## A discrete-time infectious disease model for global pandemics

Abdul-Aziz Yakubu<sup>a,1</sup>

The ongoing global pandemic of coronavirus (COVID-19), an infectious disease caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has raised concerns about the effectiveness of current preventive pharmaceutical and nonpharmaceutical interventions (1). In addition, the upward global trends in the numbers of emerging and reemerging infectious diseases, as evidenced by the reported cases of Ebola, Zika, Chikungunya, SARS, West Nile virus, and other serious infections, have dramatically expanded the demand for mathematical models of infectious diseases across multiple entities that include the pharmaceutical industry, health and medical organizations, and local and international governments, and that span the public and private sectors (2, 3). With this increased demand comes the opportunity to meaningfully reassess the variety of existing mathematical epidemic models. Such an assessment is an important step in capturing the ways in which these models contribute to the understanding of infectious disease surveillance data and the policies, programs, and practices that emerge from these data (1).

Mathematical models of infectious diseases are powerful tools that are used in extending societal understanding and forecasting of disease transmission dynamics and for evaluating the effects of different interventions and changing on-the-ground conditions for epidemiological outcomes. Thus, it is important that we make use of the full range of the available models and disease data to study disease dynamics. Mathematical models can be classified based on how they model variability, chance and uncertainty, time, space, and the structure of the population. On the data side, disease surveillance data are reported at discrete time intervals, for example, daily, weekly, monthly, or yearly disease incidence or number of disease-induced deaths (1). However, many of the existing infectious disease models are continuous-time models that implicitly assume the availability of a continuous stream of these data. While these models have produced useful information, insights, and interventions, it may be worthwhile to consider discrete-time infectious disease models and other models that are more closely aligned with the discrete nature of disease surveillance data.

The discrete-time version of the Kermack-McKendrick model, a system of difference equations introduced in ref. 1, is more compatible with the data that are available to the modeling community. As a result, parameters of the model can be related directly to disease surveillance data without additional model assumptions. Furthermore, the discrete-time model in ref. 1 is very easy to implement computationally. To investigate the factors that determine both magnitude of the "bell-shaped geometry" associated with most disease epidemics and their termination within a given population (see Fig. 2), in 1927, Kermack-McKendrick introduced an age-of-infection model (4), that is, a model in which the infectivity of an individual depends on the time since the individual became infective. To describe the classic Kermack-McKendrick model, we consider a population that is partitioned into the following three nonintersecting classes by an infectious disease: the class of susceptible individuals or susceptibles (S), infected individuals or infectious (I), and recovered individuals or removed (R) (4, 5). Fig. 1 is the flowchart of the Kermack-McKendrick SIR epidemic model. Childhood diseases, such as chickenpox, smallpox, rubella, and mumps, are modeled by SIR disease models. To account for how the number of individuals in each of the three classes changes continuously, the classic Kermack-McKendrick model was formulated as an initial value problem for a

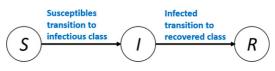


Fig. 1. Flowchart for the classic Kermack–McKendrick SIR model.

<sup>a</sup>Department of Mathematics, Howard University, Washington, DC 20059

Author contributions: A.-A.Y. wrote the paper.

The author declares no competing interest.

Published under the PNAS license.

See companion article, "The discrete-time Kermack–McKendrick model: A versatile and computationally attractive framework for modeling epidemics," 10.1073/pnas.2106332118.

<sup>1</sup>Email: ayakubu@howard.edu.

Published October 8, 2021. PNAS 2021 Vol. 118 No. 42 e2116845118

on November 30.

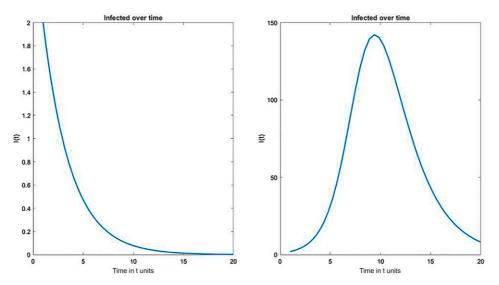


Fig. 2. Left shows prevalence decreasing monotonically. Right shows increasing prevalence to a peak before decreasing monotonically.

system of continuous-time ordinary differential equations (Newtonian derivative). The deterministic continuous-time SIR model framework was a significant milestone in the development of subsequent infectious disease models.

The process of model construction and parametrization requires model type choices and assumptions, and the Kermack-McKendrick model is based on the following three assumptions: 1) There are no births and deaths. 2) There are no emigrations and migrations. 3) All recovered individuals have complete immunity and cannot get reinfected. These assumptions are not universal, and thus the Kermack-McKendrick model has a distinctive asymptotic dynamics. In epidemiology, threshold conditions for the occurrence of disease outbreaks are common. The Kermack-McKendrick model predicts that disease prevalence decreases monotonically in the population when the model's epidemic threshold parameter value is smaller than one. However, when the epidemic threshold parameter value is bigger than one, the model predicts the occurrence of a sudden increase in disease prevalence to a peak which is followed by a continuous decline. This "bellshaped geometry" is a signature for classic disease epidemics (Fig. 2) (4, 5).

The discrete-time version of the Kermack–McKendrick model has several additional advantages. These include replication of the "bell-shaped geometry" of classic epidemic events (1). Furthermore, discrete-time infectious disease models are especially appealing for the mathematical description of a disease epidemic process, since such a process can be conceptualized as evolving through a set of discrete-time disease events. Notwithstanding its advantages, there is room for the inclusion of

https://doi.org/10.1073/pnas.2116845118

additional relevant real-world features that would help us control and prevent disease infections. For example, modelers have used the Kermack-McKendrick model to study influenza and other diseases; however, the model does not account for disease intervention protocols such as vaccination, social distancing, and mask wearing (6, 7). Furthermore, the rapid global spread of COVID-19 currently underway is merely the latest example in a long line of diseases that have been transported from their endemic region and have touched off epidemics in new populations (3, 8). Thus, adding relevant features to the more flexible and easier to implement computationally discretetime version of the Kermack-McKendrick model framework could potentially overcome features of this model that limit our capacity to control and prevent global disease pandemics. Specifically, such model extensions could include social and demographic characteristics of populations, including age, gender, race, geographical locations, and so on. These modified models and their extensions could potentially increase the precision of our model predictions in the important work of informing public health preparedness strategies for future large-scale disease outbreaks and the ongoing global COVID-19 pandemic. Dynamic compartmental models, such as the classic Kermack-McKendrick model and the discrete-time version, are also able to account for the indirect protection of nonvaccinated susceptibles against disease infections (9, 10). The ensemble of data-driven models, such as discrete-time infectious disease models or models with time steps that can be adjusted to the time interval between real disease data points, are well positioned to capture and reflect the actual disease trajectories in populations.

2 of 3 | PNAS



<sup>1</sup> O. Diekmann, H. G. Othmer, R. Planqué, M. C. J. Bootsma, The discrete-time Kermack–McKendrick model: A versatile and computationally attractive framework for modeling epidemics. Proc. Natl. Acad. Sci. U.S.A. 118, e2106332118 (2021).

<sup>2</sup> K. E. Jones et al., Global trends in emerging infectious diseases. Nature 451, 990–993 (2008).

<sup>3</sup> N. Siewe, S. Lenhart, A.-A. Yakubu, Ebola outbreaks and international travel restrictions: Case studies of Central and West Africa regions. J. Biol. Syst. 28, 431–452 (2020).

<sup>4</sup> W. O. Kermack, A. G. McKendrick, A contribution to the mathematical theory of epidemics. Proc. R. Soc. Lond. A Contain. Pap. Math. Phys. Character 115, 700–721 (1927).

<sup>5</sup> M. Martcheva, An Introduction to Mathematical Epidemiology (Texts in Applied Mathematics, Springer, New York, 2015), vol. 61.

<sup>6</sup> A.-A. Yakubu, "Introduction to discrete-time epidemic models" in Modeling Paradigms and Analysis of Disease Transmission Models, A. B. Gumel, S. Lenhart, Eds. (DIMACS Series in Discrete Mathematics and Theoretical Computer Science, American Mathematical Society, 2010), vol. 75, pp. 83–109.

7 W. H. Hamer, Epidemic disease in England. Lancet 1, 733–739 (1906).

- 8 Z. Feng, A. N. Hill, A. T. Curns, J. W. Glasser, Evaluating targeted interventions via meta-population models with multi-level mixing. *Math. Biosci.* 287, 93–104 (2017).
- 9 W. Hethcote, The mathematics of infectious diseases. SIAM Rev. 42, 599-653 (2000).
- 10 O. Diekmann, H. Heesterbeek, T. Britton, Mathematical Tools for Understanding Infectious Disease Dynamics (Princeton University Press, Princeton, NJ, 2013).

