The Limits to Learning an SIR Process: Granular Forecasting for Covid-19

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Abstract

A multitude of forecasting efforts have arisen to support management of the ongoing COVID-19 epidemic. These efforts typically rely on a variant of the SIR process and have illustrated that building effective forecasts for an epidemic in its early stages is challenging. This is perhaps surprising since these models rely on a small number of parameters and typically provide an excellent retrospective fit to the evolution of a disease. So motivated, we provide an analysis of the limits to estimating an SIR process. We show that no unbiased estimator can hope to learn this process until observing enough of the epidemic so that one is approximately two-thirds of the way to reaching the peak for new infections. Our analysis provides insight into a regularization strategy that permits effective learning across simultaneously and asynchronously evolving epidemics. This strategy has been used to produce accurate, granular predictions for the COVID-19 epidemic that has found large-scale practical application in a large US state.

1 Introduction

The so-called Susceptible-Infected-Recovered (SIR) model, proposed nearly a century ago, remains a cornerstone for the forecasting of epidemics. In numerous retrospective studies the model has been found to fit the trajectory of epidemics well, while simultaneously providing a meaningful level of interpretability. As such, the plurality of models used for forecasting efforts related to the COVID-19 epidemic are either SIR models or close cousins thereof. Surprisingly, the experience with these forecasts has illustrated that predicting the cumulative number of cases (or peak number of cases) in an epidemic, early in its course, is a challenging task.

Ultimately, this paper is motivated by producing high quality forecasts for the progression of epidemics such as COVID-19. However, we begin with a fundamental question that, despite the SIR model’s prevalence, has apparently not been asked: What are the limits of learning in epidemic models such as the SIR model? Surprisingly, we find that it is fundamentally difficult to predict the cumulative number of infections (or the ‘peak’ of an infection) until quite late in an epidemic. In particular, we show that no estimator can hope to learn these quantities until observing enough of the epidemic so that one is approximately two-thirds of the way to reaching the peak for new infections. As far as we can tell, this result is the first of its kind for epidemic modeling. We find that this hardness is driven by uncertainty in the initial prevalence of the infection in the population, which is not observable with incomplete testing and/or asymptomatic patients. On the other hand, we show that certain other important parameters in the SIR model – including the so-called reproduction number and infectious period – are actually easy to learn.
Our analysis on the limits of learning above suggests a specific regularization approach that dramatically impacts forecast performance when learning across regions. In greater detail, we consider a differentiable model, where the true infection curve is latent and follows SIR dynamics. The observations are the results of limited testing, which censors the true infection curve, and deaths. Our learning rate analysis suggests that by suitably ‘matching’ regions early in the epidemic to regions that are further along, we can build useful estimators of initial disease prevalence in the former regions. This matching is effectively enabled through a regularization strategy in our approach where certain region specific random effect terms are regularized to zero.

We demonstrate that our approach allows us to learn granular epidemic models (essentially at the level of individual counties) that are substantially more accurate than a highly referenced benchmark \[5\]. As such, our model has found practical application in a large US state. Among other uses, the forecasts inform decisions related to providing surge capacities in hospitals across the state; such decisions necessitate a granular forecast.

**Related Literature**: Extant epidemiological models are typically *compartamental* models, of which the SIR model [2] is perhaps the best known. The plurality of COVID-19 modeling efforts are founded on SIR-type models, eg. \[4,5,6,7,8,9,10,11,12,13,14\]. Some of these efforts consider generalizations to the SIR model that add additional states or ‘compartments’. For instance the (retrospectively fit) model studied in [6] considers an eight state model (as opposed to three for the vanilla SIR). Not surprisingly, learning gets harder with as the number of states increases \[15\].

Whereas the identifiability of the SIR model (in a deterministic sense) is well understood [16], this is not the case for the natural stochastic variant of this model. Specifically, calibrating a vanilla SIR model to data requires learning the so-called infectious period and basic reproduction rate. Both these parameters are relatively easy to calibrate with limited data; this is supported both by the present paper, but also commonly observed empirically; see for instance [15]. In addition to these parameters, however, one needs to measure both the initial number of infected individuals and the size of the susceptible population. Estimating the number of infected individuals poses a challenge in the presence of limited testing and asymptomatic carriers. Indeed, epidemiological models for COVID-19 typically assume that measured infections are some fraction of true infections; eg. \[4,6\]. This challenge is closely related to that of measuring the true fraction of cases that lead to fatalities (or the so-called Infection Fatality Rate) [17].

Our main theorem shows that having to learn the true initial prevalence of the infection presents a fundamental difficulty to learning SIR models with limited data. Our theoretical results are complemented by empirical findings in the literature, see \[18,19\]. Our result is the first to characterize the limits to learning an SIR process [1] and relies on a non-trivial application of the Cramer-Rao bound.

Finally, on the empirical front, we compare the quality of our predictions to publicly available predictions for the IHME model [3]: this highly publicized benchmark model has a large number of forecast vintages concurrently available which makes possible an analysis of relative prediction accuracy as a function of available data.

# 2 The Deterministic SIR Model and a Stochastic SIR Process

We begin by describing the *deterministic* SIR model. Let \(s(t), i(t)\) and \(r(t)\) be the size of the susceptible, infected and recovered populations respectively, as observed at time \(t\). The SIR model is defined by the following system of ODEs, specified by the tuple of parameters \((\alpha, \beta, \gamma)\):

\[
\begin{align*}
\frac{ds}{dt} &= -\beta \frac{s}{N} i, \\
\frac{di}{dt} &= \beta \frac{s}{N} i - \gamma i, \\
\frac{dr}{dt} &= \gamma i.
\end{align*}
\]  

(1)

The rate of recovery is specified by \(\gamma > 0\); \(1/\gamma\) is frequently referred to as the *infectious period*. \(\beta > 0\) quantifies the rate of transmission; \(\beta/\gamma \equiv R_0\) is also referred to as the *basic reproduction number*. \(N\) is the total population (assumed known). The typical exposition of this model sets \(\alpha = 1\) corresponding to the setting where the infection is entirely observed. On the other hand, the quantity \(\alpha \in (0, 1]\) corresponds to the fraction of true infections we actually observe. The role of \(\alpha\) is made clear by the following Proposition:

**Proposition 2.1.** Let \(\{s'(t), i'(t), r'(t)\} : t \geq 0\) be a solution to (1) for parameters \(\alpha = 1, \beta = \beta', \gamma = \gamma'\) and initial conditions \(i(0) = i'(0), s(0) = s'(0)\). Then, for any \(\alpha' > 0\),
We next characterize a sequence of systems of increasing size. Specifically, consider a sequence when we will consider a differentiable model inspired by the SIR process and show that a regularization strategy motivated by our learning analysis yields material performance gains.

Proposition 2.2. Fix \( N \), and let \( \hat{i}(t) \) be observed over some open set in \( \mathbb{R}_+ \). Then the parameters \((\alpha, \beta, \gamma)\) are identifiable.

A self-contained proof follows immediately from the identity theorem; a more involved argument based on identification results for non-linear systems can be found in [16].

**Stochastic SIR Process:** Of course, any real world model must incorporate noise, and we describe next a natural continuous-time Markov chain variant of the deterministic model described above, proposed by [1]. Specifically, the stochastic SIR process, \( \{(S(t), I(t), R(t)) : t \geq 0\} \), is a multivariate counting process, with RCLL paths, determined by the parameters \((\alpha, \beta, \gamma)\). The jumps in this process occur at rate \( \lambda(t) \) (to be defined) and correspond either to a new observed infection (where \( I(t) \) increments by one, and \( S(t) \) decrements by one) or to a new observed recovery (where \( I(t) \) decrements by one, and \( R(t) \) increments by one). Let \( C(t) = I(t) + R(t) \) denote the cumulative number of infections observed up to time \( t \). Denote by \( t_k \) the time of the \( k \)-th jump, and let \( T_k \) be the time between the \( k-1 \)-st and \( k \)-th jumps. Finally, let \( \hat{N}_k \triangleq I(t_k) \), and similarly define \( \hat{R}_k, \hat{S}_k \) and \( \hat{C}_k \).

The SIR process is then completely specified by:

\[
C_k - C_{k-1} \sim \text{Bern}\left(\frac{\beta S_{k-1}}{\beta S_{k-1} + \alpha N \gamma}\right)
\]

\[
T_k \sim \text{Exp}\left(\frac{\beta S_{k-1}}{\alpha N} + \gamma\right) I_{k-1}.
\]

It is well known that solutions to the deterministic SIR model [1] provide a good approximation to sample paths of the SIR process (described by [2], [3]) in the so-called fluid regime; see [20]. The next section analyzes the rate at which one may hope to learn the unknown parameters \((\alpha, \beta, \gamma)\) as a function of \( k \); our key result will illustrate that in large systems, \( \alpha \) is substantially harder to learn than \( \beta \) or \( \gamma \). In fact, an approximation suggests that the time taken to learn this parameter accurately will be approximately two-thirds of the time required to hit the peak for infections. In Section 4 we will consider a differentiable model inspired by the SIR process and show that a regularization strategy motivated by our learning analysis yields material performance gains.

3 Limits to Learning

This section characterizes the rate at which one may hope to learn the parameters of a stochastic SIR process, simply from observing the process.

**Observations:** Define the stopping time \( \tau = \inf\{k : I_k = 0\} \); clearly \( \tau \) is bounded. For clarity, when \( k > \tau \), we define \( C_k = C_{\tau}, I_k = 0, \) and \( T_k = \infty \). We define the \( k \)-th information set \( O_k = (I_0, R_0, T_1, C_1, \ldots, T_k, C_k) \) for all \( k \geq 1 \). Note that \( I_k \) and \( R_k \) are deterministic given \( C_k, I_0, \) and \( R_0 \).

We next characterize a sequence of systems of increasing size. Specifically, consider a sequence of SIR processes, indexed by \( n \), such that \( \alpha_n N_n \to \infty \) while \( \beta_n = \beta, \gamma_n = \gamma \). Moreover, let \( m_n = o(\alpha_n N_n) \) and \( \beta > \gamma \). Finally, assume that \( I_{n,0}, R_{n,0} \leq m_n \). Our main theorem characterizes the Fisher information of \( O_{m,n} \) relative to \( \alpha_n \) as \( n \) grows large.

**Theorem 3.1.** Assume \( \beta > \gamma \) are known and \( I_{n,0} \geq D \), where \( D \) is a constant that depends only on \( \beta \) and \( \gamma \). Then, the Fisher information of \( O_{m,n} \) relative to \( \alpha_n \) is

\[
\mathcal{J}_{O_{m,n}}(\alpha_n) = \Theta\left(\frac{m_n^3}{\alpha_n^4 N_n^2}\right).
\]

Components of the proof of this result are provided in Section 3.1. Now since \( \alpha_n \in (0,1] \) and since all points in this interval are regular in the terminology of [21], we have by the constrained Cramer-Rao bound (Lemma 4 in [21]):
Then, we next turn our attention to learning $\beta$ (call this time $J$ Define Lemma 3.5. Now since we know (from the fluid model (1)) that cumulative

Expected $E_Rk$ after

Define the indicator variable subscript $n$ Define $\beta$

level of accuracy, we simply need the number of events $m$ In stark contrast with Corollary 3.2, Theorem 3.4 shows that the variance in estimating $B$ where $R_t \triangleq \frac{\beta}{\gamma}S_n(t)/((\alpha_nN_n)$ to fall below 1 (the expected increment in the infection process is negative at times when $R_t < 1$); call this time $T_{2,n}$. More precisely, $T_{1,n} = \inf\{t : C_n(t) \geq (\alpha_nN_n)^{2/3}\}$ and $T_{2,n} = \inf\{t : \beta S_n(t)/((\alpha_nN_n) < \gamma\}$. Unfortunately, characterizing either of these (random) times exactly in the stochastic SIR process appears to be a difficult task and so we consider analyzing these times in the deterministic model, where we denote $T^d_{1,n} = \inf\{t : c_n(t) \geq (\alpha_nN_n)^{2/3}\}$ for the process defined by (1) and similarly $T^d_{2,n} = \inf\{t : \beta S_n(t)/((\alpha_nN_n) < \gamma\}$. We have:

**Proposition 3.3.** If $c_n(0) = O(\log(\alpha_nN_n))$,

$$\lim_{n \to \infty} \frac{T^d_{1,n}}{T^d_{2,n}} \geq \frac{2}{3}. \quad (5)$$

In summary, this suggests that the sampling requirements made precise by Corollary 3.2 can only be met at such time where we are close to reaching the peak infection rate.

We next turn our attention to learning $\beta$ and $\gamma$:

**Theorem 3.4.** Assume $\beta \in [0, \beta_{\text{max}}], \gamma \in [0, \gamma_{\text{max}}]$. Let $C_{n,0}, m_n, \alpha_n, N_n$ satisfy $m_n(m_n+C_{n,0}) \leq \alpha_nN_n$, $\sqrt{2\log m_n} \leq \frac{1}{4}$ and $\frac{\alpha_nN_n-m_n-C_{n,0}}{\alpha_nN_n} > \frac{1}{2}(\frac{\beta}{\beta+\gamma} + \frac{1}{2})$. Then, there exist estimators $\hat{\beta}_{m_n}$ and $\hat{\gamma}_{m_n}$, both functions of $O_{m_n}$, such that

$$\mathbb{E}[(\hat{\beta}_{m_n} - \beta)^2] \leq M_1\beta_{\text{max}}^2 \frac{\log m_n}{m_n} + B_1\beta_{\text{max}}^2 e^{-B_2 I_{n,0}}, \quad (6)$$

$$\mathbb{E}[(\hat{\gamma}_{m_n} - \gamma)^2] \leq M_2\gamma_{\text{max}}^2 \frac{\log m_n}{m_n} + B_1\gamma_{\text{max}}^2 e^{-B_2 I_{n,0}}, \quad (7)$$

where $B_1, B_2 > 0$ depend only on $\beta$ and $\gamma$ and $M_1, M_2 > 0$ are absolute constants.

In stark contrast with Corollary 3.2, Theorem 3.4 shows that the variance in estimating $\beta$ and $\gamma$ grows small as $m_n$ and $I_{n,0}$ grow, but is independent of $\alpha_n$ and $N_n$. Consequently, to achieve any desired level of accuracy, we simply need the number of events $m_n$ and the initial number of infections $I_{n,0}$ to exceed a constant that is independent of the size of the system $N_n$.

### 3.1 Proof of Theorem 3.1

Define $p \triangleq \frac{1}{2}(\beta \frac{\beta}{\beta+\gamma} + \frac{1}{2}) > \frac{1}{2}$, and let $P_n = \alpha_nN_n$. We fix $n$ large enough so that $m_n + C_0 \leq P_n$ and $\beta(P_n - m_n - C_0) > p$ (this is possible since $\frac{\beta}{\beta+\gamma} > p$ and $m_n = o(P_n)$). We remove the subscript $n$ henceforth.

Define the indicator variable $E_k = 1\{\tau > k\}$ on the event which the SIR process has not terminated after $k$ jumps. The following lemma states that both $E_k$ and $I_k$ can be determined from $C_k, I_0,$ and $R_0,$ which will allow us to decouple variables in $O_{m}$ in the analysis of the Fisher information. The result follows from the definitions of $\tau, E_k$, and $C_k$; the details can be found in the Appendix.

**Lemma 3.5.** Define $r_k \triangleq \frac{I_0 + k + 2R_0}{2}$ for all $k \geq 0$. For all $k, E_k = 1\{C_k > r_k\}$. Moreover, when $E_k = 1, I_k = 2C_k - k - I_0 - 2R_0 > 0$.

The next lemma writes an exact expression for $J_{O_{m}}(\alpha)$.
Lemma 3.6. The Fisher information of the observations \( O_m \) with respect to the parameter \( \alpha \) is

\[
\mathcal{J}_{O_m}(\alpha) = \sum_{k=1}^{m} \Pr(E_{k-1} = 1) \mathbb{E} \left[ \frac{N^2 C_{k-1}^2}{P^2 (P - C_{k-1})((P - C_{k-1}) + \frac{2}{3}P)} | E_{k-1} = 1 \right]. \tag{8}
\]

Proof. We first define conditional Fisher information and state some known properties.

Definition 3.7. Suppose \( X, Y \) are random variables defined on the same probability space whose distributions depend on a parameter \( \theta \). Let \( g_{X|Y}(x|y, \theta) = \frac{\partial}{\partial \theta} \log f_{X|Y}(x|y, \theta) \) be the square of the score of the conditional distribution of \( X \) given \( Y = y \) with parameter \( \theta \) evaluated at \( x \). Then, the conditional Fisher information is defined as \( \mathcal{J}_{X|Y}(\theta) = \mathbb{E}_{X|Y} \left[ g_{X|Y}(X, Y, \theta) \right] \).

Property 3.8. \( \mathcal{J}_{X_1, \ldots, X_n}(\theta) = \mathcal{J}_{X_1}(\theta) + \sum_{i=2}^{n} \mathcal{J}_{X_i|X_1, \ldots, X_{i-1}}(\theta). \)

Property 3.9. If \( X \) is independent of \( Z \) conditioned on \( Y \), \( \mathcal{J}_{X|Y,Z}(\theta) = \mathcal{J}_{X|Y}(\theta). \)

Property 3.10. If \( X \) is deterministic given \( Y = y \), \( g_{X|Y}(X, y, \theta) = 0. \)

Property 3.11. If \( \theta(\eta) \) is a continuously differentiable function of \( \eta \), \( \mathcal{J}_X(\eta) = \mathcal{J}_X(\theta(\eta))(\frac{d\theta}{d\eta})^2. \)

Since \( I_0 \) and \( R_0 \) are known and not random, the Fisher information of \( O_m \) is equal to the Fisher information of \((1, C_1, T_2, C_2, \ldots, T_m, C_m)\). Then, Property 3.8 implies

\[
\mathcal{J}_{O_m}(\alpha) = \mathcal{J}_{T_1}(\alpha) + \mathcal{J}_{C_1|T_1}(\alpha) + \mathcal{J}_{T_2|T_1, C_1}(\alpha) + \mathcal{J}_{C_2|T_1, C_1, T_2}(\alpha) + \cdots + \mathcal{J}_{C_m|T_1, C_1, \ldots, T_m}(\alpha).
\]

Note that for any \( k \), \( C_k \) and \( T_k \) only depend on \( C_{k-1} \). Indeed, since \( C_{k-1} \) determines \( E_{k-1} \), if \( E_{k-1} = 0 \) (the stopping time has passed), then \( C_k = C_{k-1} \) and \( T_k = \infty \). When \( E_{k-1} = 1 \), the distributions of \( C_k \) and \( T_k \) are given in (2-3). Since \( \beta, \gamma, I_0, R_0 \) are known, \( S_{k-1} = P - C_{k-1} \), and \( I_{k-1} \) can be determined from \( C_{k-1} \) (Lemma 3.5), the distributions of \( C_k \) and \( T_k \) are determined by \( C_{k-1} \). Therefore, we use Property 3.9 to simplify the above equation to

\[
\mathcal{J}_{O_m}(\alpha) = \sum_{k=1}^{m} \Pr(E_{k-1} = 1) \mathbb{E} \left[ g_{C_k|C_{k-1}}(C_k, C_{k-1}, \alpha) + g_{T_k|C_{k-1}}(T_k, C_{k-1}, \alpha) | E_{k-1} = 1 \right] \Pr(E_{k-1} = 1).
\]

The last step is to evaluate \( g_{C_k|C_{k-1}}(C_k, C_{k-1}, \alpha) \) and \( g_{T_k|C_{k-1}}(T_k, C_{k-1}, \alpha) \). When \( E_{k-1} = 1 \), the distributions of \( C_k \) and \( T_k \) conditioned on \( C_{k-1} \) have a simple form provided in (2-3), Property 3.11 allows for straightforward calculations, resulting in (8). See Appendix for details of this last step.

Proof of Theorem 2.1. We show both upper and lower bounds for \( \mathcal{J}_{O_m}(\alpha) \) starting from Equation (8). For the upper bound, we have that \( C_k \leq k + I_0 + R_0 \) by assumption, \( C_k \leq 3m \). Moreover, by assumption, \( C_k \leq m + C_0 \leq \frac{m^2}{2} \). Plugging these into (8) results in

\[
\mathcal{J}_{O_m}(\alpha) \leq \sum_{k=0}^{m-1} \Pr(E_{k-1} = 1) \frac{N^2 (3m)^2}{P^2 (P - \frac{1}{2}P)((P - \frac{1}{2}P) + \frac{3}{2}P)} \leq H_1 \frac{m^3 N^2}{\alpha^4},
\]

for a constant \( H_1 \). For the lower bound, we first prove a lower bound for \( \Pr(E_m = 1) \).

Lemma 3.12. There exists a constant \( D \) that only depends on \( \beta \) and \( \gamma \) such that if \( \frac{\beta (P - m - C_0)}{\beta (P - m - C_0) + P} > p \) and \( I_0 \geq D \), then \( \Pr(E_m = 1) \geq \frac{1}{2} \).

This result relies on an interesting stochastic dominance argument and can be found in the Appendix. Then, similarly to the upper bound, \( \mathcal{J}_{O_m}(\alpha) \geq H_2 \frac{m^2}{\alpha^2} \), follows from using \( \Pr(E_m = 1) \geq \frac{1}{2} \) and the fact that \( C_k \geq \frac{k + I_0 + 2R_0}{2} \) when \( E_k = 1 \) (Lemma 3.5). Combining the upper and lower bounds finish the proof.
4 Forecasting in the real world

This section describes a practical SIR model that can incorporate a number of real-world features and datasets. We then consider an approximation to the MLE estimate for this model. Finally we propose an approach to overcome the difficulty in learning α. We present comprehensive experimental results at a ‘regional’ granularity (essentially close to county level) that illustrate strong relative merits.

Discrete-time SIR  Recall the stochastic SIR process, \((S(t), I(t), R(t)) : t \geq 0\), a multi-variate counting process determined by parameters \((α, β, γ)\). We now allow \(β\) to be time-varying, yielding a counting process with jumps \(C_k - C_{k-1} \sim \text{Bern}\{βS_{k-1}/(βS_{k-1} + αNγ)\}\) and rate \(λ(t) = (β(t)S(t)/αN + γ)I(t)\). We obtain discrete-time SIR processes, \((S_i[t], I_i[t], R_i[t]) : t \in \mathbb{N}\) for regions \(i \in \mathcal{I}\) by considering the Euler-approximation to this counting process (e.g. \([22]\)). Specifically, let \(ΔI[t] = I[t] - I[t-1]\), and define \(ΔS[t]\) and \(ΔR[t]\) analogously. The discrete-time approximation to the SIR process is then given by:

\[
\begin{align*}
ΔS_i[t+1] &= -β_i[t](S_i[t]/α_iN_i)I_i[t] + ν_i^S[t], \\
ΔI_i[t+1] &= β_i[t](S_i[t]/α_iN_i)I_i[t] - γI_i[t] + ν_i^I[t], \\
ΔR_i[t+1] &= γI_i[t] + ν_i^R[t],
\end{align*}
\]

where \(ν_i^S[t], ν_i^I[t], ν_i^R[t]\) are appropriately defined martingale difference sequences.

Model Parameters and Covariates  For each region \(i\), the observability parameter \(α_i\) and reproduction factor \(β_i\) must be learned from data, but we assume the total population \(N_i\) and recovery rate \(γ\) are known (a typical assumption; for COVID-19, \(γ \sim 1/4\)). Demographic and mobility factors influence the reproduction rate of the disease. To model these effects, we estimate \(β_i[t]\) as a mixed effects model incorporating these covariates: \(β_i[t] = \exp(X_i[t]θ) + ϵ_i\), where \(X_i[t] = (Z_i, M_i[t]) \in \mathbb{R}^{d_1+d_2}\) is a set of observed covariates for region \(i\) partitioned into static \(Z_i \in \mathbb{R}^{d_1}\) and time-varying \(M_i[t] \in \mathbb{R}^{d_2}\). We estimate the parameters \(ϵ_i\), representing stationary per-region random effects, and \(θ \in \mathbb{R}^{d_1+d_2}\), a vector of fixed effects shared across regions.

4.1 Learning and a Differentiable Model

In the real world, the SIR model is a latent process – we never directly observe any of the state variables \(S_i[t], I_i[t], R_i[t]\). Instead, we observe \(C_i[t] = I_i[t] + R_i[t] = α_iN_i - S_i[t]\). Note that other processes (deaths, hospitalizations, etc.) that depend on the latent state may also be observable, and can easily be incorporated into this learning scheme. The MLE problem for parameters \((θ, α, ϵ)\) is simply \(\max_{θ,α,ϵ} \sum_{i,t} \log P(C_i[t]|θ, α, ϵ)\).

This is a difficult non-linear filtering problem (and an interesting direction for research). We therefore consider an approximation: Denote by \(\{(s_i[t], i[t], r_i[t]) : t \in \mathbb{N}\}\) the deterministic process obtained by ignoring the martingale difference terms in the definition of the discrete time SIR process. We consider the approximation

\[
C_i[t] = α_iN_i - S_i[t] \sim (α_iN_i - s_i[t])ω_i[t]
\]

where \(ω_i[t]\) is log-normally distributed with mean 1 and variance \(\exp(σ^2) - 1\). Under this approximation, the MLE problem is now transformed to

\[
\min_{θ,α,ϵ} \sum_{i,t} \left( \log C_i[t] - \log (α_iN_i - s_i[t]) \right)^2
\]

which constitutes a differentiable model. We solve (9), (or a weighted version) using Adam \([23]\).

Working around the limits to learning  Theorem \([3,4]\) asserts that \(β\) is easy to learn via MLE, suggesting that the parameters \(θ, \{ϵ_i\}\) underlying our model of \(β\) can be estimated as well. However, Corollary \([3,2]\) illustrates that we cannot estimate \(\{α_i\}\) in reasonable time or accuracy from infections alone. This necessitates augmenting the estimation problem (9) with auxiliary information.

1Adam was run for 20k iterations, with learning rate tuned over a coarse grid. A weighted version of the loss function in (9) with weights for \((i, t)\)th observation set to \(C_i[t]\) worked well.
We propose to take advantage of heterogeneity across regions: infection curves start at different times in each region, and we typically have access to some set $P[t] \subseteq \mathcal{I}$ of regions that have already experienced enough infections to reliably estimate $\alpha_i$ for $i \in P[t]$ via MLE. At a high level, our strategy will be to identify the set $P[t]$, estimate $\alpha_i$ for $i \in P[t]$, then extrapolate these estimates to obtain $\alpha_i$ for $i \notin P[t]$. We define the set

$$P[t] = \{ i \in \mathcal{I} : C_i[t] - C_i[t-1] \leq -\gamma_1 \max_{\tau \leq t} (C_i[\tau] - C_i[\tau-1]) \}$$

(10)

where $\gamma_1$ is a hyperparameter. In the fluid model this identifies the first time $t$ where $d^2 s(t)/dt^2 < 0$.

We expect $\alpha_i$ to vary across regions due to different demographics (that impact asymptomatic rates) and testing policies. Given a set of parameters $\{\hat{\alpha}_i : i \in P[t]\}$ estimated via MLE, a natural approach to estimate $\alpha_i$ for regions $i \notin P[t]$ is by matching to regions in $P[t]$ with similar covariates. It is also worth noting that since in large systems in the fluid regime, we may show that $C_i(t)/N_i \rightarrow \alpha_i$ as $t \rightarrow \infty$, we also enforce $\alpha_i \geq C_i[t]/N_i$.

**Overall Learning Algorithm (‘Two-Stage’)**  
So motivated, given data up to time $T$, we now define our overall learning algorithm ‘Two-Stage’, as follows:

$$\alpha_i = \begin{cases} 
\exp(\phi^T Z_i + \delta_i), & \text{if } i \in P[T] \\
\max \left\{ \exp(\phi^T Z_i), \gamma_2 C_i[T]/N_i \right\}, & \text{otherwise}
\end{cases}$$

where $\gamma_2$ is a hyper-parameter and $\delta_i$ are region specific random effect terms. We then estimate the model parameters $(\theta, \phi, \delta, \epsilon)$ by minimizing the following specialization of (9):

$$\min_{(\theta, \phi, \delta, \epsilon)} \sum_{i,t} \left( \log C_i[t] - \log (\alpha_i (\phi, \delta_i) N_i - s_i[t]) \right)^2$$

(11)

The hyper-parameters $\gamma_1, \gamma_2$ and the learning rates are tuned via cross-validation.

### 4.2 Experimental results

We use our estimates to forecast cumulative infections in the US at the level of sub-state ‘regions’, corresponding broadly to public health service areas. The median state has seven regions. The static covariates $Z_i$ for each region $i$ comprise of 60 demographic features from multiple data providers, ranging from socio-economic to health indicators. The time varying covariates $M_i(t)$ correspond to mobility data inferred from aggregated cell phone data. All datasets are widely available and are in active use in other models. The Appendix provides a detailed description of these covariates.

This section focuses on two goals: First, we perform an ablation study focused on the impact of assumed structure on the parameter $\alpha_i$. This allows us to understand whether and why optimizing (11) provides improved performance. Second, and perhaps more importantly, our overall goal was simply to produce accurate forecasts; here we compare the performance of our approach to published forecast performance for the most broadly cited forecaster for COVID-19, [3].

**The impact of learning $\alpha$:** We compare four approaches to learning $\alpha$: 
*Default*: Ignoring the modeling of $\alpha$ altogether by setting $\alpha_i = 1 \forall i \in \mathcal{I}$ (the ‘default’ SIR choice); 
*MLE*: Learn $\alpha_i$ for each region via the MLE approximation (9); 
*Two-Stage*: The two-stage approach specified by (11); 
*Idealized*: Using a value of $\alpha$ computed in-sample (i.e. by looking into the future) via (9).

Figure 1 shows weighted mean absolute percentage error (WMAPE) over regions, with weights proportional to infections on May 21, 2020, for two metrics relevant to decision making: cumulative infections by May 21, 2020 and maximum daily infections, for regions that have peaked by May 21, 2020. Model vintages vary along the x-axis so that moving from left to right models are trained on data approaching the target date of May 21.

*Default* fails, with a WMAPE over 70% even on May 21 (an in-sample fit). At the other extreme, *Idealized* exhibits consistently low error even for early model vintages. This bears out the prediction of Theorem [3, 4] given $\alpha, \beta$ is easy to learn even early in the infection with few samples. *MLE* performs poorly until close to the target date of May 21 at which point sufficient data is available to learn $\alpha$. This empirically illustrates the difficult of learning $\alpha$, as described in Corollary [3, 2].

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[3]: We do not show this model in Figure 1 as the WMAPEs are substantially larger than the scale shown.
Turning to our proposed approach, we see that Two-Stage significantly outperforms MLE far away from the test date. Close to the test date the two approaches are comparable. For maximum daily infections – perhaps the most critical metric for capacity planning – MLE drastically underperforms Two-Stage far from the test date. Our approach to learning from peaked regions significantly mitigates the difficulty of learning $\alpha$. As further evidence, Figure 2 shows how well $\alpha$ from the April 21 vintage predicts $\alpha$ in the final May 21 vintage, for regions that have peaked by May 21. As Proposition 3.3 predicts, the April 21 $\alpha$ are close to the May 21 $\alpha$ values for peaked regions, in both MLE and Two-Stage models. For non-peaked regions, the April 21 MLE $\alpha$ estimates vary much more than the Two-Stage estimates, and tend to underestimate the true $\alpha$ value.

**Overall model performance** Finally, we compare our analyzed models to the widely used IHME model [3]. Figure 3 compares state-level WMAPE for MLE, Two-Stage and IHME models, for vintages stretching back 28 days. The IHME model is, in effect, an SI model with carefully tuned parameters. We report published IHME forecasts; 10 vintages of that model were reported between April 21 and May 21. Two Stage dominates IHME across all model vintages.

---

3Due to IHME only providing state-level predictions. Additionally IHME only offers deaths predictions for these vintages; we show WMAPE on deaths for IHME and WMAPE on infections for MLE and Two-Stage.
Figure 3: WMAPE for predicting state-level cumulative cases on May 21, 2020, comparing MLE and the Two-Stage approach against IHME.

**Broader Impact**

In contrast with the plurality of available forecast models (which make predictions at the level of a state), the models we construct here are at a much finer level of granularity (essentially, the county) and provide accurate predictions early in an epidemic. As a result our models can guide the deployment of resources in states with heterogeneity in resource availability and prevalence. The model was deployed in such an operational fashion in a large US state, where it was used to proactively place hospital resources (mobile surge capacity) in areas where we anticipated large peaks in infections. Many of these predictions were proven accurate and timely in hindsight. This would not be possible with a state-level forecast (let alone a forecast with limited predictive power). While not every state was able to make such data driven decisions in resource deployment, we believe that in the event of a second outbreak, the approach we develop here can serve this need broadly.

**Acknowledgements**

The authors express their gratitude to Danial Mirza, Suzana Iacob, El Ghali Zerhouni, Neil Sanjay Pendse, Shen Chen, Celia Escribe and Jonathan Amar for their excellent support in data collection and organization.

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Appendices

Appendices A and B contain the proofs of Theorems 3.1 and 3.4 respectively. Appendix C contains the proofs of Propositions 2.1, 2.2, and 3.3 each in their own subsections. Appendix D contains a result justifying the use of (10) as a sufficient condition in reliably estimating \( \alpha \). Appendix E contains a description of the covariates used in the practical SIR model.

A Proof of Theorem 3.1

We finish the sections of the proof that were not included in the main paper. This includes the proof of Lemma 3.5, Lemma 3.12 and calculations for Lemma 3.6.

We define \( \lambda(\alpha, k - 1, C_{k-1}) = \left( \frac{\beta(\alpha N - C_{k-1})}{\alpha N} + \gamma \right) I_{k-1} \) and \( \eta(\alpha, C_{k-1}) = \frac{\beta(\alpha N - C_{k-1})}{\beta(\alpha N - C_{k-1}) + \alpha N \gamma} \).

Thus, for \( k \leq \tau \), \( \lambda(N, k - 1, C_{k-1}) \) is the mean of the \( k \)-th inter-arrival time and \( \eta(\alpha, C_{k-1}) \) is the probability that the arrival in the \( k \)-th instance is a new infection rather than a recovery.

A.1 Proof of Lemma 3.5

Proof. Suppose \( k < \tau \) i.e. \( E_k = 1 \). Then, \( k \) is equal to total number of jumps that have occurred so far (the number of movements from S to I and from I to R). The number of individuals that have moved from S to I is \( C_k - I_0 - R_0 \), and the number of movements from I to R is \( C_k - I_k - R_0 \). Therefore, \( k = 2C_k - I_0 - I_k - 2R_0 \). Since \( I_k > 0 \), \( C_k > \tau_k \).

Suppose \( k \geq \tau \) i.e \( E_k = 0 \). Then, \( k \) is greater than or equal to the total number of jumps, which is still equal to \( 2C_k - I_0 - I_k - 2R_0 \). Hence \( C_k \leq \tau_k \) in this case.

\[ \square \]

A.2 Proof of Lemma 3.12

Proof. Let \( X_k \sim \text{Bern}(p) \) for \( k = 1, 2, \ldots \). Let \( \{A_k : k \geq 0\} \) be a stochastic process defined by:

\[
A_k = \begin{cases} 
C_0 & \text{if } k = 0 \\
C_0 + X_1 + \cdots + X_k & \text{if } A_i > r_i \forall i < k \\
A_{k-1} & \text{otherwise.}
\end{cases}
\]

Let \( \tau_A = \min\{k : A_k \leq \tau_k\} \) be the “stopping time” of this process.

Claim A.1. \( \Pr(\tau \leq m) \leq \Pr(\tau_A \leq m) \).

The proof of this claim involves showing the process \( \{A_k\} \) is stochastically less than \( \{C_k\} \); the proof can be found in Section A.2.1. We now upper bound \( \Pr(\tau_A \leq m) \). \( \tau_A \leq m \) if and only if \( A_k \leq \tau_k \) for some \( k \leq m \). Before this happens, \( A_k = C_0 + X_1 + \cdots + X_k \). Therefore, if \( \tau_A \leq m \), it must be that \( C_0 + X_1 + \cdots + X_k \leq \frac{k + I_0 + 2R_0}{2} \) for some \( k \leq m \).

\[
\Pr(\tau_A \leq m) \leq \sum_{k=1}^{m} \Pr\left( C_0 + X_1 + \cdots + X_k \leq \frac{k + I_0 + 2R_0}{2} \right) \tag{13}
\]

\[
= \sum_{k=1}^{m} \Pr\left( X_1 + \cdots + X_k < pk \left( 1 - \frac{2pk - k + I_0}{2pk} \right) \right). \tag{14}
\]
Since $E[X_1 + \cdots + X_k] = pk$, using the Chernoff bound (multiplicative form: $\Pr(\sum_{i=1}^k X_i \leq (1-\delta)\mu) \leq \exp(-\delta^2\mu/2)$) gives

$$\Pr(\tau_A \leq m) \leq \sum_{k=1}^m \exp \left( -\frac{pk}{2} \left( \left( 1 - \frac{1}{2p} \right)^2 + \frac{I_0}{2pk} \right) \right)$$

(15)

$$= \sum_{k=1}^m \exp \left( -\frac{pk}{2} \left( 1 - \frac{1}{2p} \right)^2 - \frac{I_0}{2} \left( 1 - \frac{1}{2p} \right) - \frac{I_0^2}{8pk} \right)$$

(16)

$$\leq \sum_{k=1}^m \exp \left( -\frac{pk}{2} \left( 1 - \frac{1}{2p} \right)^2 - \frac{I_0}{2} \left( 1 - \frac{1}{2p} \right) \right)$$

(17)

$$\leq \exp \left( -\frac{1}{2} - \frac{1}{4p} \right) I_0 \sum_{k=1}^m \exp \left( -\frac{pk}{2} \left( 1 - \frac{1}{2p} \right)^2 \right)$$

(18)

$$\leq B_1 \exp(-B_2 I_0),$$

(19)

for constants $B_1 = \sum_{k=1}^\infty \exp \left( -\frac{pk}{2} \left( 1 - \frac{1}{2p} \right)^2 \right)$, $B_2 = \frac{1}{2} - \frac{1}{4p} > 0$. ($B_1$ is a constant since it is a geometric series with a ratio smaller than 1, since $p > 1/2$.) Let $D$ be the solution to $B_1 \exp(-B_2 D) = \frac{1}{2}$. Then, if $I_0 \geq D$, $\Pr(E_m) = 1 - \Pr(\tau \leq m) \geq 1 - \Pr(\tau_A \leq m) \geq \frac{1}{2}$.

A.2.1 Proof of Claim B.5

Definition A.2. For scalar random variables $X, Y$, we say that $X$ is stochastically less than $Y$ (written $X \leq_{st} Y$) if for all $t \in \mathbb{R}$,

$$\Pr(X > t) \leq \Pr(Y > t).$$

(20)

For random vectors $X, Y \in \mathbb{R}^n$ we say that $X \leq_{st} Y$ if for all increasing functions $\phi : \mathbb{R}^n \to \mathbb{R}$,

$$\phi(X_1, \ldots, X_n) \leq_{st} \phi(Y_1, \ldots, Y_n).$$

(21)

We make use of the following known result for establishing stochastic order for stochastic processes.

Theorem A.3 (Veinott 1965). Suppose $X_1, \ldots, X_n, Y_1, \ldots, Y_n$ are random variables such that $X_1 \leq_{st} Y_1$ and for any $x \leq y$,

$$(X_k | X_1 = x_1, \ldots, X_{k-1} = x_{k-1}) \leq_{st} (Y_k | Y_1 = y_1, \ldots, Y_{k-1} = y_{k-1})$$

(22)

for every $2 \leq k \leq n$. Then, $(X_1, \ldots, X_n) \leq_{st} (Y_1, \ldots, Y_n)$.

Proof of Claim B.5 Because of the condition $\frac{\beta(P-m-C_0)}{\beta(P-m-C_0) + \beta(0)} > p$, for $k \leq m$ and $k \leq \tau$, $C_k - C_{k-1} \sim \text{Bern}(q)$ for $q > p$. First, we show $(A_0, A_1, \ldots, A_m) \leq_{st} (C_0, C_1, \ldots, C_m)$ using Theorem A.3, $C_0 \leq_{st} A_0$ since $C_0 = A_0 = I_0$. We condition on $A_{k-1} = x$ and $C_{k-1} = y$ for $x \leq y$, and we must show $A_k \leq_{st} C_k$. (We do not need to condition on all past variables since the both processes are Markov.) If $x \leq r_{k-1}$, then $A_k = A_{k-1} = x \leq y = C_{k-1} \leq C_k$. Otherwise, the process $A_k$ has not stopped, and neither has $C_k$ since $y \geq x$. Then, $A_k \sim x + \text{Bern}(p)$ and $C_k \sim y + \text{Bern}(q)$ for some $q \geq p$. Clearly, $A_k \leq_{st} C_k$ in this case. We apply Theorem A.3 which implies $A_m \leq_{st} C_m$.

Define the function $u : \mathbb{R}^{m+1} \to \{0,1\}, u(x_0, x_1, \ldots, x_m) = 1\{\cup_{k=1}^m \{x_k \leq r_k\}\}$. Then, $u(A_0, A_1, \ldots, A_m) = 1$ if and only if $\tau_A \leq m$, and $u(C_0, C_1, \ldots, C_m) = 1$ if and only if $\tau \leq m$. $u$ is a decreasing function. Therefore, $u(A_0, A_1, \ldots, A_m) \geq_{st} u(C_0, C_1, \ldots, C_m)$. Then, $\Pr(\tau \leq m) = \Pr(u(C_0, C_1, \ldots, C_m) \geq 1) \leq \Pr(u(A_0, A_1, \ldots, A_m) \geq 1) = \Pr(\tau_A \leq m)$ as desired. □
A.3 Calculations for Lemma 3.6

**Derivation of** $\mathbb{E}_{C_k}[g_{C_k}(C_k, C_{k-1}, \alpha)|E_{k-1} = 1]$. When $E_{k-1} = 1$, we have $C_k \sim C_{k-1} + \text{Bern}(\eta(\alpha, C_{k-1}))$. Therefore, $\mathbb{E}_{C_k}[g_{C_k}(C_k, C_{k-1}, \alpha)|E_{k-1} = 1] = J_{C_k \sim \text{Bern}(\eta(\alpha, C_{k-1}))}(\alpha)$. We reparameterize to write the Fisher information as:

$$
\mathbb{E}_{C_k}[g_{C_k}|C_{k-1}(C_k, C_{k-1}, \alpha)|E_{k-1} = 1] = J_{C_k \sim \text{Bern}(\eta)}(\eta) \left( \frac{\partial}{\partial \alpha} \eta(\alpha, C_{k-1}) \right)^2 \tag{23}
$$

Substituting, $\eta(\alpha, C_{k-1}) = \frac{\beta(\alpha N - C_{k-1})}{\beta(\alpha N - C_{k-1}) + \gamma N}$ to derive

$$
\frac{\partial}{\partial \alpha} \eta(\alpha, C_{k-1}) = \frac{\beta N(\beta(\alpha N - C_{k-1}) + \gamma N) - \beta N(\alpha N - C_{k-1})(\beta + \gamma)}{(\beta N(\alpha N - C_{k-1}) + \gamma N)^2}
$$

$$
= \frac{\beta N \gamma C_{k-1}}{(\beta(\alpha N - C_{k-1}) + \gamma N)^2} \tag{25}
$$

Therefore, $\mathbb{E}_{C_k}[g_{C_k}|C_{k-1}(C_k, C_{k-1}, \alpha)|E_{k-1} = 1] = \frac{\beta(\alpha N - C_{k-1}) + \gamma N)^2}{(\beta(\alpha N - C_{k-1}) + \gamma N)^2} \left( \frac{\beta N \gamma C_{k-1}}{(\beta(\alpha N - C_{k-1}) + \gamma N)^2} \right)^2 \tag{26}
$$

Also, $\frac{1}{(1-\eta)} = \frac{(\beta(\alpha N - C_{k-1}) + \gamma N)^2}{(\alpha N - C_{k-1})\beta \alpha N \gamma}$.

**Derivation of** $\mathbb{E}_{T_k}[g_{T_k}|C_{k-1}(T_k, C_{k-1}, \alpha)|E_{k-1} = 1]$. Similarly, conditioned on $E_{k-1} = 1, T_k \sim \text{Exp}(\lambda(\alpha, k-1, C_{k-1}))$. Therefore, $\mathbb{E}_{T_k}[g_{T_k}|C_{k-1}(T_k, C_{k-1}, \alpha)] = J_{T_k \sim \text{Exp}(\lambda(\alpha, k-1, C_{k-1}))}(\alpha)$. We reparameterize to write

$$
\mathbb{E}_{T_k}[g_{T_k}|C_{k-1}(T_k, C_{k-1}, \alpha)] = J_{T_k \sim \text{Exp}(\lambda)}(\lambda) \left( \frac{\partial}{\partial \alpha} \lambda(\alpha, k-1, C_{k-1}) \right)^2 \tag{27}
$$

Use $\lambda(\alpha, k-1, C_{k-1}) = (\frac{\beta(\alpha N - C_{k-1})}{\alpha N} + \gamma)(2C_{k-1} - (k - 1) - I_0 - 2R_0)$ to derive

$$
\frac{\partial}{\partial \alpha} \lambda(\alpha, k-1, C_{k-1}) = \frac{N \beta C_{k-1}(2C_{k-1} - (k - 1) - I_0 - 2R_0)}{\alpha N^2}
$$

$$
\frac{1}{(\alpha N - C_{k-1} + \gamma N)(2C_{k-1} - (k - 1) - I_0 - 2R_0)} \tag{29}
$$

Substituting, $\mathbb{E}_{T_k}[g_{T_k}|C_{k-1}(T_k, C_{k-1}, \alpha)] = \frac{\beta N C_{k-1}}{(\alpha N(\beta(\alpha N - C_{k-1}) + \gamma N)^2} \tag{30}
$$

$$
= \frac{\beta N C_{k-1}}{(P(\beta - C_{k-1}) + \gamma P)^2} \tag{31}
$$

Derivation of $J_{O_m}(\alpha)$. Using the expressions derived above for $\mathbb{E}_{C_k}[g_{C_k}|C_{k-1}(C_k, C_{k-1}, \alpha)|E_{k-1} = 1]$ and
\[ E_T \left[ g_{T_{k} | C_{k-1}} (T_k, C_{k-1}, \alpha) \right], \text{ we get} \]
\[ E_{C_k} \left[ g_{C_k | C_{k-1}} (C_k, C_{k-1}, \alpha) | E_{k-1} = 1 \right] + E_{T_k} \left[ g_{T_k | C_{k-1}} (T_k, C_{k-1}, \alpha) \right] \]
\[ = \frac{\beta N^2 C_k^{-1}}{P(P - C_{k-1})(\beta(P - C_{k-1}) + P)^2} \left( \frac{\beta N C_{k-1}}{P(\beta(P - C_{k-1}) + \gamma P)} \right)^2 \]
\[ = \frac{P^2(P - C_{k-1})(|P - C_{k-1}| + 2 \gamma P)}{N^2 C_k^{-1}} \]

Thus,
\[ J_{O_m}(\alpha) = \sum_{k=1}^{m} E \left[ g_{C_k | C_{k-1}} (C_k, C_{k-1}, \alpha) | g_{T_k | C_{k-1}} (T_k, C_{k-1}, \alpha) | E_{k-1} = 1 \right] \Pr(E_{k-1} = 1) \]
\[ = \sum_{k=1}^{m} E \left[ \frac{N^2 C_k^{-1}}{P^2(P - C_{k-1})(|P - C_{k-1}| + 2 \gamma P)} | E_{k-1} = 1 \right] \Pr(E_{k-1} = 1). \]

**B Proof of Theorem 3.4**

Fix an instance in which the assumptions of the theorem statement hold. We remove the subscript \( n \),
and let \( P = \alpha N \). Let \( p = \frac{1}{2} \left( \frac{3}{\beta^+ \gamma} + \frac{1}{2} \right) > \frac{1}{2} \), Let \( \hat{A} = \frac{C_{k-1} - C_0}{m} \) be an estimator for \( \frac{\beta}{\beta^+ \gamma} \), \( \hat{B} = \frac{C_{k-1} - C_0}{m} \) be an estimator for \( \frac{1}{\beta^+ \gamma} \) for \( \hat{S}_m = \sum_{k=1}^{\min(m, \tau)} I_{k-1} T_k \). Let \( \hat{\beta} = \hat{A} / \hat{B} \) and \( \hat{\gamma} = 1 / \hat{B} - \hat{\beta} \).

This first lemma follows from [19] of the proof of Lemma 3.12.

**Lemma B.1.** If \( \frac{\beta}{\beta^+ \gamma} \frac{P - m - C_0}{P} > p \), \( \Pr(\tau < m) \leq B_1 e^{-B_2 I_0} \), where \( B_1, B_2 > 0 \) are constant that depend only on \( \beta \) and \( \gamma \).

The next two lemmas give a high probability confidence bound for estimators \( \hat{\beta} \) and \( \hat{\gamma} \).

**Lemma B.2.** For any \( m, I_0 \) where \( \frac{\beta}{\beta^+ \gamma} \frac{P - m - C_0}{P} > 1/2 \), for any \( \delta > 0 \),
\[ \Pr \left( \frac{C_{m} - C_0}{m} \notin \left[ \frac{\beta}{\beta + \gamma} (1 - \delta) \frac{P - m - C_0}{P}, \frac{\beta}{\beta + \gamma} (1 + \delta) \right], \tau \geq m \right) \leq 2 \exp(-m \delta^2/(4 + 2 \delta)). \]

**Lemma B.3.** Let \( \hat{S}_m = \sum_{k=1}^{\min(m, \tau)} I_{k-1} T_k \). Then
\[ \Pr \left( \frac{\hat{S}_m}{m} \notin \left[ \frac{(1 - \delta)}{\beta + \gamma}, \frac{(1 + \delta)}{\beta + \gamma} \frac{P - m - C_0}{P}, \tau \geq m \right] \leq 2 e^{-m \delta^2/(4 + 2 \delta)}. \]

The next proposition combines the two estimators from the above lemmas and into estimators \( \hat{\beta} \) and \( \hat{\gamma} \).

**Proposition B.4.** Assume \( \beta > \gamma > 0 \). Let \( I_0 \leq m < P \) such that \( \frac{\beta}{\beta^+ \gamma} \frac{P - m - C_0}{P} > p \). Let \( z = \frac{P - m - C_0}{P} \). Then, for any \( 0 < \delta < 1 \), with probability \( 1 - 4 e^{-m (\delta - \ln(1 + \delta))} - 4 e^{-m \delta^2/(4 + 28)} \)
\[ 2 B_1 e^{-B_2 I_0}, \]
\[ \hat{\beta} \in \left[ \frac{(1 - \delta) z^2}{1 + \delta}, \frac{1 + \delta}{1 - \delta} \right], \quad \hat{\gamma} \in \left[ \frac{z}{1 + \delta} + \beta \frac{(1 - \delta) z - (1 + \delta)^2}{(1 + \delta)(1 - \delta)}, \frac{1 + \delta - (1 - \delta)^2 z^2}{(1 - \delta)(1 + \delta)} \right], \]

where \( B_1, B_2 > 0 \) are constants that depend on \( \beta \) and \( \gamma \).

We first show Theorem 3.4 using these results. We then prove Lemma B.2, Lemma B.3, and Proposition B.4 in Appendix B.2.
B.1 Proof of Theorem 3.4

Proof. Let $\delta = \sqrt{5 \log m \over m}$. First, we claim that the probability in Proposition B.4 is greater than $1 - 8 m^{-2} B_1 e^{-B_2 I_0}$. Note that $\ln(1 + \delta) \leq \delta - \delta^2 \over 2 + \delta^3$, implying $\delta - \ln(1 + \delta) \geq \delta^4 (1 - \delta)$. Since $\delta \leq {1 \over 4}$,

$$4 e^{-m (\delta - \ln(1 + \delta))} \leq 4 e^{-m \delta^2 \over 2} \leq {4 \over m}. \tag{40}$$

Using $\delta \leq {1 \over 4}$ again,

$$4 e^{-m \delta^2 / (4 + 2 \delta)} \leq 4 e^{-m \delta^2 \over 2} = {4 \over m}. \tag{41}$$

Hence, the bound in B.4 holds with probability greater than $1 - 8 m^{-2} B_1 e^{-B_2 I_0}$.

Since we assume $m(m + C_0) \leq P$ and $z = 1 - m + C_0 \over P$,

$$1 - z \leq {1 \over m}. \tag{42}$$

Let $R$ be the event that the confidence bounds (38)-(39) hold. Note that $1 + \delta \leq 1 + 3 \delta$ and $1 + z \leq 2$.

Similarly,

$$\beta^2 (1 + 3 \delta - (1 - 3 \delta) z^2) \leq \beta^2 ((1 - z) + 3 \delta (1 + z))^2 \tag{43}$$

$$\leq \beta^2 \left( {1 \over m} + 6 \sqrt{5 \log m \over m} \right)^2 \tag{44}$$

$$\leq \beta^2 M_3 \log m \over m \tag{46}$$

for an absolute constant $M_3 > 0$. The second last step uses (42) and $1 + z \leq 2$.

Similarly,

$$\beta^2 (1 + 3 \delta - (1 - 3 \delta) z^2) \leq \beta^2 \left( {1 \over m} + 6 \sqrt{5 \log m \over m} \right)^2 \tag{44}$$

Using the fact that $(1 - \delta)(1 + \delta) \geq {1 \over 2}$,

$$1 \over 1 - \delta - z \over 1 + \delta \leq 2 ((1 - z) + \delta (1 + z)) \leq 2 \left( {1 \over m} + 2 \sqrt{5 \log m \over m} \right). \tag{48}$$

$$z (1 + \delta - (1 - \delta)^2 z^2 \over (1 - \delta)(1 + \delta) - (1 - \delta) z (1 + \delta)^2 \over (1 + \delta)(1 - \delta) \right)^2 \tag{49}$$

$$\leq 2 (1 - z) + 4 \delta (1 + z) + 4 \delta \over 1 - \delta \leq 1 + \delta \over 1 + \delta \tag{50}$$

$$\leq 2 (1 - z) + 8 \delta + (1 + 3 \delta) - (1 - 3 \delta) z^2 \tag{51}$$

$$\leq 2 (1 - z) + 8 \delta + (1 - z^2) + 6 \delta (1 + z^2) \tag{52}$$

$$\leq (1 - z) (3 + z) + \delta (8 + 6 (1 + z^2)) \tag{53}$$

$$\leq {4 m \over 20} \sqrt{5 \log m \over m}. \tag{54}$$
Substituting back into (47) results in
\[
\mathbb{E}[(\hat{\gamma} - \gamma)^2 | R] \leq \left( \gamma \left( \frac{2}{m} + 4\sqrt{\frac{5 \log m}{m}} \right) + \beta \left( \frac{4}{m} + 20\sqrt{\frac{5 \log m}{m}} \right) \right)^2
\] (55)
\[
\leq M_4 \beta^2 \frac{\log m}{m},
\] (56)
for an absolute constant \(M_4\), since \(\beta > \gamma\).
Therefore,
\[
\mathbb{E}[(\hat{\beta} - \beta)^2] \leq \mathbb{E}[(\hat{\beta} - \beta)^2 | R] + \beta^2 \max \Pr(\tilde{R})
\leq M_3 \beta^2 \frac{\log m}{m} + \beta^2 \max \left( \frac{8}{m} + 2B_1 e^{-B_2 I_0} \right)
\leq M_1 \beta^2 \frac{\log m}{m} + 2B_1 \beta^2 e^{-B_2 I_0},
\] (58)
for an absolute constant \(M_1 > 0\). Similarly,
\[
\mathbb{E}[(\hat{\gamma} - \gamma)^2] \leq M_4 \beta^2 \max \left( \frac{8}{m} + 2B_1 e^{-B_2 I_0} \right)
\leq M_2 \beta^2 \frac{\log m}{m} + 2B_1 \gamma^2 e^{-B_2 I_0}.
\] (60)
\[
\] (61)
\[\square\]

### B.2 Proofs of Intermediate Results

#### B.2.1 Proof of Lemma B.2

**Proof.** Fix \(m\), let \(z := \frac{\nu - m - C_0}{p} = \frac{\nu}{p - \gamma} z\). Then \(p \geq \frac{1}{2}\). Define three stochastic processes \(\{A_k : k \geq 0\}, \{B_k : k \geq 0\}, \{\tilde{C}_k : k \geq 0\}\):

\[
A_k = \begin{cases} C_0 & \text{if } k = 0 \\ A_{k-1} + \text{Bern}(p) & \text{otherwise}. \end{cases}
\] (62)

\[
B_k = \begin{cases} C_0 & \text{if } k = 0 \\ B_{k-1} + \text{Bern}(p/z) & \text{otherwise}. \end{cases}
\] (63)

\[
\tilde{C}_k = \begin{cases} C_0 & \text{if } k = 0 \\ \tilde{C}_{k-1} + \text{Bern} \left\{ \frac{\beta (p - \tilde{C}_{k-1})}{\beta (p - \tilde{C}_{k-1}) + p} \right\} & \text{otherwise}. \end{cases}
\] (64)

Note that \(\tilde{C}_k\) is a modified version of \(C_k\) where \(\tilde{C}_k\) still evolves after the stopping time.

**Claim B.5.** \(A_m\) is stochastically less than \(\tilde{C}_m\) (\(A_m \leq_{st} \tilde{C}_m\)); \(\tilde{C}_m\) is stochastically less than \(B_m\) (\(\tilde{C}_m \leq_{st} B_m\)); that is, for any \(\ell \in \mathbb{R}\),
\[
\Pr(B_m \leq \ell) \leq \Pr(\tilde{C}_m \leq \ell) \leq \Pr(A_m \leq \ell).
\] (65)

This claim follows from Theorem [A.3] using a similar argument to Claim B.5.

Let \(A_k = C_0 + X_1 + X_2 + \ldots + X_k\) where \(X_i \sim \text{Bern}(p)\) are independent. We provide the left tail bound for \(C_m\). Note that when \(\tau \geq m\), \(\tilde{C}_m \equiv \tilde{C}_m\). Hence,
\[
\Pr(C_m \leq mp(1 - \delta) + C_0, \tau \geq m) = \Pr(\tilde{C}_m \leq mp(1 - \delta) + C_0, \tau \geq m)
\leq \Pr(\tilde{C}_m \leq mp(1 - \delta) + C_0)
\leq \Pr(A_m \leq mp(1 - \delta) + C_0).
\] (66)

Using the Chernoff bound gives,
\[
\Pr(A_m \leq mp(1 - \delta) + C_0) = \Pr(C_0 + X_1 + \cdots + X_m \leq pm(1 - \delta) + C_0)
= \Pr(X_1 + \cdots + X_m \leq pm(1 - \delta)) \leq \exp \left( - \frac{m \frac{1}{2} \delta^2}{2} \right).
\] (69)

\[
\] (70)
Therefore,
\[
\Pr \left( \frac{C_m - C_0}{m} \leq \frac{p}{z} (1 - \delta) z, \tau \geq m \right) = \Pr(C_m \leq mp(1 - \delta) + C_0, \tau \geq m) \quad (72)
\]
\[
\leq \exp \left( -\frac{mp}{2} \delta^2 \right) \leq \exp(-m\delta^2/4). \quad (73)
\]

Let \( B_k = C_0 + Y_1 + \ldots + Y_k \) where \( Y_i \sim \text{Bern}(p/z) \) are independent. Similarly, for the upper tail bound, we have
\[
\Pr \left( \frac{C_m - C_0}{m} \geq \frac{p}{z} (1 + \delta), \tau \geq m \right) = \Pr(C_m \geq mp/z(1 + \delta) + C_0, \tau \geq m) \quad (74)
\]
\[
\leq \Pr(B_m \geq mp/z(1 + \delta) + C_0) \quad (75)
\]
\[
\leq \Pr(C_0 + Y_1 + \ldots + Y_m \geq mp/z(1 + \delta) + C_0) \quad (76)
\]
\[
\leq \exp(-mp/z \delta^2) \leq \exp(-m\delta^2/(4 + 2\delta)) \quad (77)
\]
due to the multiplicative Chernoff bound \( \Pr(Z \geq E[Z](1 + \delta)) \leq e^{-\frac{2}{1+\delta} \delta^2} \) where \( Z \) is the sum of i.i.d Bernoulli random variables.

Combine upper and lower tail bounds and note that \( p/z = \frac{\beta}{\beta + \gamma} \). Then, we can conclude, for any \( \delta > 0 \),
\[
\Pr \left( \frac{C_m - C_0}{m} \notin \left[ \frac{\beta}{\beta + \gamma}(1 - \delta) z, \frac{\beta}{\beta + \gamma}(1 + \delta) \right], \tau \geq m \right) \leq 2 \exp(-m\delta^2/(4 + 2\delta)). \quad (78)
\]

**B.2.2 Proof of Lemma B.3**

**Proof.** Conditioned on \( (I_0, C_0, I_1, C_1, \ldots, I_{m-1}, C_{m-1}) \) with \( \tau \geq m \), we have
\[
I_{k-1} T_k \sim \text{Exp} \left( \frac{\beta}{\beta + \gamma} \right)
\]
are independent exponential random variables.

Theorem 5.1 in [24] gives us a tail bound for the sum of independent exponential random variables: let \( X = \sum_{i=1}^n X_i \) with \( X_i \sim \text{Exp}(a_i) \) independent, then for \( \delta > 0 \),
\[
\Pr(X \geq (1 + \delta)\mu) \leq \frac{1}{1 + \delta} e^{-a_i \mu(\delta - \ln(1 + \delta))} \leq e^{-a_i \mu(\delta - \ln(1 + \delta))} \quad (79)
\]
\[
\Pr(X \leq (1 - \delta)\mu) \leq e^{-a_i \mu(\delta - \ln(1 + \delta))} \quad (80)
\]
where \( \mu = E[X], a_i = \min_{1 \leq i \leq n} a_i \).

Let \( \tilde{S}_{m|\tilde{c}, \tilde{f}} \) be \( \tilde{S}_m \) conditioned on \( (I_0, C_0, I_1, C_1, \ldots, I_{m-1}, C_{m-1}) \) with \( \tau \geq m \). Let \( \mu = E[\tilde{S}_{m|\tilde{c}, \tilde{f}}] = \sum_{k=1}^m \frac{1}{\beta(P - C_{k-1})/P + \gamma} \), \( a* = \min_{1 \leq k \leq m} \beta(P - C_{k-1})/P + \gamma \). It is easy to verify the following facts
\[
\mu a* \geq \sum_{k=1}^m a* \left( \frac{P - m - C_0}{P} \right) \quad (81)
\]
\[
\frac{1}{\beta + \gamma} \leq \frac{\mu}{m} \leq \frac{1}{\beta + \gamma} \frac{P}{P - m - C_0} \quad (82)
\]

Combining these with Eqs. (79) and (80), we have
\[
\Pr \left( \frac{\tilde{S}_{m|\tilde{c}, \tilde{f}}}{m} \notin \left[ \frac{(1 - \delta)}{\beta + \gamma} \frac{P}{P - m - C_0}, \frac{P}{\beta + \gamma} \right], \tau \geq m \right) \leq 2 \exp(-m\delta^2/(4 + 2\delta)). \quad (83)
\]

Combining these with Eqs. (79) and (80), we have
\[
\Pr \left( \frac{\tilde{S}_{m|\tilde{c}, \tilde{f}}}{m} \notin \left[ \frac{(1 - \delta)}{\beta + \gamma} \frac{P}{P - m - C_0}, \frac{P}{\beta + \gamma} \right], \tau \geq m \right) \leq 2 \exp(-m\delta^2/(4 + 2\delta)). \quad (84)
\]
Therefore,
\[
\Pr\left(\frac{\hat{S}_m}{m} \notin I, \tau \geq m \right) = \int_{\tilde{C}, \tilde{I} \tau \geq m} \Pr\left(\frac{\hat{S}_m}{m} \notin I \mid \tilde{C}, \tilde{I}, \tau \geq m \right) f(\tilde{C}, \tilde{I} \tau \geq m) \Pr(\tau \geq m)
\]
\[
\leq 2e^{-m\frac{p-m-C_h}{p}(\delta - \ln(1+\delta))} \Pr(\tau \geq m)
\]
\[
\leq 2e^{-m\frac{p-m-C_h}{p}(\delta - \ln(1+\delta))}.
\]

B.2.3 Proof of Proposition B.4

Proof. Let \( \hat{\beta} = \frac{C_h - C_h}{S_m} \), \( z = \beta - \frac{C_h - C_h}{\beta + \gamma} \). Suppose \( x \in \frac{\beta}{\beta + \gamma} [1 - \delta, 1 + \delta], y \in \frac{\beta}{\beta + \gamma} [1 - \delta, 1 + \delta] \). Then,
\[
\frac{x}{y} \in \left[ \beta \frac{(1 - \delta)z^2}{1 + \delta}, \frac{1 + \delta}{1 - \delta} \right]
\]

Similarly, let \( \hat{\gamma} = \frac{m}{S_m} - \hat{\beta} \). Suppose \( a \in (\beta + \gamma)[1 + \frac{1}{1 + \delta}, 1 + \frac{1}{1 + \delta}], b \in \beta [(1 - \delta)z^2, 1 + \delta] \). Then
\[
a - b \in \left[ \gamma \frac{1}{1 + \delta} + \beta \frac{(1 - \delta)z - (1 + \delta)^2}{(1 + \delta)(1 - \delta)} , \gamma \frac{1}{1 + \delta} + \beta \frac{1 + \delta - (1 - \delta)^2z^2}{(1 - \delta)(1 + \delta)} \right].
\]

Then, for any sets \( U_1, U_2 \),
\[
\Pr(\hat{\beta} \in U_1, \hat{\gamma} \in U_2) \geq 1 - \Pr(\hat{\beta} \notin U_1) - \Pr(\hat{\gamma} \notin U_2)
\]
\[
\geq 1 - \Pr(\hat{\beta} \notin U_1, \tau > m) - \Pr(\hat{\beta} \notin U_2, \tau > m) - 2 \Pr(\tau < m)
\]
\[
\geq 1 - 4e^{-m(\delta - \ln(1+\delta))} - 4e^{-m(\delta - \ln(1+\delta))} - 2B_1 e^{-B_2 t_0},
\]
where the last step uses Lemma [B.1], Lemma B.2 and Lemma B.3 using the intervals (88) and (89) for \( U_1 \) and \( U_2 \) respectively.

C Proofs of Propositions

C.1 Proof of Proposition 2.1

Proof. As in [25] [26], the solution \( \{ (s'(t), i'(t), r'(t)) : t \geq 0 \} \) with \( \alpha = 1 \) can be written:
\[
s'(t) = s'(0) e^{-\xi'(t)}
\]
\[
i'(t) = N - s'(t) - r'(t)
\]
\[
r'(t) = r(0) + \frac{\gamma'N}{\beta'} \xi'(t)
\]
\[
\xi'(t) = \frac{\beta'}{N} \int_0^t i'(t^*) dt^*
\]

Making the appropriate substitutions yields the following equivalent system:
\[
i'(t) = N - s'(0) \exp\left(-\frac{\beta'}{N} \xi(t)\right) - r(0) - \frac{\gamma'N}{\beta'} \xi'(t)
\]
\[
\xi'(t) = \frac{\beta'}{N} \int_0^t i'(t^*) dt^*.
\]
Therefore, it remains to show that for $\alpha' > 0$, $\{(s(t), i(t), r(t)) : t \geq 0\} \triangleq \{(\alpha' s(t), \alpha' i(t), \alpha' r(t)) : t \geq 0\}$ is a solution for (97) and (98). Starting with (97),

\[
i'(t) = N - s'(0) \exp(-\xi'(t)) - r'(0) - \frac{\gamma' N}{\beta'} \xi'(t)
\]

(99)

\[
\alpha' i'(t) = \alpha' \left( N - s'(0) \exp(-\xi'(t)) - r'(0) - \frac{\gamma' N}{\beta'} \xi'(t) \right)
\]

(100)

\[
= \alpha' N - \alpha s'(0) \exp(-\xi(t)) - \alpha' r(0) - \frac{\gamma' \alpha N}{\beta'} \xi(t)
\]

(101)

where $\xi(t) = \xi'(t) = \frac{\beta'}{N \alpha'} \int_0^t \alpha' i' (t^*) dt^*$. Noting that $\xi'(t) = \xi(t)$ and substituting $i(t) = \alpha' i'(t)$ yields the equations below, clearly showing that $\{(s(t), i(t), r(t)) : t \geq 0\}$ satisfy (97) and (98):

\[
i(t) = \alpha' N - s(0) \exp(-\xi(t)) - r(0) - \frac{\gamma' \alpha N}{\beta'} \xi(t)
\]

(102)

\[
\xi(t) = \frac{\beta'}{N \alpha'} \int_0^t i(t^*) dt^*.
\]

(103)

\begin{proof}
Consider initial conditions $(s(0), i(0), 0)$, as in [25, 26], the analytical solution is given by

\[
s(t) = s(0)e^{-\xi(t)},
\]

\[
i(t) = \alpha N - s(t) - r(t),
\]

\[
r(t) = \gamma \alpha N \xi(t),
\]

\[
\xi(t) = \frac{\beta}{\alpha N} \int_0^t i(t^*) dt^*.
\]

Consider two SIR models with parameters $(\alpha, \beta, \gamma)$ and $(\alpha', \beta', \gamma')$, and initial conditions $(s_0, i_0, 0)$ and $(s'_0, i'_0, 0)$ respectively. We claim that infection trajectories $i(t)$ and $i'(t)$ being identical on an open set $[0, T]$ implies the parameters and initial conditions are identical as well.

Assume $i(t) = i'(t)$ for all $t \in [0, T)$; then, given the exact solution above it follows that

\[
\alpha N - s_0 e^{-\frac{\beta}{\alpha N} x} - \gamma x = \alpha' N - s'_0 e^{-\frac{\beta'}{\alpha' N} x} - \gamma' x, \quad \text{for all } x \in \left[0, \int_0^T i(t) dt \right]
\]

(104)

As functions of $x$, both the RHS and LHS in the equality above are holomorphic, and hence, using the identity theorem, we conclude that $\alpha N = \alpha' N, s_0 = s'_0, -\frac{\beta}{\alpha N} = -\frac{\beta'}{\alpha' N}, \gamma = \gamma'$, which completes the proof.
\end{proof}

\begin{proof}
The crux of the problem is summarised in two smaller results, bounding $T_{1,n}^d$ and $T_{2,n}^d$ respectively. To ease our exposition, we will let $P_n = \alpha_n N_n$ and drop the index $n$ in these helper results.

\begin{proposition}
There exists a constant $\nu_1$ that only depends on $\gamma, \beta$ such that

\[
T_1^d \geq \frac{1}{\beta - \gamma} \left( \frac{2}{3} \log \frac{\nu_1 P}{c(0)^{3/2}} + \log \frac{\nu_1^{2/3}}{c(0)} \left( 1 - \frac{c(0)}{P^{2/3}} \right) \right).
\]

\end{proposition}
Proposition C.2. Let \( \rho_1 = 1 - \frac{1}{\log \log P} \), there exists a constant \( \nu_2 \) that only depends on \( \gamma, \beta \) and a constant \( C = O(1) \), such that

\[
T_2^d \leq \frac{1}{\beta \rho_1 - \gamma} \log \frac{\nu_2 P}{i(0)} + \frac{C}{1 - \rho_1}.
\]

The argument follows directly by taking the limit of the bounds we provide in Propositions C.1-C.2. Specifically, using that the constants \( \nu_1, n, \nu_2, n \) do not change with \( n \), we arrive at

\[
\lim_{n \to \infty} \frac{T_2^d n}{T_1^d n} \leq \lim_{n \to \infty} \frac{\frac{1}{2 \rho_1 - \gamma} \log \frac{\nu_2 P}{i(0)} + \frac{C}{1 - \rho_1}}{\frac{1}{\beta - \gamma} \log P_n + \log \nu_2 - \log i(0)} \\
= \lim_{n \to \infty} \frac{\beta - \gamma}{\beta \rho_1 - \gamma} \left( \frac{1}{2} \log P_n + \frac{4}{3} \log \nu_1 - 2 \log c_n(0) + \log \left( 1 - \frac{c_n(0)}{P_n^{1/3}} \right) \right) \\
+ \lim_{n \to \infty} \frac{\beta - \gamma}{3} \log P_n + \frac{4}{3} \log \nu_1 - 2 \log c_n(0) + \log \left( 1 - \frac{c_n(0)}{P_n^{1/3}} \right)
\]

Since \( c_n(0) = O(\log(P_n)) \) by assumption (and \( i_n(0) \leq c_n(0) \)), and \( C_n = O(1) \) by Proposition C.2, the limits of the two summands above are \( 3/2 \) and \( 0 \) respectively, which concludes the proof.

C.3.1 Proof of Proposition C.1

Proof of Proposition C.1 Define \( \tilde{i}(t) \) such that \( \tilde{i}(0) = i(0) \) and \( \frac{di}{dt} = (\beta - \gamma)\tilde{i} \), implying

\[
\tilde{i}(t) = i(0) \exp\{ (\beta - \gamma)t \}.
\]

Since \( \frac{di}{dt} \geq \frac{di}{dt} \) for all \( t, \tilde{i}(t) \geq i(t) \) for all \( t \). Then, for all \( t \),

\[
\frac{ds}{dt} = -\beta \frac{s}{P} \tilde{i} \geq -\beta i \geq -\beta \tilde{i}.
\]

Hence we can write

\[
s(t) \geq s(0) + \int_0^t -\beta \tilde{i}(t')dt'
\]

\[
= s(0) - \beta i(0) \int_0^t \exp\{ (\beta - \gamma)t' \} dt'
\]

\[
= s(0) - \frac{\beta i(0)}{\beta - \gamma} (\exp\{ (\beta - \gamma)t \} - 1)
\]

Since \( s(0) - s(T_1^d) = P^{2/3} - c(0) \), setting \( t = T_1^d \) and solving for \( T_1^d \) in the inequality above results in

\[
T_1^d \geq \frac{1}{\beta - \gamma} \log \left( \frac{\beta - \gamma}{\beta c(0)} (P^{2/3} - c(0)) \right)
\]

\[
\geq \frac{1}{\beta - \gamma} \log \left( \frac{\beta - \gamma}{\beta c(0)} (P^{2/3} - c(0)) \right)
\]

\[
= \frac{1}{\beta - \gamma} \left( \log \frac{\beta - \gamma}{\beta c(0)} (P^{2/3}) + \log \frac{\beta - \gamma}{\beta c(0)} (1 - \frac{c(0)}{P^{2/3}}) \right)
\]

\[
= \frac{1}{\beta - \gamma} \left( \frac{2}{3} \log \frac{\nu_1 P}{c(0)^{3/2}} + \log \frac{\nu_1^{2/3}}{c(0)} (1 - \frac{c(0)}{P^{2/3}}) \right)
\]

for \( \nu_1 = \left( \frac{\beta - \gamma}{\beta} \right)^{3/2} \) as desired.
C.3.2 Proof of Proposition C.2

For \( \rho \in [0, 1/2] \), let \( t_\rho \) be the time \( t \) when \( s(t) = \rho \). \( \rho \) will represent the fraction of the total population that is susceptible. Since \( \rho \leq 1/2 \), \( i \) is increasing for the time period of interest.

Let \( \beta > \gamma \), \( P \) be fixed. Let \( \rho_1 = 1 - \frac{1}{\log \log P} \) and \( \rho_2 = \frac{7}{8} \). We assume \( P \) is large enough that \( \rho_1 > \rho_2 \), hence \( t_{\rho_1} < t_{\rho_2} \). \( T_d = t_{\rho_2} \).

**Lemma C.3.** For any \( \rho \in [0, 1/2] \), \( i(t_\rho) \geq P(1 - \rho) \frac{\beta \rho - \gamma}{\rho} - \frac{c(0)}{2} \).

**Proof of Lemma C.3.** Fix \( \rho \). At time \( t_\rho \), the total number of people infected is \( c(t_\rho) = i(t_\rho) + r(t_\rho) = P(1 - \rho) \), by definition. At any time \( t \leq t_\rho \), the rate of increase in \( i \) is \( \frac{\beta \rho - \gamma}{\rho} \geq \frac{\beta \rho - \gamma}{\rho} \) of the rate of increase in \( c \). Therefore, \( i(t_\rho) - i(0) \geq (\frac{\beta \rho - \gamma}{\rho}) (c(t_\rho) - c(0)) \) and \( i(t_\rho) \geq (\frac{\beta \rho - \gamma}{\rho}) P(1 - \rho) - \frac{\beta \rho - \gamma}{\rho} c(0) + i(0) \). Using the fact that \( i(0) \geq \frac{c(0)}{2} \) and rearranging terms gives the desired result.

**Lemma C.4.** For \( t \in [t_{\rho_1}, t_{\rho_2}] \), where \( \rho_2 > \rho_1 \) for \( \rho_1, \rho_2 \in [0, 1/2] \), \( t_{\rho_2} - t_{\rho_1} \leq \frac{P(\rho_2 - \rho_1)}{P_2 g(t_{\rho_1})} \).

**Proof of Lemma C.4.** The difference in \( s \) between \( t_{\rho_1} \) and \( t_{\rho_2} \) is \( s(t_{\rho_1}) - s(t_{\rho_2}) = P(\rho_2 - \rho_1) \). As a consequence of the mean value theorem, \( \frac{s(t_{\rho_2}) - s(t_{\rho_1})}{t_{\rho_2} - t_{\rho_1}} \leq \max_{t \in [t_{\rho_1}, t_{\rho_2}]} \left\{ \frac{ds}{dt} \right\} \). Using these two expressions,

\[
\frac{P(\rho_2 - \rho_1)}{t_{\rho_2} - t_{\rho_1}} \geq \min \left\{ \frac{ds}{dt} \right\} = \min \left\{ \beta \frac{s(t)}{P} i(t) : t \in [t_{\rho_1}, t_{\rho_2}] \right\} \geq \beta \rho_2 i(t_{\rho_1})
\]

The desired expression follows from rearranging terms.

**Lemma C.5.** For any \( \rho \leq \min \{ \frac{7}{8}, 1/2 \} \), \( t_\rho \leq \frac{1}{\log \log P} \frac{1}{P} \), for \( \nu_2 = \frac{2(\beta - \gamma)}{\beta} \).

The proof of this lemma follows the exact same procedure as the proof of Proposition C.1

**Proof of Lemma C.5.** We proceed in the same way as the proof of Proposition C.1 except in this case we will lower bound \( s(0) - s(t) \). We achieve this by letting \( \tilde{i} \) be defined to grow slower than \( i \), so it is used as a lower bound. Define \( \tilde{i}(t) \) such that \( \tilde{i}(0) = i(0) \) and \( \tilde{i}^{(1)}(0) = (\beta \rho - \gamma) \tilde{i} \), implying

\[
\tilde{i}(t) = i(0) \exp \{(\beta \rho - \gamma)t\}.
\]

Since \( \frac{d\tilde{i}}{dt} \leq \frac{ds}{dt} \) when , \( \tilde{i}(t) \leq i(t) \) for all \( t < t_{\rho_2} \). In addition, when \( t < t_{\rho_2} \), \( \frac{\tilde{i}(t)}{P} \geq \frac{7}{8} \geq \rho \). Then, for \( t < t_{\rho_2} \),

\[
\frac{ds}{dt} = -\beta \frac{s}{P} \tilde{i} \leq -\beta \rho \tilde{i}.
\]

Hence we can write

\[
s(t) \leq s(0) + \int_0^t -\beta \rho \tilde{i}(t')dt'
\]

\[
= s(0) - \beta \rho \tilde{i}(0) \int_0^t \exp \{(\beta \rho - \gamma)t'\}dt'
\]

\[
= s(0) - \beta \rho i(0) (\exp \{(\beta \rho - \gamma)t\} - 1)
\]

Since \( s(t_\rho) = \rho P \),

\[
\rho P \leq s(0) - \frac{\beta \rho i(0)}{(\beta \rho - \gamma)} (\exp \{(\beta \rho - \gamma)t_\rho\} - 1).
\]
Solving for \( t_p \) results in
\[
    t_p \leq \frac{\log\left(\frac{\beta \rho - \gamma}{\beta \rho N(0)}(s(0) - \rho P) + 1\right)}{\beta \rho - \gamma} \leq \frac{1}{\beta \rho - \gamma} \log\left(\frac{\nu_2}{\nu} P\right) \quad (122)
\]
where \( \nu_2 = \frac{2(\beta \rho - \gamma)}{\beta} \), using the fact that \( \rho \leq 1/2 \).

**Proof of Proposition C.2** Using the results from Lemmas C.3, C.5
\[
    t_{c_2} = t_{p_1} + (t_{p_2} - t_{p_1})
\]
\[
    \leq \frac{1}{\beta \rho_1 - \gamma} \log\left(\frac{\nu_2}{\nu} P\right) + \frac{P(\rho_1 - \rho_2)}{\beta \rho_2 \rho_1(t_{p_1})} \leq \frac{1}{\beta \rho_1 - \gamma} \log\left(\frac{\nu_2}{\nu} P\right) + \frac{\rho_2 P(\rho_1 - \rho_2)}{\beta \rho_1 - \gamma} - \frac{\rho_1}{\beta \rho_1 - \gamma}
\]
\[
    = \frac{1}{\beta \rho_1 - \gamma} \log\left(\frac{\nu_2}{\nu} P\right) + \frac{C}{\beta \rho_1 - \gamma}
\]
where \( C = \frac{\rho_1 - \rho_2}{\beta \rho_1 - \gamma} - \frac{\rho_2}{\beta \rho_1 - \gamma}. \) Note that, as required in the statement, \( C = O(1) \). Indeed,
\[
    C = \frac{(\rho_1 - \rho_2)}{\beta \rho_1 - \gamma} - \frac{\rho_2}{\beta \rho_1 - \gamma} = \frac{1 - \rho_2}{\beta \rho_2 - \beta \rho_1(\rho_1 - \rho_2)} \left(\frac{c(1)}{\log \log P}\right)
\]
and so, as \( P \) grows large, \( C \) tends to \((1 - \rho_2)/\rho_2(\beta - \gamma)\) (recall that \( c(0) = O(\log \log P) \)).

**D Sufficient Condition for \( P[t] \)**

In the practical model, \( P[t] \) represents the regions that have enough observations to reliably estimate \( \alpha \) at time \( t \). In this section, we justify why the definition \( (10) \) is a sufficient condition. Let \( T_{3,n}^d = \inf\{t : \frac{d^2 s}{dt^2} > 0\} \) be the time at which the rate of new infections is highest in the deterministic SIR model.

**Proposition D.1.** As \( n \to \infty \), \( T_{1,n}^d \leq T_{3,n}^d \).

**Proof.**
\[
    \frac{d^2 s}{dt^2} = -\frac{\beta}{\alpha_n N_n} \left( \frac{ds}{dt} i + \frac{di}{dt} s \right)
\]
\[
    = -\frac{\beta}{\alpha_n N_n} \left( \frac{-\beta s}{\alpha_n N_n} \right) + \left( \frac{\beta s}{\alpha_n N_n} - \gamma \right) i s
\]
\[
    = -\beta s \left( \frac{-\beta i + \beta s - \gamma \alpha_n N_n}{\alpha_n N_n} \right)
\]
\[
    = \beta^2 s \left( i - s + \frac{\gamma}{\beta} \alpha_n N_n \right)
\]
From \( (122) \), we see that \( \frac{d^2 s}{dt^2} > 0 \) if and only if
\[
    s < \frac{\gamma}{\beta} \alpha_n N_n + i.
\]
At \( t \leq T_{1,n}^d \), \( c(t) \leq (\alpha_n N_n)^{2/3} \) which implies \( i(t) \leq (\alpha_n N_n)^{2/3} \) and \( s(t) \geq \alpha_n N_n - (\alpha_n N_n)^{2/3} \). We assume \( n \) is large enough so that \( 2(\alpha_n N_n)^{-1/3} < 1 - \frac{\gamma}{\beta} \). Then, \( (128) \) cannot hold:
\[
    2(\alpha_n N_n)^{-1/3} < 1 - \frac{\gamma}{\beta}
\]
\[
    \frac{\gamma}{\beta} + (\alpha_n N_n)^{-1/3} < 1 - (\alpha_n N_n)^{-1/3}
\]
\[
    \frac{\gamma}{\beta} \alpha_n N_n + i(t) \leq \frac{\gamma}{\beta} \alpha_n N_n + (\alpha_n N_n)^{2/3} < \alpha_n N_n - (\alpha_n N_n)^{2/3} \leq s(t).
\]

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Therefore, $T_{d_{1,n}} \leq T_{d_{3,n}}$. 

E Covariate Details for Practical SIR Model

For observed COVID-19 cases, we use publicly available case data from the ongoing COVID-19 epidemic provided by [27]. $X_i[t]$ consists of static demographic covariates and time-varying mobility features that affect the disease transmission rate.

The dynamic covariates proxy mobility by estimating the daily fraction of people staying at home relative to a region-specific benchmark of activity in early March before social distancing measures were put in place. We also include a regional binary indicator of the days when the fraction of people staying home exceeds the benchmark by 0.2 or more. These data are provided by SafeGraph, a data company that aggregates anonymized location data from numerous applications in order to provide insights about physical places. To enhance privacy, SafeGraph excludes census block group information if fewer than five devices visited an establishment in a month from a given census block group. Documentation can be found at [28].

The static covariates capture standard demographic features of a region that influence variation in infection rates. These features fall into several categories:

- Fraction of individuals that live in close proximity or provide personal care to relatives in other generations. These covariates are reported by age group by state from survey responses conducted by [29].
- Family size from U.S. Census data, aggregated and cleaned by [30].
- Fraction of the population living in group quarters, including colleges, group homes, military quarters, and nursing homes (U.S. Census via [30]).
- Population-weighted urban status (US Census via [30]).
- Prevalence of comorbidities, such as cardiovascular disease and hypertension ([31]).
- Measures of social vulnerability and poverty (U.S. Census via [30]; [32]).
- Age, race and occupation distributions (U.S. Census via [30]).