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Mathematical Modelling of the Dynamics of COVID-19 Disease Transmission

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Authors' contributions

This work was carried out in collaboration between both authors. Author ABO designed the study, performed the analysis and wrote the protocol. Author EE managed the analyses and wrote the first draft of the manuscript. Both authors read and approved the final manuscript.

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Abstract

We construct a simple mathematical model that describes the dynamics of the transmission of COVID-19 disease in a human population. It accounts for the various phases of the disease and its mode of contact through infectious humans and surfaces. The contribution of asymptomatic humans in the dynamics of the disease is well represented. The model is a system of ordinary differential equations that describes the evolution of humans in a range of COVID-19 states due to emergence of an index case. The analysis includes establishment of the basic reproduction

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number, R_0, where, $R_0 < 1$ signifies a disease free state that is locally asymptotically stable. A key result in this study shows some long term damped oscillatory behaviour that do not seem to end soon.

Keywords: COVID-19; modelling; symptomatic; asymptomatic; surface virus.

1 Introduction

COVID-19 was identified and named by the World Health Organisation (WHO) on January 10, 2020 following an ealier virus borne infection episode in Wuhan, China in December, 2019. The COVID-19 pandemic is considered as the biggest global threat worldwide because of thousands of confirmed infections, accompanied by thousands deaths over the world [1]. Hence, the WHO declared it to be a Public Health Emergency of International Concern [2]. Some SIR models have been proposed and analysed in [3, 4].

In [1] an epidemiological compartmental model that takes into account a super-spreading phenomenon of some individuals including fatality and hospitalized classes was proposed. The sensitivity analysis of their model shows that the most sensitive parameters to the basic reproduction number are infection rate of humans , the rate at which exposed humans become infectious and the disease related death rate. Increase in the infection rate and the rate at which exposed individuals become infectious increase the basic reproduction number , and in contrast, the disease related death rate and the basic reproduction number are inversely related.

A more comprehensive approach was adopted by [5] where the Wuhan meat market is represented in the model with a continuous flow of the Corona virus from bat population.

Here we propose a simple mathematical model incorporating some of these features. However, instead of considering a continuous flow of the virus from a bat population we consider the dynamics of the disease following the emergence of an index case. Section 1 includes a brief introduction where as the formulation of the model is given in section 2. In section 3, we present the model analysis, numerical simulations and discussion. The paper was rounded up by a brief conclusion in section 4.

2 Model formulation

The model is intended to describe the progression of the disease over a period of infection following the introduction of an index case in an entirely susceptible population. We make assumptions usually associated with other infectious disease models, whereby birth and natural death processes as well as the disease kinetics are considered. We employ the principle of mass action including a correction term that describes the logistic population growth rate in the absence of the disease. The total human population, $N(t)$, is divided into 5 classes namely, a non-infectious susceptible class, $C(t)$, a non-infectious latent class, $L(t)$, an infectious symptomatic class, $S(t)$, an infectious asymptomatic class $A(t)$ and a non-infectious Recovered class $R(t)$; Hence

$$N = C + L + S + A + R. \tag{2.1}$$

The State variables in the model are real and non-negative. Their description is given in Table 2 and the movement between compartments is summarised in Fig. 1, with the individual pathways discussed below.

Table 1. List of model variables

State variable	Description
N	Total human population
C	Susceptible human population
L	Exposed, non-infectious human population
S	Symptomatic, infectious human population
A	Asymptomatic, infectious human population
R	Recovered, non-infectious human population
V	Number of viruses on surfaces

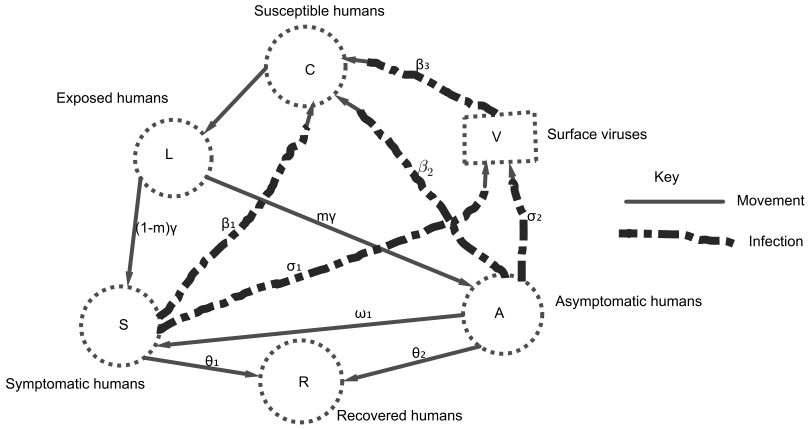

Fig. 1. Pathway diagram of the COVID-19 model showing (a) the progression (solid) and transmission (dashed) of the disease between compartments; the variable names are listed in Table 1. The connecting arrows are labelled with the associated rate constants, where the natural death of each of the classes 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 $\beta_3 VC$, where β_1, β_2 and β_3 are rate constants. The fractions $\frac{S}{N}$ and $\frac{A}{N}$ are the probabilities that the contacts are with symptomatic and asymptomatic humans. We note that humans in class L are in the exposed stage of infection and are not infectious. Susceptible humans are recruited into the population through a constant birth rate, λ_1 with a correction term, $\theta_3 N^2$, stopping the population from growing without limit in the absence of the disease, where θ_3 is per capita resource availability for the human population. Incubating humans become infectious after a mean latency time, γ_1 where a proportion, m of them become asymptomatic. This assumption is different from that of [5], where they suggested two incubation period even though they meant a single incubation period. All human classes "die naturally" at per capita rate, μ_1 while some individuals in the S class die at an additional rate $\alpha_1 S$ from the disease. We also assume that COVID-19 patients recover without being reinfected since there has not been concrete evidence to that effect. Surface viruses die at rate $\gamma_2 V$ while symptomatic and asymptomatic humans contribute to the emergence of surface viruses at rates $\sigma_1 S$ and $\sigma_2 A$ respectively with σ_1 and σ_2 as rate constants. The proposed model consistent with the above assumptions is given as

$$\frac{dC}{dt} = \lambda_1 N - (\beta_1 \frac{S}{N} + \beta_2 \frac{A}{N} + \beta_3 V + \mu_1)C - \theta_3 N^2, \tag{2.2}$$

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

Symbol	Description	Value	Unit	Source
λ_1	Per capita human birth rate	0.0000433 •	day^{-1}	[5]
β_1	Infectious contact rate of symptomatic and susceptible humans	0.05 •	day^{-1}	[5]
β_2	Infectious contact rate of asymptomatic and susceptible humans	0.124 •	day^{-1}	[5]
β_3	Infectious contact rate of surface viruses and susceptible humans	0.00000123 •	$virus^{-1}day^{-1}$	[5]
α_1	Disease induced death rate of symptomatic humans	0.043 ∗	day^{-1}	[6]
μ_1	Per capita human death rate	0.0000357 •	day^{-1}	[5]
γ_1	Transition rate from exposed state to infectious state	0.000479 •	day^{-1}	[5]
γ_2	Motality rate of viruses on surfaces	0.01	day^{-1}	[5]
m	Proportion of exposed humans becoming asymptomatic	0.005 •	nondimensional	[5]
θ_1	Recovery rate of symptomatic humans	0.0987 •	day^{-1}	[5]
θ_2	Recovery rate of asymptomatic humans	0.854 •	day^{-1}	[5]
θ_3	Per capita resource avilability for the human population	0.00024 •	$human^{-1}day^{-1}$	[7]
ω_1	Loss of asymptomatic status	0.035	$human^{-1}day^{-1}$	Assumed
σ_1	Contribution of symptomatic humans to surface viruses	0.00398 •	$virus\ human^{-1}day^{-1}$	[5]
σ_2	Contribution of asymptomatic humans to surface viruses	0.001 •	$virus\ human^{-1}day^{-1}$	[5]

$$\frac{dL}{dt} = (\beta_1\frac{S}{N} + \beta_2\frac{A}{N} + \beta_3 V)C - (\gamma_1 + \mu_1)L, \tag{2.3}$$

$$\frac{dS}{dt} = (1-m)\gamma_1 L + \omega_1 A - (\theta_1 + \alpha_1 + \mu_1)S, \tag{2.4}$$

$$\frac{dA}{dt} = m\gamma_1 L - (\theta_2 + \omega_1 + \mu_1)A, \tag{2.5}$$

$$\frac{dR}{dt} = \theta_1 S + \theta_2 A - \mu_1 R, \tag{2.6}$$

$$\frac{dV}{dt} = \sigma_1 S + \sigma_2 A - \beta_3 VC - \gamma_2 V, \tag{2.7}$$

$$\frac{dN}{dt} = (\lambda_1 - \mu_1)N - \alpha_1 S - \theta_3 N^2, \tag{2.8}$$

where (2.8) is derived from adding (2.2)−(2.6). The system is to be investigated subject to the initial conditions

$$t = 0, \quad N = N_0, \quad L = L_0, \quad C = N_0 - L_0, \quad S = A = R = V = 0.$$

2.1 Parameter values and nondimensionalisation

All the model dimensional parameters listed in Table 2 are non-negative real numbers with $\lambda > \mu$ and $0 < m < 1$. We note that the values of these parameters are still undergoing a process of development due to the newness of COVID-19 Virus. However, some of the data are assumed and others are estimates obtained from related mathematical models. Since the variable N is the sum of the human compartment values, it is convenient to re-express the compartment values as population fractions using

$$\hat{C} = \frac{C}{N},\ \hat{L} = \frac{L}{N},\ \hat{S} = \frac{S}{N},\ \hat{A} = \frac{A}{N},\ \hat{R} = \frac{R}{N},$$

so that

$$\hat{C} + \hat{L} + \hat{S} + \hat{A} + \hat{R} \quad = \quad 1. \tag{2.9}$$

We define the remaining variables as

$$\hat{V} = \frac{V}{V_0},\ \hat{N} = \frac{N}{N_0},\ \hat{t} = \frac{t}{t_0},$$

which lead to the following time derivatives

$$\begin{aligned}
\frac{d\hat{C}}{\hat{dt}} &= t_0\lambda_1(1-\hat{C}) - (t_0\beta_1\hat{S} + t_0\beta_2\hat{A} + t_0\beta_3V_0\hat{V})\hat{C} - t_0\theta_3N_0(1-\hat{C})\hat{N} + t_0\alpha_1\hat{C}\hat{S},\\
\frac{d\hat{L}}{\hat{dt}} &= t_0\beta_1\hat{S}\hat{C} + t_0\beta_2\hat{A}\hat{C} + t_0\beta_3V_0\hat{V}\hat{C} - t_0\gamma_1\hat{L} - t_0\lambda_1\hat{L} + t_0\theta_3N_0\hat{L}\hat{N} + t_0\alpha_1\hat{L}\hat{S},\\
\frac{d\hat{S}}{\hat{dt}} &= (1-m)t_0\gamma_1\hat{L} + t_0\omega_1\hat{A} - t_0\theta_1\hat{A} - t_0\lambda_1\hat{S} + t_0\theta_3N_0\hat{S}\hat{N} + t_0\alpha_1\hat{S}^2,\\
\frac{d\hat{A}}{\hat{dt}} &= mt_0\gamma_1\hat{L} - t_0\theta_2\hat{A} - t_0\omega_1\hat{A} - t_0\lambda_1\hat{A} + t_0\theta_3N_0\hat{A}\hat{N} + t_0\alpha_1\hat{A}\hat{S},\\
\frac{d\hat{R}}{\hat{dt}} &= t_0\theta_1\hat{S} + t_0\theta_2\hat{A} - t_0\lambda_1\hat{R} + t_0\theta_3N_0\hat{R}\hat{N} + t_0\alpha_1\hat{R}\hat{S},\\
\frac{d\hat{V}}{\hat{dt}} &= t_0\sigma_1\frac{N_0}{V_0}\hat{S}\hat{N} + t_0\sigma_2\frac{N_0}{V_0}\hat{A}\hat{N} - t_0\beta_3N_0\hat{V}\hat{C}\hat{N} - t_0\gamma_2\hat{V},\\
\frac{d\hat{N}}{\hat{dt}} &= t_0(\lambda_1-\mu_1)\hat{N} - t_0\alpha_1\hat{S}\hat{N} - t_0\theta_3N_0\hat{N}^2.
\end{aligned} \tag{2.10}$$

We define the following dimensionless parameters by rescaling time with the recovery rate of symptomatic humans

$$\begin{aligned}
t_0 &= \frac{1}{\theta_1},\ \lambda = \frac{\lambda_1}{\theta_1},\ a = \frac{\beta_1}{\theta_1},\ b = \frac{\beta_2}{\theta_1},\ \mu = \frac{\mu_1}{\theta_1},\ \theta = \frac{\beta_3V_0}{\theta_1},\ \alpha = \frac{\alpha_1}{\theta_1},\\
\gamma &= \frac{\gamma_1}{\theta_1},\ h = \frac{\theta_3N_0}{\theta_1},\ \omega = \frac{\omega_1}{\theta_1},\ d = \frac{\sigma_1N_0}{\theta_1V_0},\ e = \frac{\sigma_2N_0}{\theta_1V_0},\ g = \frac{\gamma_2}{\theta_1},\ \eta = \frac{\beta_3N_0}{\theta_1},\ q = \frac{\theta_2}{\theta_1},
\end{aligned} \tag{2.11}$$

and by substituting these new parameters into (2.10) and dropping the hats for notational clarity we get

$$\frac{dC}{dt} = \lambda(1-C) - (aS + bA + \theta V)C - h(1-C)N + \alpha CS, \tag{2.12}$$

$$\frac{dL}{dt} = (aS + bA + \theta V)C - (\lambda+\gamma)L + hLN + \alpha LS, \tag{2.13}$$

$$\frac{dS}{dt} = (1-m)\gamma L + \omega A - (\lambda+1)S + hSN + \alpha S^2, \tag{2.14}$$

$$\frac{dA}{dt} = m\gamma L - (\lambda + \omega + q)A + hAN + \alpha AS, \tag{2.15}$$
$$\frac{dR}{dt} = S + qA - \lambda R + hRN + \alpha RS, \tag{2.16}$$
$$\frac{dV}{dt} = dSN + eAN - \eta CVN - gV, \tag{2.17}$$
$$\frac{dN}{dt} = (\lambda - \mu)N - \alpha SN - hN^2. \tag{2.18}$$

3 Model Analysis

3.1 Determining the basic reproduction number, R_0

The basic reproduction number is the expected number of secondary infections due to the introduction of a single COVID-19 infection case. Using the next generation matrix approach [8, 9], we consider a small perturbation from the disease free state $C = 1, L = 0, S = 0, A = 0, R = 0, V = 0, N = \frac{\lambda - \mu}{h}$ by investigating the linearised system

$$P' = FP - MP, \tag{3.1}$$

where, $P' = \frac{dP}{dt}$ and

$$F = \begin{bmatrix} 0 & a & b & \theta \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \quad M = \begin{bmatrix} y_1 & 0 & 0 & 0 \\ -y_6 & y_2 & -\omega & 0 \\ -y_7 & 0 & y_3 & 0 \\ o & -y_4 & -y_5 & y_8 \end{bmatrix}, \quad P = \begin{bmatrix} L \\ S \\ A \\ V \end{bmatrix};$$

where, FP represents the emergence of new infections, MP the transition of these infections between compartments and P the "pool of infection". The constants $y_i's$ are non-negative real numbers expressed in terms of the model parameters as follows:

$$\begin{aligned} y_1 &= \mu + \gamma, \quad y_2 = \mu + 1, \quad y_3 = \mu + \omega + q, \\ y_4 &= \frac{d(\lambda - \mu)}{h}, \quad y_5 = \frac{e(\lambda - \mu)}{h}, \quad y_6 = (1 - m)\gamma, y_7 = m\gamma, \quad y_8 = \frac{\eta(\lambda - \mu)}{h} + g. \end{aligned} \tag{3.2}$$

This method assumes that there is a non-negative matrix $G = FM^{-1}$ that guarantees a unique, positive and real eigenvalue strictly greater than all others. Computing the inverse of M yields

$$G = \frac{1}{g_0} \begin{bmatrix} g_1 & g_2 & g_3 & g_4 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{3.3}$$

where,

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

The characteristic equation of (3.3) in terms of the eigenvalue, $\lambda^\star$, shows that three of the eigenvalues vanish leaving the expression

$$\lambda^\star_{max} = \frac{g_1}{B_4}, \tag{3.5}$$

which expressed in terms of the model parameters gives

$$R_0 = \frac{\gamma\left(agh\left(m\omega + r_2(\mu+\omega+q)\right) + r_1(a\eta+dq)(m\omega+r_2) + m(\mu+1)\left(bgh + r_1(b\eta+e\theta)\right)\right)}{(\mu+1)(\mu+\gamma)(\mu+\omega+q)\left(gh+\eta r_1\right)}. \tag{3.6}$$

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.

3.2 Positivity, existence and uniqeness of solution

The model is described in the domain

$$\begin{aligned} \Gamma \in \mathbb{R}^7 &= \{C, L, S, A, R, V, N : C \geq 0, L \geq 0, S \geq 0, A \geq 0, R \geq 0, V \geq 0, \\ N &> 0, C + L + S + A + R = 1\}, \end{aligned} \tag{3.7}$$

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 $N < \frac{\lambda-\mu}{h}$, then $\frac{dC}{dt} \geq 0$. If $N = 0$, then $\frac{dN}{dt} = 0$. But if $N > 0$ assuming $\lambda > \mu$ i.e. $\lambda_1 > \mu_1$, then with appropriate initial conditions, $\frac{dN}{dt} > 0$ for 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 $t \in [0, \infty)$.

3.3 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,

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

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

$$N(t) = \frac{KN_0}{N_0 + (K - N_0)\, e^{-rt}} \tag{3.9}$$

and as $t \longrightarrow \infty$, $N(t) \longrightarrow K = \frac{\lambda-\mu}{h}$, 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

$$J_{df} = \begin{bmatrix} -\mu & 0 & y_{10} & -b & 0 & -\theta & 0 \\ 0 & -y_1 & a & b & 0 & \theta & 0 \\ 0 & y_6 & -y_2 & \omega & 0 & 0 & 0 \\ 0 & y_7 & 0 & -y_3 & 0 & 0 & 0 \\ 0 & 0 & 1 & q & -\mu & 0 & 0 \\ 0 & 0 & y_4 & y_5 & 0 & -y_8 & 0 \\ 0 & 0 & -y_9 & 0 & 0 & 0 & -r_1 \end{bmatrix}. \tag{3.10}$$

Its characteristic polynomial equation with eigenvalues (σ^*) is given by

$$(\sigma^* + \mu)^2(\sigma^* + r_1)\left(\sigma^{*4} + B_1\sigma^{*3} + B_2\sigma^{*2} + B_3\sigma^* + B_4\right) = 0, \tag{3.11}$$

where the $y_i s, i = 1, 2, 3, ..., 8$, B_4 and r_1 are as defined above. The other constants are expressed as follows:

$$\begin{aligned}
y_9 &= \frac{\alpha r_1}{h}, \quad y_{10} = \alpha - a, \quad k_1 = a\omega y_7 y_8, \quad k_2 = b y_2 y_7 y_8, \quad k_3 = \theta\omega y_4 y_7, \\
k_4 &= \theta y_2 y_5 y_7, \quad k_5 = \theta y_3 y_4 y_6 \quad k_6 = a y_3 y_6 y_8, \quad k_7 = y_2 y_8, \quad k_8 = y_1 y_3, \\
k_9 &= y_1 y_8, \quad k_{10} = y_2 y_2, \quad k_{11} = y_3 y_8, \quad k_{12} = y_1 y_2, \quad B_1 = y_1 + y_2 + y_3 + y_4, \\
B_2 &= (k_8 + k_{12})(1 - R_0) + k_7 + k_9 + k_{10} + \frac{1}{k_{11}}(k_1 + k_2 + k_3 + k_4 + k_5) \\
&+ \frac{1}{k_7}(k_1 + k_3 + k_4 + k_5 + k_6) \\
B_3 &= y_2 k_{11} + y_1(k_7 + k_{10} + k_{11})(1 - R_0) + \frac{1}{y_8}(k_3 + k_4 + k_5) \\
&+ \frac{1}{y_3}(k_1 + k_2 + k_3 + k_4 + k_6) + \frac{1}{y_2}(k_1 + k_3 + k_5 + k_6) \\
B_4 &= y_1 y_2 y_3 y_8(1 - R_0)
\end{aligned} \tag{3.12}$$

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 $R_0 > 1$.*

Proof. We note that the value of R_0 is strictly positive as expressed by (3.6), and from the definitions of the constants in (3.2) and (3.4), we observe that the coefficients of the quartic polynomial of (3.11) are all positive and non zero; so by the Descartes' rule of signs there are no positive real eigenvalues, this means there are 4 negative real eigenvalues, 2 negative real eigenvalues and 1 pair of complex conjugates with negative real parts, or 2 pairs of complex conjugate eigenvalues with negative real parts. We need to show that Routh Hurwitz stability conditions for a fourth order polynomial as stated in [10] can be expressed in our case as satisfying the condition

$$\psi = B_1 B_2 B_3 - ({B_3}^2 + {B_1}^2 B_4) > 0 \tag{3.13}$$

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

$$\begin{aligned}
\psi &= D_1(1 - R_0)^2 + D_2(1 - R_0) + k_1 E + k_2 F + k_3 G \\
&+ k_4 H + k_5 J + k_6 L + L_0.
\end{aligned} \tag{3.14}$$

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:

$$\begin{aligned}
B_5 &= y_2^2 + y_3^2 + y_8^2 + k_{11}, \ B_6 = y_1 y_2 y_3, \ B_7 = y_1 + y_2, \ B_8 = y_1 + y_3, \ B_9 = y_1 + y_8, \\
B_{10} &= y_2 + y_3, \ B_{11} = y_2^2 + y_3^2, \ B_{12} = B_{11} + y_8^2, \ B_{13} = k_7 + k_{10} + k_{11}, \\
B_{14} &= k_8 + k_{10} + k_{12} + B_{11}, \ B_{15} = B_1 + C_2, \ B_{16} = y_1 + y_2 + y_3, \ B_{17} = B_{10} + y_8, \\
B_{18} &= y_2 y_3 y_8, \ B_{19} = 2k_{10} C_7 y_8 + k_{10}(k_{10} + y_8^2) + k_{10} y_8(y_2 + C_1) + k_{11}(y_3 + C_2), \\
B_{20} &= C_5 + B_{10} k_5 y_1 \left[k_5(k_4 + k_{11}) + k_7\right], \ B_{21} = k_6 k_{12}(k_7 + k_{11} + B_{13}), \ B_{22} = y_1 y_2 y_3, \\
B_{23} &= k_7 + k_{10} + y_8^2, \ B_{24} = y_1 y_2 y_8, \ C_1 = y_2 + y_8, \ C_2 = y_3 + y_8, \ C_3 = B_7 + y_3,
\end{aligned}$$

$$
\begin{aligned}
C_4 &= 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,\\
C_6 &= B_{18}^3y_1(B_{13} + y_8 + y_8^2 + k_7 + k_{11}),\ C_7 = B_{18}^3\left[B_{13}C_2 + y_2^2(k_{10}y_2 + k_7^2)\right],\\
C_8 &= kk_{11}k_{12}\left[B_{10}(k_9 + 2k_{10} + 2k_{11}) + 2C_1k_7 + k_{11}(C_2 + y_3)\right],\\
C_9 &= B_{22}y_8\left[B_9k_7y_8 + 2B_{18}(B_{10} + y_3) + 2C_1k_{10}y_3 + 2k_7^2\right],\\
C_{10} &= B_{18}C_2(B_{10}y_2^2 + 2C_1B_{18}) + B_{18}k_{11}(B_{10}y_2^2 + B_{18}k_{11}),\ C_{11} = y_3 + 2y_8,\\
C_{12} &= k_3\left[B_{10}(2B_7k_9 + k_{12}y_3 + 2k_9y_3) + C_1y_2^2y_3 + k_{10}(B_{13} + y_3^2)\right],\\
C_{13} &= k_1\left[2k_{11} + k_{11}B_{16}(y_2 + B_{10}) + k_{12}\right],\\
C_{14} &= B_{18}^3\left[B_9k_9 + B_{22}(1 + y_2) + 2B_{24} + B_{18}(2 + C_2) + C_2y_2^2 + k_{10}y_3 + k_7\right],\\
D_1 &= B_{13}B_{14}k_{11}^2k_{12}^2,\ D_2 = k11k_{12}(B_{19}B_{20}B_{21},\\
E_1 &= 2B_{10}k_{11}(B_7k_5B_{16}k_6) + C_2k_5y_2\left[B_{10}y_1 + C_2(2y_2 + y_3)\right],\\
E_2 &= C_2k_5y_2\left[C_2(B_7 + B_{10}) + (2C_2y_2 + C_2y_3 + k_8 + k_{12})\right],\\
E_3 &= B_{10}k_3k_{12}(B_7k_9 + 2k_9y_3 + k_{12}y_3) + C_1y_2^2 + k_1k_{10}(B_{13} + y_3^2),\\
E_4 &= 2B_{10}B_{16}k_2k_7 + C_{10} + B_{10}k_{12}\left[k_9y_8(2 + k_8 + k_{11}) + 2B_{18}C_2y_8\right],\\
E_5 &= k_3\left[2B_7B_{10}k_9 + B_{10}y_3(2k_9 + k_{12}) + C_1y_2^2 + k_{10}(B_{13} + y_3^2)\right] + C_{11},\\
E_6 &= B_1B_{10}y_8\left[k_9(2 + k_8 + k_{11}) + 2B_{18}C_2\right] + C_{12} + C_{13},\\
F_1 &= C_{19} + B_{18}\left[2B_{18}(k_7 + k_{10} + y_2^2) + B_5y_8 + y_7\right],\\
F_2 &= k_3\left[2B_{10}B_{24} + B_1k_{10}y_2 + 2k_7y_2(B_{11} + k_{10})\right] + C_4k_2k_7y_8,\\
F_3 &= k_4\left[k_{12}(B_{13} + k_{10}) + y_2^2(k_7 + k_{10}) + B_{11}B_{18} + y_8 +_{10} k_7y_2 + B_{18}C_2y_3\right],\\
F_4 &= k_5\left[B_{13}k_{12} + k_{10}(k_7 + k_{11}) + k_{10}(k_{10} + B_6) + B_{23}y_2^2 + B_{10}B_{18}\right] + 2B_{16}B_{18}k_6,\\
G_1 &= B_{18}y_1(4B_{17}B_{18} + C_{11}k_7y_8 + B_{13}k_9 + k_7^2 + k_{10}^2),\\
G_2 &= B_{18}\left[B_{10}k_{10}^2 + C_{11}k_7^2 + 4B_{13}B_{18} + 3B11B_{18} + B_{18}(k_7 + 2y_3^2) + C_2k_{10}^2 + k_5k_{11}y_3\right],\\
G_3 &= k_4\left[3B_{10}B_{18} + C_2(2B_{10}k_{12} + B_{10}k_{11} + 3k_{10}y_2 + 2y_2^3)\right],\\
G_4 &= k_5\left[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\right],\\
G_5 &= k_6\left[3B_{22}C_2 + B_6(B_{10} + y_3) + 2C_{11}y_3 + C_2k_{10}y_3(B_{10} + 2k_5)\right],\\
G_6 &= k_3\left[B_{10}y_8 + B_{17}k_8y_2 + k_{10}(B_{10}C_1 + k_{11} + y_3^2) + k_{11}(B_{16} + y_{11})\right],\\
H_1 &= B_{18}k_{12}\left[B_7C_2y_8 + y_8(B_{17} + C_{11}y_8 + 3y_3) + B_{18}\right],\\
H_2 &= B_{18}y_2\left[3B_7B_{17} + k_{10}y_3(1 + C_{11}) + k_9^2(1 + y_8) + C_2k_{11}(C_2 + y_3)\right],\\
H_3 &= k_5\left[2B_{17}B_{22} + B_{11}k_9 + B_{10}(3B_{18} + 2k_{10}y_2 + k_7y_2 + y_3^2) + y_3^2(C_{11}y_3 + y_8^2)\right],\\
H_4 &= k_6y_3\left[2B_{16}k_7 + C_{11}k_8 + B_{10}(k_{10} + C_2k_{11}\right],\\
H_5 &= k_3\left[B_{22}C_4 + B_{18}(B_{17} + y_8) + C_2y_2^3 + B_{10}k_{10}y_3 + k_7(k_{11}) + k_{12})\right],\\
J_1 &= B_{18}y_1\left[B_6C_{11} + 3B_{17}B_{18} + k_{10}(k_{10} + k_{11})\right],\\
J_2 &= B_{18}\left[B_{17}k_{10}^2 + 3B_{18}(B_{13} + y_2^2) + B_{18}(B_{11} + y_2^2) + C_2k_{11}^2\right],\\
J_3 &= k_5k_6^2\left[2 + B_7(C_2k_{10}) + 2C_{11}y_2^3 + k_{11}(k_7 + y_3^2)\right],\\
J_4 &= B_{18}\left[B_{17}k_{10}^2 + 3B_{18}(B_{14} + y_3^2) + B_{18}(C_2k_{11}^2 + 2y_2^2)\right],\\
J_5 &= k_5\left[B_{18}(B_{17} + y_2) + B_{16}B_{22} + y_3(B_6 + B_{10}y_2^2 + k_{10} + k_{11})\right],\\
E &= E_1 + E_2 + E_3 + E_4 + E_5 + E_6,\ F = F_1 + F_2 + F_3 + F_4,\\
G &= G_1 + G_2 + G_3 + G_4 + G_5 + G_6,\ H = H_1 + H_2 + H_3 + H_4 + H_5,\\
J &= J_1 + J_2 + J_3 + J_4 + J_5,\\
L &= B_{18}k_{11}\left[y_1(2B_{13} + k_9 + y_8^2) + y_2(2B_{13} + y_3^2 + y_8^2) + C_2k_{11} + C_4k_6k_{11}y_3\right],
\end{aligned}
$$

$$L_0 = C_6 + C_7 + C_8 + C_{14} + y_2^2(B_{10} + C_1) + k_8.$$

Since D_1, D_2, E, F, G, H, J, L, L_0, k_i, $i = 1, 2, ..., 6 > 0$, it follows that $\psi > 0$, if $R_0 < 1$. Thus, the disease-free equilibrium is locally asymptotically stable if $R_0 < 1$. The coefficient B_1 is positive and we observe that if $R_0 > 1$, B_4 is negative, wherein the signs of B_2 and B_3 cannot immediately be ascertained. We note that there is only one sign change If B_2 is positive and B_3 is negative, or B_2 and B_3 are both negative or positive. But there are three sign changes if B_2 is negative and B_3 is positive. Therefore the sequence of coefficients, 1, B_1, B_2, B_3, B_4 has only one sign change in the worst scenario. By using Descartes' rule of sign there must exist at least one positive real eigenvalue, we conclude that the disease free state is unstable if $R_0 > 1$.

When $R_0 = 1$, (3.11) has one zero eigenvalue, which shows that $R_0 = 1$ is a bifurcation surface in $(\beta_1, \beta_2, \beta_3, \gamma_1, \gamma_2, \sigma_1, \sigma_2, \theta_1, \theta_2, \lambda_1, \mu_1, \omega_1, m)$ parameter space.

3.4 Numerical solution

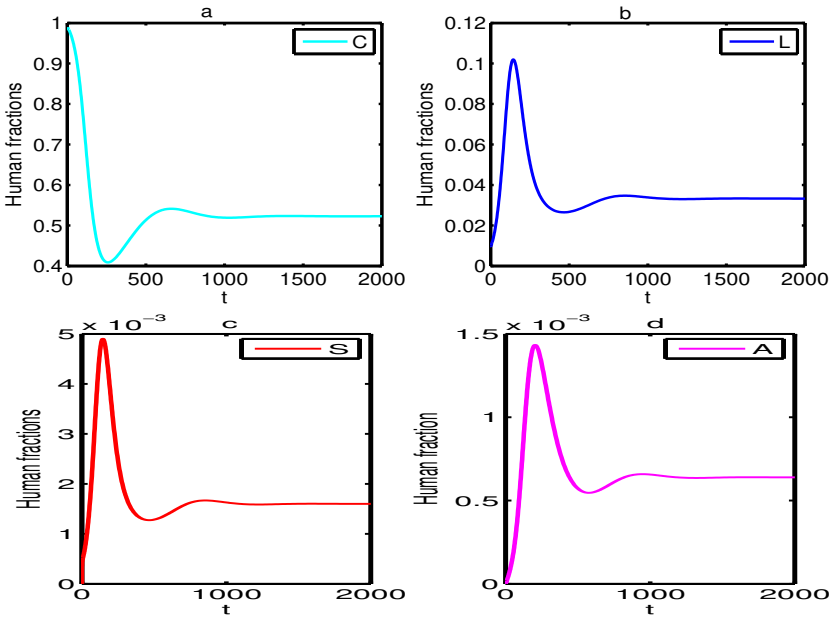

Fig. 2. Results showing the effect of the initial infected humans on evolution of infection where $t = 1$, represents approximately 10 days in real time. The initial conditions used are $C = 1$, $L = 0.01$, $S = 0$, $A = 0$, $R = 0$, $V = 0$, $N = 1$ and the parameter values are given above

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; $\lambda = 0.00439$, $a = 1.57$, $b = 1.26$, $\mu = 0.00362$, $\theta = 0.000137$, $\alpha = 0.00463$, $\gamma = 0.0485$, $h = 0.000767$, $\omega = 0.000357$, $d = 0.00403$, $e = 0.0101$, $g = 0.101$, $\eta = 0.000125$, $q = 0.00865$, $m = 0.005$. 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.

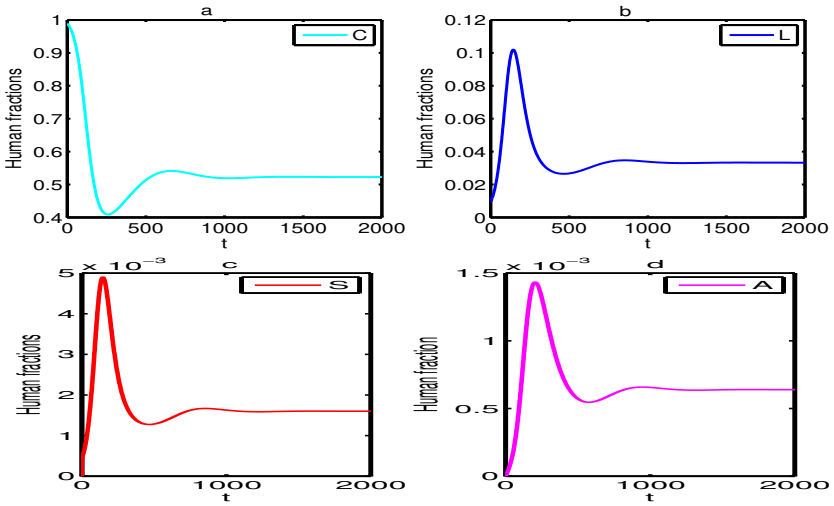

Fig. 3. Results showing the effect of the initial infected humans on evolution of infection where $t = 1$, represents approximately 10 days in real time. The initial conditions used are $C = 1$, $L = 0.01$, $S = 0$, $A = 0$, $R = 0$, $V = 0$, $N = 1$ and the parameter values are given above

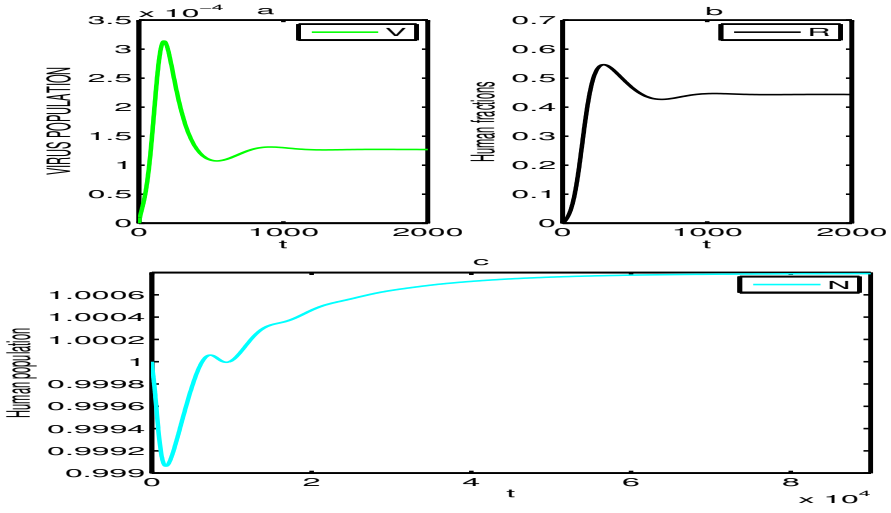

Fig. 4. Results showing the human population recovered humans and number of surface viruses. The values used for the simulations are the same as those in Fig. 2. except that we have used $\lambda = 0.00439$, $a = 1.57$ and $h = 0.000767$ in Fig. 4c.

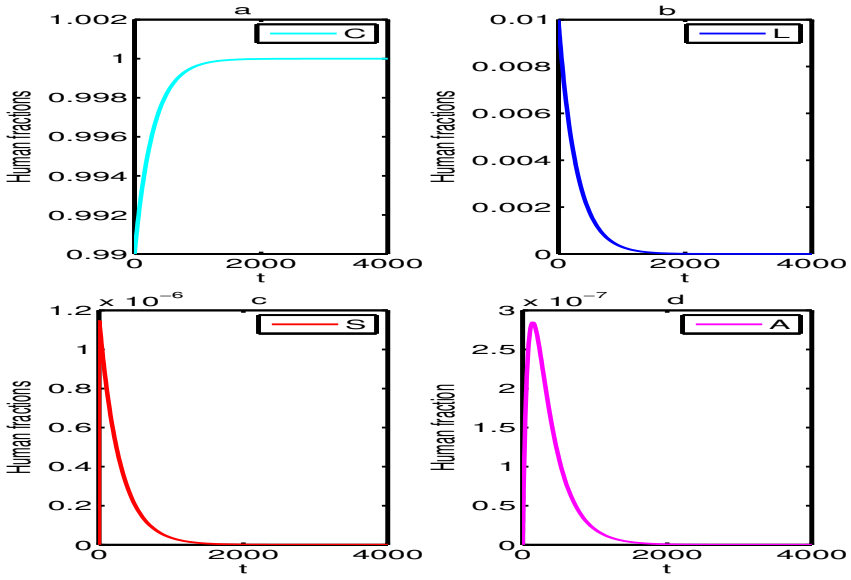

Fig. 5. Results showing the disease free state when $R_0 < 1$. The parameter values used to obtain these results are $\lambda = 0.00439$, $a = 1.57$, $b = 1.26$, $\mu = 0.00362$, $\theta = 0.000137$, $\alpha = 0.00463$, $\gamma = 0.000118$, $h = 0.000767$, $\omega = 0.000357$, $d = 0.00403$, $e = 0.0101$, $g = 0.101$, $\eta = 0.000125$, $q = 0.00865$, $m = 0.005$.

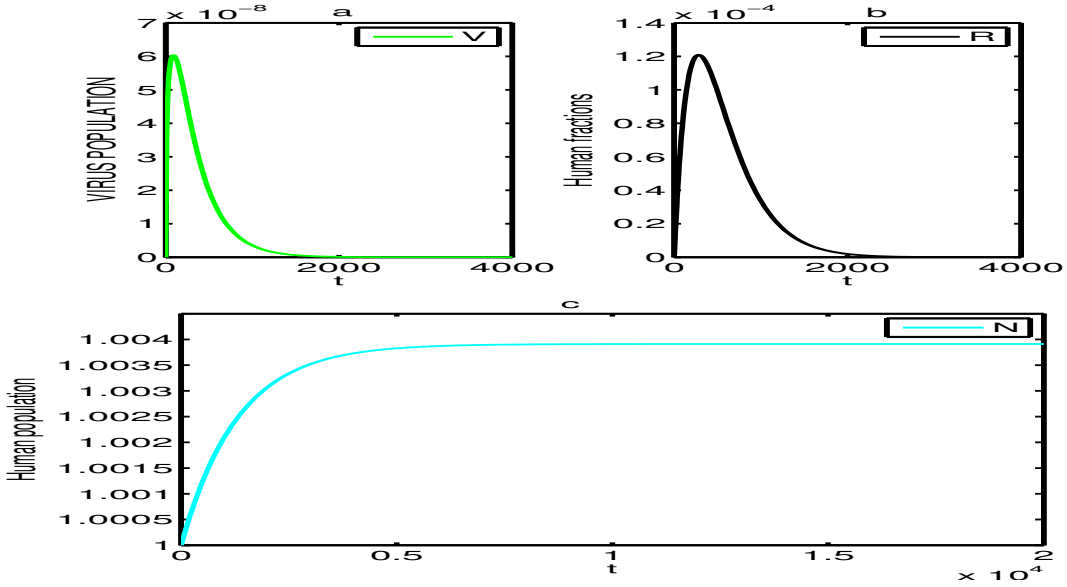

Fig. 6. Results showing the disease free state precisely,R_0=0.9982 for viruses of surfaces, recovered humans and the total human population. Parameter values are the same as those in Fig. 5

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 $R_0 < 1$.

3.5 Discussion

Our model describes the transmission of COVID-19 in an entirely susceptible human population due to the introduction of an index case. Although accurate parameter values for the pandemic are yet to be ascertained but the available parameter values we have used in our model show a prospective endemic nature of the disease. This is supported by the value of R_0 given as 2.1 using (3.6), which has earlier been estimated in [5] as 2.4829. The numerical simulations show characteristic features of long term solutions that need to be carefully studied using other forms of analysis including asymptotic due to the inherent large number of small parameters. This will draw out key time-scales in which events occur during the evolution of the disease. This could be a possible future work in understanding the transmission of the disease.

We note that the nondimensional parameters, γ, a and b are very important parameters with γ being the most effective in the control of the disease as may be deduced from the basic reproduction number. An increase or decrease in γ increases or decreases R_0 reliably. This is demonstrated in 5 and 6 where we have used γ to drive the disease into extinction. Since $\gamma = \frac{\gamma_1}{\theta_1}$ an effective strategy to significantly reduce γ is to reduce γ_1 and increase θ_1. That is, reducing the rate at which infected humans become infectious and facilitating the recovery rate of symptomatic humans. Increasing the value of b to a large extent will increase R_0 but reducing b to zero may not significantly reduce R_0 noting that asymptomatically infected individuals are much less likely to transmit the virus than those who develop symptoms [11, 12]. Similarly, $\lim_{a\to 0} R_0$ depends largely on other parameters and reducing a alone may not have significant impact on disease control. However, a combined downsize of a and d reduces R_0 faster. An implication of this is that prevention of exposed humans from infectiousness should be combined with efforts to reduce contact with viruses on surfaces.

Our results show a direct relationship between R_0 and the transition rate from exposed state to infectiousness as shown in the work of [1]. However, the disease related death rate has no relevance in our model as can be deduced from the basic reproduction number, R_0. Although, deases related death rate appears to play a disease control role in [1], it is quite misleading to comprehend that an increase in disease related death would reduce the basic reproduction number. However, such result may be mathematically tractable, it may lack some biological and epidemiological relevance in that we do not need to increase death of humans in order to control a disease.

4 Conclusions

In this work we propose a simple mathematical model on the transmission of COVID-19 that originally surfaced in Wuhan, China and has drastically affected most small and even the greatest economies of the world. The model focuses on the transmission dynamics of the disease in a totally susceptible population due to the introduction of an index case. The results show some long term damped oscillatory behaviour that do not seem to end soon. An analysis of the basic reproduction number shows that a control measure targeting the management of exposed humans preventing them from being infectious to susceptible humans and surfaces could cause effective hindrance to disease transmission. Testing and early detection of infectious cases as well as the quarantine

method including the use masks and social distancing are measures that could possibly reduce disease transmission. COVID-19 is relatively new and a lot needs to be done in terms of estimation of parameters that drive the dynamics of the disease. The inherent long term solutions in the numerical solutions need to be properly studied.

Due to the mode of transmission and individual behavioural pattern caused by various social, cultural and religious factors, the sure time to eradication of the disease is the manufacturing of an appropriate vaccine followed by proper sensitisation of the people on the need to take the vaccine. The World Health Organization may not have been quite proactive even as we want to appreciate its effort in maintaining Global health. The world would have lost far less if this COVID-19 was contained in China. We suggest that in case of any epidemic originating in any country, a condition be imposed by the WHO such that only entry but no exit is allowed in the country while medical experts and aids from other countries are deployed to contain the epidemic in that country.

Competing Interests

Authors have declared that no competing interests exist.

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