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# Mathematical Epidemiological Models: A Comprehensive Review of Classical, Extended, Network, Spatial and Frractional Approaches

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**Abstract**

A methodical framework for characterizing, evaluating, and predicting the spread of infectious diseases is provided by mathematical epidemiological models. In addition to discussing the historical origins of epidemic modeling, this work presents popular models including SIS, SIR, SIRS, SEIR, SEIRD, SEIRV, and SEAIHRD formulations in a logical manner. We outline the mathematical formulas, compartmental organization, interpretation, and common applications for each model. We also examine reaction-diffusion (spatial) models that represent geographic dissemination and network-based models that capture numerous interactions. Particular attention is paid to the basic reproduction number $R_0$ and its computation utilizing the next-generation matrix technique.

*Keywords:* Epidemic models, basic reproduction number, fractional order, analytical epidemiology

## 1. Introduction

Infectious diseases have long been the leading cause of death worldwide. With the development of hygiene and urban sanitation, thanks also to the advent of anti-infectives and vaccination programs, they have gradually lost ground in developed societies. This victorious fight against infections had, by the end of the 1960s, convinced many officials that "the chapter of infectious diseases was closed" [1]. In 1980, the success of the global smallpox eradication program allowed the WHO to declare the disease eradicated. Throughout the 20th century, we observe in industrialized countries a significant drop in mortality caused by infectious diseases and an increase in life expectancy, which continues [2].

Mathematical modelling is sometimes the only possible approach to explore different scenarios. This step constitutes a real public health tool. It allows testing, without loss of time or cost, the control measures that are considered: preventive measures, isolation of patients, treatments, vaccinations,... The model is nevertheless not reality and is not supposed to reproduce it in full. It must best reproduce the characteristics of the phenomenon studied according to the objectives set for the study framework. Modeling then involves applying mathematical tools to a fragment of reality. The modeling step turns out to be the most delicate, the longest and often the most perilous. Indeed, it is necessary to understand the real problem well in order to try to propose a suitable model. If this step is neglected or omitted, if the constraints are not well placed, then we end up with a mathematical formulation that does not correspond to the problem. The resolution of the mathematical problem then provides a solution not adapted to the concrete problem. Finally, if the problem is well posed, the next step consists in solving it, that is to say, analyzing the model with the aim of understanding, predicting and acting.

The first mathematical models describing an infectious disease date back to Bernoulli in 1766, he considers the mortality induced by small pox (smallpox) and the effects of inoculation, which had been discovered at the time [3]. International literature often identifies as "founding father of epidemiology" a Frenchman, Pierre Charles Louis who created the "digital method" in medicine. Another "founding father" was the Englishman William Farr (1807-1883), a student of Pierre C. Louis, who specified, as early as 1838 (Farr, 1838), the notion of risk, and showed the importance of longitudinal analyses ("cohorts") to evaluate risks. He received in 1839 the task of collecting morbidity data systematically and is considered as the founder of epidemiological surveillance [4]. The historical roots of social epidemiology are also French, with the demonstration by Louis René Villermé in 1826 that the greatest mortality observed in certain neighborhoods was more explained by the poverty of its inhabitants than by "insalubrity" of their homes which was generally the cause mentioned [5, 6].

Since the very beginning of epidemic modeling, the basic elements for the description of infectious diseases have been the three epidemiological classes: susceptible, infected and withdrawn, which are often denoted by:

- S: individuals who are healthy and can be infected.
- I: individuals who are infected and capable of transmitting the disease.
- R: individuals who are safe, because either they have been infected and now cured, or they are vaccinated.

A fundamental distinction can be made between these diseases, those that confer lifelong immunity and those that do not. The first case leads to SIR type models, and the second to SIS models [7].

Recently, the emergence of complex epidemics such as COVID-19 has underlined the importance of adding exact clinical and epidemiological aspects in mathematical models. High-resolution compartmental models, such as the SEAIHRD framework, clearly distinguish between asymptomatic and symptomatic infections, hospitalization, recovery, and death. These models are particularly useful for healthcare planning and assessing the impact of nonpharmaceutical actions on hospital capacity constraints [8].

In addition to these findings, researchers revealed that typical compartmental models rely on the premise of homogeneous mixing, which indicates that all persons are equally likely to interact with one another. In reality, social relationships are organized and diversified. To overcome this limitation, network-based epidemiological models were created, in which individuals are represented as nodes in a graph and contacts as edges. These models take into account clustering, superspreading events, and how network topology affects epidemic thresholds. Similarly, reaction-diffusion and spatial models were developed to account for the global spread of infectious diseases, combining local transmission dynamics with spatial mobility and diffusion.

The goal of this research is to provide a comprehensive and unified examination of mathematical epidemiological models, ranging from traditional compartmental systems to more contemporary network-based and spatial formulations. We describe each model's compartmental organization, mathematical equations, epidemiological motivation. The role and computation of the basic reproduction number $R_0$ are discussed. This review, which combines historical context and mathematical formulation, is intended to serve as a resource for graduate students and researchers new to the field of mathematical epidemiology, as well as practitioners looking for appropriate modeling tools for practical applications.

## 2. Historical Background

In 1766, Bernoulli created the first mathematical models to describe an infectious disease. He took into account the mortality caused by smallpox and the effects of vaccination, which were then known [3]. Pierre Charles Louis, a Frenchman who invented the "digital method" in medicine, is frequently referred to as the "founding father of epidemiology" in international literature. The Englishman William Farr (1807–1883), a pupil of Pierre C. Louis, was another "founding father" who defined the concept of risk as early as 1838 (Farr, 1838) and demonstrated the value of longitudinal analyses ("cohorts") to assess hazards. He is regarded as the father of epidemiological surveillance and was given the task of methodically gathering morbidity data in 1839. The origins of social epidemiology can also be traced back to France, where Louis René Villermé demonstrated in 1826 that the highest mortality rates found in some neighborhoods could be better explained by the poverty of their residents rather than the "insalubrity" of their homes, which was typically cited [4].

The beginnings of "analytical epidemiology", in which risk factors for diseases are sought from observations made on populations, date back to the mid-19th century, when the English society of epidemiology was founded (1850). Three seminal works are usually cited: that of Panum, who in 1846 studied the dynamics of measles in the Faeroe Islands, identified the direct person-to-person mode of transmission and provided an estimate of the incubation time; that of Snow, who in 1854, conducted an epidemiological study during the cholera epidemic in London, at the end of which, thanks to the comparison of the frequencies of the disease in neighborhoods served by different water networks, he concludes that there was a transmissible agent at the origin of cholera and that it was carried by water [9].

At about the same time, in 1847, Semmelweis in Vienna discovered that puerperal fever was a disease carried, in the absence of proper hygiene, by the hands of women's caregivers during their deliveries. These three discoveries have in common that they have directly led to practical applications in public health. In addition, they open the series of epidemiological discoveries made before the discovery of the corresponding biological mechanisms; it was indeed only 20 years later that Pasteur highlighted the existence of explanatory infectious agents of communicable diseases [10].

At the beginning of the 20th century, Sir Ronald Ross, discoverer of plasmodium falciparum, a parasite causing malaria, published a series of works using mathematical models to study the spread and control of malaria. In 1911, he gave the first mathematical model of malaria transmission [11].

Since then, mathematical modeling has become an essential tool in the analysis of infectious disease dynamics. Indeed, Ross will use the above model to show that in order to eradicate malaria, it is enough to reduce the amount of infectious mosquitoes below a certain threshold. It is the birth of what we now call mathematical epidemiology, the theory of happenings or even pathometry.

Later, Alfred Lotka (1925) and Vito Volterra (1926) independently proposed the predator-prey model, or the Lotka-Volterra model (cited in the second part), which today plays a decisive role in population dynamics and is considered as a basic model. Let us also recall that it was Lotka who, in 1923, made an exhaustive mathematical study of Ross's model [12].

- A.G. McKendrick was a British army military doctor. He served under Ronald Ross in 1901 in Sierra Leone during an anti-malaria campaign.

- Ross encouraged the young McKendrick to apply mathematical techniques to medical problems.

In 1911, Ross wrote to McKendrick:

We will eventually establish a new science.
But first of all you and I must open the door,
so anyone can then enter if they wish.

In 1927, W. O. Kermack and A. G. McKendrick applied the ideas of Ronald Ross to study the dynamics of human infectious disease transmission. More precisely, Kermack and Mckendrick applied Ross's ideas to diseases whose transmission dynamics depend on the frequency and intensity of interactions between susceptible (healthy) individuals and infected and infectious individuals. Their fundamental result published in 1927 continues to play, like the Lotka-Volterra model in population dynamics, a central role in the mathematical theory of infectious diseases. Noting S the population of susceptible, I that of infected and by R the population of cured or "removed/recovered" [11, 13, 14].

## 3. Basic Notation and Modeling Assumptions

$S(t)$ stands for susceptible people, $E(t)$ for exposed (infected but not infectious), $I(t)$ for infectious (symptomatic), $H(t)$ for hospitalized, $R(t)$ for recovered, $D(t)$ for dead, and $V(t)$ for immunized. $\beta$ (transmission rate), $\gamma$ (recovery rate), $\sigma$ (progression rate from exposed to infectious), $\alpha$ (disease-induced mortality), $\omega$ (waning immunity rate), $v$ (vaccination rate), $h$ (hospitalization rate), $\kappa$ (relative infectiousness of asymptomatic individuals), and $p$ (fraction progressing to asymptomatic infection) are examples of typical parameters.

## 4. Classical Compartmental Models

### 4.1. SIS Model

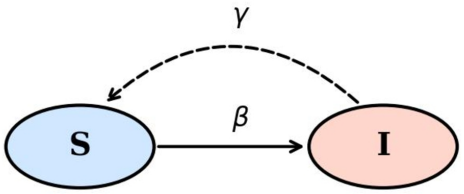


Figure 1. SIS compartmental diagram

The SI model is a rudimentary epidemiological model with no known impact in mathematical modeling of the spread of an epidemic. It is limited to a simple logic: the only observable event is the infection of a susceptible individual. To obtain the system of differential equations mathematically representing the SI model, we reason in relation to the flow of individuals entering or leaving each compartment $S$ and $I$.

The equations for the SIS model are as follows:

$$\frac{dS}{dt} = -\beta SI + \gamma I,$$
$$\frac{dI}{dt} = \beta SI - \gamma I.$$

with $S + I = N$ (constant population).

Typical applications include bacterial infections and some sexually transmitted infections where reinfection is common.

### 4.2. SIR Model

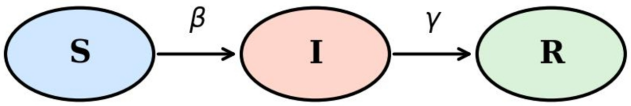


Figure 2. SIR compartmental diagram

Chronologically, the SIR model for Susceptible-Infected-Recovered (Removed in Anglo-Saxon appellation) is a precursor and classic model in epidemiological modeling. It is one of the simplest mathematical models of disease spread. In the presence of an epidemic, the SIR model recommends dividing the population into three (3) basic categories or compartments based on the state of the disease. This model is not absolute but relies on simplifying assumptions but no less realistic in the epidemiological context. One of these assumptions is to assume that any individual or group of individuals are vulnerable and susceptible to contracting the disease. They are potentially contaminable but not symptomatic. In other words, there is no innate immunity. This category of individuals forms the compartment $S$ of susceptible.

The SIR model's dynamics are governed by the following differential equations:

$$\begin{aligned} \frac{dS}{dt} &= -\beta SI, \\ \frac{dI}{dt} &= \beta SI - \gamma I, \\ \frac{dR}{dt} &= \gamma I. \end{aligned}$$

with $S + I + R = N$.

Key insight: the epidemic grows when $S$ is above a threshold and declines once susceptible depletion reduces effective transmission.

### 4.3. SIRS Model

**Motivation:** The SIRS model includes waning immunity; recovered individuals become susceptible again.

The SISR model is a variant of the SIR model, where an individual who has been infected can become susceptible again. It involves three processes: A first in which a susceptible individual becomes infected upon contact with a sick individual, with a transmission rate $\beta$; a second where an infected individual recovers (with immunization) spontaneously, with a cure rate $\gamma$, and a last one where an infected individual can spontaneously return to the class of susceptible, with a certain rate $\omega$.

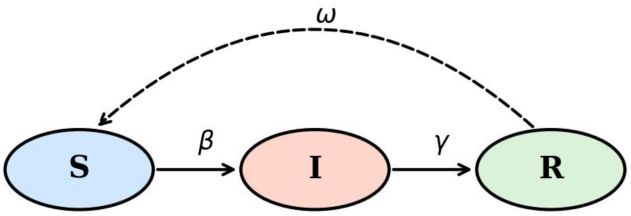


Figure 3. SIRS compartmental diagram (dashed arrow indicates immunity loss)

The evolution equations of this model are given by the following system:

$$\begin{aligned}\frac{dS}{dt} &= -\beta SI + \omega R,\\ \frac{dI}{dt} &= \beta SI - \gamma I,\\ \frac{dR}{dt} &= \gamma I - \omega R.\end{aligned}$$

Applications include respiratory viruses with partial/temporary immunity (e.g., seasonal influenza-like dynamics).

## 5. Models with Latency, Mortality, and Vaccination

### 5.1. SEIR Model

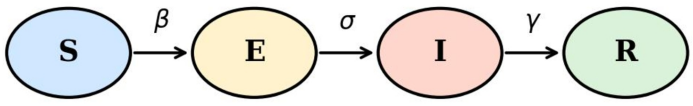


Figure 4. SEIR compartmental diagram

The two main classes of individuals are susceptible and infected persons, but other classes may also be present. For example, in this model, in addition to susceptible people, infected and recovered, another class is present, it is the class of exposed individuals, whose individuals are not contagious, which allows taking into account the incubation duration.

By designating the number of susceptible, exposed, infected and cured individuals as $S$, $E$,

$I$ and $R$, respectively, then the evolution equations for these variables are:

$$\begin{aligned}
\frac{dS}{dt} &= -\beta SI, \\
\frac{dE}{dt} &= \beta SI - \sigma E, \\
\frac{dI}{dt} &= \sigma E - \gamma I, \\
\frac{dR}{dt} &= \gamma I.
\end{aligned}$$

Applications include diseases with a meaningful incubation period (e.g., measles, Ebola, COVID-19).

### 5.2. SEIRD Model

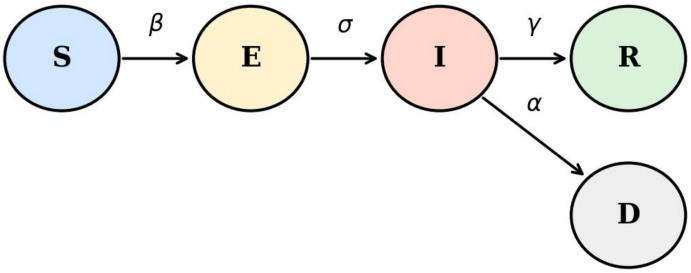


Figure 5. SEIRD compartmental diagram

The initials match: Susceptible-Infected-Exposed-Recovered-Dead. It is a predictive model used to study and predict the evolution of a virus. Based on different differential equations all linked to each other by the intervention of different variables, the model is then adapted according to these variables. There are variables that allow setting up the environment in which we will study the evolution of the epidemic: the population, the initial number of infected and the number of deaths.

The equations for the SEIRD model are as follows:

$$\begin{aligned}
\frac{dS}{dt} &= -\beta SI, \\
\frac{dE}{dt} &= \beta SI - \sigma E, \\
\frac{dI}{dt} &= \sigma E - (\gamma + \alpha) I, \\
\frac{dR}{dt} &= \gamma I, \\
\frac{dD}{dt} &= \alpha I.
\end{aligned}$$

Applications include high-fatality outbreaks where mortality reporting is central to calibration and planning.

### 5.3. SEIRV Model

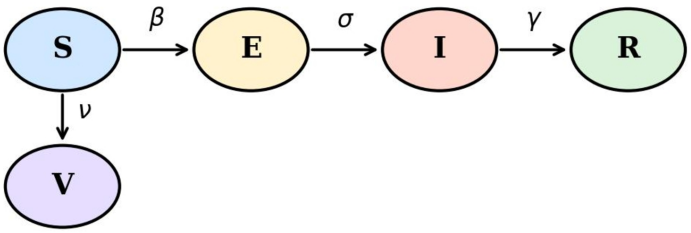


Figure 6. SEIRV compartmental diagram

A thorough framework for researching disease dynamics is provided by the SEIRV model, which has sections for susceptible, exposed, infected, recovered, and vaccinated individuals. The COVID-19 pandemic also made use of these models.

Model equations (one common form):

$$\begin{aligned}
\frac{dS}{dt} &= -\beta SI - vS, \\
\frac{dV}{dt} &= vS, \\
\frac{dE}{dt} &= \beta SI - \sigma E, \\
\frac{dI}{dt} &= \sigma E - \gamma I, \\
\frac{dR}{dt} &= \gamma I.
\end{aligned}$$

### 5.4. SEAIHRD Model

The SEAIHRD model captures asymptomatic infection, hospitalization, and mortality–features that are essential for many modern epidemics (e.g., COVID-19) and for health-system planning.

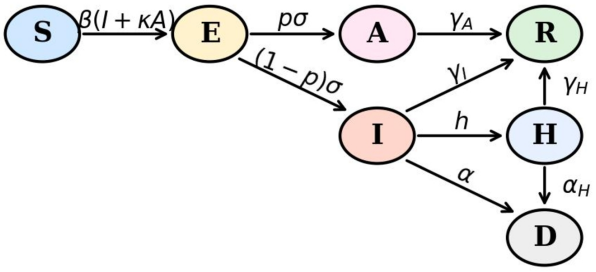


Figure 7. SEAIHRD compartmental diagram for COVID-19

Model equations (general form):

$$\begin{aligned}
\frac{dS}{dt} &= -\beta S(I + \kappa A),\\
\frac{dE}{dt} &= \beta S(I + \kappa A) - \sigma E,\\
\frac{dA}{dt} &= p\sigma E - \gamma_A A,\\
\frac{dI}{dt} &= (1-p)\sigma E - (\gamma_I + h + \alpha)I,\\
\frac{dH}{dt} &= hI - (\gamma_H + \alpha_H)H,\\
\frac{dR}{dt} &= \gamma_A A + \gamma_I I + \gamma_H H,\\
\frac{dD}{dt} &= \alpha I + \alpha_H H.
\end{aligned}$$

Here $\kappa$ scales the infectiousness of asymptomatic individuals relative to symptomatic ones; $p$ is the fraction progressing to asymptomatic infection; $h$ is the hospitalization rate; $\alpha$ and $\alpha_H$ are disease-induced death rates for $I$ and $H$, respectively.

## 6. Network-Based Epidemiological Models

**Motivation:** Classical compartmental models assume homogeneous mixing (each individual contacts every other with equal probability), which may be unrealistic. Network models represent individuals as nodes and potentially infectious contacts as edges, capturing heterogeneity, clustering, and superspreading.

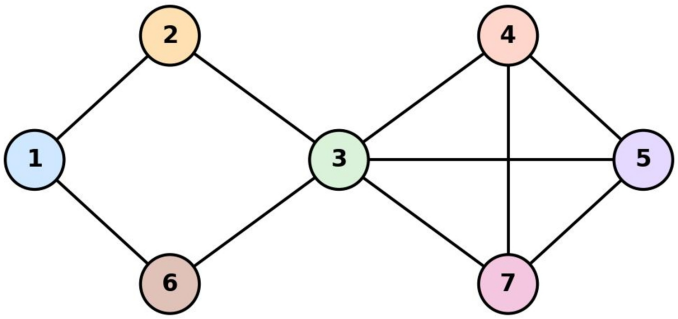


Figure 8. Conceptual network used to represent heterogeneous contact patterns

A common node-level SIS/SIR-type formulation is:

$$\frac{dI_i}{dt} = \beta(1 - I_i)\sum_j A_{ij} I_j - \gamma I_i,$$

where $A_{ij}$ is the adjacency matrix encoding contacts.

Network structure can strongly influence invasion conditions and outbreak size; for many network models, thresholds depend on spectral properties of $A$ [15].

# 7. Spatial (geographical) spread of an epidemic

## 7.1. The phenomenon of diffusion

Diffusion refers to the action that an expansive agent must spread through a favorable environment and reach a state or uniform distribution. The phenomenon of diffusion is found in several fields ranging from chemistry, telecommunications, information sciences, material sciences to biological and epidemiological sciences. In Nature, diffusion refers to the tendency towards spatial spreading of particles, atoms or molecules through an energetic excitation. Epidemics are characterized by their widespread spread from a geographical point of view and reach several continents.

In the case of the spread of epidemics, two important factors describe the phenomenon of disease diffusion: territoriality or diffusion environment involving the geometry and size of the latter (concepts that are also found in solving the diffusion equations) and the fundamental mode of diffusion in the spread of diseases.

## 7.2. Spatial models with diffusion

Reaction diffusion equations are one of the most important mathematical tools for modeling spatial movements. These can be continuous in space and time. They are represented by second-order partial differential equations. The feedback diffusion equations allow for better modeling of the spatial propagation resulting from a diffusion process. This type of equation consists of two parts: - a diffusion component represented by the second derivative of the unknown function (or functions) and a reaction part.

## 7.3. Migratory phenomenon and metapopulation

In addition to the inevitable epidemiological situations of susceptible population and infected population, spatio-temporal propagation models are governed by two components: a diffusion component that embodies spatial dynamics, a dissemination component related to the nature of migration from one point to another. This concept is based on the notion of metapopulation. Indeed, a given population is socially subdivided into a certain number of subsets or sub-populations called “patches”. On a reduced scale, a patch responds to the principle of compartmentalization distributed between “Susceptible”, “Infected” and “Recovered”. However in the case of patches, the structure becomes multi-compartment

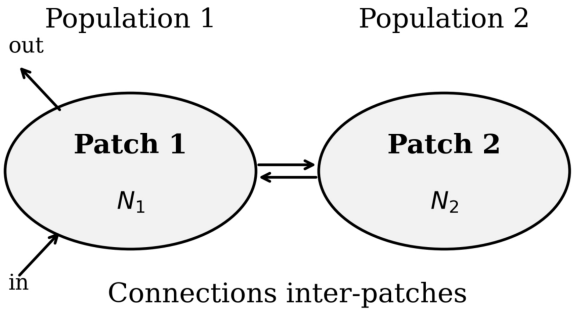


Figure 9. A type of organization in metapopulation

### 7.4. Implementation of the reaction-diffusion equation

The literature offers different approaches in implementing the feedback diffusion equation. It is developed below a rather simple discrete approach applied to a one-dimensional directional movement illustrated by the following diagram relative to the variable $x$ provided with a discretization step $\Delta t$ and the time variable $t$ provided with a temporal discretization step $\Delta t$.

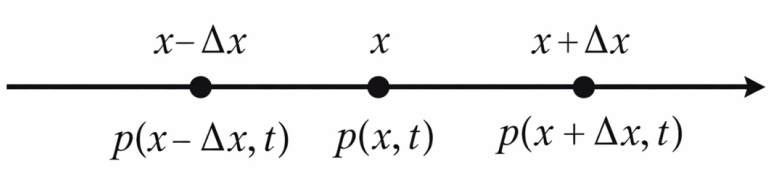


Figure 10. Conceptual spatial patch / diffusion structure

On the axis of the movement path, we set three positions of the mobile agent respectively at points $x$, $x - \Delta x$ and $x + \Delta x$. We define the following probabilities

- $p(x,t)$: probability that an individual is present at position $x$ at time $t$.
- $p(x,t+\Delta t)$: probability that an individual is present at position $x$ at time $t+\Delta t$.
- $p(x-\Delta x,t)$: probability that an individual is present at position $x-\Delta x$ at time $t$.
- $p(x+\Delta x,t)$: probability that an individual is present at position $x+\Delta x$ at time $t$.

A reaction–diffusion SIR model may be written as:

$$\begin{aligned}\frac{\partial S}{\partial t} &= D_S \Delta S - \beta SI,\\ \frac{\partial I}{\partial t} &= D_I \Delta I + \beta SI - \gamma I,\\ \frac{\partial R}{\partial t} &= D_R \Delta R + \gamma I,\end{aligned}$$

where $\Delta$ is the Laplacian operator and $D_S$, $D_I$, $D_R$ are diffusion coefficients.

These models can produce traveling-wave solutions and allow analysis of spatial heterogeneity, hotspots, and the impact of movement restrictions.

## 8. Fractional-Order Epidemic Models

Classical epidemiological models are often expressed as integer-order ordinary differential equations, which implicitly assume that the rate of change in each compartment is determined solely by the system's current state. While this assumption is mathematically convenient, it may not fully convey the complexities of real-world epidemic processes. Many infectious diseases have transmission dynamics that include memory effects, varied contact patterns across time, and nonlocal temporal behavior that cannot be properly captured by integer-order derivatives.

To solve these constraints, fractional-order epidemic models have been proposed as a natural development of classical models. In these models, the first-order time derivative is substituted by a fractional derivative of order $0 < \alpha \leq 1$. Fractional derivatives incorporate memory by allowing the system's evolution to be based on its whole history, with a weighting that typically follows a power-law kernel. This trait makes fractional models ideal for explaining processes in which previous states influence current dynamics, such as varied infectious durations, delayed behavioral reactions, and long-term immune effects.

The Caputo fractional derivative is the most widely employed in epidemiological modeling due to its compliance with classical beginning conditions. For a smooth function $f(t)$, the Caputo derivative of order $\alpha$ is defined as

$$^{C}D_t^{\alpha} f(t) = \frac{1}{\Gamma(1-\alpha)} \int_0^t \frac{f(s)}{(t-s)^{\alpha}} ds, \quad 0 < \alpha < 1,$$

where $\Gamma(\cdot)$ denoted the Gamma function. When $\alpha = 1$, the classical integer-order derivative is recovered.

A fractional-order SIR model, for example, can be written as

$$\begin{aligned} ^{C}D_t^{\alpha} DS(t) &= -\beta S(t) I(t), \\ ^{C}D_t^{\alpha} DI(t) &= \beta S(t) I(t) - \gamma I(t), \\ ^{C}D_t^{\alpha} DR(t) &= \gamma I(t), \end{aligned}$$

where the parameters $\beta$ and $\gamma$ retain their classical epidemiological interpretation. The fractional order $\alpha$ controls the strength of memory effects in the system. Smaller values of $\alpha$ correspond to stronger memory and slower system response, often leading to a smoother epidemic curve and delayed peaks compared to the integer-order case.

Fractional epidemic models have been proven to provide better data fitting for certain infectious diseases, especially when epidemic curves show sub-exponential growth or long-tail decay. These characteristics are frequently seen in real-world outbreaks, where behavioral changes, varying contact rates, and intervention methods cause memory and persistence effects. As a result, fractional models can provide a more accurate depiction of epidemic dynamics than classical models.

The analysis of fractional-order epidemiological models has more mathematical hurdles than integer-order systems. Standard approaches from ordinary differential equations, such as phase-plane analysis and classical stability theory, must be modified for the fractional context. The stability of equilibria is often explored using fractional versions of Lyapunov methods. The requirement for asymptotic stability depends on both the eigenvalues of the Jacobian matrix and the fractional order $\alpha$. In particular, a disease-free equilibrium that is stable in the integer-order model may show distinct convergence qualities in the fractional case.

The formulation and interpretation of the basic reproduction number $R_0$ in fractional-order models is still an active research topic. In many research, $R_0$ is derived using the same next-generation matrix structure as integer-order models, with the expectation that $R_0$ will continue to operate as a disease invasion threshold.

Fractional-order models have been used to study a number of infectious diseases, such as influenza, COVID-19, HIV, and tuberculosis. Because of its flexibility, researchers can include more complexity in a single mathematical framework, such as time-varying transmission rates, spatial affects, and control measures. Despite their advantages, fractional models have drawbacks that need to be carefully considered in real-world research, including problems with parameter estimate, computational cost, and biological interpretation of the fractional order.

In conclusion, by incorporating memory and nonlocal temporal effects, fractional-order epidemic models expand upon traditional compartmental frameworks. They offer a potent alternative for modeling complex epidemic dynamics, particularly when conventional integer-order models are unable to capture features of observed data. Fractional modeling is expected to become more important in mathematical epidemiology as theoretical knowledge and computational capabilities advance.

## 9. The Basic Reproduction Number $R_0$

**Definition 1.** *The basic reproduction number $R_0$ is the expected number of secondary infections caused by a typical infectious individual introduced into a wholly susceptible population. It acts as a threshold parameter: if $R_0 > 1$ the disease can invade; if $R_0 < 1$ the disease tends to die out [16].*

### 9.1. R0 for Simple Models

For the classical SIR model (with mass-action incidence), $R_0 = \frac{\beta}{\gamma}$ (after appropriate normalization by population size if needed).

For the SIS model, $R_0$ similarly compares infection and recovery rates and determines persistence.

### 9.2. Next-Generation Matrix Method (NGM) [17]

For multi-compartment models, $R_0$ is most systematically computed using the next-generation matrix method. Let x collect the infected-state variables (e.g., $E$ and $I$, or $E$, $A$, $I$, $H$). Write the infected subsystem as:

$$\frac{dx}{dt} = F(x) - V(x),$$

where $F(x)$ contains terms corresponding to NEW infections entering infected compartments and $V(x)$ contains transfers between infected compartments and removals (recovery, death, etc.).

Step 1: Find the disease-free equilibrium (DFE), typically with $I = 0$ (and $E = 0$, $A = 0$, $H = 0$) and $S = S*$ (often $S* = N$).

Step 2: Compute the Jacobians at the DFE:

$$F = \left.\frac{\partial F}{\partial x}\right|_{DFE} \text{ and } V = \left.\frac{\partial V}{\partial x}\right|_{DFE}.$$

Step 3: Form the next-generation matrix $K = FV^{-1}$. Then:

$$R_0 = \rho(K),$$

where $\rho(\cdot)$ is the spectral radius (dominant eigenvalue magnitude). This approach generalizes across SEIR-type models, vaccination models, and many network-structured formulations.

### 9.3. Example: SEIR Model via NGM

For SEIR, infected compartments are $(E, I)$. New infections enter $E$ only. At the DFE, $S* = N$. One obtains:

$$F = \begin{pmatrix} 0 & \beta S^* \\ 0 & 0 \end{pmatrix}, V = \begin{pmatrix} \sigma & 0 \\ -\sigma & \gamma \end{pmatrix}.$$

Hence $K = FV^{-1}$ and $R_0$ reduces to: $R_0 = \frac{\beta S^*}{\gamma}$ (under standard assumptions), with $S^*$ normalized appropriately depending on the incidence formulation (mass action vs. standard incidence).

## 10. Discussion and Model Selection

Model choice depends on disease biology and data availability. SIS is appropriate when immunity is absent; SIR when immunity is long-lasting; SIRS when reinfection is possible. SEIR-type models are essential when incubation is non-negligible. SEIRD is used when mortality is a key outcome. SEIRV supports vaccination policy analysis. SEAIHRD is suitable when asymptomatic transmission and hospitalization are important for planning and calibration. Network models are recommended when contact heterogeneity is central, while reaction–diffusion models are used for spatial propagation.

## 11. Conclusion

This survey provided mathematical formulations, compartmental diagrams, and interpretation for a broad family of epidemic models. Across frameworks, the reproduction number $R_0$ remains the central threshold concept and can be computed systematically using the next-generation matrix method. The integrated presentation is intended to support doctoral-level research and model selection for real applications.

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