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Modelling the COVID-19 Pandemic: Asymptomatic Patients, Lockdown and Herd Immunity *

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1. INTRODUCTION

1.1 Background

The mathematical modelling of the spread of epidemics has a long history, stretching back over several centuries. The “modern” approach to the modelling of epidemics can be said to have begun with Kermack and McKendrick (1927), which first enunciated the principle that infected persons pass on the disease to susceptible persons at a rate proportional to the number of contacts between the two groups, the so-called SIR model. Over the years, various refinements of the basic model have been proposed. The literature on disease modelling is truly enormous. Indeed, a survey paper Hethcote (2000) published in 2000 already had more than 200 references. Today it would be many times that number.

Traditionally, epidemiological models have grouped people into two, three, or four groups, usually denoted by Susceptible (S), Exposed (E), Infected (I), and Removed (R). Note that many authors use the symbol R to denote “recovered.” However, in this paper we wish to make a distinction between those who recover and are immune to further disease, and those who die from the disease. In traditional models, contact between a member of the Infected group I and another person belonging to the susceptible group S leads to the latter person becoming infected with a certain probability. Depending on the model, the susceptible person either becomes infected straightaway (the SIR model), or enters an intermediate stage called Exposed (E) (SEIR model). In the latter scenario, it is assumed that contact between persons belonging to the E and S groups does not lead to fresh infections, because members of the E group do not carry a sufficient viral load to infect others through contact.

However, one of the characteristic features of the coronavirus, or COVID-19, pandemic that is currently sweeping the world, is that many of the persons who contract the disease, in some cases a majority, are “asymptomatic,” or A. The members of this group do not manifest any external symptoms, and for the most part, recover with no visible impact from the disease. However, unlike in traditional epidemiological models, contact between a person in the A group and another in the S group does lead to the latter getting infected, with a certain probability. In addition, as in other models, contact between a person in the I group and another in the S group also leads to the latter getting infected, with a certain probability. To the best of the authors’ knowledge, the first paper to formulate
and analyze a model that captures this phenomenon is Robinson and Stilianakis (2013). However, the analysis of
the model in Robinson and Stilianakis (2013) is not so complete as it is currently available for the SEIR model. The
authors of Robinson and Stilianakis (2013) did not give a name to their model. In the present paper, we adopt
the model of Robinson and Stilianakis (2013) and refer to it as the SAIR (Susceptible, Asymptomatic, Infected, Removed) model.

1.2 Organization and Contributions of the Paper

The paper is organized as follows: We begin by reviewing
two classical models, namely the SIR and the SEIR
models, and analyze the stability of these models using
Lyapunov stability theory. This analysis closely follows
Korobeinikov and Wake (2002); Korobeinikov and Maini
(1996). We then carry out a complete analysis of the SAIR
(Susceptible, Asymptomatic, Infected, Removed) model,
which represents a more realistic model for the COVID-19
pandemic than currently existing SEIR models. In order
to establish the stability properties of the SAIR model, we
extend the classical Krasovskii-LaSalle theory of Lyapunov
stability, from the case where the Lyapunov function V
is positive definite, to the case where V is only positive
semidefinite. This extension is of independent interest. In
the remainder of the paper, we first present methods for
estimating the parameters in the SAIR model based on the
evolution of the pandemic. Then we present the outcomes of applying our theories to actual data from the COVID-19
pandemic in eight countries from around the world, including India.

In order to conform to page limits, various theorems are
presented without proofs.

2. REVIEW OF THE SIR AND SEIR MODELS

We begin with the SIR model. In this model, the popula-
tion is divided into three groups, denoted as S (Susceptible), I (Infected), and R (Removed). Note that many
authors use R to denote “Recovered.” However, in our
model, the group R also includes those who die from the
disease. Also, it is assumed that the total population size
is constant, so that S + I + R represent the fraction of
the population within each group. Therefore

\[ S, I, R \geq 0, S + I + R = 1. \]

In the absence of births and deaths, the equations that
govern the SIR model are

\[ \dot{S} = -\beta IS, \dot{I} = \beta IS - \gamma I, \dot{R} = \gamma I. \]

As expected, we have that \( \dot{S} + \dot{I} + \dot{R} = 0 \). Therefore we
can ignore anyone of the three equations and focus only
on the other two. Most authors ignore R and study

\[ \dot{S} = -\beta IS, \dot{I} = \beta IS - \gamma I, \]

where \( \beta, \gamma > 0 \) are parameters of the disease under study. Now let us analyze the dynamics in (2). Because we are
ignoring R, this dynamical system evolves over the simplex

\[ S_2 = \{ (S, I) : S \geq 0, I \geq 0, S + I \leq 1 \}. \]

It can be seen that any point \((S, 0)\) where \( S \in [0, 1] \) is
an equilibrium of the system (2). Therefore there is a
continuum of equilibria.

A thorough analysis of this equation is carried out in
Hethcote (1976); see Equation (2.5) and thereafter.

**Theorem 1.** (See (Hethcote, 1976, Theorem 2.2.)) Define \( \sigma := \beta/\gamma \) to be the “basic reproduction ratio.” Consider
the system (2) starting at an initial condition \((I_0, S_0)\). If \( \sigma S_0 \leq 1 \), then \( I(t) \downarrow 0 \) as \( t \to \infty \). If \( \sigma S_0 > 1 \), then \( I(t) \) increases at first and then decreases to 0, while \( S(t) \downarrow S_\infty \), where \( S_\infty \) is the unique solution in \((0, 1/\sigma)\) of

\[ 1 - S_\infty + \frac{\ln(S_\infty/S_0)}{\sigma} = 0. \]

The SEIR model differs from the SIR model in that there
is an additional group, known as Exposed (E). These are
people whose viral load is not sufficient to infect anyone
through contact. The SEIR model is described by

\[ \dot{S} = -\beta IS, \dot{E} = \beta IS - \gamma E, \dot{I} = \gamma E - \delta I, \dot{R} = \delta I. \]

3. THE SAIR MODEL

As mentioned earlier, the distinguishing feature of the
COVID-19 pandemic is the presence of a large number of
asymptomatic patients, who do not manifest any external
symptoms, but are still capable of infecting susceptible
persons. To capture this phenomenon, the following model
is presented in Robinson and Stilianakis (2013):

\[ \dot{S} = -\beta A S - \beta I S, \]

\[ \dot{A} = \beta A S + \beta I S - \gamma A - \delta A, \]

\[ \dot{I} = \delta A - \gamma I, \]

\[ \dot{R} = \gamma A + \gamma I. \]

In the above model, \( S, A, I, R \) denote the susceptible,
asymptomatic, infected, and removed populations respect-
ively. Interactions between \( A \) and \( S \) lead to the person
from \( S \) moving to \( A \) at the rate of \( \beta_A \), while interactions
between \( I \) and \( S \) lead to the person from \( S \) moving to \( A \)
at the rate of \( \beta_I \). Note that persons from \( S \) move only to
\( A \) and do not move directly to group \( I \). The persons in
\( A \) move to the group \( R \) at the rate \( \gamma_A \), and to the
\( I \) at the rate \( \gamma_I \). Finally, persons in group \( I \) move
to group \( R \) at the rate \( \gamma_I \).

It is easy to verify that the set

\[ S_3 := \{ (S, A, I) \in \mathbb{R}^3_+ : S + A + I \leq 1 \} \]

is an invariant set of (5), and that the set of equilibria is

\[ \{ (S, 0, 0), S \in [0, 1] \}. \]

**Theorem 2.** Define

\[ M_0 := \{ (S, A, I) \in S_3 : A = 0, I = 0 \}. \]

For the system (5), we have that

\[ (S(t), A(t), I(t)) \rightarrow M_0 \text{ as } t \rightarrow \infty. \]

This theorem is proved using the rather non-traditional
Lyapunov function \( V = S + A + I \).

Now let us impose the simplifying assumptions

\[ \beta_A = \beta_I = \beta, \gamma_A = \gamma_I = \gamma. \]
in (5). This leads to
\[ \dot{S} = -\beta AS - \beta IS, \dot{A} = \beta AS + \beta IS - \gamma A - \delta A, \dot{I} = \delta A - \gamma I, \tag{9} \]
and of course \( \dot{R} = \gamma A + \gamma I \). We refer to this model as the simplified SAIR model, to distinguish it from the more general SAIR model of (5). The main features of the simplified SAIR model are:

1. The infectivity of asymptomatic and infected patients is the same. Papers such as Wölfel et al. (2020); He et al. (2020); Liu et al. (2020); Li et al. (2020) show that asymptomatic patients show the same amount of viral shedding as infected patients.
2. The removal rate of asymptomatic and infected patients is the same. This says that the recovery rate of asymptomatic patients is that same. Papers such as Wölfel et al. (2020); He et al. (2020); Liu et al. (2020); Li et al. (2020) show that asymptomatic patients show the same amount of viral shedding as infected patients.

The assumptions made allow us to derive closed-form solutions to the simplified SAIR model, analogous to Theorem 1 for the SIR model. Define a new variable
\[ M := A + I. \]
Then it readily follows from (9) that
\[ \dot{S} = -\beta MS, \dot{M} = \beta MS - \gamma M, \dot{R} = \gamma M, \tag{10} \]
which is just the “SIR” model of (1) with \( M \) playing the role of \( I \). Recall the simplified SAIR model
\[ \dot{S} = -\beta AS - \beta IS, \dot{A} = \beta AS + \beta IS - \gamma A - \delta A, \dot{I} = \delta A - \gamma I. \tag{11} \]

It can be seen that there are only three parameters to be estimated here, namely \( \beta, \gamma, \delta \). Now we discuss the estimation is to be done. We estimate them in the order \( \gamma, \delta, \beta \).

To estimate \( \gamma \), we observe that the removal rate of persons in group \( I \) is given by \( \dot{R}_I = \gamma I \). Both \( \dot{R}_I \) and \( I \) can be measured. We express this in integral form as
\[ \dot{R}_I(T) - \dot{R}_I(0) = \gamma \int_0^T I(t) dt. \]
In effect one makes a “phase portrait” of \( \dot{R}_I \) versus \( I \). Computing \( \gamma \) using a least-squares approach with the above relationship for various values of \( T \) gives an estimate that is more robust to the discrete nature of \( \dot{R}_I \).

Next we derive a method to estimate \( \delta \), using the data after lockdown. Suppose a “perfect” lockdown is implemented at time \( T_L \), which causes \( \beta = 0 \) after that time. In this case, the simplified SAIR model becomes
\[ \dot{S} = 0, \dot{A} = -(\gamma + \delta) A, \dot{I} = \delta A - \gamma I. \]
In particular, it follows that
\[ A(T_L + t) = A(T_L) \exp(-(\gamma + \delta)t), \forall t \geq 0. \]
Also, the last equation in the model can be rewritten as
\[ \dot{I} + \gamma I = \delta A. \]
Combining these two relationships gives
\[ \log((\dot{I} + \gamma I)(T_L + t)) = \log(\delta A(T_L)) - (\gamma + \delta)t, \forall t \geq 0. \]
Therefore, ideally the plot of \( \log((\dot{I} + \gamma I)(T_L + t)) \) should be a straight-line with intercept \( \log(\delta A(T_L)) \) and slope \(-(\gamma + \delta)\). By computing the slope we can estimate \( \gamma + \delta \), and by combining this with the earlier estimate for \( \gamma \), we can get an estimate for \( \delta \). Note in passing that, once there

| Country    | \( \gamma^{-1} \)     | Country    | \( \gamma^{-1} \)     |
|------------|------------------------|------------|------------------------|
| USA        | 50 ± 3                 | Brazil     | 20 ± 1                 |
| Italy      | 30 ± 2                 | India      | 20 ± 2                 |
| Iran       | 11 ± 1                 | Japan      | 11 ± 1                 |
| France     | 21 ± 2 or 100 ± 4      | Switzerland| 35 ± 2 or 19 ± 1       |

Table I.

is an estimate, it is possible to determine the fraction of asymptomatic patients by setting
\[ A(T_L + t) = \left(\frac{1}{(\gamma + \delta)}(1 + \gamma I)(T_L + t), \forall t \geq 0. \]
Finally we come to estimating \( \beta \), which turns out to be the most involved part. Define \( \sigma = \beta/\gamma \) and \( M = A + I \), and observe that, before the lockdown, we have
\[ \frac{d}{dt} \log S = -\sigma, \frac{dM}{ds} = -1 + \frac{1}{S\sigma}. \]
After some manipulations which are contained in the full paper, we can write
\[ I(t) = \frac{(S_0 - 1)(\sigma - 1)}{2 \left(1 - \sigma S_0 + \sigma(S_0 - 1) \exp((\beta - \gamma)t)\right)} \exp((\beta - \gamma)t) \tag{12} \]
Since we can measure \( I(t) \) as a function of \( t \), and we have an estimate for \( \gamma \) is available at this point, the above equation can be used to estimate \( \beta \).

4. NUMERICAL RESULTS

Now we present the outcomes of applying the parameter estimation techniques.

Table 1 shows the estimated \( \gamma^{-1} \) (with units of days) for various countries.

Figures 1 and 2 show the values of \( \gamma \) for various countries, by plotting \( R_I(t) \) versus \( \int_0^T I(t) dt \) as a function of \( T \), for various countries. It can be seen that, for six out eight countries, the plot is nearly linear, thus indicating a robust estimate for \( \gamma \). However, for France and Switzerland, the graph is far from linear. We believe that this is because we ignored the incubation period of the virus. If we were to take this into account, we would have to modify the dynamics as a delay-differential equation, in the form
\[ \dot{R}_I(t) = \gamma I(t - \tau), \]
where \( \tau \) is the incubation period. Thus
\[ R(T) - R(0) = \int_0^{T-\tau} I(t) dt. \]
Figure 3 shows the estimates of \( \gamma \) for these two countries using various values for the delay \( \tau \). It can be seen that, as \( \tau \) is increased, the plot becomes more linear. It is not clear why this should be an issue only for two out of eight countries.

Figure 4 shows the outcomes for estimating \( \delta \) for various countries.

Figures 5 and 6 show the outcomes for estimating \( \beta \) for various countries. This figure shows clearly that the “lockdown” has been implemented with quite varied levels of thoroughness in different countries.

Once we have fitted the parameters, we have fitted the past history and made future forecasts for various countries in Figure 7. The quality of our estimates can be seen in this figure.
Fig. 1. Removal (recovery+death) frequency $\gamma$ for various countries.

5. DISCUSSION AND FUTURE RESEARCH

In this paper, we undertook the task of completely analyzing the SAIR model which was introduced in Robinson and Stilianakis (2013) to incorporate asymptomatic patients. As a part of this, we established the global attractivity of the equilibria in the SAIR model, both with and without vital dynamics. We then validated our model by fitting observed data in eight countries. Our analysis shows that the model built upon our estimated parameters does an excellent job of explaining the evolution of the pandemic across these countries. We have also applied a similar analysis to the situation in Delhi. This analysis shows clearly the impact of implementing the “lockdown” in the Delhi area.

There is no shortage of interesting open problems to be tackled. We list some of them below: Some of these are listed below. The full paper gives descriptions of these problems.

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Fig. 3. Delay difference plots for: France (left) and Switzerland (right), in order to find the correct $\gamma$

Fig. 4. Time $t = 0$ is 14th February 2020. Extracting $\delta$ (using the decaying exponential in the analytical solution 10-30 days post lockdown).

- Nonlinear observers for the SAIR model.
- Lyapunov stability analysis of SAIR and compartment models with vital dynamics:
- Refinements of the SAIR model
- Sensitivity to estimation errors

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