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# COVID-19 TRANSMISSION ESTIMATES AND FORECASTS

## A MODEL TO TEST SAMPLE SELECTION BIAS IN A 'LOW RISK' CRISIS

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### Abstract

This paper surveys estimates of the transmission features of the novel coronavirus, and then proposes a model to address sample-selection bias in estimated determinants of infection. Containment assumptions of the infection forecasting models depend on assumed effects of policies and self-regulating behavior. In the commons dilemma of the pandemic, the perceived 'low risks' of unregulated marginal choices do not reflect the full social cost, implying non-pharmaceutical interventions (NPI) to reduce mortality can enhance social welfare. As more economic activity renews with liftings of restrictive NPI (RNPI), a critical question concerns the ability of milder NPI (MNPI) and voluntary precautions to mitigate the risk of greater infections and deaths while also limiting the pandemic's economic damage and its social costs. Ineffective NPI could lead to continued COVID-19 waves and new types of crises, worsened expectations and delayed economic recoveries. From the central range of surveyed estimates of transmission and alternative herd-immunity-threshold estimates, a 'worst-case' virus guidepost suggests eventual deaths of around 25 to 41 million worldwide and 1.1 to 1.7 million in the U.S. needed to reach herd immunity with no vaccine or treatment. The most optimistic study surveyed (theoretical model from a non-reviewed preprint study) combined with the low end of the range of the estimated mortality rate suggests 6 to 9 million deaths worldwide and 250 to 370 thousand in the U.S. to reach herd immunity. Successes in the mix of NPI, treatments, and vaccine can limit the eventual global death toll of the virus. Improved estimation models for forecasting and decision making may assist in better targeting the local timings and mix of NPI. Diagnostic tests for the virus have been largely limited to symptomatic cases, causing possible sample selection bias. A recursive bivariate probit model of infection and testing is proposed along with several possible applications from cross-section or panel-data estimation. Multiple potential explanatory variables, data sources, and estimation needs are specified and discussed.

Keywords: COVID, coronavirus, sample selection bias, bivariate probit, public goods, macroeconomic

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## 1. Introduction

This paper surveys and analyzes estimates of the transmission features of the virus, and then proposes a model to address sample-selection bias in estimated determinants of infection.

The global surge of the pandemic has sharply lowered economic activity due to the rapid shift in expectations and precautionary behavior coupled with non-pharmaceutical interventions (NPI) for virus containment. In the commons dilemma of the pandemic, the perceived 'low risks' of unregulated marginal choices do not reflect the full social cost, with a socially suboptimal outcome (Eichenbaum et al 2020), implying NPI to reduce mortality can enhance social welfare. Restrictive NPI (RNPI) includes business shutdowns, travel restrictions, and shelter-in-place- and nonessential-stay-at-home orders. Milder NPI (MNPI) includes less restrictive measures such as: masking, enhanced exposure notification for quarantine, shielding of the vulnerable, social distancing in public spaces and gatherings, enhanced population testing, and messages for self-regulating behavior. As more economic activity renews with liftings of RNPI, bringing new and reelevated COVID-19 risks, a critical question concerns the ability of MNPI and voluntary precautions to mitigate the risk of greater infections and deaths while also limiting the pandemic's economic damage and its social costs.

Ineffective NPI could lead to continued COVID-19 waves and flare-ups and new types of crises worldwide, worsening expectations and delaying economic recoveries. A worst-case virus assessment guidepost requires only the herd immunity threshold equation or estimate, estimates of the reproduction ratio, and the estimated mortality rate. The analysis in section 2 suggests eventual deaths of around 25 to 41 million worldwide and 1.1 to 1.7 million in the U.S. needed to reach herd immunity with no ultimately successful treatments, vaccine, and NPI. The most optimistic study surveyed (theoretical model from a non-reviewed preprint study) combined with the low end of the range of the estimated mortality rate suggests 6 to 9 million deaths worldwide and 250 to 370 thousand in the U.S. to reach herd immunity. Successes in RNPI, MNPI, and voluntary behavior have limited the high mortality threat to date, as more focus now shifts to MNPI and self-regulating behavior, with RNPI fallbacks, while hopes for future

treatment or a vaccine continue. Improved model estimates and forecasts may enhance policymaking on the local timings and mix of NPI.

Diagnostic testing for the novel coronavirus has been largely limited to symptomatic cases while many unreported mild or asymptomatic infections have been found. To address and test sample selection bias in estimating effects on observed infections, a recursive bivariate probit (RBP) model is proposed in section 3 along with several possible applications in section 4 from cross-section or panel-data estimation. Multiple potential explanatory variables, data sources, and estimation needs are specified and discussed. Case-level scoring models built from anonymized data may assist in advanced and under-privileged or constrained clinical diagnostic settings. Key estimated marginal effects on local infection rates include those from RNPI and MNPI to compare with other model results.

## **2. Estimates and Assessments of SARS-Cov-2 Transmission, Mortality, and Pandemic Policies**

Policymakers in the COVID-19 crisis face many tradeoffs and uncertainties worldwide. Institutions must consider different scenarios of the pandemic with precautionary reduced spending and investment, ongoing policy interventions, and the resulting macro and local economic effects. Containment assumptions of the infection forecasting models depend on assumed effects of policies and self-regulating behavior (e.g. Chowdhury et al 2020), which especially impact longer-term projections.

RNPI, MNPI, and voluntary increases in precautionary behavior have played simultaneous roles in containment. Varying data and measurements have allowed some first estimations of the effects of NPI and behavior on infection incidence and transmission rates, finding large effects (Courtemanche et al 2020; Unwin et al 2020; Jarvis et al 2020). Ferguson et al 2020 calibrates a model from UK and multi-country data and forecasts the effects of more vs. less restrictive virus ‘suppression’ vs. ‘mitigation’ policy interventions, testing the effects of intervention cycles. Andersen et al (2020) exploit a natural experiment: Denmark and Sweden were similarly exposed with only Denmark mandating significant restrictions. Estimated aggregate spending dropped about 25 percent in Sweden and only an additional 4 percent in Denmark, suggesting economic contraction was due largely to the virus itself and resulting

precautionary behavior. Economic recoveries may generally depend on consumer and business fears and expectations, likely reacting to NPI successes or failures in containment and hopes for a successful PI. Calibrations of the parameters in forecasting models require continued estimates of the marginal effects of NPIs and other determinants of transmission, infections, and mortality.

Virus transmission propensity depends on the critical base reproduction ratio of the virus,  $R_0$  (new cases produced by a typical single infection in a fully susceptible population). This reflects the intensity of effective contact between the infected and susceptible populations, referred to as the transmission coefficient or effective contact rate,  $\beta$ .  $R$  is equal to  $\beta \times d$ , where  $d$  is the average duration of infectiousness. Transmission rates (the speed of spread) depend on both  $R$  and the ‘serial interval’ (average time between onset in primary and secondary cases); i.e., the speed of a pandemic depends on how many other people each case infects and how long it takes for infection between people to spread.<sup>1</sup> Base transmission rates due to differing  $\beta$ 's can vary widely across local environments depending on population density, local transit, customary gatherings, etc. This has generally led to much lower infection rates in rural versus urban areas, for example (although highly connected groups may dominate transmission in any environment). Unwin et al (2020) estimated an  $R_0$  of 5.0 for New York versus 0.3 for Montana, for example.  $R$  is also of course lowered by traditional quarantining of cases and exposures (from contact tracing), and by RNPI, MNPI and voluntary social distancing.

The relationship between the incubation period (how long it takes for symptoms to appear) and the serial interval, and the duration of infectiousness, have both had important implications for the relative spread of the virus (SARS-Cov-2). The median serial interval of the seasonal flu exceeds the average incubation period, and the range of flu incubation periods is distributed comparatively narrowly, as shown in Fig. 1. In contrast, the estimated SARS-Cov-2 serial interval is generally somewhat less than the estimated median incubation period, and the range of incubation periods is distributed across a

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<sup>1</sup> The serial interval is important for epidemiological estimates of  $R_0$  based on available information as it unfolds.

considerably longer number of days. The median or average incubation period for SARS-Cov-2 is around 5-6 days, but it can vary from 1 to 14 days (WHO 2020). The virus has been characterized by a high  $R_0$ , a long incubation period, and a relatively shorter serial interval (Xie & Chen 2020; Van Buesekom 2020).

Furthermore, although ultimate statistical outcomes are less severe across a population of the typical flu-infected versus the SARS-Cov-2 infected, a comparatively larger share experience no or only mild symptoms from SARS-Cov-2 (which most shrug off without seeking treatment or testing). Symptoms across these respective infected populations are thus more bimodal with SARS-Cov-2. Most people with the flu know they have it and naturally self-quarantine; likewise, with the original SARS in 2003 and its severe symptoms. Not so with SARS-Cov-2, a critical difference.

These key features of the virus clearly elevate the relative public health risk. Each implies a larger scope for 'asymptomatic' or pre-symptomatic transmission. More of the infected population simply passes the virus along to others unwittingly. In addition, a relatively low serial interval exacerbates the health system pressures when COVID-19 cases surface and flow through in faster cycles.

The range of current estimates of the general  $R_0$  for SARS-Cov-2 considerably exceeds the  $R_0$  of the widely studied seasonal flu, as shown in Fig. 2. Compared with 1.3 for the flu, the estimates for SARS-Cov-2 range from the low 2's to 5.7 or more. The effective contact rate of SARS-Cov-2 is elevated by its high unwitting non-quarantining spread, and the infectiousness duration of someone with SARS-Cov-2 (14 days or more after onset) may be at least twice that of the flu.<sup>2</sup>

The higher general base  $R_0$  well above 1.0 implies not only that a single case infects more people in an exponential spread, but the ultimate overall need for virus extinction, developed herd immunity plus vaccine efficacy, is also higher. This reflects the basic herd immunity threshold (HIT) equation of  $1 -$

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<sup>2</sup> Research on the severity and duration of SARS-Cov-2 'viral shedding' is another important area of research. Bullard, et al (2020) estimate that infectiousness may not last beyond 8 days after the onset of symptoms for those with a specific SARS-Cov-2 gene value.

$1/R_0$  (Fine et al 2011).<sup>3</sup> Higher values of  $R_0$  increase the HIT at the rate of  $1/R_0^2$ . Herd immunity will not convey biological immunity to all or stop transmission, but it tends to abate the spread of a virus toward population extinction. NPI policies and behavioral social distancing can also similarly abate the virus at any time if and where they suppress  $R$  below 1.0. The studies summarized in Fig. 2 have confirmed expectations that strong NPI social distancing has managed to lower the virus's  $R$  considerably. Within the strongest estimated effects, Unwin et al (2020) estimate that post social-distancing  $R$  recently reached below 1.0 in about half of U.S. states, correlated with a sharp drop in observed mobility; however, the same study estimates that the suppressions of  $R$  will become temporary to the extent fully normal activity resumes, leading to many more deaths. Without continued caution, local conditions may allow the virus to resume previous exponential spread, and in any area new viral seedings from elsewhere may elevate transmission, particularly if a highly connected group with few precautions becomes infected.

The above observations from the range of empirical studies are consistent with the devastating mass outbreak in NYC,<sup>4</sup> and with recent random antibody test results from New York State and elsewhere showing a large share of undetected infections (Hendrickson & Strum 2020). Li et al (2020) concluded that 86% of all infections in China were undocumented before travel restrictions and undocumented infections were the source of 79% of the documented cases. Such findings also of course lower the estimated virus mortality rate.

Estimates of total (detected and undetected) virus mortality have been generally around 1% (Preidt 2020), although recent estimates suggest it may be as low as 0.5% (Hamilton 2020). A 0.5 to 1% mortality rate is about 5 to 10 times higher than that of influenza, and with SARS-Cov-2 it can impact a

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<sup>3</sup> Simplifying assumptions make the equation complex to apply in many aspects of public health practice (Fine et al). Surviving SARS-Cov-2 infection is also assumed to generally convey immunity, similar to other diseases; observed reinfections have been rare, but research continues.

<sup>4</sup> During a bad flu season, the city could expect to see around 50,000 flu cases and 50 resulting deaths over the 6-week peak in January-February, as calculated from CDC estimates of 'flu-like illnesses' and the detailed data reported by the New York State Health Department. Over the 6-week period ending April 25, 2020 (6 weeks after the first reported NYC COVID-19 death) the city's bolstered health system saw the flood of COVID-19 cases lead to 11,544 deaths. This is limited to the city of New York only as of April 25. It does not include the thousands of additional deaths in the suburbs and surrounding jurisdictions and thousands more after April 25.

much larger share of the population infected from latent spread. Social distancing and case quarantining have reduced observed mortality, and there have been many reports of undercounting of COVID-19 deaths. Higher mortality rates have especially impacted vulnerable (pre-existing conditions and older) and under-privileged populations (e.g. Borjas 2020) and can similarly affect medical staff, first responders or others with high exposure and inadequate personal protective equipment (PPE). On the other hand, although there have clearly been many tragic deaths among the young also, the statistical mortality rate from SARS-Cov-2 has been considerably lower across younger groups without vulnerable conditions.

These numbers suggest that from the personal standpoint of most individuals, the virus may well be perceived as fairly ‘low risk.’ Although worse than the flu, 0.5 to 1% is much lower than the approximate 10% death rate from the original SARS virus in 2003, for example. This, along with strong incentives to resume normal economic activities, clearly elevates the common problem of SARS-Cov-2. Public health messaging drawing on insights from behavioral science may be most effective in helping to maintain voluntary social-distancing and support for further MNPI and any needed RNPI (Bonell et al. 2020). An important simple message could emphasize the need for a large share of the population to acquire the virus to suppress it ‘naturally,’ with national mortality well beyond the already grim toll. The virus has clearly shown its capabilities as an individually ‘low risk’ but collective social disaster.

Applying the benchmark HIT equation, examples from the estimates of  $R_0$  cited in Figure 2, and the estimated mortality rate, Table 1 shows estimates of the implied ‘worst case’ number of deaths in the U.S. and worldwide. These are the approximate numbers of deaths needed to reach herd immunity with no vaccine. They are labelled ‘worst case’ because they estimate the approximate eventual outcomes with no ultimately successful treatments, vaccine, and NPI. The shaded part of Table 1 focuses on the central part of the distribution of surveyed estimates. Using a mortality rate of 0.75%, this area of the table implies approximately 25 to 41 million worst-case deaths worldwide and 1.1 to 1.7 million in the U.S.

In addition to estimates from the basic HIT equation, Table 1 also includes some alternative HIT estimates based on two recent preprint (not peer reviewed) papers that argue for a lower HIT than implied

by the classic equation. Although most epidemiologists have centered on 60 to 70% as the likely HIT range for SARS-Cov-2, these alternative more optimistic models and arguments theorize that the HIT is reduced with highly susceptible or exposed groups becoming infected and immune first, preventative measures, and facilitating population connections (Britton et al 2020; Gomes et al 2020).

Although some have advocated it as a social strategy, herd immunity from natural spread through the population is not generally a social goal. Rather, total immunity = herd immunity + vaccinated immunity or effective treatment is the necessary ultimate target for virus suppression. In addition to mitigating health-systems overload, effective social distancing and NPI precautions ‘buy time’ until vaccinated immunity or successful treatment can substitute to ultimately suppress the virus effects and keep the eventual death toll down. Allen, et al (2020) refer to a maintained normalcy with ‘no social distancing’ approach as the “surrender” strategy.<sup>5</sup>

Maintaining MNPI measures may be better for long-run economic growth. SARS-Cov-2 transmission is insidious, inviting commons-dilemma fueled complacency. Remaining health threats with further economic repercussions, including “second wave” or “saw tooth” behavior in the virus and local economies, should not be under-estimated, even as more locations in the U.S. and globally continue to relax RNPI. Latent infections and transmission may cause outbreaks that can overwhelm any local health care system and economy. COVID-19, like the 1918 pandemic, is likely having a significant negative impact on many businesses and long-term economic growth in any event (Eichenbaum et al 2020; Andersen et al 2020; Garrett 2007; Brainerd & Siegler 2003). Evidence across cities from the 1918 pandemic suggests that cities that intervened earlier and more aggressively if anything grew faster after the pandemic (Correia et al 2020). For a variety of reasons, low- and middle-income countries are the most vulnerable to SARS-Cov-2 and high mortality globally (Khalatbari-Soltani et al. 2020), and they also suffer from the reduced global economic growth. Because of many global connections that can only

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<sup>5</sup> Only Sweden has adopted anything close to this in policies, but their population has also had much voluntary social distancing and precautionary behavior, as observed from Andersen et al (2020) cited earlier and other studies.

be limited at significant additional economic costs, worldwide herd immunities plus vaccine efficacy or effective treatment and quarantines are likely needed to fully suppress the threats of the virus to the U.S. Expansion of trade wars and many other socioeconomic and political unintended consequences may follow from less effective policymaking, further slowing both U.S. and global economic growth in both the short and long term.<sup>6</sup>

In the prospects for the “time buying” success of NPI, the unprecedented efforts across the globe to achieve one or more effective treatments or vaccine should not be counted out. Based on the constraints evident from past virus-containment and pharmaceutical experiences, no single 100% ‘cure’ for the pandemic is likely, and no satisfactory vaccine may be found (Picheta 2020). Successes in the mix of NPI, treatments, and vaccine, however, can mitigate the eventual global toll of SARS-Cov-2.

### **3. A Proposed Complementary Model for Estimating Determinants of Infection Rates Across Individuals or Local Areas Correcting for Sample-Selection Bias**

Estimation of the effects of NPI and other factors in local transmission is proposed in a recursive bivariate-probit model to address sample-selection bias, a method used in other economic and biostatistics applications (Marra et al 2014; Greene 2003/12; Cuddeback et al 2004). Applicable to individual or local-area data, the proposed model is complementary to other methods and broader testing.<sup>7</sup> Data to estimate the model are now widely reported for local areas across U.S. states and counties, for example. Individual-level case data also exists, although these developing data must be anonymized.

The key local-area unknown is the total infection rate, i.e. the full infected proportion across or within local-area population(s). The numerator includes the observed test-positive cases plus many unobserved pre-symptomatic and asymptomatic infections in individuals never tested (or who previously tested negative but could test positive now if tested again). We can only observe whether some random

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<sup>6</sup> To be sure, many trade strategies of all countries are being rethought in various ways in the anticipated wake of the COVID-19 crisis.

<sup>7</sup> A bivariate logistic approach is also possible, although not common. Broader testing has been widely advocated to enhance quarantining and better target NPI measures (e.g. Berger, Herkenhoff & Mongey 2020; Begley 2020).

individual from the population is infected based not on their true infection status, but on the observed outcome of the sample-selection variable, tested or not tested. The selection process into observability has been nonrandom. This creates the well-known problem of sample selection bias in the estimated parameters of a single-equation model of infection incidence.

The explanatory variables are likely to differ somewhat in the infection-incidence equation and the sample-selection equation, as there are factors that affect testing incidence but not the incidence of infections. Consider the following plausible determinants of the Infection versus Testing likelihoods in the general bivariate probit model specification:

$$(1) \quad y \sim \text{Pr}(\text{Infected}) = f(\text{Pop. Density, Living Facilities, Mass Transit, Work Conditions, Medical, First Responder, Income, Social Distance Practices, Recent Infectious Contacts ...}) + v$$

$$(2) \quad s \sim \text{Pr}(\text{Tested}) = g(\text{Infected, Flu, Living Facilities, Medical, First Responder, Age, Vulnerable Health Condition, Income, Pop. Density, Work Conditions, Social Distance Practices, Recent Infectious Contacts ...}) + v$$

$$\rho = \text{Corr}(v, v) \neq 0 \quad \text{nonzero correlation of error terms}^8$$

The conceptual variables specified in (1) and (2) are largely self-explanatory effects on the effective contact rate and transmission vulnerability; these variables are specified in more detail in Table 2 below, with the expected signs on the variables. The variables are also more or less difficult to capture or measure depending on the nature of available data and level of information sought, and some may not be measurable easily or at all. “Flu” in the testing equation indicates the possibility of an individual likely to

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<sup>8</sup> Nonzero correlation of the error terms is likely for several reasons, particularly with nonrandom testing: 1) Unobserved factors impacting infection likelihood also affect testing likelihood. 2) Most of the population’s enhanced fear to enter any medical or testing facility (for fear of contracting the virus) plus many doctors’ needs to triage limited test kits in favor of more likely cases, has tended to keep spurious test attempts down. 3) Diagnostic tests include false negatives. If someone tests negative for the virus, and particularly if they also test negative for the flu, they are often simply sent home and instructed to quarantine for 14 days. Only those with worsening symptoms, i.e. the “sickest of the sick,” will typically return later for a repeated SARS-Cov-2 test, even though some number in the “least sick of the sick” also have the virus. Since retesting is more likely to be performed on those truly infected with SARS-Cov-2, this boosts the likelihood of testing, based on this unobserved process, in a positive correlation with the likelihood of infection (retested cases could be dropped from estimation, but this would raise another potential sample selection bias).  $v$  is correlated with  $v$ . Simply estimating equation (1) alone will result in coefficient estimates that are biased and inconsistent.

be given a SARS-Cov-2 test due to similar symptoms from the flu. If the presence of seasonal flu or other flu strain is always tested first, then testing for SARS-Cov-2 is sequential. Flu testing may also be given simultaneously or not at all. The predicted effect of “Flu” on net is ambiguous. Recent Infectious Contacts is an important variable clinically and in local areas. Some sampled large reductions in transmission across local areas attributed to NPI may reflect high infectious contacts from recent international travel (Miller et al 2020), and the benefits of NPI may well differ in less exposed areas.

Notice that (1) does not include Age or Vulnerable Health Condition (controlling for Living Facilities). This maintains the simplifying assumption that other than in more dense living facilities such as nursing homes and high-density housing and neighborhoods, pre-existing health-vulnerability conditions primarily affect the likelihood of infection severity, rather than the incidence of infection. A more complete model, beyond the scope of this paper, would also include an equation predicting the likelihood of infection severity, with age and health vulnerability (correlated with other variables such as income and race; Borjas 2020) expected to have large effects.

The postulated model specifications in (1) and (2) lead to the recursive bivariate probit model specification (Maddala 1986; Filippini et al. 2018) given that the endogenous variable, Infected, appears in (2). Using the standard latent-variable specification for limited-dependent variables, this model is:

$$(3) \quad y^*_i = \beta'_1 X_{1i} + v_i \quad , \quad y_i = 1 \text{ if } y^*_i > 0 \quad , \quad y_i = 0 \text{ otherwise}$$

$$(4) \quad s^*_i = \delta y_i + \beta'_2 X_{2i} + v_i \quad , \quad s_i = 1 \text{ if } s^*_i > 0 \quad , \quad s_i = 0 \text{ otherwise}$$

$$[v_i, v_i] \sim \Phi_2[(0, 0), (1, 1), \rho], \rho \in [-1, 1]$$

where  $i$  is the individual index;  $y^*_i$  and  $s^*_i$  are latent continuous variables for which only the binary variables  $y_i$  and  $s_i$  are observable;  $X_{1i}$  and  $X_{2i}$  are the explanatory vectors of exogenous variables specified above in (1) and (2) above and Table 2 below; and  $[v_i, v_i]$  is the vector of error terms discussed above and here explicitly specified in  $\Phi_2$  — the bivariate standard normal distribution with the correlation  $\rho$ . For other previous applications of selection-bias correction methods in epidemiology, medical care and

biostatistics, see e.g. Wirth & Tchetgen Tchetgen 2014, Cheung 2001, Zimmerman Murphy 1987, Rosenman et al 2010, Crealey & O'Neill 2018, and Marra et al 2014. Marra et al derive associated Lagrange multiplier and Wald tests for the hypothesis of absence of unobserved confounding (in this case unobserved variables associated with both testing and infection). For estimation of marginal effects in the bivariate probit model, see Greene (1996).

Table 2 shows many of the potential data sources and defines the explanatory variables in (1) and (2) more specifically, and their postulated effects. Equations (3) and (4) are specified, as usual, at the individual level, as this is where the endogenous selection and outcome variables of Tested and Infected are observed. Case-level data, however, although potentially valuable across various applications, is not needed to estimate equations (3) and (4) as described below. Case-level data requires both anonymizing and careful assessment of the distributions of each of the individual-level data elements to remove erroneous data, address missing information, and so on. Such data could include additional explanatory variables beyond those listed in Table 2, including clinical/medical-condition indicators. Organizations have announced plans and proceeded to assemble case-level data to make it more widely available.<sup>9</sup>

As shown in Table 2, group-level local-area data is widely available from multiple data sources. For the set of endogenous and explanatory variables postulated, these data also have some missing information, differences in reporting, etc. but these data are readily accessed at the current time. With respect to individual-level reporting errors, the Central limit Theorem suggests that for a sizable local-area population the reported group-level means from individual data should tend to be normally

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<sup>9</sup> Ciox, a group partnering with LabCorp, for example, announced on April 10, 2020 that they are performing anonymization “with an initial data set based on LabCorp's nearly 500,000 completed COVID-19 tests” (Eddy 2010). Two weeks later, Cerner Corporation announced a similar plan to offer “select U.S. health systems and academic research centers complimentary access to critical de-identified COVID-19 patient data to help fight the pandemic. This offering will provide eligible health care researchers free access to Cerner's COVID-19 data set to support epidemiological studies, clinical trials and medical treatments related to COVID-19, in line with applicable laws and guidelines” (Cerner Corporation 2020). See Jason (2020) who also describes these efforts and another similar one from Health Catalyst. The WHO also has a Global COVID-19 Clinical Data Platform, although it is limited to hospitalized patients. Similarly, the CDC has a database, COVID-NET that was “implemented to produce robust, weekly, age-stratified COVID-19-associated hospitalization rates.” (Garg et al. 2020). Organizations first providing the anonymized-case databases may also request feedback from qualified initial users.

distributed. For decision-making beyond the clinical setting, policy choices are primarily focused on local areas and types of activity, particularly which parts of a state can ‘re-open’ and remain as such under what kinds of conditions. For these reasons also, local-area-data modeling and projections that are faster to produce have significant clear advantages.

To estimate equations (3) and (4) with local-area infection and testing rates, the reported rates represent a fractional group incidence of the underlying Bernoulli distributed 0’s and 1’s. A recursive bivariate fractional probit model (along the lines of Schwiebert 2018) may be needed for estimation across multiple local areas. Alternatively, synthetic assignment of 0’s and 1’s to represent the local-area rates allows a straightforward estimation of equations (3) and (4). Other estimation and data considerations for estimating this specific model are described in the Appendix.

#### **4. Potential Applications of the Recursive Bivariate Probit Model Estimates**

*Scorecard Models:* With anonymized case-level data, an estimated scoring model can rank order the likelihood that an individual with symptoms is infected with SARS-Cov-2. A preliminary scorecard-type model estimation has been reported for the virus using patient and control- and validation- group clinical and other data (Tordjman et al. 2020).<sup>10</sup> These authors found no clinical variables that were statistically significant. A scoring model controlling for sample selection bias could be valuable in different ways in both advanced and under-privileged health systems. With insufficient diagnostic tests available, a scorecard may be able to assist providers in triaging the available tests. It may also help assess more uncertain cases that have tested negative, or patients that can only be evaluated remotely. More aggressive quarantining may be advised or pursued for those with a higher likelihood of infection.

If a stratified random sample of testing has been performed across local areas, it may be used together with individual- or area-scorecard results. Scorecard results can assess and rank order the distributions of infection-risk characteristics within and across local areas. Such complementary findings

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<sup>10</sup> This is a pre-print study prior to peer review. The authors used univariate and binary logistic analysis to derive their score, apparently with no determined need or no attempt to control for possible sample selection bias.

may allow more precise prediction of the probabilities of infection across local areas, improving identification of emerging and potential nearby outbreaks, including areas currently showing fewer cases. Individual- or area-scorecard model results may also be used to better target stratification in random testing. Areas with higher infection-risk characteristics may be targeted for higher sample weights. Scorecards may also be used for initial estimates of area infection rates prior to random sampling.

*Hazard-Models:* Scorecard or projection models can be built using panel data (relatively easier to collect and assemble at the local-area level). Hazard models, such as Cox proportional hazards, or panel-logit or panel-probit which estimate a discrete hazard rate, allow complementary predictions of infection rates over time based on baseline hazard paths and time-varying covariates.

Panel data models also allow the use of fixed- and random-effects estimates to capture unobserved fixed characteristics and time varying behavioral changes (although fixed- or random-effects used exclusively do not estimate the effects of specific policy-relevant variables). Fitting the bivariate probit model with panel data is described, for example, by Roodman (2011) and Miranda (2010). Mulkey (2015) describes methods for computing a bivariate probit model on panel data with correlated random effects. See also Vella & Verbeek (1999) and Verbeek (1990).

*Improved Assumptions in Epidemiological Forecasting Models:* Epidemiological (typically SEIR infectious disease) models remain the key tools for forecasting the future paths of infection across local areas, particularly given the potentials for rapid exponential spread due to the high general  $R_0$  of SARS-Cov-2. Empirical hazard-model results may be able to provide useful complementary benchmarks and insights in refining parameter assumptions within the epidemiological models.

As discussed, the most pressing but very uncertain state and local policy questions today concern the effectiveness of various RNPI and MNPI gradations of social distancing in reducing the spread of infection. Courtemanche et al. (2020), for example, used county data across the U.S. to estimate the marginal impacts on reported infection growth rates of specific local-area gradations of RNPI social-

distancing measures, controlling for unobserved fixed effects.<sup>11</sup> This study included testing incidence as a control variable in the estimation, which implicitly assumes that  $\rho = 0$ , a testable assumption in the RBP model. Testing incidence was also measured at the state instead of county level.

*Commons-Dilemma Policy Assessments and Macroeconomic Scenarios:* Voluntary social distancing behavior and reasonable compliance with MNPI and CDC guidelines could be just as or more important than RNPI. The U.S. and many other nations are increasingly relying in practice on more voluntary and uncoordinated mass behavioral changes (Weible et al. 2020) to help limit the mortality toll of the pandemic. Estimates of this effectiveness, together with creative NPI strategies (e.g. Ferguson et al. 2020; de Walque et al. 2020; Mello & Wang 2020), may help improve model projections, policy decisions, and ultimate mortality and economic outcomes in the ‘new normal’ of the COVID-19 crisis and any similar pandemics in the future.

Macroeconomic forecasters and economists have attempted to address the interdependency of the path(s) of the virus with national and local macroeconomic scenarios – a complex problem of the virus behavior, the social psychology of expectations and precautionary behavior and the commons dilemma, and the extent, effectiveness, and duration of stress-counteracting macroeconomic policies. The key question today is whether the macroeconomic effects of the pandemic will be short- or longer-lived.

## 5. Conclusion

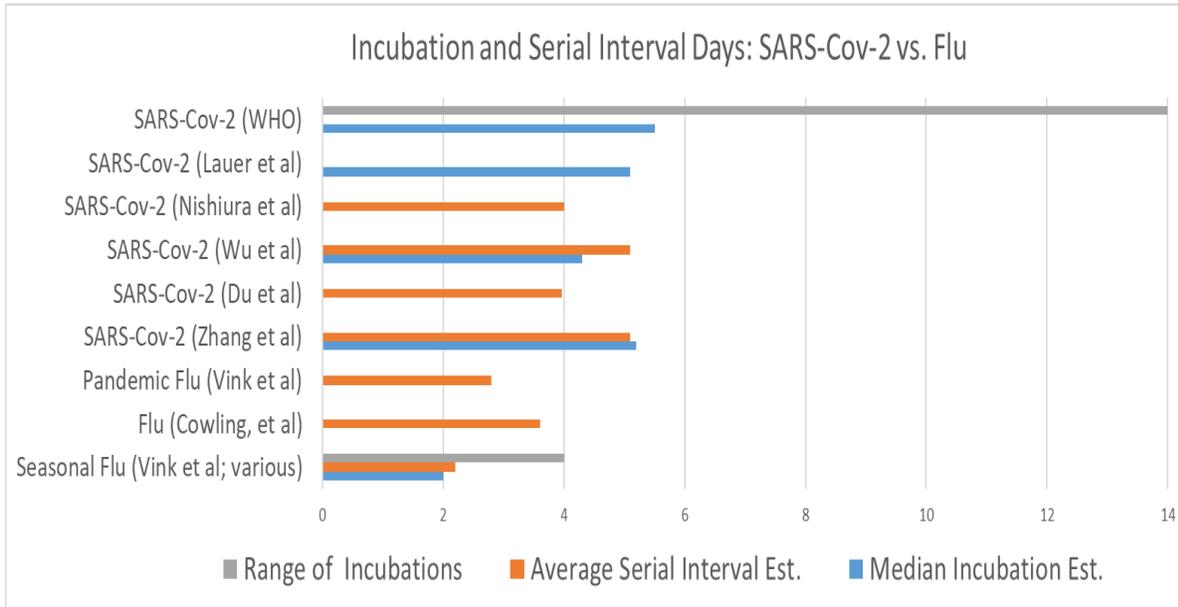
This paper’s survey and assessment of estimates of the transmission features of SARS-Cov-2 highlights the importance of continued estimation of NPI and other covariate effects on the virus’s behavior and mortality outcomes. The recursive bivariate probit model proposed can be used to address sample-selection bias in infection data, with multiple applications suggested. Longer-term projections of infection forecasting models especially depend on assumed effects of NPI policies and behavior.

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<sup>11</sup> In qualifying their conclusions, the authors note the changing nature of voluntary social distancing over time. Random effects may be better able to help quantify this, which could be important.

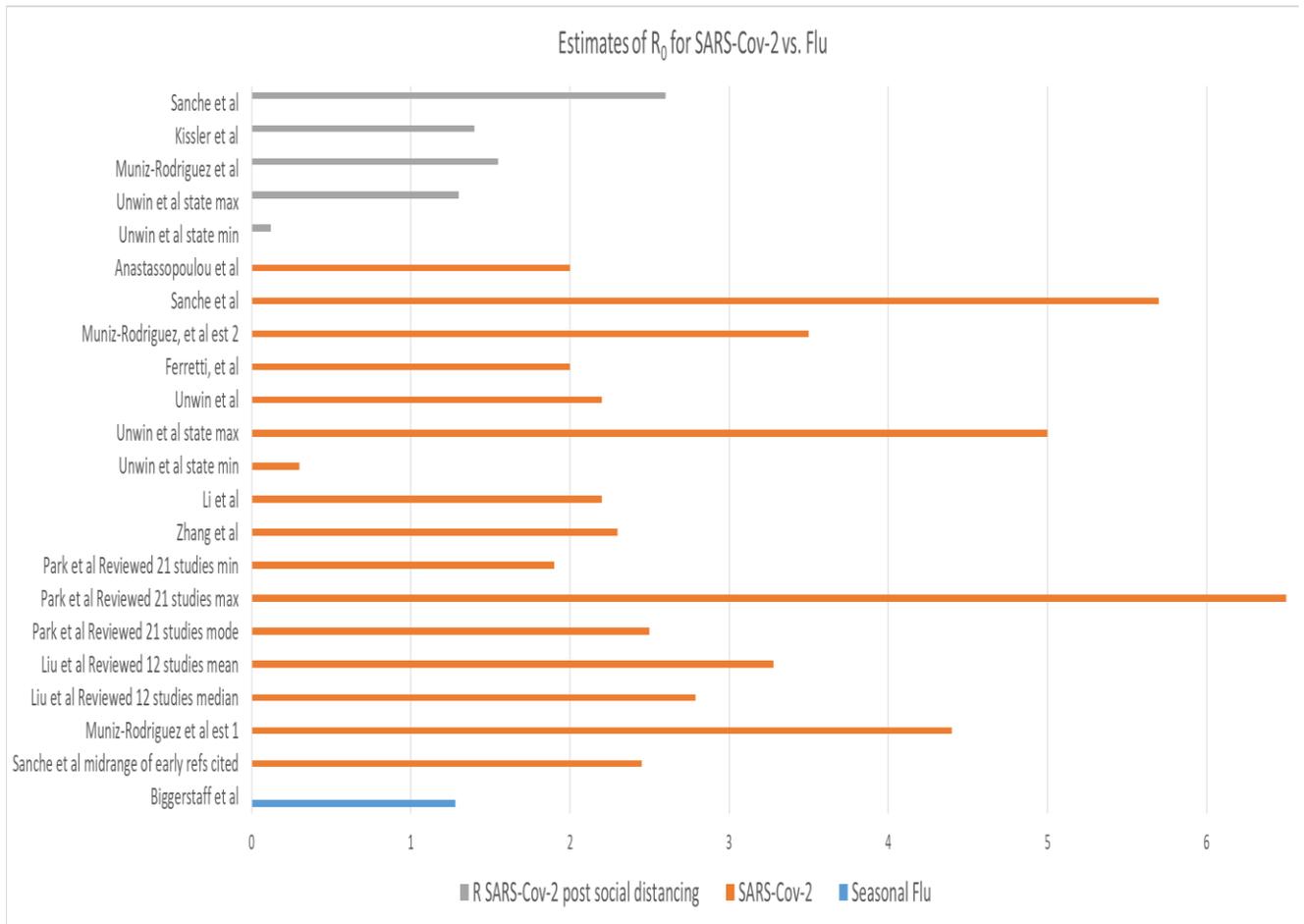
History may provide some additional insight on current pandemic policies. Policies across individual U.S. states today parallel previous global experiences in pandemic policymaking with two alternative directions (Baekkeskov 2016). One policy (correlating with states with lower  $R_0$  and more limited outbreaks) emphasizes a national ‘theme,’ which tends to be followed with countervailing information ignored or ‘underreaction’ (Maor 2014) until such information becomes overwhelming. This has placed national and many state pandemic policies on a path of first ‘ignore,’ then ‘shut down,’ then ‘reopen.’ This policy appears most conducive to recurring waves of the virus *unless* MNPI and voluntary measures are sufficiently effective, particularly in higher  $R_0$  areas, while lower base and seeding conditions in lower  $R_0$  areas remain protective. The other policy approach follows a more local ‘knowledge-and-validation community’ where accruing information leads toward better consensus on appropriate policies. The timing of recent policy changes in the U.S., rapid spread of RNPI, then relaxing RNPI in all states some weeks in advance of that recommended by most experts, also shows a ‘domino effect’ – i.e. the national-theme-led policy has affected all states. If MNPI and voluntary precautions (with local RNPI if needed) are sufficiently effective while base conditions remain protective, a ‘successful muddle through’ could result, with continued local outbreaks and a growing death toll, but dampened waves of both the virus and any further macroeconomic costs. If MNPI and voluntary behavior are too weak in containing the virus, however – a critical empirical question – then more extreme outcomes of the virus and various socioeconomic effects may occur. Hopes for a ‘muddle through’ in the spring and summer of 2008 in the U.S. were followed by a panic-shift of the financial crisis in September. Bipartisan U.S. macroeconomic stimulus packages passed with extraordinary speed during the national pandemic ‘shut down’ phase in mid-March through early April. The recent-decade history of political polarization and dysfunction in U.S. policymaking, however, has led to high planning uncertainty and social costs (Straka & Straka 2020). U.S. pandemic policies seem likely to continue to reflect the national election year, which may increase the reliance on MNPI and voluntary behavior.

**Fig. 1. Estimated Incubation and Serial Interval Days: SARS-Cov-2 vs. Flu**



Source: Reported Statistical Estimates and WHO (see references).

**Fig. 2. Estimates of  $R_0$ : SARS-Cov-2 vs. Seasonal Flu**



Source: Existing Statistical Estimates (see references).

**Table 1. SARS-Cov-2  $R_0$  and its Effect on Herd Immunity Threshold and Worst-Case Death Toll**

R0 examples	Herd Immunity Threshold	Approx Worst Case Tot US Deaths	Approx Worst Case Tot Global Deaths	Approx Worst Case Tot US Deaths	Approx Worst Case Tot Global Deaths	Approx Worst Case Tot US Deaths	Approx Worst Case Tot Global Deaths
	1 - 1/R0	1% Mortality rate	1% Mortality rate	0.75% Mortality rate	0.75% Mortality rate	0.5% Mortality rate	0.5% Mortality rate
5.7	82%	2,720,000	64,320,000	2,040,000	48,240,000	1,360,000	32,160,000
3.28	70%	2,290,000	54,220,000	<del>1,720,000</del>	<del>40,660,000</del>	1,150,000	27,110,000
2.79	64%	2,120,000	50,040,000	<del>1,590,000</del>	<del>37,530,000</del>	1,060,000	25,020,000
2.2	55%	1,800,000	42,550,000	<del>1,350,000</del>	<del>31,910,000</del>	900,000	21,270,000
R0 assumed	Speculative Alt. Herd Immunity	From two preprint studies (not peer reviewed). Under specific theoretical assumptions about highly susceptible or exposed groups becoming infected and immune first, preventative measures in place, and population connection heterogeneities, transmission may decline at lower threshold levels.					
2.5	43%	1,410,000	33,540,000	<del>1,060,000</del>	<del>25,160,000</del>	710,000	16,770,000
2.5 - 3.0	15%	490,000	11,700,000	370,000	8,780,000	250,000	5,850,000

Table 2. Variables in the Bivariate Probit Model and Potential Data Sources

<i>Endogenous Variable</i>			<i>Case Level Data Sources</i>	<i>Local-Area Data Sources</i>
Infected			Discussion in text	Johns Hopkins Coronavirus Resource Center, State Health Depts. <a href="https://coronavirus.jhu.edu/us-map">https://coronavirus.jhu.edu/us-map</a> <a href="https://github.com/CSSEGISandData/COVID-19">https://github.com/CSSEGISandData/COVID-19</a> <a href="https://usafacts.org/visualizations/coronavirus-covid-19-spread-map/">https://usafacts.org/visualizations/coronavirus-covid-19-spread-map/</a> cdc.gov/coronavirus/2019-ncov/cases-updates/cases-in-us.html
Tested			Discussion in text	Johns Hopkins Coronavirus Resource Center, State Health Depts Covidtracking.com at <i>The Atlantic</i> <a href="https://github.com/CSSEGISandData/COVID-19">https://github.com/CSSEGISandData/COVID-19</a> cdc.gov/coronavirus/2019-ncov/cases-updates/testing-in-us.html
<i>Explanatory Variable</i>	<i>Pr (Infected)</i>	<i>Pr (Tested)</i>	<i>Case Level Data Sources</i>	<i>Local-Area Data Sources</i>
Population Density	+	+	Area data	US Current Pop. Survey (CPS), <a href="https://usafacts.org/visualizations/coronavirus-covid-19-spread-map/">https://usafacts.org/visualizations/coronavirus-covid-19-spread-map/</a>
Dense Living Facilities	+	+	Discussion in text	American Housing Survey (AHS)
Dense Mass Transit	+	?	Area data	American Community Survey; APTA Fact Book, National Transit Database, www.bts.gov, CPS, AHS
Dense Work Conditions	+	+	Discussion in text	<a href="http://www.dol.gov">www.dol.gov</a> , American Working Conditions Survey, www.bls.gov
Medical	+	+	Discussion in text	<a href="http://www.bls.gov">www.bls.gov</a> , phpartners.org
First Responder	+	+	Discussion in text	www.bls.gov
Income Related <sup>a</sup>	+ / +	- / +	Discussion in text	CPS, USDA <a href="https://www.ers.usda.gov/data-products/county-level-data-sets/">https://www.ers.usda.gov/data-products/county-level-data-sets/</a>
Flu	?	?	Discussion in text	State Health Depts., CDC
Age: Older <sup>b</sup>	?	+	Discussion in text	CPS, AHS
Health Condition of Pre-existing Vulnerable <sup>b</sup>	?	+	Discussion in text	<a href="http://www.healthdata.org">www.healthdata.org</a> , countyhealthrankings.org, State govt. county health profiles (e.g. Health.Pa.gov), <a href="http://www.cdc.gov">www.cdc.gov</a> , ruralhealthinfo.org
Social Distance Practices	-	-	Area data	Aggregated anonymous geo-location data from digital devices (see e.g. Wladawsky-Berger 2020; Google; Unacast social distancing scoreboard); Berkley Interpersonal Contact Study (BICS; Feehan & Mahmud 2020); stay-at-home and business-closure measures (Courtemanche et al. 2020)
Recent Infectious Contacts	+	+	Individual exposures	Transit-in data, e.g. US International Air Passenger and Freight Stats., Airport proximity

## Notes to Table 2

- a Low income is assumed associated with more dense neighborhoods and closer living quarters and less access to testing (Borjas 2020). High Income is assumed associated with greater likelihood of prior domestic and overseas travel and greater access to testing.
- b These vulnerability factors increase the COVID-19 severity risk, but their effect on SARS-Cov-2 incidence likelihood is unclear, all else constant, especially with more self-quarantining conducted by those more vulnerable to severe COVID-19 illness.

**Figure 3. Local Population Components of SARS-Cov-2 Susceptible, Infected, and Recovered**

<b>Last Population Count or Estimate</b>		Recent In / Out Migration		Recent New Births
<b>Current Population not counting recent deaths</b>				
Asymptomatic Not Immune	Asymptomatic Antibody Immune (Recovered)	<i>SARSCov2 Suspected Never Hospitalized</i>		
Tested SARSCov2 Negative or Not Tested; Antibody Tested or Not Tested				
Known Infected Never Hospitalized	Known Infected Hospitalized, Released	Known Infected Hospitalized Still	Deceased from COVID-19	Deceased from Other Causes
Tested SARSCov2 Positive; Antibody Tested or Not Tested		Tested SARSCov2 Positive		Tested or Not Tested

## Appendix

Estimation of the recursive bivariate probit model with either synthetic assignment of 0's and 1's to represent local-area rates or fractional bivariate probit requires estimates of the local-area population. Figure 3 describes components of the Susceptible, Infectious, and Recovered groups within a local-area population (and the associated mixtures of testing status), beginning from the last known population count or estimate (e.g., from the 2019 CPS estimate for a specific county).

*Estimation of Area Population:* The relevant current population of any local area is equal to:

$$\text{CURR. POP.} = \text{Last Estimate} - \text{Out-Migration} - \text{Deceased|COVID-19, Other} + \text{In-Migration} + \text{Births}$$

The values of each of the variables on the right side above, except for Last Estimate, are dynamic and can change daily. For this reason, it seems generally reasonable to assume that Last Estimate provides a sufficiently close estimate of the Current Population, which implicitly assumes that  $\text{Out-Migration} + \text{Deceased} = \text{In-Migration} + \text{Births}$ . This is reasonable in general to simplify (although though more recent data on deaths and births, and migration should be used if available and relevant). If a local area with a large outbreak experienced sizable out-migration (fleeing the outbreak before expected travel restrictions) or deaths, and very little or no in-migration, this suggests the possible need for assessment and adjustment of the assumed area denominator.<sup>12</sup> The best denominator to use depends on the main purpose of the estimation, and the timing of out-migrations.<sup>13</sup>

The widely tracked and reported known infected cases in a local area include all those who tested positive for SARS-Cov-2, including the deaths from resulting COVID-19 (in addition to deaths many areas now also report still-active cases and other information). As depicted in Figure 3, most of this group are at varying stages of recovery or stages of becoming non-infectious (typically thought to be reached about 4 to 6 weeks after release or testing).<sup>14</sup>

*Addressing the Different Types of Testing:* Figure 3 also describes two types of SARS-Cov-2 testing, and this is relevant for the estimation of the selection equation for Pr (Tested). The number of

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<sup>12</sup> Journalists have documented this in the case of Wuhan, for example; it may have also occurred elsewhere as the growing severity of the virus and a metropolitan-area outbreak became clear.

<sup>13</sup> If the purpose pertains to the infection rate of the total exposed population, then using Last Estimate should be reasonably close to the total exposed; however, any diagnostic SARS-Cov-2 tests performed elsewhere on out-migrated individuals are censored from the observed area data. If the main purpose pertains to the current residing population of the area, it is more important to adjust the denominator to reflect materially relevant out-migration.

<sup>14</sup> Many of those infected may have lasting physical damages, making them the 'wounded' share of the infected population, which only future research may be able to estimate. Global experience has also shown that some recovered patients have become re-infected, for reasons that are unclear. The share of the infected population that does not become antibody immune is generally believed (or hoped) to be small, but not yet fully known.

available tests has continued to grow, with varying accuracy, although accuracy levels have generally improved. Most available kits are a diagnostic test (DT) for the presence of active virus. For simplicity, we can assume that most or all diagnostic testing performed as reported for a local area is reasonably similar in type and similarly allocated in a non-random, largely clinical diagnostic manner (and that any divergences from this are negligible or immaterial). Various states in the U.S. have provided more detailed information on the type of tests in use.<sup>15</sup>

Less available but statistically useful kits allow testing a population for the presence of antibodies. Those who previously had the virus typically develop the presence of antibodies. The antibody tests (AT) help to estimate how close a population may stand from the estimated ‘herd immunity’ threshold, for example, and as referenced in the text they can also allow more accurate estimation of the ultimate mortality rate. The AT types currently available have had more problems and may not be reliable (Vogel 2020), although accuracy has improved.<sup>16</sup> Most states in the U.S. are reporting any AT performed separately from the DT.

*Addressing Data Reporting Discrepancies:* Data reporting discrepancies need to be considered for relevance and possible adjustments if needed. Not all sources are reporting and tracking the incidence of SARS-Cov-2 infections and testing in the same way or with the same timeliness. For example, Meyer & Madrigal (2020) report data discrepancies between state health departments’ testing data and recently started CDC reports on the incidence of testing by state. Most states have reported confirmed test-positive cases as their total case count, with some discrepancies.<sup>17</sup> For a discussion of COVID-19 data reporting needs, see Pearce et al 2020. When comparing confirmed COVID-19 deaths with those from the flu, Faust (2020) has shown the difference in reporting of confirmed COVID-19 deaths versus confirmed flu deaths – many observers have not accounted for the CDC’s inclusion of total deaths from all ‘flu like’ illnesses (including pneumonia); actual confirmed annual flu deaths in the U.S. have been far lower than those from COVID-19 to date.

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<sup>15</sup> ‘Kits’ must include swabs and other materials needed for any test, and available trained personnel as well.

<sup>16</sup> New York State has reported a specificity of 97-100% for the AT used in its population testing. The CDC recently issued guidance on AT, warning of the need to interpret results properly in view of probabilities conditional on an area of high or low infections.

<sup>17</sup> Virginia, for example, also includes “probable cases” in its count of total cases.

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