Practical guide to fitting an SIR model

Sang Woo Park

March 16, 2019

Contents

1 Introduction

Over the last few decades, a plethora of computational tools has been developed to study epidemic time series. Central to these methods are mechanistic models. These models allow us to study the qualitative dynamics of a system and infer underlying mechanisms that drove the observed patterns in the data. For example, we now know that the complex dynamics of measles outbreaks are driven by seasonal transmission patterns (Earn et al., 2000).

The Susceptible-Infected-Recovered (SIR) model is one of the most basic mechanistic models. It describes how individuals move from susceptible (S) to infected (I) and to recovered (R) states in a homogeneously mixing population:

$$\begin{aligned} \frac{dS}{dt} &= b(t) - \left(\beta(t)\frac{I}{N} + \mu\right) S \\ \frac{dI}{dt} &= \beta(t) S \frac{I}{N} - (\gamma + \mu) I \\ \frac{dR}{dt} &= \gamma I - \mu R \end{aligned} \tag{1}$$

where $b(t)$ is the natural birth rate (or recruitment rate to susceptible population), $\beta(t)$ is the transmission rate, γ is the recovery rate, and μ is the natural death rate. Despite its simplicity, the SIR model can be applied to studying a wide range of diseases, such as measles, plague, HIV, and influenza. The main difficulty in analyzing these systems comes from estimating transmission rate, $\beta(t)$.

One way to estimate the transmission rate $\beta(t)$ is by matching the deterministic solution of the system (1) with the observed data by minimizing their distance, measured by sum of squares difference or negative log-likelihood. This method is referred to as trajectory matching. Similarly, it is possible to match gradients of the system (1), where the gradients of the observed time series can be obtained by approximating the data with a smooth curve, such as piecewise polynomials, to the data and taking its derivative (Ellner et al., 2002). While these methods are computationally efficient and easy to implement, they do not allow us to take demographic stochasticity into account explicitly.

On the other hand, Monte Carlo methods allow us to model *all* sources of variation (e.g., observation error and process error) explicitly. For example, Sequential Monte Carlo methods, also known as particle filtering, provides a way of approximating the likelihood through a series of simulations (particles) as long as there is a process model and an observation model. Approximate likelihood can be maximized by iterated filtering (Ionides et al., 2011, 2015) or can be incorporated into a Bayesian scheme using partiler Monte Carlo Markov Chain (MCMC). Likewise, Bayesian Monte Carlo methods based on renewal equations are able to integrates over state space. These methods are computationally expensive and require discretization of the system, which may be undesirable.

This is not at all an exhaustive list of all existing methods. Generalized profiling balances bewteen trajectory matching and gradient matching by fitting a deterministic model (Hooker et al., 2010).

Laplace approximation based methods are also available.

Of all these methods, the time series SIR (TSIR) method has been particularly successful in studying dynamics of ??? diseases, especially measles.

There are many ways to estimate underlying parameters of the SIR model (1); yet their differences are unclear because they all rely on different set of assumptions. Which one should one use? Here, we compare TSIR each method against simulated data and study how differences in the assumptions affect our conclusions. Then, we apply each method to measles time series from Boston and discuss implications.

2 Data

In order to estimate the parameters of the SIR model, it is important to know what kinds of data are available. Epidemic time series can be classified into three categories: incidence, mortality, and prevalence. Incidence is defined as the number of newly infected individuals generated over a reporting period. Then, true incidence of the SIR model (1) between time t and $t - t_{\text{rep}}$, where t_{rep} is the length of reporting time step, can be obtained by integrating total infection rate over the reporting period:

$$\int_{t-t_{\text{rep}}}^{t} \beta(s) S \frac{I}{N} ds. \tag{2}$$

Similarly, mortality case, which is defined as the number of individuals that died during a reporting period, can be obtained by integrating total death rate over the reporting period:

$$\int_{t-t_{\text{rep}}}^{t} \gamma I ds. \tag{3}$$

Finally, prevalence is defined as the number of infected individuals that are present in the population and corresponds to the state variable I. Reported incidence, mortality, and prevalence cases can be drawn from a binomial (or beta-binomial) distribution with reporting rate ρ.

When reporting period is equal to mean generation time, incidence and prevalence are often assumed to be equivalent (Fig. 1). Indeed, for the simple SIR model (1), it can be shown that incidence, prevalence, and mortality are similar to each other under Euler approximation when reporting period is equal to mean generation time (see Appendix). Their deterministic dynamics are sufficiently similar that we expect these three reports to be nearly indistinguishable from each other when demographic stochasticity and observation error is introduced. However, this is not necessarily true for more complicated models. For a more realistic model with exposed period (SEIR model), incidence is similar to prevalence when reporting period is equal to mean *generation time* whereas mortality is similar to prevalence when reporting period is equal to mean *infectious period*. **[SWP:** *TODO: confirm*]

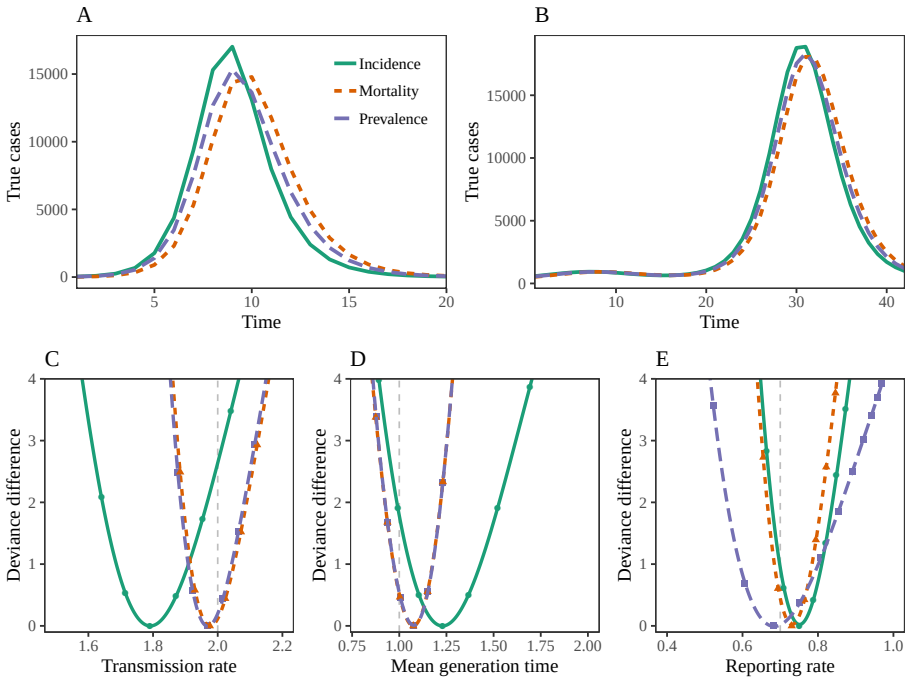


Figure 1: **Comparison of incidence, prevalence, and mortality from dynamical and estimation perspective.** A: dynamics of incidence, mortality and prevalence of the SIR model for a single outbreak (no natural birth/death and fixed transmission rate) when mean generation time ie equal to reporting period. The parameters and initial conditions are $\beta = 2$, $\gamma = 1$, $N = 1 \times 10^5$, $I(0) = 10$, and $S(0) = N - 10$. B: dynamics of incidence, mortality and prevalence of the SIR model with sinusoidal transmission rate ($\beta(t) = b_0(1 + b_1 \cos(2\pi t/26))$) when mean generation time ie equal to reporting period. The parameters and initial conditions are $b_0 = 500/26$, $b_1 = 0.15$, $\gamma = 1$, $\mu = 1/(50 \times 26)$, $N = 5 \times 10^6$, $I(0) = 0.0001N$, $S(0) = 0.05N$. C, D, E: deviance difference (profile likelihood minus the maximum likelihood) of each parameters when incidence, mortality, and prevalence are fit to same time series using trajectory matching. Grey dashed line represents the true value.

Even if incidence, mortality, and prevalence are expected to be exhibit similar dynamics other when reporting period is equal to mean generation time, it is important to distinguish them when we are estimating underlying parameters of the SIR model, especially when mean generation time $1/\gamma$ is not exactly known. To demonstrate this idea, we first simulate an epidemic time series representing prevalence over time by solving the SIR model numerically and calculating prevalence, $I(t)$, for 20 reporting periods, where reporting period is equal to the mean generation time. Then, we draw a beta-binomial random

variable at each time step with a reporting rate of 70% and an overdispersion parameter of 10. Using this time series, we try to estimate four parameters – transmission rate β, recovery rate γ, reporting rate ρ, and an initial number of infected individuals $I(0)$ – by treating the same time series as if it were incidence, mortality, and prevalence report (Fig. 1).

We find that fitting prevalence and mortality curves to the same time series yields almost identical estimates of the transmission rate β and the recovery rate γ (as well as related uncertainty in the estimates) whereas fitting incidence and mortality curves to the same time series yields consistent estimates of the reporting rate ρ (as well as related uncertainty in the estimates). As mortality is approximately a lagged reflection of prevalence regardless of reporting period (reported mortality case at time t is approximately equal to $\rho\gamma I(t - t_{\text{rep}})t_{\text{rep}}$), it is possible to generate a mortality curve that matches its corresponding prevalence curve by adjusting the reporting rate ρ. This explains why estimates of dynamical parameters, β and γ, are similar whether we treat the same time series as prevalence or mortality. On the other ahnd, incidence report no longer matches prevalence report when reporting period is not equal to the mean generation time (i.e., when γ is being estimated); therefore, fitting incidence curve yields a different estimate of the dynamical parameters, β and γ, from fitting mortality or prevalence curves. Fitting incidence and mortality curve yields similar estimates of the reporting rate ρ because they both contain equivalent information about the final size of an epidemic: total *true* incidence and total *true* mortality is equal to the final size of an epidemic.

Even when mean generation time is exactly known, we find that estimates of transmission rate β and reporting rate ρ depends on our assumptions about the data. We find that fitting mortality and prevalence yields consistent estimates of both β and ρ in this case, whereas fitting incidence yields similar but still different estimates. Treating incidence report as prevalene report (and vice versa) should be avoided.

[SWP: *Say something more*]

3 TSIR model

3.1 Model

The time-series SIR (TSIR) method was developed by Bjørnstad et al. (2002) to estimate seasonally varying transmission rates of measles. It relies on reconstructing the susceptible dynamics from birth and case reports and using linear regression, conditional on knowing "true susceptible dynamics", to estimate transmission rates. Initial conditions are often estimated to improve goodness of fit.

The TSIR method begins by discretizing the infection process, assuming that disease generation-time is equal to reporting interval:

$$S_{t+1} = B_t + S_t - I_{t+1}I_{t+1} \quad = \beta_t S_t \frac{I_t^\alpha}{N_t} \tag{4}$$

where α is often referred to as a conversion factor from continuous-time model to discrete-time model or a heterogeneity parameter [CITE]. True incidence, I_t, is inferred from the observed incidence, y_t, and estimated reporting rate ρ_t: $I_t = y_t/\rho_t$. Taking log on both sides, estimation of transmission rates β_t becomes a regression problem:

$$\log I_{t+1} = \log \beta_t + \log S_t + \alpha \log I_t - \log N_t + \epsilon_t. \tag{5}$$

The estimation process is deterministic, but the TSIR method is analogous to fitting a stochastic model: equation ?? models step-ahead prediction as a function of previous state and process error, ϵ_t. Coupled with a deterministic susceptible reconstruction and estimation of reporting rate, the TSIR method attributes all errors to one-step process noise and oversmooths the likelihood surface (figure ?). Several studies have associated dynamics of the deterministic model (equation ??) with the inferred parameters from the regression but they are intrinsically different models.

3.2 Probability of infection

The TSIR model assumes that probability of infection is a nonlinear function of prevalence: $\beta I^\alpha/N$. This contrasts with the linear assumption of the Euler approximation ($\beta I/N$) or the exponenital assumption of the hazard-based approximation ($1-\exp(-\beta I/N)$). Inclusion of the parameter α has been previously attributed to heterogeneity in the population or changes from continuous to discrete time model. However, there is still a room for clearer understanding of the actual role of the parameter α: for example, a few studies recognize that the estimated α does not always correspond to the best predictive α and resorted to assuming $\alpha = 0.97$.

Glass et al derived an analytical expression for optimal values of α by comparing the changes in the infected population at equilibrium using a second order Taylor expansion. Based on their approximation, optimal value of α equals 1 when there is no birth term. Instead, when we rewrite the TSIR model as

$$\log\left(\frac{I_{t+1}}{S_t}\right) = \log\left(\frac{\beta I_t^\alpha}{N_t}\right) + \epsilon_t. \tag{6}$$

it becomes much clearer that the TSIR model is trying to match the log probability of infection (when an epidemic is ongoing, changes in the susceptible population S_t through natural birth or death is negeligible over a generation, and I_{t+1}/S_t can be interpreted as the probability of infection over a generation). Then, we almost always expect α to be less than one because we expect the probability of infection (i.e., probability of finding a new host) to experience density dependence.

Though the difference in the interpretation of α is subtle, this perspective allows us to derive a useful connection between the discrete-time model and the continuous-time model. A naive estimate of basic reproductive number from the

TSIR model is β. However, as discussed earlier, we often prefer to use hazard-based approach to approximate the continuous-time model. Then, we can find $\hat{\beta}$ such that $1 - \exp(-\hat{\beta} I/N)$ matches $\beta I^\alpha/N$ and use $\hat{\beta}$ as our estimate of the basic reproductive number instead. A simple way to match two functions is to match their values at the mean observed incidence. This corrections reduces bias in the estimate of the reproductive number by a large amount.

3.3 Process model

The TSIR model is often associated with three different process models: deterministic ($I_{t+1} = \beta_t S_t I_t^\alpha/N$), poisson ($I_{t+1} \sim \text{Poisson}(\beta_t S_t I_t^\alpha/N)$), and negative binomial ($I_{t+1} \sim \text{NB}(\beta_t S_t I_t^\alpha/N, I_t)$).

3.4 Susceptible reconstruction

One of the main assumptions behind the TSIR model is that the dynamics susceptible population S_t must be known. Assuming that all newborn infants become infected eventually, it is possible to recover the reporting rate, ρ_t, as well as the dynamics of the susceptible population as a deviation from its mean, $Z_t = S_t - \bar{S}$, by fitting a regression model to between cumulative births and cumulative cases:

$$\sum_{t=1}^{N} B_t = \sum_{t=1}^{N} \frac{C_{t+1}}{\rho_{t+1}} + Z_{t+1} - Z_1, \tag{7}$$

where C_t is the number of observed cases. Then, the mean susceptible population $\bar{S}$ can be estimated using profile likelihood with the TSIR regression.

While this method of reconstructing the susceptible dynamics has been successfully used with the TSIR method, several questions remain to be answered. First, how sensitive is our inference to regression methods? Second, does Z_t represent deviation from the overall population mean $\bar{S}$ or from some sort of moving average (assuming that population changes over time)? Likewise, how does changes in population size or reporting rate over time affect our estimate of ρ_{t+1} or Z_t? Finally, how do assumptions about the population size affect estimate of the transmission rate?

We first simulate an epidemic using the continuous deterministic SIR model for 10 years and apply 5 different regression models available from the `tsiR` package to test their ability to infer reporting rate and susceptible dynamics (Fig. **??**). We find that estimates of transmission rates are highly sensitive to regression methods. The gaussian regression is particularly inaccurate in boundaries. Other methods provide similar estimates. In general, estimates of susceptible dynamics appear to be robust regardless of the regression method.

Then, we compare estimates of susceptible dynamics and reporting rate under different scenarios (Fig. 3). We allow for population size to stay constant or increase over time and also allow for reporting rate to stay constant or increase over time; these combinations yields 4 different scenarios. We find that estimating time-varying reporting rate is difficult. We also find that Z_t estimated

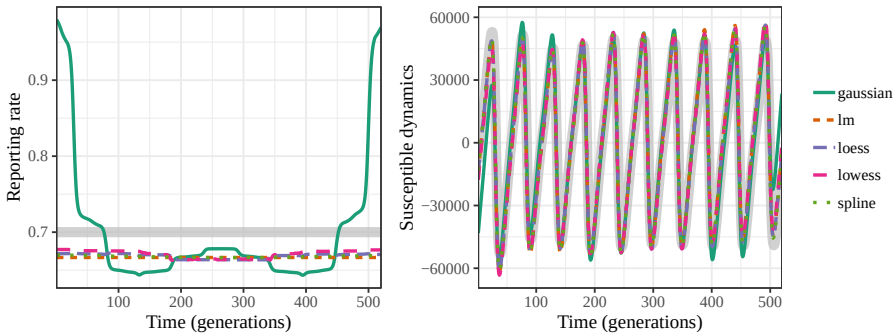


Figure 2: [**SWP:** *TODO*]

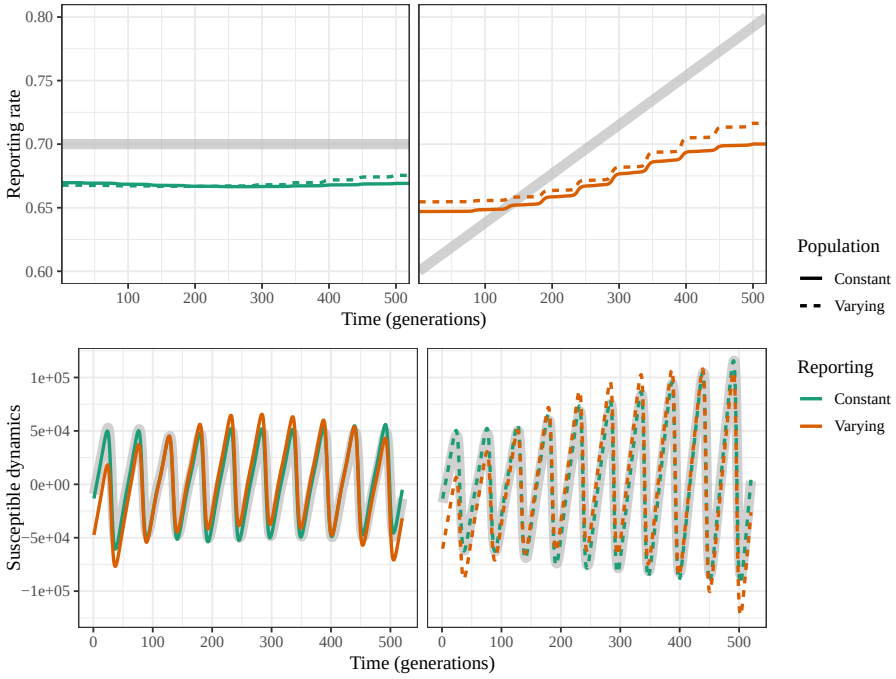


Figure 3: [**SWP:** *TODO*]

form regression better matches $S_t - \bar{S}$ rather than $S_t - \sigma N$, i.e., any long term dynamics in the susceptible population is captured in Z_t.

3.5 Splines

4 Other fitting methods

4.1 Trajectory matching

4.2 Gradient matching

4.3 Particle filtering

4.4 Renewal equation

5 Numerical comparison of different methods

5.1 Single outbreak

5.2 Time-varying transmission rate

6 Case study

7 Discussion

8 Appendix

8.1 Equivalence of incidence, mortality, and prevalence

First, recall that mortality case can be written as

$$\int_{t-t_{\text{rep}}}^{t} \gamma I ds. \tag{8}$$

When reporting period is equal to mean generation time, i.e., $t_{\text{rep}} = 1/\gamma$, it follows that:

$$\int_{t-t_{\text{rep}}}^{t} \gamma I ds \approx \gamma I(t - t_{\text{rep}}) t_{\text{rep}} = I(t - t_{\text{rep}}) \tag{9}$$

Likewise, we can derive a similar relation for incidence:

$$\begin{aligned} I(t + t_{\text{rep}}) &= I(t) + \int_{t}^{t+t_{\text{rep}}} \frac{dI(s)}{ds} ds \\ &= I(t) + \int_{t}^{t+t_{\text{rep}}} \beta(s) S \frac{I}{N} ds - \int_{t}^{t+t_{\text{rep}}} \\ &\approx \int_{t}^{t+t_{\text{rep}}} \beta(s) S \frac{I}{N} ds \end{aligned} \tag{10}$$

Here, we assumed that individuals leaving infected class I from natural mortality is negligible over the reporting period.

[SWP: *TODO*]

9 Fitting continuous models

9.1 Trajectory matching

Trajectory matching is one of the simplest fitting methods. As its name suggests, the goal is to find a set of parameters such that the deterministic trajectory of the ODE model best matches the observed data. This method assumes that there is only measurement error and no process error. The measurement model can be written as:

$$y_t \sim \text{NegBinomial}\left(\mu_t = \int_{t-\Delta t}^{t} \beta(s) S \frac{\hat{I}}{N} ds, \phi\right), \tag{11}$$

where ρ is reporting rate and ϕ is dispersion parameter. We prefer this model compared to the binomial or beta-binomial model because fitting binomial or beta-binomial models may not work if there are any irregular patterns in the data.

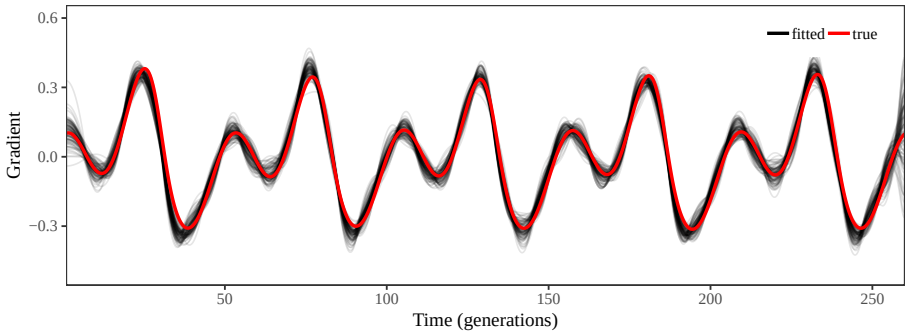


Figure 4: This is not a caption.

9.2 Gradient matching

When *all* state variables are observed, we can fit smooth curves to the data and obtain an estimate of the gradients of the ODE by taking the derivatives of the estimated smooth functions. Gradient matching methods seek to estimate the rate equation using nonparametric regression. However, for most epidemics, it is impossible to have data of all state variables. When only incidence is provided, we have no direct observations of any state variables. If we can reconstruct the dynamics of the susceptible population and assume that incidence is sufficiently similar to prevalence, we can still be able to apply gradient mathing methods.

Observe that

$$\frac{d \log \hat{I}}{dt} = \beta(t) \frac{S}{N} - (\gamma + \mu), \tag{12}$$

If we can obtain a reasonable estimate of the gradient $Z_i = d \log \hat{I}/dt(t_i)$, we can write

$$Z_i + (\gamma + \mu) = \exp\left(\log \beta(t_i) + \log S_i - \log N\right) + \epsilon_i \tag{13}$$

and estimate β, given that we can reconstruct the susceptible dynamics S_i. Estimate of β can be done using nonparametric regression by treating $\log S_i$ and $\log N$ as offset terms.

First, we want to test whether gradients can be reliably estimated from incidence data alone by simulating a deterministic SIR model with measurement error. We use natural cubic spline with knots placed every 6 biweeks. It works generally well but there is systematic bias. When gradients are at their local maxima (and minima), we tend to overestimate (and underestimate) the gradients. This is because incidence precedes prevalence???

If we simply construct confidence intervals based on equation ?? we underestimate uncertainty significantly by ignoring the uncertainty in the estimated gradients Z_i.

Conditions:

- See Jost and Ellner (???)

- sampled sufficiently
- not too much error
- Brunel Parameter estimation of ODE's via nonparametric estimators

9.3 Generalized profiling

Generalized profiling combines trajectory matching with gradient matching.

9.4 Spectral matching

What is this? Read Reuman et al. 2006

10 Fitting discrete time models

10.1 Particle filtering

Particle filtering provides a way of approximating the likelihood for stochastic models. In this paper, we focus on mif2 method.

$$\begin{aligned} S_{t+\Delta t} &= B_t + S_t - S_t\left(1 - \exp\left(-\beta_t \frac{I_t}{N_t}\Delta t\right)\right) \\ I_{t+\Delta t} &= S_t\left(1 - \exp\left(-\beta_t \frac{I_t}{N_t}\Delta t\right)\right) + I_t - I_t(1 - \exp(-(\gamma+\mu)\Delta t)) \end{aligned} \tag{14}$$

Tries to account for variation in ... but still ... When are these assumptions appropriate?

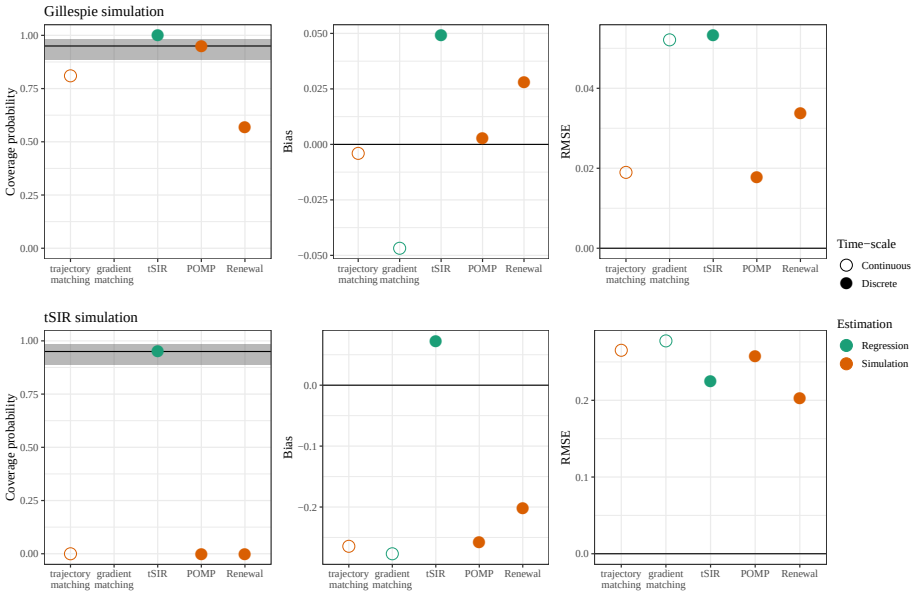
10.2 Incidence-based models

Li et al. (2018) compared incidence-based models across several Bayesian platforms. In our case, we can write a similar model as:

$$\begin{aligned} S_{t+\Delta t} &= B_t + S_t - I_{t+\Delta t} \\ \phi_t &= \sum_{i=1}^{\ell} k(i) I_{t-\ell+i} \\ I_{t+\Delta t} &\sim \text{BetaBin}(1 - e^{-\phi_t}, S_t, \delta_P) \\ \text{Obs}_t &\sim \text{NegBin}(P_{rep}, \sum I_t, \delta_{obs}) \end{aligned} \tag{15}$$

When reporting period is same as the mean generation time, we want to simulate the model on a finer scale.

Disadvantage: unable to model mechanisms; difficult to deal with prevalence or mortality data. Advantage: flexible generation time distribution.



11 Results

11.1 Statistical inference

References

Bjørnstad, O. N., B. F. Finkenstädt, and B. T. Grenfell (2002). Dynamics of measles epidemics: estimating scaling of transmission rates using a time series SIR model. *Ecological Monographs 72*(2), 169–184.

Earn, D. J., P. Rohani, B. M. Bolker, and B. T. Grenfell (2000). A simple model for complex dynamical transitions in epidemics. *Science 287*(5453), 667–670.

Ellner, S. P., Y. Seifu, and R. H. Smith (2002). Fitting population dynamic models to time-series data by gradient matching. *Ecology 83*(8), 2256–2270.

Hooker, G., S. P. Ellner, L. D. V. Roditi, and D. J. Earn (2010). Parameterizing state–space models for infectious disease dynamics by generalized profiling: measles in Ontario. *Journal of The Royal Society Interface 8*(60), 961–974.

Ionides, E. L., A. Bhadra, Y. Atchadé, A. King, et al. (2011). Iterated filtering. *The Annals of Statistics 39*(3), 1776–1802.

Ionides, E. L., D. Nguyen, Y. Atchadé, S. Stoev, and A. A. King (2015). Inference for dynamic and latent variable models via iterated, perturbed Bayes maps. *Proceedings of the National Academy of Sciences 112*(3), 719–724.

Li, M., J. Dushoff, and B. M. Bolker (2018). Fitting mechanistic epidemic models to data: a comparison of simple Markov chain Monte Carlo approaches. *Statistical methods in medical research 27*(7), 1956–1967.

12 Appendix

12.1 Derivation of growth rate in discrete-time SIR model

Derivation:

$$\begin{aligned} S_{t+\Delta t} &= S_t - S_t(1 - \exp(-\beta I_t \Delta t/N)) \\ I_{t+\Delta t} &= S_t(1 - \exp(-\beta I_t \Delta t/N)) + I_t - I_t(1 - \exp(-\gamma \Delta t)) \end{aligned} \tag{16}$$

Assuming tat S_t is approximately equal to N, we get

$$I_{t+\Delta t} = N_t(1 - \exp(-\beta I_t \Delta t/N)) + I_t \exp(-\gamma \Delta t) \tag{17}$$

When $I_t \approx 0$, $\exp(-\beta I_t \Delta t/N) \approx 1 - \beta I_t \Delta t/N$ and so

$$\begin{aligned} I_{t+\Delta t} &\approx I_t \beta \Delta t + I_t \exp(-\gamma \Delta t) \\ &= I_t(\beta \Delta t + \exp(-\gamma \Delta t)) \end{aligned} \tag{18}$$

Substituting $\hat{\gamma}$, we get

$$I_{t+\Delta t} = I_t \left(1 + \beta \Delta t - \frac{\Delta t}{\mu}\right), \tag{19}$$

Therefore,

$$I_t = I_0 \left(1 + \beta \Delta t - \frac{\Delta t}{\mu}\right)^{t/\Delta t} \tag{20}$$

and the initial growth rate is given by

$$r = \frac{1}{\Delta t} \log \left(1 + \beta \Delta t - \frac{\Delta t}{\mu}\right). \tag{21}$$

12.2 Probability of infection

In both the TSIR model and the hazard-based model, transition from the susceptible comparment, S, to the infected compartment, I, is represented as a product of number of susceptible individuals and probability of infection between two time steps t and $t + \Delta t$. The TSIR model assumes that the probability of infection is a linear function of prevalence I_t. This formulation is based on the Euler approximation to the solution of the ordinary differential equation. On the other hand, the hazard-based model assumes that the probability of infection is an inverse exponential function of prevalence I_t. Besides their differences in the relationship between prevalence and probability of infection, there are two more assumptions that we need to consider: (1) force of infection remains constant over two time steps and (2) new susceptible individuals have zero probability of infection between the two time steps.

12.3 Infectious period

Geometric distribution with probability of $1-\exp(-\gamma\Delta t)$ and unit of Δt. Then, mean infectious period and generation interval is given by

$$\frac{\Delta t}{1-\exp(-\gamma\Delta t)}. \tag{22}$$

An important but often overlooked component is the variance of the distribution. Squared coefficient of variation for this distribution is equal to $\exp(-\gamma\Delta t)$, which necessarily depends on pre-specified time-step Δt. If we want to match the mean of the distribution to a fixed value μ regardless of our choices of Δt, we obtain

$$\hat{\gamma} = -\frac{1}{\Delta t}\log\left(1-\frac{\Delta t}{\mu}\right) \tag{23}$$

Then,

$$\mathrm{CV}_{\text{Infectious period}}(\Delta t)^2 = 1-\frac{\Delta t}{\mu}. \tag{24}$$

We expect changes in variation in infectious period to affect variation in stochstic realizations.

This means that the relationship between r and $\mathcal{R}$ changes. For this generation-interval distribution, we have

$$\mathcal{R} = 1+\frac{\exp(r)-1}{(1-\exp(-\gamma\Delta t))} \tag{25}$$

Substituting

$$r = \frac{1}{\Delta t}\log\left(1+\beta\Delta t-\frac{\Delta t}{\mu}\right), \tag{26}$$

as well as $\hat{\gamma}$, we get

$$\mathcal{R} = 1+\frac{\mu}{\Delta t}\left(1+\beta\Delta t-\frac{\Delta t}{\mu}\right)^{1/\Delta t} \tag{27}$$

Then, we can obtain a relationship between $\mathcal{R}$ of a discrete-time system and that of a corresponding continuous time system, assuming that contact rate β and mean generation-time μ are the same:

$$\mathcal{R}_{\text{discrete}} = 1+\frac{\mu}{\Delta t}\left(1+\frac{(\mathcal{R}_{\text{continuous}}-1)\Delta t}{\mu}\right)^{1/\Delta t} \tag{28}$$

On the other hand, we may want to match choose β and μ to match true r and $\mathcal{R}$.