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SIRSi compartmental model for COVID-19 pandemic with immunity loss

Cristiane M. Batistela\textsuperscript{a}, Diego P.F. Correa\textsuperscript{b}, Átila M Bueno\textsuperscript{c}, José Roberto C. Piqueira\textsuperscript{a,∗}

\textsuperscript{a} Polytechnic School of University of São Paulo - EPUSP, São Paulo, SP, Brazil
\textsuperscript{b} Federal University of ABC - UFABC, São Bernardo do Campo, SP, Brazil
\textsuperscript{c} São Paulo State University – UNESP, São Paulo, SP, Brazil

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The coronavirus disease 2019 (Covid-19) outbreak led the world to an unprecedented health and economic crisis. In an attempt to respond to this emergency, researchers worldwide are intensively studying the dynamics of the Covid-19 pandemic. In this study, a Susceptible - Infected - Removed - Sick (SIRSi) compartmental model is proposed, which is a modification of the classical Susceptible - Infected - Removed (SIR) model. The proposed model considers the possibility of unreported or asymptomatic cases, and differences in the immunity within a population, i.e., the possibility that the acquired immunity may be temporary, which occurs when adopting one of the parameters (γ) other than zero. Local asymptotic stability and endemic equilibrium conditions are proved for the proposed model. The model is adjusted to the data from three major cities of the state of São Paulo in Brazil, namely, São Paulo, Santos, and Campinas, providing estimations of duration and peaks related to the disease propagation. This study reveals that temporary immunity favors a second wave of infection and it depends on the time interval for a recovered person to be susceptible again. It also indicates the possibility that a greater number of patients would get infected with decreased time for re-infection.

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\section{1. Introduction}

The Wuhan Municipal Health Commission reported a cluster of 27 pneumonia cases on December 31, 2019, in Wuhan city, Hubei Province in China. On January 1, 2020 the World Health Organization (WHO) set up an Incident Management Support Team for dealing with the emergency. On January 5, 2020 WHO published the first news of a new viral disease outbreak. On January 7, 2020 the causative agent was identified and named as severe acute respiratory syndrome corona virus 2 (SARS-CoV-2) and the disease was named as Covid-19. On January 13, 2020 the first case, outside China, was reported in Thailand. On January 22, 2020 WHO stated that there was evidence of human-to-human transmission, and approximately 7 weeks later, on March 13, 2020, WHO declared Covid-19 a pandemic [1,2].

In Brazil, the first confirmed Covid-19 case was reported on February 26, 2020. By June 25, 2020 there were 1,188,631 confirmed cases with 53,830 deaths in Brazil [3] and 9,292,202 confirmed cases and 479,133 deaths globally according to [4].

Most patients were asymptomatic carriers and resolved spontaneously; however, some, particularly those with comorbidities, developed fatal complications [2], resulting in the rapid spread of the disease. This global emergency brings with it important and yet unanswered questions related to the dynamic behavior of the contagion and its mitigation and control strategies. As a result, measures carried out to contain the contagion such as social distancing, quarantine, and complete lock-down of areas have been studied [5–10].

In Mexico, on March 18, 2020, the Mexican Health Secretariat reported that the pandemic was going to last 90 days, with 250,656 expected cases. On the next day, the Health Secretariat informed that approximately 9.8% (24,594) of the patients would need hospitalization and 4.2% (10,528) of the total cases would be critical and need intensive care. The number of available Health Care units at that time was 4291 with 2053 ventilators [11]. The situation in Mexico led to the adoption of non-pharmaceutical interventions, such as washing hands, social distancing, cough/sneeze etiquette, etc.

\textsuperscript{1} Reported on 2:25pm CEST.
In Rio de Janeiro, Brazil, social distancing measures were introduced on March 17, 2020 [8], and the government of the state of São Paulo, Brazil, decreed quarantine on March 23, 2020. By June 25, 2020, the State of São Paulo had 248,587 confirmed cases with 13,759 deaths.

An important question related to patients’ immunity after recovery was raised. Differences in immunity after recovery have been reported in humans [12], and in experiments with rhesus macaques [13]. The experiment in [12] collected plasma from 175 recovered Covid-19 patients, and SARS-CoV-2-specific neutralizing antibodies (NAbs) were detected from day 10 to 15, after the onset of the disease and remained thereafter. Nonetheless, the NAbs levels were variable in the cohort. Among them, 52 (≈30%) patients developed low levels of these antibodies, in 10 (≈6%) the NAbs levels were undetectable, and 25 (≈14%) developed high levels.

In [14], data suggests an imbalance between the control of virus replication and activation of the adaptive immune responses. In some patients, lung cells remain vulnerable to infection due to immune system deficiencies. The virus continues replicating while the immune system attacks infected cells, also killing healthy cells in the vicinity, thereby severely inflaming the lung tissues. This seems to be the mechanism, which makes some patients severely ill weeks after their initial infection. Additionally, SARS-CoV-2 probably induces immunity like other coronaviruses; however, this mechanism is not fully understood [15].

The economic crisis due to the pandemic is another important issue. According to [16] the mortality rate of Covid-19 is not necessarily correlated with the global economic crisis, since governments, companies, consumers, and media have reacted to the economic shock. However, a global recession seems to be inevitable, and its duration and intensity will depend on the success of the measures taken to prevent the spread of the disease.

Taking the whole scenario into account, researchers worldwide are intensively studying and developing mathematical models of the Covid-19 outbreak. The knowledge on the dynamics of this pandemic is important to estimate the duration and peaks of the outbreak.

Macro-modeling of propagation of infectious diseases has been a field of research since the Kermack and McKendrick proposition [17–21]. It is a dynamic model that classifies individuals in a population as Susceptible - Infected - Removed (SIR) [22]. Since then, several modifications of the SIR model [23,24] such as, the Susceptible - Exposed - Infected - Removed (SEIR) model, [25,26] have been proposed for epidemic modeling.

In addition, studies considering time delay [27] and fractional-order [28–30] dynamical systems have been applied to disease propagation and could be useful to model the Covid-19 outbreak, and other biological systems [31,32].

In [33] a SIRD (SIR with delay) model had been adjusted to Covid-19 spread in China, Italy, and France. The results have shown that the recovery rates were similar for the three countries. In [34], a mixed analytical-statistical inverse problem is used to predict the Covid-19 progression in Brazil. A SIRu model, Susceptible - Infected - Removed - Unreported, was used to estimate the direct problem and a Bayesian parameter was used for the inverse problem.

Compartmental models considering immigration and home isolation, or quarantine, were proposed by [10]. All the situations presented an infection-endemic equilibrium with results showing that home isolation or lock down mitigates the infection probability.

In this work the proposed model is a modification of the original compartmental SIR model of Kermack and McKendrick [17–19], which includes a sick (S_{ck}) population compartment representing nodes of the networks that manifest the symptoms of the disease.

Although, several relevant questions are raised about the spread of the disease, we will focus on two of them in this study. The first one is associated with the group of asymptomatic individuals, who constitute the majority of the cases of the disease, and the second is related to the possibility that the immunity acquired by an infected person may be temporary.

As the mechanism of the spread of this disease is associated with the number of infected people and contact of this group to the susceptible group, it is important that the group of asymptomatic or unreported individuals be studied. Asymptomatic individuals are the group of infected people who do not manifest the symptoms, but can spread the disease by establishing effective contact with susceptible individuals.

To infer the behavior of asymptomatic individuals, the infected compartment will be divided into infected people who manifest the symptoms (sick) and those who do not, based on public data.

In addition, the influence of acquired immunity among individuals who have recovered from the infection is also being studied. If the acquired immunity is permanent, then the pattern of spread is described as temporal. On the other hand, if the immunity is temporary, it is important to understand the time interval after which the individual becomes susceptible again, changes in the dynamics of disease spread, and, the influence of the time interval for this reinforcement on the temporal response.

The proposed Susceptible - Infected - Removed - Sick (SIRS) model also considers birth and death of individuals in the given population and introduces a feedback from those in the recovered group who did not gain immunity or lost their immunity after a period of time.

The proposed SIRSi model presents both a disease free and an endemic equilibrium. The influence of the re-susceptibility feedback is investigated either analytically or numerically.

The parameters of the proposed SIRSi model are numerically fit to the pandemic situation of three cities in São Paulo State, in Brazil, namely, São Paulo, the capital of the State, Santos, on the coast and approximately 80 Km away from São Paulo, and Campinas, in the interior of the state and approximately 90 Km distant from the capital São Paulo.

The paper is organized as follows, Section 2 presents the SIRSi compartmental mathematical model. Section 3 presents the equilibrium points and stability conditions, showing the possible existence.

2. A modified SIR model with birth and death cases

The proposed SIRSi model can be seen in Fig. 1. In this model, the susceptible population $S$ is infected at a rate $\alpha$ when contacted by an infected individuals from $I$. The susceptible compartment also receives a population, at a rate $1/\gamma$, who did not gain complete immunity after recovering or who lost their immunity after a period of time.

The compartment $R$ represents the infectious population in the incubation stage prior the onset of symptoms. Infection transmission during this period has been reported in [35–38]. The infected

![Fig. 1. Epidemiological SIRSi model.](image-url)
population can be asymptomatic or symptomatic. The period between infection and onset of symptoms \(1/\beta_2\) ranges from 3 to 38 days with a median of 5.2 days. Once the infected individual is tested positive and the case is documented, it is moved to the \(S_{\text{ick}}\) compartment and those who do not develop severe symptoms become asymptomatic.

[39] reported that the estimate of the number of infections originating from undocumented cases is as high as 86%, including infected individuals with mild, limited and lack of symptoms. In other recent studies [40,41], it was found that, 20% - 40% of positive tested patients were asymptomatic.

In this work, and [42], under-reporting of asymptomatic cases is considered. The size of this population could be 7 times bigger than that of the documented cases and this group recovers within the period \(1/\beta_1\).

Some of the individuals within this population could develop symptoms. In [43], it has been reported that the average time between infection and the onset of symptoms can be 4.6 days. Once the case is documented it should be moved to the \(S_{\text{ick}}\) compartment, which consists of those patients with severe symptoms seeking medical attention. In [44] it was reported that this population could represent up to 19.9% of the total documented cases, of which 13.8% are severe cases and 6.1% require intensive care. The sick population recovers within the period \(1/\beta_3\) or moved to asymptomatic (see Fig. 1).

The effect of social distancing measures in the infected patients and corresponding deaths toll are shown in Fig. 1. The parameter \(\theta\) is introduced (1), and subject to the constraint \(0 < \theta < 1\). Consequently, the SIRS model is given by (1),

\[
\begin{align*}
\dot{S} &= \lambda - \alpha(1 - \theta)S - \delta S + \gamma I \\
\dot{I} &= \alpha(1 - \theta)S - (\beta_1 + \beta_2)I \\
\dot{S}_{\text{ick}} &= \beta_1 I - (\beta_3 + \sigma)S_{\text{ick}} \\
\dot{R} &= \beta_2 I + \beta_3 S_{\text{ick}} - (\gamma + \delta)R,
\end{align*}
\]

(1)

where \(\lambda\) and \(\delta\) are the birth and death rates, respectively.

The number of documented cases is a key information that should be noted from (1) because the number of confirmed cases is publicly available, and will be used to fine-tune the model. Although data on the number of new cases are added daily, it tends to be less representative of the dynamics.

3. Disease-free and endemic equilibrium points

Here, the model given by (1) is considered, such that \(x = f(x)\), where \(x = (S, I, S_{\text{ick}}, R)^T, x \in \mathbb{R}^4, f : \mathbb{R}^4 \rightarrow \mathbb{R}^4\) is the right-hand side of (1), and \(\alpha, \beta_1, \beta_2, \beta_3, \sigma, \gamma, \theta \in \mathbb{R}^+\) are the parameters.

To investigate the influence of the introduction of feedback from the recovered individuals with no immunity, the equilibrium points from (1) must be determined and their stability must be discussed.

Assuming the realistic hypothesis that \(\alpha \neq 0\), i.e., susceptible can be converted into infected, the equilibrium points are \(f(x^*) = 0\).

Using the Hartman-Grobman Theorem [45] the local stability of the equilibrium points can be determined by the eigenvalues of the Jacobian matrix computed on each equilibrium point. The Jacobian \(J = \frac{df}{dx}\), of (1) is given by (2).

\[
J = \begin{bmatrix}
-\delta - \alpha(1 - \theta) & -\alpha(1 - \theta) & 0 & \gamma \\
\alpha(1 - \theta) & S^* \alpha(1 - \theta) - (\beta_2 + \beta_1) & 0 & 0 \\
0 & \beta_2 & - (\beta_3 + \sigma) & 0 \\
0 & \beta_1 & \beta_3 & - (\delta + \gamma)
\end{bmatrix}
\]

(2)

The disease-free (Section 3.1) and the endemic (Section 3.2) equilibrium points are determined in the following section.

3.1. Disease-free equilibrium points

The disease-free equilibrium is a state corresponding to the absence of infected individuals, i.e., \(I^* = 0\). Applying this condition to (1), there is a disease-free equilibrium point \(x^* = (S^*, I^*, S_{\text{ick}}^*, R^*)^T\), belonging to the first octant of \(\mathbb{R}^4\) given by:

\[
P_1 = (S^*, I^*, S_{\text{ick}}^*, R^*)^T = (\lambda/\delta, 0, 0, 0)^T.
\]

(3)

Considering \(P_1\), the corresponding Jacobian [45] \(J_{P_1} = \frac{df}{dx}\) is

\[
J_{P_1} = \begin{bmatrix}
-\delta & -\alpha(1 - \theta) & 0 & \gamma \\
\alpha(1 - \theta) & 0 & 0 & 0 \\
0 & \beta_2 & - (\beta_3 + \sigma) & 0 \\
0 & \beta_1 & \beta_3 & - (\delta + \gamma)
\end{bmatrix}
\]

(4)

By the Laplace determinant development [46], the eigenvalues of (4) are the elements in the diagonal, that is, \(\xi_1 = -\delta, \xi_2 = \alpha(1 - \theta)/(\lambda/\delta) - (\beta_1 + \beta_2), \xi_3 = - (\beta_3 + \sigma)\) and \(\xi_4 = - (\delta + \gamma)\).

The eigenvalues \(\xi_1, \xi_2, \xi_3\), and \(\xi_4\) are real and negatives, indicating asymptotically stable directions. Consequently, \(\xi_2\) determines the equilibrium stability. If \(\alpha(1 - \theta)/(\lambda/\delta) < (\beta_1 + \beta_2)\), the eigenvector associated indicates an asymptotically stable direction. For \(\alpha(1 - \theta)/(\lambda/\delta) > (\beta_1 + \beta_2)\), the equilibrium point \(P_1\) is unstable, with the onset of a bifurcation in the parameter space.

3.2. Endemic equilibrium points

The endemic equilibrium points are characterized by the existence of infected people in the population, that is, \((I^* \neq 0)\).

Therefore, there is an endemic equilibrium point \(P_2 = x^* = (S^*, I^*, R^*, S_{\text{ick}}^*)^T\) in the first octant of \(\mathbb{R}^4\) given by

\[
S^* = \frac{\beta_1 + \beta_2}{\alpha(1 - \theta)} \\
I^* = \frac{1}{\phi}(\delta + \gamma)(\beta_1 + \sigma)[\alpha(1 - \theta)\lambda - (\beta_1 + \beta_2)\delta] \\
S_{\text{ick}}^* = \frac{1}{\phi} \beta_2 (\delta + \gamma)[\lambda \alpha(1 - \theta) - (\beta_1 + \beta_2)\delta] \\
R^* = \frac{1}{\phi} (\beta_1 \beta_3 + \beta_2 \beta_3 + \beta_1 \sigma)[\alpha(1 - \theta)\lambda - (\beta_1 + \beta_2)\delta],
\]

(5)

where \(\phi = \alpha(1 - \theta)\beta_1 \beta_3 \delta + \beta_2 \beta_3 \delta + \beta_1 \sigma + \beta_2 \sigma + \beta_2 \gamma\).

Consequently, the existence condition for the endemic equilibrium \(P_2 = x^* = (S^*, I^*, R^*, S_{\text{ick}}^*)^T\) in \(\mathbb{R}^4\) is given by

\[
(\alpha - 1) \frac{\lambda}{\delta} > \beta_1 + \beta_2.
\]

(6)

Considering \(P_2\), the corresponding Jacobian [45] \(J_{P_2} = \frac{df}{dx}\) is

\[
J_{P_2} = \begin{bmatrix}
-\delta - \alpha(1 - \theta) & -\beta_1 & 0 & \gamma \\
\alpha(1 - \theta) & 0 & 0 & 0 \\
0 & \beta_2 & - (\beta_3 + \sigma) & 0 \\
0 & \beta_1 & \beta_3 & - (\delta + \gamma)
\end{bmatrix}
\]

(7)

with eigenvalues \(\xi\) given by

\[
\xi^4 + a_1 \xi^3 + a_2 \xi^2 + a_3 \xi + a_4 = 0,
\]

(8)

with

\[
a_1 = \beta_1 + 2 \delta + \gamma + \sigma + \alpha(1 - \theta) \\
a_2 = \alpha(1 - \theta)(\beta_1 + \beta_2 + \beta_3 + \delta + \gamma + \sigma) + 2 \delta (\beta_1 + \sigma) + \gamma \beta_1 + 2 \delta + \gamma \sigma + \beta_2 \sigma + \beta_2 \gamma \sigma \\
a_3 = \alpha(1 - \theta)(\beta_1 \beta_3 + \beta_2 \beta_3 + (\beta_1 + \beta_2 + \beta_3)\delta + (\beta_2 + \beta_3)\gamma + (\beta_1 + \beta_3 + \delta + \gamma) \sigma + \beta_3 \delta^2 + \beta_2 \gamma \sigma + \delta \gamma \sigma + \beta_2 \gamma \sigma \\
a_4 = \alpha(1 - \theta)(\beta_1 \beta_3 + \beta_2 \beta_3 + \beta_1 \sigma + \beta_2 \delta \sigma + \beta_2 \gamma \sigma).
\]

(9)
Any further effort to analytically obtain $\xi$ eigenvalues becomes difficult due to the complexity of the coefficients. A possible alternative approach is to perform numerical calculations.

However, some insight for the model with feedback $\gamma$ can be obtained, in terms of bifurcations and stability, analyzing the eigenvalues when $\gamma = 0$ and $\gamma \neq 0$.

### 3.2.1. Eigenvalues for $\gamma = 0$

Endemic equilibrium exists even with $\gamma = 0$. In this case, the eigenvalues are

$$\begin{align*}
\xi_1 &= -\delta, \\
\xi_2 &= -(\beta_1 + \sigma), \\
\xi_3 &= \frac{1}{2}(\beta_1 + \beta_2)(-\alpha(1 - \theta)\lambda + \sqrt{\Delta}), \\
\xi_4 &= \frac{1}{2}(\beta_1 + \beta_2)(-\alpha(1 - \theta)\lambda - \sqrt{\Delta}),
\end{align*}$$

with $\Delta = 4\delta(\beta_1 + \beta_2)^3 + (\alpha(1 - \theta))\lambda^2 - 4\lambda\alpha(1 - \theta)(\beta_1 + \beta_2)^2$.

Consequently, $\xi_1$ and $\xi_2$ are real and negative, indicating asymmetrically stable directions. The conclusion is the same for $\xi_3$, because it is real and negative or complex with negative real part.

The eigenvalue $\xi_4$ must be analyzed more accurately because it can be the conjugated to $\xi_4$ in a complex case, i.e., in situations with $\Delta < 0$, indicating asymptotic stability. However, in a real case, i.e. when $\Delta > 0$, if $\alpha(1 - \theta)\lambda < \sqrt{\Delta}$, the endemic equilibrium point is unstable.

However, in the case of instability due to $\xi_4$, the existence condition (6) for the endemic equilibrium is not satisfied. Consequently, if the endemic equilibrium point $P_2$ exists, it is asymptotically stable.

### 3.2.2. Eigenvalues for $\gamma \to \infty$

Analyzing how $\gamma$ changes the endemic situation, in a case with $\gamma \to \infty$ is described by

$$\begin{align*}
S^* &= \frac{\beta_1 + \beta_2}{\alpha(1 - \theta)}, \\
I^* &= \frac{(\beta_1 + \sigma)}{\alpha(1 - \theta)\beta_2\sigma}(\alpha(1 - \theta)\lambda - (\beta_1 + \beta_2)\delta), \\
S_{\text{lk}}^* &= \frac{\beta_1 + \beta_2}{\alpha(1 - \theta)},
\end{align*}$$

following the same existence condition given by (6).

Under the assumption $\gamma \to \infty$, the coefficients from Eq. (8) can be approximated by

$$\begin{align*}
a_1 &\approx \gamma, \\
a_2 &\approx \gamma(I^*\alpha(1 - \theta) + \beta_3 + \delta + \sigma) = \gamma b_2, \\
a_3 &\approx \gamma(I^*\alpha(1 - \theta)(\beta_3 + \beta_2 + \sigma) + \delta(\beta_3 + \sigma)) = \gamma b_3, \\
a_4 &\approx \gamma I^*\alpha(1 - \theta)\beta_2\sigma = \gamma b_4.
\end{align*}$$

and, consequently, (8) is rewritten as

$$\begin{align*}
\xi_1^4 + \gamma\xi_1^3 + \gamma b_2\xi_1^2 + \gamma b_3\xi_1 + \gamma b_4 = 0. \\
\end{align*}$$

Assuming that at least one root $\xi$ goes to infinity as $\gamma \to \infty$,

$$\xi_4^4 + \gamma\xi_4^3 + \gamma b_2\xi_4^2 + \gamma b_3\xi_4 + \gamma b_4 = 0. \\
\xi_4^3 + \gamma b_2\xi_4^2 + \gamma b_3\xi_4 + \gamma b_4 = 0.$$

### 4. Parameter fitting by the least-squares method

In this section the parameters of the proposed SIRS$\xi$ model (1) (see Fig. 1) are numerically adjusted to fit the curve of confirmed symptomatic cases of three major cities in the state of São Paulo - Brazil, using publicly available data from the State Data Analysis System - SEADE (Sistema Estadual de Análise de Dados$^2$) [47]. The total populations in each of the cities were obtained from the same source and it is shown in Table 1.

| City          | Total population in 2020 |
|---------------|--------------------------|
| São Paulo     | 11.869.660               |
| Campinas      | 1.175.501                |
| Santos        | 428.703                  |

Birth and death rates were calculated using $\lambda$ and $\delta$ respectively. Linear interpolation was necessary as the data from the public repository were out of date. Results are shown in Fig. 2.

One of the first measures to contain the spread of the virus was the imposition of social distancing. This intervention, represented by $\theta$ in the model, focuses on reducing the possible contact between infected and susceptible individuals, and hence it is introduced as a factor of the transmission rate, i.e., $\alpha(1 - \theta)$. The time series corresponding to the daily measures of this index along with their mean for each one city considered are shown in Fig. 3. Although this time variation resembles a 7-day periodic function, especially on the second half of the register, the mean of this measure is used as a representative value.

Information about the duration of antibody response to SARS-CoV-2 is limited; however, currently it is known that the immunity acquired is temporary. Recent research highlights that immune response and antibody protection after recovery may depend on the severity of the infection, in some cases this protection can last for as little as 12 weeks while in others, no antibody protection is observed [49–54]. To assess the influence of this behavior on disease spread, we set the feedback parameter $\gamma$ at constant values $\gamma \in [0.00, 0.01, 0.02, 0.03, 0.04]$ in order to map possible scenarios especially those related to second waves of infection.

The disease transmission rate of symptomatic individuals prior to hospitalization is estimated to be between 1.12 and 1.19, while that of asymptomatic cases is between 0.1 and 0.6. In the model (1) this parameter is represented by the product $\alpha(1 - \theta)$, thus, making $\alpha \in [0.1, 1.0]$. The mean time between infection and onset-of-symptoms for confirmed cases, $1/\beta_2$, is 2 days, making $\beta_2 \in [0.5, 0.6]$. The time from onset-of-infection to full recovery, $1/\beta_1$, is considered to be between a few days to two weeks (5 to 15 days). The time period for a symptomatic patient to overcome the disease, $1/\beta_3$, and the time between hospitalization to death, $1/\sigma$, are both considered to be between 5 to 20 days [43,44,55,56].

The trust-region reflective least-squares algorithm [57–60] along with a 4th-order Runge-Kutta integrator were used to fit parameters in model (1) to actual data collected from public repositories for each one of the three cities into consideration. All parameters and initial conditions computed are normalized with respect to the total population in each case. For fitting, we set $S_0 \in [0.1], I_0 \in [0.1]$ with initial guess $S_0 = 99.9\%$ and $I_0 = 0.1\%$. The initial condition for $S_{\text{lk}}$ and $R$ was set to zero. Results are shown in Tables 2–7, and the temporal evolution of the $S_{\text{lk}}$ compartment for each city, for both, the fitted model and the public data, is show in Figs. 6, 8 and 10.

1. www.seade.gov.br,
In order to assess the influence of the parameter $\gamma$ in the endemic equilibrium, the eigenvalues were plotted for the set of fit parameters found, for $\gamma \in [0, 2)$. In Fig. 4 the eigenvalues are shown for the endemic equilibrium of each city, and for each one of the fit sets.

For $\gamma = 0$ the eigenvalues are stable for São Paulo and Santos, and as $\gamma$ increases, the eigenvalues move towards the left-hand side of the complex plane, whereas for Campinas the eigenvalues are unstable for $\gamma = 0$. In Fig. 5a closer view of the eigenvalues around the origin is shown.
5. Numerical experiments

In this section numerical experiments conducted using the MATLAB-Simulink [61] for two different conditions are described. First, the SIRSI model is fit with the real data for the Sicks population and different values of the parameter $\gamma$. Consequently, the simulation for the infected population $I$ can be inferred from the proposed model.

The numerical experiments, as in Section 4, were conducted for three major cities in the state of São Paulo, namely, São Paulo, Campinas, and Santos.

The initial condition is $x_0 = (S_0, I_0, S_{ick}, R_0)^T$, where $S_0$ and $I_0$ are the normalized susceptible and infected populations, which are considered as free parameters as they can be modified by the fitting algorithm.
Table 4
Fitted parameter for Santos.

|      | Fit 1     | Fit 2     | Fit 3     | Fit 4     | Fit 5     |
|------|-----------|-----------|-----------|-----------|-----------|
| α    | 9.131e-01 | 9.479e-01 | 9.835e-01 | 7.840e-01 | 9.355e-01 |
| β₁   | 1.500e-01 | 1.384e-01 | 1.932e-01 | 1.652e-01 | 1.529e-01 |
| β₂   | 2.440e-01 | 2.261e-01 | 2.459e-01 | 1.768e-01 | 2.034e-01 |
| β₃   | 5.070e-02 | 7.930e-02 | 1.112e-01 | 9.574e-02 | 1.469e-01 |
| σ    | 7.973e-02 | 5.095e-02 | 2.750e-02 | 6.898e-02 | 2.503e-02 |
| λ    | 2.693e-05 | 2.693e-05 | 2.693e-05 | 2.693e-05 | 2.693e-05 |
| δ    | 2.710e-05 | 2.710e-05 | 2.710e-05 | 2.710e-05 | 2.710e-05 |
| θ    | 4.860e-01 | 4.860e-01 | 4.860e-01 | 4.860e-01 | 4.860e-01 |
| γ    | 0.000e+00 | 1.000e-02 | 2.000e-02 | 3.000e-02 | 4.000e-02 |

Table 5
Fitted initial conditions for Santos.

|      | Fit 1     | Fit 2     | Fit 3     | Fit 4     | Fit 5     |
|------|-----------|-----------|-----------|-----------|-----------|
| S₀   | 1.006e+00 | 9.927e-01 | 1.004e+00 | 1.017e+00 | 9.685e-01 |
| I₀   | 1.141e-05 | 1.141e-05 | 1.168e-05 | 1.852e-05 | 1.402e-05 |
| S₀ₐ₀ | 0.000e+00 | 0.000e+00 | 0.000e+00 | 0.000e+00 | 0.000e+00 |
| R₀   | 0.000e+00 | 0.000e+00 | 0.000e+00 | 0.000e+00 | 0.000e+00 |

Table 6
Fitted parameter for Campinas.

|      | Fit 1     | Fit 2     | Fit 3     | Fit 4     | Fit 5     |
|------|-----------|-----------|-----------|-----------|-----------|
| α    | 7.473e-01 | 7.472e-01 | 7.466e-01 | 7.464e-01 | 7.384e-01 |
| β₁   | 1.350e-01 | 1.352e-01 | 1.354e-01 | 1.355e-01 | 1.368e-01 |
| β₂   | 1.930e-01 | 1.934e-01 | 1.934e-01 | 1.934e-01 | 1.923e-01 |
| β₃   | 5.631e-02 | 5.582e-02 | 5.225e-02 | 5.281e-02 | 5.965e-02 |
| σ    | 1.995e-01 | 1.994e-01 | 1.899e-01 | 1.906e-01 | 1.996e-01 |
| λ    | 3.353e-05 | 3.353e-05 | 3.353e-05 | 3.353e-05 | 3.353e-05 |
| δ    | 4.509e-05 | 4.509e-05 | 4.509e-05 | 4.509e-05 | 4.509e-05 |
| θ    | 4.842e-01 | 4.842e-01 | 4.842e-01 | 4.842e-01 | 4.842e-01 |
| γ    | 0.000e+00 | 1.000e-02 | 2.000e-02 | 3.000e-02 | 4.000e-02 |

Table 7
Fitted initial conditions for Campinas.

|      | Fit 1     | Fit 2     | Fit 3     | Fit 4     | Fit 5     |
|------|-----------|-----------|-----------|-----------|-----------|
| S₀   | 1.008e+00 | 1.008e+00 | 1.007e+00 | 1.007e+00 | 1.005e+00 |
| I₀   | 8.749e-06 | 9.148e-06 | 9.364e-06 | 9.614e-06 | 1.776e-06 |
| S₀ₐ₀ | 0.000e+00 | 0.000e+00 | 0.000e+00 | 0.000e+00 | 0.000e+00 |
| R₀   | 0.000e+00 | 0.000e+00 | 0.000e+00 | 0.000e+00 | 0.000e+00 |

5.1. Simulation results for São Paulo

The Fig. 6 shows that the SIRSi model gives a good adjustment for the data of confirmed cases in the infected population.

Considering that the acquired immunity is permanent, i.e., γ = 0, and that the isolation rate is constant, the peak of the infection occurs soon after July 2020, and the disease will not persist, until the end of the year.

On the other hand, assuming that immunity is not permanent and adopting a reinfection rate γ = 0.01 such that every 100 days the recovered individual becomes susceptible again, the model predicts a decrease in the confirmed cases and a new wave of infection in the first half of 2022.

Decreasing the time interval in which a recovered person becomes susceptible (γ = 0.02) to 50 days, the model indicates a second wave of infection in the first half of 2021. Decreasing the time interval in which a recovered person becomes susceptible (γ = 0.04) to 25 days, the model indicates that number of confirmed infected will be reduced by almost two thirds by the end of this year and the number of infected cases with continue to reduce. In this case, the number of confirmed cases will remain higher than the others.

In Fig. 7, the infected compartment I inferred from the SIRSi model is presented, showing that the peak of infection is close to July 2020.

Increasing γ reduces the time for a recovered person to become susceptible again, causing the peaks in Fig. 7 to increase, when compared to the curves for lower values of γ. This behavior, however, cannot be observed in Fig. 6, indicating that the increase in re-susceptibility feedback gain γ possibly contributes to the increase of asymptomatic or unreported infected cases.

In addition, it seems that if recovered individuals acquire permanent immunity γ = 0, the number of infected people tends to zero by the end of 2020. For γ = 0.01, there is a small increase in January 2022. For γ = 0.02, a new wave of confirmed cases can be seen in Fig. 6, and the increase in the infected population is also observed in Fig. 7.
For São Paulo, the numerical experiments show that considering any reinfection rate, there will be confirmed infected cases and unreported infected cases until 2023, indicating the need for a control strategy and study on preventive inoculation.

5.2. Simulation results for Santos

Fig. 8 shows the SIRSi model adjusted to the data of confirmed cases in Santos. Assuming that the immunity acquired is permanent, $\gamma = 0$, and isolation rate is constant, the peak of the confirmed cases in Santos will occur very close to July 2020, similar to that in São Paulo (see Fig. 6).

Adopting a nonzero reinfection rate, such that a recovered individual becomes susceptible again in 100 days ($\gamma = 0.01$), a second wave of infection can be seen in the coastal city around July 2021, one year before the second wave predicted for São Paulo with the same value for the re-susceptibility feedback gain $\gamma$.

Considering $\gamma = 0.02$, in which an infected person becomes susceptible again in a time interval of 50 days, the second wave of confirmed cases occurs at the beginning of the first half of 2021 and the number of confirmed infected is reduced to one third of the peak value.

These situations should be analyzed with caution and it is suggested to study the influence of the flow of people between these
cities, since the second wave of infection in the city of Santos occurs prior to São Paulo.

When \( \gamma = 0.04 \), after the peak of the confirmed cases, a second wave can be observed in the numerical results before the end of 2020, delaying the reduction of the confirmed cases.

For Santos, the numerical experiments show that when \( \gamma = 0 \) and \( \gamma = 0.03 \), the number of confirmed cases tends to zero in the beginning of 2023.

The infected compartment \( I \) inferred from the SIRSi model is presented in Fig. 9, showing that the peak of infection is close to July 2020.

The increase in re-susceptibility feedback gain \( \gamma \), reduces the time for an infected person to be susceptible again, causing higher peaks when compared to the curves for lower values of \( \gamma \). This behavior does not occur in Fig. 8 indicating that the increase in feedback possibly contributes to the increase in asymptomatic or unreported infected cases. This situation is similar to that observed for São Paulo.

In addition, when \( \gamma = 0 \), the number of infected people \( I \) tends to zero before the end of the 2020 (see the curve for \( \gamma = 0 \) in Fig. 9).

When \( \gamma = 0.01 \), a new wave of infection can be seen in 2021, and when \( \gamma = 0.02 \) the peak of the second wave of infection is near January 2021 (see Fig. 9, \( \gamma = 0.01 \) and \( \gamma = 0.02 \)).

Unlike São Paulo, the highest peak of infection among the unreported cases occurs when \( \gamma = 0.03 \). This behavior suggests a more
detailed study of the dynamics, because together with the situation in which the infected individual acquires permanent immunity, these rates suggest that the number of confirmed cases (see Fig. 8) and asymptomatic infected individuals tends to zero more quickly.

In the situation in which a recovered person becomes susceptible in 25 days, it is observed that the infection persists in the population for a longer time, as shown by the curve with $\gamma = 0.04$ in Fig. 9 justifying an accurate set of public strategies.

5.3. Simulation results for Campinas

Fig. 10 shows the SIRSi model results adjusted to the confirmed cases of infected people data in Campinas.

For permanent acquired immunity, $\gamma = 0$, and constant isolation rate, the peak of confirmed infection cases occur in the beginning of the second half of 2020.

Considering the re-susceptibility feedback gain $\gamma > 0$, in Fig. 10, it seems that with the increase in $\gamma$ the time necessary for the number of confirmed cases tending to zero is slightly higher, which is different from that of Santos and São Paulo. However, Campinas does not present a second wave of infection, even with the variation of $\gamma$.

The general behavior of Campinas, concerning the sensitivity analysis for $\gamma$, presents different results from that of Santos and São Paulo. It can be noticed in Fig. 10 that the observed data are far from the peak of infection predicted by the SIRSi model. At this point, more data is needed for any further qualitative analysis.

Observing the eigenvalues for the city of Campinas (See Figs. 4 and 5) it can be noticed that they are all real, indicating that there is no oscillatory behavior in the dynamics of the model. Depending on the new data this situation might change.

The infected compartment of Campinas presents the peak of infection close to the beginning of the second half of 2020, Fig. 11. Increase in the re-infection parameter causes an increase in the infection peak as shown in Fig. 10 indicating that the increase in feedback possibly contributes to the increase in either asymptomatic or unreported infected cases.

6. Conclusions

The proposed SIRSi model was fit to publicly available data of the Covid-19 outbreak, providing estimates on the duration and peaks of the outbreak. In addition, the model allows us to infer information related to unreported and asymptomatic cases.

The proposed model with re-susceptibility feedback adjusted to the confirmed infection data, suggests the possibility that recovered patients may have temporary immunity $\gamma > 0$ or even permanent $\gamma = 0$.

Considering the situation in which immunity is temporary, there is a second wave of infection which, depending on the time interval for a recovered person to be susceptible again, indicates a second wave with a greater or lesser number of reinfection patients.

If the time interval is larger (larger $\gamma$), the second wave of infection will have a greater number of infected people when compared to that of a shorter time interval.

The qualitative behavior for São Paulo and Santos is similar in terms of the sensitivity analysis of the re-susceptibility feedback gain $\gamma$. The bigger the value of $\gamma$, the shorter the time for a recovered person to become susceptible to reinfection, increasing the number of unreported or asymptomatic cases.

It is suggested to collect more data for the city of Campinas because the data of the confirmed infected cases presents a certain distance from the peak of the infection. The dynamics of the model may undergo some significant changes, given the sensitivity of the model to disturbances.

It is inferred from the proposed model that if the acquired immunity is temporary, a second wave of infection is a serious possibility. In addition, the number of asymptomatic patients increases if the acquired immunity lasts for a short period of time.

Availability of data and materials

Data are publicly available with [47,48].

Declaration of Competing Interest

There is no conflict of interest between the authors.
Credited authorship contribution statement

Cristiane M. Batistela: Conceptualization, methodology, validation, writing - original draft, visualization. Diego P.F. Correa: Conceptualization, methodology, software, validation, investigation, writing - original draft. Átila M Bueno: Conceptualization, methodology, validation, investigation, writing - original draft. José Roberto C. Piqueira: Conceptualization, validation, formal analysis, resources, writing - review & editing, project administration, funding acquisition.

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