to LaSalle invariant set principle, E^* is globally asymptotically stable.

3.2.3 Existence of epidemic equilibrium

Theorem 3.8 *For system (2), there is only one endemic equilibrium $E_4 = (S_4^*, I_{p4}^*, I_{h4}^*, I_{s4}^*, I_{a4}^*, P_4^*)$ if and only if $q_5 < q < q_8$, here $q_5 = \frac{\mu_0 P_0}{\Lambda_s-(\mu_p-\mu_0)I_{p4}^*}$, $q_8 = \frac{P_0Q_1}{Q_1I_{p4}^*+(\Lambda_s-\mu_pI_{p4}^*)Q_2}$.*

Proof *From the third, fourth, and fifth equations of the system (2), eliminating I_a, I_s, and I_h in turn, we get*

$$I_{p4}^* = \frac{K_0p}{\frac{C_h}{\mu_h+\delta_h}+\frac{C_s}{\mu_s+\delta_s}+\frac{C_a}{\mu_a+\delta_a}}, I_s = \frac{C_s(\mu_h+\delta_h)}{C_h(\mu_s+\delta_s)}I_h, I_a = \frac{C_a(\mu_h+\delta_h)}{C_h(\mu_a+\delta_a)}I_h,$$

where

$C_h = [K_1(1-K_0)p\beta + K_3(1-p)\beta_1](\mu_p+\delta_p)$, $C_s = [K_2(1-K_1)(1-K_0)p\beta + K_4(1-K_3)(1-p)\beta_1](\mu_p+\delta_p)$, $C_a = [(1-K_2)(1-K_1)(1-K_0)p\beta + (1-K_4)(1-K_3)(1-p)\beta_1](\mu_p+\delta_p)$.

From the first and second equations of system (2), we have

$$S = \frac{\Lambda_s-\mu_pI_{p4}^*-\mu_hI_h-\mu_sI_s-\mu_aI_a}{\mu_0}, P = \frac{(\mu_p+\delta_p)I_{p4}^*}{K_0pS}.$$

In the sixth equation of the system (2), substituting the aforementioned expressions for S, I_s, I_a, and P results in

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$$\frac{qQ_1}{\mu_0}(\frac{Q_1}{\mu_0}-Q_2)I_h^2+[-\frac{2q(\Lambda_s-\mu_pI_{p4}^*)Q_1}{\mu_0^2}+\frac{q(\Lambda_s-\mu_pI_{p4}^*)Q_2}{\mu_0}-\frac{(qI_{p4}^*-P_0)Q_1}{\mu_0}]I_h+[\frac{q(\Lambda_s-\mu_pI_{p4}^*)^2}{\mu_0^2}+\frac{(qI_{p4}^*-P_0)(\Lambda_s-\mu_pI_{p4}^*)}{\mu_0}]=0,$$

where $Q_1=\mu_h+\mu_s\frac{C_s(\mu_h+\delta_h)}{C_h(\mu_s+\delta_s)}+\mu_a\frac{C_a(\mu_h+\delta_h)}{C_h(\mu_a+\delta_a)}$, $Q_2=1+\frac{C_s(\mu_h+\delta_h)}{C_h(\mu_s+\delta_s)}+\frac{C_a(\mu_h+\delta_h)}{C_h(\mu_a+\delta_a)}$, $P_0-qI_{p4}^*>0,\Lambda_s-\mu_pI_{p4}^*>0$.

The above quadratic equation with respect to I_h *is written as*

$$B_2I_h^2+B_1I_h+B_0=0, \tag{4}$$

where

$B_2=\frac{qQ_1}{\mu_0}(\frac{Q_1}{\mu_0}-Q_2)=\frac{qQ_1}{\mu_0}[(\frac{\mu_h}{\mu_0}-1)+(\frac{\mu_s}{\mu_0}-1)\frac{C_s(\mu_h+\delta_h)}{C_h(\mu_s+\delta_s)}+(\frac{\mu_a}{\mu_0}-1)\frac{C_a(\mu_h+\delta_h)}{C_h(\mu_a+\delta_a)}]>0$, $B_1=-\frac{2q(\Lambda_s-\mu_pI_{p4}^*)Q_1}{\mu_0^2}+\frac{q(\Lambda_s-\mu_pI_{p4}^*)Q_2}{\mu_0}-\frac{(qI_{p4}^*-P_0)Q_1}{\mu_0}$, $B_0=\frac{q(\Lambda_s-\mu_pI_{p4}^*)^2}{\mu_0^2}+\frac{(qI_{p4}^*-P_0)(\Lambda_s-\mu_pI_{p4}^*)}{\mu_0}$, $\Delta_h=\frac{\{q[(\Lambda_s-\mu_pI_{p4}^*)Q_2+Q_1I_{p4}^*]-P_0Q_1\}^2}{\mu_0^2}\geq 0$.

Solving the equation (4) *gives* $I_{h41}^*=\frac{-B_1-\sqrt{\Delta_h}}{2B_2}, I_{h42}^*=\frac{-B_1+\sqrt{\Delta_h}}{2B_2}$. *The following three scenarios are covered.*

(1)When $B_0<0$,*that is* $q<\frac{\mu_0P_0}{\Lambda_s-(\mu_p-\mu_0)I_{p4}^*}=q_5$, *then equation* (4) *has only one positive root* $I_{h42}^*=\frac{-B_1+\sqrt{\Delta_h}}{2B_2}=\frac{\Lambda_s-\mu_pI_{p4}^*}{Q_1}$, *but* $S=\frac{\Lambda_s-\mu_pI_{p4}^*-\mu_hI_h-\mu_sI_s-\mu_aI_a}{\mu_0}=\frac{\Lambda_s-\mu_pI_{p4}^*-Q_1I_{h42}^*}{\mu_0}=0$ *at this time. Therefore, there is no endemic equilibrium.*

(2)When $B_0=0$, *that is* $q=q_5$, *there is a unique positive root* $I_h^*=-\frac{B_1}{B_2}=\frac{\Lambda_s-\mu_pI_{p4}^*}{Q_1}$ *if* $B_1<0$. *And* $B_1<0$ *if and only if* $q>\frac{\mu_0P_0Q_1}{\mu_0Q_1I_{p4}^*-(\Lambda_s-\mu_pI_{p4}^*)(\mu_0Q_2-2Q_1)}=q_7$, $P_0-qI_{p4}^*>0$ *if and only if* $q<\frac{P_0}{I_{p4}^*}=q_6$, *after calculation, we get* $q_7<q_5<q_6$ *satisfying* $B_1<0$, *however,* $S=\frac{\Lambda_s-\mu_pI_{p4}^*-Q_1I_h^*}{\mu_0}=0$, *there is no endemic equilibrium.*

(3)When $B_0>0$,*that is* $q>q_5$, *the sufficient condition for the existence of positive roots of the equation* (4) *is* $B_1<0,\Delta_h\geq 0$. *Here are two scenarios.*

(i) When $B_1<0,\Delta_h=0$, *that is* $q>q_7, q=\frac{P_0Q_1}{Q_1I_{p4}^*+(\Lambda_s-\mu_pI_{p4}^*)Q_2}=q_8$, *satisfying* $q_5<q_8<q_6$, *there is a unique positive root* $I_{h42}^*=\frac{\Lambda_s-\mu_pI_{p4}^*}{Q_1}$, *but there is no endemic equilibrium for* $S=\frac{\Lambda_s-\mu_pI_{p4}^*-Q_1I_{h42}^*}{\mu_0}=0$.

(ii) When $B_1<0,\Delta_h>0$, *we have* $q\in(q_5,q_8)\bigcup(q_8,q_6)$ *from* $q<q_6$, *the equation* (4) *has two different positive roots* $I_{h41}^*=\frac{q[\Lambda_s-(\mu_p-\mu0)I_{p4}^*]-\mu_0P_0}{q(Q_1-\mu_0Q_2)}, I_{h42}^*=\frac{\Lambda_s-\mu_pI_{p4}^*}{Q_1}$.

(a)When $q\in(q_5,q_8)$, *we get* $S_4^{**}=\frac{\Lambda_s-\mu_pI_{p4}^*-Q_1I_{h42}^*}{\mu_0}=0, S_4^*=\frac{P_0Q_1-q[Q_1I_{p4}^*+Q_2(\Lambda_s-\mu_pI_{p4}^*)]}{q(Q_1-\mu_0Q_2)}>0$, *thus, there is a unique endemic equilibrium* $E_4=(S_4^*,I_{p4}^*,I_{h4}^*,I_{s4}^*,I_{a4}^*,P_4^*)$, *here* $I_{h4}^*=I_{h42}^*, I_{s4}^*=\frac{C_s(\mu_h+\delta_h)}{C_h(\mu_s+\delta_s)}I_{h4}^*, I_{a4}^*=\frac{C_a(\mu_h+\delta_h)}{C_h(\mu_a+\delta_a)}I_{h4}^*$.

(b)When $q\in(q_8,q_6)$, *we have* $S_4^{**}=\frac{\Lambda_s-\mu_pI_{p4}^*-Q_1I_{h42}^*}{\mu_0}=0, S_4^*=\frac{P_0Q_1-q[Q_1I_{p4}^*+Q_2(\Lambda_s-\mu_pI_{p4}^*)]}{q(Q_1-\mu_0Q_2)}<0$. *Hence, there is no endemic equilibrium.*

In conclusion, the unique endemic equilibrium $E_4=(S_4^*,I_{p4}^*,I_{h4}^*,I_{s4}^*,I_{a4}^*,P_4^*)$ *exists when* $q_5<q<q_8$.

Assume $q_5\geq q_3$, that is $\frac{\Lambda_s-(\mu_p-\mu_0)I_{p4}^*}{\Lambda_s+\mu_pI_{p4}^*+2\sqrt{\Lambda_s\mu_pI_{p4}^*}}\leq R_1\leq\frac{\Lambda_s-(\mu_p-\mu_0)I_{p4}^*}{\Lambda_s+\mu_pI_{p4}^*-2\sqrt{\Lambda_s\mu_pI_{p4}^*}}$. Denote $R_1^*=\frac{\Lambda_s-(\mu_p-\mu_0)I_{p4}^*}{\Lambda_s+\mu_pI_{p4}^*+2\sqrt{\Lambda_s\mu_pI_{p4}^*}}$, $R_1^{**}=\frac{\Lambda_s-(\mu_p-\mu_0)I_{p4}^*}{\Lambda_s+\mu_pI_{p4}^*-2\sqrt{\Lambda_s\mu_pI_{p4}^*}}$. It is easy to derive $R_1^*<1, R_1^{**}>1$ by calculation, therefore, $q_5\geq q_3$ if and only if $R_1^*\leq R_1\leq R_1^{**}$.

The following Theorem 3.9 can be condensed by combining Theorem 3.2, Theorem 3.5 and Theorem 3.8.

Theorem 3.9 . *For system* (2), *we have:*

(1)If $q_3\leq q_5<q<q_8$, *that is* $R_1^*\leq R_1\leq R_1^{**}$, *the endemic equilibrium* E_4 *does not coexist with the disease-free equilibrium, nor with boundary equilibria.*

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(2)If $q_3 > q_5, q_5 < q < q_8$, that is $R_1 < R_1^* < 1$ or $R_1 > R_1^{**} > 1$.

(i) When $R_1 < R_1^* < 1$, the endemic equilibrium E_4 does not coexist with the disease-free equilibrium, nor with boundary equilibria.

(ii)When $R_1 > R_1^{**} > 1$, there are four cases as follows.

(a)If $q_8 \leq q_3$, then the endemic equilibrium E_4 coexists with the boundary equilibria E_{31}, E_{32}.

(b)If $q_8 > q_3$, and $q_5 < q < q_3$, then the endemic equilibrium E_4 coexists with the boundary equilibria E_{31}, E_{32}.

(c)If $q_8 > q_3$, and $q_3 < q < q_8$, then the endemic E_4 does not coexist with either the disease-free equilibrium or boundary equilibria.

(d)If $q_8 > q_3$, and $q = q_3$, then only the boundary equilibrium E_3 exists.

where $R_1^* = \frac{\Lambda_s - (\mu_p - \mu_0) I_{p4}^*}{\Lambda_s + \mu_p I_{p4}^* + 2\sqrt{\Lambda_s \mu_p I_{p4}^*}} < 1$, $R_1^{**} = \frac{\Lambda_s - (\mu_p - \mu_0) I_{p4}^*}{\Lambda_s + \mu_p I_{p4}^* - 2\sqrt{\Lambda_s \mu_p I_{p4}^*}} > 1$.

Table 1: Existence of equilibria of system (2)

q	R_1	Equilibrium
$0 < q < q_1$	$-$	$(BE)E_1$
$q = q_1$	$-$	$(DFE)E_0$
	$R_1 > 1$	$(DFE)E_0 \quad and \quad (BE)E_2$
$q_1 < q < q_3$	$R_1 > 1$	$(BE)E_{31}, E_{32}$
$q = q_3$	$R_1 > 1$	$(BE)E_3$
$q_5 < q < q_8$	$R_1^* < R_1 < R_1^{**}$	$(EE)E_4$
	$R_1 < R_1^* < 1$	$(EE)E_4$
$q_5 < q < q_8 \leq q_3$	$R_1 > R_1^{**} > 1$	$(EE)E_4 \quad and \quad (BE)E_{31}, E_{32}$
$q_5 < q < q_3 < q_8$	$R_1 > R_1^{**} > 1$	$(EE)E_4 \quad and \quad (BE)E_{31}, E_{32}$
$q_5 < q = q_3 < q_8$	$R_1 > R_1^{**} > 1$	$(BE)E_3 \quad and \quad (EE)E_4$
$q_3 < q < q_8$	$R_1 > R_1^{**} > 1$	$(EE)E_4$

Boundary equilibrium(BE), endemic equilibrium(EE), and disease-free equilibrium (DFE), respectively, are denoted.

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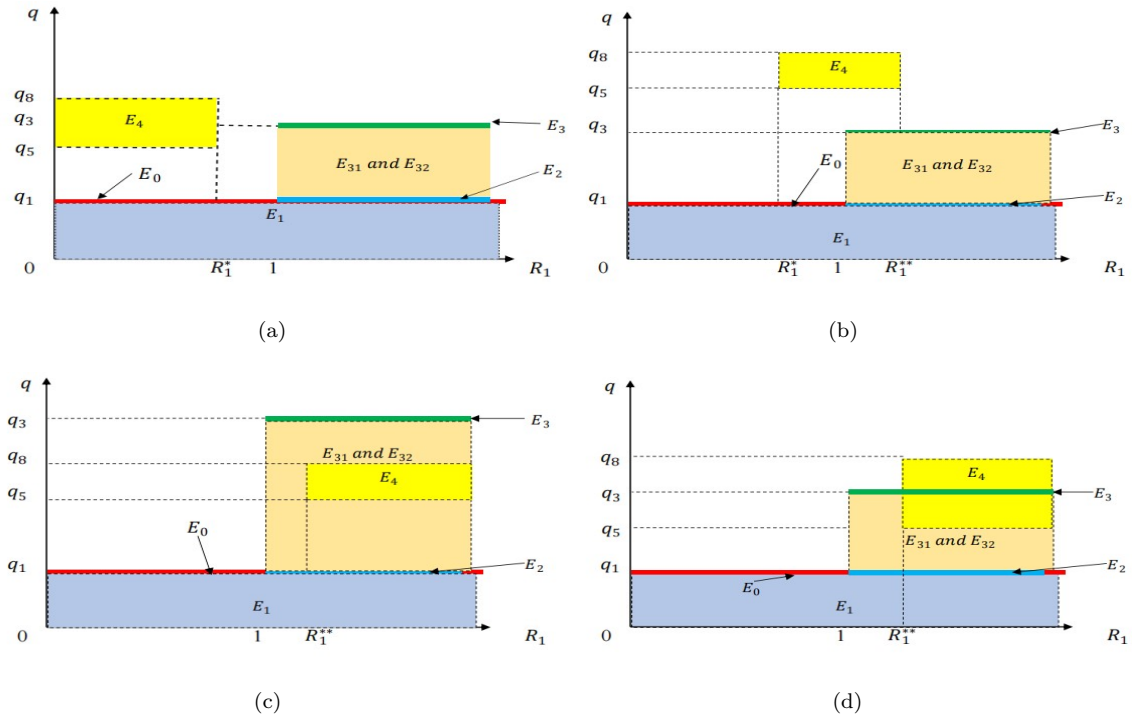

Figure 3: Existence of equilibria in system (2).

According to the results of the analysis of Tabel 1 and Theorem 3.9 above, the existence of equilibria of system (2) is influenced by the threshold $R_1 = \frac{P_0 K_0 p}{c(\mu_p+\delta_p)}(\frac{\mu_p}{\mu_0} - 1)$ and individual inhalation of air pollutants q. In other words, by adjusting threshold R_1 and human pollutant inhalation q, the equilibrium of system can be controlled so that, to the greatest extent possible, only disease-free equilibrium exist in the system, minimizing the harm caused by air pollution and achieving the goal of stopping the epidemic of respiratory diseases.

3.3 Bifurcation analysis

Theorem 3.10 *When $q = q_1, R_1 = 1$ and $\sigma \neq 0$, system (2) occurs fold bifurcation at disease-free equilibrium E_0.*

Disease-free equilibrium E_0 exists if and only if $q = q_1$, When $a_3 = 0$, i.e., $R_1 = 1$, the characteristic equation corresponding to E_0 is

$$\lambda(\lambda + \mu_h + \delta_h)(\lambda + \mu_s + \delta_s)(\lambda + \mu_a + \delta_a)(\lambda^2 + a_1\lambda + a_2) = 0,$$

where $a_1 > 0, a_2 > 0, a_1^2 - 4a_2 > 0$. Thus the characteristic equation has five negative characteristic roots and one zero characteristic root.

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Let $X_1 = S - \frac{\Lambda_s}{\mu_0}, X_2 = I_p, X_3 = I_h, X_4 = I_s, X_5 = I_a, X_6 = P$, system (2) can be deformed as

$$\begin{cases} \frac{dX_1}{dt} = - K_0 p(X_1 + \frac{\Lambda_s}{\mu_0})X_6 - (1-K_0)p(X_1 + \frac{\Lambda_s}{\mu_0})X_6\beta(X_3+X_4+X_5) - (1-p)(X_1 + \frac{\Lambda_s}{\mu_0})X_6\beta_1(X_3+X_4+X_5) \\ \qquad + \delta_p X_2 + \delta_h X_3 + \delta_s X_4 + \delta_a X_5 - \mu_0 X_1, \\ \frac{dX_2}{dt} = K_0 p(X_1 + \frac{\Lambda_s}{\mu_0})X_6 - (\mu_p + \delta_p)X_2, \\ \frac{dX_3}{dt} = K_1(1-K_0)p(X_1 + \frac{\Lambda_s}{\mu_0})X_6\beta(X_3+X_4+X_5) + K_3(1-p)(X_1 + \frac{\Lambda_s}{\mu_0})X_6\beta_1(X_3+X_4+X_5) - (\mu_h + \delta_h)X_3, \\ \frac{dX_4}{dt} = K_2(1-K_1)(1-K_0)p(X_1 + \frac{\Lambda_s}{\mu_0})X_6\beta(X_3+X_4+X_5) + K_4(1-K_3)(1-p)(X_1 + \frac{\Lambda_s}{\mu_0})X_6\beta_1(X_3+X_4+X_5) \\ \qquad - (\mu_s + \delta_s)X_4, \\ \frac{dX_5}{dt} = (1-K_2)(1-K_1)(1-K_0)p(X_1 + \frac{\Lambda_s}{\mu_0})X_6\beta(X_3+X_4+X_5) + (1-K_4)(1-K_3)(1-p)(X_1 + \frac{\Lambda_s}{\mu_0})X_6\beta_1(X_3 \\ \qquad + X_4 + X_5) - (\mu_a + \delta_a)X_5, \\ \frac{dX_6}{dt} = - cX_6 - q(X_1 + X_2 + X_3 + X_4 + X_5), \end{cases} \tag{5}$$

Let $F_k(X_1, X_2, X_3, X_4, X_5, X_6) = \frac{dX_k}{dt} (k = 1, 2, 3, 4, 5, 6)$. Taking the partial derivatives of $F_k(X_1, X_2, X_3, X_4, X_5, X_6)(k = 1, 2, 3, 4, 5, 6)$ separately, we can obtain the matrix $H = \left((\frac{\partial F_i}{\partial X_j})_{ij} \right)_{1 \le i,j \le 6}$, substituting the origin into H gives

$$H = \begin{pmatrix} -\mu_0 & \delta_p & \delta_h & \delta_s & \delta_a & -K_0 p\frac{\Lambda_s}{\mu_0} \\ 0 & -\mu_p - \delta_p & 0 & 0 & 0 & K_0 p\frac{\Lambda_s}{\mu_0} \\ 0 & 0 & -\mu_h - \delta_h & 0 & 0 & 0 \\ 0 & 0 & 0 & -\mu_s - \delta_s & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_a - \delta_a & 0 \\ -q & -q & -q & -q & -q & -c \end{pmatrix},$$

If the eigenvalue of H corresponds to an eigenvector x and the adjoint eigenvector is y, we know from the Center Manifold Theorem[27] that

$$G(x, y) = \Big(G_1(x, y), G_2(x, y), G_3(x, y), G_4(x, y), G_5(x, y), G_6(x, y) \Big)^T,$$

where

$G_1(x, y) = -K_0 p X_1 Y_6 - (1-K_0)p\frac{\Lambda_s}{\mu_0}\beta(X_3+X_4+X_5)Y_6 - (1-p)\frac{\Lambda_s}{\mu_0}\beta_1(X_3+X_4+X_5)Y_6 - K_0 p X_6 Y_1 - (1-K_0)p\frac{\Lambda_s}{\mu_0}\beta(Y_3+Y_4+Y_5)X_6 - (1-p)\frac{\Lambda_s}{\mu_0}\beta_1(Y_3+Y_4+Y_5)X_6,$

$G_2(x, y) = K_0 p X_1 Y_6 + K_0 p X_6 Y_1,$

$G_3(x, y) = K_1(1-K_0)p\frac{\Lambda_s}{\mu_0}\beta(X_3+X_4+X_5)Y_6 + K_3(1-p)\frac{\Lambda_s}{\mu_0}\beta_1(X_3+X_4+X_5)Y_6 + K_1(1-K_0)p\frac{\Lambda_s}{\mu_0}\beta(Y_3+Y_4+Y_5)X_6 + K_3(1-p)\frac{\Lambda_s}{\mu_0}\beta_1(Y_3+Y_4+Y_5)X_6,$

$G_4(x, y) = K_2(1-K_1)(1-K_0)p\frac{\Lambda_s}{\mu_0}\beta(X_3+X_4+X_5)Y_6 + K_4(1-K_3)(1-p)\frac{\Lambda_s}{\mu_0}\beta_1(X_3+X_4+X_5)Y_6 + K_2(1-K_1)(1-K_0)p\frac{\Lambda_s}{\mu_0}\beta(Y_3+Y_4+Y_5)X_6 + K_4(1-K_3)(1-p)\frac{\Lambda_s}{\mu_0}\beta_1(Y_3+Y_4+Y_5)X_6,$

$G_5(x, y) = (1-K_2)(1-K_1)(1-K_0)p\frac{\Lambda_s}{\mu_0}\beta(X_3+X_4+X_5)Y_6 + (1-K_4)(1-K_3)(1-p)\frac{\Lambda_s}{\mu_0}\beta_1(X_3+X_4+X_5)Y_6 + (1-K_2)(1-K_1)(1-K_0)p\frac{\Lambda_s}{\mu_0}\beta(Y_3+Y_4+Y_5)X_6 + (1-K_4)(1-K_3)(1-p)\frac{\Lambda_s}{\mu_0}\beta_1(Y_3+Y_4+Y_5)X_6,$

$G_6(x, y) = 0.$

At this time, system (5) is

$$\dot{x} = Hx + \frac{1}{2}G(x, y) + \frac{1}{6}C_1(x, y, z) + O(||x||^4), \tag{6}$$

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where

$$x=(X_1,X_2,X_3,X_4,X_5,X_6)^T\in R^6, y=(Y_1,Y_2,Y_3,Y_4,Y_5,Y_6)^T\in R^6.$$

It is easy to find that when $R_1=1$ i.e., $a_3=0$, the characteristic roots of H are

$$\begin{aligned}\lambda_{t1} &= 0,\lambda_{t2}=-(\mu_h+\delta_h)<0,\lambda_{t3}=-(\mu_s+\delta_s)<0,\lambda_{t4}=-(\mu_a+\delta_a)<0,\\ \lambda_{t5} &= \frac{-a_1-\sqrt{a_1^2-4a_2}}{2}<0,\lambda_{t6}=\frac{-a_1+\sqrt{a_1^2-4a_2}}{2}<0,\end{aligned}$$

therefore, system (2) may occur fold bifurcation at disease-free equilibrium E_0. Note that $\mu_2=\mu_h,\delta_2=\delta_h,\mu_3=\mu_s,\delta_3=\delta_s,\mu_4=\mu_a,\delta_4=\delta_a$.

The eigenvector corresponding to the eigenvalue $\lambda_{t1}=0$ is $\tilde{p}=(\tilde{p_1},\tilde{p_2},0,0,0,1)$ and the adjoint eigenvector is $\tilde{q}=(\tilde{q_1},\tilde{q_2},\tilde{q_3},\tilde{q_4},\tilde{q_5},1)$, where

$$\begin{cases}
\tilde{p_1}=-\frac{c\mu_p}{q(\mu_p-\mu_0)}+\frac{c+\mu_p}{q(\mu_p-\mu_0)}A_{11}-\frac{1}{q(\mu_p-\mu_0)}A_{11}^2<0,\tilde{p_2}=\frac{c\mu_0}{q(\mu_p-\mu_0)}-\frac{c+\mu_0}{q(\mu_p-\mu_0)}A_{11}+\frac{1}{q(\mu_p-\mu_0)}A_{11}^2>0,\\
\tilde{q_1}=-\frac{q}{\mu_0}+\frac{q(c+\mu_p+\delta_p)}{c\mu_0(\mu_p+\delta_p)}A_{11}-\frac{q}{c\mu_0(\mu_p+\delta_p)}A_{11}^2<0,\tilde{q_2}=-\frac{q(\mu_0+\delta_p)}{\mu_0(\mu_p+\delta_p)}+\frac{q(c+\mu_0+\delta_p)}{c\mu_0(\mu_p+\delta_p)}A_{11}-\frac{q}{c\mu_0(\mu_p+\delta_p)}A_{11}^2<0,\\
\tilde{q}_{i+1}=\frac{A_{i1}}{A_{i2}}+\frac{A_{i3}A_{11}}{A_{i4}}+\frac{A_{i5}A_{11}^2}{A_{i6}}(i=2,3,4),\\
A_{11}=\frac{a_1}{3}+\frac{-\frac{a_1^2}{9}+\frac{a_2}{3}}{[-\frac{a_1^3}{27}+\frac{a_1a_2}{6}+\frac{a_2}{3}\sqrt{\frac{a_2}{3}-\frac{a_1^2}{12}}]^{\frac{1}{3}}}-[-\frac{a_1^3}{27}+\frac{a_1a_2}{6}+\frac{a_2}{3}\sqrt{\frac{a_2}{3}-\frac{a_1^2}{12}}]^{\frac{1}{3}}<0,\\
A_{i1}=q\Big\{\mu_0^2(c+\mu_p+\delta_p-\mu_i-\delta_i)+\mu_0[c(\delta_p-\mu_i)+\mu_i(\mu_i+\delta_i-\delta_p)+\mu_p(\delta_i-\mu_p-\delta_p)]+\mu_p[c(\mu_i-\mu_p-\delta_p)\\
\quad+\mu_i(\mu_p+\delta_p-\mu_i-\delta_i)]\Big\}<0,\\
A_{i2}=[\mu_0^2+\mu_p(\mu_i+\delta_i)](c-\mu_i-\delta_i)(\mu_p+\delta_p-\mu_i-\delta_i)+\mu_0\Big\{c(\mu_p+\delta_p)(\mu_p-\mu_0)+(\mu_i+\delta_i)[c(\mu_i+\delta_i)-\delta_p]\\
\quad-\mu_p(\mu_p+\delta_p)+(\mu_i+\delta_i)[\mu_p(\mu_p+\delta_p)+(\mu_i+\delta_i)(\delta_p-\mu_i-\delta_i)]\Big\}>0,\\
A_{i3}=\mu_0^2\Big\{c\mu_0(\mu_p+\delta_p)(c+\mu_p+\delta_p+\mu_0-\mu_i)+\delta_i(\mu_p+\delta_p-\mu_i-\delta_i)[c(c+\mu_p+\delta_p-\mu_i-\delta_i)-(\mu_p+\delta_p)(\mu_i+\delta_i)]\Big\},\\
A_{i4}=\frac{c^2\mu_0^3(\mu_p+\delta_p)}{q(\mu_p-\mu_0)}\Big\{\mu_0(\mu_p+\delta_p)(\mu_p-\mu_0)+(\mu_p+\delta_p-\mu_i-\delta_i)[\mu_0^2+\mu_p(\mu_i+\delta_i)]+\mu_0[(\mu_i+\delta_i)^2-\mu_p(\mu_p+\delta_p)\\
\quad-\delta_p(\mu_i+\delta_i)]\Big\}>0,\\
A_{i5}=\mu_0^2[\delta_i(\mu_i+\delta_i-\mu_p-\delta_p)(c-\mu_i-\delta_i)-c\mu_0(\mu_p+\delta_p)],\\
A_{i6}=A_{i4}+\frac{c\mu_0^3(\mu_p+\delta_p)(\mu_i+\delta_i)}{q(\mu_p-\mu_0)}\Big\{\mu_0^2(\mu_i+\delta_i-\mu_p-\delta_p)+\mu_0[\mu_p(\mu_p+\delta_p)+\delta_p(\mu_i+\delta_i)-(\mu_i+\delta_i)^2]+\mu_p(\mu_i\\
\quad+\delta_i)(\mu_i+\delta_i-\mu_p-\delta_p)\Big\}>0.
\end{cases}$$

We get $G(\tilde{p},\tilde{q})=\Big(G_1(\tilde{p},\tilde{q}),G_2(\tilde{p},\tilde{q}),G_3(\tilde{p},\tilde{q}),G_4(\tilde{p},\tilde{q}),G_5(\tilde{p},\tilde{q}),G_6(\tilde{p},\tilde{q})\Big)^T$, where

$G_1(\tilde{p},\tilde{q})=-K_0p(\tilde{p_1}+\tilde{q_1})-[(1-K_0)p\beta+(1-p)\beta_1]\frac{\Lambda_s}{\mu_0}(\tilde{q_3}+\tilde{q_4}+\tilde{q_5})$,

$G_2(\tilde{p},\tilde{q})=K_0p(\tilde{p_1}+\tilde{q_1})$,

$G_3(\tilde{p},\tilde{q})=[K_1(1-K_0)p\beta+K_3(1-p)\beta_1]\frac{\Lambda_s}{\mu_0}(\tilde{q_3}+\tilde{q_4}+\tilde{q_5})$,

$G_4(\tilde{p},\tilde{q})=[K_2(1-K_1)(1-K_0)p\beta+K_4(1-K_3)(1-p)\beta_1]\frac{\Lambda_s}{\mu_0}(\tilde{q_3}+\tilde{q_4}+\tilde{q_5})$,

$G_5(\tilde{p},\tilde{q})=[(1-K_2)(1-K_1)(1-K_0)p\beta+(1-K_4)(1-K_3)(1-p)\beta_1]\frac{\Lambda_s}{\mu_0}(\tilde{q_3}+\tilde{q_4}+\tilde{q_5})$,

$G_6(\tilde{p},\tilde{q})=0$.

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The following system is produced by limiting the system to a one-dimensional center manifold:

$$\dot{X} = \sigma X^2 + O(|X|^3), X \in R^1, \sigma = \frac{1}{2}\langle \tilde{q}, G(\tilde{p}, \tilde{q})\rangle.$$

When the condition $\sigma \neq 0$ is satisfied, the local topological equivalence of system (5) has the following form: $\dot{X} = \xi + \sigma X^2$. That is, system (2) occurs fold bifurcation at disease-free equilibrium E_0 when $q = q_1, R_1 = 1$ and $\sigma \neq 0$.

4 Sensitivity analysis about the number of infected

Sensitivity indices can be used to determine how much the state variables have changed relatively to the parameters. They might be either positive or negative for these indicators. The absolute value of the index reveals the intensity of the connection, and its positive and negative values reveal positive and negative correlations. The PRCC approach is then used to investigate how the parameters affect various patient categories.

Table 2: Ranges for parameters

Parameters	Range	Sources	Parameters	Range	Sources
Λ_s	[1000,5000],[0.00005,0.005]	[9, 8]	K_3	[0.05,0.75]	
K_4	[0.1,0.7]	[22, 23]	μ_0	[0.00003,0.00006],[0.00006,0.005]	[9, 8]
μ_h	[0.00003,0.00008],[0.00007,0.015]	[26]	μ_s	[0.00004,0.0001],[0.00007,0.015]	[26]
μ_a	[0.00003,0.00006],[0.00006,0.005]	[9]	δ_h	[1/30,1/3]	[22, 24, 25]
δ_s	[1/30,1/3]	[22, 24, 25]	δ_a	[1/14,1/3]	[22, 24]
β_1	[3e-10,2.5e-8],[0.000005,0.0001]	[9]	K_0	[0.1,0.7]	
K_1	[0.1,0.7]		K_2	[0.1,0.7]	
μ_p	[0.00007,0.015]	[8]	δ_p	[0.05,0.3]	[8]
p	[0.0001,0.01]	[8]	β	[0.00001,0.001]	
P_0	[0.00001,0.01]	[8]	c	[0.00001,0.01]	[8]
q	[0.0001,0.01]	[8]			

Table 2 displays the range of parameter values. The significance of numerous characteristics on various state variables of system (1) and system (2) is depicted in Figure 4 and Figure 5, respectively.

Combining Figure 4, a significant negative correlation can be seen between the cure rate $(\delta_h, \delta_s, \delta_a)$ and the total number of patients not seen, the number of symptomatic infected individuals who were not seen and the number of asymptomatic infections who were not seen, respectively. However, a positive correlation is presented between the number of patient pairs and infection rate β_1, whereas a significant positive correlation is presented between the cure rate $(\delta_h, \delta_s, \delta_a)$. Meanwhile, the total number of patients not seen show a negative correlation with K_3, but there is a positive correlation between the number of patients and the proportion of patients who go to the hospital (K_3).

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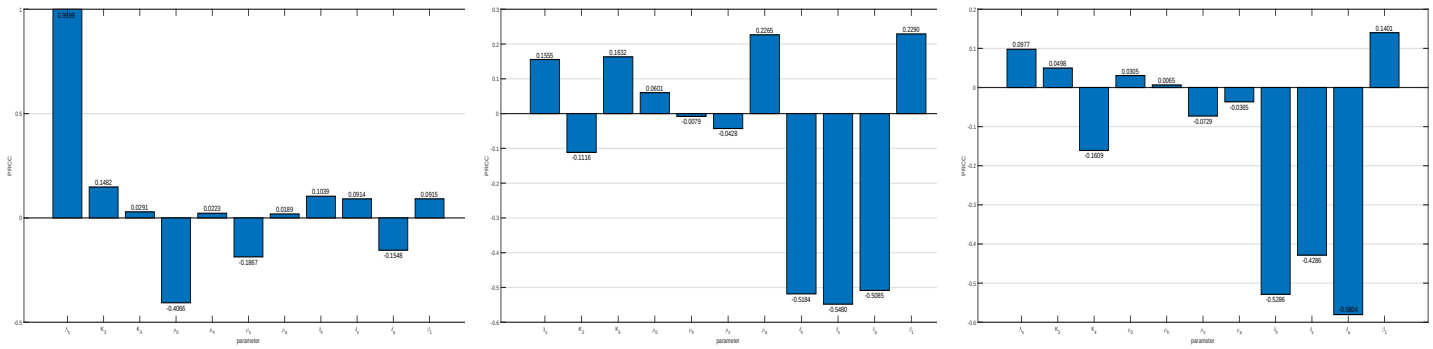

(a) (b) (c)

Figure 4: The significance analysis diagram of parameters to $(a)I_h, (b)I_s, (c)I_a$ of system (1).

Figure 5(a) shows that, to varying degrees, the parameters p, c, and q have a substantial correlation with the number of patients I_p. Combining Figure 5, we find a strong positive correlation between the total number of patients I_v and daily air pollutant emissions P_0, the natural clearance rate of air pollutants c, as well as a strong negative correlation between I_v and pollutant inhalation q. This suggests that an increase in daily air pollutant emissions P_0 and the natural clearance rate of air pollutants c causes an increase in the overall number of viral respiratory patients I_v; especially, an increase in the inhalation of air pollutants q causes a decrease in the total number of I_v because an increase in the conversion of susceptible people to allergic respiratory disease due to the inhalation of more air pollutants causes an increase in the conversion of susceptible people to allergic respiratory disease. In addition, Figure 5 (c)(d) demonstrate a strong negative correlation between δ_h, and δ_s and the number of unattended patients, indicating that an increase in the cure rate of attending patients and symptomatic infected patients will result in a reduction in the overall number of unattended patients.

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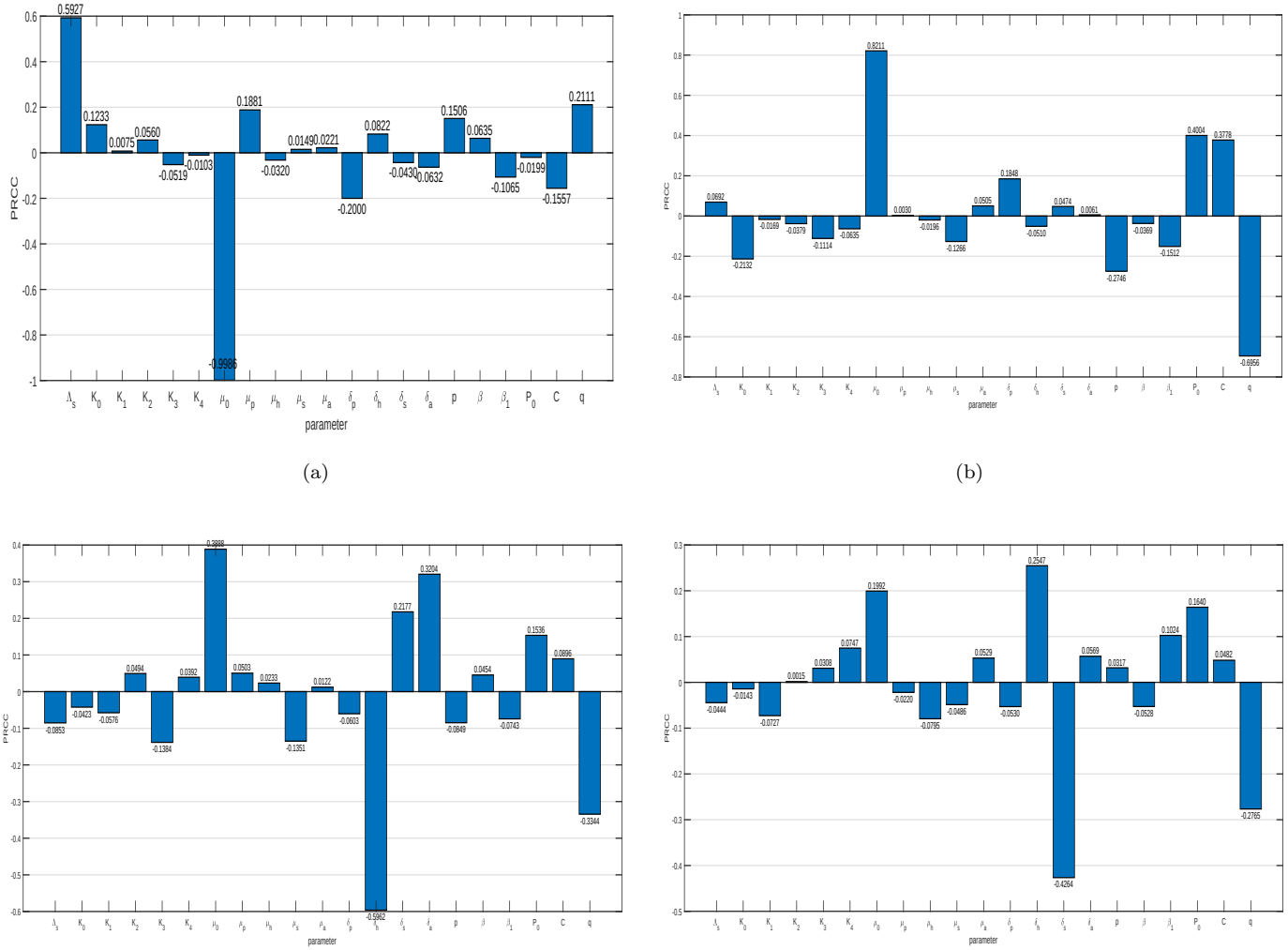

(a) (b)

(c) (d)

Figure 5: The significance analysis diagram of parameters to $(a)I_p, (b)I_h, (c)I_s, (d)I_a$ of system (2).

5 Numerical simulation

In this section, we investigate alternative values of these parameters in order to examine the influence of parameters that are more closely connected with the number of patients in each group on disease transmission. Figures 6 through 11 display the findings.

For system (1), we take the initial values $S(0) = 80, I_h(0) = 5, I_s(0) = 5$, and $I_a(0) = 5$. As shown in Figure 6, at low levels of air pollution, the infection rate β_1 of viral respiratory diseases is a significant determinant of the spread of the disease. An increase in β_1 results in a marked increase in the peak number of patients I_h, I_a, and I_s, as well as a larger final number of patients. In addition, a rise in cure rate δ_h decreases the peak number of viral respiratory disease patients, the duration to peak and the overall number, and it has very big impact on patients I_h(see Figure 7).

For system (2), initial values of $S(0) = 70, I_p(0) = 3, I_h(0) = 3, I_s(0) = 3, I_a(0) = 3$, and $P(0) = 2$ are taken to investigate the effect of individual parameter changes on disease transmission. Evidently, the final number and peak value

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of I_p are not significantly impacted by the shift in daily air pollution emissions P_0. However, when P_0 fall, so do the overall number and peak numbers of I_v, particularly I_p, as shown in Figure 8. An increase in air pollution inhalation q, as indicated in Figure 9, decreases the number of patients with viral respiratory disease but has no effect on the total number of patients.

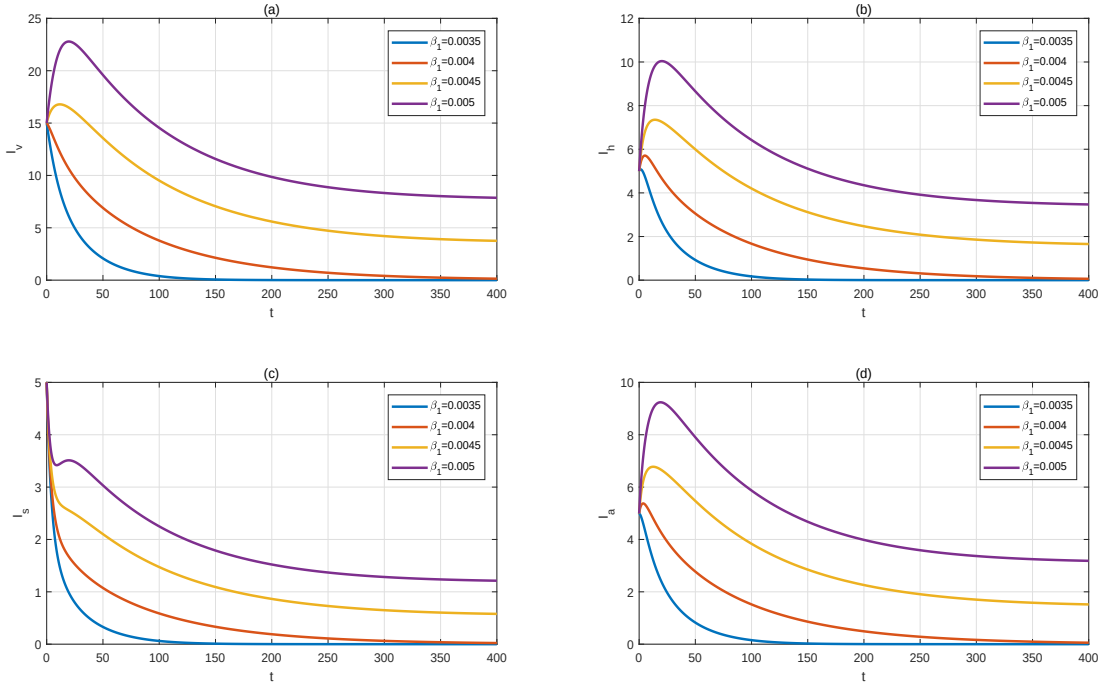

Figure 6: Effect of different infection rates β_1 on the number of patients (a)I_v, (b)I_h, (c)I_s, (d)I_a in model (1)

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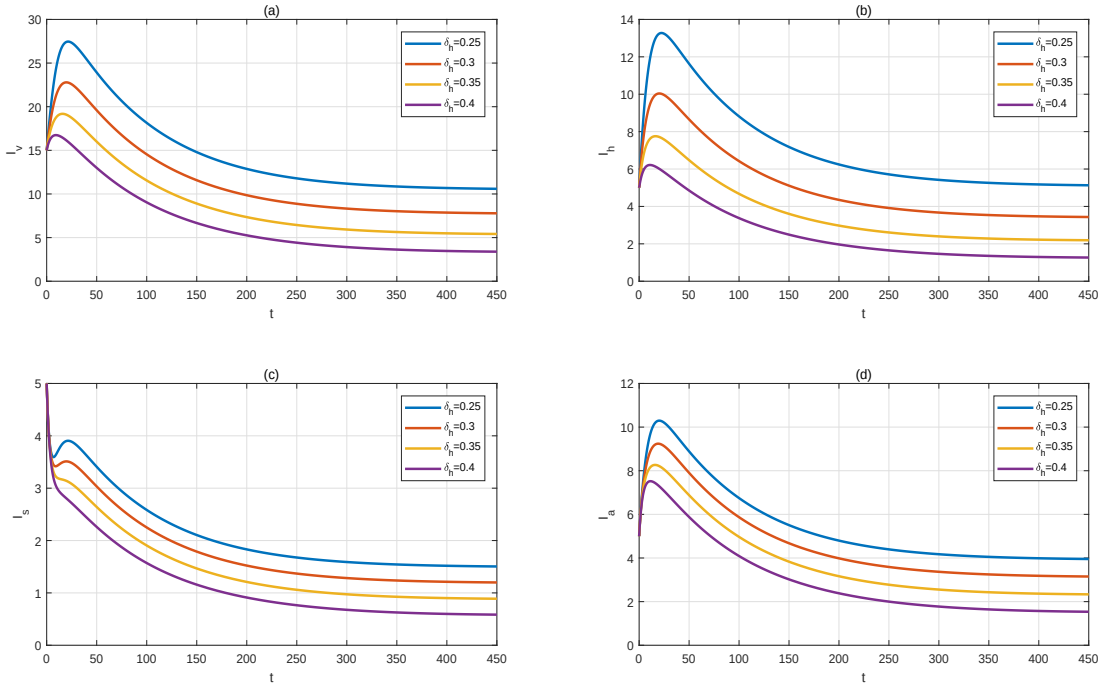

Figure 7: Effect of different visit cure rates δ_h on the number of patients (a)I_v, (b)I_h, (c)I_s, (d)I_a in model (1)

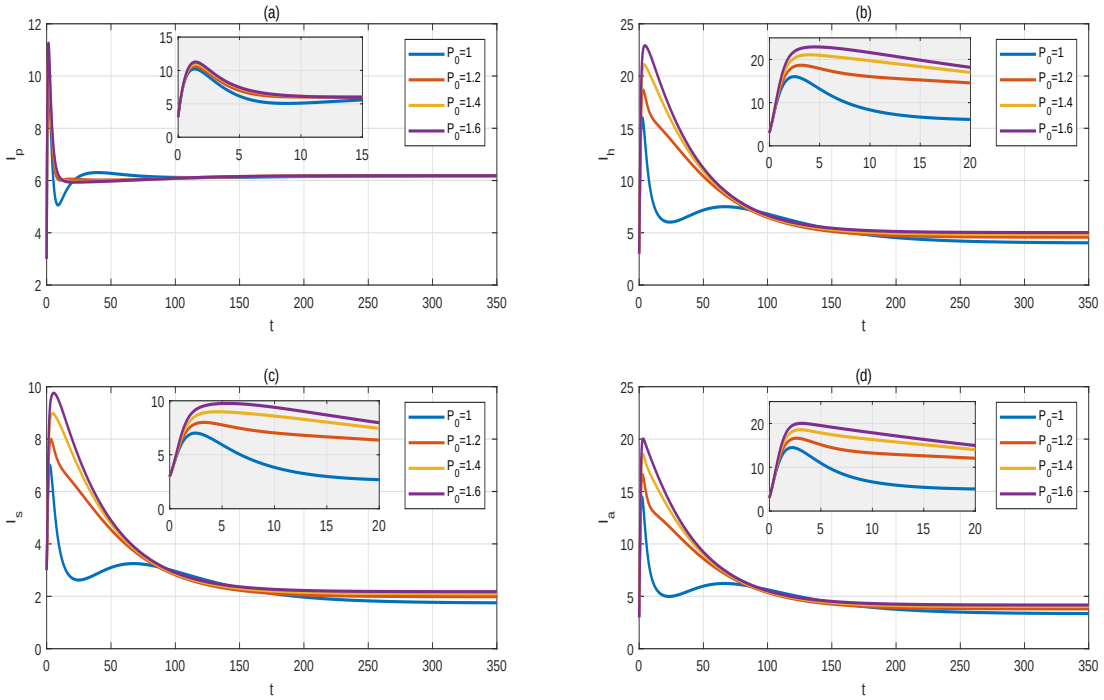

Figure 8: Effect of different daily air pollution emissions P_0 on the number of patients (a)I_p, (b)I_h, (c)I_s, (d)I_a.

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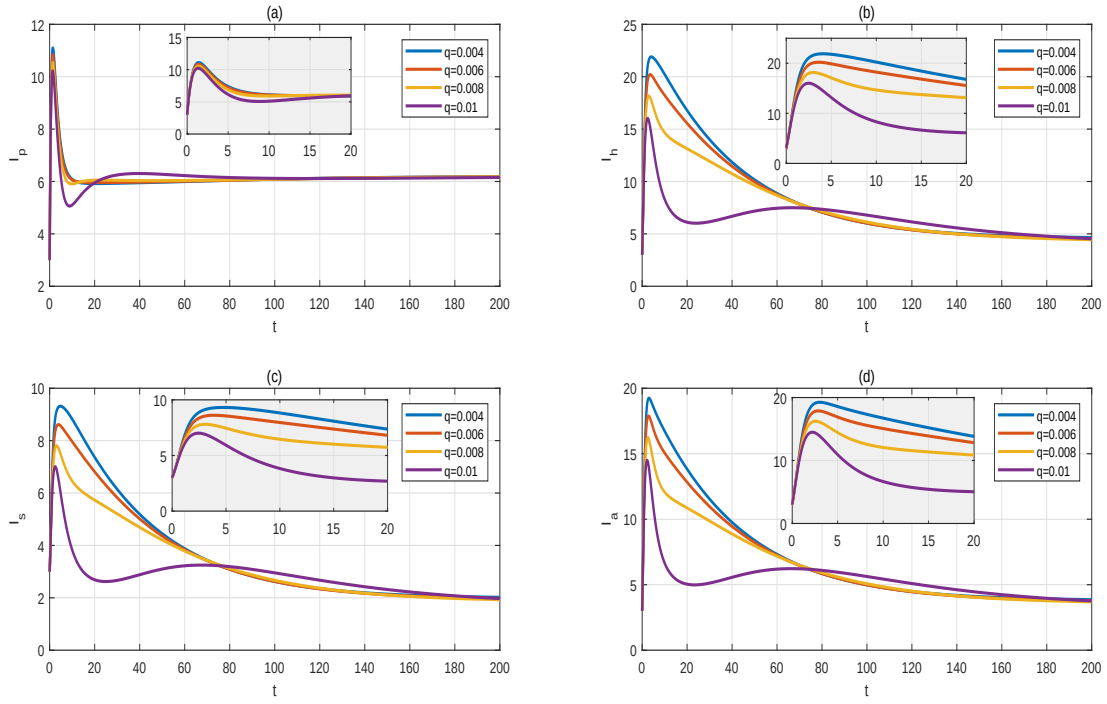

Figure 9: Effect of different air pollution inhalation levels q on the number of patients (a)I_p, (b)I_h, (c)I_s, (d)I_a.

We provide simulation results in Figures 10 and 11 so that one can more clearly observe how parameters K_3 and K_4 affect viral respiratory diseases at various degrees of air pollution. Initial values of $S(0) = 80, I_p(0) = 5, I_h(0) = 3, I_s(0) = 1, I_a(0) = 8$, and $P(0) = 2$ are selected.

In Figure 10, we choose parameters $A_s = 0.38, K_0 = 0.4, K_1 = 0.6, K_2 = 0.8, K_4 = 0.4, \mu_0 = 0.008, \mu_p = 0.02, \mu_h = 0.012, \mu_s = 0.018, \mu_a = 0.008, \delta_p = 0.45, \delta_h = 0.25, \delta_s = 0.2; \delta_a = 0.3, p = 0.08, \beta = 0.03, \beta_1 = 0.005, P_0 = 1, c = 0.6, q = 0.01$, and K_3 as shown in the figure. We can see that at lower levels of air pollution, the influence of K_3 changes on the spread of viral respiratory diseases is more pronounced. An increase in K_3 causes an increase in the peak in patients selected for hospitalization I_h, while decreasing the peak in I_s, I_a.

And we choose parameters $A_s = 0.5, K_0 = 0.2, K_1 = 0.6, K_2 = 0.8, K_3 = 0.4, \mu_0 = 0.005, \mu_p = 0.02, \mu_h = 0.03, \mu_s = 0.04, \mu_a = 0.02, \delta_p = 0.25, \delta_h = 0.35, \delta_s = 0.2, \delta_a = 0.45, p = 0.15, \beta = 0.003, \beta_1 = 0.002, P_0 = 1, c = 0.2, q = 0.01$ in Figure 11. The number of hospitalized patients I_h and the number of asymptomatic individuals who aren't hospitalized I_a aren't significantly impacted by the change in K_4 in the event that the disease eventually goes extinct, regardless of whether air pollution levels are high or low. The peak of I_s, however, is greatly raised by an increase in K_4.

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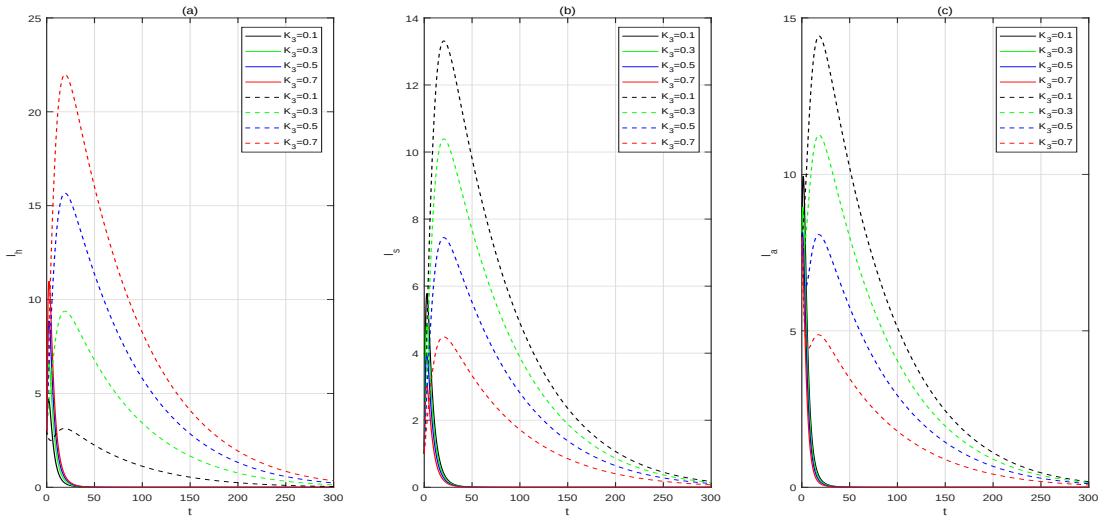

Figure 10: Contrasting the effects of K_3 on people with viral respiratory infections in two models, where the dashed line represents patients in model (1) and the solid line represents patients in model (2).

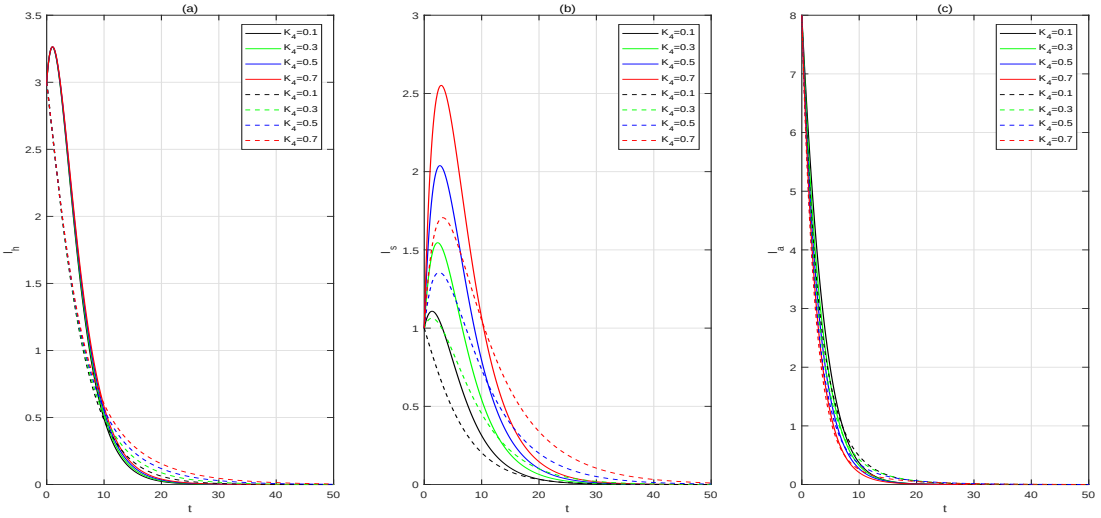

Figure 11: Contrasting the effects of K_4 on people with viral respiratory infections in two models, where the dashed line represents patients in model (1) and the solid line represents patients in model (2).

6 Conclusion and discussion

In order to study the effect of air pollution on the transmission of viral respiratory diseases in a heterogeneous population, this paper takes into account the sensitivity of individuals to air pollutants, their awareness of consultation, and the presence or absence of symptoms and classifies patients with allergic respiratory diseases caused by inhalation of

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air pollutants and patients with respiratory viral infections (specifically, patients with consultation, symptomatic patients without consultation, and asymptomatic patients without consultation). When the level of air pollution is low and does not produce allergic reactions in people, only the effect of individual heterogeneity on the transmission dynamics of viral respiratory infections is taken into account when constructing the $SI_hI_sI_aS$ model. The differential equation describing the change in air pollutant concentration is added at higher levels of air pollution, and a $SI_pI_hI_sI_aSP$ respiratory disease model is established to obtain the threshold R_1 for the equilibrium state of the system. The effects of this threshold and pollutant inhalation on the kinetics of disease transmission are then examined.

The basic reproduction number R_0, the disease-free equilibrium E_0^*, and the endemic equilibrium E_1^* are all derived by studying the system (1). From the stability analysis results, we can deduce that the endemic equilibrium, which indicates that viral respiratory diseases are persistently prevalent in the population, is globally asymptotically stable when $R_0 > 1$, and the disease-free equilibrium, which is where viral respiratory diseases are extinct, is globally asymptotically stable when $R_0 < 1$.

For system (2), it is demonstrated that when the threshold $R_1 < 1$ and pollutant inhalation $q = q_1$ are reached, an equilibrium E_0 exists where both viral and allergic respiratory diseases are eliminated. If either $R_1 > 1, q = q_1$ or $q < q_1$ or $R_1 > 1, q_1 < q < q_3$ holds and the stability condition of boundary equilibrium is satisfied, allergic respiratory disease will persist and viral respiratory disease will disappear. The existence of the endemic equilibrium is complicated; under certain conditions, it can coexist with the boundary equilibria (E_{31} and E_{32}) or exist alone. When $q_5 < q < q_8, R_1 < R_1^{**}$ or $q_3 < q < q_8, R_1 > R_1^{**}$,there is a unique endemic equilibrium E_4. In the case of inhalation $q_5 < q < min\{q_3, q_8\}$ and $R_1 > R_1^*$, E_4 coexists with E_{31} and E_{32}.

Sensitivity analysis of system (1) reveals that the number of patients seen and the number of patients not seen have a relatively high positive correlation with the infection rate β_1, the total number of patients not seen has a strong negative correlation with the cure rate (δ_h, δ_s, and δ_a), and and the number of patients seen and the number of patients not seen shows a positive and negative correlation with the proportion of patients seen K_3, respectively. In other words, when the infection rate β_1 increases, the number of patients seen and the number of patients not seen will increase; the higher the cure rate is, the lower the number of patients will be.

According to sensitivity analysis of system (2), the number of patients has a strong positive correlation with the air pollutant inhalation rate q; the number of viral patients I_v has a strong positive correlation with the natural clearance rate of pollutants c; the number of I_p has a strong negative correlation with both the natural clearance rate c and the cure rate δ_p; the number of unattended patients has a strong negative correlation with the cure rate δ_p; and the number of unattended patients has a strong negative correlation with the cure rate δ_p.The number of unattended patients has a strong correlation with the parameters K_0, K_3, as well as the cure rates δ_h, δ_s.

Numerical simulation allows us to observe an interesting phenomenon. Although higher levels of air pollution may trigger an epidemic of allergic respiratory disease, the increase in the proportion of susceptible individuals affected by air pollution at this time who become asymptomatic with respiratory viral infections (i.e., K_2 decreases) does not have a significant effect on the spread of allergic respiratory disease and viral respiratory disease. The peak number of patients I_s can rise as a result of a increase in the proportion of susceptible people not affected by air pollution who develop into symptomatic patients not seen (i.e., a increase in K_4). At lower air pollution levels, no allergic respiratory disease occurs, and at this time, the proportion of symptomatic patients with viral respiratory disease decreases, causing the peak number

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of symptomatic patients to increase with a weaker change than at higher air pollution levels. In conclusion, the proportion of I_s increases regardless of air pollution level, leading to an increase in the peak number of symptomatic patients, with the change in the peak number of patients being less pronounced at lower levels of air pollution than at higher levels of air pollution.

To sum up, the dynamic behavior of the system is more complex when the level of air pollution is high, and there are more equilibrium states, but due to the variation of pollutant inhalation, the disease-free equilibrium is only a more ideal state. Combined with the results of numerical simulation, a more realistic and feasible way to control the spread of viral respiratory diseases is to reduce the amount of pollutant inhalation, by wearing masks, reducing travel in bad weather, reducing pollutant emissions, and by increasing the natural clearance rate of air pollutants. In the case of low levels of air pollution, increasing the attendance rate, reducing the infection rate, and increasing the cure rate, can effectively inhibit the spread of viral respiratory diseases, such as by strengthening outpatient consultation, raising awareness of consultation, reducing contact with patients, improving medical care, and enhancing immunity.

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