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A review of epidemiologic models: from SIR to distributed delays

Natali Hritonenko · Victoria Hritonenko · Yuri Yatsenko

Abstract The review surveys and compares state-of-the-art epidemiologic models starting from the celebrated SIR model to recent SEIR Erlang models and integral models with distributed delays. The authors emphasize the flexibility and advantages of integral models in describing government efforts to control the current COVID -19 pandemic.

Keywords epidemiologic models · Erlang SEIR models · integral models · distributed delay · COVID pandemic control

Mathematics Subject Classification (2010) 45-02 · 92C50 · 92D30 · 97M10

1 Introduction

This Special Issue "Surveys in Mathematical Biology and Medicine: Modelling, Simulations and Control" is timely and important. The authors appreciate the invitation and efforts of their long-time colleague and friend Professor Sebastian Aniţa to organize this issue.

The paper briefly surveys the eighty-year history of epidemiologic modeling in its development, starting from the classic SIR model and including recent models with realistic distributions of latent and infection periods. The authors focus on mathematical logic of models and how they match real epidemic processes. Extensive epidemiologic research [20], [30], has conceded that the standard SIR model and its first modifications (SIS, SEIR, SMEIRS, and other) do not adequately describe delays in real disease transmission and, thus, cannot satisfactorily explain dynamics of the majority of real epidemics. To describe more realistic infection distributions, multicompartment epidemiological models were suggested and analyzed [15], [30]. Such models are described by larger ODE systems but provide more flexibility in describing transition dynamics. Starting 2020, the epidemic models from SIR family have been intensively used to describe COVID-19 propagation and control without addressing the model suitability [1], [2], [8], [14],

[18]. This paper argues that specific features of COVID-19 control require new refined models.

An alternative way to develop realistic descriptions of epidemic propagation and control is based on using integral and integrodifferential equations [12], [13], [28]. Integral dynamic models with distributed delay have been known for almost a hundred years and successfully used in many applications [3], [4], [23], [24], [25], [26], [27], [35]. This paper substantiates that integral models are more natural and flexible for the control of COVID-19. They open new possibilities to describe and optimize the economic control of epidemics [10].

2 SIR, SEIR, SLIAR, and Other Models

This section briefly surveys the evolution of epidemiologic models starting from the classic SIR model and its major modifications. We follow common notations in the literature.

2.1 Standard SIR model

To focus on epidemics, most SIR-family models abstract from new births and non-epidemic-related deaths. The classic *SIR model* of [28] considers a constant population of N individuals. At each time t, the population is divided into three groups (stages): Susceptible (S), Infectious (I), and Recovered or dead (R) individuals:

$$S(t) + I(t) + R(t) = N, \tag{2.1}$$

where S is the number of susceptible individuals, I is the number of infectious individuals, and R is the number of recovered (or dead) individuals. These numbers evolve in time as

$$I'(t) = \beta(t)I(t)\frac{S(t)}{N} - \gamma I(t), \tag{2.2}$$

$$S'(t) = -\beta(t)I(t)\frac{S(t)}{N}, \tag{2.3}$$

$$R'(t) = \gamma I, \qquad 0 \le 1 < \infty. \tag{2.4}$$

All recovered individuals are assumed to become immune. Therefore, the only susceptible fraction S/N of the population can become infected. By (2.2)-(2.4), individuals leave the "Susceptible" stage and move to "Infected" at the rate $\beta(t)I(t)S(t)/N$, while the infected individuals move to the "Recovered or dead" stage at a constant exponential Poisson rate γ. The average duration τ of the stage "Infected" is $\tau = 1/\gamma$. The analytic solution to the nonlinear ODE system (2.2)-(2.4) is obtained in [11].

The most critical parameter in (2.2)-(2.4) is the transmission rate β, at which an infected individual contaminates others. It equals the product of the average number of contacts of one individual per time unit and the probability of transmitting disease during one contact.

Recent epidemic-economic models [1], [2], [14], use the variable rate $\beta(t)$ to capture government control actions and behavioral changes such as lock-down, social distance, and similar.

The fraction $0 < \delta < 1$ of "Recovered or dead" is assumed to die, so, the number of dead is

$$D(t) = \delta R(t). \tag{2.5}$$

The death rate δ is considered as a fixed parameter in most SIR-like models, but in reality it can fluctuate with time. The model (2.1)-(2.5) is known as the *SIRD model*.

In epidemiology, the *reproduction number* is the expected number of new infections generated by one infected individual. In SIR model (2.1)-(2.5), it is calculated as

$$\mathfrak{R}_t = \beta(t)/\gamma^* S(t)/N. \tag{2.6}$$

At $\mathfrak{R}_t > 1$, the modeled epidemic grows, otherwise it weakens. The *basic reproduction number*

$$\mathfrak{R}_0 = \beta(t)/\gamma \tag{2.7}$$

describes the expected number of new infections generated by one infected individual in a totally susceptible large population, i.e., when $S(t) \ll N$.

If no recovered individuals become immune after infection, then we obtain the *SIS* (*susceptible-infectious-susceptible*) model:

$$I'(t) = \beta(t)I(t)\frac{S(t)}{N} - \gamma I(t), \tag{2.8}$$

$$S'(t) = -\beta(t)I(t)\frac{S(t)}{N} + \gamma I(t). \tag{2.9}$$

2.2 SEIR model

The *SEIR* (*susceptible-exposed-infectious-recovered*) *model* includes the separate stage "Exposed", which contains individuals in a latent state of infection (that are infected but not yet infectious). The non-infected individuals are either susceptible (S) or recovered (R).

Here and thereafter, the dependence on time t will be omitted for notational simplicity (unless it leads to ambiguity). Also, let the total population size be $N = 1$, then, the unknown functions S, E, I, R are fractions (proportions) of the total population. The SEIR model is described by four nonlinear ODEs [13], [15], [22]:

$$S' = \upsilon - \beta SI - \mu S, \tag{2.10}$$

$$E' = \beta SI - (\sigma + \mu)E, \tag{2.11}$$

$$I' = \sigma E - (\gamma + \mu)I, \tag{2.12}$$

$$R' = \gamma I, \qquad 0 \le t < \infty. \tag{2.13}$$

where S is the proportion of the population that is susceptible, E is the proportion of the population in the latent stage, and I is the proportion of infectious individuals. The model assumes that the exposed individuals eventually become sick and move to the "Infectious" stage at the exponential rate $\sigma > 0$. The parameter β is the transmission rate, υ is the natural per capita birth rate, and μ is the natural per capita death rate. Then, the mean durations of the latent (E) and infectious (I) periods are $1/(\sigma + \mu)$ and $1/(\gamma + \mu)$, respectively.

As [7] stipulates, *epidemic models* (in contrast to *endemic models*) consider short time horizons and assume no birth or natural death, only death by removal from the infectious stage. Then, at $\upsilon = \mu = 0$, the SEIR model (2.10)-(2.13) is reduced to

$$S' = -\beta SI, \tag{2.14}$$

$$E' = \beta SI - \sigma E, \tag{2.15}$$

$$I' = \sigma E - \gamma I, \tag{2.16}$$

$$R' = \gamma I, \qquad 0 \le t < \infty. \tag{2.17}$$

As in the SIR model (2.1)-(2.4), a fraction $0 < \delta < 1$ of "Recovered" dies of disease. So, the number of dead is $D = \delta R$.

The SEIR model was a step ahead for modeling of diseases with latent periods. However, comprehensive research [7], [20], [30], concluded that most epidemics did not follow assumptions of the SEIR model. As a result, the SEIR model does not fit well real epidemiological data.

Confusingly for general scientific community, prominent epidemiologists disagree on the name of the SEIR model. Some of them consider the term *latent* as a better description of the *exposed* (infected but not yet infectious) individuals. The "Latently infected" stage is similar to "Exposed" and may include some individuals with lower infectivity. So, the SEIR model (2.10)-(2.13) can be referred to as a *SLIR model* [5], [6], [7].

2.3 SEIRS model

Many categories of diseases do not guarantee a permanent immunity [21]. To include such cases, the SEIR model (2.10)-(2.13) has been elevated to the *SEIRS (susceptible-exposed-infectious-recovered-susceptible) model* [13], [22], described by four nonlinear ODEs:

$$S' = \upsilon - \beta SI - \mu S + \eta R, \tag{2.18}$$

$$E' = \beta SI - (\sigma + \mu)E, \tag{2.19}$$

$$I' = \sigma E - (\gamma + \mu)I, \tag{2.20}$$

$$R' = \gamma I - (\eta + \mu)R, \qquad 0 \le t < \infty. \tag{2.21}$$

This model describes a *nonpermanent* (temporary) *immunity*, when the recovered individuals eventually lose the immunity and move back to the "Susceptible" stage at the exponential rate $\eta > 0$.

2.4 SLIAR model

An important development of the SEIR epidemic model is the SLIAR (susceptible-latently infected-symptomatic-asymptomatic infectious-removed) model used to describe the propagation of respiratory diseases.

The SLIAR model [5] includes a separate "Latent" stage, in which there is some reduced infectivity (represented by a factor $\varepsilon < 1$). The SLIAR model is described by the system of six nonlinear ODEs:

$$S' = -\beta S(I + \varepsilon L + \rho A), \tag{2.22}$$

$$L' = \beta S(I + \varepsilon L + \rho A) - \kappa L, \tag{2.23}$$

$$I' = p\kappa L - \alpha I, \tag{2.24}$$

$$A' = (1 - p)\kappa L - \eta A, \tag{2.25}$$

$$R' = (1 - \delta)\alpha I + \eta A, \tag{2.26}$$

$$D' = \delta\alpha I, \qquad 0 \le t < \infty. \tag{2.27}$$

In the model (2.22)-(2.27), the latent individuals eventually go to the "Asymptomatic" and "Infectious" stages with the exponential rate $\kappa > 0$. In doing so, the fraction p of the latent individuals goes to the "Infectious" stage, while $(1 - p)$ goes to the "Asymptomatic" stage with reduced infectivity described by the factor $\rho < 1$.

The asymptomatic individuals eventually move to the "Recovered" stage at the exponential rate $\eta > 0$. The infectious individuals eventually move to the "Recovered" and "Dead" stages at the exponential rate $\alpha > 0$. The fraction $\delta < 1$ of the "Infectious" dies and $(1 - \delta)$ recovers.

The SLIAR model assumes no natural birth and death, but the death by infection only. The mean latent, infective, and asymptomatic periods are $1/\kappa$, $1/\alpha$ and $1/\eta$, respectively. Compared to the SEIR (SLIR) models, the SLIAR model (2.22)-(2.27) emphasizes infection by asymptomatic individuals, which has been reported to substantially contribute to COVID-19 propagation [34].

3 Epidemic Models with Additional Compartments

Regrettably, a later thorough analysis showed that the above models fail to explain the dynamics of most real epidemics [20], [30]. It has been noticed by epidemiologists starting 1939.

Indeed, SIR-like models are simplified descriptions of real epidemiological processes. Such approximations have their application domains, are acceptable in some real situations, though can be improved when necessary.

The key weakness of the above models is that the used ODEs describe the so-called memoryless dynamic systems with exponentially distributed exit times. For clarity, let us consider the standard SIR model (2.1)-(2.4) in the special case of a small epidemic in a large population, i.e., when $S \ll N$. Following the equation (2.2), if $n(t)$ individuals enter the stage I at a fixed time t_1 and eventually exit at the rate γ, then the number of individuals remaining in stage I at time $t_1 + \tau$ exponentially decreases in τ as

$$n(t_1\tau) = n(t)\exp(-\gamma\tau). \tag{3.1}$$

The formula (3.1) describes the so-called *infectious period distribution* (*IPD*) in the SIR model. It directly follows from the general solution to the linearized ODE (2.2) with respect to I. Stated differently, each individual remains in the stage I with the probability $p(\tau) = \gamma\exp(-\gamma\tau)$ at the time τ after entering this stage.

The exponential IPD (3.1) appears to be unrealistic in epidemiological practice. It has too fat right tail. Following (3.1), some individuals will remain in stage I at any distant time in the future, which is impossible. In reality, all individuals exit all model stages in the *finite* time. As result, such models underestimate the basic reproductive number R_0 of epidemic processes.

To override those challenges, the *epidemiologic models with additional compartments* were constructed. Such models are described by epidemiologists as a mathematical device or *mathematical trick* to make the model-generated IPDs more realistic [15], [20], [30]. The trick (also known as "*linear chain trick*" or "*boxcar formulation*") allows to obtain better epidemiologic models with more realistic IPDs while remaining in the ODE framework. Stated another way, such models attempt to implement *continuous-time distributed delays* using ODEs. A more natural way to achieve that would be to switch to delay-differential or integral equations, which is discussed in Section 4.

3.1 SIR model with realistic infection distributions

Applied to the standard SIR model (2.1)-(2.4), the *linear chain trick* is as follows.

In order to include a more realistic IPD, the modified SIR model divides the "Infectious" stage of the SIR model into n substages $I_1, ..., I_n$. Then,

newly infected individuals enter the first substage I_1, pass through each successive substage, and move to the "Recovered" stage as they leave the n-th substage I_n. Lloyd [30] points out that these additional substages have no analogues in reality.

The obtained *SIR model with realistic distributions* [30] is described by the following system of $n+1$ nonlinear ODEs:

$$S' = \mu - \beta SI - \mu S, \qquad I = \sum_{k=1}^{n} I_k, \tag{3.2}$$

$$\begin{aligned} I_1' &= \beta SI - (n\gamma + \mu) I_1, \\ I_i' &= n\gamma \beta S I_i - (n\gamma + \mu) I_i, \qquad i = 2, ..., n. \end{aligned} \tag{3.3}$$

Here, I is the total number of infective individuals, which equals the sum of the infective individuals, I_j, in each of the n substages. The variable S and parameters β and γ have the same meanings as in the SIR model (2.1)-(2.5). Per-capita birth and death rates are both equal to μ and there is no mortality due to the disease. Thus, the population size N remains constant, and the number of recovered individuals is $R = N - S - I$. The mean duration of infection period is the same $1/(\gamma + \mu)$ at any value of n.

In the model (3.2)-(3.3), the probability of individuals to remain in the stage I is not exponential $p(\tau) = \gamma \exp(-\gamma\tau)$ anymore. To demonstrate that, we consider this model in the case of small epidemics in a large population, at $S \ll N$ and $\mu = 0$. Let $p_i(\tau)$ denote the probability of an individual to be in the i-th stage. Analogously to the standard SIR model (2.1)-(2.4), we obtain $p_1(\tau) = \gamma \exp(-\gamma\tau)$ for the first stage I_1. Next, the sequential solution of the ODE (3.3) at $i = 2, 3, ..., n$ gives the formula

$$p_i(\tau) = \gamma \frac{(n\gamma\tau)^{i-1}}{(i-1)!} e^{-n\gamma\tau} \qquad i = 2, ...n. \tag{3.4}$$

Finally, the probability to be infectious (remain in the stage I) at the time τ after entering the stage is *gamma-distributed*:

$$\sum_{i=1}^{n} p_i(\tau) = \gamma e^{-n\gamma\tau} \sum_{i=1}^{n} \frac{(n\gamma\tau)^{i-1}}{(i-1)!} = 1 - F(\tau; n, \gamma), \tag{3.5}$$

where $F(\tau; n, \gamma)$ is the regularized *gamma function* with parameters n and γ. The gamma distribution with the integer shape parameter n is also known as the *Erlang distribution* [15].

The gamma distribution is determined by its mean γ and the integer shape parameter n. For almost all values of n, except for small ones, the IPD (3.5) is close to the normal distribution. As the number of substages n increases, the IPD becomes more closely centered around its mean γ. The limit of the distribution (3.5) at $n \to \infty$ is the *Dirac delta distribution* $\delta(\gamma)$, which corresponds to all individuals having exactly the same infectiveness duration γ.

3.2 The Erlang SEIR model

A multi-compartment SEIR model, known as the *Erlang SEIR model*, is a generalization of the SEIR model [15], [20]. It divides the E and I stages into m and n substages, respectively. All m latent substages are identical, and so are all n infectious substages. The goal is to obtain more realistic distributions of both the latent and infectious periods.

The Erlang SEIR model is described by a system of $m+n+1$ ODEs,

$$S' = \mu - \beta SI - \mu S, \qquad I = \sum_{k=1}^{n} I_k, \tag{3.6}$$

$$E_1' = \beta SI - (m\sigma + \mu)E_1, \tag{3.7}$$

$$E_j' = m\sigma E_{j-k} - (m\sigma + \mu)I_k, \qquad j = 2, ..., m, \tag{3.8}$$

$$I_1' = m\sigma E_m - (n\gamma + \mu)I_1, \tag{3.9}$$

$$I_k' = n\gamma\beta SI_k - (n\gamma + \mu)I_k, \qquad k = 2, ..., n, \tag{3.10}$$

where E_j is the proportion of the population that is in the j-th latent compartment, I_k is the proportion of the population that is in the k-th infectious compartment.

The variable S and parameters β, σ, and γ have the same meanings as in the SEIR model (2.10)-(2.13). The transition rates ($m\sigma$ and $n\gamma$) between substages are scaled by the numbers of compartments, the average durations $1/(\sigma+\mu)$ and $1/(\gamma+\mu)$ of the E and I stages do not depend on the numbers m and n of compartments.

Similarly to the reasoning for the multi-compartment SIR model (3.1)-(3.3) in Section 3.1, one can prove that the resulting latent and infectious periods have the Erlang distributions (gamma distributions with integer shape parameter), see [15], [20], for details.

3.3 SLIAR models with additional compartments

Multi-compartment versions of the *SLIAR* model (2.22)-(2.27) also exist and divide each of I, L, and A stages into several identical substages [7].

Here, we restrict ourselves with the recent $SL_1L_2I_1I_2A_1A_2R$ model with eight compartments, which is offered and calibrated for COVID-19 epidemic in [6]. The model is described by the following eight ODEs:

$$S' = -\beta S(I_1 + I_2 + \varepsilon L_1 + \varepsilon L_1 + \rho A_1 + \rho A_1), \tag{3.11}$$

$$L_1' = \beta(I_1 + I_2 + \varepsilon L_1 + \varepsilon L_1 + \rho A_1 + \rho A_1) - \kappa L_1, \tag{3.12}$$

$$L_2' = \kappa(L_1 - L_2), \tag{3.13}$$

$$I_1' = p\kappa L_2 - \alpha I_1, \tag{3.14}$$

$$I_2' = \alpha(I_1 - I_2), \tag{3.15}$$

$$A_1' = (1-p)\kappa L_2 - \eta A_1, \tag{3.16}$$

$$A_2' = \eta(A_1 - A_2), \tag{3.17}$$

$$R' = \alpha I_2 - \eta A_2, \tag{3.18}$$

where the notations of the SLIAR model (2.22)-(2.27) are used and indexed for two substages (compartments) in each stage L, I, and A.

A SEIR model with six compartments (susceptible, exposed, asymptomatically infected, symptomatically infected, recovered, and dead individuals) was used to describe the dynamics of COVID-19 lockdown in Mexico.

4 Distributed-delay integral models vs ODEs

Integral dynamic models have been commonly used for the description of population dynamics, including epidemiology, starting the beginning of 20^{th} century (recall, for example, Volterra and Lotka integral models). Analytic comparison of integral epidemiological models with distributed delay and the comprehensive ODE-based compartmental models of previous sections is provided by Breda et al. [13] and Champredon et al. [15], who demonstrate that those two model classes produce the same dynamics in special cases. Below we briefly review these two papers.

It appears that all major ideas about applying interval dynamic models to epidemics were originated in the celebrated paper [28] (Kermack and McKendrick, 1927). To illustrate that, Breda et al. [13] analyze the model of an epidemic in a closed population described by the following equations

$$F(t) = \int_0^\infty F(t-s)S(t-s)A(s)ds, \tag{4.1}$$

$$S'(t) = -F(t)S(t), \tag{4.2}$$

with respect to $S(t)$ and the new unknown variable, the *force of infection*, $F(t)$. The force of infection is the probability (pet unit time) that a susceptible individual becomes infected. It corresponds to $\beta I(t)$ in the SIR and SEIR models of Sections 2 and 3.

The given function $A(s)$ is the "infection force" of one individual infected s time units ago. Breda et al. [13] emphasize that the standard SIR and SEIR model can be obtained from the integral model (4.1)-(4.2) at $A_{SIR}(s) = \beta e^{-\gamma s}$ and $A_{SEIR}(s) = \beta\frac{\gamma}{\gamma-\sigma}(e^{-\sigma s} - e^{-\gamma s})$, respectively.

Solving the linear ODE (4.2) in S and substituting the result to (4.1) produces one *nonlinear renewal equation*

$$y(t) = \int_0^\infty (1 - e^{-y(t-s)})S(-\infty)A(s)ds, \tag{4.3}$$

with respect to the *cumulative force* of infection $y(t) = \int_0^\infty F(s)ds$ [13].

As Champredon et al. [15] stress, the renewal (integral) and ODE approaches are based on different conceptualizations of population dynamics. The renewal approach focuses on cohorts of infectious individuals and the way how they spread infection through time, while the ODE approach counts individuals in different stages.

Champredon et al. [15] construct the model of an epidemic in a population with natural reproduction as a renewal equation

$$i(t) = R_0 S(t) \int_0^\infty i(s) g(s) ds, \tag{4.4}$$

$$S'(t) = \mu - i(t) - \mu S(t), \tag{4.5}$$

with respect to $S(t)$ and the *incidence* $i(t)$. The incidence is the rate at which new infections occur in the population and corresponds to the flow rate $\beta S(t) I(t)$ in the SIR and SEIR models.

The given *intrinsic generation-interval distribution* $g(s)$ is the probability that an individual survives and is infectious s days after becoming infected, i.e., remains in the stage I. For SIR model, with additional compartments, $g(s)$ is given by (3.5).

Champredon et al. [15] demonstrate that the renewal model (4.4)-(4.5) and the SEIR Erlang model (3.6)-(3.10) produce the same epidemic dynamics when the *interval distribution* $g(s)$ is the same. It confirms the well-known fact that integral equations with distributed delay can be converted to ODEs of higher dimension. However, the integral models with delays are more general and can describe other patterns of infectivity and latency, including those obtained in clinical data. Those patterns will not be necessarily exponentially or gamma distributed.

Moreover, the integral equations naturally describe delays that occur in epidemic models with a variable transmission rate β. Economic models [1], [2], and others use the variable changing rate $\beta(t)$ to capture government control of COVID-19. The ODE-based models of Sections 2 and 3 do not consider the variable transmission rate β at all.

5 Conclusion: Toward economic-epidemiologic models for COVID-19 control

An intensive research in modeling the COVID-19 pandemic and governmental attempts to control it started in 2020 [1], [2], [8], [9], [14], [17], [18], [19], [29], [32], [33], and many others. Most related economic models use the SIR or SEIR epidemic models (2.1)-(2.4) and (2.10)-(2.13) with a variable transmission rate $\beta(t)$. This rate changes over time to capture government control actions such as lockdowns, shelter-in-place orders, social distancing and related behavioral changes.

Moreover, some economic-related models of COVID-19 [1], [2], and others assume that the transmission rate $\beta(t)$ possesses a discrete jump at the

time of the introduction of a lockdown. All the above models of Sections 2 and 3 are not suitable for accurate modeling of such sharp changes in the transmission rate. Indeed, if a lockdown was introduced yesterday, it affects only a small portion of today contacts while most contacts already occurred during preceding latent and infection periods. However, the change of $\beta(t)$ in any of the above ODE models will affect ALL contacts between the S and I classes.

At the best of the authors' knowledge, no systematic research has been done to analyze the variable transmission rate $\beta(t)$ in any of the above epidemiologic models. The variable transmission rate leads to specific delays in disease propagation that cannot be adequately described in the ODE framework. A preliminary analysis demonstrates that those delays essentially affect both the transition dynamics after a change of $\beta(t)$ and the asymptotic behavior. The distributed delayed nature of the infectiveness process becomes critical for adequate modeling when the transmission rate $\beta(t)$ sharply changes.

Integral and integrodifferential equations are more natural and flexible for the description and control of epidemics. Study of the integral epidemiologic models with delays has mostly focused on model stability and bifurcations, see, e.g., [16], [22], [31]. Relations between integral and ODE-based biological models have been studied for the past fifty years, but still have many open issues. Specifically, the existing integral models (described in Section 4 and similar ones) are not perfect for the purposes of COVID control.

The major required modification of the integral models for COVID-19 is to assume the *finite delay duration* (a finite length of *process prehistory*). In reality, all epidemic stages (classes) have a finite length that depends on the nature of disease. Ignoring this fact in both ways (assuming *no memory* or the *infinite memory*) makes models less accurate. The *finite memory* of processes leads to new phenomena in modeled dynamics. The *integral dynamic models with finite memory* have been successfully used in many applications [24], [25], [26] and can bring new possibilities to control COVID-19 mitigation and economy. However, the construction of such models is out of the scope of this paper and is an interesting topic for prospective research.

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Authors

Natali Hritonenko,
Department of Mathematics,
Prairie View A &M University,
Prairie View, TX 77446, United States,
E-mail: nahritonenko@pvamu.edu

Victoria Hritonenko,
Dept. of Sciences, Mathematics & Biotechnology,
UC Berkeley, CA 77030, United States,
E-mail: hritonenko@berkeley.edu

Yuri Yatsenko,
Dunham College of Business,
Houston Baptist University,
Houston, TX 77074, United States,
E-mail: yyatsenko@hbu.edu