Dynamics of Covid-19 Transmission using Mathematical model

Md. Abu Salek[1] and Md. Shahidul Islam[2]

[1]Departmental of Mathematics, Bhawal Bodre Alam Government College, Gazipur, Bangladesh
[2]Department of Mathematics, University of Dhaka, Dhaka 1000, Bangladesh
[1]E-mail: salekphd@gmail.com, [2]mshahidul11@yahoo.com

[1]Corresponding Author

Abstract:

We broaden a dynamical model to recognize the underlying dynamics of COVID-19 contamination at the population in the recent world. As reported by the World Health Organization, a novel coronavirus (2019-nCoV) was identified as the causative virus of Wuhan pneumonia of unknown aetiology by Chinese authorities on 7 January 2020. The virus was named as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) by International Committee on Taxonomy of Viruses on 11 February 2020. This study aimed to develop a mathematical model for calculating the transmissibility of the virus. The model, which integrates the remedy of people, the infections of latent and medicinal people, is rigorously analyzed to gather insight into its dynamical features. The phenomenon resulted because of the exogenous infection of COVID-19 disease. The mathematical assessment is famous that the version exhibits even as COVID-19 treatment stays of infected elegance. It's far demonstrated that, in the absence of preparation, the version has a sickness-loose equilibrium (DEF) this is globally asymptotically stable, and the associated reproduction threshold is less than fraternal cases. Similarly, the model has a unique endemic equilibrium (EEP), for a special case, whenever the related duplicate threshold amount exceeds solidarity. For a unique case, the EEP is operating the use of the important manifold theorem of Castillo-Chavez.

Keywords: COVID-19 Model, SIR Model, equilibria, stability, Castillo-Chavez theorem, disease dynamics, disease-endemic equilibrium.

I. Introduction

A differential equation which describes some physical process is often referred to as a mathematical version of the method. Again, a differential equation is a mathematical equation that quotes a few features of one or extra variables with its derivatives, differential equation arises every time a deterministic relation involving some constantly various portions and their rates of alternate in space and time. Those equations occupy the area at the centre level of

both natural and carried out mathematics. Assessing the severity of COVID-19 is crucial to determine the appropriateness of mitigation strategies and to enable planning for health-care needs as epidemics unfold. However, crude case fatality ratios obtained by dividing the number of deaths by the number of cases can be misleading. First, there can be a period of 2–3 weeks between a person developing symptoms, the case subsequently being detected and reported, and observation of the final clinical outcome. During a growing epidemic, the final clinical outcome of most of the reported cases is typically unknown. Simply dividing the cumulative reported number of deaths by the cumulative number of reported cases will, therefore, underestimate the true case fatality ratio early in an epidemic. This effect was observed in past epidemics of respiratory pathogens, including severe acute respiratory syndrome (SARS) and H1N1 influenza, and as such is widely recognized. Thus, many of the estimates of the case fatality ratio that have been obtained to date for COVID-19 correct for this effect. Additionally, however, during the exponential growth phase of an epidemic, the observed time lags between the onset of symptoms and outcome (recovery or death) are censored, and naive estimates of the observed times from symptom onset to outcome provide biased estimates of the actual distributions. Ignoring this effect tends to bias the estimated case fatality ratio downwards during the early growth phase of an epidemic. As a public fitness and medical laboratory scientist, I have experienced several “new” and reemerging microbial outbreaks spanning three a long time. Over the approaching days, weeks, months or even years, we will study more about the biology of this new SARS-CoV-2 virus, inclusive of higher estimates of case fatality, duplicate prices, and other crucial information. Extra instances are possibly to be diagnosed within the coming days, such as extra instances within the United States. Given what has taken place formerly with MERS-CoV and SARS-CoV, it’s probably to spread from character-to-individual, which includes inside the United States of America. Searching for out legit assets for new statistics and comply with CDC recommendations and recommended health precautions (e.g. hand washing, up to date vaccinations, and tour alerts this newsletter became compiled from a spread of resources by the author, with the majority of facts from the CDC and WHO (and their related assets). That is a rising, swiftly changing situation, and CDC will provide updated statistics as it will become available, in addition to updated steerage. Coronaviruses are anywhere. They are the second main reason for the common bloodless (after rhinoviruses) and until current a long time, rarely prompted any ailment extra serious than a common cold in humans. The primary coronavirus became isolated in 1937. A few reason illnesses in human beings and others circulate among different animals, which includes camels, cats and bats. When you consider that its discovery, associated coronaviruses have been found to infect livestock, pigs, horses, turkeys, cats, dogs, rats, and mice. The first human coronavirus turned into cultured inside the 1960s from nasal cavities of humans with the common cold. The four important classes of coronavirus are recognized through the Greek messages, beta, delta and gamma. Most effective alpha and beta coronaviruses are recognized to contaminate human beings. These viruses spread thru the air and are chargeable for approximately 10-30 in keeping with cent of colds international. Lengthy acknowledged to cause higher respiratory infections, coronaviruses were not felt to seriously reason pneumonia till noticeably recently. Seven human coronaviruses (HCoVs) have now been identified: HCoV-229E, HCV-OC43, HCV-

NL63, HCoV-HKU1, SARS-CoV (which reasons severe acute respiratory syndrome), MERS-CoV (centre East breathing syndrome), and now SARS-CoV-2. All but 2019-nCoV look like installed human pathogens with worldwide distribution, causing the top and decrease respiration tract infections, mainly in children. Usually, HCoV infection follows a seasonal pattern much like that of influenza, even though Hong Kong researchers determined that HCoV-NL63 infections especially occurred in early summer and autumn. At the same time as there aren't many coronaviruses that purpose lower respiration disease in people, they can have critical results for those they infect. Coronaviruses are zoonotic, meaning they may be transmitted among animals and people, but maximum infect only their animal host. Rarely, animal coronaviruses can evolve to contaminate and unfold among people. This was the case with intense Acute respiration Syndrome Coronavirus (SARS-CoV) and the centre of East respiration Syndrome Coronavirus (MERS-CoV). SARS killed nearly 10% of the 8,096 folks who fell ill in 29 international locations. A total of 774 humans died, consistent with the sector fitness corporation. MERS is even extra deadly, claiming extra than 30% of humans it infects. Unlike SARS, outbreaks of MERS are nevertheless taking place, NIAID Director Anthony Fauci says. When you consider that 2012, MERS has triggered 2,494 showed instances in 27 nations and killed 858 humans. SARS-CoV and MERS-CoV generally spread between folks who have been in close touch, which resulted in many fatalities of healthcare workers. Those viruses unfold just like the flu virus, and past MERS-CoV and SARS-CoV outbreaks were complicated, requiring comprehensive public fitness responses. The rapid public fitness responses to those outbreaks have been able to fast determine that the SARS-CoV genome series isn't the same as all other recognized coronaviruses and that it turned into first transmitted from civet cats to people, even though bats have been determined to be the reservoir. Scientists also speedy determined that MERS-CoV spread from dromedary camels to human beings. Consequently, it's important to in short talk coronavirus genetics. On 31 December 2019, the world fitness business enterprise (WHO) China use workplace became informed of instances of pneumonia of unknown aetiology (unknown purpose) detected in Wuhan city, Hubei Province of China, and WHO pronounced that a unique coronavirus (2019-nCoV), which turned into named as excessive acute respiratory syndrome coronavirus 2 (SARS-CoV-2) by international Committee on Taxonomy of Viruses on 11 February 2020, became diagnosed as the causative virus through Chinese authorities on 7 January [1]. Its miles stated that the virus is probably batted foundation [2], and the transmission of the virus may be related to a seafood marketplace (Huanan Seafood Wholesale marketplace) exposure [3, 4]. The genetic capabilities and a few scientific findings of the infection were reported recently [4–6]. Potentials for worldwide unfold thru commercial air journey has been assessed [7]. Public fitness issues are being paid globally on what number of human beings are infected and suspected. Consequently, it's far urgent to expand a mathematical version to estimate the transmissibility and dynamic of the transmission of the virus. There were numerous kinds of research specializing in mathematical modelling [3, 8]. These researches centred on calculating the fundamental replica wide variety (R0) using the serial durations and intrinsic growth charge [3, 9, 10], or using everyday differential equations and Markov Chain Monte Carlo techniques [8]. However, the bat beginning, and the transmission path shape the seafood market to humans have been no longer taken into consideration inside the

posted fashions. Through calculating the posted statistics, our model showed that the transmissibility of SARS-CoV-2 might be higher than MERS within the Middle East nations, just like SARS, however, decrease than MERS in the Republic of Korea. Since the goal of this observes changed into to offer a mathematical model for calculating the transmissibility of SARS-CoV-2, the R_0 changed into envisioned based totally on restrained statistics published in the literature. More information has been needed to estimate the transmissibility correctly. In this study, we developed a Bats-Hosts-Reservoir-People transmission network model for simulating the potential transmission from the infection source (probably be bats) to the human infection. Since the Bats-Hosts-Reservoir network was hard to explore clearly and public concerns were focusing on the transmission from Huanan Seafood Wholesale Market (reservoir) to people, we simplified the model as Reservoir-People (RP) transmission network model. The next-generation matrix approach was adopted to calculate the basic reproduction number (R_0) from the RP model to assess the transmissibility of the SARS-CoV-2.

In this paper, we have formulated the transmission dynamics of COVID-19 in the presence of treatment and investigated its role in the dynamics of the disease.

II. Formulation of model

Coronaviruses, so named due to the fact they seem like halos (called coronas) when regarded below the electron microscope, are a massive family of RNA viruses. The standard conventional coronavirus genome is an unmarried strand of RNA, 32 kilobases lengthy, and is the largest acknowledged RNA virus genome. Coronaviruses have the best-known frequency of recombination of any effective-strand RNA virus, promiscuously combining genetic statistics from different assets when a host is inflamed with multiple coronaviruses. In different words, these viruses mutate and exchange at a high price that could create havoc for each diagnostic detection as well as therapy (and vaccine) regimens. Coronaviruses have an uncommon replication manner, which entails a 2-step replication mechanism. Many RNA virus genomes comprise a single open studying frame (ORF) that is then translated as a single polyprotein this is then catalytically cleaved into smaller practical viral proteins, but coronaviruses can include up to 10 separate ORFs. Maximum ribosomes translate the largest this kind of ORFs, referred to as replicas, which alone is two times the scale of many different RNA viral genomes. The replicase gene encodes a series of enzymes that use the rest of the genome as a template to provide a hard and fast of smaller, overlapping messenger RNA molecules, which are then translated into the structural proteins the building blocks of latest viral debris.

For an in-depth, complete description of coronavirus replication and pathogenesis. Following the classical assumptions, we formulate a deterministic, compact mental, mathematical model to describe the transmission dynamics of measles. The population is homogeneously mixing

and reflecting the demography of a typical developing country, as it experiments an exponentially increasing dynamic. In other to describe the model equations, the total population (*N*) is divided into three classes: Susceptible (S), exposed (E), quarantined (Q), infected (I) and recovered (R). Here we shall detail the transitions among these five classes as depicted in Figure 1.

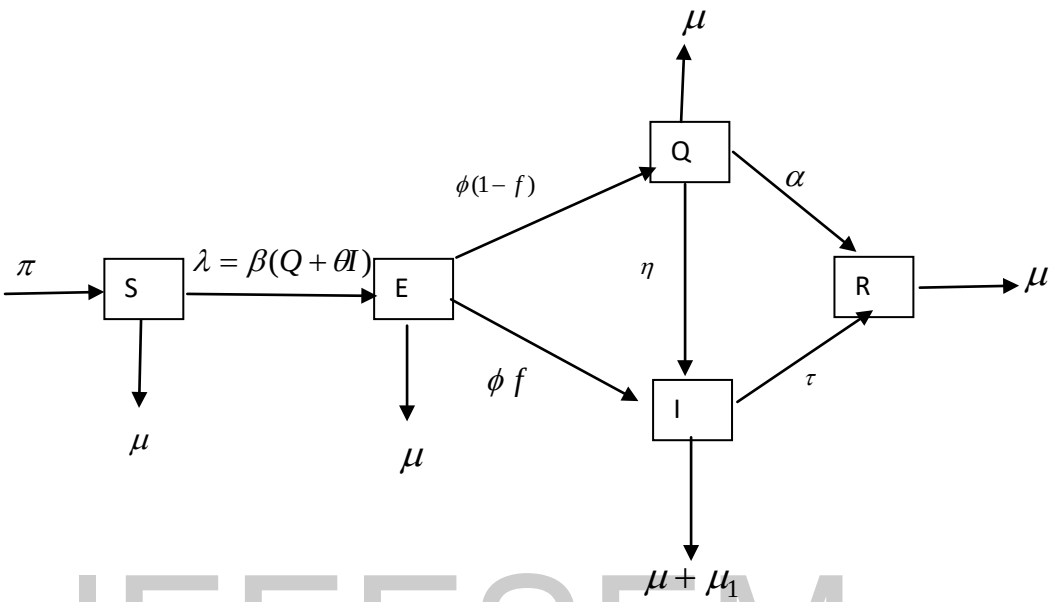


Figure 1: Model diagram of COVID – 19.

The class S of susceptible is increased by birth or immigration at a rate π. It is decreased by infection following contact with quarantined and infected individuals at a rate β, and diminished by force of infection, λ and natural death at a rate μ. The class E is decreased by the fraction of newly infected individuals at a rate ϕ, and natural death at a rate μ. The class Q of quarantined individuals is generated by the fraction of exposed individuals and decreased by the progression of infected individuals, at a rate η, by the recovery rate α of quarantined individuals and natural death rate. The class of infected individuals is decreased by the treatment rate τ and natural death rate and disease-induced death rate μ_1.

The recovered individuals are increased by the recovered individuals of quarantined and the infected individuals who are getting treatment and diminished by the natural death rate. The model assumes that recovered individuals become permanently immune to the disease. This generates a class *R* of individuals who have complete protection against the disease. The transmissions between model classes can now be expressed by the following system of first-order differential equations:

$$\frac{dS}{dt} = \pi - \lambda S - \mu S$$
$$\frac{dE}{dt} = \lambda S - (\phi + \mu) E$$
$$\frac{dQ}{dt} = \phi(1 - f)E - (\eta + \alpha + \mu)Q \qquad (1)$$
$$\frac{dI}{dt} = \phi f E + \eta Q - (\tau + \mu + \mu_1) I$$
$$\frac{dR}{dt} = \alpha Q + \tau I - \mu R$$

where $\lambda = \beta(Q + \theta I)$ is the force of infection.

Table 1: Description of Variables and parameters of the COVID-19 Model

Variables	Description
S	Susceptible individuals
E	Latently infected (exposed) individuals
Q	Quarantined individuals
I	Infected individuals
R	Recovered individuals
π	Recruitment rate into the population
μ	Natural death rate
μ_1	The death rate due to infection
β	Probability rate of transmission
ϕ	The rate at which exposed individuals become infected
f	Fractions of exposed individuals become infected
α	The recovery rate for quarantined individuals
η	Progression rate of quarantined individuals
τ	Treatment rate of infected individuals
θ	infectiousness of infected individuals, where $\theta > 1$

Table 2: Data summary of Parameters of the COVID-19 Model

Parameters	**Values**
π	2000 [collected]
μ	0.02 [collected]
μ_1	0.1 [collected]
η	0.08 (assumed)
τ	0.85 (assumed)
α	0.9 (assumed)

f	0.5(assumed)
ϕ	0.7 [collected]

Since the model monitors the human population, all the associated parameters and state variables are non-negative $t \geq 0$. It is easy to show that the state variables of the model remain non-negative for all non-negative initial conditions. Consider the biologically feasible region.

$$\Omega = \left\{ (S, E, Q, I, R) \in R_+^5 : N \leq \frac{\pi}{\mu} \right\}$$

From the model equation (1) it will be shown that the region is positive. The total population of individuals is given by

$$N = S + E + Q + I + R$$

III. Analysis of model

Disease Free Equilibrium (DFE): The equilibrium points of the system can be obtained by equating the rate of changes of zero, given by ε_0,

$$\therefore \ \varepsilon_0 = (\frac{\pi}{\mu}, 0, 0, 0, 0) \qquad (2)$$

The stability of the DFE will be analyzed using the next generation method [13]. The non-negative matrix F (of the new infection terms) and the non-singular M-matrix V (of the remaining transfer terms) are given, respectively by,

$$F(\varepsilon_0) = \begin{pmatrix} 0 & \frac{\beta\pi}{\mu} & \frac{\beta\theta\pi}{\mu} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

$$\text{and,} \quad V = \begin{pmatrix} (\phi + \mu) & 0 & 0 \\ -(1-f)\phi & \eta + \alpha + \mu & 0 \\ -f\phi & -\eta & (\tau + \mu + \mu_1) \end{pmatrix}$$

The associated reproduction number, denoted by R_0, is given by $R_0 = \rho(FV^{-1})$, where ρ denotes the spectral radius (dominant eigenvalue in magnitude) of the next generation matrix FV^{-1}. It follows that

$$FV^{-1} = \begin{bmatrix} -\frac{\beta\pi\phi(f-1)}{\mu(\mu+\phi)(\alpha+\eta+\mu)} + \frac{\beta\theta\pi\phi(\alpha f + f\mu + \eta)}{\mu(\mu+\phi)(\alpha+\eta+\mu)(\mu+\mu_1+\tau)} & \frac{\beta\pi}{\mu(\alpha+\eta+\mu)} + \frac{\beta\theta\pi\eta}{\mu(\alpha+\eta+\mu)(\mu+\mu_1+\tau)} & \frac{\beta\theta\pi}{\mu(\mu+\mu_1+\tau)} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

$$\therefore R_0 = \frac{\beta\pi\phi\{\theta(\alpha f + f\mu + \eta) + (1-f)k_3\}}{\mu k_1 k_2 k_3} \tag{3}$$

where, $k_1 = \phi + \mu$, $k_2 = \eta + \alpha + \mu$, $k_3 = \tau + \mu + \mu_1$.

Lemma: The Disease-free equilibrium ε_0 of the model (1) is locally asymptotically stable (LAS) if $R_0 < 1$ and unstable if $R_0 > 1$.

The threshold quantity R_0 is the reproduction number for the model. The epidemiological implication of Lemma 1 is that COVID-19 spread can be effectively controlled in the community (when $R_0 < 1$) if the initial sizes of the populations of the model are in the basin of attraction of the disease-free equilibrium ε_0 [14].

IV. Global stability of DFE of the COVID-19 model

Let,

$$\frac{dX}{dt} = H(X, Z)$$
$$\frac{dZ}{dt} = G(X, Z),\ G(X, 0) = 0. \tag{4}$$

Where,

$X = (X, 0)$ and $Z = (E, Q, I)$ with the components of $X \in R^1$ denoting the uninfected population and the components of $Z \in R^3$ denoting the infected population [15].

The disease-free equilibrium is now denoted as,

$$\varepsilon_0 = (X^*, 0, 0), \quad X^* = \left\{\frac{\Lambda}{\mu}, 0, 0\right\}$$

Now, $\frac{dX}{dt} = H(X, 0)$, X^* is globally asymptotically stable (GAS)

$$G(X, Z) = Pz - \hat{G}(X, Z), \hat{G}(X, Z) \geq 0 \text{ for } (X, Z) \in \Omega. \tag{5}$$

Where $P = D_Z G\left(X^*,0\right)$ is an M-matrix (the off-diagonal elements of P are non-negative) and Ω is the region where the model makes biological sense. If the system (4) satisfies the conditions of (5) then the theorem below holds.

Theorem 1: The fixed point $\varepsilon_0\left(X^*,0\right)$ is a globally asymptotically stable equilibrium of system (4) provided that $R_0 < 1$ and the assumptions in (5) are satisfied.

Proof:

From the system (1) we have,

$$H\left(X,0\right) = \begin{pmatrix} \pi - \mu S \\ 0 \\ 0 \\ 0 \\ -\mu R \end{pmatrix}$$

$$\begin{aligned} G\left(X,Z\right) &= P\left(Z\right) - \hat{G}\left(X,Z\right) \\ \Rightarrow \hat{G}\left(X,Z\right) &= P\left(Z\right) - G\left(X,Z\right) \end{aligned} \qquad (6)$$

Where,

$$P\left(Z\right) = \begin{pmatrix} -\left(\phi+\mu\right) & 0 & 0 \\ \left(1-f\right)\phi & -\left(\eta+\alpha+\mu\right) & 0 \\ f\phi & \eta & -\left(\tau+\mu+\mu_1\right) \end{pmatrix}$$

and $$G\left(X,Z\right) = \begin{pmatrix} \beta\left(Q+\theta I\right)S - \phi E - \mu E \\ \phi\left(1-f\right)E - \eta Q - \alpha Q - \mu Q \\ \phi f E + \eta Q - \tau I - \left(\mu+\mu_1\right)I \end{pmatrix}$$

Putting values $P\left(Z\right)$ and $G\left(X,Z\right)$ in (6) no equation and we obtain,

$$\hat{G}\left(X,Z\right) = \begin{pmatrix} \beta\left(Q+\theta I\right)S \\ 0 \\ 0 \end{pmatrix} \geq 0 \qquad (7)$$

It is clear that $\hat{G}\left(X,Z\right) = 0$ for all $\left(X,Z\right) \in \Omega$ we also note that matrix P is an M-matrix since it's off-diagonal elements are non-negative [16].

V. Endemic Equilibrium Point (EEP) of the COVID-19 model

Let, $\varepsilon_1 = \left(S^*, E^*, Q^*, I^*, R^*\right)$ represents any arbitrary endemic equilibrium of the model. Solving the equations of the system (1), the model has the following endemic equilibrium points (EEP),

$$S^* = \frac{\pi}{\lambda+\mu},\ E^* = \frac{\lambda\pi}{(\mu+\phi)(\lambda+\mu)},\ Q^* = \frac{\lambda\pi\phi(1-f)}{(\mu+\phi)(\lambda+\mu)(\alpha+\eta+\mu)},$$

$$I^* = \frac{\lambda\pi\phi(\alpha f + f\mu + \eta)}{(\mu+\phi)(\mu+\mu_1+\tau)(\lambda+\mu)(\alpha+\eta+\mu)},\ R^* = \frac{\pi\lambda\phi\{\tau(f\mu+\alpha+\eta)+\alpha(1-f)(\mu+\mu 1)\}}{\mu(\mu+\phi)(\mu+\mu_1+\tau)(\lambda+\mu)(\alpha+\eta+\mu)}$$

Existence of Endemic Equilibrium Point (EEP):

In this section, the possible existence and stability of endemic (positive) equilibria of the model (i.e. equilibria where at least one of the infected of the model is non-zero) will be considered.

Let, $\varepsilon_1 = \left(S^*, E^*, Q^*, I^*, R^*\right)$ represents any arbitrary endemic equilibrium of the model (1). Solving the equations of the system at a steady stage gives, the following EEP

$$\varepsilon_1 = \left(\frac{\pi}{\lambda+\mu}, \frac{\lambda\pi}{(\mu+\phi)(\lambda+\mu)}, \frac{\lambda\pi\phi(1-f)}{(\mu+\phi)(\lambda+\mu)(\alpha+\eta+\mu)}, \frac{\lambda\pi\phi(\alpha f + f\mu+\eta)}{(\mu+\phi)(\mu+\mu_1+\tau)(\lambda+\mu)(\alpha+\eta+\mu)}, \frac{\pi\lambda\phi\{\tau(f\mu+\alpha+\eta)+\alpha(1-f)(\mu+\mu_1)\}}{\mu(\mu+\phi)(\mu+\mu_1+\tau)(\lambda+\mu)(\alpha+\eta+\mu)}\right) \tag{8}$$

The expression for λ, at the endemic steady state, is given by

$$\lambda = \beta\left(Q^* + \theta I^*\right) \tag{9}$$

For mathematical convenience, the expression in (9) is re-written,

$$\lambda = \frac{\beta\pi\lambda\phi\{\theta(\alpha f + f\mu+\eta)+(1-f)k_3\}\mu}{(\lambda+\mu)k_1k_2k_3\mu}$$

And we get,

$$\lambda^2 - \lambda\mu(R_0 - 1) = 0$$
$$\Rightarrow\ \lambda = \mu(R_0 - 1) > 0 \tag{10}$$

The components of the unique endemic equilibrium ε_1 can be obtained by substituting the unique value of λ, given into the expression in (9). Thus, the following has been established.

Lemma: The model has a unique endemic equilibrium, given by ε_1, whenever $R_0 > 1,\ \lambda > 0$.

VI. Local stability of EEP

The unique EEP is locally asymptomatically stable if the following theorem hold.

Theorem 2: (Castillo-Chavez and Song)
Consider the following general system of ordinary differential equations with a parameter φ.

$$\frac{dx}{dt} = f(x,\varphi),\ f : \Re^n \times \Re \to \Re \text{ and } f \in C\left(\Re^n \times \Re\right)$$

where 0 is an equilibrium of the system (i.e. $f(0,\varphi) = 0$ for all φ and assume

A1: $A = D_x f(0,0) = \left(\frac{\partial f_i}{\partial x_j}(0,0)\right)$ is the linearization matrix of the system (1) around the equilibrium 0 with φ evaluated at 0. Zero is a simple eigenvalue of A and other eigenvalues to A have negative real parts.

A2: Matrix A has a right eigenvector w and a left eigenvector v (each corresponding to the zero eigenvalues).

Let, f_k be the k component of f and

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0)$$

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \varphi}(0,0)$$

The local dynamics of the system around the equilibrium point 0 is determined by the sings of a and b. Particularly, $a < 0, b > 0$ the system does not show backward bifurcation $R_0 = 1$. In these case, $0 < \varphi << 1, 0$ becomes unstable and there exists a positive locally asymptotically stable equilibrium [17].

Proof: The proof of the theorem, which is based on the use of centre manifold theory. The backward bifurcation phenomenon of the model is numerically illustrated below. It is convenient to let $S = x_1, E = x_2, Q = x_3, I = x_4, R = x_5$, so that $N = x_1 + x_2 + x_3 + x_4 + x_5$. Further, by introducing the vector notation $X = (x_1, x_2, x_3, x_4, x_5)^T$, the model can be written in the form,

$\frac{dx}{dt} = F(x)$, where $F = (f_1, f_2, f_3, f_4, f_5)^T$, as follows

$$\begin{aligned}
\frac{dx_1}{dt} &= f_1 = \pi - \beta(x_3 + \theta x_4)x_1 - \mu x_1 \\
\frac{dx_2}{dt} &= f_2 = \beta(x_3 + \theta x_4)x_1 - \phi x_2 - \mu x_2 \\
\frac{dx_3}{dt} &= f_3 = \phi(1-f)x_2 - \eta x_3 - \alpha x_3 - \mu x_3 \\
\frac{dx_4}{dt} &= f_4 = \phi f x_2 + \eta x_3 - \tau x_4 - \mu x_4 - \mu_1 x_4 \\
\frac{dx_5}{dt} &= f_5 = \alpha x_3 + \tau x_4 - \mu x_5
\end{aligned} \qquad (11)$$

The Jacobian of the system at the DFE (ε_0) is given by,

$$J(\varepsilon_0) := \begin{bmatrix} -\mu_1 & 0 & -\frac{\beta\pi}{\mu} & -\frac{\beta\theta\pi}{\mu} & 0 \\ 0 & -\mu-\phi & \frac{\beta\pi}{\mu} & \frac{\beta\theta\pi}{\mu} & 0 \\ 0 & \phi(1-f) & -\eta-\alpha-\mu & 0 & 0 \\ 0 & \phi f & \eta & -\mu-\mu l-\tau & 0 \\ 0 & 0 & \alpha & \tau & -\mu \end{bmatrix}$$

To analyze the dynamics of the model and we compute the eigenvalues of the Jacobian of the equations at the disease-free equilibrium (DEF). It can be shown that this Jacobian has a left eigenvector is given by $V=(v_1,v_2,v_3,v_4,v_5)^T$ where,

$$v_1=0,\ v_2=\frac{\mu k_3 v_4}{\pi\beta\theta},\ v_3=\left(\frac{k_3+\theta\eta}{\theta k_2}\right)v_4,\ v_4=free,\ v_5=0$$

The right eigenvector is given by, $W=(w_1,w_2,w_3,w_4,w_5)^T$

$$w_1=-\frac{1}{\mu}(\beta x_1 w_3+\beta\theta x_1 w_4)$$

$$w_2=\text{free},\quad w_3=\frac{\phi(1-f)w_2}{k_2},\quad w_4=\frac{\phi f k_2+\eta\phi(1-f)}{k_2 k_3}w_2,$$

$$w_5=\frac{\{\alpha\phi k_3(1-f)+\tau\theta f k_2+\tau\eta\theta(1-f)\}}{k_2 k_3\mu}w_2$$

Computations of a and b:

$$a=\sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0)=2\beta v_2 w_1(w_3+\theta w_4)<0 \qquad (12)$$

$$b=\sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \varphi}(0,0)=\frac{\pi v_2(\theta w_4+w_3)}{\mu}>0 \qquad (13)$$

This result is summarized below.

Theorem 3: The model (1) has a unique EEP, which is locally asymptotically stable (LAS) wherever $R_0 > 1$, and $a < 0,\ b > 0$.

It should be noted that using the Castillo-Chavez and Song theorem the co-efficient, a is given below,

$$a = 2\beta v_2 w_1 \left(w_3 + \theta w_4\right) < 0$$

since all the model parameters and the eigenvectors w_i (i = 2, 3, ...,) and v_i (i = 1, 2, ...) are non-negative and $0 < \theta < 1$.

VII. Global stability of EEP by non-linear Lyapunov function

Theorem 5: The unique EEP

$$\varepsilon_1 = \left(\frac{\pi}{\lambda+\mu}, \frac{\lambda\pi}{(\mu+\phi)(\lambda+\mu)}, \frac{\lambda\pi\phi(1-f)}{(\mu+\phi)(\lambda+\mu)(\alpha+\eta+\mu)}, \frac{\lambda\pi\phi(\alpha f + f\mu + \eta)}{(\mu+\phi)(\mu+\mu_1+\tau)(\lambda+\mu)(\alpha+\eta+\mu)}, \frac{\pi\lambda\phi\{\tau(f\mu+\alpha+\eta)+\alpha(1-f)(\mu+\mu_1)\}}{\mu(\mu+\phi)(\mu+\mu_1+\tau)(\lambda+\mu)(\alpha+\eta+\mu)} \right)$$

of the model is globally asymptotically stable (GAS), whenever $R_0 > 1$.

Let, $R_0 > 1$, so the EEP ε_1 exists. Consider the following non-linear Lyapunov function,

So that $\dot{L} \le 0$ for $R_0 > 1$. Hence, L is a Lyapunov function of the system on Ω. In other words,

$$\lim_{t\to\infty} (S, E, Q, I, R) = \left(S^*, E^*, Q^*, I^*, R^*\right)$$

Thus, by the Lyapunov function L and LaSalle's Invariance Principle every solution to the equation in the model approaches ε_1 as $t \to \infty$ for $R_0 > 1$ [13].

VIII. Numerical Simulation and Discussions

In Fig. 2, The effect of the COVID-19 transmission dynamics is monitored by simulating the model with the parameters from Table 2. COVID-19 transmission decreases gradually with the time where $R_0 < 1$ $\left(\text{with } \beta = 0.9,\ \beta = 0.09,\ \beta = 0.009\right)$. This transmission has to observe as a latent stage with a certain time. In Fig. 3, one infected COVID-19 patient takes medicine at the rate $\left(\tau = 0.5\right)$ in proper time, his infection is decreases rapidly and the infection can be eradicated from the community within a very short time. If he takes treatment at a moderate rate $\left(\tau = 0.05\right)$, he cures of the disease gradually day by day with time. On the other hand, if he takes treatment at the low rate $\left(\tau = 0.005\right)$, he will be cured of the disease at a very slow rate but he gets remove from COVID-19 disease at a certain period.

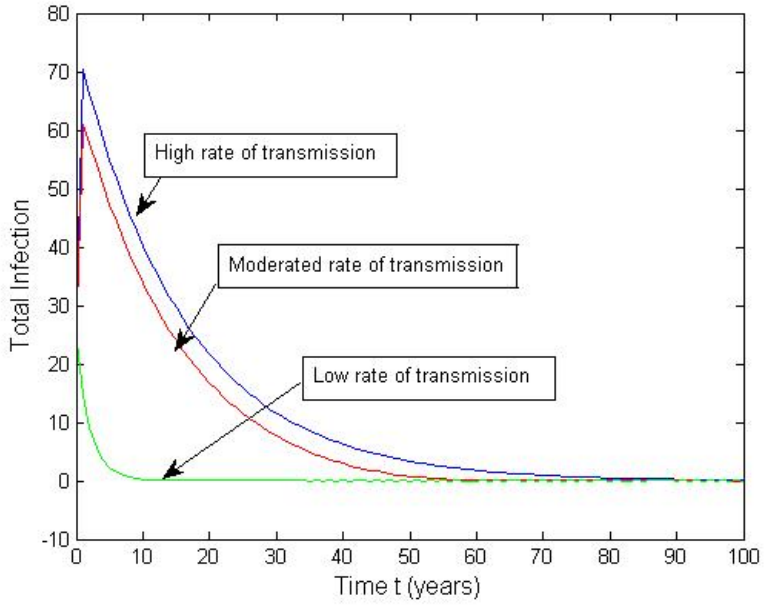


Figure 2: Diagram of COVID-19 transmission rate

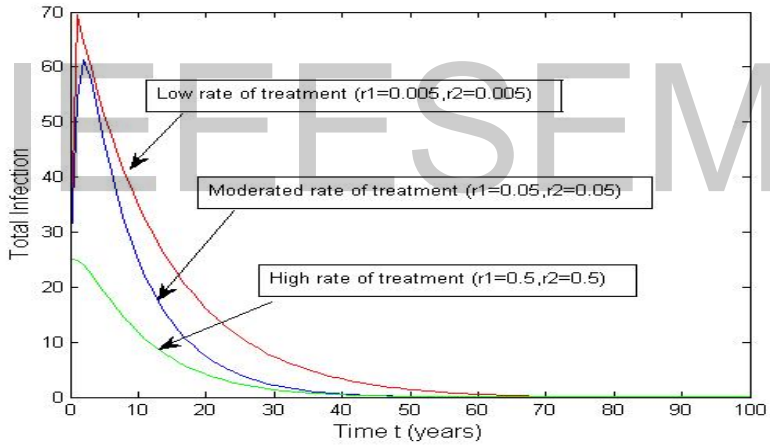


Figure 3: Diagram of COVID-19 treatment

In Fig. 4, it is shown that when $R_0 = 0.8544 < 1$ TB infected person gets remove from COVID-19 rapidly after a certain period with proper treatment and COVID-19 disease is stable for the value of R_0. On the other hand, in Fig. 5, it is shown that when $R_0 = 4.7466 > 1$ COVID-19 infected person spreads there infection at a stable stage with a certain time and COVID-19 disease is unstable for the value of R_0 and the disease can't eradicate from the community permanently.

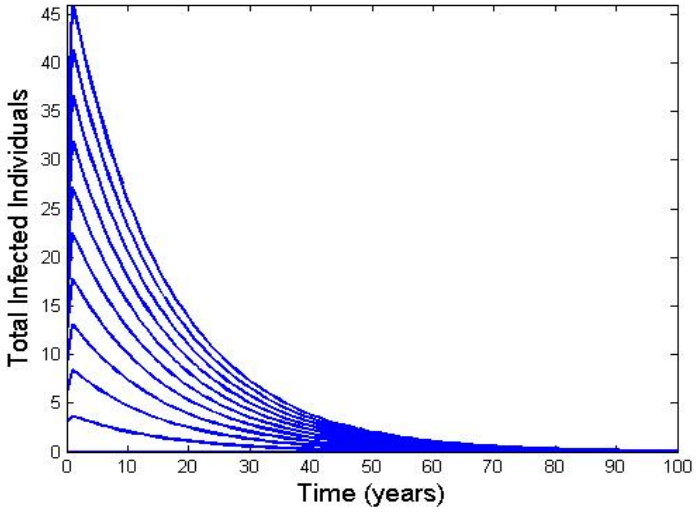


Figure 4: Simulation of the model showing the total number of infected individuals as a function of time, using the parameters in Table 2 where $R_0 = 0.8544 < 1$

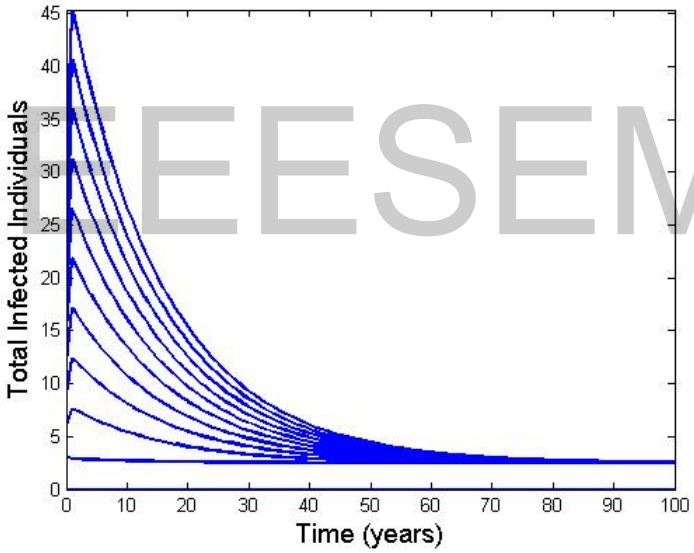


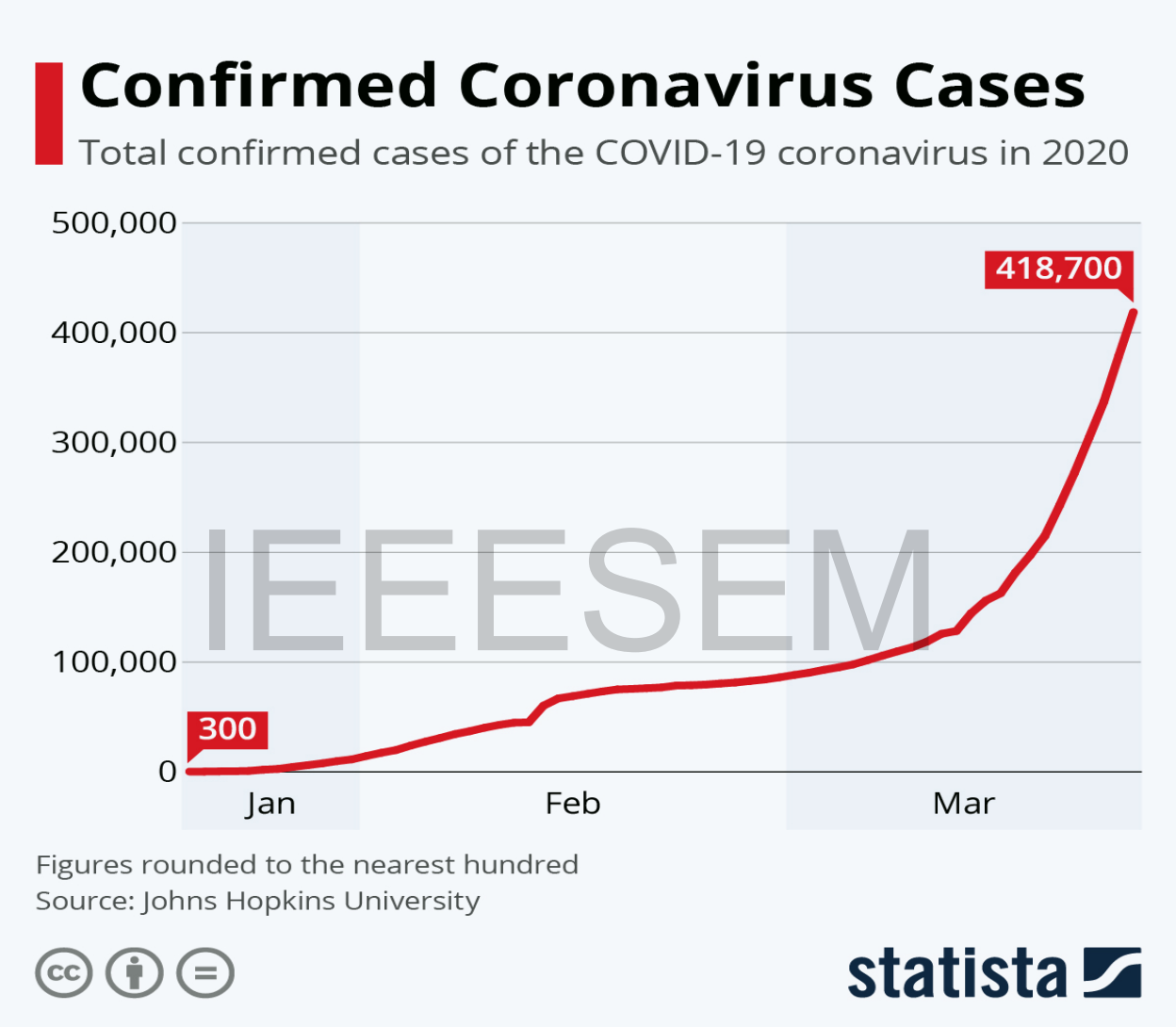
Figure 5: Simulation of the model showing the total number of infected individuals as a function of time, using the parameters in Table 2 where $R_0 = 4.7466 > 1$

IX. Discussion

Approximately eighty presents of cases are moderate, in step with one have a look at, however, older humans with preexisting health troubles are extra vulnerable to excessive headaches like difficulty respiratory and lung infections. Flowing charts and pix lay out what to realize as the outbreak keeps to development. The unfold of the coronavirus from the

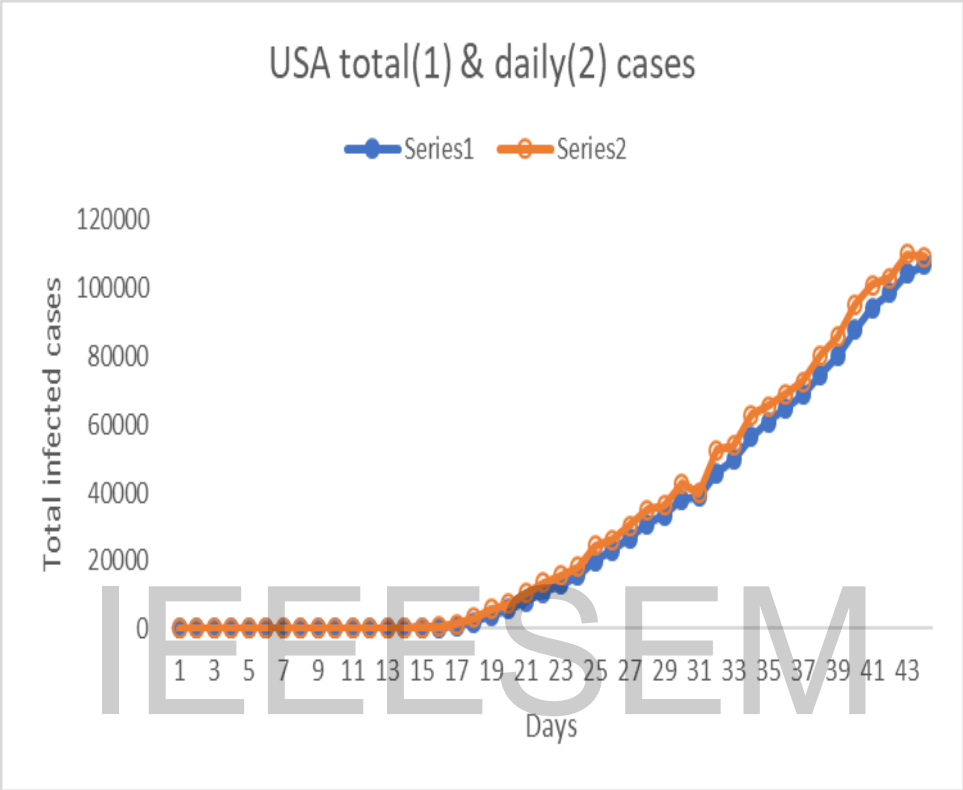
Chinese language town of human maintains at a fast tempo, with over 415 thousand confirmed cases globally as of 24 March. The virus has been confirmed in a hundred and seventy international locations/areas. So far, Johns Hopkins College has tracked almost 19 thousand deaths resulting from the virus.

This chart shows the rate at which the coronavirus case total has shot up worldwide.

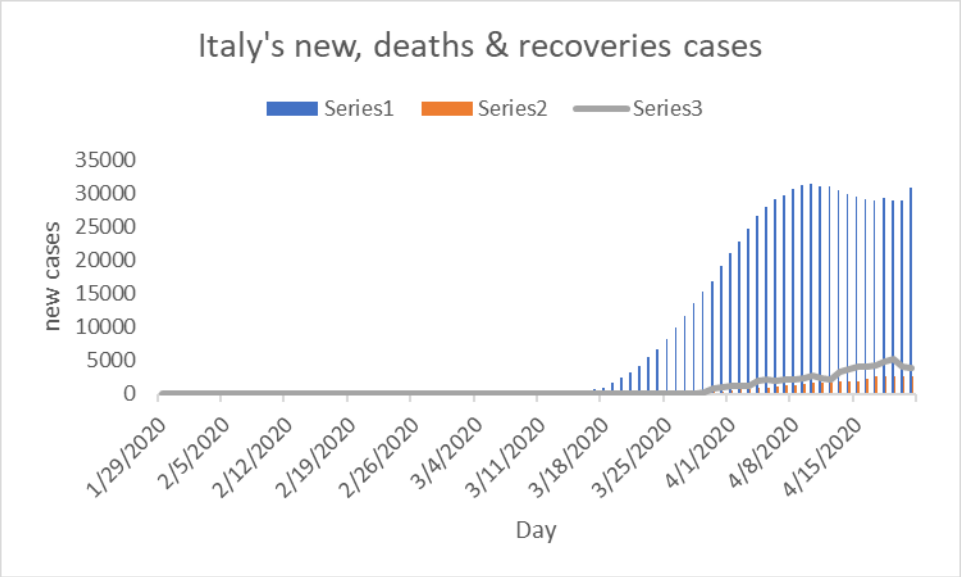


The true number of infected people is probably still higher than the official total since some mild and asymptomatic cases are likely not tested and counted. The sharing equipment determined through the share button on the top or aspect of articles. Copying articles to proportion with others is a breach of ft.com T&Cs and copyright coverage. e-mail licensing@feet.com to buy additional rights. Subscribers can also share up to 10 or 20 articles consistent with the month the usage of the present article provider. Greater facts may be observed at the humanitarian prices of the coronavirus outbreak keep to mount, with greater than 522,000 human beings inflamed globally. The range of humans showed to have died due to the virus has now handed 27,500. The virus's proliferation has been declared a deadly disease with the aid of the sector fitness company that means its miles spreading

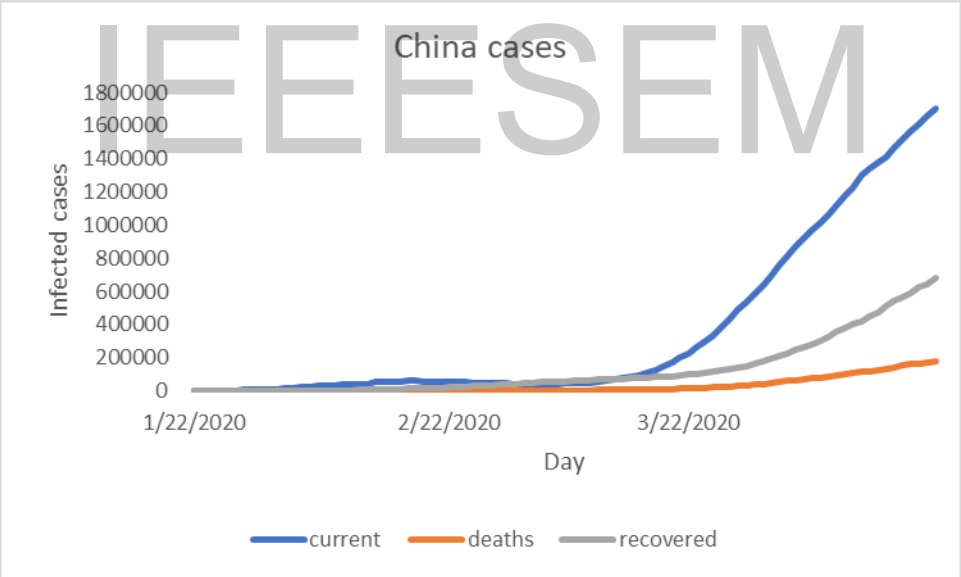
unexpectedly in special parts of the sector. More than 199 nations have shown cases to this point dated on 28 March 2020. The epicentre of the coronavirus is now Europe, with the biggest number of showed instances in Italy, and demise tolls developing extra speedy in Italy and Spain than they did in China on the equal degree of the outbreak.



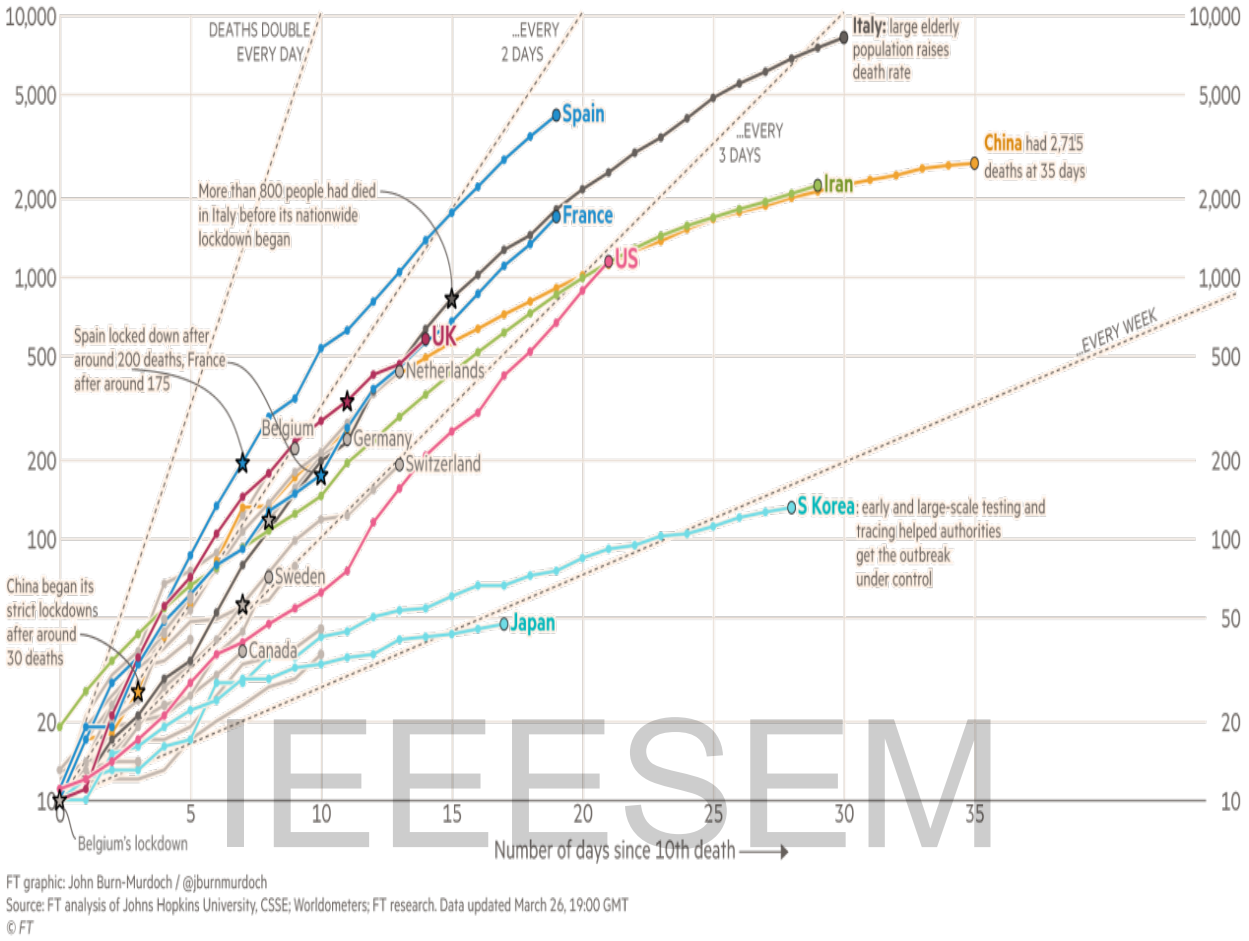
In the country USA cases, we see that the total infected cases and daily infected cases are adjacent to each other, i.e., in USA cases of COVID 19 is the more critical situation than any other countries of the world.



In the country, Italy cases we see that the total infected cases and daily infected cases are not adjacent to each other, i.e., in Italy cases of COVID 19 is the second more critical situation than any other countries of the world.



In the country China cases, we see that the total infected cases and daily infected cases are not adjacent to each other, i.e., in China cases of COVID 19 is the third more critical situation than any other countries of the world, but at first, infected COVID 19 was found in China.

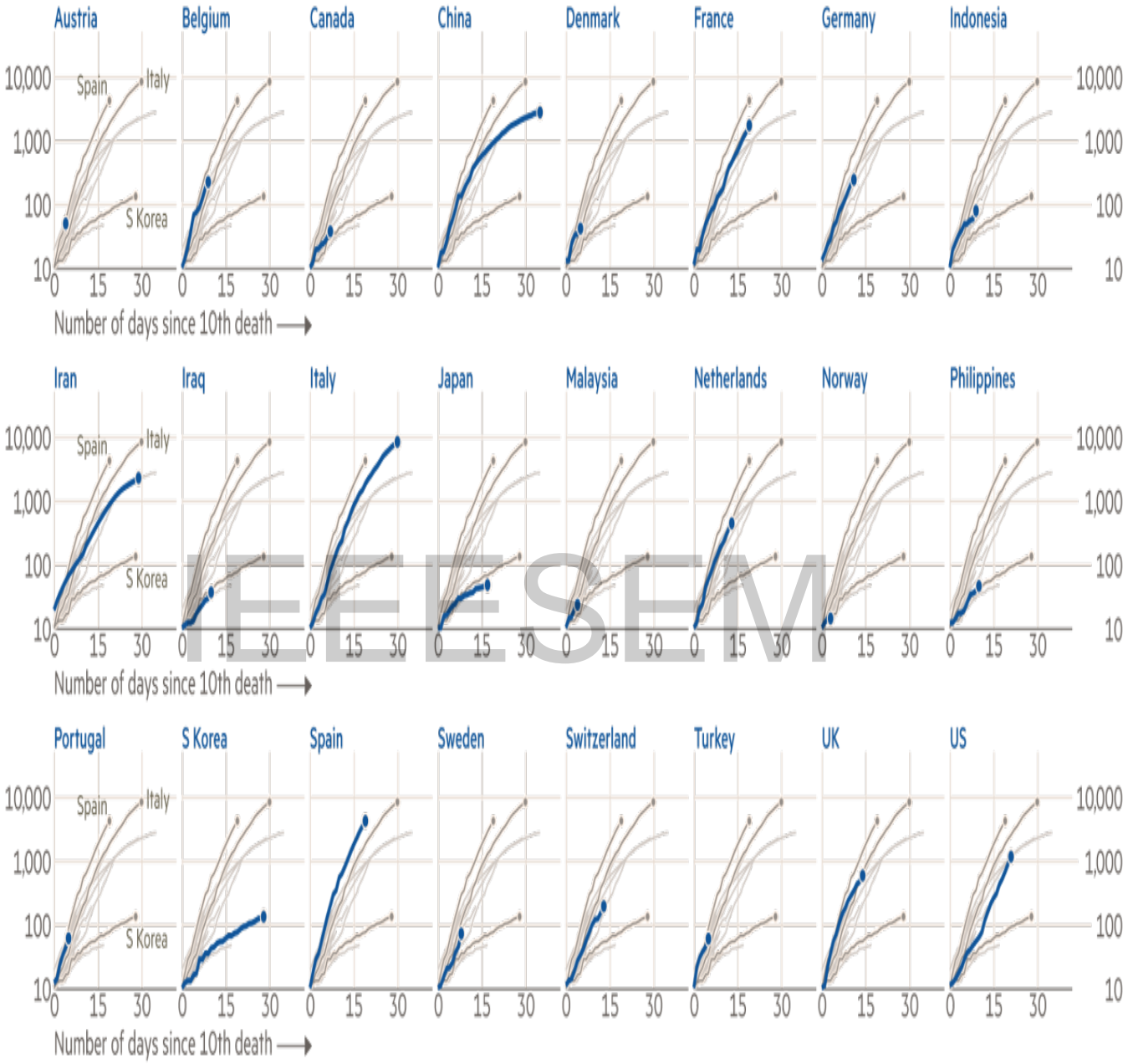


FT graphic: John Burn-Murdoch / @jburnmurdoch
Source: FT analysis of Johns Hopkins University, CSSE; Worldometers; FT research. Data updated March 26, 19:00 GMT
© FT

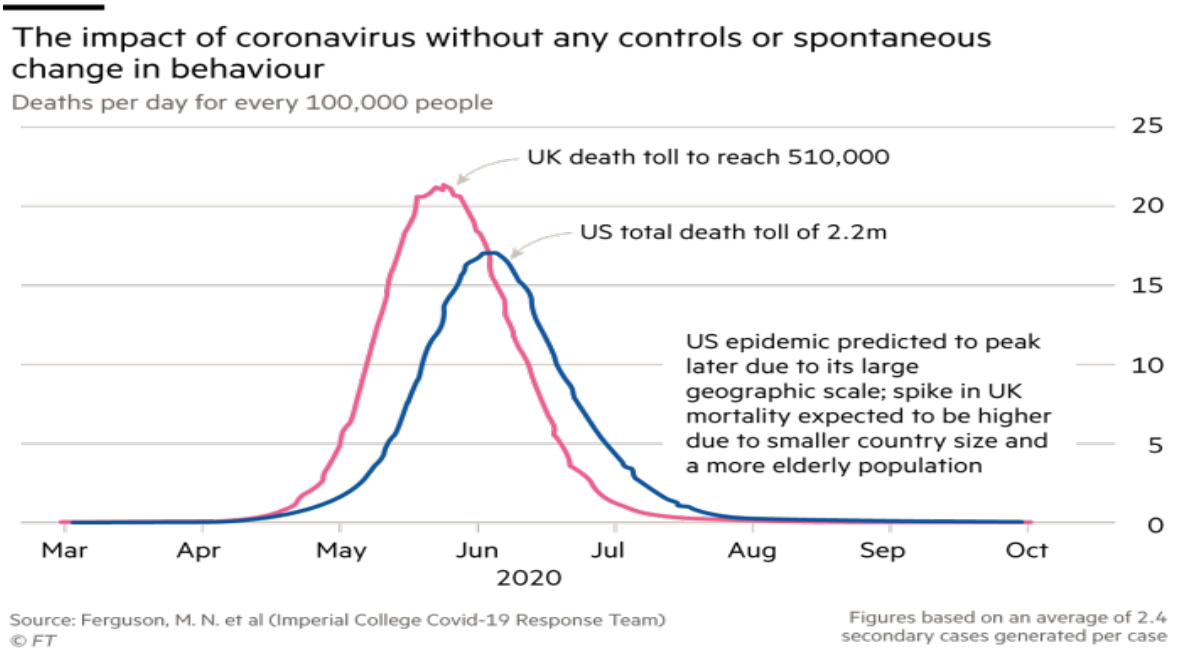
The sharing tools found thru the share button at the top or side of articles. Copying articles to percentage with others is a breach of feet.com T&Cs and Copyright coverage. E-mail licensing@ft.com to buy extra rights. Subscribers might also proportion up to ten or 20 articles consistent with month using the gift article service. The data for this map comes from a dashboard maintained through the Johns Hopkins college centre for structures science Engineering, which has mixed records from the arena health employer, the USA centres for disorder manage and Prevention, the European Centre for Disease Prevention and manage, the Chinese language centres for ailment manipulate and Prevention. It additionally contains records from the Chinese language scientific network website DXY, which aggregates live situation reviews from the Chinese language countrywide fitness commission and local CCDC. Case numbers have now exceeded a hundred in 35 nations. The region now debts for 75% of the latest day by day instances.

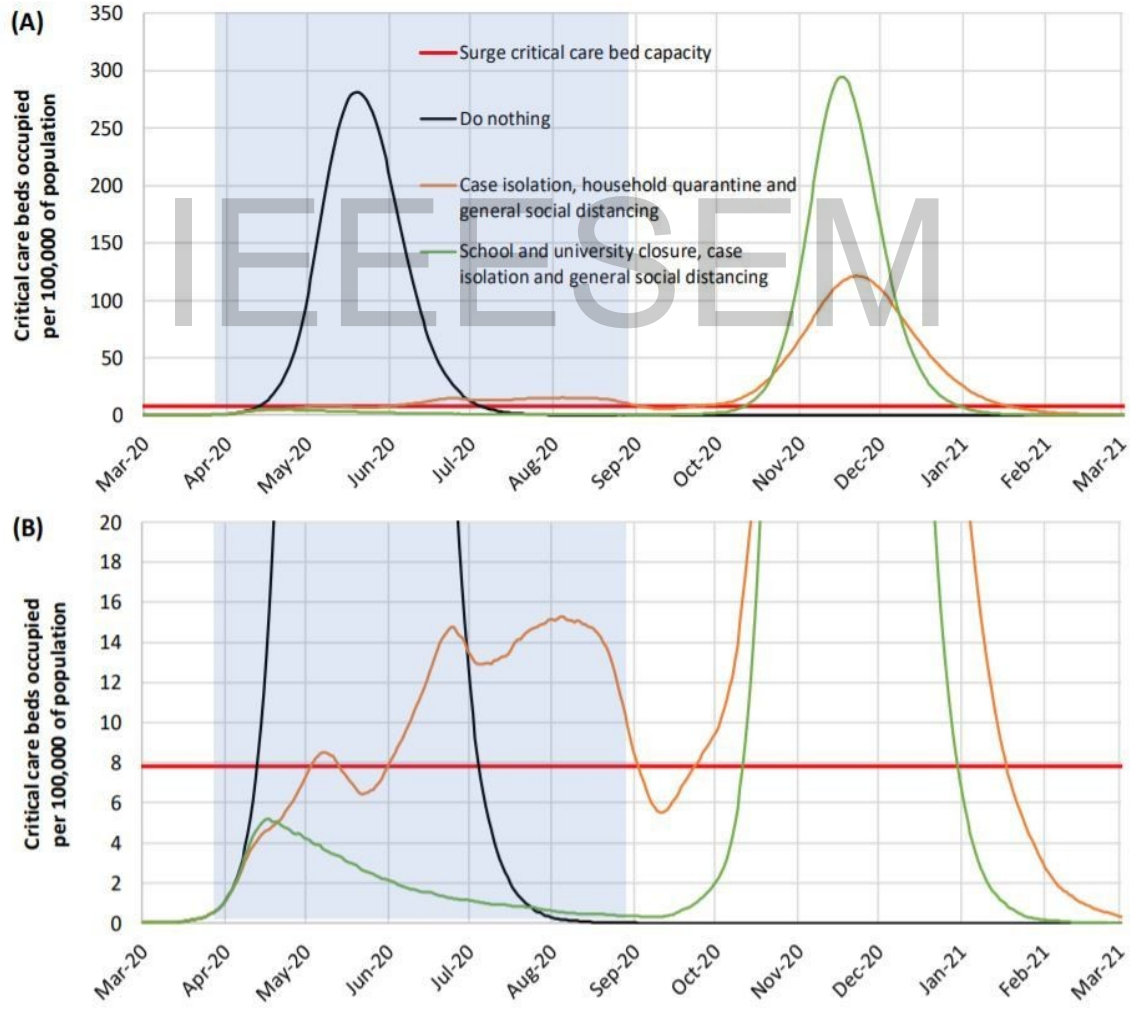
More than 20 countries now have at least 10 deaths

Cumulative number of deaths, by number of days since 10th death



FT graphic: John Burn-Murdoch / @jburnmurdoch, inspired by Philippe Straforelli
Source: FT analysis of Johns Hopkins University, CSSE; Worldometers; FT research. Data updated March 26, 19:00 GMT
© FT



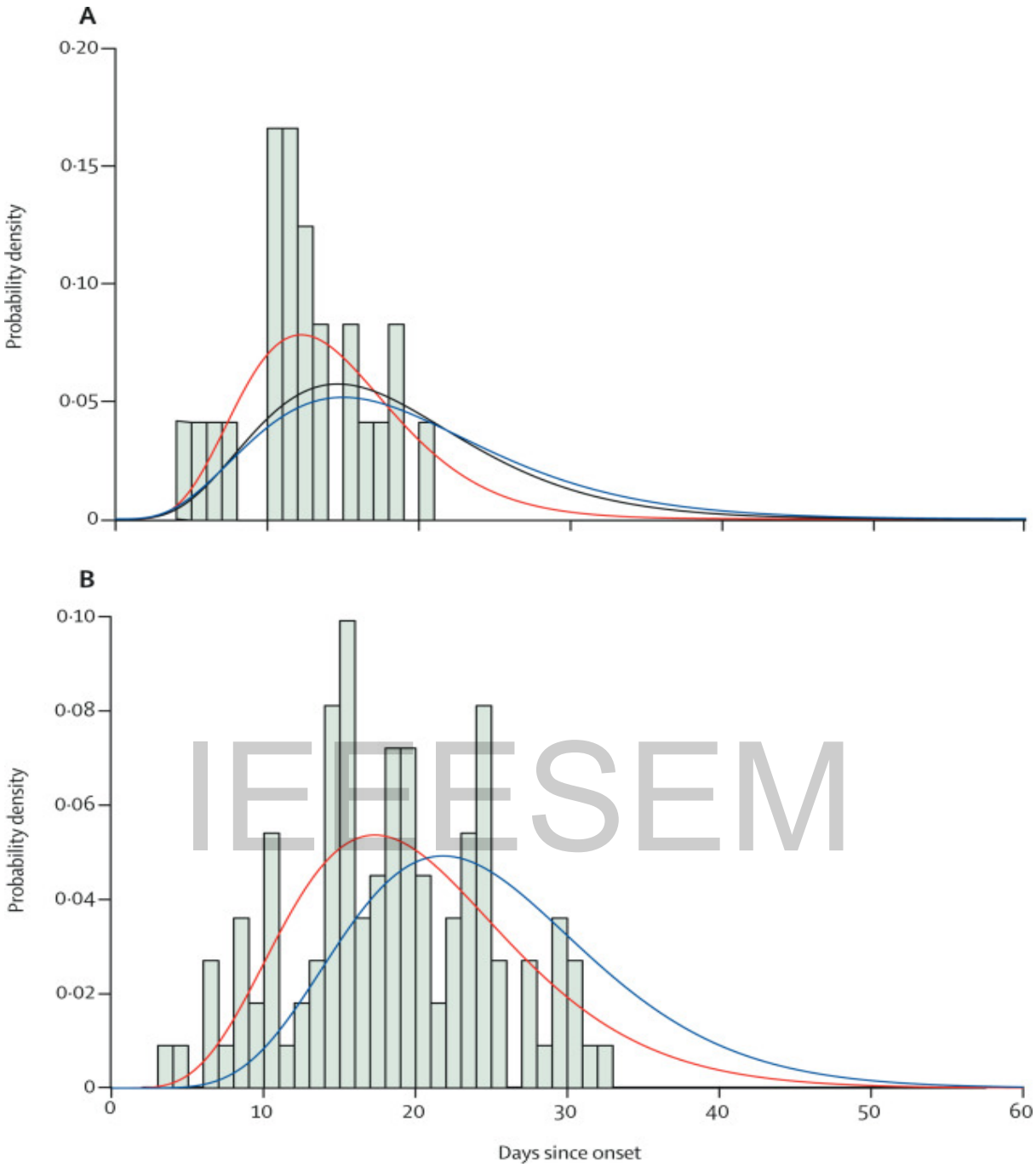


X. Result

We rigorously analyzed (mathematically and numerically) the dynamics of COVID-19 in the model. Some mathematical and epidemiological findings of the study are given below:

1. The model has a disease-free equilibrium (DFE) which is asymptotically stable $R_0 < 1$ and unstable if $R_0 > 1$. The model is also globally asymptotically stable for a special case when $r_1 = r_2 = 0$.
2. When $R_0 = 0.8544 < 1$ the rate of infected individuals increases and after a certain time it smoothly decreases.
3. And lastly, the prevalence is very high when $R_0 = 4.7466 > 1$.

Using data on 24 deaths that occurred in mainland China and 165 recoveries outside of China, we estimated the mean duration from onset of symptoms to death to be 17·8 days (95% credible interval [CrI] 16·9–19·2) and to hospital discharge to be 24·7 days (22·9–28·1). In all laboratory-confirmed and clinically diagnosed cases from mainland China (n=70 117), we estimated a crude case fatality ratio (adjusted for censoring) of 3·67% (95% CrI 3·56–3·80). However, after further adjusting for demography and under-ascertainment, we obtained the best estimate of the case fatality ratio in China of 1·38% (1·23–1·53), with substantially higher ratios in older age groups (0·32% [0·27–0·38] in those aged <60 years *vs* 6·4% [5·7–7·2] in those aged ≥60 years), up to 13·4% (11·2–15·9) in those aged 80 years or older. Estimates of case fatality ratio from international cases stratified by age were consistent with those from China (parametric estimate 1·4% [0·4–3·5] in those aged <60 years [n=360] and 4·5% [1·8–11·1] in those aged ≥60 years [n=151]). Our estimated overall infection fatality ratio for China was 0·66% (0·39–1·33), with an increasing profile with age. Similarly, estimates of the proportion of infected individuals likely to be hospitalised increased with age up to a maximum of 18·4% (11·0–37·6) in those aged 80 years or older. In the subset of 24 deaths from COVID-19 that occurred in mainland China early in the epidemic, with correction for bias introduced by the growth of the epidemic, we estimated the meantime from onset to death to be 18·8 days (95% credible interval [CrI] 15·7–49·7; with a coefficient of variation of 0·45 (95% CrI 0·29–0·54). With the small number of observations in these data and given that they were from early in the epidemic, we could not rule out many deaths occurring with longer times from onset to death, hence the high upper limit of the credible interval. However, given that the epidemic in China has since declined, our posterior estimate of the meantime from onset to death, informed by the analysis of aggregated data from China, is more precise (mean 17·8 days [16·9–19·2]).



4. Figure 2Onset-to-death and onset-to-recovery distributions

XI. Conclusion

A deterministic model for the transmission dynamics of COVID-19 in the population level is designed and rigorously analyzed. Some of the main findings of the study include the following:

(i) The model exhibits DFE is locally asymptotically stable.

(ii) The model has an EEP which is locally and globally asymptotically stable.

Bibliography

1. World Health Organization. Coronavirus. World Health Organization, cited January 19, 2020. Available: https://www.who.int/health-topics/coronavirus.

2. Zhou P, Yang XL, Wang XG, Hu B, Zhang L, Zhang W, et al. A pneumonia outbreak associated with a new coronavirus of probable bat origin. Nature. 2020. https://doi.org/10.1038/s41586-020-2012-7.

3. Li Q, Guan X, Wu P, Wang X, Zhou L, Tong Y, et al. Early transmission dynamics in Wuhan, China, of novel coronavirus-infected pneumonia. N Engl J Med. 2020. https://doi.org/10.1056/NEJMoa2001316.

5. Huang C, Wang Y, Li X, Ren L, Zhao J, Hu Y, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. Lancet. 2020. https://doi.org/10.1016/S0140-736(20)30183-5.

6. Chan JF, Yuan S, Kok KH, To KK, Chu H, Yang J, et al. A familial cluster of pneumonia associated with the 2019 novel coronavirus indicating person-to-person transmission: a study of a family cluster. Lancet. 2020. https://doi.org/10.1016/S0140-6736(20)30154-9.

6. Zhu N, Zhang D, Wang W, Li X, Yang B, Song J, et al. A novel coronavirus from patients with pneumonia in China, 2019. N Engl J Med. 2020. https://doi.org/10.1056/NEJMoa2001017.

7. Bogoch II, Watts A, Thomas-Bachli A, Huber C, Kraemer MUG, Khan K.Pneumonia of unknown etiology in Wuhan, China: Potential for International Spread Via Commercial Air Travel. J Travel Med. 2020. https://doi.org/10.1093/jtm/taaa008.

8. Wu JT, Leung K, Leung GM. Nowcasting and forecasting the potential domestic and international spread of the 2019-nCoV outbreak originating in Chen et al. Infectious Diseases of Poverty (2020) 9:24 Page 7 of 8 Wuhan, China: a modelling study. Lancet. 2020. https://doi.org/10.1016/S0140-6736(20)30260-9.

9. Zhao S, Lin Q, Ran J, Musa SS, Yang G, Wang W, et al. Preliminary estimation of the basic reproduction number of novel coronavirus (2019-nCoV) in China, from 2019 to 2020: a data-driven analysis in the early phase of the outbreak. Int J Infect Dis. 2020. https://doi.org/10.1016/j.ijid.2020.01.050.

10. Zhao S, Musa SS, Lin Q, Ran J, Yang G, Wang W, et al. Estimating the unreported number of novel coronavirus (2019-nCoV) cases in China in the first half of January 2020: a data-driven Modelling analysis of the earlyoutbreak. J Clin Med. 2020. https://doi.org/10.3390/jcm9020388.

11. World Health Organization. Novel Coronavirus – China. World Health Organization. cited January 20, 2020. Available: https://www.who.int/csr/don/12-january-2020-novel-coronavirus-china/en/. Accessed 20 Jan 2020.

12. Chen T, Rui J, Wang Q, Zhao Z, Cui J-A, Yin L. A mathematical model for simulating the transmission of Wuhan novel Coronavirus. bioRxiv. 2020:2020.2001.2019.911669. Accessed 13 Feb 2020.

13. Jannatun Nayeem and Chandra N. Podder (2014). A Mathematical study on the Vaccination impact on the disease dynamics of HBV. IOSR Journal of Mathematics, e-ISSN: 2278-5728, P-ISSN: 2319-756X, Vol-10, p-26-44.

14. S.O. Adewale, C.N. Podder and A.B Gumble (2009). MATHEMATICAL ANLYSIS OF A TB TRANSMISSION MODEL WITH DOTS. Canadian applied mathematics quarterly, vol-17.

15. Hong bin Guo and Michael Y. LI (2006). Global stability in a Mathematical Model of Tuberculosis. CANADIAN APPLIED MATHEMATICS QUARTERLY Volume 14, Number 2.

16. A. Loweell et al.(1996). Tuberculosis. Havard University Press, Cambridge, M. A.

17. C. Castillo Chavez et. al.(2005). A model of TB with exogenous re-infection.Theor.POP. Biol 132, 235-239.