Mathematical Modelling of the Dynamics of COVID-19 Disease Transmission

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Introduction

On January 10, 2020, the World Health Organisation (WHO) recognised and designated COVID-19 in response to an earlier virus-borne infection outbreak that occurred in Wuhan, China in December 2019. Due to thousands of confirmed illnesses and thousands of deaths worldwide, the COVID-19 pandemic is regarded as the greatest threat to the international community [1]. As a result, it was deemed a Public Health Emergency of International Concern by the WHO [2]. In [3, 4], a few SIR models have been put forth and examined.

An epidemiological compartmental model, which includes hospitalised classes and deaths, was developed in [1] and accounts for the super-spreading phenomenon of certain individuals. Their model's sensitivity analysis reveals that the variables most sensitive to the fundamental reproduction number are the human infection rate,

- ## Model formulation

The goal of the model is to explain how the disease develops over the course of an infection once an index case is introduced into a community that is fully susceptible. We adopt the same assumptions as other models of infectious diseases, taking into account the kinetics of the disease as well as the processes of birth and natural death. We use the mass action principle with a corrective term describing the logistic population growth rate if the disease didn't exist. The entire human population, denoted as N (t), is split up into five classes: the infectious symptomatic class S(t), the infectious asymptomatic class A(t), the non-infectious Recovered class R(t), and the non-infectious susceptible class C(t). This means that N = C + L + S + A + R.(2.1)
The variables of the State

Table 1. List of model variables

Variable of stateSynopsis NThe total population of humans, C the susceptible population, L the exposed, non-infected population, and S the symptomatic, infectious population Human population, asymptomatic, infectious R; recovered, non-infectious Vquantity of viruses present on surfaces

Fig. 1. Pathway diagram of the COVID-19 model showing (a) the progression (solid) and transmission (dashed) of the disease between compartments; the variable names are listed in Table 1. The connecting arrows are labelled with the associated rate constants, where the natural death of each of the classes are not shown for clarity

Susceptible humans get infected by contacting infectious humans and viruses from surfaces at rates

$\beta_1 \frac{S}{N} C$, $\beta_2 \frac{A}{N} C$ and $\beta_3 V C$, where β_1, β_2 and β_3 are rate constants. The fractions $\frac{S}{N}$ and $\frac{A}{N}$ are

the likelihoods that the contacts are with persons who exhibit symptoms or not. It should be noted that class L persons are not infectious; rather, they are in the exposed stage of infection. In the absence of the disease, θ3N 2, which represents the per capita resource availability for the human population, prevents the population from growing infinitely and attracts susceptible persons into the population through a steady birth rate, λ1. After a mean latency period of η1, a fraction of incubating persons become asymptomatic, and after that, they become infectious. This presumption differs from that of [5], wherein the authors proposed two incubation periods, despite intending only one. Every human class "dies naturally" at a rate of μ1 per capita, yet certain people

$$\frac{dC}{} = \lambda N - (\beta$$

$S + \beta A + \beta V + \mu)C - \vartheta N^2,$ (2.2)

dt 1

^1N $_2N$ 3 1 3

Table 2. Model parameters and their dimensions. Values marked with bullet (•) are assumed values in other mathematical models and those marked with asterisk (∗) are obtained from experimental sources

$$g_0 = y_1y_2y_3y_8,\ g_1 = ah_2 + bh_3 + \vartheta h_4,\ g_2 = ah_5 + \vartheta h_6,\ g_3 = ah_7 + bh_8 + \vartheta h_9,\ g_4 = \vartheta h_{10},$$
$$h_1 = y_2y_3y_8,\ h_2 = y_3y_6y_8 + \omega y_7y_8,\ h_3 = y_2y_7y_8,\ h_4 = y_3y_4y_6 + y_2y_5y_7 + \omega y_4y_7, \quad (3.4)$$
$$h_5 = y_1y_3y_8,\ h_6 = y_1y_3y_4,\ h_7 = \omega y_1y_8,\ h_8 = y_1y_2y_8,\ h_9 = y_1y_2y_5 + \omega y_1y_4,\ h_{10} = y_1y_2y_3.$$

The characteristic equation of (3.3) in terms of the eigenvalue, λ^*, shows that three of the eigenvalues vanish leaving the expression

$$= \frac{g_1}{B_4}, \quad (3.5)$$

which expressed in terms of the model parameters gives

$$R = \frac{\gamma(agh(m\omega + r_2(\mu + \omega + q)) + r_1(a\eta + dq)(m\omega + r_2) + m(\mu + 1)(bgh + r_1(b\eta + e\vartheta)))}{(\mu + 1)(\mu + \gamma)(\mu + \omega + q)(gh + \eta r_1)}. \quad (3.6)$$

where $r_1 = (\lambda - \mu) > 0$ and $r_2 = (1 - m) > 0$. This follows from section2.1, where we have assumed that natural birth rate is greater than natutal death rate and $m \in (0, 1)$. The condition $m = 1$, signifies all cases are asymptomatic while $m = 0$, is a situation where all infectious humans are sick.

• Positivity, existence and uniqeness of solution

The model is described in the domain

$$\Gamma \in R^7 = \{C, L, S, A, R, V, N : C \geq 0, L \geq 0, S \geq 0, A \geq 0, R \geq 0, V \geq 0,$$
$$N > 0, C + L + S + A + R = 1\}, \quad (3.7)$$

Suppose at $t = 0$ all variables are non-negative, then $C(0) + L(0) + S(0) + A(0) + R(0) = 1$ and $V(0) = 0$. If $L = 0$, and all other variables are in Γ, then $\frac{dL}{dt} \geq 0$. This is also the case for all other variables in (2.14)—(2.17). But if $C = 0, \lambda > \mu$ and $N < \frac{\lambda - \mu}{}$, then $\frac{dC}{dt} \geq 0$. If $N = 0$, then $\frac{dN}{dt} = 0$.

h

But if $N > 0$ assuming $\lambda > \mu$ i.e. λ

$> \mu$, then with appropriate initial conditions, $\frac{dN}{dt} > 0$ for

1 1

all values of $t > 0$. We note that the right-hand side of (2.12)—(2.18) is continuous with continuous partial derivatives, so solutions exist and are unique. The model is therefore mathematically and biologically well posed with solutions in Γ for all $t \in [0, \infty)$.

• Steady state solution and stability analysis

It can be shown from the system that the disease free state is $(C, L, S, A, R, V) = (1, 0, 0, 0, 0, 0,)$. In the absence of infection, $S = 0$ and $A = 0$. Substituting these into the right hand side of (2.17)
— (2.13) in that order, we obtain $V = 0$, $R = 0$ and $L = 0$. Further substitution of the values of S, A and V into (2.12), we obtain $C = 1$. At the disease free state $C = N$, meaning all humans are entirely susceptible and we obtain from (2.18) the following logistic equation,

$$\frac{dN}{} = rN\left(1 - \frac{N}{}\right), \tag{3.8}$$

where $r = \lambda - \mu$ and $K = \frac{\lambda-\mu}{}$. The solution of (3.8)is given as

$$N(t) = N + (K - N)e_{-rt} \tag{3.9}$$

and as $t \longrightarrow \infty$, $N(t) \longrightarrow K = \frac{\lambda-\mu}{}$, the carying capacity of the environment. The disease free state is locally asymptotically stable when $R_0 < 1$ and unstable for $R_0 > 1$.

We derive sufficient conditions for local stability of the disease free state from all initial conditions $\in \Gamma$. The Jacobian matrix obtained by linearising system (2.12)—(2.18) about the disease free equilibrium point, $(C, L, S, A, R, V, N) = \left(1, 0, 0, 0, 0, 0, \frac{\lambda - \mu}{h}\right)$ is

We note the linear factorisation = (3.11) clearly yields negative real eigenvalues, however, from the quartic equation, no such deduction can immediately be made.

Lemma 3.1. *The disease-free equilibrium is locally asymptotically stable if* $R_0 < 1$ *and unstable if* $R_0 > 1$.

Evidence. The quartic polynomial of (3.11) has positive and non-zero coefficients, as observed by the definitions of the constants in (3.2) and (3.4). According to Descartes' rule of signs, there are no positive real eigenvalues, which means that there are either four negative real eigenvalues, two negative real eigenvalues, one pair of complex conjugates with negative real parts, or two pairs of complex conjugate eigenvalues with negative real parts. We must demonstrate that the fourth order polynomial Routh Hurwitz stability criteria, as described in [10], can be written in our situation in a way that satisfies the requirement.

$$\psi = B_1B_2B_3 - (B_3^2 + B_1^2B_4) > 0 \tag{3.13}$$

We need to express ψ as a finite sum of positive terms involving the model parameters. Using Maple to undertake the tedious algebra, we are able to show that ψ is indeed a sum of positive terms given by

$$\psi = D_1(1 - R_0)^2 + D_2(1 - R_0) + k_1E + k_2F + k_3G + k_4H + k_5J + k_6L + L_0. \tag{3.14}$$

The Maple input file used in obtaining the results is not included here due to its size but can be made available on request. However, expressions for the constants are stated as follows:

$B_7 + y_8$, $C_5 = 2B_{10}B_{13}k_3y_1 + aB_{10}k_4y_1(B_{13} + k_7 + k_{10})$, $C_5 = y_1y_2y_8$,

$B^3\ y_1(B_{13} + y_8 + y^2 + k_7 + k_{11})$, $C_7 = B^3\ [B_{13}C_2 + y^2(k_{10}y_2 + k^2)]$,

$kk_{11}k_{12}\ [B_{10}(k_9 + 2k_{10} + 2k_{11}) + 2C_1k_7 + k_{11}(C_2 + y_3)]$, $B_{22}y_8\ \ B_9k_7y_8 +$

$2B_{18}(B_{10} + y_3) + 2C_1k_{10}y_3 + 2k^2$,

$B_{18}C_2(B_{10}y^2 + 2C_1B_{18}) + B_{18}k_{11}(B_{10}y^2 + B_{18}k_{11})$, $C_{11} = y_3 + 2y_8$,

$k_1\ [2k_{11} + k_{11}B_{16}(y_2 + B_{10}) + k_{12}]$,

$[B_9k_9 + B_{22}(1 + y_2) + 2B_{24} + B_{18}(2 + C_2) + C_2y^2 + k_{10}y_3 + k_7]$,

$B_{13}B_{14}k^2\ k^2$, $D_2 = k11k_{12}(B_{19}B_{20}B_{21}$,

$2B_{10}k_{11}(B_7k_5B_{16}k_6) + C_2k_5y_2\ [B_{10}y_1 + C_2(2y_2 + y_3)]$, $C_2k_5y_2\ [C_2(B_7 + B_{10}) +$

$(2C_2y_2 + C_2y_3 + k_8 + k_{12})]$,

$B_{10}k_3k_{12}(B_7k_9 + 2k_9y_3 + k_{12}y_3) + C_1y^2 + k_1k_{10}(B_{13} + y^2)$,

$2B_{10}B_{16}k_2k_7 + C_{10} + B_{10}k_{12}\ [k_9y_8(2 + k_8 + k_{11}) + 2B_{18}C_2y_8]$,

$$B_1B_{10}y_8\,[k_9(2 + k_8 + k_{11}) + 2B_{18}C_2] + C_{12} + C_{13},\ C_{19} + B_{18}^{2}\,[2B_{18}(k_7 + k_{10} + y^2) + B_5y_8 + y_7]\,,$$

$$k_3\,[2B_{10}B_{24} + B_1k_{10}y_2 + 2k_7y_2(B_{11} + k_{10})] + C_4k_2k_7y_8,$$

$$k_4\,[k_{12}(B_{13} + k_{10}) + y^2(k_7 + k_{10}) + B_{11}B_{18} + y_8 + {}_{10}\,k_7y_2 + B_{18}C_2y_3]\,,$$

$$k_5\,[B_{13}k_{12} + k_{10}(k_7 + k_{11}) + k_{10}(k_{10} + B_6) + B_{23}y^2 + B_{10}B_{18}] + 2B_{16}B_{18}k_6,$$

$$B_{18}y_1(4B_{17}B_{18} + C_{11}k_7y_8 + B_{13}k_9 + k^2 + k^{2}_{7}\,),$$

$$B_{18}\,[B_{10}k^2 + C_{11}k^2 + 4B_{13}B_{18} + 3B11B_{18} + B_{18}(k_7 + 2y^2) + C_2k^2 + k_5k_{11}y_3]\,,$$

$$k_4\ \ 3B_{10}B_{18} + C_2(2B_{10}k_{12} + B_{10}k_{11} + 3k_{10}y_2 + 2y^3)\ \ ,$$

$$k_5\,[B_{10}(B_{22} + B_{24} + 2B_{26} + k_{10}y_2) + C_2y_2(B_5 + C_2 + k_{10}) + C_{11}k_{11}y_2]\,,\ k_6[3B_{22}C_2 + B_6(B_{10} + y_3) + 2C_{11}y_3 + C_2k_{10}y_3(B_{10} + 2k_5)],$$

$$k_3\ \ B_{10}y_8 + B_{17}k_8y_2 + k_{10}(B_{10}C_1 + k_{11} + y^2) + k_{11}(B_{16} + y_{11})\ \ ,\ B_{18}k_{12}\,[B_7C_2y_8 + y_8(B_{17} + C_{11}y_8 + 3y_3) + B_{18}],$$

$$B_{18}y_2\,[3B_7B_{17} + k_{10}y_3(1 + C_{11}) + k^2(1 + y_8) + C_2k_{11}(C_2 + y_3)]\,,$$

$$k_6y_3\,[2B_{16}k_7 + C_{11}k_8 + B_{10}(k_{10} + C_2k_{11}]\,,$$

$$k_3\ \ B_{22}C_4 + B_{18}(B_{17} + y_8) + C_2y^3 + B_{10}k_{10}y_3 + k_7(k_{11}) + k_{12})\ \ ,\ B_{18}y_1\,[B_6C_{11} + 3B_{17}B_{18} + k_{10}(k_{10} + k_{11})]\,,$$

$$B_{18}\,[B_{17}k^2 + 3B_{18}(B_{13} + y^2) + B_{18}(B_{11} + y^2) + C_2k^2\ ]\,,$$

$$B_{18}\,[B_{17}k^2$$

$$+ 3B_{18}(B_{14} + y^2) + B_{18}(C_2k^2$$

$$+ 2y^2)\,]\,,$$

$$k_5\ \ B_{18}(B_{17} + y_2) + B_{16}B_{22} + y_3(B_6 + B_{10}y^2 + k_{10} + k_{11})\ \ ,\ E_1 + E_2 + E_3 + E_4 + E_5 + E_6,\ \ F\ = F_1 + F_2 + F_3 + F_4,$$

$$G_1 + G_2 + G_3 + G_4 + G_5 + G_6,\ H = H_1 + H_2 + H_3 + H_4 + H_5,\ J_1 + J_2 + J_3 + J_4 + J_5,$$

$$B_{18}k_{11}\,[y_1(2B_{13} + k_9 + y^2) + y_2(2B_{13} + y^2 + y^2) + C_2k_{11} + C_4k_6k_{11}y_3]\,,$$

$$L_0\ \ =\ \ C_6 + C_7 + C_8 + C_{14} + y^2(B_{10} + C_1) + k_8.$$

Assuming that R0 < 1, ψ > 0 since D1, D2, E, F, G, H, J, L, L0, ki, i = 1, 2,..., 6 > 0. R0 < 1 indicates that the disease-free equilibrium is thus locally asymptotically stable. The coefficient B1 is positive, and we find that B4 is negative if R0 > 1, meaning that the signs of B2 and B3 are not immediately apparent. It is observed that there is a single sign change. If B2 and B3 are both positive or negative, or if B2 is positive and B3 is negative. Nevertheless, if B2 is negative and B3 is positive, there are three sign changes. Therefore, in the worst case, there is just one sign change in the sequence of coefficients, 1, B1, B2, B3, and B4. We determine that the disease-free condition is unstable if R0 > 1 by applying Descartes' rule of sign, which states that there must be at least one positive real eigenvalue.

In the case when R0 = 1, (3.11) has a single zero eigenvalue, indicating that R0 = 1 is a bifurcation surface in the parameter space of (β1, β2, β3, γ1, γ2, σ1, σ2, θ1, θ2, λ1, μ1, ω1, m).

- **Numerical solution**

Fig. 2. Results showing the effect of the initial infected humans on evolution of infection where t = 1, represents approximately 10 days in real time. The initial conditions used are C = 1, L = 0.01, S = 0, A = 0, R = 0, V = 0, N = 1 and the parameter values are given above

The numerical solution is obtained by using MATLAB's ode15s, a variable order Runge-Kutta method with a relative tolerance of 10^{-8} and absolute tolerance of 10^{-9}. The dimensionless parameters used for the simulations are defined in (2.11) with numerical values; λ = 0.00439, a = 1.57, b = 1.26, μ = 0.00362, ϑ = 0.000137, α = 0.00463, γ = 0.0485, h = 0.000767, ω = 0.000357, d = 0.00403, e = 0.0101, g = 0.101, η = 0.000125, q = 0.00865, m = 0.005. Even though some of these data are different from the original data their adjusted values are significantly within the same order of magnitudes. At time t = 0 we have the following initial conditions in the proportions: C = 0.99, L = 0.01, S = 0, A = 0, R = 0, V = 0, N = 1. This is a situation where

the entire susceptible human population is exposed to a small fraction of infected humans. The program was run in MATLAB with different sets of initial conditions, and the qualitative form of the steady state solutions were the same, although the system gets to a steady state faster as initial value of L increases.

Fig. 3. Results showing the effect of the initial infected humans on evolution of infection where t = 1, represents approximately 10 days in real time. The initial conditions used are C = 1, L = 0.01, S = 0, A = 0, R = 0, V = 0, N = 1 and the parameter values are given above

Fig. 6. Results showing the disease free state precisely, R_0=0.9982 for viruses of surfaces, recovered humans and the total human population. Parameter values are the same as those in Fig. 5

In Fig. 3a, the proportion of susceptible human population drops and picks, and later drops before finally increasing to a steady state This behaviour assumes an opposite trajectory in Fig. 3b,c,d and Fig. 4a,b where the level of infection, recovery and viruses on environmental surfaces pick and drop and later pick before dropping to a steady state. In 4c, the human population drops in a fast time scale due to the disease related death caused by early invasion of the virus. While Fig. 3 and Fig. 4 show prospect of the disease being endemic for $R_0 > 1$, Fig. 5 and Fig. 6 demonstrate a situation of disease eradication when $R_0 < 1$.

Discussion

Our model explains how the introduction of an index case causes COVID-19 to spread throughout a completely susceptible human population. The available parameter values we employed in our model indicate a prospective endemic character of the disease, even if precise values for the pandemic have not yet been determined. This is corroborated by the value of R0, which is 2.1 utilising (3.6) and was previously estimated as 2.4829 in [5]. Due to the enormous number of minor factors that are inherent in the numerical simulations, they display characteristic traits of long term solutions that require thorough study utilising different types of analysis, including asymptotic. This will highlight the important time ranges at which certain events take place as the illness progresses. This might be a project for the future to learn more about how the illness spreads.

We observe that the three nondimensional parameters—γ, a, and b—are highly significant, with γ being the most useful in managing the illness, as may be inferred from the fundamental reproduction number. R0 is consistently increased or decreased with a rise or fall in. This is seen in figures 5 and 6, when the disease is eradicated with γ. Reducing γ1 and increasing θ1 is an effective technique to considerably lower γ, since γ = η1. In other words, slowing down the spread of infection among diseased people and speeding up the healing process for those who are ill. Since those who are infected without symptoms are far less likely to spread the virus than those who do, raising the value of b to a substantial extent will raise R0, while lowering b to zero might not have a significant impact [11, 12]. Comparably, lim R0 is mostly dependent on other factors, therefore lowering a by itself might not have a major effect on disease control (a→0). R0 is decreased more quickly with a combined downsizing of a and d, though. This implies that attempts to lessen contact with viruses on surfaces should be coupled with measures to avoid infectiousness in persons who have been exposed.

According to the findings of [1], there is a direct correlation between R0 and the rate at which an exposed condition becomes infectious. However, as can be inferred from the fundamental reproduction number, R0, the disease-related death rate is irrelevant in our

model. It is erroneous to believe that a rise in disease-related mortality would lower the basic reproduction number, even if [1] suggests that disease-related death rates may have a function in disease control. Even while such a result would be theoretically tractable, it might not have any biological or epidemiological significance because disease control does not require a rise in human mortality.

Conclusions

In this study, we present a basic mathematical model of the COVID-19 transmission, which originated in Wuhan, China, and has had a major impact on the majority of small and even large economies worldwide. Because an index case is introduced, the model concentrates on the dynamics of disease transmission in a population that is completely susceptible. The findings display some long-term damped oscillatory behaviours that don't appear to be ending anytime soon. A basic reproduction number analysis reveals that a control mechanism aimed at managing exposed persons to keep them from spreading to susceptible humans and surfaces could effectively impede the spread of disease. In addition to the quarantine, testing and early diagnosis of infectious cases

The usage of masks and social separation are two strategies that may help stop the spread of disease. Since COVID-19 is still a relatively novel disease, there is still much to be done in terms of estimating the parameters that govern its behaviour. It is necessary to thoroughly examine the numerical solutions' innate long-term solutions.

Owing to the disease's mechanism of transmission and unique behavioural patterns brought on by a variety of social, cultural, and religious factors, the development of a suitable vaccination and effective public education about the vaccine's importance are the surefire steps to its elimination. Even while we wish to recognise the World Health Organization's efforts to maintain global health, it may not have been as proactive as it could have been. If China had been able to limit this COVID-19, the world would have suffered significantly less. In the event that an epidemic breaks out in any nation, we propose that the WHO impose a restriction that permits only entry and prohibits all other forms of departure from the nation while medical personnel and humanitarian aid from other nations are sent in to help contain the outbreak.

Competing Interests

The authors have stated that there are no conflicting interests.

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