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Stochastic modelling of the dynamics of infections caused by the SARS-CoV-2 and COVID-19 under various conditions of lockdown, quarantine, and testing

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A B S T R A C T

We develop a mathematical model for the transmission and spread of infections caused by the severe acute respiratory syndrome coronavirus 2 (SAR-CoV-2) which causes the coronavirus disease 2019 (COVID-19), a disease that has since been classified by the World Health Organization, as a global pandemic. We focus attention on virus transmissions in a closed population and hence use a compartmental epidemic model to study the inherent dynamics of infections between the various subgroups of the population. We assume random interactions between members from different subgroups and hence we employ stochastic modelling techniques. In the absence of a vaccine for this novel coronavirus, governments worldwide have put in place various intervention strategies, including travel bans, lockdowns, screening, testing, quarantine, etc. in order to reduce and hopefully eliminate the transmission and spread of the COVID-19 virus. These interventions are built into our model and we investigate their effects and effectiveness. In particular, we observe that the two subgroups containing infectious individuals, namely; the subgroup comprising of unidentified asymptomatic individuals as well as; the subgroup comprising of un-quarantined symptomatic individuals; pose the greatest risk of the transmission and spread of infections. We therefore also observe from our results that; rapid (realtime) mass testing as well as effective (or mandatory) quarantine of all infected individuals are the fundamentally critical and necessary steps in reducing the internal transmissions and spread of the COVID-19 virus. In particular, our results indicate that lockdowns are only important to the extent that, if implemented effectively, they help in reducing the rate of transmissions (i.e. help to ‘flatten’ the transmission curves) and hence allow policy makers and healthcare practitioners to put in place the important processes of realtime mass testing and mandatory quarantine, including hospitalization and treatment. Our results are presented over a single wave (or part of a single wave) of COVID-19 infections in a given population. If conditions are repeated, the results would correspondingly extend to multiple waves of infection.

Introduction

The transmission and spread of infectious diseases, especially those diseases that manifest into global pandemics, remains the greatest threat to mankind. Even when such diseases exist as localized epidemics within a given population, the risk of such a population being wiped off the face of the Earth would be catastrophic. It is therefore incumbent upon all of us to contribute in any and every possible way in the global efforts to stop the transmission and spread of pandemics whenever they arise. This research is therefore conducted in that spirit, to add to the growing body of knowledge with regards to the transmission dynamics of the deadly COVID-19 virus.

A significant number of recent research articles and results on transmission and spread of the COVID-19 virus have been localized to the context of specific countries or similar such geo-political spaces, see for example [1–4] and the multiple references therein. We have adopted an approach that develops a universal and robust mathematical model for the analysis of the transmission dynamics of the COVID-19 virus. Our model therefore applies to any country (or a subsection thereof) and similarly applies to any population size. Practically all the mathematical modelling research that has been conducted thus far on the transmission and spread of the COVID-19 virus have used compartmental models and employed deterministic or discrete stochastic models. A reasonable survey of the such models which have been used thus far for the study of the transmission dynamics of the COVID-19 virus is given, say, in [4]. We follow a continuous time stochastic modelling approach and implement a novel reduction of the compartmental variables. We also employ the more realistic modelling of the rate of contact between ‘healthy’ and infectious individuals as a time varying function.

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The compartmental modelling approach in studying epidemics and population dynamics can be traced back to the early 20th century from the pioneering works of Kermack and McKendrick [5]. Equally broad overviews of these methods are given in the recent text such as [6] and [7]. A comprehensive account of stochastic processes and stochastic differential equations can be found in [8].

We also take this opportunity to clear the possible confusion regarding quarantine versus isolation processes. Quarantine refers to the process in which an individual who has been in close contact with a person who carries the infectious disease (COVID-19 in this case) is kept away from other individuals. Isolation refers to the process in which an infected individual (someone who is sick with COVID-19 in this case) or an asymptomatic individual who has tested positive for COVID-19 is kept away from other individuals, even in the infected/positive individual's private home.

Given its encompassing nature, especially noting that quarantined individuals may or may already be infected, the term quarantine is now globally accepted to refer to either quarantine or isolation processes. This is the approach adopted in this paper! The term isolation will therefore only be used, in this paper, to refer to those circumstances in which the context specifically refers to the need to keep known infected (or known COVID-19 positive) individuals away from others.

Model assumptions

We limit our attention to the spread and transmissions of the COVID-19 virus in a closed population of size \( N \). This is a reasonable assumption in light of the following considerations;

- All individuals from each subgroup of the population, including those who die from the infection (or the disease), are counted as part of the population;
- A travel ban is imposed (except for possible repatriations described in the next bullet) and hence eliminating the possible population size changes due to migration;
- Repatriation of citizens is allowed in either direction, the numbers are small and we assume that the inbound repatriates cancel out those outbound.

We track the transmission and spread of infections in time starting at time \( t = 0 \) which coincides with the day on which a lockdown of the population group comes into effect. The subsequent transmission and spread of the virus, over the period \( t > 0 \), is strictly therefore attributed to internal transmission, arising from the contact between infected individuals and ‘healthy’ individuals. In particular, we assume that;

- All inbound repatriates are quarantined for a period of time equivalent to the generally accepted incubation period of the COVID-19 virus (currently 14 days) and hence are incapable of transmitting or spreading the virus;
- All quarantine processes are strictly controlled and hence, except for healthcare workers and other essential services providers (who are expected to at all times have proper personal protective equipment (PPE)), there is otherwise no contact as would transmit from unidentified asymptomatic individuals as well as from unidentified symptomatic individuals from the indeed infectious but otherwise hospitalized individuals;
- Infections are therefore transmitted to ‘healthy’ individuals only from unidentified asymptomatic individuals as well as from un-quarantined symptomatic individuals.

It is important to remark at this point that evidence exists (especially in Europe) of healthcare providers (such as doctors and nurses) who have been infected by the COVID-19 virus. We do not see this as a weakness in our assumption as this has widely been attributed to the lack of appropriate PPE for such workers. The provision of appropriate PPE for essential services providers remains the responsibility of governments. This explains why such essential services providers across the globe have repeatedly threatened to down tools if this important matter of PPE is not adequately addressed by their respective governments. Our assumptions are therefore based on governments acting responsibly in line with what is expected of them. If governments cannot provide such assurances, then the fight against this pandemic is already lost and all other well-intentioned interventions would come down to nought.

The model

We use a compartmental epidemic model of the SEIQHRD type, see for example [2–4]. Our model is modified to SEUQHRD based on the basic realization that, with regards to COVID-19, all the four subgroups, \( E, U, Q, \) and \( H \) are infected, indeed infectious, and hence can be clustered into the broad subgroup, of infectious individuals, \( I = E + U + Q + H \). It would therefore be anomalous for a COVID-19 epidemic model to have, as separate subgroups, the clusters (\( E, I, Q, H \)) and hence our correction of this to \( (E, U, Q, H) \). Here;

- \( S \) represents the ‘healthy’ (or Susceptible) subgroup;
- \( E \) represents the (Exposed) subgroup which in our case will comprise of unidentified, infected, and asymptomatic individuals;
- \( U \) represents the (Un-quarantined), infected, and symptomatic individuals;
- \( Q \) represents the (Quarantined), infected, and symptomatic individuals;
- \( H \) represents the (Hospitalized), infected, and symptomatic individuals whether with mild or with severe symptoms;
- \( R \) represents the (Recovered) individuals who are assumed to have recovered from infection with immunity, given the currently agreed medical evidence especially with regards to antibodies;
- \( D \) represents (Deaths) from infections.

The schematic flow diagram of the compartmental SEUQHRD model is shown in Fig. 1.

In Fig. 1, the directed arrows are interpreted as follows;

- \( S \to E \) and \( S \to U \) represent the rates of infection of ‘healthy’ individuals (\( S \)) resulting from contact with infected individuals (\( E \) and \( U \));
- \( E \to U \) and \( E \to Q \) represent the rates at which asymptomatic individuals (\( E \)) develop symptoms;
\[ \rho(t, x) = \frac{\partial}{\partial t} \rho(t, x) = -\frac{1}{2} \sum_{i,j} \frac{\partial^2}{\partial x_i \partial x_j} [\eta_{ij}(x, t) \rho(x, t)]. \tag{1} \]

In our case, this would be a PDE in 8 variables given that the vector \( x \) contains 7 variables and also that the time \( t \) adds another variable. It would be impractical to attempt to solve such a PDE, even via numerical techniques. Fortunately, the PDE in Eq. (1) converts to the system of stochastic differential equations given in Eq. (3),

\[ dX(t) = c(X(t), t) dt + \sigma W(t), \tag{2} \]

where \( W(t) \) is a collection of Wiener processes. Given our intended application of these stochastic differential equations to modelling the spread of epidemics, and given the fact that such spread of epidemic diseases mirrors a diffusion process (spreading in any direction) it therefore follows that the drift (or convective) terms must vanish. This implies that, for our application, the drift coefficient is zero, i.e. \( c = 0 \). In stochastic language, the symmetric diffusion in any direction is referred to as a symmetric random walk, in which case the stochastic process is known as Brownian motion. The random distributions (or noise terms) required in our stochastic model will therefore all be of the form,

\[ \text{noise term} = \sigma_k dW_k(t), \quad \text{with} \quad \sigma_k = \sqrt{\eta_k}. \tag{3} \]

where the index \( k \in \{1, 2, 3, 4, 5\} \) corresponds to each of the five stochastic rate processes referred to earlier, e.g. stochastic infection rate, etc.

### Governing equation

It is not a coincidence that, in Fig. 1, we clustered the compartments into four broad categories, this was intended to lay the foundation for developing a reduced set of governing equations, reduced from seven down to four. In particular, the clusters in Fig. 1 reduce the epidemic model from a SEUQRHD to a SIRD model in which, as already noted before, \( I = E + U + Q + H \). This novelty means that we can reduce the number of equations to four, in the unknown quantities \( S, I, R \) and \( D \). This reduction is made possible via the existing ability of mining the statistical data related to the proportions of the different subgroups. Such concerted data mining efforts are indeed expected of governments and other interested organizations, such as the World Health Organization, especially given the need to account for all COVID-19 related infections, hospitalizations, recoveries, and deaths at all times. Most hospitals across the world, including in highly developed nations, for example are faced with critical shortages of ventilators and other related medical resources and facilities. The need to fully account, in real time, for all the crucial data and statistics related to the COVID-19 pandemic can therefore not be overstated. Given the prevalence of such data, we can therefore deduce the following proportions;

- \( E = \xi I \), with \( 0 \leq \xi \leq 1 \), gives the asymptomatic subgroup as a proportion of the entire group of infected individuals;
- \( U + Q = \rho I \), with \( 0 \leq \rho \leq 1 \), gives the symptomatic subgroup as a proportion of the entire group of infected individuals; in particular
- \( U = \rho_0 I \), where \( 0 \leq \rho \leq 1 \) gives the probability of a symptomatic individual to be un-quarantined; and
- \( Q = \rho(1 - \xi) I \), gives the probability of a symptomatic individual to be in quarantine;
- \( H = (1 - \rho - \xi) I \), with \( 0 \leq (\rho + \xi) \leq 1 \), gives the hospitalized subgroup as a proportion of the entire group of infected individuals.

We intend to introduce dimensionless parameters and hence also to analyse the relevant non-dimensional equations. The final form of our equations shall therefore be non-dimensional. This enables our results, and model, to be transferable to any country of locality. We are be able to illustrate the development of the final, non-dimensional and stochastic, governing equations by using deterministic equations as a starting point without losing any generality.
Deterministic equations

The relevant deterministic rates in relation to 1, are;

- $\lambda$ represents the rate of infection of ‘healthy’ individuals ($S$) resulting from contact with infected individuals ($E$ and $U$);
- $\alpha$ represents the rate at which asymptomatic individuals ($E$) develop symptoms, moving with probability $\rho$ to ($U$) and with probability $(1 - \rho)$ to ($Q$);
- $\mu$ represent the rates at which symptomatic individuals ($U$ and $Q$) die of infection;
- $\gamma$ represents the rate at which symptomatic individuals ($U$ and $Q$) recover;
- $\delta = (1 - \mu - \gamma)$ represents the rate at which symptomatic individuals ($U$ and $Q$) are hospitalized;
- $\mu_H$ represent the rates at which hospitalized individuals ($H$) die of infection; and
- $\gamma_H = 1 - \mu_H$ represents the rate at which hospitalized individuals ($H$) recover.

In particular,

$$\lambda = \lambda_E + \lambda_U = \frac{r_H}{N} E + \frac{r_U}{N} (E + U),$$

where $r$ represents the number of contacts per unit time and $\beta$ is the probability of transmitting the infection per contact. This leads to the evolutions equations,

$$\frac{dS}{dt} = -\lambda S,$$  
$$\frac{dE}{dt} = \lambda_S S - \alpha p E - \alpha(1 - p) E = \lambda_E E - \alpha E,$$  
$$\frac{dU}{dt} = \lambda_U S + \alpha(1 - p) E - (\delta + \gamma + \mu) U = \lambda_U S + \alpha p E - U,$$  
$$\frac{dQ}{dt} = \alpha(1 - p) E - (\delta + \gamma + \mu) Q = \alpha(1 - p) E - Q,$$  
$$\frac{dH}{dt} = \delta(U + Q) - (\gamma_H + \mu_H) H = \delta(U + Q) - H,$$  
$$\frac{dR}{dt} = \gamma_H U + \gamma_H T H,$$  
$$\frac{dD}{dt} = \mu U + \mu Q + \mu_H H,$$

subject to initial conditions,

$$S(0) = S_0, \quad E(0) = E_0, \quad U(0) = U_0, \quad Q(0) = Q_0, \quad H(0) = H_0,$$
$$R(0) = R_0, \quad D(0) = D_0.$$  
(11)

The reduces equations are,

$$\frac{dS}{dt} = -\frac{r_H}{N} S,$$  
$$\frac{dI}{dt} = \frac{r_U}{N} S - (\mu + \gamma) I - (\gamma_H + \mu_H)(1 - \rho - \xi) I,$$  
$$\frac{dR}{dt} = \rho I + \gamma_H (1 - \rho - \xi) I,$$

with ($\gamma_H + \mu_H$) and subject to initial conditions,

$$S(0) = S_0, \quad I(0) = I_0, \quad R(0) = R_0, \quad D(0) = D_0.$$  
(16)

We can further reduce the number of parameters in the model by introducing dimensionless quantities, say:

$$S^* = S/N, \quad I^* = I/N, \quad R^* = R/N, \quad D^* = D/N, \quad t^* = (\mu + \gamma)t.$$  

After dropping the asterisks, this leads to the non-dimensional equations,

$$\frac{dS}{dt} = -R_0 I S,$$  
$$\frac{dI}{dt} = R_0 I S - \rho I - \frac{(1 - \rho - \xi)}{\mu + \gamma} I,$$  
$$\frac{dR}{dt} = \frac{1}{\mu + \gamma} \left[ \rho \mu + \mu_H(1 - \rho - \xi) \right] I,$$

where,

$$R_0 = \frac{r_H(\rho q + \xi)}{\mu + \gamma},$$  
$$R_0 = r \times (\rho q + \xi) \times \frac{1}{\mu + \gamma},$$

is the basic reproduction number. By writing,

$$R_0 = r \times (\rho q + \xi) \times \frac{1}{\mu + \gamma},$$

$R_0$ is interpreted as; number of contacts per unit time $\times$ probability of transmitting the infection per contact $\times$ length of time in appropriate infectious stages.

To analyse the stability conditions of the equilibrium states, we can drop the equations in $R$ and $D$ since they are fully determined by the knowledge of $I$. It is clear that the only disease free equilibrium point (DFEP) is,

$$(S^0, I^0, R^0, D^0) = (1, 0, 0, 0),$$

where it should be noted that $N^* = 1$ represents the, non-dimensional, total population size. By analysing the eigenvalues of the Jacobian matrix at the equilibrium point, we notice that the DFEP is locally asymptotically stable if,

$$\frac{1}{\mu + \gamma} \left( \rho \mu + \mu_H(1 - \rho - \xi) \right) I < 1.$$  
(24)

Stochastic model

We can modify the deterministic equations by introducing stochastic variations into our model via the arguments presented in the previous sections, see for example [9]. In particular, the stochastic model, in dimensionless terms, reads,

$$dS = -R_0 I S (dt + \sigma_d W_d).$$  
(25)

$$dI = R_0 I S (dt + \sigma_d W_d) - \left[ \left( \rho + \frac{(1 - \rho - \xi)}{\mu + \gamma} \right) I \right] dt,$$

$$dR = \frac{1}{\mu + \gamma} \left[ \rho \mu (dt + \sigma_d W_d) + \left( \mu_H(1 - \rho - \xi) I \right) (dt + \sigma_H W_H) \right],$$

$$dD = \frac{1}{\mu + \gamma} \left[ \rho \mu (dt + \sigma_d W_d) + \left( \mu_H(1 - \rho - \xi) I \right) (dt + \sigma_H W_H) \right].$$

The introduction of random processes (stochastic variations) into the above equations comes naturally. For example, Eq. (25) now adds appropriate stochastic variation to the probability of transmitting the infection per contact, $\beta(\rho q + \xi)$.

The system of stochastic differential equations (25)–(28) is much more straightforward to solve than the corresponding Fokker–Planck differential equation. We solve the system of stochastic differential equations numerically via finite difference methods subject to appropriate initial conditions,

$$S(0) = S_0, \quad I(0) = I_0, \quad R(0) = R_0, \quad D(0) = D_0, \quad S_0 + I_0 + R_0 + D_0 = 1.$$  
(29)

Modelling of interventions for reducing the transmission and spread of infections

Travel bans

Travel bans are a commonsensical approach to managing the spread of infectious diseases both locally and across borders. For example when the COVID-19 virus was first reported in China in December 2019, countries around the world could have immediately imposed travel bans on individuals travelling from China. Indeed, governments could have immediately put in place measures to test all individuals arriving on their shores. This way, the virus would remain localized
to its point of origin. Unfortunately however, the time delays arising from; the incubation periods of new viruses; the detection of illness and disease; the identification of the new virus; and the ease of contemporary global travel coupled with global connectedness; mean that it may already be too late to contain the infections to a particular locality at the time of identification of a new virus. Travel bans, therefore, must still be accompanied by supplementary interventions.

Given that the previous two iterations of coronaviruses (the SARS-CoV of 2002 and the MERS-CoV of 2012) did not lead to global pandemics but instead remained localized to specific regions, governments around the world may therefore have underestimated the ability of the SARS-CoV-2 of 2019 to rapidly spread into a deadly global pandemic. This may explain the delayed responses around the world, including the delays in instituting travel bans.

Modelling a lockdown

As with travel bans, lockdowns result in hugely adverse and widespread socio-economic effects. However, the havoc caused by global pandemics, especially the large scale loss of life must at all times outweigh social discomforts and economic considerations. It would not make much sense to insist on continuing with non-essential social gatherings when such events may subsequently lead to loss of many lives. Similarly, it would not make much sense for governments to still want to balance the budget when people are starving because they suddenly find themselves with no source of income, or when hospitalized individuals are dying because they are in need of otherwise non-existent expensive medical equipment and facilities, etc. It would be imperative, for example, for governments to raise their debt ceiling if necessary and as much as possible, for the provision of stimulus packages to their ‘locked down’ citizens, if only to ensure the continued existence of humanity.

Once a viral infection has been introduced into a population either locally or imported from outside, a lockdown would usually be among the fundamentally important first steps to take in order to provide the necessary conditions to assess the scale of the spread of infections. The basic reproduction number $R_0$ is a very important measure in this direction. $R_0$ represents the number of secondary infections caused by a single infected individual who is introduced into a population consisting of only susceptible (S) individuals. If for example $R_0 = 2$, then each infected individual will infect two other individuals, who in turn will each infect two other individuals, etc. leading (in the absence of any interventions) to exponential growth of infected individuals,

$$I(t) = 2^{t−1}, \quad t \geq 1.$$  

Even with $R_0 = 1$ and in the absence of any interventions, we would still have exponential growth of infected individuals,

$$I(t) = 2^{t−1}, \quad t \geq 1.$$  

This explains why the current COVID-19 statistics of most affected countries reflect exponential growth. In order to reduce this dramatic increase in the number of infected individuals, a lockdown would be required as this naturally also reduces the $R_0$ value, by minimizing the number of contacts between individuals. In a perfect lockdown, in which there is absolutely no contact between individuals, the $R_0$ value should instantly drop to zero,

$$R_0 = \begin{cases} R_0 \geq 1, & \text{before lockdown,} \\ 0, & \text{post lockdown.} \end{cases}$$  

meaning that no new infections can happen post such an ideal lockdown. The lockdown period could then be used to ensure that already infected individuals receive appropriate healthcare, the lockdown being lifted only after it is determined that no infected individuals still exist within the population.

To illustrate, we apply our model to the case of a perfect lockdown. Starting with any percentage of infected individuals, the expectation is that the proportion of ‘healthy’ individuals must remain constant (since there are no contacts with infected individuals) and that the number of infected individuals must gradually decrease, as they recover or die. Fig. 2 shows the predictions of our model under perfect lockdown conditions, $R_0 = 0$, starting from an initial condition of 80% infected individuals and 20% ‘healthy’ individuals.

Fig. 3 shows the time evolution of the infectious subgroups under the same conditions as Fig. 2.

Our results are indeed in line with expectations, the proportion of previously uninfected individuals stays constant at 20% and the proportion of infected individuals decreases gradually as individuals recover or die, Figs. 2 and 3.

It is particularly important to note the possibility and ability to tell, from the model results, whether or not the infections would have vanished from the population by a certain date post lockdown. For example, our hypothetical example above (given the very high initial proportion of infected individuals, 80%) shows that after 21 days the infectious individuals (especially from the high risk asymptomatic and un-quarantined subgroups) are still prevalent in the population.

In Figs. 2 and 3, the following set of parameter values are used

$$\rho = 0.3, \quad \xi = 0.6, \quad q = 0.1, \quad y = 0.04, \quad \mu = 0.01, \quad \mu_H = 0.1, \quad \sigma_i = 0.01, \quad \sigma_R = 0.01, \quad \sigma_f = 0.0001, \quad \sigma_{\mu H} = 0.0001, \quad \sigma_{\sigma H} = 0.001.$$  

Unless otherwise stated, the set of parameter values as given in Eq. (33) are the values that will be used in the results throughout this article.
We notice, from Fig. 4, that even when the initial infection rate is greatly reduced to 1% of the population, the number of asymptomatic individuals, say at the 21 day mark, would still be significantly high in the population, at around 0.05%. In a population of 60million, this would represent 30,000 individuals still carrying the infection as asymptomatic individuals.

Unfortunately a perfect lockdown is not possible, anywhere, and especially not in developing countries. People would still need to be in close contact for various essential needs. Relevant lockdown rules and policies must therefore be put in place to ensure that the lockdown conditions are as close as possible to the ideal situation. Such rules and policies include:

- Prohibition on all social gatherings (we will refer to this as social distancing);
- Mandatory physical distancing including mandatory provision and use of personal protective equipment (PPE) in all unavoidable social, public, and private spaces; e.g. at funerals, in grocery stores, in banks, in pharmacies, etc.;
- Mandatory provision, by employers, of proper PPE to all essential services personnel whose responsibilities do not allow for physical distancing from other individuals (indeed including physical distancing from infected individuals) such as doctors, nurses, law enforcement, grocery store employees, etc.

The model that we use to account for such a lockdown takes into account that contacts between ‘healthy’ and infected individuals would gradually decrease with time,

\[ R_0(t) = \begin{cases} R_0, & \text{before lockdown}, \\ R_\infty + (R_0 - R_\infty) \exp(-\kappa t), & \text{post lockdown}, \ t \geq 0, \end{cases} \tag{34} \]

where \( R_\infty > 0 \) is the long term value of \( R_0 \) in keeping with the understanding that it would not be possible to ensure the ideal lockdown scenario with \( R_\infty = 0 \). The parameter \( \kappa \) is a measure of the effectiveness of the lockdown rules, regulations, and policies and adherence thereto. An ineffective lockdown, in which rules, regulations, and policies are routinely and easily violated means that \( \kappa \approx 0 \) in which case \( R_0(t) \) stays near the pre-lockdown value of \( R_0 \) and hence infections would continue to grow, exponentially.

Results

**Ineffective lockdown**

We keep the parameter values of Fig. 4 except we now investigate how an ineffective lockdown would manifest. In particular, we now employ the time varying equation for \( R_0 \), i.e. Eq. (34) and consider variations in the lockdown parameter \( \kappa \). \( R_\infty \) will be kept at the value of \( R_\infty = 0.1 \) throughout this article.

Fig. 5 gives the time varying basic reproduction number starting from \( R_0 = 10 \), with \( R_\infty = 0.1 \) and \( \kappa = 0.0001 \).

We display the corresponding evolution of the population subgroups in Fig. 6.

As is evident from Fig. 6, the proportion of infectious individuals, especially from the asymptomatic subgroup (E), remains very high even after 30 days. Indeed the proportion of infected individuals records massive growth from its initial 1% to nearly 28% at 30 days, with nearly 18% of the total population now being in the asymptotic (E) subgroup at day 30.

**More effective lockdown**

A stricter enforcement and observance of the lockdown rules, regulations, and policies corresponds to a higher value of \( \kappa \). Fig. 7 gives the time varying basic reproduction number starting from \( R_0 = 10 \), with \( R_\infty = 0.1 \) and \( \kappa = 0.01 \).
Fig. 6. Evolution of population subgroups under ineffective lockdown conditions.

Fig. 7. Time varying basic reproduction number, \( R_0(t) \), with \( R_0 = 10 \), \( R_\infty = 0.1 \), and \( \kappa = 0.01 \).

The corresponding evolution of the population subgroups is shown in Fig. 8.

In stark contrast with the disastrous growth in the number of infectious individuals under conditions of an ineffective lockdown as was demonstrated in from Fig. 6, we observe the complete opposite behaviour with regards when a more effective lockdown is in place, see Fig. 8. In particular, and as can be observed from Fig. 8, such an effective lockdown leads to a significant decrease in non-essential contact between individuals and hence the number of infectious individuals correspondingly also decreases, significantly, nearly vanishing from the population after 30 days under the conditions of Fig. 8.

Limited testing

In our model, \( \xi \) (the proportion of asymptomatic individuals from within the entire group of infected individuals) represents the testing parameter. High values of \( \xi \) (say \( \xi \approx 1 \)) correspond to a high volume of asymptomatic individuals and hence also to a limited/weak testing regime. Mass testing, with rapid results, would drastically reduce the proportion of asymptomatic individuals and hence corresponds to low \( \xi \) values (\( \xi \approx 0 \)). Individuals who show symptoms of COVID-19 would

Fig. 8. Evolution of population subgroups under stricter lockdown conditions.
be easier to identify, indeed, would be expected to voluntarily present themselves to hospitals or similar medical facilities for testing and/or treatment; this especially given the very real risk of an untimely death from COVID-19. The focus of testing processes would therefore be largely focused on asymptomatic individuals, who may remain unaware that they are infectious and hence pose a risk to themselves and others.

We keep the parameter values of Fig. 8 except we now investigate the effects of limited testing, or slow and non-realtime testing in which test results take several days. In our model, this amounts to using high values of $\xi$, which naturally also implies possible changes to the parameter $\rho$ since we must always ensure that $0 \leq \xi + \rho \leq 1$. In particular, high values of $\xi$ will correspondingly imply lower values of $\rho$.

Fig. 9 gives the time varying basic reproduction number starting from $R_0 = 10$, with $R_\infty = 0.1$ and $\kappa = 0.01$. The corresponding evolution of the population subgroups is shown in Fig. 10.

It is evidently clear from Fig. 10 that it would take a long time for the high volume of asymptomatic individuals to decreases significantly. Indeed the proportion of infected individuals records significant growth from its initial 1% to nearly 8% at 30 days, with nearly 7% of the total population now being in the asymptotic (E) subgroup at day 30.

Mass testing

Very low values of $\xi$ correspond to a mass testing regime. It is crucially important that the roll out of mass testing be supplemented rapid and realtime test results, otherwise the mass testing efforts would in fact be wasted, especially in time consuming contact tracing processes. Fig. 11 gives the time varying basic reproduction number starting from $R_0 = 10$, with $R_\infty = 0.1$ and $\kappa = 0.01$.

The corresponding evolution of the population subgroups is shown in Fig. 12. Fig. 12 shows that, under the mass testing regime, the high risk groups (E and U) have almost vanished from the population, the majority of infected individual being now confined to quarantine. Evidently, coupled with the proposed strategy for mandatory quarantine of all individuals who test positive for the virus, very limited resources would need to be dedicated to the laboriously time consuming processes of contact tracing. Indeed the need for large scale contact tracing would not exist!

Ineffective quarantine

Voluntary quarantine methods such as self isolation are ineffective. It takes a lot of responsibility, indeed near superhuman capabilities,
for individuals to maintain a strict self isolation schedule and this unfortunately does not always work given the nature of human beings and their various needs. In fact, self isolation would not work in densely populated neighbourhoods which are generally located in very poor communities.

Even if a self isolating individual may not come into direct personal contact with other persons, they may indirectly spread the infections by contaminating any of a number of surfaces, sometimes unconsciously, thereby spreading infections. In addition to proper hygiene and hand washing, we are, for example, repeatedly cautioned against touching our faces. The fact that the absolute majority of individuals fail to abide by this simple caution shows that self isolation is indeed equally a myth.

Until such a time when a vaccine for the COVID-19 virus or a cure for the COVID-19 disease is discovered, all individuals who test positive for the COVID-19 virus must be placed in mandatory (or involuntary) quarantine until such a time as they recover!

In our model, high values of \( q \) correspond to ineffective quarantine. Fig. 13 gives the time varying basic reproduction number starting from \( R_0 = 10 \), with \( R_\infty = 0.1 \), \( \kappa = 0.01 \), \( \xi = 0.3 \), \( \rho = 0.6 \), and \( q = 1 \).

Fig. 14. Evolution of population subgroups under ineffective quarantine conditions \( \xi = 0.3, \rho = 0.6 \) and \( q = 1 \).
Fig. 15. Time varying basic reproduction number, \( R_0(t) \), with \( R_0 = 10, R_\infty = 0.1, \kappa = 0.01, \xi = 0.3, \rho = 0.6, \) and \( q = 0 \).

Fig. 14 shows that, under an ineffective quarantine regime, the proportion of high risk infectious groups (E and U) remains significantly high within the population and that it will take a long time for this proportion to decrease significantly.

Mandatory quarantine

Mandatory (or involuntary) quarantine methods are the most effective methods of ensuring that individuals who test positive for the COVID-19 virus do not transmit and spread the virus.

In our model, mandatory quarantine corresponds to low values of \( q \), i.e. \( q \approx 0 \). Fig. 15 gives the time varying basic reproduction number starting from \( R_0 = 10 \), with \( R_\infty = 0.1 \) and \( \kappa = 0.01 \).

The corresponding evolution of the population subgroups is shown in Fig. 16.

Fig. 16 shows that, under the effective (mandatory) quarantine regime, the proportion of high risk infectious groups (E and U) has significantly decreased within the population, and is on track to vanish completely from the population in a reasonable amount of time, say from around day 30.

Discussion and conclusion

We have successfully developed a stochastic epidemic model for modelling the transmission and spread of the COVID-19 virus within a given population over a single or multiple waves. Our model uses a novel treatment of the infective group (I) which enables us to reduce the number of equations governing the transmission dynamics of the COVID-19 virus from the seven equations that would correspond to a full SEUQHRD compartmental model down to four equations for an equivalent SIRD model.

We employ dimensionless parameters and corresponding non-dimensional equations and hence our model applies to any setting, country, or population group. Similarly, being a stochastic model which takes into account data variability and random diffusion processes, our model applies to any population size, including to small populations which may indeed face extinction. Deterministic models would be incapable of modelling or predicting such phenomena.

The number of parameters in our model reduces significantly to: 6 rate parameters \( \rho, \xi, q, r, \mu, \) and \( \mu_H \), which can be directly inferred from the available data in a given population, 1 contact parameter \( \kappa \) that measures the level of adherence to lockdown regulations, and 5 stochastic variation parameters \( \sigma_\rho, \sigma_\xi, \sigma_q, \sigma_r, \sigma_H, \) and \( \sigma_H \). In the near future we aim to supplement this research with parameter estimation models to accurately determine the value of stochastic variability parameters from a given data set. Similarly we aim to deploy ideas from stochastic optimal stopping problems, [10–12] to better determine more accurate time-frames for processes such a lockdowns, this especially given that governments across the world continue to use arbitrary lockdown time-frames, ranging from as low as 15 days, which unfortunately have to be regularly shifted, causing widespread anxiety given that citizens are then unable to make any realistic plans.

We have otherwise successfully demonstrated, in summary, that; Lockdowns are crucially important in reducing the rate of transmission and spread of the COVID-19 virus, but only if managed efficiently, otherwise the number of infectious individuals that are outside of hospitals or quarantine facilities would remain significantly high at the end of the lockdown period;

With an efficient lockdown in place, it is important to use the period of the lockdown to carry out mass testing, with rapid and real-time test results, in order to identify and properly manage any infected individuals as this would greatly assist in eliminating any further transmissions and spread of the infections, both during and post lockdown, by such identified and infected individuals to the as yet still ‘healthy’ individuals within the population;
Once the infected individuals have been identified via, say, the mass testing, mandatory quarantine methods would be the most effective way to reduce and indeed eliminate further transmissions and spread of the virus, both during and post lockdown, by such identified and infected individuals to the as yet still ‘healthy’ individuals within the population;

An effective management of these three broad interventions would ensure that the lockdown may be lifted in reasonable time and hence further mitigate against socio-economic turmoils that come with lockdowns.

Declaration of competing interest

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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