A Bayesian Latent Variable Model for Semi-parametric Identification and Analysis of COVID-19 Infection Rates

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Abstract

In this paper we present an original approach for measuring infections as a latent variable and making use of serological surveys to provide ground truth identification. Compared to existing approaches, our model makes much fewer assumptions and has a simpler form to permit extensive covariate adjustment and mediation analysis. To show the utility of the model, we analyzed COVID-19 case and test data from the fifty United States from March to July of 2020. With our modeling framework we are able to move beyond associations via linear models to more direct testing of hypothesized mechanisms for COVID-19 transmission as a function of human behavior and preferences. We are able to show with this analysis how social distancing measures, Trump approval rating, and mask-wearing are associated with COVID-19 infections through mediated changes in cell phone mobility and opinion polling.[1].

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[1]A reproducible version of this paper is available as an Rmarkdown file at https://github.com/CoronaNetDataScience/covid_model

The COVID-19 pandemic has led to a significant increase in disease modeling as the demands of a worldwide emergency spurred substantial innovation. Accurately modeling COVID-19 and similar diseases is no simple feat, however, due to multiple forms of selection and measurement bias that are difficult to overcome. The approach we present in this paper differs from existing work by conceptualizing the relative level of infections as a time-varying latent variable and applying Bayesian techniques to obtain a posterior distribution over likely estimates. Furthermore, we directly incorporate serological survey data into the model in a way that allows us to identify the scale of the latent variable without having to make extensive assumptions about transmission dynamics.

This approach has two advantages for applied research. First, the minimal set of assumptions makes the model more robust by incorporating as much uncertainty as possible about the actual infection rate. Existing approaches based on the SIR/SEIR framework often require multiple hard-coded values due to the large number of dynamic compartments that must be estimated. By employing a Bayesian model with weakly informative priors, we can obtain estimates that are more fully a reflection of our underlying uncertainty about the state of the world, rather than having to employ simulation inference or other tools to try to test for the reality of assumptions.

Second, conceptualizing the infection rate as a latent variable within a generative modeling framework permits us to examine covariate relationships in a manner that is more nuanced. As we show in this paper, it is possible for us to employ a wide range of covariates that predict the infection rate and consequently dramatically reduce the uncertainty of our estimates without having to assume any prior relationship between covariates and the attack rate. Second, we are able to examine mediation relationships between covariates and the infection rate, which permits us to be more specific about the pathways through which covariates may be associated with infections. Especially given how difficult it is to achieve causal identification with observational epidemiological data, we believe that employing causal graphs to test for plausible mediation relationships is a way to improve our understanding of potential disease transmission dynamics.

To apply the model, we estimate the infection rate in the United States from March to July of 2020, a crucial time in the pandemic as substantial uncertainty existed about how to respond to the disease. This uncertainty manifested itself in rapidly changing patterns of human behavior and also political conflict as leaders diverged over their understanding of the disease's threat. While we have increasing evidence about the relationship between partisan identities and individual beliefs about COVID-19 (Fan, Orhun, and Turjeman 2020; Grossman et al. 2020), data about partisan identity, along with other social and economic covariates, are rarely included in efforts to model and predict the spread of the disease in the population (Seth Flaxman 2020; Sharma et al. 2020; Haug et al. 2020a). As a result, we have difficulty understanding the causal

pathways through which political and social variables affect the course of the pandemic, and in turn are affected by it. In this paper we disaggregate the effects of COVID-19 policies, partisanship and demographic factors on disease outcomes by examining the way that these variables are mediated by individual mobility and fear of the virus. Even when we cannot make exclusive claims of causal identification, we can still learn in much more detail about time-varying associations between covariates and the disease and the plausible pathways through which beliefs affected actions.

We show with this model that political partisanship in the United States had a very strong association with the spread of the pandemic by increasing or reducing people's fear of the virus and also by changing their mobility patterns. A 1-SD increase in a state's 2016 vote share for Donald Trump is associated with a cumulative increase of 0.5% to 0.7% of a state's population infected by SARS-CoV-2 mediated by decreasing people's concern over the pandemic. A 1-SD increase in a state's vote share for U.S. President Donald Trump is also associated with a 0.3% to 0.5% increase in a state's infections mediated by increased mobility. We show that offsetting these increases in infections from mobility associated with partisanship would require at least 100 additional days of a state-wide stay-at-home order.

We also find evidence that political activity on the left is positively associated with the spread of the disease, with states that saw a 1-SD increase in social justice protests following the death of George Floyd witnessing an increase in infections as high as 0.4% over time. On the other hand, we do not find that the protests reduced people's fears of the disease or changed mobility patterns, suggesting that the spread of the disease happened solely through increased personal contact at the protests and subsequent chains of transmission rather than by changing behavioral patterns over long term.

1 Background

As more and more data has become available on observed case counts of the SARS-CoV2 coronavirus, there have been increasing attempts to infer how contextual factors like government policies, partisanship, and temperature affect the disease's spread Brzezinski et al. (2020). The temptation to make inferences from the observed data, however, can result in misleading conclusions. Modeling approaches that fully account for disease dynamics like the SIR/SEIR specifications are very powerful but also require more information than is known about disease progression in the population, requiring researchers to rely on assumptions that are difficult to know with complete confidence (Seth Flaxman 2020; Neil M Ferguson 2020). For this reason, in this paper we present a retrospective Bayesian model that can adjust for testing bias by estimating the unobservable infection rate up to an unidentified constant. Furthermore, by incorporating informative

priors based on serological surveys of infection prevalence, it is possible to put an informative prior on the unobserved infection rate and estimate both recent disease trends and the effect of covariates on the historical spread of the disease.

We can summarize the problem of modeling COVID-19 (and diseases more generally) in terms of two main challenges. The first is the challenge of modeling the spread of the disease given the limitations of testing and reported deaths, which could obfuscate the effect of any covariates with data reporting issues. Second, employing observational data requires nuanced comparisons to be made. To learn the effect of a stay-at-home order, for instance, we would want to compare two regions with similar demographic, social and political characteristics as these could be also influencing human behavior, masking the effect of the stay-at-home order. For example, regions with less political partisanship may be more likely to take prudent behaviors to mitigate COVID-19 and also may be more likely to see stay-at-home orders implemented.

A vast and expanding literature documents connections between many political, economic and social factors with human behavior related to the COVID-19 pandemic (Abouk and Heydari 2021; Adolph et al. 2021; Allcott et al. 2020; Ashraf 2020; Barceló et al. 2022; Bo et al. 2021; Brauner et al. 2020; Courtemanche et al. 2020; Dave, Friedson, Matsuzawa, and Sabia 2020; Fellows, Slayton, and Hakim 2020; Flaxman et al. 2020; Haug et al. 2020b; Islam et al. 2020; Murray and Jilani-Hyler, n.d.; Perra 2021; Sebhatu et al. 2020; M. Sharma et al. 2021; Zheng et al. 2020). While existing studies have shown these associations primarily through surveys and other individual-level analyses, it is difficult to test whether these factors jointly have an effect on COVID-19 infections. The reason for this difficulty is due to how these variables affect human behavior in general equilibrium. For example, non-pharmaceutical interventions (NPIs) like stay-at-home orders have been associated with reduced infections, but stay-at-home orders were also implemented in a rapidly changing environment as public health policies, new suppression practices like masking and the health of the economy varied. People have faced myriad influences on their choices during the pandemic, and even if we have a strong reason to believe that a certain factor should influence their behavior, estimating that effect when many other contravening and contrasting factors were at play is challenging.

At the same time, estimating these general equilibrium effects even within the limitations of available data is very important to learn what factors are associated with the spread of COVID-19 in realistic conditions. For example, some argued that masking would lead to increased infections because it would reduce concern over the risk of infection (Abaluck et al. 2020). Evaluating this hypothesis ultimately requires general equilibrium analysis as it involves competing influences on human behavior. In other words, is the moral hazard of being falsely protected a greater threat than the positive benefits of reducing infections via masking? Being able to sort, rank and understand socio-economic, political and healthcare-related factors behind the disease's spread

is crucial to better understand why and how COVID-19 overwhelmed countries' disease control systems.

In this paper, we seek to address these questions by collecting a rich set of important covariates, implementing models to adjust for bias in COVID-19 data and employ mediation analysis to understand the pathways that covariates affect the spread of the pandemic. We believe that doing so contributes to our emerging understanding of the factors that contributed to the spread of the pandemic, especially with respect to factors that we believe tend to be ignored in epidemiological modeling of COVID-19.

For example, in this paper we show that political partisanship may be equally as important to the spread of the pandemic as more conventional factors like the implementation of social-distancing measures. Political scientists have investigated to what extent partisanship has inhibited preventive measures against the COVID-19 pandemic as President Trump has argued against public health policies like face masks. Research has already shown that Republicans are less likely than Democrats to practice public health behaviors like hand washing (Gadarian, Goodman, and Pepinsky 2020), to practice social distancing (Andersen 2020; Alcott et al. 2020; Painter and Qiu 2020), and to comply with policies targeted against COVID-19 (Fan, Orhun, and Turjeman 2020; Grossman et al. 2020).

While partisanship in favor of President Trump and the Republican party has received the most attention, other types of political mobilization have also come under scrutiny. Of particular note were the protest movements against police brutality that spread across the United States in the summer and fall of 2020. Existing research suggests the protests have not had an adverse effect on COVID-19 infections (Dave, Friedson, Matsuzawa, Sabia, and Safford 2020), though it is again limited by the observational bias we describe later. As such, it is clear that political motivations on both the left and the right have at times led to reduced compliance with COVID-19 precautions, though it is not clear how severe these factors are, especially when compared to other covariates known to affect COVID-19.

2 Methods

Fitting models that can differentiate causal pathways is difficult because of the complexity of disease modeling with biased observed data. To address this crucial problem, we present a new Bayesian latent variable model that has a similar aim as epidemiological disease-tracking models in that it is designed explicitly to model disease dynamics. However, our model is a significant simplification of the compartmental models employed by epidemiologists to study disease, and in particular SARS-CoV2 (Peak et al. 2020; Riou et al. 2020; Robert Verity 2020; Perkins et al. 2020; Jose Lourenco 2020; Ruiyun Li 2020; Neil M Ferguson 2020; Carleton and Meng 2020; Sajadi et al. 2020; Dudel et al. 2020; Tasnim, Hossain, and Mazumder 2020; Seth Flaxman

2020; Brzezinski et al. 2020). These models suppose different classes (compartments) of individuals in the population, denoted S for susceptible, I for infectious, and \$R\$ for removed (other compartments may be added, such as E for exposed).

While these models are a powerful expression of the progress of a disease in the population, these models often struggle to provide straightforward estimates when employing empirical data. COVID-19 data, unfortunately, has serious flaws that are increasingly well-known, including limited testing and under-reporting of hospitalizations and deaths (Larremore et al. 2020; Sánchez-Romero et al. 2021). When such data is unavailable, modelers can compensate by simulating plausible random values or using informative prior distributions, but this makes the model estimates tied to the particular set of values used (Grinsztajn et al. 2021). As a result, the challenges in the estimation of compartmental models with empirical data restrict the ability to employ more rigorous forms of covariate adjustment.

By contrast, this paper endeavors to estimate a much simpler quantity than the entire evolution of the outbreak. We believe that many researchers and the general public often only want to learn about what has already happened, or the *empirical* infection rate (also called the attack rate in the epidemiological literature). For a number of time points $t \in T$ since the outbreak's start and states $c \in C$, we aim to identify the following quantity:

$$f_t\left(\frac{I_{ct}}{S_{ct} + R_{ct}}\right)$$

where I_{ct} denotes the number infected with SARS-CoV-2 at time t and S_{ct} and R_{ct} denote those who remain susceptible to the virus and those who have either died or recovered. In our model, we collapse S_{ct} and R_{ct} to a single quantity–those who are not infected–so we can focus exclusively on identifying I_{ct}.

However, even with this simplification, we do not have estimates of the actual infected rate I_{ct}, only positive COVID-19 cases a_{ct} and numbers of COVID-19 tests q_{ct} due to the aforementioned measurement issues. Given this limitation, the aim of the model is to backwards infer the infection rate I_{ct} as a latent process given observed test and counts. Modeling the latent process is necessary to avoid bias in using only observed case counts as a proxy for I_{ct}. The reason for this is shown in Figure 1 in which a covariate X_{ct}, such as a stay-at-home order, is hypothesized to affect the infection rate I_{ct}. Unfortunately, increasing infection rates can cause both increasing numbers of observed counts a_{ct} and tests q_{ct}. As more people are infected, more tests are likely to be done, which will increase the number of cases independently of the infection rate. As a result, due to the back-door path from the infection rate I_{ct} to case counts a_{ct} via the number of tests q_{ct}, it is impossible to infer the association of X_{ct} on I_{ct} from the observed data alone without modeling the latent

infection rate.

Figure 1: Directed Acyclic Graph Showing Confounding of Covariate X_{ct} on Observed Tests q_{ct} and Cases a_{ct} Due to Unobserved Infection Rate I_{ct}

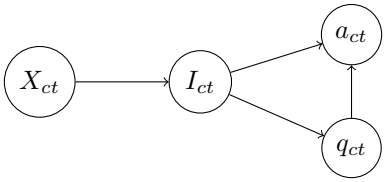


Figure shows the relationship between a covariate X_{ct} representing a policy or social factor influencing the infection rate I_{ct}. Because the infection rate I_{ct} influences both the number of reported tests q_{ct} and reported cases a_{ct}, any regression of a covariate X_{ct} on the reported data will be biased.

To estimate the process in Figure 1, we assume that the unobserved state-specific cumulative infection rate I_{ct} can be modeled as a time-varying Beta-distributed random variable with a mean parameter $\mu \in (0, 1)$ and shape parameter $\phi > 0$. We also assume that the over-time change in the disease can be modeled as a 3-order polynomial time trend that is a function of the number of post-outbreak time periods $T_O < T$, where an outbreak begins at the first reported case in a given area.

It is important to note that the reason we employ a cubic function is based on theoretical considerations. In our model, the polynomial represents the rate of infection increase in the absence of any other covariates, or equivalently the *counterfactual* rate of infections. We know from the SIR/SEIR simulations that, in the absence of any countervailing measures, epidemics occur in ever-increasing waves until the herd immunity threshold is reached, although the curve is unlikely to be symmetric as a simpler quadratic function would require. As such, we employ this function because it represents a credible baseline for what the epidemic would do if no other factors impeded its spread. We further allow the polynomial trends to vary by states hierarchically, i.e., the information about the trends is partially pooled across states.

We define the conditional distribution of the unobserved infection rate I_{ct} as:

$$\Pr(I_{ct} \mid t = T) \sim \text{Beta}(\mu\phi, (1 - \mu)\phi) \tag{1}$$

$$\mu = g^{-1}(\alpha_1 + \beta_{O1} \sum_{c=1}^{C} \sum_{t=1}^{T-14} a_{ct} + \tag{2}$$

$$\beta_{I1} t_o + \beta_{I2} t_o^2 + \beta_{I3} t_o^3 + \beta_C X_{ct}) \tag{3}$$

This parameterization of the Beta distribution in terms of μ and ϕ follows from the Beta regression literature (Ferrari and Cribari-Neto 2004) so that we can model the expected value $E[I_{ct}]$ directly via μ. As such, we

use $g^{-1}(\cdot)$, the inverse logit function, to scale the linear model in μ to the $(0, 1)$ interval. For the parameters, $\beta_{O1} \sum_{c=1}^{C} \sum_{t=1}^{t-14} a_{ct}$ are the sum of observed cases in the country with a 14-day lag, which represents the possibility of cross-border spread in infections. The three β_{Ii} are polynomial coefficients of the number of post-outbreak time periods t_o.

The parameter vector β_C represents the effect of independent covariate matrix X_{ct} on the latent infection rate. These are our main variables of interest, and have effects in addition to the polynomial time trends. Finally, the parameter ϕ becomes a dispersion parameter governing the variability of latent infection rate.

Because we do not have measures of I_{ct}, we need to used the observed data, tests q_{ct} and cases a_{ct}, to infer I_{ct}. First, we propose that the number of infections will almost certainly increase the number of tests as states try to stop the disease's spread via surveillance. Second, we can assume that a rising infection rate is associated with a higher ratio of positive results (reported cases) conditional on the number of tests, that is, COVID-19 is causing positive test results. We model both of these observed indicators, tests and cases, jointly to simultaneously adjust for the infection rate's influence on both factors. It is this joint modeling that permits us to directly incorporate testing bias. In fact, our model learns about the infection rate from the level of tests.

To model the number of tests, we assume that each state has an unobserved level of testing capacity, which increases at a non-linear rate during the course of the epidemic. We employ a quadratic function of testing capacity to express the concept of diminishing marginal returns. States were able to ramp up testing once PCR tests were approved by the FDA, but faced constraints due to shortages of supplies, personnel and labs. The cumulative number of observed tests q_{ct} for a given time point t and state c and as a fraction of the states' population, c_p, then has a binomial distribution:

$$q_{ct} \sim \text{Binomial}(c_p, g^{-1}(\alpha_2 + \beta_b I_{ct} + \beta_{cq1} L_t + \beta_{cq2} L_t^2)). \tag{4}$$

The parameters β_{cq1} and β_{cq2} represent the quadratic increase in testing capacity that varies by state c. We similarly allow for partial pooling of these coefficients as testing capacity will show a limited level of variability across states. The parameter β_b then represents the independent contribution of the level of infections I_{ct} on the total number of requests demanded marginal of testing capacity. The intercept α_2 indicates how many tests would be performed in a state with an infection rate of zero and at time $t = 0$, and as such is likely to be very low.

The binomial model for the number of observed tests q_{ct} provides some information about I_{ct}, but not enough for useful estimates. We can learn much more about I_{ct} by also modeling the number of observed cases a_{ct} as

another binomial random variable expressed as a proportion of the state population, c_p:

$$a_{ct} \sim \text{Binomial}(c_p, g^{-1}(\alpha_3 + \beta_a I_{ct})), \tag{5}$$

where $g^{-1}(\cdot)$ is again the inverse logit function, α_3 is an intercept that indicates how many cases would test positive with an infection rate of zero (approximately equal to the false positive rate of the test), and β_a is a parameter that determines how hard it is to find the infected people and test them as opposed to people who are not actually infected. The multiplication of this parameter and the infection rate determines the cumulative number of cases, a_{ct}, as a proportion of the state population, c_p.

To summarize the model, infection rates determine how many tests a state is likely to undertake and also the number of positive tests they receive as cases. This simultaneous adjustment helps takes care of mis-interpreting the observed data by not taking into account varying testing rates, which has made it hard to generalize findings concerning the disease and also led some policy makers to claim that rising case rates are solely due to increasing numbers of tests. It also allows us to learn the likely location of the infection rate conditional on what we observe in terms of tests and cases.

Because sampling from a model with a hierarchical Beta parameter can be difficult, we simplify the likelihood by combining the beta distribution and the binomial counts into a beta-binomial model for tests:

$$q_{ct} \sim \text{Beta-Binomial}(c_p, \mu_q \phi_q, (1 - \mu_q)\phi_q) \tag{6}$$

$$\mu_q = g^{-1}(\alpha_2 + \beta_b I_{ct} + \beta_{cq1} L_t + \beta_{cq2} L_t^2) \tag{7}$$

and cases:

$$a_{ct} \sim \text{Beta-Binomial}(q_{ct}, \mu_a \phi_a, (1 - \mu_a)\phi_a) \tag{8}$$

$$\mu_a = g^{-1}(\alpha_3 + \beta_a I_{ct}). \tag{9}$$

where I_{ct} is now equal to the linear model vector μ shown in (3) and mapped to $(0, 1)$ via the inverse logit function.

2.1 Identifiability

This model contains an unobserved latent process I_{ct}, and as such the model as presented is not identified from the data alone without further information. For example, the parameters that control the influence of the infection rate on tests and cases could increase and the latent infection rate could decrease without the probability of the observed data changing.

There two further steps taken to identify this model which we believe represent very limited additional assumptions, especially compared to existing modeling approaches. First, we must require that I_{ct} is a non-decreasing quantity. The number of infected people cannot decrease in an epidemic, but the model as expressed does not require that to be true.[2] We can eliminate that possibility from the model by imposing an ordered constraint on I_{ct}:

$$I_{ct} = \begin{cases} I_{ct} & \text{if } t = 1 \\ I_{ct-1} + e^{I_{ct}} & \text{if } 1 < t < T \end{cases} \tag{10}$$

This transformation forces I_{ct} to be no less than I_{ct-1}. At the same time, we do not need to impose any constraints on the covariates themselves, allowing us to sample those in an unconstrained space before we transform I_{ct}.

However, we also need some information about the empirical scale of testing bias to produce identified estimates of I_{ct}. We could do so by adding a prior to the model about the plausible range of total infections to reported cases, though we prefer to use information that is more precise. The Centers for Disease Control's serology surveys conducted during the pandemic represent an empirical way of relating I_{ct} to plausible estimates of infections at varying time points. We include a list of these surveys in the supplementary information. By including this information, we also implicitly account for many of the variables explicitly parameterized in compartmental models such as reporting delays. Because we have an estimate of the number infected at time t that is independent of reported cases and tests, the model will find the parameter estimates that are most likely given the observed differences between the surveys and the reported data.

Because we model the infection rate as a cumulative count, it is straightforward to include this information in the model. For a given state c and time point t for which we have survey information, we model the count of infected S^P_{ct} as a proportion of the total subjects in each serology survey S^N_{ct} with the Binomial distribution:

[2] See https://www.google.com/covid19/mobility/

$$S_{ct} \sim \text{Binomial}(S_{ct}^N, g^{-1}(I_{ct})) \tag{11}$$

It is important that the serology surveys enter the model in this fashion so that we can model the survey count stochastically. This is necessary to propagate uncertainty in the CDC sample size through to our estimates of I_{ct}. This uncertainty matters as well because the serology surveys exhibit random noise and do not always increase over time, as can be seen in the supplementary information. By modeling the relationship as a probabilistic one, we are making the weaker assumption that the infected rate is probably close to the serology estimate, but the two do not need to be the identical. The combined posterior estimates for I_{ct} will then be weighted with the case and test likelihoods to produce the most credible estimate of I_{ct}.

As we show in the supplemental information with simulations, no other identification restrictions are necessary to estimate the model beyond weakly informative priors assigned to parameters.

These are:

$$\beta_a \sim \text{Normal}(30, 10), \tag{12}$$

$$\beta_{qci} \sim \text{Normal}(\mu_{qi}, \sigma_{qi}), \tag{13}$$

$$\sigma_{qi} \sim \text{Exponential}(1), \tag{14}$$

$$\mu_{qi} \sim \text{Normal}(0, 20), \tag{15}$$

$$\beta_C \sim \text{Normal}(0, 5), \tag{16}$$

$$\beta_{Ii} \sim \text{Normal}(\mu_{Ii}, \sigma_{Ii}), \tag{17}$$

$$\mu_{Ii} \sim \text{Normal}(0, 10), \tag{18}$$

$$\sigma_{Ii} \sim \text{Exponential}(1), \tag{19}$$

$$\alpha_1 \sim \text{Normal}(0, 10), \tag{20}$$

$$\alpha_2 \sim \text{Normal}(0, 10), \tag{21}$$

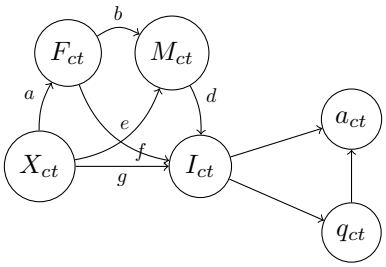
$$\alpha_3 \sim \text{Normal}(0, 10) \tag{22}$$

where the normal distribution is parameterized in terms of mean and standard deviation.

The priors to note are the hierarchical regularizing prior put on the varying testing adjustment parameters β_{qci} and varying polynomial trends β_{Ii} with shared means and standard deviations. This partial pooling

permits a reasonable degree of heterogeneity in the parameters while still constraining overall dispersion. Relatively informative priors are put on the hierarchical variance parameters σ_{qi} and σ_{Ii} to suggest that while state heterogeneity does exist, we do still expect the states' estimates to be within a given range.

Figure 2: Directed Acyclic Graph for Latent Infection Rate with Mediators



This figure adds mediators M_{ct} (mobility data) and F_{ct} (fear of COVID-19) that mediate the relationship between state-level covariates X'_{ct} and the latent infection rate I_{ct}. Because beliefs precede actions, F_{ct} is causally prior to M_{ct} and can affect infections both via reducing mobility (path abd) and directly apart from mobility (path ae), such as by encouraging individuals to remain socially distant.

We also extend this model in order to analyze the mediation of a subset of covariates X'_{ct} by adding mediators M_{ct} for mobility and F_{ct} for fear of the disease to the causal diagram, as in Figure 2. Figure 2 has several paths due to the fact that the influence of covariates X_{ct} affects the two mediators differently. Given that beliefs and preferences precede actions, the covariates X'_{ct} first influence I_{ct} along the ae and abd path through perceptions of how dangerous the disease is. These beliefs both affect the chance of an individual getting infected and thus I_{ct} directly on the path ae, such as by causing an individual to adopt social distancing behaviors, and also on an indirect path abd by which an increase in a people's fear of the disease reduces mobility as people prefer to stay home.

In addition to pathways through the fear mediator F_{ct}, a covariate could influence infections along the pathway through mobility ed without increasing or decreasing fear. This situation could arise if government policies forced people to stay at home against their will and despite their unconcern about the disease. Finally, a covariate could have an unmediated direct effect g on the infection rate. The total effect of a covariate X_{ct} on the spread of the disease is then the sum of all the paths, $abd + af + ed + g$. To calculate the indirect effects and direct effects given the use of the inverse logit function $g^{-1}(\cdot)$, we employ the chain rule as in Winship and Mare (1983) to calculate the marginal effect of covariates with respect to different pathways to I_{ct}.

Adding the mediators to the model is relatively simple as they do not have link functions and can be included as Normal distributions (i.e., OLS regression) as in Yuan and MacKinnon (2009). It should be noted that there are in fact five mobility covariates as explained in the following section, and so we explicitly model the

covariance in mobility via a multivariate Normal distribution with a covariance matrix parameter Σ_m.

To add our mediation covariates M_{ct} and F_{ct}, which we describe in more detail in the next section, we multiply the following likelihoods with the joint posterior:

$$M_{ct} \sim MVN(\alpha_m + \beta_m X'_{ct}, \Sigma_m) \tag{23}$$

$$F_{ct} \sim N(\alpha_f + \beta_f X'_{ct}, \sigma_f) \tag{24}$$

We also include all of M_{ct} and F_{ct} as linear predictors in (3).

We fit this model using Markov Chain Monte Carlo in the Stan software package (Carpenter et al. 2017). We run the sampler for 1000 iterations with 500 warmup iterations and two chains to test for convergence.

3 Data

The only data required to fit the model, in addition to the covariates of interest and serology surveys, are observed cases and tests for COVID-19 by day. In this section, we fit the model to numbers of COVID-19 case counts on US states and territories provided by The New York Times. By doing so, we can use the differences in trajectories across states to help identify the effect of state-level covariates on the infection rate. We supplement these observed case counts with testing data by day from the COVID-19 Tracking Project. We then take the 7-day rolling average of both series to account for reporting fluctuations and weekly reporting effects.

We note that COVID-19 cases and deaths are available at the county level in the US. We do not use this reduced level of aggregation for two reasons. First, and most importantly, our aim is to better understand the mechanisms of COVID transmission, which requires us to have access to daily polling data which is not available at the country level. Second, we note that what data is available is much more prone to measurement error due to issues with reporting that vary by county (Stoto et al. 2022). Aggregating to the state level can reduce this idiosyncratic measurement error and permit more stable inferences, especially when looking at day-to-day changes in these covariates .

To analyze the effect of suppression policies, we use data on counts of social distancing policies, restrictions on mass gatherings, restrictions on businesses, mandatory mask orders, restrictions on government services, and stay-at-home orders from the CoronaNet Government Response dataset (Cheng et al. 2020). For each type

of policy, we include a variable representing the count of policies in that category effective for a particular day. For each update to an existing policy, we code it as +1 if the update increases the scope of the policy or -1 if it decreases the scope of the policy (down to a minimum of 0). While this is a simplification of the underlying data, we are still able to capture relative complexity over time without having to make judgments about stringency or other qualitative criteria. We then interact these policy counts with a linear trend to examine time-varying policy effects. We separately include policies designed to increase health resources like personal protective equipment (PPE) and also policies requiring mask use as we do not examine time-varying effects of these covariates.

The policy data is plotted by state in Figure 3. As can be seen, there is a rise in policies after the pandemic begins in the middle of March, though the number of policies varies across categories. The count of policies is an admittedly imperfect measure though it communicates more information about policy activity than a simple binary coding. Generally speaking, states imposed many more policies designed to increase their access to PPE for health staff than they were willing to take on lockdowns, social distancing, and restrictions on businesses and government services. This difference likely has to do with the increased cost and salience of these policies vis-a-vis relatively less politically difficult options like gathering more masks and face shields for health care workers (Cheng et al. 2020).

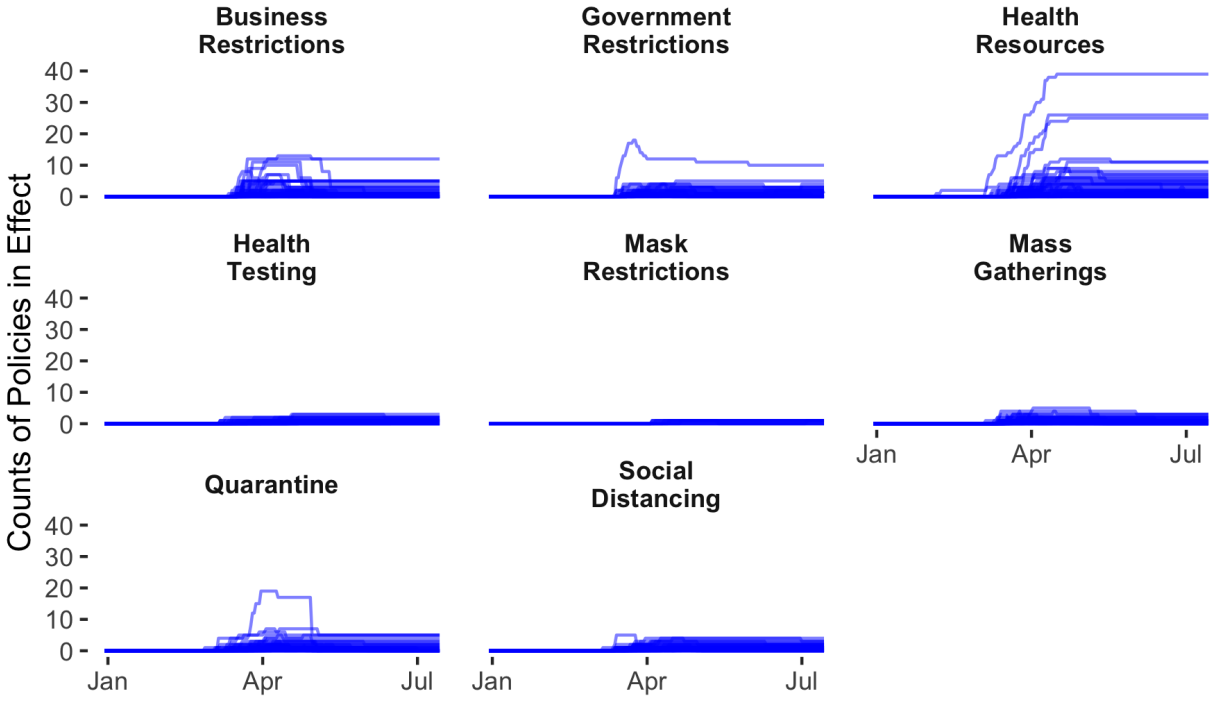


Figure 3: Count of Policies in Effect by Day and by State from the CoronaNet Dataset

To better understand over-time factors that may also affect COVID-19, we include polling data from Civiqs and YouGov at the state level. From Civiqs we include state-level polling averages by day for the percentage of respondents favoring Trump, percentage reporting the economy is "very good", and the percentage reporting that they are "extremely concerned" about the coronavirus. From YouGov we use a poll from May 8th reporting average number of respondents who said they used masks by U.S. state. As this poll does not vary over time, we set the mask prevalence at one-half the minimum value of the poll prior to the WHO's revision of guidance concerning wearing masks on April 3rd, and equal to the poll's values thereafter. As described in the previous section, the poll asking respondents whether they are "extremely concerned" about COVID-19 represents our fear mediator, and is also included as a separate outcome with other covariates as predictors.

To better understand the mediating effects of suppression policies, we include Google mobility data[3] for retail, residential, parks, workplaces, transit and retail establishments. These estimates are by day and aggregated to the state level. They are measured in terms of an index that is initialized with a value of 100 at the index start on February 15th, 2020. To test for mediation, we include these as predictors of the infection rate, and separately fit a likelihood with each mobility covariate as an outcome and the other covariates as predictors.

We note that it is important to measure mediation for mobility because mobility is hypothesized to affect the spread of COVID-19 (Gao et al. 2020). As such, measuring the simultaneous effect on mobility for covariates in our model is important as the covariates could be affecting mobility, which subsequently affects COVID-19 spread. Ignoring this association would result in post-treatment bias that deflates the effect of predictors in the model, though our main interest in including these variables is because this mediation is substantively interesting to decompose.

To measure protest activity, we include a covariate reflecting the proportion of a state's population engaged in social justice protests following the death of George Floyd on May 25, 2020. This data is drawn from publicly available information about the number and size of protests from three online sources: Wikipedia protest data, the Count Love protest web-crawling web site,[4] and list of protests compiled by Ipsos.[5] For protests present in only one of the three sources, we used information on both size and location. If a protest was present in three sources, we averaged reported protest size. If the sources had contradictory information about the type of protest, we had research assistants re-code the protest using secondary sources. For protests for which size was not available, we imputed missing data using random forest algorithms (Stekhoven and Bühlmann 2012).

All time-varying covariates–polling, protests, policies and mobility data–are lagged by 14 days to account

[3]See https://www.google.com/covid19/mobility/
[4]https://countlove.org/
[5]See https://www.ipsos.com/en-us/knowledge/society/Protests-in-the-wake-of-George-Floyd-killing-touch-all-50-states

for the likely delay in events showing up in reported cases. This 14-day lag comes from the epidemiology literature (Seth Flaxman 2020) and is meant to take into the account the amount of time required for people to be infected, be tested and then have the test results reflected in case counts.

We further add in non-varying state-level data on Donald Trump's vote share for the 2016 election from the MIT Election Lab, a 2019 estimate of state GDP from the Bureau of Economic Analysis, the 2018 percentage of foreign born residents, population under 18 years of age and population density from the U.S. Census Bureau, 2019 state-level average data on air pollution,[6] cardiovascular deaths per capita, percentage of residents under age 18, number of dedicated health care providers, public health funding, and smoking rates provided by the United Health Foundation ("America's Health Rankings 2019 Report" 2019). All variables are standardized to permit comparability.

We employ state-level data rather than country-level data because our aim is to have a rich adjustment set of covariates. While some of our data is available as well at the country level, crucial covariates such as polling about fears of COVID-19 and the state of the economy are only available at the state level. We believe that obtaining quality estimates of these crucial variables is more important than the statistical power we would obtain from dis-aggregation.

This is particularly true because in general we cannot make claims of causal identification as we can with our claims of statistical identification of the latent infection rate. COVID-19 is not a very likely candidate for meeting any kind of assumption about ignorable selection into treatment; it is a disease that is indirectly caused by human behavior. Our identification strategy primarily relies on including as many relevant adjustment variables as is prudent to isolate factors which are likely to or known to have an effect on COVID-19 spread and could be confounding variables.

In addition, even when we cannot ensure causal identification, we can still learn important aspects of the underlying relationships by partitioning the variance via mediation analysis. Doing so allows us to isolate the part of the association which we have a strong theoretical reason to believe is causally related to the spread of COVID-19, such as via influencing people's fears over the severity of the virus in their area. While using observational data necessarily means there are inferential concerns we cannot rule out, we can still learn substantially from variables that we have a strong prior reason to believe are related to the outcome.

[6] Defined as average exposure of the general public to particulate matter of 2.5 microns or less ($PM_{2.5}$) measured in micrograms per cubic meter (3-year estimate).

4 Results

We first report the model's estimates of infected counts for the U.S. population as a whole in Figure 4. Panel A in this plot shows the cumulative total both for reported cases (thin black line) and for the model's estimate of total infected (blue line). The interval in this plot, as with all figures presented, are the 5% and 95% quantiles of the empirical posterior distribution. As can be seen, the model estimates that there are approximately 3-4 times as many infected people in the United States as reported cases, with the total cumulative number of infected persons reaching 15 million with around 500,000 infected as of mid-July. Early expert estimates are shown as black points in panel A, revealing that even epidemiologists largely under-estimated the spread of the disease in its early stages, largely due to limitations in testing and case reporting.

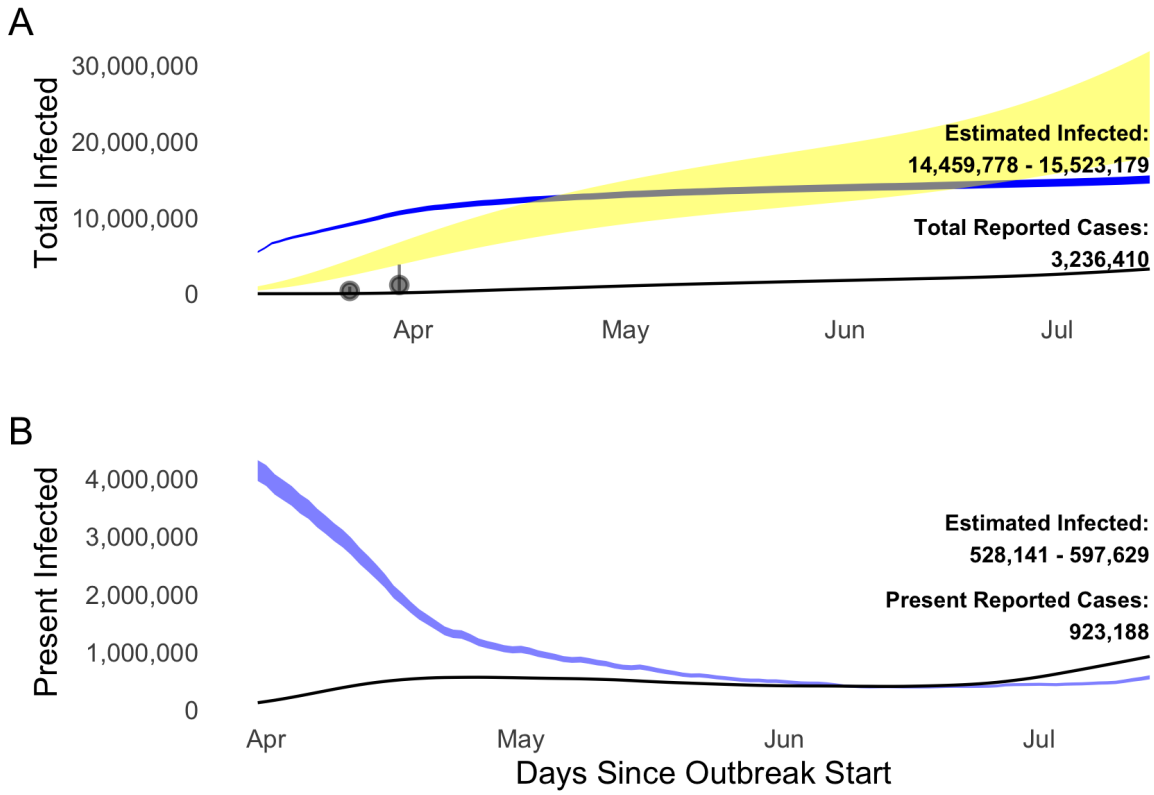
We compare these estimates with a popular COVID-19 forecaster employing SEIR models from Gu (2020) by plotting their estimates as a yellow ribbon on the plot. As can be seen, the trajectories are similar although they diverge slightly at the end of the series in mid-July. On the whole it would seem that our estimate of infected individuals is on the conservative end compared to other approaches–in other words, while we do not know for certain what the true number is, we are unlikely to be under-estimating the total. Furthermore, our intervals are far more precise than other approaches, which is likely because we employ extensive covariate adjustment to better infer human behavior during the course of the pandemic.

Panel B in the plot shows our estimates of infected individuals, excepts that it adjusts the cumulative number with a 19-day lag to account for the approximate time that recovery from COVID-19 requires (deaths are first subtracted). This plot displays an imperfect but useful formulation of the likely number of people infected at any given time point. As of July 14, it would appear that there were approximately 500 thousand infected individuals in the United States, while the number peaked at about four million in late April.[7]

By comparison, Figure 5 shows the cumulative totals of estimated infections by state. Plot A in this figure has the count of infections by state, while plot B shows the percentage of the population infected by state. Both the overall S-shape of the epidemic can be seen along with the substantial heterogeneity in infections, with early infected states like New York and New Jersey still in the top quartile of states with infections even though they successfully reduced the rate of disease spread.

In addition to the estimation of the cumulative count of infected individuals, the model provides further useful information by parameterizing the relationship between the unobserved infection rate and the number

[7]While it is interesting to note that the model shows fewer infected people than observed cases for this plot at the end, this difference is merely an artifact of the 19-day lag. It is difficult to put a precise number on the presently infected as states do not always report how many recover from COVID-19.



Blue 5% - 95% HPD intervals show estimated infected and the black line shows observed cases from he New York Times. These estimates are based on CDC seroprevalence data and a Bayesian model of how cases and tests are influenced by infection rates. Black dots in Panel A show early expert estimates of COVID-19 prevalence in the United States. Yellow ribbon shows 5% - 95% predicted cumulative infections from covid19-projections.com hybrid SEIR model.

Figure 4: Total Cumulative and Present COVID-19 Infections in the United States

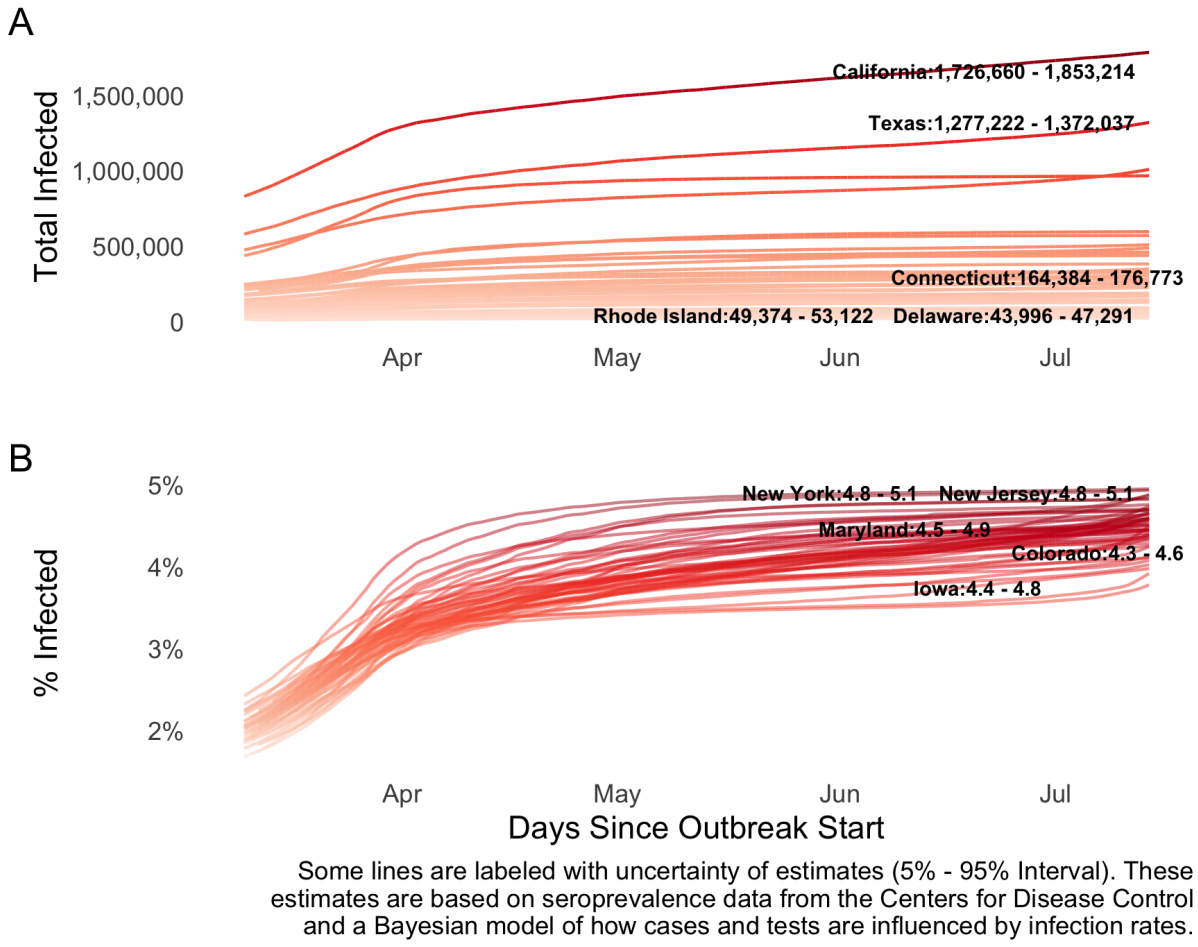


Figure 5: Average Cumulative Count of Infected People by U.S. State as of July 14th

of tests conducted in a given state. These individual parameters are shown in Figure 6. The scale of the y axis shows the number of people that a state was able to test relative to each person infected. The plot shows that some states have been able to test far more people than have been infected (New York, New Jersey), while other states like Pennsylvania and Oregon have tested barely twice as many as those who have been infected. The fact that new outbreaks have been seen in Texas and Arizona suggests that this shortfall in testing likely disguised early outbreaks that could have been detected otherwise.

In addition, we know from Figure 6 that because New York and New Jersey are quite high in the test/case distribution, having tested around four individuals per infected person, the high infection rates in Figure 5 for these states are not an artifact of more rigorous testing. The model is successfully able to separate the bias of increased testing from the actual level of new infections.

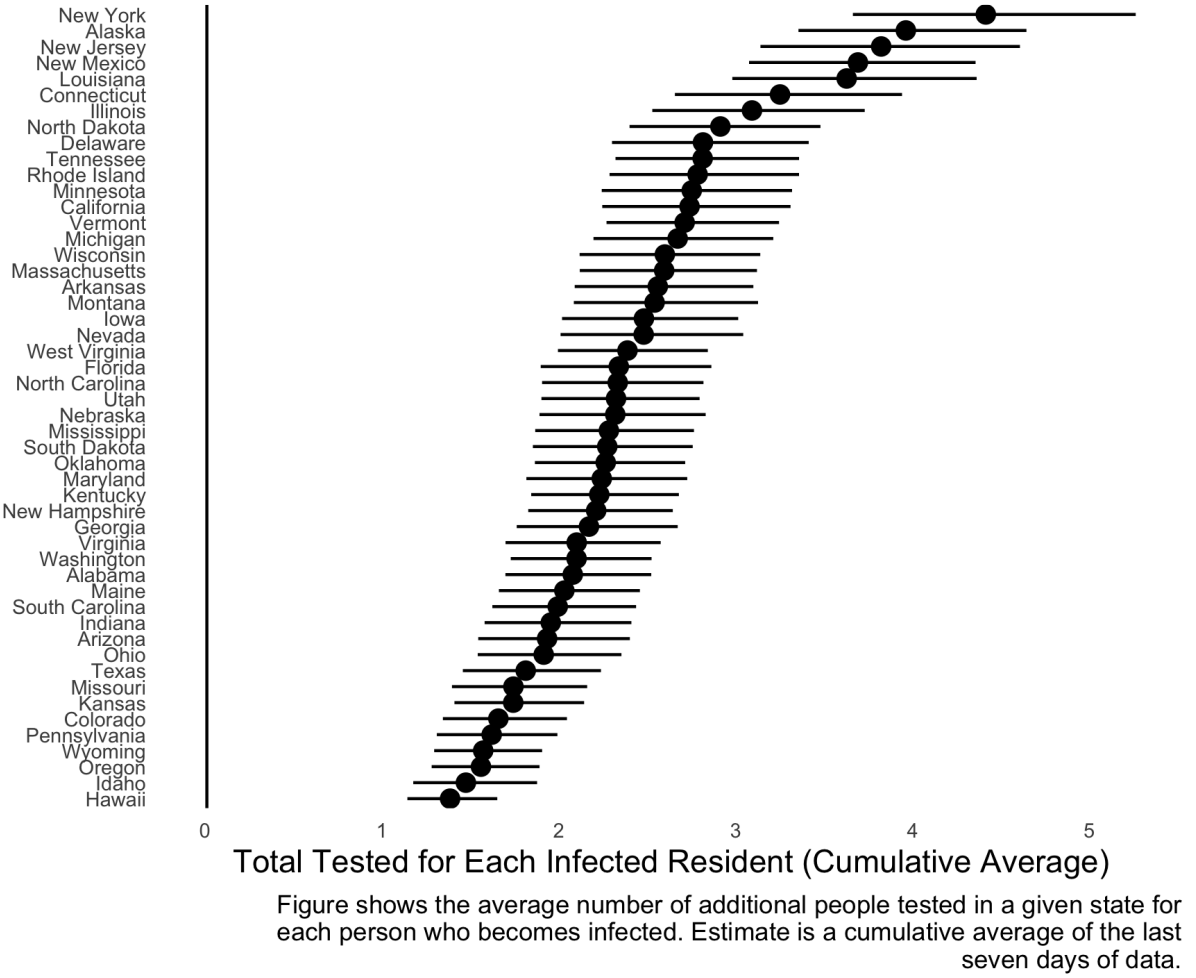


Figure 6: Measuring States' Testing Rates Relative to Infection Rates

We would note that this information is also helpful to policy makers and others trying to make sense of

observed case counts given the limitation in testing thus far. Our estimates help take into account these known biases and adjust them based on differences between states and within states in terms of disease trajectories. We believe this model can be used to help understand disease trends and factors associated with it even in the relatively data-poor environment many countries find themselves in. Unlike SEIR/SIR approaches, we do not employ information about hospitalization and death reporting delays, the infection-age distribution, or initial seeds. While these other outcomes can provide additional information about the progress and severity of the disease, they also considerably complicate inference.

To calculate the effect of covariates on the infection rate, we report here average cumulative marginal effects by state, i.e., by how much a given covariate increased the proportion infected for a one-unit increase in the covariate over time. We report cumulative marginal effects rather than the sample average marginal effect because the outcome monotonically increases, and so the marginal effect at any one point in time is not as meaningful a statistic. The way to interpret the coefficients presented is how a 1-unit (1-SD) change would affect the infection rate if that increase were sustained for an average state's entire time series (March to July).

We first show the association of mobility types with the infection rate. In Figure 7 we show the marginal effect of a 1-SD increase in different types of Google mobility on the infection rate expressed as a fraction of a state's population. In line with the growing research on cellphone mobility and the epidemic, there are strong positive effects of some types of mobility on the spread of the disease, especially residential, workplace, and retail mobility. Movement in parks, on the other hand, is negatively associated with COVID-19 occurrence. While these results are somewhat surprising given that both residential and workplace mobility are very large, other results confirm with prior suspicions that outdoor activities like attending parks are relatively low-risk for COVID exposure. In fact, increased mobility in parks is associated with reduced infections, probably because it substitutes for more high-risk types of mobility.

To interpret these coefficients correctly, it is important to take into account the multivariate normal distribution that was used to model each of these mobility measures as one joint distribution. The residual correlations for the mobility measures model are shown in Figure 8. These correlations are intuitive, with transit positively correlated with other mobility measures except residential (people tend to be at home if they are not in transit). What is quite important is that workplace and residential mobility are strongly inversely correlated at -0.88; in other words, people tend to be at home if they are not working and vice versa. As a result, the effect of residential and workplace mobility on COVID-19 is complicated due to this displacement effect. The fact that residential mobility is positively associated with infections once this displacement effect is taken into account accords with the modeling literature that warned that stay-at-home orders would paradoxically

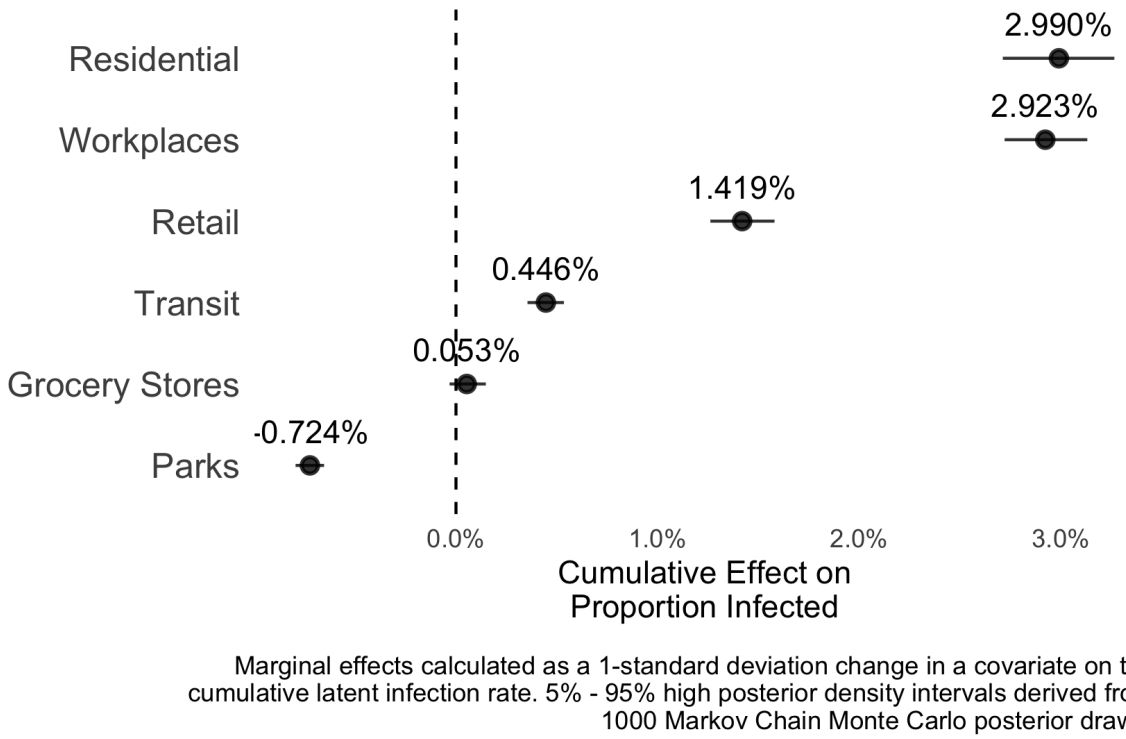


Figure 7: Effect of Google Mobility Data on COVID-19 Spread

increase infections in the home as people were kept in close quarters with each other (Neil M Ferguson 2020). We believe these strong correlations provide compelling evidence for employing the multivariate normal distribution in our model so that we do not assume these measures are conditionally independent. At the same time, it does render the interpretation of mediation effects somewhat more complicated as the model is explicitly taking into account that changes in one type of mobility are likely to displace or effect other types of mobility.

We next turn to an analysis of the rest of the covariates used to predict the latent infection rate. Figure 9 shows the marginal effect of all other covariates in the model on the latent infection rate expressed as average cumulative marginal effects. The estimates are further broken out in terms of mediation. The mobility effect is equivalent to the ed path in Figure 2, i.e., it is the path from the covariates to mobility that does not go through increased fear of COVID-19 measured by daily polls. The fear of COVID-19 pathway, on the other hand, is equivalent to the $abd + ae$ paths, or the sum of the path from fear through mobility and the path from fear to infections apart from mobility. In other words, a covariate's effect mediated by increased fear of COVID-19 can both immediately impact the outcome by heightening sensitivity to the severity of the pandemic and affect the outcome by reducing an individual's willingness to engage in dangerous types of mobility. The direct effects, which represent the unexplained effect of covariates independent of either

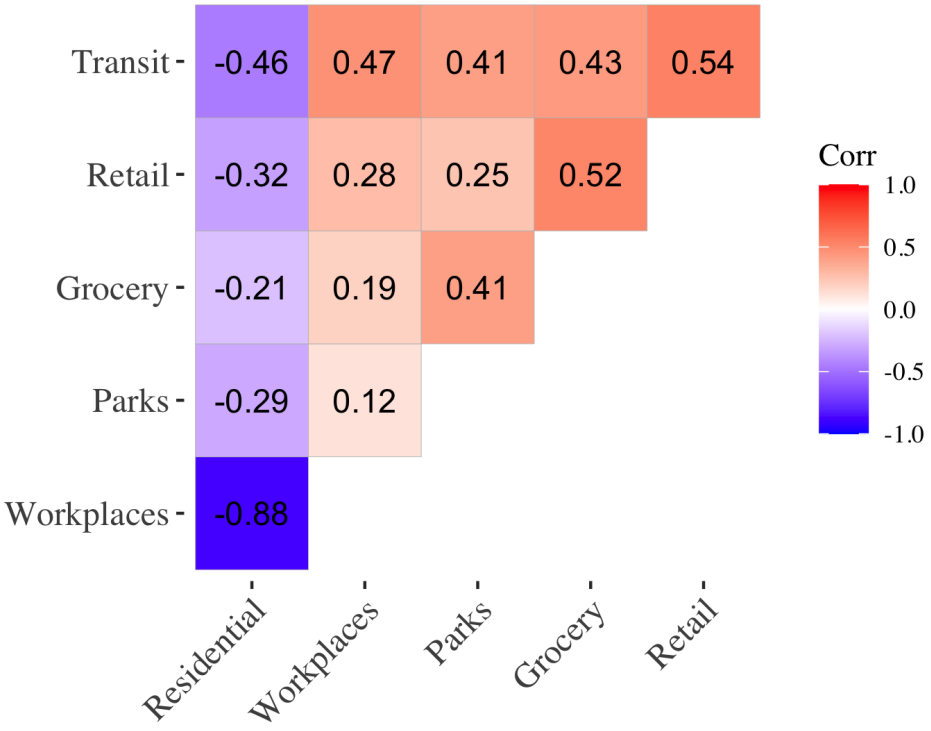


Figure 8: Estimated Correlation of Mobility Measures

concern over COVID-19 or changes in mobility, are then equivalent to the g path in Figure 2, and the total effects are the sum of all paths. The direct and indirect effects are disaggregated in panel A while the total effects are shown in panel B.

The use of mediation analysis shows substantial heterogeneity in the types of associations and whether direct and indirect effects tend to complement or substitute each other. First, it is important to note that the single strongest associations in panel B come from the YouGov mask-wearing poll, the Civiqs concern over coronavirus poll and the economy poll, and the percent of a state's residents that are foreign-born. As these are cumulative average marginal effects, that number reflects what an *average* state might experience; the effect could well be larger for states with higher infection rates than average. On the other hand, as these effects are cumulative, they reflect a state that experienced a sustained increase in the covariates and so it might overstate the effects somewhat.

In terms of total effects in panel B, the factors that are most strongly associated with reducing infections include concern over COVID-19, wearing masks, concern over the state of the economy, higher per capita income and higher population density. Conversely, a higher percentage of foreign-born, more social justice protests, PPE policies and greater concentrations of $PM_{2.5}$ are associated with more infections. Some of these

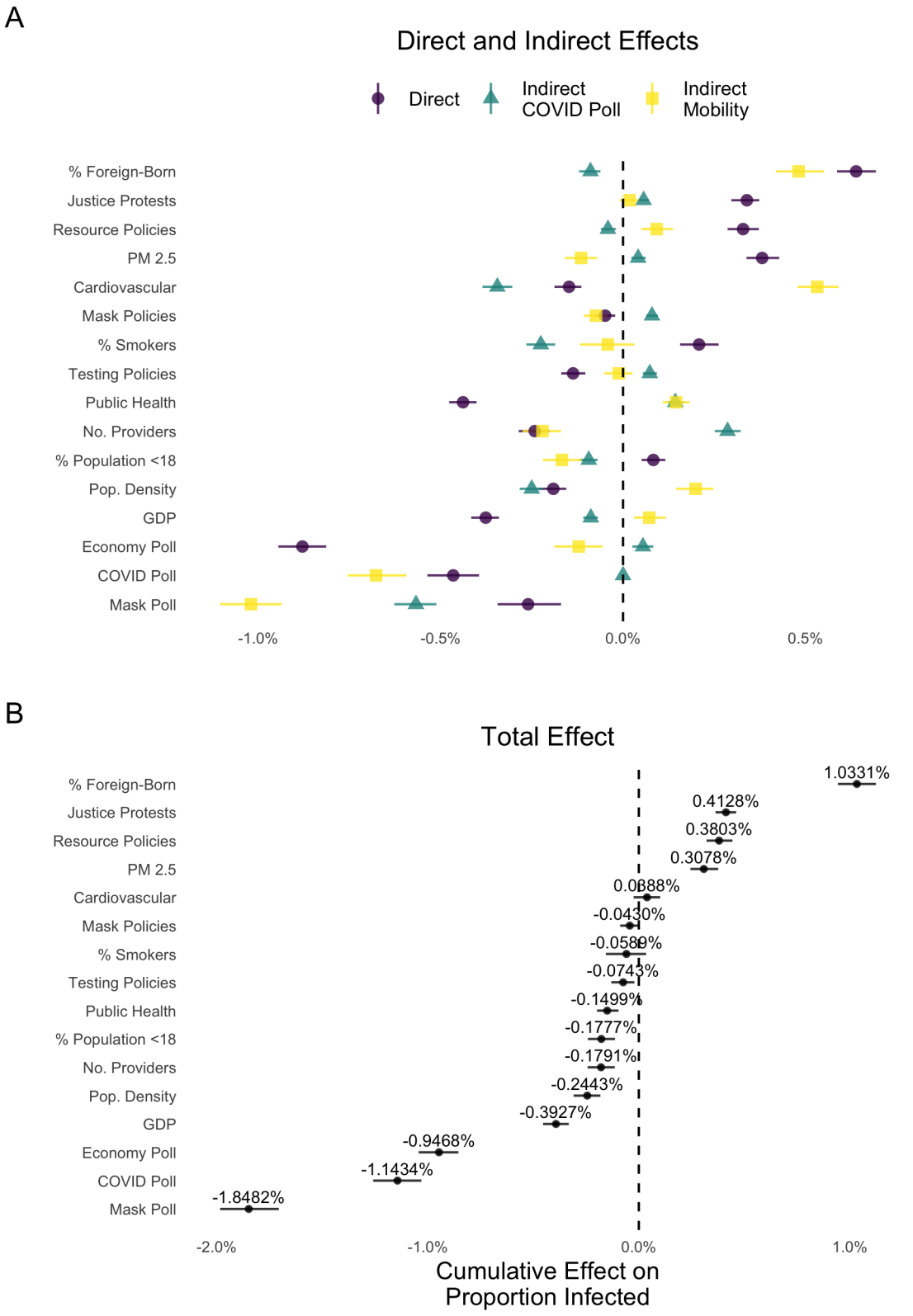


Figure 9: Marginal Effects of Covariates on Latent Infection Rates for U.S. States

associations would correspond to what is known about the virus, such as mask-wearing reducing infections while large gatherings like protests increasing infections.

Mediation analysis is helpful at understanding what may be driving these associations. We can learn more about the meaning of the results when we can identify effects through pathways which we have a theoretical reason to believe matter for fighting the epidemic: individual concern over COVID-19 and individual mobility. For example, while the association with percent foreign-born is quite strong, we know that the bulk of this association arose through an unspecified mechanism. Indirectly, the percentage foreign born is associated with *reduced* COVID-19 spread via the fear pathway. Instead, it is most likely the case that the percentage of foreign born is a proxy for international travel which led to initial outbreaks. This proxy was the reason the covariate was introduced into the model as it is difficult to otherwise capture travel patterns that may have introduced the virus earlier.

In contrast to other research, we find that social justice protests are positively associated with COVID-19 spread, though the effect is of medium size. Furthermore, as we report cumulative marginal effects, it is unlikely that states experienced protests every day in the sample, suggesting that the reported effect is more of an upper bound for what most states experienced. We do not find much evidence, as Dave, Friedson, Matsuzawa, Sabia, and Safford (2020) suggest, that the positive effect of the protests was offset by reduced mobility by non-protesters as the indirect effects are almost zero. On the other hand, the effect of the protests could have been much worse if it had decreased people's fears of the virus or induced risky travel patterns. In this case, the direct effect is relatively easy to surmise: close contact through the protests which spread infections. It is important to note as well that this effect exists even controlling for both Trump vote share and Trump approval rating, so it is not simply a proxy for state-level partisanship.

There are other interesting associations in Figure 7. States with more people with cardiovascular issues tended to see more infections due to risky mobility patterns but fewer infections due to increased concern over COVID-19. States with more public health spending tended to have a strongly negative direct effect on the spread of infections, as we might expect given that public health practices can help spread information about the virus, but this association was partially offset by reduced concern over the pandemic and riskier travel patterns. Similarly, the number of health providers has a strong negative association with the virus that is entirely offset by reduced concern over the pandemic. Both of these covariates have as a consequence very small total effects, suggesting that the different pathways are obscuring the complex ways with which state health care resources affected the course of the pandemic.

The economy poll is another interesting case as on the whole it is strongly negatively associated with infections. As the percentage of people who believed the economy was in a good state rose, infections tended to decrease.

Furthermore, this effect is primarily a direct effect, though there is some association with reduced infections via mobility. This result is theoretically interesting as trade-offs over the economy were often framed as a willingness to combat the epidemic versus the economic consequences of social distancing (Bonaccorsi et al. 2020). The empirical analysis shows that this trade-off may exist and that fears over an economic downturn increased infections, though not by changing mobility patterns or concern over the severity of the disease. We might speculate that this association results from increased willingness to comply with economically costly social distancing behaviors when the economy is believed to be on sound footing, though without further analysis we cannot say for sure.

What is clear is that the strongest time-varying factors present in the model concern individual behavior more than policies or state preparedness. Considering that the percentage of foreign residents (i.e., exposure to international travel) and per capita income were determined long before COVID-19 arrived, the most important manipulable factors are those involving beliefs, such as in the strength of the economy and the relative threat of COVID-19, along with personal behaviors like mask-wearing.

It is also interesting to note contrasting direct and indirect effects in panel A of Figure 9. The large effect from the COVID poll primarily comes from mobility data; people who are more concerned about COVID are less likely to frequent places where they could contract the disease. The mask poll is associated with repressing COVID through mobility, fear of COVID-19, and as a direct effect (presumably reduced spread through airways). The fact that all of these associations align suggests that the idea that masks would encourage risky behavior is in fact untrue (Abaluck et al. 2020). Finally, states with a larger proportion of smokers do tend to see more infections on a direct pathway, presumably by increasing people's risk to severe disease, but this effect is largely offset by increased fear in these states of the disease. This fascinating result shows how competing direct and indirect effects can mask an important empirical association we would expect given prior knowledge.

We next turn to the prominence of partisanship variables in explaining the spread of the disease, which we did not include in the previous figures as we interacted Trump vote share and within-state changes in approval polls in our model. Instead, we explore this interaction graphically in Figure 10. In this figure, the effect of Trump 2016 vote share is plotted conditional on the relative level of daily Trump approval polling on the x axis. The effects are shown aggregated in panels A and B and disaggregated across mobility types in panel C. Panel A shows that in general, the effect of partisanship for Trump has both direct and indirect effects, with the direct effect highly conditional on the above/below polling average of approval for Trump in a given state (which has a maximum swing of about +/- 4 pp). When Trump approval rose, states with high Trump vote share witnessed fewer infections later on. These high conditional associations are

likely due to the rally-around-the-flag effect in which Trump's approval rating spiked when the epidemic first appeared in March and April, leading to an association between high approval levels and low infection counts in conservative areas of the country.

However, it is important to note opposite effects through the mediated pathways. Panels B and C in Figure 10 shows that Trump vote share mediated through mobility and fear is strongly positive in terms of infection counts. While the effects are not as large as the direct effects, they are still substantial. Trump vote share's effect on COVID-19 mediated through these important channels shows that pro-Trump states tend to implement social-distancing behaviors at lower rates, as previous research has shown, with consequent relative increases in infections. Furthermore, these associations are relatively constant given Trump approval polls, although there is a more stronger association for combined Trump approval polling and Trump vote share in dampening fears over COVID-19; i.e., in states that voted the most for Trump, when Trump approval reached it highest point then fear of COVID-19 declined the most (and infections consequently increased).

On the whole, this finding points to very strong associations between partisanship and the spread of the COVID-19, comparable or greater than the demographic and socio-economic factors in the model. States with higher Trump vote shares have seen significantly fewer infections via unexplained pathways, but very importantly, this decrease did not come through reduced mobility nor increased concern over COVID-19. The direct relationship is likely an artifact of the pattern of the early spread of the virus. After all, it is well-known that the early states that were infected with COVID tended to vote against Trump, although partisanship is not why they were more vulnerable to COVID initially. We believe that pro-Trump states received fortuitous outcomes by happening to not be on major travel routes from early COVID-19 hot spots; rising Trump approval in these states occurred as pro-Trump residents believed their president's dismissal of the virus' threat. In other words, the unexplained direct effect justified the relative inattention to important behaviors that could prevent infection. Given the increase in COVID-19 infections in the last two months in heavily Republican states, it would seem that this tendency would lead pro-Trump states to suffer in the long run as behavior caught up with initial conditions.

Finally, we can also use estimates of cell phone mobility on COVID-19 to understand how NPIs have had mediated effects on the disease through increasing or decreasing mobility. Figure 11 shows the disaggregated mediation effects for two types of policies, restrictions on businesses and stay-at-home orders. The plots reveal how indirect mediation effects change substantially over time. Panel A shows that business restrictions had a powerful suppressive effect on workplace mobility and to a lesser extent retail establishments during the early part of the epidemic, though that association weakened over time. By contrast, panel B indicates that stay-at-home orders have had more durable effects on mobility that have suppressed the disease, particularly

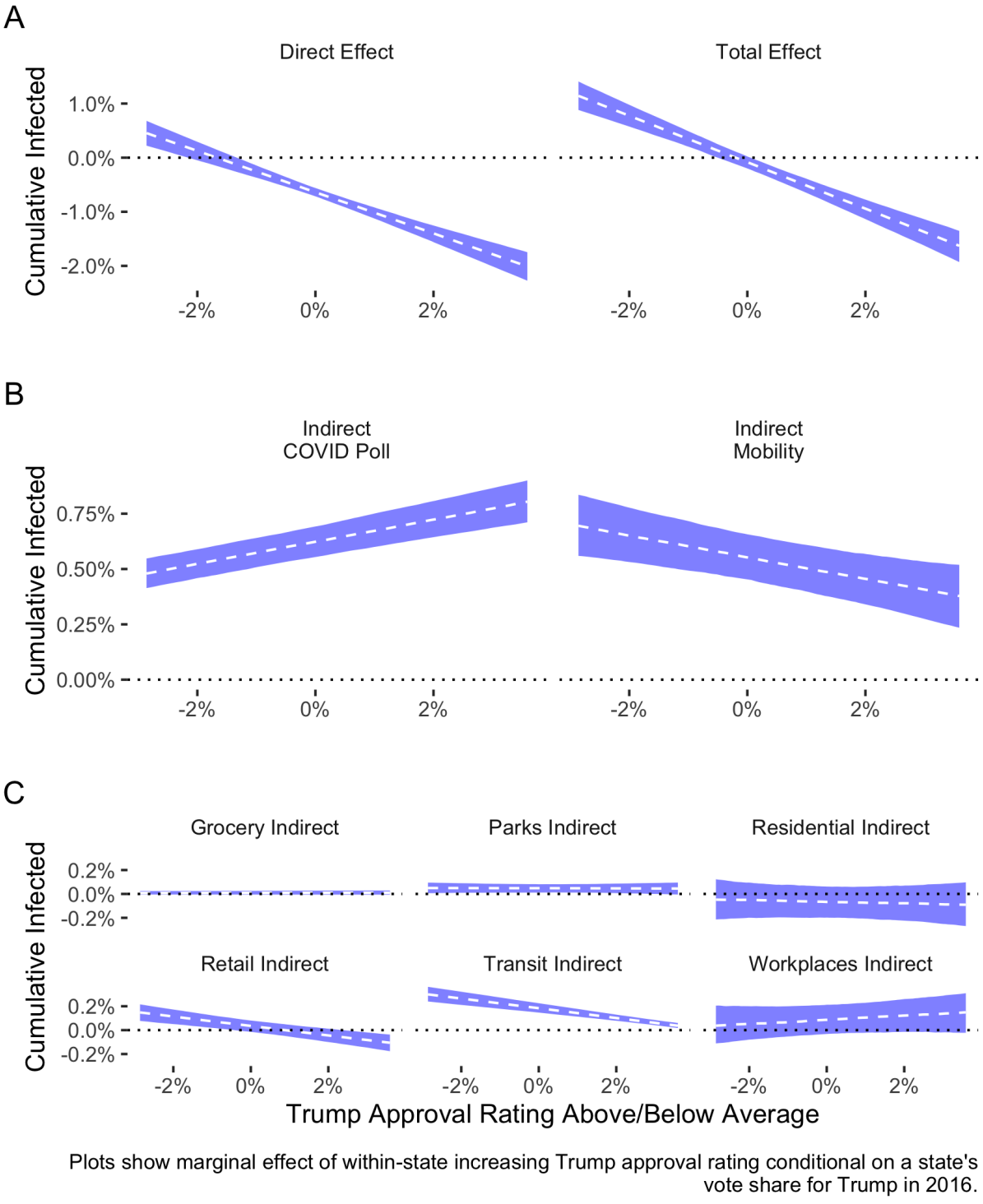


Figure 10: Marginal Effects of Trump Vote Share in 2016 Conditional on State Approval Polls

in retail establishments, workplaces and transit. Furthermore, these effects seem to be increasing rather than decreasing over time. On the other hand, stay-at-home orders seem to be increasing disease infections via increasing residential mobility, trade-offs that were noted in some early epidemiological modeling of COVID-19 (Seth Flaxman 2020).

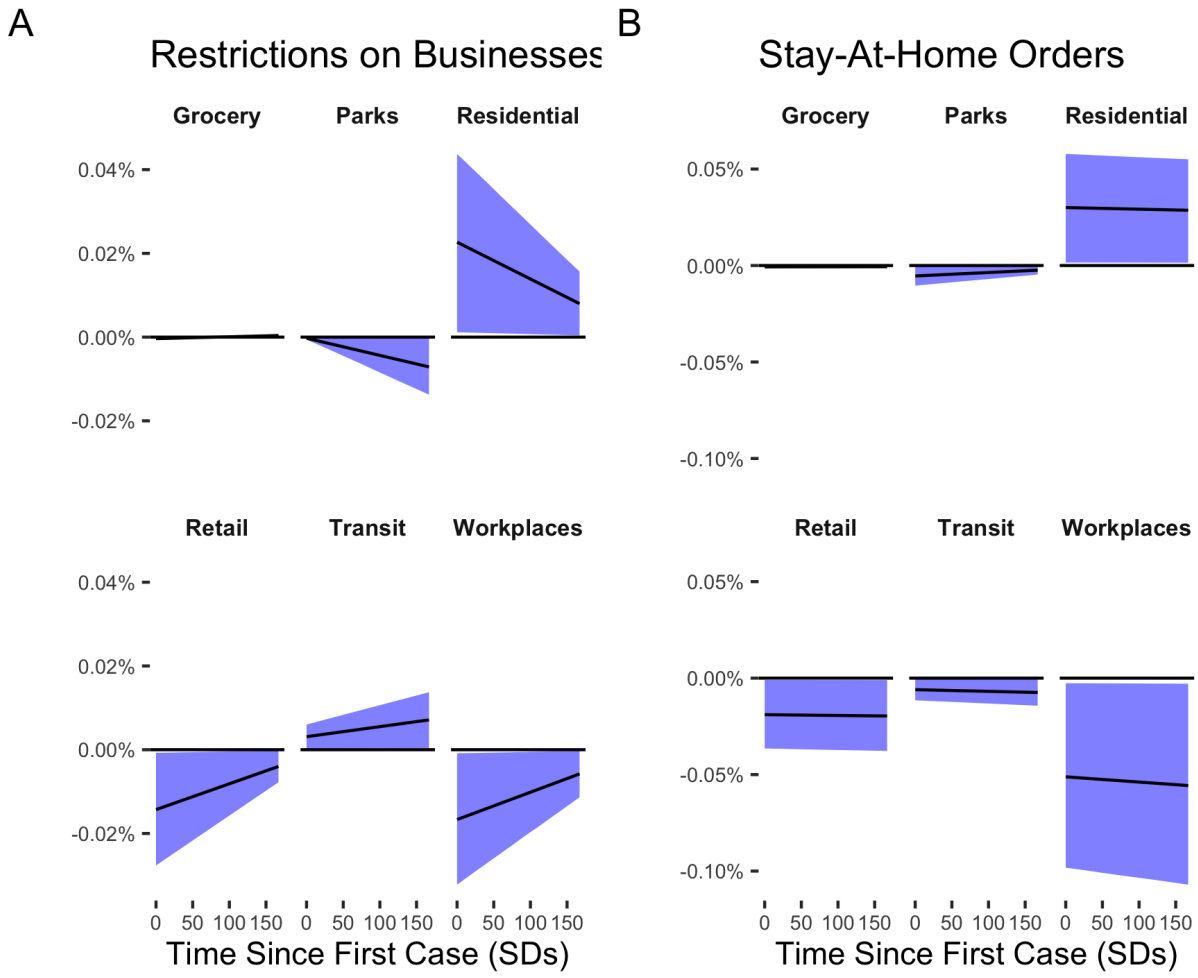


Figure 11: Mediated Effects of Lockdowns on Google Mobility Data

To compare the associations of NPIs to that of partisanship we can consider a policy of a given length of implementation. Both business restrictions and stay-at-home orders reduced infections primarily via reducing retail mobility and workplace mobility, with stay-at-home orders with associations twice as high per day compared to business restrictions. For stay-at-home orders, a policy implementation of the full sample period days is associated with a total reduction in infections of -0.59% (95% UI -0.03%, -1.14%). If we consider

that a 1-SD increase in Trump's 2016 vote share is associated with an increase in the infection rate of 0.38% (95% UI 0.23%, 0.51%) via mobility alone, it appears that a stay-at-home order would need to be implemented for at least 100 days to compensate the partisanship association, especially when Trump approval polls were rising.

5 Conclusion

Our results show that sociopolitical covariates like partisanship are equally as important predictors of the disease's spread as are NPIs designed to counter the pandemic. These results suggest that future research take into account these covariates even if they are not traditionally included in epidemiological studies. The politicization of the pandemic undermined measures to combat and led to reduced concern over the virus in the United States, with serious consequences for individuals' exposure to SARS-CoV-2. We find that politicization on the right end of the political spectrum has the strongest association with increased spread of COVID-19, although left-leaning political activity aimed at ending policy brutality via protests is also associated with increased spread, though at a lower scale.

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