

# Thinking About the Basic Reproduction Number

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In epidemiology, the basic reproduction number,  $R_0$ , also called the basic reproduction ratio, is the expected number of secondary infections produced by a typical infected individual throughout its period of infectiousness in a completely susceptible population [Diekmann et al., 2010].  $R_0$  serves as a threshold condition for epidemics, and is often used as a summary statistic to estimate the true infection rate  $\beta$ .

As a motivating example, early  $R_0$  estimates in certain countries for the COVID-19 pandemic were in the range of 2.0 – 6.5 [Vegvari et al., 2022]. Because values of  $|R_0| > 1$  imply epidemic-level transmission, these estimates played an important role in informing public health responses. In particular, projections based on early  $R_0$  estimates were frequently cited in support of non-pharmaceutical interventions (as this was prior to the development of the vaccine), including travel restrictions and mobility controls, as part of efforts to reduce transmission and prevent healthcare system overload.

A number of recent observational studies and commentaries emphasize that  $R_0$  is a highly model-dependent quantity whose definition, estimation, and interpretation depend strongly on underlying assumptions. These works highlight parameterized derivations, such as deterministic model-based (next generation matrix and survival function), threshold-based (Jacobian analysis) and data-driven (Bayesian MCMC and likelihood estimation amongst others) approaches. As a starting point, the next-generation model (NGM) framework is described, providing a mechanistic characterization of epidemic invasion in compartmental models. Other deterministic and data-driven methods are introduced subsequently.

In the NGM approach, epidemics are typically modeled as non-spatial and deterministic, with transmission dynamics governed by compartmental structure which can be modeled using systems of ordinary differential equations (ODEs) that describe inflow and outflow rates between compartments, the classic example being the SIR model. The population may be divided into multiple states (compartments) and stratified by characteristics (such as treatment status, age, or risk group). The other assumption is that the initial phase of an outbreak occurs in an otherwise fully susceptible population system. At this stage, the system is linearized around the disease free equilibrium (DFE). For example, consider the SIR model:

$$\dot{S} = -\beta \frac{SI}{N}, \quad \dot{I} = \beta \frac{SI}{N} - \gamma I, \quad \dot{R} = \gamma I,$$

where  $\beta$  is the transmission rate and  $\gamma$  is the recovery rate. The DFE is given by

$$(S^*, I^*, R^*) = (N, 0, 0),$$

corresponding to a fully susceptible population with no infected individuals.

The NGM approach is motivated by viewing transmission as a renewal process across successive generations of infected individuals [Diekmann et al., 1990]. Rather than tracking infection continuously like in time series forecasting, the focus is on how one generation of infected individuals produces the next. This leads to the definition of a linear next-generation operator, which maps the current distribution of infected individuals to the expected distribution of newly infected individuals in the next generation.

Mathematically, the long-term behavior of this generational process is governed by the spectral radius of the next-generation operator.

In compartmental ODE models, the next-generation operator has a direct matrix representation. The population is partitioned into infected compartments, and the linearized infected subsystem at the DFE is decomposed as

$$\dot{x} = (F - V)x,$$

where  $F$  represents the rate of appearance of new infections in each infected compartment, and  $V$  represents all other transitions among infected compartments, including progression, recovery, and death.  $V$  is invertible and describes the expected movement of individuals through infected states in the absence of new infections. The next generation matrix is defined as

$$K = FV^{-1},$$

where the  $(i, j)$ -th entry of  $K$  gives the expected number of new infections in compartment  $i$  produced by a single individual initially introduced into compartment  $j$ . The basic reproduction number  $R_0$  is defined as the spectral radius of  $K$  [Diekmann et al., 2010], because repeated multiplication by  $K$  corresponds to successive generations of infection, and the dominant eigenvalue of  $K$  determines whether infection grows or dies out.

The survival function approach provides a parsimonious definition of  $R_0$  based off a probabilistic perspective of epidemic modeling. The basic reproductive number is then given by

$$R_0 = \int_0^\infty b(a) F(a) da,$$

where  $F(a)$  is the probability that an infected individual remains infectious for at least time  $a$ , and  $b(a)$  is the expected number of secondary infections generated per unit time by an individual who has been infected for duration  $a$ . In other words,  $R_0$  is the expected total number of secondary infections produced by a typical infected individual over their entire infectious lifetime, and does not depend on assumptions related to deterministic state transitions, where exponentially distributed infectious periods or constant transmission rates may be required.

The Jacobian-based threshold approach [Heffernan et al., 2005] is conceptually similar to the NGM framework, but the two methods differ in interpretation. Jacobian analysis characterizes local stability through growth rates of perturbations near the DFE, whereas the NGM explicitly constructs a biologically motivated mapping from one generation of infected individuals to the next. As a result, the dominant eigenvalue of the Jacobian may not coincide with the basic reproduction number. The NGM framework is specifically designed to ensure that  $R_0$  corresponds to the dominant eigenvalue.

In data-driven approaches,  $R_0$  is estimated via statistical strategies depending on the strength of assumptions [Boonpatcharanon et al., 2022] of the underlying data collected. Initially, at the ascent of an epidemic, data related to reported cases, and biological knowledge of the disease is limited. This motivates the the serial distribution, an ordered sequence of data, commonly time

series, where observations are not independent, but rather correlated over time or position. In epidemiology, this distribution is the time that an infected individual becomes symptomatic to the time in which that individual infects another person who then becomes infective. The serial distribution can be estimated through maximum likelihood estimation (MLE).

One class of data-driven methods consists of simplified real-time estimators (White–Pagano, Sequential Bayesian, Incidence Decay, Incidence Decay with Exponential Adjustment) “based on simplifications of the full ODE epidemiological models ... [this] simplification is necessitated by the fact that the full data is unobservable” [Boonpatcharanon et al., 2022]. As a result, they are computationally efficient and suitable for rapid assessment, though sensitive to biases. For the frequentist approaches, estimation of  $R_0$  is computed through the least squares optimizer of the serial distributions of two simplified models that denote relationships between  $R_0$  and other compartments in the SIR model. In Bayesian approaches, a mildly informative prior is updated sequentially: that is, SIR is the model and the mean of the serial distribution is known, and the infected equation can be updated at time intervals.

Plug-n-Play and full Bayesian Markov chain Monte Carlo (MCMC) comprise the second class of statistical approaches that uses the full epidemic models, allows for noise and unobserved states, and handles the unknown serial distribution through Bayesian theory, which is thematically about belief and updating this belief through observation. It follows that observed incidence data is linked to the unfolding epidemic process through an observation model. In Plug-n-Play, hidden Markov (HMM) or state-space models are used to estimate  $R_0$ , which are usually reported with credible intervals, not just a point estimate. A Markov process describes a sequence of two events in which the probability of each event depends only on the state attained in the previous event. HMM assumes that there is such a Markov process whose behavior is not directly observable. Inference is performed using MCMC methods, which generate estimates of  $R_0$  from the joint posterior distribution of the specified model’s parameters (for example with SIR or SEIR assumptions) and latent states, conditional on the observed data, and the probability of the transitions in the Markov process remain without a closed form. In the fullBayes approach, the main difference is that a prior is placed on the data to determine its distribution as well. The likelihood (for a compartmental model, like SEAIR) can be iteratively calculated upon updates of data. The joint prior distributions are made up of independent and identical realizations from the gamma distribution, which belong to the exponential family of distributions and therefore have a closed form for priors. Later priors are proposed through a Metropolis step. A key strength of this approach is the accuracy of prediction, as these methods can accommodate complex epidemic features such as asymptomatic transmission, reporting delays, and partial observability [Boonpatcharanon et al., 2022]. Indeed, the mean squared error (MSE) of  $R_0$  updates from the paper indicate that Plug-n-Play, Incidence Delay (ID) and Sequential Bayes (SeqB) have the best convergence in the shortest amount of time. While other models may obtain lower MSE at later times, the goal is to make the most accurate estimate of  $R_0$  as early in the pandemic as possible. Because there are various tradeoffs in all 6 models, with the former 3 emerging as the most robust, the choice of estimators should be a suite flexible to various scenarios: Plug-n-play does not require knowledge of the serial distribution and provides close to true estimates under different model structures quickly, while SeqB and ID should be implemented using a range of known serial intervals, to provide sensitivity analysis and confidence in  $R_0$  estimation [Boonpatcharanon et al., 2022].

It should be noted that deterministic approaches and classical compartmental models can be fit to observed data, but their ability to capture real epidemic dynamics may be limited because they typically assume fixed parameters, full observability, and do not explicitly model stochastic

noise or reporting processes, while the latter stochastic models are better suited to real epidemic data, which comes in the form of highly imperfect daily reported cases. Statistical models explicitly model stochasticity and various latent variables such as hidden infections and testing variability in their quantification of  $R_0$  through prediction intervals and probabilities of outbreak growth/decline. Taken together, the literature highlights that while the basic reproduction number plays a central role in theoretical epidemiology, its estimation and interpretation are inherently challenging, and ultimately, all methods that estimate  $R_0$  result in tradeoffs in interpretability and flexibility. Deterministic model-based approaches, such as survival function formulations, next generation matrix methods and Jacobian survival analysis, provide rigorous and interpretable definitions of  $R_0$  within specified modeling frameworks, but are simplified, and thus, highly biased and inflexible. Data-driven estimation methods extend these ideas to fitting the model from observed incidence data, which still pose challenges related to over-parameterization (high variance and overfitting), biological interpretation, and computational cost.

Ultimately the only safe conclusion to be drawn is that  $R_0$  is not a fixed or intrinsic property about a population, but a model-dependent quantity that reflects assumptions about population structure, behavior, and transmission mechanisms [Delamater et al., 2019]. Different modeling choices applied to the same outbreak data can yield substantially different estimates of  $R_0$ , underscoring that it is not uniquely identifiable from data alone. Similarly, early estimates are particularly sensitive to reporting delays and unobserved transmission, and historically,  $R_0$  has been underestimated [Cepelewicz, 2021]. There is a shift towards estimating  $R_t$ , the real time value reproduction number, which serves as a “real-time indicator of the epidemic’s spread and how well interventions are working” [Delamater et al., 2019], and may yield more accurate and interpretable results.

At a personal level, I believe that while compartmental models provide convenient mathematical abstraction and contextual interpretation, the assumption that individuals occupy discrete epidemiological states is an oversimplification of underlying biological processes, such as waning immunity or continuous variation in susceptibility. Therefore, in order to account for irreducible error and the extreme detail and magnitude in which real-world processes evolve, we need to realize that the data we collect and the world in which we live in is only a random sample from an unknown and highly complex distribution; as such, it is a beautiful thing that many processes cannot be interpreted or completely known, but remain nonetheless predictable, and it is perfectly acceptable to pursue unparametric methods in the mainstream.

## References

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