Dynamic analysis and optimal control of a class of SISP respiratory diseases

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ABSTRACT
In this paper, the actual background of the susceptible population being directly patients after inhaling a certain amount of PM$_{2.5}$ is taken into account. The concentration response function of PM$_{2.5}$ is introduced, and the SISP respiratory disease model is proposed. Qualitative theoretical analysis proves that the existence, local stability and global stability of the equilibria are all related to the daily emission $P_0$ of PM$_{2.5}$ and PM$_{2.5}$ pathogenic threshold $K$. Based on the sensitivity factor analysis and time-varying sensitivity analysis of parameters on the number of patients, it is found that the conversion rate $\beta$ and the inhalation rate $\eta$ has the largest positive correlation. The cure rate $\gamma$ of infected persons has the greatest negative correlation on the number of patients. The control strategy formulated by the analysis results of optimal control theory is as follows: The first step is to improve the clearance rate of PM$_{2.5}$ by reducing the PM$_{2.5}$ emissions and increasing the intensity of dust removal. Moreover, such removal work must be maintained for a long time. The second step is to improve the cure rate of patients by being treated in time. After that, people should be reminded to wear masks and go out less so as to reduce the conversion rate of susceptible people becoming patients.

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Highlights

- The significant difference between this paper and other respiratory diseases models lies in that a compartment of air pollutant (PM$_{2.5}$) is added. At the same time, the emission and pathogenic amount of PM$_{2.5}$ are introduced, and the emission threshold value and pathogenic amount is given through theoretical analysis and numerical simulation. It has very important practical value for the development of public health and environmental protection.

- It is worth noting that the existence, local stability and global stability of the equilibria are all related to the daily emission $P_0$ of PM$_{2.5}$ and PM$_{2.5}$ pathogenic threshold $K$. This indicates that the emission of PM$_{2.5}$ and the pathogenic threshold have a great impact on the transmission dynamics of respiratory diseases, which further confirms the correctness of our consideration in the model.
This paper also proposes an optimal control strategy for the prevention and control of respiratory diseases. The first step is to improve the PM$_{2.5}$ clearance rate. Moreover, such removal work must be maintained for a long time. The second step is to improve the cure rate. After that is to reduce the conversion rate.

1. Introduction

The rapid development of the economy has promoted the process of industrialization in China, but at the same time, it also causes serious damage to the ecological environment, and the air pollution problem has become more and more serious [22, 24, 31, 32, 44]. Meanwhile, the impact of air pollution particles on human health has also become a public health problem. Air pollution particulate matter is the fifth-ranked health risk factor in the 2017 Global Burden of Disease Assessment [7, 14]. As is known to all, the fine particulate matter (PM$_{2.5}$) is an important part of the air pollution particulate matter. It can carry toxic and harmful substances, floating in the air for a long time, has a long transportation distance. It will enter the human body along with the breath, even into the alveoli and blood, making the respiratory system and other systems damage, leading to the occurrence of respiratory diseases and other diseases [5, 9, 12, 53]. PM$_{2.5}$ monitoring data of major cities in China in 2016 are shown in Figure 1 [40]. It can be seen that PM$_{2.5}$ monitoring concentration in some areas of Hebei Province is too high, indicating that local air pollution is relatively serious, the prevention and control of respiratory diseases in this region is still grim.

At present, the pathogenesis, transmission rules and prevention strategies of human respiratory diseases caused by air pollution have been investigated and studied by many scholars through mathematical modelling and combining with actual data [4, 11, 16, 36]. At the same time, many epidemiological studies have shown that PM$_{2.5}$ can avoid the nose hair, and the function of filtration by trachea cilia cells, along with the deep breathing into

![Figure 1. PM$_{2.5}$ concentration in major cities in China in 2016. The data came from a WHO report in September 2020 [40].](image-url)
the respiratory tract. In addition, it can be in direct contact with human lung tissue cells, and it is difficult to fall off when adsorbed in the alveolar, causing the deposition of particulate matter. Through direct stimulation, it causes mechanical damage to the airway mucosal epithelial cells and alveolar wall, destroys the respiratory defense barrier, increases the susceptibility of the body (especially in the elderly and children with poor resistance), and aggravates the inflammatory response of respiratory diseases. In short, PM$_{2.5}$ may cause harm to human health and even cause disease by oxide stimulation, physical and chemical reaction and mutagenesis [2, 10, 41, 49]. Therefore, it has become a hot topic to study the impact of PM$_{2.5}$ on respiratory diseases through mathematical modelling at present.

Under normal circumstances, because of factory emissions, automobile pollution, winter heating and other reasons, air pollutants will continue to exist in the natural environment, causing harm to people’s health. But the tiny particles that float in the air and can enter the lungs don’t necessarily cause respiratory problems when inhaled by the body. Respiratory disease outbreaks occur only if the PM$_{2.5}$ concentration inhaled by humans is higher than a certain critical threshold that makes susceptible people ill [23]. This critical pathogenic threshold may be caused by the clearance of the innate immune system. This kind of situation is similar to the case that the number of pathogens ingested by human beings must be higher than the critical threshold causing the infection of susceptible individuals in order to lead to the outbreak of infectious diseases [17]. In recent years, mathematical infectious disease models have been established by many scholars of introducing immune threshold or pathogen concentration threshold function to analyse the influence of such threshold on the dynamic behaviour [20, 52]. Currently, only the association between increased PM$_{2.5}$ concentrations and increased risk of respiratory diseases has been studied in the extensive epidemiological literatures [9, 42, 54]. The specific size of PM$_{2.5}$ pathogenic threshold and its impact on the incidence and transmission of respiratory diseases has not been explored in detail. Therefore, the concentration response function $\frac{P}{K+P}$ related to PM$_{2.5}$ is drawn into in this paper, and a respiratory disease model with PM$_{2.5}$ pathogenic threshold is installed and analysed.

Industrial sources, coal sources, motor vehicle sources, dust sources, biomass combustion sources, open sources are the main sources of PM$_{2.5}$ emissions in China. These sources will emit a large amount of PM$_{2.5}$ into the natural environment every day, which will not only affect the atmospheric environment and the ecosystem, but also cause great harm to human health [27]. PM$_{2.5}$ emissions from the cement industry of various provinces in China in 2013 are presented in Figure 2. It can be seen that PM$_{2.5}$ emissions of Anhui, Jiangsu, Hunan and other provinces in China this year have more than 400,000 tons [27, 47]. The amount of PM$_{2.5}$ emission from cement industrial sources alone is already quite huge. Combined with the irregular emission from other sources, the PM$_{2.5}$ concentration in these areas has seriously exceeded the standard. Long-term exposure of susceptible people to such seriously polluted air environment can easily lead to the outbreak of respiratory diseases [27, 29, 47]. Therefore, it has become urgent to study the influence of PM$_{2.5}$ emissions threshold on human health through mathematical modelling.

In recent years, the impact of PM$_{2.5}$ on respiratory diseases and human health is investigated by many scholars through using the modelling principle of infectious disease model [4, 11, 16, 36]. At the same time, on the basis of the traditional infectious disease model,
Figure 2. PM$_{2.5}$ emissions from cement industry in China in 2013. The data come from an IHME report in February 2020 [14].

the compartment of virus or bacterial population is added by many scholars to study the propagation dynamics of the model [1, 3, 6, 19, 28, 34, 39, 50]. However, little literature has brought the compartment of PM$_{2.5}$ in respiratory disease models to establish relevant mathematical models for analysis and research. Inspired by this, based on our previous work [33], a new compartment is added to describe the dynamic behaviour of PM$_{2.5}$, and a three-dimensional SISP respiratory disease model is proposed in this paper to study the impact of PM$_{2.5}$ emissions on the outbreak and spread of respiratory diseases.

The structure of this article as follows: In Section 1, the SISP respiratory disease model is built in which the susceptible population directly got sick after inhaling PM$_{2.5}$. In Section 2, the boundedness of the solution, the existence and stability of the equilibria are investigated. In Section 3, the sensitivity of parameters to the number of patients is presented. Section 4 is the optimal control analysis. Section 5 is the numerical simulation. Result and discussion are given at the end.

2. Mathematical modelling

In actual life, major emission source emit large amounts of PM$_{2.5}$ into the air daily, and let $P_0$ denote the daily emissions. The respiratory systems of susceptible people exposed to such polluted air can be damaged by inhaling PM$_{2.5}$, which can lead to respiratory diseases and make susceptible people become patients directly [5, 9, 12, 53]. In addition, a trace amount of PM$_{2.5}$ will not cause great harm to the human body due to the role of autoimmune function. Respiratory disease outbreaks only when the PM$_{2.5}$ concentration is higher than some pathogenic threshold $K$ [9, 23, 42, 54]. In this paper, the following SISP
A respiratory disease model is built by integrating the above two actual situations:

\[
\begin{align*}
\frac{dS}{dt} &= \Lambda - \mu S + \gamma I - \frac{\beta \eta SP}{K + P}, \\
\frac{dI}{dt} &= \frac{\beta \eta SP}{K + P} - \mu I - \alpha I - \gamma I, \\
\frac{dP}{dt} &= P_0 - cP - \eta (S + I).
\end{align*}
\]

(1)

Where, \(S\) represents the number of susceptible people, \(I\) represents the number of infected people, and \(P\) represents the PM\(_{2.5}\) concentration. \(\Lambda\) is the recruitment rate of susceptible persons, \(\mu\) is the natural mortality rate of susceptible and infected persons, \(\alpha\) is the disease-induced mortality rate of infected persons, and \(\gamma\) is the cure rate of infected persons. \(\beta\) is the conversion rate of susceptible individuals becoming patients by inhaling PM\(_{2.5}\). \(\frac{P}{K + P}\) is the concentration response function related to PM\(_{2.5}\), representing the probability of each susceptible person getting sick by inhaling PM\(_{2.5}\). \(c\) is the clearance rate for PM\(_{2.5}\) and \(\eta\) is the inhalation rate for PM\(_{2.5}\) per person.

3. Qualitative analysis

3.1. Boundedness of solutions

**Theorem 3.1:** The solutions \((S_0, I_0, P_0)\) of system (2.1) with initial value \(S(0) > 0, I(0) \geq 0, P(0) > 0\) are positive for all \(t > 0\). All solutions of system (2.1) are uniformly bounded on

\[
\Omega = \left\{(S, I, P) \in \mathbb{R}^3_+ : 0 \leq S + I = N \leq \frac{\Lambda}{\mu}, 0 \leq P \leq \frac{P_0}{c}\right\}.
\]

**Proof:** First, the total derivative of the function \(N(t) = S(t) + I(t)\) along the solution of system (1) can be obtained, we have

\[
\frac{dN(t)}{dt} = \frac{dS(t)}{dt} + \frac{dI(t)}{dt} = \Lambda - \mu [S(t) + I(t)] - \alpha I(t) \leq \Lambda - \mu N(t).
\]

So, we get \(N(t) \leq \frac{\Lambda}{\mu} - (\frac{\Lambda}{\mu} - N(0))e^{-\mu t}\) for all \(t \geq 0\). Hence, \(\lim_{t \to \infty} \sup N(t) \leq \frac{\Lambda}{\mu}\).

From the third equation of system (1), it can be known

\[
\frac{dP(t)}{dt} = P_0 - cP(t) - \eta [S(t) + I(t)] \leq P_0 - cP(t).
\]

Hence, we can obtain \(P(t) \leq \frac{P_0}{c} - (\frac{P_0}{c} - P(0))e^{-ct}\) for all \(t \geq 0\). Therefore \(\lim_{t \to \infty} \sup P(t) \leq \frac{P_0}{c}\).

To sum up, it can be concluded that the positive invariant set of system (1) is

\[
\Omega = \left\{(S, I, P) \in \mathbb{R}^3_+ : 0 \leq S + I = N \leq \frac{\Lambda}{\mu}, 0 \leq P \leq \frac{P_0}{c}\right\}.
\]
3.2. Existence of equilibria

Theorem 3.2: For system (1), we have:

1. There is a disease-free equilibrium $E_0 = (S_0, 0, 0)$ if $P_0 = \eta \frac{\Lambda}{\mu}$, here $S_0 = \frac{P_0}{\eta} = \frac{\Lambda}{\mu}$.
2. There is no endemic equilibrium if $K > K_1$, here
   \[ K_1 = \left\{ \frac{\sqrt{(\eta \Lambda - \mu P_0) [\beta \eta (\mu + \alpha) + \mu (\mu + \alpha + \gamma)]} - \sqrt{\beta \eta^2 \Lambda \alpha}}{c \mu^2 (\mu + \alpha + \gamma)} \right\}^2. \]
3. There is unique endemic equilibrium $E^*_1 = (S^*_1, I^*_1, P^*_1)$ if $P_0 < \eta \frac{\Lambda}{\mu}$, $K = K_1$, where
   \[ S^*_1 = \frac{\Lambda - (\mu + \alpha) I^*_1}{\mu}, \quad P^*_1 = \frac{\mu P_0 - \eta \Lambda + \eta \alpha I^*_1}{\mu c}, \quad I^*_1 = \frac{\eta \Lambda [\beta \eta (\mu + 2\alpha) + \mu (\mu + \alpha + \gamma)]}{2 \eta \alpha [\beta \eta (\mu + \alpha) + \mu (\mu + \alpha + \gamma)]} > 0. \]
4. There is an endemic equilibrium $E^*_2 = (S^*_2, I^*_2, P^*_2)$ if $\eta \frac{\Lambda}{\mu} < P_0 < \eta \frac{\Lambda}{\mu} (1 + \frac{\alpha}{\mu + \alpha})$, where
   \[ S^*_2 = \frac{\Lambda - (\mu + \alpha) I^*_2}{\mu}, \quad P^*_2 = \frac{\mu P_0 - \eta \Lambda + \eta \alpha I^*_2}{\mu c}, \quad I^*_2 = \frac{\eta \Lambda [\beta \eta (\mu + 2\alpha) + \mu (\mu + \alpha + \gamma)]}{2 \eta \alpha [\beta \eta (\mu + \alpha) + \mu (\mu + \alpha + \gamma)]} > 0. \]
5. There are two different endemic equilibria $E^*_2 = (S^*_2, I^*_2, P^*_2)$ and $E^*_3 = (S^*_3, I^*_3, P^*_3)$ if $P_0 < \eta \frac{\Lambda}{\mu}$, $K_5 < K < K_1$, where
   \[ K_5 = \frac{(\eta \Lambda - \mu P_0) [\beta \eta (\mu + \alpha) + \mu (\mu + \alpha + \gamma)]}{c \mu^2 (\mu + \alpha + \gamma)}, \quad S^*_3 = \frac{\Lambda - (\mu + \alpha) I^*_3}{\mu}, \quad P^*_3 = \frac{\mu P_0 - \eta \Lambda + \eta \alpha I^*_3}{\mu c}, \quad I^*_3 = \frac{\eta \Lambda [\beta \eta (\mu + 2\alpha) + \mu (\mu + \alpha + \gamma)]}{2 \eta \alpha [\beta \eta (\mu + \alpha) + \mu (\mu + \alpha + \gamma)]} - \sqrt{\Delta} > 0. \]

Proof: To solve the equilibrium of system (1), let
\[
\begin{align*}
\lambda - \mu S + \gamma I - \frac{\beta \eta S P}{K + P} &= 0, \\
\frac{\beta \eta S P}{K + P} - \mu I - \alpha I - \gamma I &= 0, \\
P_0 - c P - \eta (S + I) &= 0.
\end{align*}
\]
If $P = 0$, and $I = 0$, then $S = \frac{P_0}{\eta} = \frac{\Lambda}{\mu}$. Hence, system (1) has a disease-free equilibrium $E_0 = (S_0, 0, 0)$, where $S_0 = \frac{P_0}{\eta} = \frac{\Lambda}{\mu}$.
Add the first and second equations to get $S = \frac{\Lambda - (\mu + \alpha) I}{\mu}$. So we obtain $P = \frac{\mu P_0 - \eta \Lambda + \eta \alpha I}{\mu \sigma}$ from the third equation.

Substituting these two equations into the first equation, one has

$$AI^2 + BI + C = 0,$$

where

$$A = \eta \alpha [\beta \eta (\mu + \alpha) + \mu (\mu + \alpha + \gamma)],$$

$$B = [\beta \eta \mu P_0 (\mu + \alpha) + \mu^2 (\mu + \alpha + \gamma)(cK + P_0)] - \eta \Lambda [\beta \eta (\mu + 2 \alpha) + \mu (\mu + \alpha + \gamma)],$$

$$C = \beta \eta \Lambda (\eta \Lambda - \mu P_0),$$

$$\Delta = \left[\beta \eta \mu P_0 (\mu + \alpha) + \mu^2 (\mu + \alpha + \gamma)(cK + P_0)\right] - \eta \Lambda [\beta \eta (\mu + 2 \alpha) + \mu (\mu + \alpha + \gamma)]^2 \right]$$

$$-4 \beta \eta^2 \alpha \eta (\eta \Lambda) \left[\beta \eta (\mu + \alpha) + \mu (\mu + \alpha + \gamma)\right].$$

To take the existence of the endemic equilibrium $E^* = (S^*, I^*, P^*)$, then $S^*$, $P^*$ must be positive, and the roots $I^*$ of the quadratic Equation (2) must also be positive. Therefore, the quadratic Equation (2) has no real roots if $\Delta < 0$. The quadratic Equation (2) has one real root $I_1^* = -\frac{B}{2A} > 0 (B < 0)$ if $\Delta < 0$. The quadratic equation (2) has two different real roots $I_2^* = -\frac{B + \sqrt{\Delta}}{2A}$ and $I_3^* = -\frac{B - \sqrt{\Delta}}{2A}$ when $\Delta > 0$.

Let’s simplify the Equation (2) to $AI^2 + C = -BI$, and then the existence of the equilibrium in system (1) is discussed by using the symbolic-graphic combination.

1. There is only one intersection between functions $f_1(I) = AI^2 + C$ and $f_2(I) = -BI$ on the right side of $I$ if $B > 0$, $C > 0$.
2. There is no intersection between functions $f_1(I)$ and $f_2(I)$ on the right side of $I$ if $B < 0$, $C > 0$ and $\Delta$. There is an intersection $I_1^* = -\frac{B}{2A} > 0$ between functions $f_1(I)$ and $f_2(I)$ if $\Delta = 0$. There are two intersections $I_2^* = -\frac{B + \sqrt{\Delta}}{2A}$ and $I_3^* = -\frac{B - \sqrt{\Delta}}{2A}$ between functions $f_1(I)$ and $f_2(I)$ if $\Delta$.
3. There is only one intersection $I_2^* = -\frac{B + \sqrt{\Delta}}{2A}$ between functions $f_1(I)$ and $f_2(I)$ on the right side of $I$ if $B > 0$, $C < 0$.
4. There is only one intersection $I_2^* = -\frac{B + \sqrt{\Delta}}{2A}$ between functions $f_1(I)$ and $f_2(I)$ on the right side of $I$ if $B < 0$, $C < 0$.

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

1. System (1) has a disease-free equilibrium $E_0 = (S_0, 0, 0)$ if $P_0 = \eta \frac{\Delta}{\mu}$, and the factory emissions threshold is recorded as $P_0$, where $S_0 = \frac{P_0}{\eta} = \frac{\Lambda}{\mu}$.
2. When $P_0 \neq \eta \frac{\Delta}{\mu}$, two cases discussed below:
   (a) When $P_0 > \eta \frac{\Delta}{\mu}$, system has an endemic equilibrium $E_2^* = (S_2^*, I_2^*, P_2^*)$ if $\Delta > 0, \Lambda - (\mu + \alpha)I_2^* > 0, \mu P_0 - \eta \Lambda + \eta \alpha I_2^* > 0$. We know that $\Delta > 0$ and $\Lambda - (\mu + \alpha)I_2^* > 0$ are always true. And $\mu P_0 - \eta \Lambda + \eta \alpha I_2^* > 0$ is always true from $P_0 > \eta \frac{\Delta}{\mu}$. Then, we have $I_2^* < \frac{\Lambda}{\mu + \alpha}$ by $\Lambda - (\mu + \alpha)I_2^* > 0$. Therefore, $I_2^*$ is meaningful if and only if $\frac{\Lambda}{\mu + \alpha} > \frac{\mu P_0 - \eta \Lambda}{\eta \alpha}$ is true, and then there is an endemic
equilibrium $E^*_2$. So $\eta \frac{\Delta}{\mu} < P_0 < \eta \frac{\Delta}{\mu}(1 + \frac{\alpha}{\mu + \alpha})$ is a guarantee that $\frac{\Delta}{\mu + \alpha} > \frac{\mu P_0 - \eta \Delta}{\eta \alpha}$ is always true. Hence, system (1) has only one endemic equilibrium when $\eta \frac{\Delta}{\mu} < P_0 < \eta \frac{\Delta}{\mu}(1 + \frac{\alpha}{\mu + \alpha})$.

(b) When $P_0 < \eta \frac{\Delta}{\mu}(C > 0)$, there are two cases:

1. There is an endemic equilibrium $E^*_1 = (S^*_1, I^*_1, P^*_1)$ if $B < 0$,

   \[
   \Delta = 0, \left\{ \begin{array}{c}
   \frac{\Lambda - (\mu + \alpha) I^*_1}{\mu P_0 - \eta \Lambda + \eta \alpha I^*_1} > 0 \\
   \end{array} \right.
   \]

   We can acquire $K_1 = \frac{\sqrt{(\eta \Lambda - \mu P_0) [\beta \eta (\mu + \alpha) + \mu (\mu + \alpha + \gamma)]}}{\sqrt{\eta \Delta^2}}$ from $\Delta = 0$.

   And then we have $K < \frac{\beta \eta^2 \Lambda \alpha + (\eta \Lambda - \mu P_0) [\beta \eta (\mu + \alpha) + \mu (\mu + \alpha + \gamma)]}{\sqrt{\eta \Delta^2}} = K_2$ from $B < 0$.

   According to $\Lambda - (\mu + \alpha) I^*_1 > 0$ and $P_0 < \eta \frac{\Delta}{\mu}$, one has

   \[
   \eta \Lambda \beta \alpha (\mu + \alpha) + 2 \eta \Lambda \mu \alpha (\mu + \alpha + \gamma) + \mu^2 (\mu + \alpha) (\mu + \alpha + \gamma) cK \\
   > (\eta \Lambda - \mu P_0) \left[ \beta \eta (\mu + \alpha)^2 + \mu (\mu + \alpha) (\mu + \alpha + \gamma) \right] \\
   > \left( \frac{\eta \Lambda - \mu \Lambda \frac{\eta}{\mu}}{\eta \Lambda - \mu \frac{\eta}{\mu}} \right) \left[ \beta \eta (\mu + \alpha)^2 + \mu (\mu + \alpha) (\mu + \alpha + \gamma) \right] = 0.
   \]

   Then, $P_0 < \eta \frac{\Delta}{\mu}$ guarantees that $\Lambda - (\mu + \alpha) I^*_1 > 0$ is always true.

   And then we get $K < \frac{\beta \eta^2 \Lambda \alpha + (\eta \Lambda - \mu P_0) [\beta \eta (\mu + \alpha) + \mu (\mu + \alpha + \gamma)]}{\sqrt{\eta \Delta^2}} = K_3$ from $\mu P_0 - \eta \Lambda + \eta \alpha I^*_1 > 0$.

   In addition, we have $K = K_1$ from $K < \min(K_2, K_3) = K_2$ and $K = K_1 < K_2$.

   Hence, system has an endemic equilibrium $E^*_1$ if $P_0 < \eta \frac{\Delta}{\mu}$, $K = K_1$.

2. There are two distinct endemic equilibria $E^*_2 = (S^*_2, I^*_2, P^*_2)$ and $E^*_3 = (S^*_3, I^*_3, P^*_3)$ if $B < 0$,

   \[
   \Delta > 0, \left\{ \begin{array}{c}
   \frac{\Lambda - (\mu + \alpha) I^*_2}{\mu P_0 - \eta \Lambda + \eta \alpha I^*_2} > 0 \\
   \end{array} \right. \quad \text{and} \quad \left\{ \begin{array}{c}
   \frac{\Lambda - (\mu + \alpha) I^*_3}{\mu P_0 - \eta \Lambda + \eta \alpha I^*_3} > 0 \\
   \end{array} \right.
   \]

   According to $\Delta > 0$ and $P_0 < \eta \frac{\Delta}{\mu}$, one has

   \[
   \Delta = \left[ \left( \beta \eta \mu P_0 (\mu + \alpha) + \mu^2 (\mu + \alpha + \gamma) (cK + P_0) \right) \\
   - \eta \Lambda [\beta \eta (\mu + 2 \alpha) + \mu (\mu + \alpha + \gamma)] \right]^2 \\
   > 4 \beta \eta^2 \Lambda \alpha (\eta \Lambda - \mu P_0) [\beta \eta (\mu + \alpha) + \mu (\mu + \alpha + \gamma)] \\
   > 4 \beta \eta^2 \Lambda \alpha \left( \eta \Lambda - \mu \eta \frac{\Lambda}{\mu} \right) [\beta \eta (\mu + \alpha) + \mu (\mu + \alpha + \gamma)] = 0.
   \]

   Then, $P_0 < \eta \frac{\Delta}{\mu}$ is the guarantee that $\Delta > 0$ is always true.

   According to $C > 0$, we have $\mu P_0 - \eta \Lambda + \eta \alpha I^*_2 > 0$. Meanwhile $\mu P_0 - \eta \Lambda + \eta \alpha I^*_3 > 0$ by $I^*_2 > I^*_3$. 


Table 1. Existence of the equilibria in system (1).

| $P_0$ | $K$ | Equilibria |
|-------|-----|------------|
| $P_0 = \frac{\Lambda}{\mu}$ | – | Disease free equilibrium $E_0$ |
| $P_0 < \frac{\Lambda}{\mu}$ | $K_5 < K < K_1$ | Endemic equilibria $E_2^*$ and $E_3^*$ |
| $\frac{\Lambda}{\mu} < P_0 < \frac{\Lambda}{\mu}(1 + \frac{\alpha}{\mu + \alpha})$ | $K = K_1$ | Endemic equilibrium $E_1^*$ |
| $\frac{\Lambda}{\mu} < P_0 < \frac{\Lambda}{\mu}(1 + \frac{\alpha}{\mu + \alpha})$ | – | Endemic equilibrium $E_2^*$ |

Figure 3. Existence of the equilibria in system (1), here $P_1 = \frac{\eta \Lambda}{\mu}$, $P_2 = \frac{\Lambda}{\mu}(1 + \frac{\alpha}{\mu + \alpha})$.

On the basis of $\mu P_0 - \eta \Lambda + \eta \alpha I_2^* > 0$, we obtain

$$K < \frac{(\eta \Lambda - \mu P_0)[\beta \eta (\mu + \alpha) + \mu (\mu + \alpha + \gamma)] + (\eta \Lambda - \mu P_0)}{c \mu^2 (\mu + \alpha + \gamma)} = K_4.$$

From $\mu P_0 - \eta \Lambda + \eta \alpha I_3^* > 0$, we have $K > \frac{(\eta \Lambda - \mu P_0)[\beta \eta (\mu + \alpha) + \mu (\mu + \alpha + \gamma)]}{c \mu^2 (\mu + \alpha + \gamma)} = K_5$.

Moreover, $K_5 < K < \min\{K_2, K_4\} = K_4$. Comprehensive analysis shows that, system (1) has two endemic equilibria $E_2^*$ and $E_3^*$ if $P_0 < \frac{\Lambda}{\mu}$, $K_5 < K < K_1$.

According to the analysis results in Table 1 and Figure 3 above, it can be seen that the existence of the equilibria of the system (1) is influenced by the daily emission of PM$_{2.5}$ and the PM$_{2.5}$ pathogenic threshold. That is to say, by adjusting the PM$_{2.5}$ pathogenic threshold $K$ and the daily emission $P_0$ of PM$_{2.5}$, the existence of endemic equilibrium in the system can be controlled, so that system only has the disease-free equilibrium as far as possible, so as to reduce the harm of PM$_{2.5}$ and achieve the purpose of controlling the spread of respiratory diseases.
3.3. Local stability analysis

3.3.1. Local stability of disease-free equilibrium $E_0$ when $P_0 = \frac{\eta}{\mu}$

**Theorem 3.3:** The disease-free equilibrium $E_0$ is locally asymptotically stable when $K > \frac{\beta \eta^2 \Lambda \alpha}{c \mu^2 (\mu + \alpha + \gamma)}$.

**Proof:** The Jacobian matrix of system (1) evaluated at the equilibrium $E_0$ is given by

$$J(E_0) = \begin{pmatrix}
-\mu & \gamma & -\frac{\beta \eta S_0}{K} \\
0 & -\left(\mu + \alpha + \gamma\right) & \frac{\beta \eta S_0}{K} \\
-\eta & -\eta & -c
\end{pmatrix}.$$  

The corresponding characteristic equation is

$$\det (\lambda I - J(E_0)) = \lambda^3 + A_1 \lambda^2 + A_2 \lambda + A_3 = 0,$$

where

- $A_1 = (\mu + \alpha + \gamma) + \mu + c > 0$,
- $A_2 = (c + \mu)(\mu + \alpha + \gamma) + c \mu > 0$,
- $A_3 = c \mu (\mu + \alpha + \gamma) - \eta \frac{\beta \eta S_0}{K} \alpha$.

When $c \mu (\mu + \alpha + \gamma) - \eta \frac{\beta \eta S_0}{K} \alpha > 0$, we have $A_3 > 0$.

According to the Routh–Hurwitz criterion, if $K > \frac{\beta \eta^2 \Lambda \alpha}{c \mu^2 (\mu + \alpha + \gamma)}$, then the real parts of all eigenvalues of $E_0$ are negative. Therefore, $E_0$ is locally asymptotically stable. Theorem 3.3 can be obtained from the existence condition of disease-free equilibrium $E_0$. $\blacksquare$

3.3.2. Local stability of endemic equilibrium $E^*_1$ when $P_0 > \frac{\eta}{\mu}$, $K = K_1$

**Theorem 3.4:** The endemic equilibrium $E^*_1$ is locally asymptotically stable when $P_0 > \bar{P}$, where $\bar{P} = \eta \frac{\Lambda}{\mu} \cdot \frac{\beta \eta \alpha}{\mu (\mu + \alpha + \gamma)}$. 


**Proof:** The Jacobian matrix corresponding to the endemic equilibrium $E_1^*$ of system (1) is

$$J(E_1^*) = \begin{pmatrix} -\left(\mu + \frac{\beta \eta P_1^*}{K + P_1^*}\right) & \gamma & -\frac{\beta \eta P_1^*}{(K + P_1^*)^2} \\ \frac{\beta \eta P_1^*}{K + P_1^*} & -(\mu + \alpha + \gamma) & \frac{\beta \eta P_1^*}{(K + P_1^*)^2} \\ -\eta & -\eta & -c \end{pmatrix}.$$ 

The corresponding characteristic equation is

$$\det(\lambda I - J(E_1^*)) = \lambda^3 + B_1 \lambda^2 + B_2 \lambda + B_3 = 0,$$

where

$$B_1 = c + (\mu + \alpha + \gamma) + \left(\mu + \frac{\beta \eta P_1^*}{K + P_1^*}\right) > 0,$$

$$B_2 = c \left(\left(\mu + \frac{\beta \eta P_1^*}{K + P_1^*}\right) + (\mu + \alpha + \gamma)\right) + \left(\mu + \frac{\beta \eta P_1^*}{K + P_1^*}\right) (\mu + \alpha + \gamma) + \gamma \frac{\beta \eta P_1^*}{(K + P_1^*)^2} > 0,$$

$$B_3 = c \mu (\mu + \alpha + \gamma) + c (\mu + \alpha) \frac{\beta \eta P_1^*}{K + P_1^*} - \alpha \eta \frac{\beta \eta P_1^*}{(K + P_1^*)^2} > 0,$$

If $c \mu (\mu + \alpha + \gamma) + c (\mu + \alpha) \frac{\beta \eta P_1^*}{K + P_1^*} - \alpha \eta \frac{\beta \eta P_1^*}{(K + P_1^*)^2} > 0$, that is

$$P_0 > \frac{\eta \Delta}{\mu} \cdot \frac{\beta \eta P_1^*}{(K + P_1^*)^2} = \tilde{P},$$

then $B_3 > 0$.

Next, the sign of the real part of the eigenvalues is discussed by using the Routh–Hurtwitz criterion [13, 25]. According to the same proof method as Theorem 3.3, let

$$H_1 = B_1, \quad H_2 = \begin{vmatrix} B_1 & B_3 \\ 1 & B_2 \end{vmatrix}, \quad H_3 = \begin{vmatrix} B_1 & B_3 & 0 \\ 1 & B_2 & 0 \\ 0 & B_1 & B_3 \end{vmatrix}.$$ 

Through calculation, we have

$$H_1 = B_1 = c + (\mu + \alpha + \gamma) + \left(\mu + \frac{\beta \eta P_1^*}{K + P_1^*}\right) > 0,$$

$$H_2 = \left(\mu + \alpha + \gamma\right) + \left(\mu + \frac{\beta \eta P_1^*}{K + P_1^*}\right) \left[c + (\mu) + (\mu + \alpha) \frac{\beta \eta P_1^*}{K + P_1^*} + \alpha \eta \frac{\beta \eta P_1^*}{(K + P_1^*)^2}\right] > 0,$$

$$H_3 = B_3 H_2 > 0.$$

According to the Routh–Hurtwitz criterion, when $P_0 > \tilde{P}$, the real parts of all eigenvalues of $E_1^*$ are negative. Therefore, $E_1^*$ is locally asymptotically stable. Theorem 3.4 can be obtained by the existence condition of endemic equilibrium $E_1^*$.

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**3.3.3. Local stability of endemic equilibrium $E_2^*$ when $\eta \frac{\Delta}{\mu} < P_0 < \eta \frac{\Delta}{\mu} (1 + \frac{\alpha}{\mu + \gamma})$**

**Theorem 3.5:** If $K > \tilde{K}$, $P_0 < \tilde{P}$, the endemic equilibrium $E_2^*$ is locally asymptotically stable, where

$$\tilde{K} = \frac{\beta \eta^2 \Lambda \alpha + (\eta \Lambda - \mu P_0) [\beta \eta (\mu + \alpha) + 2(\mu + \alpha + \gamma)]}{c \mu (\mu + \alpha + \gamma)},$$

$$\tilde{P} = \frac{\beta \eta^2 \Lambda \alpha + (\eta \Lambda - \mu P_0) [\beta \eta (\mu + \alpha) + 2(\mu + \alpha + \gamma)]}{c \mu (\mu + \alpha + \gamma)}.$$
Proof: The Jacobian matrix corresponding to the endemic equilibrium $E^*_2$ of system (1) is

$$J(E^*_2) = \begin{pmatrix} -\left(\mu + \frac{\beta \eta P^*_2}{K + P^*_2}\right) & \gamma & -\frac{\beta \eta P^*_2}{(K + P^*_2)^2} \\ \frac{\beta \eta P^*_2}{K + P^*_2} & -\left(\mu + \alpha + \gamma\right) & \gamma \frac{\beta \eta P^*_2}{(K + P^*_2)^2} \\ -\eta & -\eta & -c \end{pmatrix}. $$

The corresponding characteristic equation is

$$\det(\lambda I - J(E^*_2)) = \lambda^3 + C_1\lambda^2 + C_2\lambda + C_3 = 0,$$

where

- $C_1 = c + (\mu + \alpha + \gamma) + \left(\mu + \frac{\beta \eta S^*_2}{K + P^*_2}\right) > 0,$
- $C_2 = c \left[\left(\mu + \frac{\beta \eta P^*_2}{K + P^*_2}\right) + (\mu + \alpha + \gamma)\right] + \left(\mu + \frac{\beta \eta P^*_2}{K + P^*_2}\right) (\mu + \alpha + \gamma) + \gamma \frac{\beta \eta S^*_2}{(K + P^*_2)^2} > 0,$
- $C_3 = c\mu (\mu + \alpha + \gamma) + c(\mu + \alpha) \frac{\beta \eta P^*_2}{K + P^*_2} - \alpha \eta \frac{\beta \eta S^*_2}{(K + P^*_2)^2}.$

If $K > \bar{K}$, $P_0 > \bar{P}$, then $B_0 > 0$, here

$$\bar{K} = \frac{\beta \eta^2 \Lambda \alpha + (\eta \Lambda - \mu P_0) \left[\beta \eta (\mu + \alpha) + 2 (\mu + \alpha + \gamma)\right]}{c\mu (\mu + \alpha + \gamma)},$$

$$\bar{\eta} = \frac{\Lambda}{\mu} < \bar{P} = \frac{\Lambda}{\mu} \left[1 + \frac{\beta \eta \alpha}{\beta \eta (\mu + \alpha) + 2 \mu (\mu + \alpha + \gamma)}\right] < \frac{\Lambda}{\mu} \left(1 + \frac{\alpha}{\mu + \alpha}\right).$$

Hence, the sign of the real part of the eigenvalues is explored by using the Routh–Hurtwitz criterion [13, 25]. According to the same proof method as Theorem 3.3 and 3.4, let

$$H_1 = C_1, \quad H_2 = \begin{vmatrix} C_1 & C_3 \\ 1 & C_2 \end{vmatrix}, \quad H_3 = \begin{vmatrix} C_1 & C_3 & 0 \\ 1 & C_2 & 0 \\ 0 & C_1 & C_3 \end{vmatrix}.$$

By calculating, we can get $H_1 > 0$, $H_2 > 0$, $H_3 > 0$.

According to the Routh–Hurtwitz criterion, if $K > \bar{K}$, $P_0 < \bar{P}$, then the real parts of all eigenvalues of $E^*_2$ are negative. Therefore, $E^*_2$ is locally asymptotically stable. Theorem 3.5 can be obtained by the existence condition of the endemic equilibrium $E^*_2$. 

### 3.3.4. Local stability of endemic equilibria $E^*_2$ and $E^*_3$ when $P_0 < \eta \frac{\Lambda}{\mu}, K_5 < K < K_1$

**Theorem 3.6:** If $K > \bar{K}$, $P_0 > \bar{P}$, the endemic equilibria $E^*_2$ and $E^*_3$ is locally asymptotically stable, where

$$\bar{K} = \frac{\beta \eta^2 \Lambda \alpha + (\mu P_0 - \eta \Lambda) \mu (\mu + \alpha + \gamma)}{c\mu^2 (\mu + \alpha + \gamma)}, \quad \bar{P} = \frac{\Lambda}{\mu} \cdot \frac{\beta \eta \alpha}{\mu (\mu + \alpha + \gamma)}.$$
Proof: The Jacobian matrix corresponding to the endemic equilibria $E_2^*$ and $E_3^*$ of system (1) is

$$
J(E_2^*, E_3^*) = \begin{pmatrix}
-\left(\mu + \frac{\beta \eta P_{2,3}^*}{K + P_{2,3}^*}\right) & \gamma & -\frac{\beta \eta P_{2,3}^*}{(K + P_{2,3}^*)^2} \\
\frac{\beta \eta P_{2,3}^*}{K + P_{2,3}^*} & -(\mu + \alpha + \gamma) & \frac{\beta \eta P_{2,3}^*}{(K + P_{2,3}^*)^2} \\
-\eta & -\eta & -c
\end{pmatrix}.
$$

By using the same proof method as Theorem 3.3, 3.4 and 3.5, according to Routh–Hurtwitz criterion, we know that the real parts of all eigenvalues of $E_2^*$ and $E_3^*$ are negative if

$$
K > \frac{\beta \eta^2 \Lambda \alpha + (\mu P_0 - \eta \Lambda) \mu (\mu + \alpha + \gamma)}{c \mu^2 (\mu + \alpha + \gamma)} = \bar{K}, \quad P_0 > \frac{\Lambda}{\mu} \cdot \frac{\beta \eta \alpha}{\mu (\mu + \alpha + \gamma)} = \bar{P}.
$$

While $\frac{\Lambda}{\mu} > P_0 > \frac{\Lambda}{\mu} \cdot \frac{\beta \eta \alpha}{\mu (\mu + \alpha + \gamma)}$, $K_1 > \bar{K} > K_3$. Hence, the endemic equilibria $E_2^*$ and $E_3^*$ is locally asymptotically stable. Theorem 3.6 can be obtained by the existence conditions of endemic equilibria $E_2^*$ and $E_3^*$.

3.4. Global stability analysis

In this section, the global stability of the equilibria of the system (1) will be proved by constructing the Lyapunov function and utilizing the LaSalle invariant set principle.

3.4.1. Global stability of disease-free equilibrium $E_0$ when $P_0 = \frac{\eta \Lambda}{\mu}$

Theorem 3.7: Assume that system parameters satisfy the conditions for Theorem 3.2, then the disease-free equilibrium $E_0$ of the system (1) is globally asymptotically stable in the positive invariant set $\Omega$ when $K > \frac{\beta \eta \Lambda}{c \mu}$.

Proof: First, let us construct the following Lyapunov function

$$
V(S, I, P) = \left(S - S_0 - S_0 \ln \frac{S}{S_0}\right) + I + P.
$$

Then, the total derivative of function $V(S, I, P)$ along the solution of system (1) is solved as follow

$$
\frac{dV(S, I, P)}{dt} = \left(1 - \frac{S_0}{S}\right) \dot{S} + \dot{I} + \dot{P} = \left(1 - \frac{S_0}{S}\right) \left[\Lambda - \mu S + \gamma I - \frac{\beta \eta S P}{K + P}\right] + \left[\frac{\beta \eta S P}{K + P} - (\mu + \alpha + \gamma)I\right]
+ [P_0 - cP - \eta (S + I)]
= \left(1 - \frac{S_0}{S}\right) (\Lambda - \mu S + \gamma I) + \left(\frac{\beta \eta S_0 P}{K + P} - cP\right)
- [(\mu + \alpha + \gamma) + \eta]I - \eta (S_0 - S)
$$
Suppose that system parameters satisfy the conditions for Theorem 3.2, then

Theorem 3.8: 3.4.2. Global stability of endemic equilibrium

the endemic equilibrium \( E^* \) of the system (1) is globally asymptotically stable in the positive invariant set \( \Omega \) when \( P_0 > \frac{\Lambda}{\mu (\mu + \alpha) K_1} \).

Proof: First, let us define the following Lyapunov function

\[
V(S, I, P) = \frac{1}{2} (S - S^*_1 + I - I^*_1)^2 + m \left( I - I^*_1 - I^*_1 \ln \frac{I}{I^*_1} \right) + \frac{1}{2} n (P - P^*_1)^2.
\]

Then, the total derivative of function \( V(S, I, P) \) along the solution of system (1) is calculated as follow

\[
\frac{dV(S, I, P)}{dt} = (S - S^*_1 + I - I^*_1)(\dot{S} + \dot{I}) + m \left( 1 - \frac{I^*_1}{I} \right) \dot{I} + n(P - P^*_1)\dot{P}
\]

\[
= (S - S^*_1 + I - I^*_1)[\Lambda - \mu S - (\mu + \alpha)I] + m \left( 1 - \frac{I^*_1}{I} \right) \left[ \frac{\beta \eta SP}{K + P} - (\mu + \alpha + \gamma)I \right] + n(P - P^*_1) \left[ P_0 - cP - \eta (S + I) \right).
\]

Combining the formulas \( \Lambda - \mu S^*_1 + \gamma I^*_1 - \frac{\beta \eta S^*_1 P^*_1}{K + P_1} = 0, \frac{\beta \eta S^*_1 P^*_1}{K + P_1} - (\mu + \alpha + \gamma)I^*_1 = 0, \)

\( P_0 - cP^*_1 - \eta (S^*_1 + I^*_1) = 0 \). We can get

\[
\dot{V} = -\mu(S - S^*_1)^2 - (\mu + \alpha)(I - I^*_1)^2 - ((\mu + \alpha) + \mu)(S - S^*_1)(I - I^*_1) + m\beta \eta \left( I - I^*_1 \right) \left[ \frac{PSK + P^*_1}{(K + P)(K + P^*_1)} - \frac{PS^*_1}{K + P^*_1} \right] - m(\mu + \alpha + \gamma) \left( I - I^*_1 \right)^2
\]

\[
- nc(P - P^*_1)^2 - n\eta (S - S^*_1)(P - P^*_1) - n\eta (I - I^*_1)(P - P^*_1)
\]

\[
\leq -\mu(S - S^*_1)^2 - (\mu + \alpha)(I - I^*_1)^2 - m(\mu + \alpha + \gamma) \left( I - I^*_1 \right)^2 - nc(I - I^*_1)^2
\]
Hence, let us introduce the following Lyapunov function

\[ V(S, I, P) = \frac{1}{2} (S - S_p^* + I - I_p^*)^2 + \omega_1 \left( I - I_p^* - \frac{I}{I_p^*} \ln \frac{I}{I_p^*} \right) + \frac{\omega_2}{2} (P - P_p^*)^2. \]

Then, the total derivative of function \( V(S, I, P) \) along the solution of system (1) is calculated as follow

\[
\frac{dV(S, I, P)}{dt} = (S - S_p^* + I - I_p^*) \dot{S} + I + m \left( 1 - \frac{I}{I_p^*} \right) \dot{I} + n(P - P_p^*) \dot{P}
\]

3.4.3. Global stability of endemic equilibrium \( E_p^* \) when \( \eta \frac{\Lambda}{\mu} < P_0 < \eta \frac{\Lambda}{\mu} (1 + \frac{\sigma}{\mu + \alpha}) \)

**Theorem 3.9:** Assume that system parameters satisfy the conditions for Theorem 3.2, then the endemic equilibrium \( E_p^* \) of the system (1) is globally asymptotically stable in the positive invariant set \( \Omega \) when \( P_0 < \hat{P} \), \( K > \hat{K} \), where \( \hat{P} = \eta \frac{\Lambda}{\mu} + \frac{\epsilon}{\beta \eta^2 \mu} \), \( \hat{K} = \frac{c \mu + (\mu P_0 - \eta \Lambda) \beta \eta^2 \Lambda}{\beta \eta^2 \mu} \).

**Proof:** First, let us introduce the following Lyapunov function

\[ V(S, I, P) = \frac{1}{2} (S - S_p^* + I - I_p^*)^2 + \omega_1 \left( I - I_p^* - \frac{I}{I_p^*} \ln \frac{I}{I_p^*} \right) + \frac{\omega_2}{2} (P - P_p^*)^2. \]

Then, the total derivative of function \( V(S, I, P) \) along the solution of system (1) is calculated as follow

\[
\frac{dV(S, I, P)}{dt} = (S - S_p^* + I - I_p^*) \dot{S} + I + m \left( 1 - \frac{I}{I_p^*} \right) \dot{I} + n(P - P_p^*) \dot{P}
\]
Remark 3.1: $\exists$ represents the existence of equilibria, LAS represents local asymptotic stability, and GAS represents global asymptotic stability.

From the perspective of controlling the spread of respiratory diseases, the global stability of the disease-free equilibrium is conducive to the prevention and control of respiratory diseases, while the global stability of the endemic equilibrium means long-term recurrence of respiratory diseases. According to the global stability analysis in this section and Table 2,
Table 2. The conditions for the existence, local stability and global stability of the equilibria of the system (1).

| Equilibria | LAS | GAS |
|------------|-----|-----|
| $E_0$      | $P_0 = \frac{\Lambda}{\mu}$ | $K > \frac{\beta \eta^2 \Lambda \alpha}{c \mu^2 (\mu + \alpha + \gamma)}$ | $K > \frac{\beta \eta \Lambda}{c \mu (\mu + \alpha) K_1}$ |
| $E_1^*$    | $K = K_1$ | $P_0 > \bar{p}$ | $P_0 > \frac{\Lambda c (2 \mu + \alpha)}{\mu (\mu + \alpha) K_1}$ |
| $E_2^*$ and $E_3^*$ | $P_0 < \frac{\Lambda}{\mu}, K < K_1$ | $K > \bar{K}, P_0 > \bar{p}$ | $P_0 > \frac{\Lambda c (2 \mu + \alpha)}{\mu (\mu + \alpha) K}$ |
| $E_4^*$    | $\frac{\Lambda}{\mu} < P_0 < \frac{\Lambda}{\mu} \left(1 + \frac{\alpha}{\mu + \alpha}\right)$ | $K > \bar{K}, P_0 < \bar{p}$ | $P_0 < \bar{p}, K > \bar{k}$ |

It can be seen that the main parameters affecting the global stability of the disease-free equilibrium are the daily emission $P_0$ of PM$_{2.5}$ and the PM$_{2.5}$ pathogenic threshold $K$. Parameters $P_0$ and $K$ can be adjusted to meet the conditions of global stability as far as possible to reduce the possibility of outbreak and transmission of respiratory diseases.

4. Sensitivity analysis

The variation of parameter values has a great influence on the transmission of respiratory diseases, especially on the basic reproduction number and the patients’ number [26, 45]. In this section, the significant impact factors of the patients’ number and the influence of time-varying sensitivity of multiple parameters on the patients’ number are mainly studied.

In order to elucidate the influence of simultaneous and large-scale changes of all parameters on the changes in the number of patients with respiratory diseases, Latin Hypercube Sampling and Partial Rank Correlation Coefficient are used to analyse the sensitivity of each parameter to the output variable (Number of infections $I$). The key parameters that need to be paid attention to about the influence of PM$_{2.5}$ on the number of patients are obtained, which provides the decision-making basis for designing a more reasonable control strategy of respiratory diseases [43, 45].

4.1. Sensitivity analysis of parameters to the number of patients

The range of parameter values is revealed in Table 3, and sampling time is $n = 2000$. In the sampling process, the setting parameter is the input variable and the number of patients $I$ is the output variable. If the absolute value of PRCC is larger, the influence of the parameter on $I$ will be greater [35, 43]. The dependence of the number of patients $I$ on each parameter is depicted in Figure 4.

The above PRCC analytical results indicated the dependence of the patients’ number $I$ on various parameters of the model [35, 43, 45]. From Table 3 and Figure 4, it can be seen that the influence of parameter $\Lambda$, $\beta$, $\eta$, $P_0$ on the number of patients $I$ has a high sensitivity and positive correlation. Where

$$\Lambda(|PRCC| = 0.3721), \quad \beta(|PRCC| = 0.3926),$$
$$\eta(|PRCC| = 0.3917), \quad P_0(|PRCC| = 0.2667)$$

has the most significant positive impact on $I$. Therefore, if the recruitment rate of susceptible persons is higher, the conversion rate of susceptible persons becomes patients after
Table 3. Parameters scope and PRCC value corresponding to $I$.

| Parameters | Range      | PRCC   | $p$-value |
|------------|------------|--------|-----------|
| $\Lambda$  | $[0.00005,0.005]$ | 0.3721 | $1.0273\times10^{-66}$ |
| $\mu$      | $[0.00006,0.005]$ | -0.3937 | $3.8870\times10^{-75}$ |
| $\gamma$   | $[0.05,0.3]$ | -0.2752 | $4.3426\times10^{-36}$ |
| $\beta$    | $[0.0001,0.01]$ | 0.3926 | $1.0370\times10^{-74}$ |
| $\eta$     | $[0.0001,0.01]$ | 0.3917 | $2.4061\times10^{-74}$ |
| $K$        | $[0.000008,0.001]$ | -0.1666 | $6.3926\times10^{-14}$ |
| $\alpha$   | $[0.000001,0.01]$ | -0.0453 | $0.0426$ |
| $P_0$      | $[0.000001,0.01]$ | 0.2667 | $6.6169\times10^{-34}$ |
| $c$        | $[0.000001,0.01]$ | -0.1265 | $1.3714\times10^{-08}$ |

Figure 4. The significance analysis diagram of parameters to $I$.

inhaling PM$_{2.5}$ is higher, and the greater the daily emission of PM$_{2.5}$, then the number of patients will be higher.

In addition, the influence of parameters $\mu$, $\gamma$, $K$, $\alpha$, $c$ on the number of patients are negatively correlated. Moreover,

$$\mu(\{|PRCC| = 0.3937\}), \quad \gamma(\{|PRCC| = 0.2752\}),$$
$$K(\{|PRCC| = 0.1666\}), \quad c(\{|PRCC| = 0.1265\})$$

has the most significant negative impact on $I$ [26, 43, 45]. It can be seen that if the natural mortality rate of susceptible and infected people is higher, the cure rate of infected people is higher. The clearance rate for PM$_{2.5}$ is higher, and the PM$_{2.5}$ pathogenic threshold is higher, the number of patients will be lower. Although $\alpha(\{|PRCC| = 0.0453\})$ has a negative correlation with the number of patients $I$, its influence result is not significant within the hypothesis range of this paper.

4.2. Time-varying sensitivity analysis of parameters to the number of patients

Time-varying sensitivity refers to the influence of parameters on the output variable $I$ as it changes over time. Therefore, time-varying sensitivity analysis can evaluate the incidence
and dependence of a parameter variable in a dynamic model on the output variable over a period of time [38, 46].

Since the outbreak period and time quantum of different diseases are different, the influence of parameters on diseases also changes with time [38, 51]. Based on the pathogenetic process of respiratory diseases, time-varying sensitivity analysis of parameters to the number of patients is considered from two aspects, continuous time period and single point time, respectively.

### 4.2.1. Time-varying sensitivity analysis of parameters to the number of patients in a continuous period

This section mainly studies the time-varying sensitivity analysis of parameters to the number of patients within a continuous period of 0–50. The results are shown in Figure 5, where parameter is the input variable, and the number of patients is the output variable. The range of grey interval is [-0.05,0.05], indicating that the change of the parameter in this region has no significant impact on the patients’ number $I$ [38, 46, 51].

From Figure 5, it is found that the parameters change significantly in the early stage in the pathogenesis of respiratory diseases, especially before the time $t = 10$. With the change of time, the parameters $\mu$, $\gamma$, $K$, $\alpha$, $c$ indicated negative correlation with the number of patients $I$, and maintained the negative effect. PM$_{2.5}$ pathogenic threshold $K$ has a large negative correlation, and the negative influence gradually increases and finally stabilizes. The clearance rate $c$ for PM$_{2.5}$ also has a large negative correlation with $I$, and its negative effect on $I$ decreases first, then increases and finally becomes stable. The cure rate $\gamma$ of infected persons has a great negative correlation, and its negative effect on $I$ first slightly decreases, then presents a large increase and finally stabilizes. In addition, although there is also a large negative correlation between the natural mortality rate $\mu$ and the disease-induced mortality rate $\alpha$ of infected persons, it is ignored under the assumptions of this paper.

![Figure 5. Time-varying sensitivity analysis of parameters to the number of patients $I$.](image-url)
Furthermore, parameters $\Lambda, \beta, \eta, P_0$ proved positive correlation with the number of patients $I$, and maintained the positive effect. The recruitment rate $\Lambda$ of susceptible persons has a significant positive correlation with $I$, and its positive effect increases significantly first and tends to be stable at last. The conversion rate $\beta$ and the inhalation rate $\eta$ for PM$_{2.5}$ prove the significant positive correlation, and its positive influence on $I$ decreases significantly first, then increasing and finally becomes stable. The daily emission $P_0$ of PM$_{2.5}$ has a large positive correlation, and its positive impact on $I$ decreases first, then increases and finally becomes stable.

Therefore, in order to effectively control the number of patients with respiratory diseases over a continuous period of time, the negative effects of clearance rate $c$ for PM$_{2.5}$ and the cure rate $\gamma$ of infected persons on $I$ should be mainly paid attention. Meanwhile, the positive effects of the conversion rate $\beta$ of susceptible persons become patients after inhaling PM$_{2.5}$, the inhalation rate $\eta$ for PM$_{2.5}$ and the daily emission $P_0$ of PM$_{2.5}$ on $I$ should be mainly paid attention.

4.2.2. Time-varying sensitivity analysis of parameters to the number of patients at a single point

From Figure 5, it is found that the incidence and dependence of different parameters on the number of patients $I$ in a continuous period from 0 to 50. However, at a certain moment, different parameters have a different impact on the number of patients $I$. Therefore, the influence of parameters on state variables at a certain moment will be proposed in this section. The impact of parameters on the number of patients $I$ at the time $t = 10$ is analysed as revealed in Figure 6–14. Where, the abscissa represents the residual of the linear regression between the sorted parameter and remaining parameters, the ordinate represents the residual of linear regression between the number of patients $I$ after sorting and other parameters [38, 46, 51].

As shown in Figures 6–14, the monotony of parameters $\Lambda, \gamma, \alpha, K$ is the most significant at the time $t = 10$, that is, patients with respiratory diseases are mainly influenced by the changes of these parameters at this time. Where, parameters $\Lambda, \gamma, \alpha$ shows the monotonously increasing situation, that is, the recruitment rate of susceptible people, the cure rate of patients and the mortality rate of infected people have the positive impact on patients with respiratory diseases. The parameter $K$ shows a monotonously decreasing situation, that is, the PM$_{2.5}$ pathogenic threshold at this moment has a negative impact on the number of patients $I$. In addition, the parameters $\beta, \eta, \mu, P_0$ present the relatively weak monotonously decreasing situation, and the parameter $c$ presents a relatively weak monotonously increasing situation. This indicates that these parameters are not sensitive to the influence of patients with respiratory diseases at $t = 10$.

To sum up, the parameters have different sensitivities to the number of patients with respiratory diseases at $t = 10$. From Figures 6–14, it is found that parameters $\beta, \eta, c, P_0$ are insensitive to the number of patients at the time $t = 10$, that is, the conversion rate of susceptible individuals becomes patients by inhaling PM$_{2.5}$, the inhalation rate for PM$_{2.5}$, the clearance rate for PM$_{2.5}$ and the daily emission $P_0$ of PM$_{2.5}$ have no significant influence on the number of patients at that time. Therefore, we suggest that it is of great significance to control the outbreak and transmission of respiratory diseases by adjusting more sensitive parameters for a different number of patients at a certain time.
5. Optimal control

It is well known that the control of respiratory diseases by means of constant control cannot achieve the desired effect. This is because of control parameters are time-dependent, and in order to achieve effective disease control within a limited time frame, time-related control
strategies need to be considered [15, 48]. In this section, reducing conversion rate, increasing treatment rate and improving clearance rate are considered time-dependent controls over a limited time. It is striving to reduce the number of patients at the lowest possible
control cost, and enable to decrease the PM$_{2.5}$ emissions, and ultimately achieve the goal of controlling the outbreak and spread of respiratory diseases.

First of all, three control functions related to time are introduced into the model (1), in which the control function $u_1(t)$ represented the enhanced treatment rate of patients.
The specific implementation strategy is to identify the cause of the symptoms and go to a regular hospital for medical treatment. The control function $u_{2}(t)$ represents the reduced conversion rate of susceptible persons become patients by inhalation PM$_{2.5}$. The specific measures are to strengthen physical exercise to improve their resistance, and wear masks to
reduce the inhalation of susceptible people to PM$_{2.5}$. The control function $u_3(t)$ represents the enhanced clearance rate for PM$_{2.5}$. The specific implementation strategy is to develop strict industrial emission standards, increase the intensity of dust removal. The control function $u_1(t)$ is to promote the healing from the perspective of curing patients, the control function $u_2(t)$ is to decrease the transmission of respiratory diseases from the perspective of cutting off the transmission route, the control function $u_3(t)$ is to reduce the outbreak of respiratory diseases from the perspective of removing PM$_{2.5}$. Finally, the model (1) is transformed into the following form

\[
\begin{align*}
\frac{dS}{dt} &= \Lambda - \mu S + \gamma (1 + u_1(t)) I - \beta (1 - u_2(t)) \frac{\eta SP}{K + P}, \\
\frac{dI}{dt} &= \beta (1 - u_2(t)) \frac{\eta SP}{K + P} - \mu I - \alpha I - \gamma (1 + u_1(t)) I, \\
\frac{dP}{dt} &= P_0 - c (1 + u_3(t)) P - \eta (S + I),
\end{align*}
\]  

(3)

First, the objective function corresponding to model (3) is proposed as follows

\[
J(u_1(t), u_2(t), u_3(t)) = \min_{u_1, u_2, u_3} \int_{0}^{T_f} \left[ a_1 I(t) + a_2 P(t) + bu_1^2(t) + c_1 u_2^2(t) + du_3^2(t) \right] dt
\]  

(4)

where $a_1, a_2 (0 < a_1, a_2 < 1)$, $b, c_1, d$ are positive weighting coefficient, $a_1 I(t)$ represents the number of infected persons, $a_2 P(t)$ is the concentration of PM$_{2.5}$. The item $bu_1^2$, $c_1 u_2^2$ and $du_3^2$ represent the cost corresponding to the cure control, conversion control and clearance control, respectively. Here, the control variable is squared to eliminate side
effects [15, 30]. For the objective function \( J(u_1(t), u_2(t), u_3(t)) \), the optimal control function \((u_1^*(t), u_2^*(t), u_3^*(t))\) needs to be sought to make the objective function reach the minimum value, that is, to minimize the total cost of controlling the outbreak and spread of respiratory diseases and reducing the emission of PM\(_{2.5}\), that is,

\[
J \left( u_1^* (t) , u_2^* (t) , u_3^* (t) \right) = \min \{ J (u_1 (t), u_2 (t), u_3 (t)) : (u_1 (t), u_2 (t), u_3 (t)) \in \phi \} 
\]

(5)

here, \( \phi = \{ u_i (t) : 0 \leq u_i (t) \leq 1, i = 1, 2, 3; 0 \leq t \leq t_f \}; u_i (t) \) is Lebesgue measurable.

To obtain the optimal solution, let us define the following Lagrangian function

\[
L (I, P, u_i) = a_1 I (t) + a_2 P (t) + bu_1^2 (t) + c_1 u_2^2 (t) + du_3^2 (t) .
\]

(6)

Then, the Pontryagin’s Maximal Principle is applied to determine the conditions under which effective control of respiratory diseases can be achieved in a limited time. This principle transforms the Equations (3), (4), (5), and (6) into Hamiltonian point-state minimization of the control function \( u_1 (t), u_2 (t), u_3 (t) \) [8, 30].

Hence, the Hamiltonian function is defined as follows

\[
H(t, X, U, \lambda) = L + \lambda_1 (t) \frac{dS}{dt} + \lambda_2 (t) \frac{dI}{dt} + \lambda_3 (t) \frac{dP}{dt}
\]

\[
= a_1 I (t) + a_2 P (t) + bu_1^2 (t) + c_1 u_2^2 (t) + du_3^2 (t)
\]

\[
+ \lambda_1 (t) \left\{ \Lambda - \mu S + \gamma (1 + u_1 (t)) I - \beta (1 - u_2 (t)) \frac{\eta SP}{K + P} \right\}
\]

\[
+ \lambda_2 (t) \left\{ \beta (1 - u_2 (t)) \frac{\eta SP}{K + P} - \mu I - \alpha I - \gamma (1 + u_1 (t)) I \right\}
\]

\[
+ \lambda_3 (t) \left\{ P_0 - c (1 + u_3 (t)) P - \eta (S + I) \right\} .
\]

(7)

Where, \( X(t) = (S(t), I(t), P(t)), U(t) = (u_1 (t), u_2 (t), u_3 (t)), \lambda_i (t) (i = 1, 2, 3) \) is the adjoint variable.

**Theorem 5.1:** Assume that \((X^*(t), U^*(t))\) is the optimal solution of the corresponding control system, there exists a vector function \( \lambda(t) = (\lambda_1 (t), \lambda_2 (t), \lambda_3 (t)) \) that satisfies the following equation

\[
\begin{align*}
\dot{\lambda}_1 (t) &= \mu \lambda_1 (t) + (\lambda_1 (t) - \lambda_2 (t)) \left( \beta (1 - u_2 (t)) \frac{\eta P}{K + P} \right) + \lambda_3 (t) \eta, \\
\dot{\lambda}_2 (t) &= -a_1 + (\lambda_2 (t) - \lambda_1 (t)) \left[ \gamma (1 + u_1 (t)) \right] + \lambda_2 (t) (\mu + \alpha) + \lambda_3 (t) \eta, \\
\dot{\lambda}_3 (t) &= -a_2 + (\lambda_1 (t) - \lambda_2 (t)) \beta (1 - u_2 (t)) \frac{\eta SK}{(K + P)^2} + \lambda_3 (t) c (1 + u_3 (t)) ,
\end{align*}
\]

(8)

and it has a transversality condition

\[
\lambda_i (t_f) = 0, \quad i = 1, 2, 3.
\]

(9)
Moreover, the optimal control function is given

\[
\begin{align*}
  u_1^* (t) &= \max \left\{ 0, \min \left( 1, \tilde{u}_1 \right) \right\}, \\
  u_2^* (t) &= \max \left\{ 0, \min \left( 1, \tilde{u}_2 \right) \right\}, \\
  u_3^* (t) &= \max \left\{ 0, \min \left( 1, \tilde{u}_3 \right) \right\},
\end{align*}
\]

(10)

where \( \tilde{u}_1 = \frac{\gamma (\lambda_2 - \lambda_1) \bar{I}}{2b} \), \( \tilde{u}_2 = \frac{(\lambda_2 - \lambda_1) \beta \eta \bar{S} \bar{P}}{2c_1 (K + \bar{P})} \), \( \tilde{u}_3 = \frac{c \bar{P} \lambda_3}{2d} \).

**Proof:** According to the Pontryagin’s Maximum Principle and the existence of the optimal control pair \([8, 37]\), the following conclusions can be found that

\[
\begin{align*}
  \frac{\partial H (t, X, U, \lambda)}{\partial U} &= 0, \quad \text{(optimality condition)} \\
  \lambda' &= \frac{\partial H (t, X, U, \lambda)}{\partial U}, \quad \text{(adjoint condition)} \\
  \lambda (t_f) &= 0, \quad \text{(transversality condition)}
\end{align*}
\]

(11)

Then, according to the adjoint condition, the partial derivative of the Hamiltonian function for the associated state equation with \( X = X^* \) is calculated, and one has

\[
\begin{align*}
  \dot{\lambda}_1 (t) &= \frac{\partial H (t, X, U, \lambda)}{\partial S}, \\
  \dot{\lambda}_2 (t) &= \frac{\partial H (t, X, U, \lambda)}{\partial I}, \\
  \dot{\lambda}_3 (t) &= \frac{\partial H (t, X, U, \lambda)}{\partial P},
\end{align*}
\]

(12)

To minimize the Hamiltonian function under optimal control, and the function \( H(t, X, U, \lambda) \) is differentiated on \( \phi \) with respect to \( u_1, u_2, u_3 \), and is equivalent to zero. Then, the following solutions are proved:

\[
\begin{align*}
  \frac{\partial H (t, X, U, \lambda)}{\partial u_1} &= 0 \text{ gets } \tilde{u}_1 = \frac{\gamma (\lambda_2 - \lambda_1) \bar{I}}{2b}, \\
  \frac{\partial H (t, X, U, \lambda)}{\partial u_2} &= 0 \text{ acquires } \tilde{u}_2 = \frac{(\lambda_2 - \lambda_1) \beta \eta \bar{S} \bar{P}}{2c_1 (K + \bar{P})}, \\
  \frac{\partial H (t, X, U, \lambda)}{\partial u_3} &= 0 \text{ gains } \tilde{u}_3 = \frac{c \bar{P} \lambda_3}{2d}.
\end{align*}
\]

Where \( \bar{S}, \bar{I}, \bar{P} \) are the optimal solutions corresponding to \( S, I, P \), respectively, and \( \{\lambda_1, \lambda_2, \lambda_3\} \) is the solution of system (12).
Then, according to the standard control parameters about control bounds, it can be obtained by calculation

\[
\begin{cases}
0, & \frac{\gamma (\bar{\lambda}_2 - \bar{\lambda}_1) \bar{I}}{2b} \leq 0, \\
\frac{\gamma (\bar{\lambda}_2 - \bar{\lambda}_1) \bar{I}}{2b}, & 0 < \frac{\gamma (\bar{\lambda}_2 - \bar{\lambda}_1) \bar{I}}{2b} < 1, \\
1, & \frac{\gamma (\bar{\lambda}_2 - \bar{\lambda}_1) \bar{I}}{2b} \leq 1
\end{cases}
\]

its compact form can be expressed as \( u_1^* = \max\{0, \min\{1, \frac{\gamma (\bar{\lambda}_2 - \bar{\lambda}_1) \bar{I}}{2b}\}\} \).

Similarly, the compact forms of \( u_2^* \) and \( u_3^* \) can be, respectively, expressed as

\[
u_2^* = \max \left\{ 0, \min \left( 1, \frac{(\bar{\lambda}_2 - \bar{\lambda}_1) \beta \eta \bar{S} \bar{P}}{2c_1 (K + \bar{P})} \right) \right\},
\]

\[
u_3^* = \max \left\{ 0, \min \left( 1, \frac{c \bar{P} \lambda_3}{2d} \right) \right\}.
\]

6. Numerical simulation

In this section, we conduct numerical simulation of the optimal control of respiratory diseases. The parameters are selected in the following Table 4. The weight in the objective function is \( a_1 = 0.8, a_2 = 0.5, b = 0.2, c = 0.006, d = 1000 \).

From Figure 15, it is illustrated that the effectiveness of the optimal control. Respiratory diseases will break out very quickly in a very short period if no control strategies are implemented. Under the optimal control strategy, the prevalence of respiratory diseases declined rapidly and remained at a very low level.

Figure 16 illustrates how the control strategy changes over time. In the whole pathogenesis process of respiratory diseases, improving the cure rate, reducing the conversion rate and improving the clearance rate are very helpful to reduce the incidence of respiratory diseases. As can be seen from Figure 17, in the early stage, with the reduction of the Lagrangian function, the clearance rate of PM2.5 began to decrease. Around the fifth day, the treatment rate for the patients began to decrease dramatically as the Lagrangian function decreased sharply. At about day 18, the conversion rate began to slow down as the

| Table 4. Parameter values in simulation. |
|------------------------------------------|
| Parameter | Definition | Value | Reference |
| --- | --- | --- | --- |
| \( \Lambda \) | the recruitment rate of susceptible persons | 0.05 | [13, 14, 39, 40] |
| \( \mu \) | the natural mortality rate of susceptible and infected persons | 0.01428 | [13, 14, 39, 40] |
| \( \alpha \) | the disease-induced mortality rate of infected persons | 0.02 | [13, 14, 39, 40] |
| \( \beta \) | the conversion rate of susceptible individuals become patients by inhaling PM2.5 | 0.002 | [13, 14, 39, 40] |
| \( \eta \) | the inhalation rate for PM2.5 per person | 0.01 | Estimated |
| \( \gamma \) | the cure rate of infected persons | 0.04 | [13, 14, 39, 40] |
| \( K \) | the PM2.5 pathogenic threshold | 100 | [25, 26, 27] |
| \( P_0 \) | the daily emissions of PM2.5 | 190 | [28, 29, 30] |
| \( c \) | the clearance rate for PM2.5 | 0.9 | Estimated |
Figure 15. Prevalence of respiratory diseases under optimal control strategies.

Figure 16. Diagram of control strategy $u_1, u_2, u_3$ over time. Control strategies of strengthening the treatment of patients (red line); Control strategies to reduce conversion of susceptible persons to patients (blue line); Control strategies of enhancing removal for $PM_{2.5}$ (green line).
Lagrangian function decreased. In the final stage of about 195 days, as the Lagrangian function rapidly reaches its minimum, the clearance rate of PM$_{2.5}$ and the conversion rate also decrease rapidly. Finally, all the control strategy functions return to 0 when the transversal condition is $\lambda_i(t_f) = 0$. Also, the arrows in Figure 16 and 17 are paired at each turning point.

By comparing Figure 16 and 17, it can be seen that with regard to the reduction of the cost function, in the short term, the control of the treatment of patients and the conversion control of susceptible people becomes patients are more effective than the control of PM$_{2.5}$ clearance. From the long-term perspective of the control of respiratory diseases, the control strategy for PM$_{2.5}$ removal should always be unremitting.

7. Conclusion and discussion

Under normal circumstances, polluted air contains a large amount of fine particulate matter (PM$_{2.5}$). After PM$_{2.5}$ is inhaled by susceptible people exposed to polluted air, the human respiratory system will be damaged and various respiratory diseases will be caused. In this paper, the actual situation that the susceptible population directly fell ill after inhaling PM$_{2.5}$ is taken into account. The SISP respiratory disease model is built by introducing the PM$_{2.5}$ pathogenic threshold, and adding a differential equation describing the dynamic change of PM$_{2.5}$. The influence of daily emission of PM$_{2.5}$ and PM$_{2.5}$ pathogenic threshold on the incidence and spread of respiratory diseases are studied, so as to provide an optimal control strategy for the outbreak and spread of respiratory diseases.

According to the existence analysis of the equilibrium in Table 1, it can be seen that the system (1) has a disease-free equilibrium $E_0$ if the daily emission $P_0$ of PM$_{2.5}$ is a constant.
Obviously, this is unrealistic, because PM$_{2.5}$ has many emission sources, and the new emissions change every day. There are two endemic equilibria $E_2^*$ and $E_3^*$ in the system when the daily emission $P_0$ of PM$_{2.5}$ is less than $\eta \frac{\Lambda}{\mu}$ and the PM$_{2.5}$ pathogenic threshold is between $K_5$ and $K_1$. These two endemic equilibria will degenerate into one endemic equilibrium $E_1^*$, and once the pathogenic threshold is $K_1$. The system only has an endemic equilibrium $E_2^*$ when the emission $P_0$ of PM$_{2.5}$ is between $\eta \frac{\Lambda}{\mu}$ and $\eta \frac{\Lambda}{\mu} (1 + \frac{\alpha}{\mu + \alpha})$. From the above analysis, it can be seen that the daily emission $P_0$ of PM$_{2.5}$ and the size of PM$_{2.5}$ pathogenic threshold have the great influence on the existence of the equilibria. In system (1), the existence of disease-free equilibrium is only an ideal state, while the existence of endemic equilibrium is the normal state. Therefore, the long-term exposure of the susceptible population in the air environment of excessive PM$_{2.5}$ emissions will easily induce the outbreak of respiratory diseases.

From the analysis results of the local and global stability of the system equilibrium in Table 2, on the premise that the existence condition of the system equilibrium is true, it is found that the disease-free equilibrium $E_0$ is locally asymptotically stable if the PM$_{2.5}$ pathogenic threshold $K$ is greater than $\frac{\beta \eta^2 \Lambda \alpha}{c \mu^2 (\mu + \alpha + \gamma)}$. The globally asymptotically stable occurs when $P_0$ is greater than $\bar{P}$ and PM$_{2.5}$ pathogenic threshold $K$ is greater than $\bar{K}$, the endemic equilibria $E_2^*$ and $E_3^*$ are locally asymptotically stable. The global asymptotic stability occurs when the daily emission $P_0$ of PM$_{2.5}$ is greater than $\bar{P}$ and PM$_{2.5}$ pathogenic threshold $K$ is $K_1 = \bar{K}$, the endemic equilibrium $E_1^*$ is locally asymptotically stable. The global asymptotic stability occurs when the daily emission $P_0$ of PM$_{2.5}$ is greater than $\frac{\Lambda c (2\mu + \alpha)}{\mu (\mu + \alpha) K_1}$. When the daily emission $P_0$ of PM$_{2.5}$ is less than $\bar{P}$ and PM$_{2.5}$ pathogenic threshold $K$ is greater than $\bar{K}$, the endemic equilibrium $E_2^*$ is locally asymptotically stable. The global asymptotically stable occurs when $P_0$ is less than $\bar{P}$ and $K$ is greater than $\bar{K}$. It is easy to know from the theoretical analysis that the daily emission $P_0$ of PM$_{2.5}$ and PM$_{2.5}$ pathogenic threshold $K$ have the strong influence on the stability of equilibrium. Where, the equilibrium can reach local or global asymptotic stability only if the daily emission $P_0$ of PM$_{2.5}$ is within a certain range. The higher the PM$_{2.5}$ pathogenic threshold $K$ is, the easier it is for the endemic equilibrium to reach stability, which is not conducive to the control of respiratory diseases.

According to the above sensitivity theoretical analysis, it can be seen that the influence of parameters $\Lambda$, $\beta$, $\eta$, $P_0$ on the number of patients $I$ have the high sensitivity and positive correlation. The influence of parameters $\mu$, $\gamma$, $K$, $\alpha$, $c$ on the number of patients $I$ are negatively correlation. That is to say, the greater the recruitment rate of susceptible people, the greater the conversion rate of susceptible people becomes patients after inhaling PM$_{2.5}$, the greater PM$_{2.5}$ inhalation rate, and the greater the daily emission $P_0$ of PM$_{2.5}$, the more patients will be. The greater the natural mortality rate of susceptible and infected people, the greater the cure rate of infected people, the greater the clearance rate for PM$_{2.5}$, and the greater the PM$_{2.5}$ pathogenic threshold, the littler the number of patients will be.

On the basis of the optimal control analysis, for human respiratory diseases caused by air pollution, the first step is to improve the PM$_{2.5}$ clearance rate, and the specific control strategy is to reduce the daily emission $P_0$ of PM$_{2.5}$ and increase the intensity of dust removal, etc. Next, improve the cure rate, and the specific control strategy is to treat the patients in
time. Then, to reduce the conversion rate of susceptible people becomes patients, and the specific control strategy is to remind people to wear masks in severe air pollution weather, as far as possible to avoid going out and so on. It is worth noting that the removal of PM$_{2.5}$ is a long and difficult work that needs to be sustained for a long time.

**Disclosure statement**

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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