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## Early COVID-19 pandemic modeling: Three compartmental model case studies from Texas, USA

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### Abstract

The novel coronavirus (SARS-CoV-2) emerged in late 2019 and spread globally in early 2020. Initial reports suggested the associated disease, COVID-19, produced rapid epidemic growth and caused high mortality. As the virus sparked local epidemics in new communities, health systems and policy makers were forced to make decisions with limited information about the spread of the disease. We developed a compartmental model to project COVID-19 healthcare demands that combined information regarding SARS-CoV-2 transmission dynamics from international reports with local COVID-19 hospital census data to support response efforts in three Metropolitan Statistical Areas (MSAs) in Texas, USA: Austin-Round Rock, Houston-The Woodlands-Sugar Land, and Beaumont-Port Arthur. Our model projects that strict stay-home orders and other social distancing measures could suppress the spread of the pandemic. Our capacity to provide rapid decision-support in response to emerging threats depends on access to data, validated modeling approaches, careful uncertainty quantification, and adequate computational resources.

### Keywords

SARS-CoV-2; compartmental models; decision support; uncertainty quantification

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## THE NOVEL CORONAVIRUS

SARS-CoV-2 emerged from Wuhan, China in late 2019 and sparked the Coronavirus Disease 2019 (COVID-19) pandemic as it spread worldwide in the early months of 2020. Early estimates of rapid growth with 3-day doubling times and high mortality rates painted a grim picture. Preliminary projections suggested that, without interventions, the healthcare infrastructure would be overwhelmed and COVID-19 mortality might exceed 2 million deaths in the United States alone. Public health messaging initially focused on “flattening the curve” to reduce healthcare burdens and buy time to ramp up surveillance and develop effective response strategies.

Policymakers turned to mathematical models of disease transmission to help with interpreting data, projecting healthcare demands and assessing mitigation measures. Over the last two decades, modeling has become a core tool for pandemic planning and decision support during emerging threats including the 2009 H1N1 flu pandemic, the 2015–2016 Ebola epidemic, and the 2015–2016 Zika virus pandemic [1]. Such models are designed to synthesize information on disease progression and transmission dynamics with local epidemiological data to provide situational awareness, project future spread, hospitalizations, and mortality, and evaluate possible interventions.

Here, we describe key challenges in modeling early COVID-19 spread and strategies for constructing data-driven models in the face of uncertainty to provide situational awareness and pandemic projections for three Metropolitan areas in Texas.

### COVID-19 data and uncertainty

As the COVID-19 pandemic emerged in US cities, there was great uncertainty regarding the transmission and severity of the virus. Two categories of data are required for robust modeling: (1) disease transmission and severity data, including the impact of behaviors that reduce transmission, and (2) surveillance data.

**Disease transmission and severity data.**—Pathogen natural history data describe mode of transmission, transmission rates, length of incubation or latent periods, timing and duration of symptom onset, and fatality rates. These data are typically aggregated from published studies, which are scarce for new pathogens. Given the speed in which COVID-19 was spreading, many groups chose to publish initial reports on *medRxiv* instead of waiting to disseminate following peer review.

Despite the quick response by the scientific community to share results and data, uncertainty in these key aspects of SARS-CoV-2 natural history remained. For example, early reports suggested individuals without symptoms were infectious. But it still remains unclear what fraction of infections are transmitted by these asymptomatic individuals. There is still debate on whether these cases would be more accurately described as subclinical, and how many asymptomatic cases are in fact pre-symptomatic and eventually progress to symptom onset [2], [3].

These uncertainties are very important for modeling efforts. Many models are designed to capture uncertainties in these estimates when enough research is available to derive estimates of parameter distributions. This is not always possible, and in many cases knowledge from similar pathogens is used to fill in the gaps. Many modeling groups, including our own, borrowed from a rich history of influenza modeling to make reasonable assumptions about aspects of SARS-CoV-2 natural history that were not yet fully described in the literature.

**Surveillance data.**—Disease surveillance systems typically track suspected and laboratory confirmed infection case counts and deaths. As SARS-CoV-2 spread, local, state and federal agencies realized that hospitals may approach their patient capacities, and rapidly developed additional surveillance systems to track hospital census [4].

Laboratory confirmation for SARS-CoV-2 infection required the development of novel diagnostics. Early delays in the manufacturing and distribution of testing kits hampered efforts to track the early spread of the virus. As testing ramped up, the demand caused backlogs in testing laboratories and exacerbated reporting delays. While many of these hurdles have been addressed, testing data remain an unreliable indicator of local transmission given the considerable spatiotemporal variability in testing availability and priorities and the high proportion of asymptomatic and mildly symptomatic cases [5]–[7].

Mortality data paint a more complete but time-lagged picture of severe COVID-19 prevalence. The average time to death from the date of hospitalization is 14 days [5]–[7]. Hospital census data—total number of patients hospitalized for COVID-19 related complications on a daily or weekly basis—provides an earlier indication of pandemic spread, given the estimated 5.9 day lag between symptom onset and hospitalization [5]–[7]. We expect the majority of severe COVID-19 cases will seek hospitalization, and have thus prioritized hospitalization data when estimating transmission rates for our models.

### Early COVID-19 models

There are many COVID-19 models that project cases, hospitalizations, and deaths, and they differ in their model structure, assumptions, and calibration. Early attempts to project the course of the pandemic relied on estimates from other respiratory viruses and from initial reports from China. For example, the widely cited projections of pandemic waves in the US and UK from Imperial College London used a previously developed individual-based influenza model parameterized with data from the Wuhan, China COVID-19 epidemic [3]. As US case and death count data became available, models including the high-profile dashboard produced by the Institute for Health Metrics and Evaluation (IHME) at the University of Washington calibrated projections based on local conditions [8]. Simultaneously, the pandemic research community developed a diverse portfolio of COVID-19 models that leveraged local, regional and national surveillance data as well as cell phone mobility data [9]. Many of these models now contribute to the national COVID-19 mortality forecasting ensemble, maintained by the Centers for Disease Control and Prevention [10].

### Three case studies in Texas, USA

Our research team responded to requests from policy makers in three Metropolitan Statistical Areas (MSAs) in Texas for data-informed projections of COVID-19 hospitalizations that were more locally relevant than the national and state level models available in the early epidemic. To meet these requests, the UT COVID-19 Modeling Consortium quickly adapted existing models for influenza virus epidemics to project healthcare needs for Austin-Round Rock MSA [5], Houston-The Woodlands-Sugar Land MSA [6], and Beaumont-Port Arthur MSA [7]. We summarize the findings of those three reports, with a focus on how differences in available data in these MSAs drove changes to modeling infrastructure and approach.

## METHODS

We used hospital census data (total count of in-hospital patients with confirmed COVID-19 diagnosis per day) to derive model-based estimates of key parameters governing the viral transmission rate in our models.

### Selection of Texas Metropolitan Statistical Areas

We selected Austin-Round Rock MSA, Houston-The Woodlands-Sugar Land MSA, and Beaumont-Port Arthur MSA based on availability of reliable COVID-19 hospital census data. Hereafter, we refer to these regions as Austin, Houston and Beaumont. Both Austin and Houston encompass large metropolitan areas with populations of 2.2 million and 7 million people, respectively. Beaumont has a smaller population of approximately 410,000 people. Austin has a lower estimated proportion of high-risk individuals than the other two MSAs [5]-[7].

### Data sources

We acquired hospital census data from stakeholders to inform our parameter estimation and projections:

- Dell Medical School at the University of Texas at Austin collected comprehensive daily COVID-19 hospital census data from hospitals in Austin starting March 13, 2020.
- The Southeast Texas Regional Advisory Council (SETRAC) provided COVID-19 hospital census data for Houston and Beaumont. Daily data were updated on a weekly cadence, beginning April 2, 2020.

### SARS-CoV-2 transmission model

We use a compartmental model based on SARS-CoV-2 transmission natural history to model transmission of the virus within each MSA, as described in detail in refs [5]–[7] (Figure 1). The model tracks the changing number of individuals in distinct disease compartments. Newly infected individuals move from susceptible (S) to exposed (i.e., infected but not yet symptomatic or infectious) (E), followed by an infectious period that could be symptomatic ( $I^Y$ ) or asymptomatic ( $I^A$ ). Asymptomatic individuals recover (R) without ever developing symptoms. Symptomatic individuals could either become

hospitalized ( $I^H$ ) or recovered (R). Hospitalized individuals then recover (R) or die (D). The population is divided into five age classes (0–4y, 5–17y, 18–49y, 50–65y, and +65y) and transmission is governed by age specific contact rates for home, school, work and other locations. The model assumes that there is no travel in or out of each MSA, given the COVID-19 stay-home orders at the time of analysis. The full list of parameters is provided in the Appendix and in our reports [5]–[7].

**Deterministic model.**—We used a deterministic implementation of our model for parameter estimation of the transmission rate, transmission rate reduction and start date. We also used the deterministic model to initialize simulations for the Houston and Beaumont reports as described in the following sections.

**Stochastic model.**—We used a stochastic implementation of our model to capture critical sources of uncertainty regarding the transmission dynamics and severity of the virus. Specifically, we draw random deviates from distributions rather than assuming fixed values for the following parameters: duration of the latent period, duration of the infectious period, relative infectiousness of asymptomatic cases, length of hospital stay for survivors, and length of hospital stay for non-survivors, as described in our reports [5]–[7]. We assume triangular distributions because they make minimal assumptions regarding the shape of the distributions while capturing a minimum, maximum, and mean value for the parameter.

In addition, the number of individuals transitioning from one compartment to the next at each time step is determined by a Poisson random variable to capture variability in the disease progression process.

### Model calibration and uncertainty estimation

We assumed that the initial transmission rate of SARS-CoV-2 prior to the implementation of social distancing measures ( $\beta_0$ ) was fixed and used hospital census data prior to any mandated interventions, if available, to estimate  $\beta_0$ . We further assume that shelter-in-place orders reduced the transmission rate by a fixed amount, and used hospital census data following implementation of mandated interventions to estimate this reduction ( $\kappa$ ). These parameters were estimated using non-linear least squares [5]–[7]. Briefly, for both  $\beta_0$  and  $\kappa$ , we searched the interval [0, 1] for values that minimized mean squared error in the observed hospital census data and the number of people in the hospitalized compartment of our model.

**Pre-Intervention data.**—We were able to estimate the transmission rate prior to intervention ( $\beta_0$ ) for Austin using hospital census data from March 13 to March 24, 2020. However, data from this early period were not available for Houston or Beaumont. Thus, we assumed that the baseline transmission rate would be the same for these MSAs as for Austin.

**Post-Intervention data.**—Stay-home orders were enacted on March 24, 2020 in Austin, March 27, 2020 in Houston and March 28, 2020 in Beaumont. For the Austin area, we fit our model to COVID-19 hospitalization data covering this entire range to estimate the reduction in transmission ( $\kappa$ ). In Houston and Beaumont, we fit our model to COVID-19 hospitalization data that were available beginning April 2.

**Uncertainty quantification.**—To indirectly estimate confidence intervals for  $\kappa$ , we ran 100 stochastic simulations for each possible value of  $\kappa$  between 0–100%, at 5% increments [5]–[7]. We then calculated the binomial probability that the 95% prediction interval for each simulation would contain the observed data. We report the 95% confidence interval for  $\kappa$  as the minimum and maximum values for which this binomial probability is greater than 0.05.

### Estimating the date of pandemic emergence

We estimated the date on which SARS-CoV-2 began spreading in the Austin area based on the date of the first reported COVID-19 hospital admission (March 13, 2020) and simple assumptions about the early transmission rate of the virus, prior to stay-home measures. Specifically, we assumed a three-day doubling time [5] and that approximately 4% of cases require hospitalization [5]–[7]. For the other two regions, we did not know the date of the first COVID-19 hospitalizations. Thus, we took an alternative approach in which we evaluated a range of possible start dates. For each date, we estimated  $\kappa$  using the non-linear least squares method mentioned above and selected the start date and  $\kappa$  combination that minimized the root mean squared error between the simulated and observed hospitalization counts.

### Hospitalization projections

We performed stochastic simulations using the estimated pandemic emergence date,  $\beta_0$ , and  $\kappa$  values to project the daily hospital census in each MSA. All simulations started with a single infected individual on the estimated start date of the local epidemic for the MSA under consideration. Transmission rate reduction began the day interventions took effect in each MSA: March 24, 2020 for Austin, March 27, 2020 for Houston, and March 28, 2020 for Beaumont.

### Filtering plausible simulations

Some stochastic simulations failed to result in epidemics. That is, the initial clusters of cases died out before producing a large-scale epidemic. Given that epidemics actually occurred, we filtered out such runs. For Austin, we excluded all simulations that had no cases on the date the local stay home order was enacted (March 24, 2020). For the Houston and Beaumont MSAs, we started each simulation using a deterministic implementation of our model, in which all parameters were fixed to their median values, and then switched to a stochastic model when ten daily incident cases were achieved.

### Computing resources

Parameter estimation (start date, transmission rate, transmission rate reduction) and stochastic simulations were conducted on the Frontera Supercomputer at the Texas Advanced Computing Center (TACC).

## RESULTS

We estimated healthcare demands for Austin, Houston, and Beaumont in the early stages of the COVID19 pandemic. We shared reports of these results with local officials and posted

them online [5]–[7]. Here, we review our projections and the challenges we faced while racing to provide time-sensitive situational awareness.

### Data availability and speed of analysis

We began our assessment of Austin on April 16th and delivered a completed report four days later (April 20), using COVID-19 hospitalization data from March 13th through April 16th. The Houston and Beaumont analyses required additional methods development since data were only available after April 2, likely several weeks after the pandemic emerged in these two areas. To address the missing early data, we assumed that the early transmission rate in Houston and Beaumont was identical to that estimated for Austin. These two reports took 8–9 days to complete.

### Estimation of epidemic emergence date and impact of stay-home orders

For Austin, we estimated initial transmission rate ( $\beta_0$ ) and impact of stay-home orders ( $\kappa$ ) using nonlinear least squares fitting. For Houston and Beaumont, we estimated the date of epidemic emergence and  $\kappa$  using a combination of nonlinear least squares fitting and a search across possible start dates. We estimated that COVID-19 began spreading in Austin, Houston, and Beaumont on February 15, February 10, and February 27, 2020, respectively. Furthermore, the local stay-home orders reduced COVID-19 transmission rates by 94% (95% CI: 55–100%) in Austin, by 95% (95% CI: 80–100%) in Houston, and by 85% (95% CI: 70–100%) in Beaumont (Figure 2). We used stochastic simulations to indirectly derive confidence intervals for  $\kappa$ , and applied two methods to restrict simulations to only those in which the epidemic eventually grew exponentially. In Austin, we filtered out all simulated outbreaks that died out after a few cases; in the other areas, we initialized our simulations using a deterministic model that did not allow for stochastic fade out. The first method produced larger confidence intervals than the second method (Figure 2).

### COVID-19 hospitalization projections

In our initial reports, we projected COVID-19 hospitalizations, ICU patients and ventilator demand through mid-August, 2020 [5]–[7]. The projections are initialized on the estimated emergence date of the pandemic. In Figure 3, we provide excerpts from those projections through May 15. Our 95% prediction intervals captured reported hospitalizations for Austin and Houston, where hospitalizations had seemingly begun to plateau before we generated our projections. Beaumont hospitalizations did not plateau until after our projections were made, and dipped below the lower 95% prediction bound shortly after the release of our report.

### Issue with uncertainty quantification

Our stochastic simulations of COVID-19 transmission in Austin produced two types of outcomes. Roughly 90% resulted in epidemic trajectories that mirrored the reported hospitalizations; the remaining 10% produced outbreaks that faded out before the epidemic began growing exponentially. This resulted in wide prediction intervals that do not fully convey the bimodal nature of the variation (Figure 3A). The narrower prediction intervals for Houston and Beaumont relative to Austin stem from a change in our methodology (Figures

3B and 3C). We employed a deterministic version of the model until there were at least ten daily incident cases and then switched to a stochastic version to ensure that all simulations progress to the point of wide community spread.

### Computational resource utilization

We utilized TACC's Frontera Supercomputer for all computations. Each model run – solving the system of equations defining the compartmental model for a number of time steps under a single set of parameters – was assigned to one of the 56 cores on a single Frontera Intel Xeon Platinum 8230 node. All runs were independent (trivially parallel) and were batched as single node jobs. We conducted 6,330 model runs (excluding development and debugging runs). A single deterministic fitting run was used to estimate  $\kappa$  for Austin, and 29 deterministic fitting runs were used to select the best  $\kappa$  and pandemic emergence date combinations for Houston (17 possible dates considered) and Beaumont (12 possible dates considered). A total of 6,300 stochastic simulations were performed with estimated parameters, of which 4,200 were hybrid deterministic and stochastic runs (2,100 each for Houston and Beaumont) and 2,100 were purely stochastic runs (Austin). Table 1 outlines the wall clock time per run and the total time needed to complete the results summarized in this paper.

## DISCUSSION

Our rapid development of models to provide COVID-19 situational awareness in Texas highlighted the importance of (1) data availability, (2) modeling infrastructure, (3) reliable uncertainty quantification, and (4) computational resources.

### Data availability.

The authors of this study include national experts in modeling the transmission dynamics of viruses who were tapped by federal, state and local policymakers and public health agencies to provide urgent analyses as the COVID-19 pandemic emerged across the US. Throughout the pandemic they have served on the Austin-wide COVID-19 leadership team, providing model-based projections and policy guidance for city leaders, all area healthcare systems and public health officials throughout the pandemic. Through these efforts, the research team has had unprecedented access to comprehensive daily COVID-19 hospitalization data starting from the first reported case on March 13, 2020.

Data for Houston and Beaumont were provided by the Southeast Texas Regional Advisory Council (SETRAC), which helps to coordinate emergency healthcare responses across the Texas Gulf Coast, an area that includes Houston and Beaumont and is prone to hurricanes and flooding. SETRAC quickly pivoted its data sharing infrastructure to collect and disseminate COVID-19 hospitalization data. However, comprehensive COVID-19 hospital reporting was not required until April 2020. Thus, earlier data for estimating baseline transmission rates in March 2020 were not available for these two regions.

Throughout the pandemic, our unique partnership with healthcare systems in Austin has provided critical data for forecasting the pandemic and providing actionable decision support [9], [11], [12]. Although our models are scalable to other cities and states, lack



of access to granular, standardized, and reliably reported COVID-19 hospitalization data has been a major impediment to such efforts.

### **Modeling infrastructure.**

We define modeling infrastructure broadly as a combination of expert knowledge, established methods and models (including software), and a data pipeline for model inputs. In March 2020, researchers at the University of Texas at Austin partnered with Texas Advanced Computing Center to establish the UT COVID-19 Modeling Consortium which brought these complementary needs under one roof. The consortium includes experts across diverse fields, including infectious disease modeling, optimization, statistics, social sciences and software development. Moreover, many of its members have a long track record of collaborating on translational research bringing epidemic science and data to the frontline of public health.

The rapid pivot from influenza modeling to COVID-19 modeling was made possible by key similarities between the two viruses. First, influenza and SARS-CoV-2 are spread by similar types of contacts and have similar risk factors [3]. This similarity allowed us to make use of the age-specific contact matrices and risk structures that we had previously developed for influenza modeling. Second, both viruses have latent periods and can be described by similar compartmental model frameworks. Given these similarities, we were able to adapt the influenza model for COVID-19 by altering the values of key parameters describing the disease progression and transmission dynamics of SARS-CoV-2. New data have since shown more differences between these two viruses, and we have been able to incorporate these updates quickly through software modifications.

### **Uncertainty quantification.**

Reliable uncertainty quantification is crucial to accurately communicating plausible outcomes to the public and to policy makers. We incorporate uncertainty directly with our stochastic simulations, where some parameters are drawn from distributions and where transitions between disease states have a random component. The result of these stochastic simulations is an array of possible epidemic trajectories that capture plausible outcomes for a given parameter set. We found that our methodology for initializing epidemic simulations produced too many unrealistic simulations and inflated our uncertainty in projecting hospitalizations, even after the most unrealistic simulations were removed. We updated our simulation initialization methodology to produce more realistic simulations, and we believe these results to better capture the realistic uncertainty in our model projections. In our full reports, we also explore multiple possible transmission rate reduction scenarios both inside and outside of our estimated confidence bounds to highlight the dependence of future trajectories on individual behavior and intervention policies [5]–[7].

### **Computational resources.**

We made extensive use of TACC's Frontera supercomputer for both model development and analysis. Once our workflow was optimized, we were able to produce the approximately 2,000 simulations needed for each report in under one hour by taking advantage of the highly parallel processing capabilities of Frontera CPU nodes. This resource coupled with

the real-time multifaceted support of TACC staff allowed us to complete the analysis and reports within four to nine days of data receipt. Computational resources were never a limiting factor.

In the months since the release of these three reports, pandemic policies, human behavior, and, consequently, the pandemic itself have evolved. We have updated our models continually to reflect our changing understanding of the situation, to make use of new data sources, and to support decision making at multiple scales, including the evaluation of stay-home orders, physical distancing recommendations, masking requirements, and testing priorities. Stemming from these early reports, we now maintain three dashboards that provide real-time COVID-19 situational awareness and short-term mortality and healthcare projections across Texas and the US [9]. Our collaborative consortium and access to the world-class computational resources at TACC has enabled and accelerated our ability to provide actionable models for the public and decision-makers.

## CONCLUSION

Access to expertise, data, modeling infrastructure, and computational resources has enabled rapid and high impact modeling in support of front-line COVID-19 response efforts. Academic groups such as the UT COVID-19 Modeling Consortium can rapidly expand access to expertise, modeling infrastructure and computational resources by leveraging collaborator networks and engaging with stakeholders. However, data access relies on external public health and clinical systems. These systems have been heavily stressed by the pandemic in ways that have impeded data collection and dissemination. While hospitalization data have proven to provide a robust signal for COVID-19 prediction models, these data are not consistently reported. This restricts the application of our most reliable prediction models to a select set of cities that report hospitalization data; localities without access to hospitalization data may have reduced situational awareness. Now in the tenth month since the emergence of SARS-CoV-2, reporting of more traditional surveillance data including confirmed case and mortality data access are still often inconsistent and missing key context for interpretation (e.g. testing policies and test sensitivity and specificity).

In addition to advocating for continued investment in the basic and applied science resources that enable the work of UT COVID-19 Modeling Consortium, we also advocate for investment in public health systems that facilitate disease surveillance and case reporting. These systems include decision making frameworks to guide resource allocation and data collection policy during public health crises, infrastructure for data dissemination (e.g. data repositories), and trained personnel who can collect, aggregate and disseminate not only surveillance data but the metadata that allow stakeholders to contextualize those data. Expanding these critical resources while solidifying connections between researchers, technologists, clinicians, emergency responders, and public health and governmental authorities will be essential to preventing and containing future pandemic threats.

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## APPENDIX

**Table A1.**

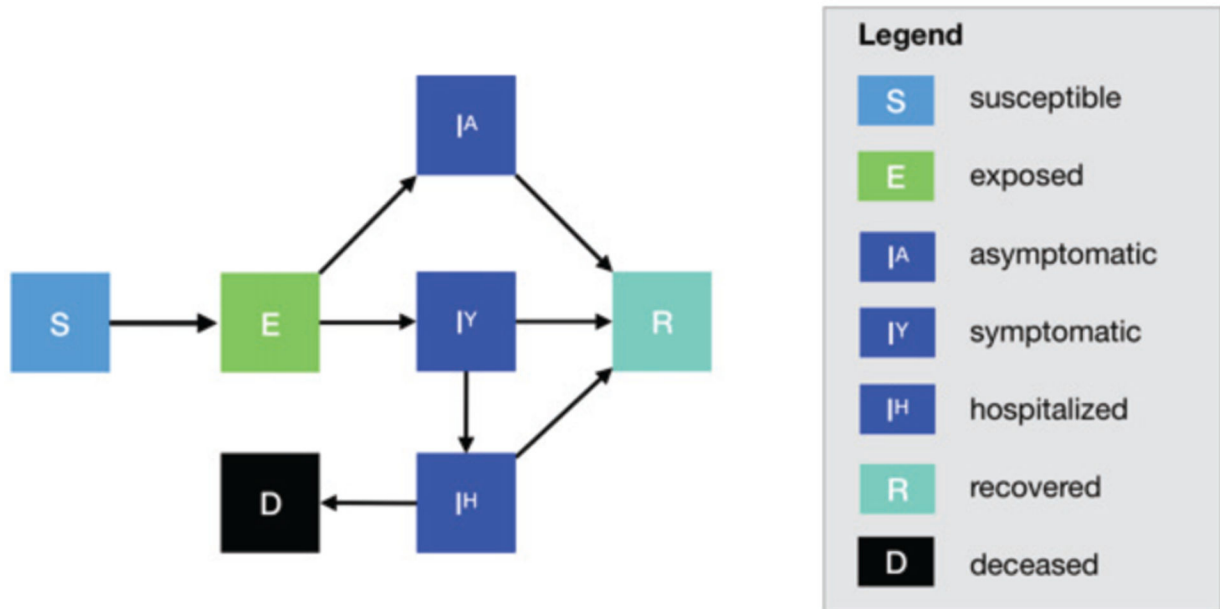
Model parameters

Symbol	Quantity	Value
$\beta_0$	transmission rate	0.035
$\frac{1}{\gamma^Y}$	symptomatic recovery rate	<i>Triangular(21.2, 22.6, 24.4)</i>
$\frac{1}{\gamma^A}$	asymptomatic recovery rate	<i>Triangular(21.2, 22.6, 24.4)</i>
$\frac{1}{\gamma^H}$	hospitalized recovery rate	1/14
$\tau$	symptomatic proportion	82.1
$\frac{1}{\sigma}$	exposure rate	<i>Triangular(5.6, 7, 8.2)</i>
$P$	proportion of pre-symptomatic transmission	12.6
$\omega^E$	relative infectiousness, exposed	$\frac{\left(\frac{YHR}{\eta} + \frac{1 - YHR}{\gamma^Y}\right)\omega^Y\sigma P}{1 - P}$
$\omega^A$	relative infectiousness, asymptomatic	0.47
$IFR_l$	low risk infection fatality ratio	[0.00092, 0.0022, 0.034, 0.25, 0.64]
$IFR_h$	high risk infection fatality ratio	[0.0092, 0.022, 0.34, 2.5, 6.4]
$YFR_l$	low risk symptomatic fatality ratio	[0.0011, 0.0027, 0.041, 0.31, 0.78]
$YFR_h$	high risk symptomatic fatality ratio	[0.011, 0.027, 0.41, 3.1, 7.8]
$YHR_l$	low risk hospitalization ratio	[0.028, 0.022, 1.3, 2.9, 3.4]
$YHR_H$	high risk hospitalization ratio	[0.28, 0.22, 13, 29, 34]
$HFR$	hospitalization fatality ratio	[4.0, 12, 3.1, 11, 23]
$h$	high-risk proportion, age specific	[8.2825, 14.1121, 16.5298, 32.9912, 47.0568]
$\eta$	symptom onset to hospitalization rate	0.1695
$\pi$	symptomatic hospitalization rate	$\frac{(\gamma^Y * YHR)}{\eta + (\gamma^Y - \eta)YHR}$
$\mu$	rate from hospitalization to death	1/14
$\nu$	hospitalization fatality rate	[0.039, 0.12, 0.030, 0.10, 0.23]
$ICU$	proportion hospitalized in ICU	[0.15, 0.20, 0.15, 0.20, 0.15]
$Vent$	proportion in ICU needing ventilation	0.67
$d_{ICU}$	duration of ICU stay (days)	10
$d_v$	duration of ventilation (days)	10

Values given as five-element vectors are age-stratified with values corresponding to 0–4, 5–17, 18–49, 50–64, 65+ year age groups, respectively. Adapted from references [5]–[7], with permission.

## REFERENCES

1. Rivers C et al. , “Using ‘outbreak science’ to strengthen the use of models during epidemics,” *Nat. Commun.*, vol. 10, no. 1, pp. 3102–3102, 2019, DOI: 10.1038/S41467-019-11067-2. [PubMed: 31308372]
2. Jiang X-L et al. , “Transmission Potential of Asymptomatic and Paucisymptomatic Severe Acute Respiratory Syndrome Coronavirus 2 Infections: A 3-Family Cluster Study in China,” *J. Infect. Dis.*, vol. 221, no. 12, pp. 1948–1952, Jun. 2020, DOI: 10.1093/infdis/jiaa206. [PubMed: 32319519]
3. Ferguson NM et al. , “Impact of non-pharmaceutical interventions (NPIs) to reduce COVID-19 mortality and healthcare demand,” *Imperial.Ac.Uk*, no. March, pp. 3–20, 2020, DOI: 10.25561/77482.
4. “COVID-19 Guidance for Hospital Reporting and FAQs For Hospitals, Hospital Laboratory, and Acute Care Facility Data Reporting. Updated October 6, 2020.” <https://www.hhs.gov/sites/default/files/covid-19-faqs-hospitals-hospital-laboratory-acute-care-facility-data-reporting.pdf>.
5. Wang X, Pasco R, Pierce K, Du Z, Fox S, and Meyers LA, “COVID-19 Healthcare Demand Projections: Austin, Texas.”
6. Pierce K et al., “COVID-19 Healthcare Demand Projections: Houston-The Woodlands-Sugar Land MSA, Texas.”
7. Pierce K et al., “COVID-19 Healthcare Demand Projections: Beaumont-Port Arthur MSA, Texas.”
8. IHME COVID-19 health service utilization forecasting team and C. J. Murray, “Forecasting the impact of the first wave of the COVID-19 pandemic on hospital demand and deaths for the USA and European Economic Area countries,” *medRxiv*, Apr. 2020, DOI: 10.1101/2020.04.21.20074732.
9. “UT Austin COVID-19 Modeling Consortium.” <https://covid-19.tacc.utexas.edu/> (accessed Oct. 23, 2020).
10. “COVID-19 Forecasts: Deaths | CDC.” [Online], <https://www.cdc.gov/coronavirus/2019-ncov/covid-data/forecasting-us.html> (accessed Sep. 09, 2020)
11. Duque D, Morton DP, Singh B, Du Z, Pasco R, and Meyers LA, “Timing social distancing to avert unmanageable COVID-19 hospital surges.,” *Proc. Natl. Acad. Sci. U. S. A.*, vol. 117, no. 33, pp. 1987319878, Aug. 2020, DOI: 10.1073/pnas.2009033117.
12. Wang X et al. , “Impact of Social Distancing Measures on Coronavirus Disease Healthcare Demand, Central Texas, USA,” *Emerg. Infect. Dis.*, vol. 26, no. 10, Oct. 2020, DOI: 10.3201/eid2610.201702.



**Figure 1. Compartmental model of COVID-19 transmission in a US city.**

Each age and risk subgroup is modeled with a separate set of compartments. Upon infection, susceptible individuals (S) progress to the exposed (E) compartment and then to either symptomatic infectious ( $I^Y$ ) or asymptomatic infectious ( $I^A$ ) compartments. All asymptomatic cases eventually progress to a recovered class where they remain protected from future infection (R); symptomatic cases are either hospitalized ( $I^H$ ) or recover. Mortality (D) varies by age group and risk group and is assumed to be preceded by hospitalization. Used, with permission, from [5]–[7].

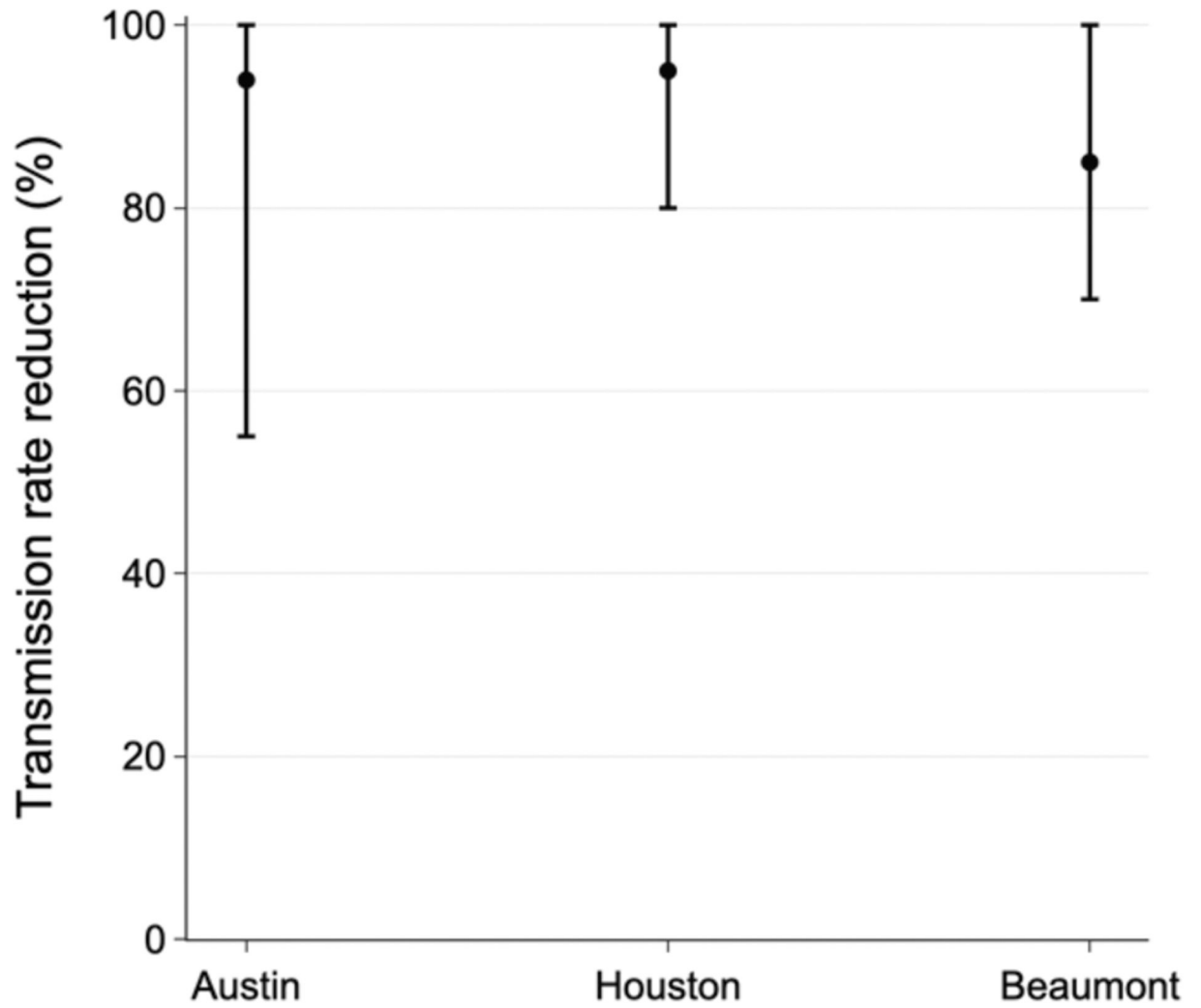
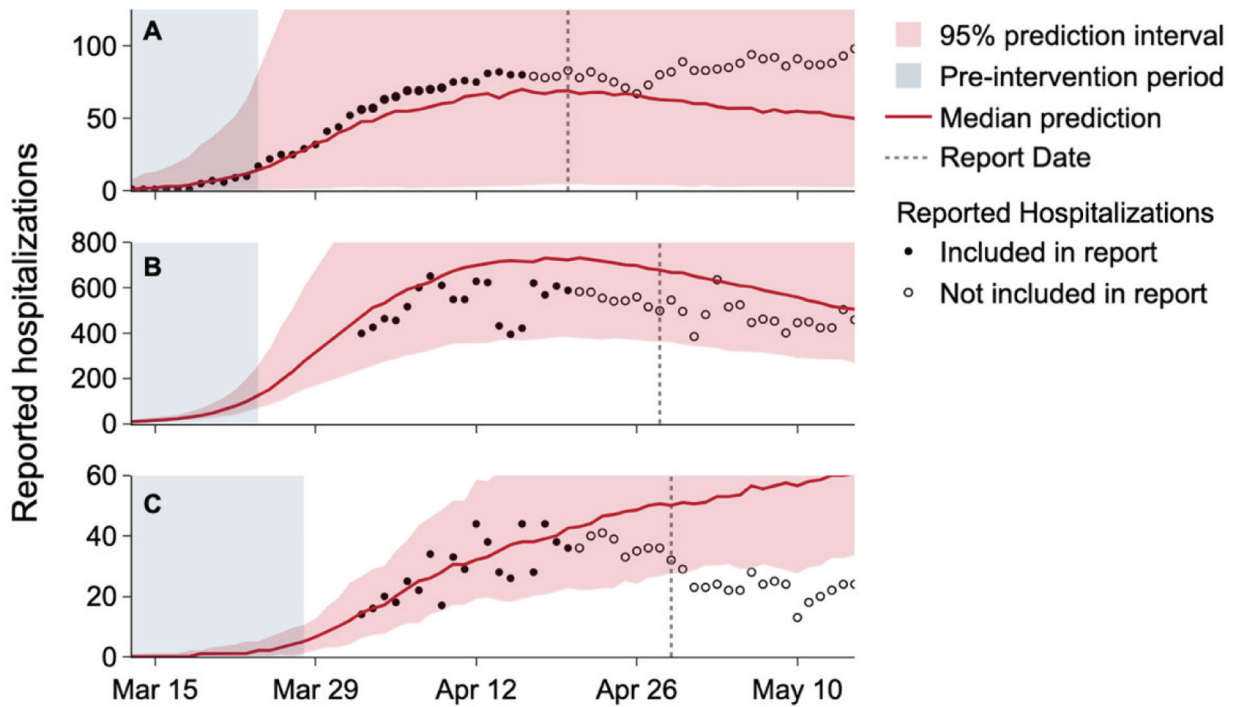


Figure 2. Estimated reduction in transmission rate during April 2020 stay-home orders ( $\kappa$ ) for the Austin, Houston and Beaumont areas.

Bars indicate 95% confidence intervals for our estimates of  $\kappa$ .



**Figure 3. Observed and projected COVID-19 hospitalization in the (A) Austin, (B) Houston, and (C) Beaumont areas from March 13-May 15, 2020.**

Filled points indicate reported hospital census data included in parameter estimation for our reports; empty circles indicate hospital census data reported after parameter estimation was complete; red lines and shading indicate median predictions and 95% prediction intervals; grey shading indicates the period prior to local stay-home orders; solid vertical lines mark the dates when reports were released.

**Table 1.**

Model run types and wall clock times

Run type	N runs	single core wall clock time (s) <sup>a</sup>	single node wall clock time (s) <sup>b</sup>
deterministic fitting	30	189	189
stochastic	2,100	6.31	240
hybrid	4,300	12.6	968
total	6,330	208	1,397

<sup>a</sup> Average single core wall clock time on Frontera Intel Xeon Platinum 8280 from 10 benchmark runs

<sup>b</sup> Average wall clock time multiplied by the number of 56 core Intel Xeon Platinum 8280 nodes required for the run volume specified

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