



Potential impacts of statewide relaxation of COVID-19 policies, the B.1.1.7 variant, and vaccination in Austin – March 2021

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Overview

To support public health decision-making and healthcare planning, we developed a model for the five-county Austin-Round Rock Metropolitan Statistical Area (henceforth *Austin*) that can provide real-time estimates of the prevalence and transmission rate of COVID-19 and project healthcare needs into the future.

The model incorporates key epidemiological characteristics of the disease, demographic information for Austin, and local mobility data from anonymized cell phone traces. It uses daily COVID-19 hospitalization data to estimate the changing transmission rate and prevalence of disease. The framework can be readily applied to provide pandemic situational awareness and short-term healthcare projections in other cities around the US.

In this report, we use COVID-19 hospitalization data for Austin from March 13, 2020 to March 5, 2021 to estimate the state of the pandemic in early March and project hospitalizations up to June of 2021. We consider the combined impact of the following factors:

- the March 10th statewide relaxation of nonpharmaceutical interventions [1]
- the emergence of the B.1.1.7 variant [2,3]
- the citywide rollout of the SARS-CoV-2 vaccine [4], and
- travel and mixing occurring during the K-12 and college spring break period

The projections are based on multiple assumptions about the age-specific severity of COVID-19 and the role of asymptomatic infections in the transmission of the virus. The graphs below do not present the full range of uncertainty for the city of Austin, but are intended to provide basic insight into the changing risks of COVID-19 transmission and potential healthcare surges in Austin.

Our results suggest that if transmission rates estimated on March 5th, 2021 persist and vaccines are administered at a rate of 35,000 per week (totalling ~51% of the MSA population fully vaccinated by June 1, 2021), COVID-19 hospitalizations will continue to decline, and there will be approximately 550 (95% CrI: 350-950) COVID-19 hospitalizations and 100 (95% CrI: 75-155) COVID-19 deaths between March 5 and June 1, 2021. However, the statewide lifting of NPI's, the emerging threat of the B.1.1.7 variant, and spring break activities could cause large COVID-19 surges. Specifically, we estimate the following for the three-month period between March 5 and June 1, 2021:

- On its own, the spread of the B.1.1.7 variant could increase the number of COVID-19 hospitalizations by 20% and deaths by 10%. In this scenario, the probability of exceeding an ICU capacity of 200 patients before June 1 is <1%.
- The combination of the statewide policy change and spring break could lead to triple the number of COVID-19 hospitalizations and double the number of COVID-19 deaths. This assumes that transmission rebounds similarly to the patterns observed in May 2020 following Phase 1 and 2 of Open Texas [5,6]. In this scenario, there is a 35% chance that hospitalizations will rise to the level of triggering Stage 5 and a 22% chance of exceeding the ICU capacity of 200 patients before June 1.
- The combination of the B.1.1.7 and behavioral relaxation could produce a large spring wave that threatens Austin's healthcare systems in the absence of intervention. In this scenario, there is a 54% chance that hospitalizations will rise to the level of triggering Stage 5 and a 47% chance of exceeding the ICU capacity of 200 patients before June 1.

We are posting these results prior to peer review to provide intuition for both policy makers and the public regarding both the immediate threat of COVID-19 and the importance of heightened social distancing and transmission reducing-precautions as vaccine distribution continues, including keeping physical distance from others, wearing cloth face coverings and self-isolating when symptomatic.

Austin COVID-19 model

The appendix below describes the model in detail. In short, we use mathematical equations to track the changing numbers of individuals who are susceptible (not yet infected), infected, hospitalized, recovered, and deceased. The model incorporates key features of the virus and uses iterated filtering [7] to estimate daily transmission rates in Austin from a combination of local hospital data (COVID-19 admissions, discharges and deaths) as well as SafeGraph mobility trends (cell phone-based estimates of hours spent at home and daily trips to public points-of-interest such as grocery stores, restaurants, bars and parks [8]). We use the estimated transmission rates to project COVID-19 hospitalizations, ICU visits, and deaths several months ahead. The model makes the following assumptions:

- Epidemic seeding: February 17th, 2020 with 1 infected adult
- Transmission rates are modulated by age-specific contact patterns
- Following infection, cases go through multiple stages of infection:
 - Stage 1: Pre-symptomatic and non-contagious for an average of 2.9 days
 - Stage 2: Pre-symptomatic contagious for an average of 2.3 days (44% of transmission events occur during this period)
 - Stage 3: Symptomatic contagious or asymptomatic contagious for an average of 4 days. The model assumes that 43% of all infections are asymptomatic and that asymptomatic cases are 67% as infectious as symptomatic cases.
- Cases may be hospitalized and/or die at rates that depend on their age and risk group.
 - The overall infection hospitalization rate (IHR) is 4.2%
 - The overall infection fatality rate (IFR) is 0.54%
- The duration of hospital stays are estimated from the local hospitalization data and can change through time.
- Vaccination is modeled by reducing susceptibility to infection of fully vaccinated individuals by 94% [9].

COVID-19 in Austin through March 5, 2021

We track COVID-19 spread in Austin through a metric called the effective reproduction number, $R(t)$. This indicates the contagiousness of the virus at a given point time and roughly corresponds to the average number of people a typical case will infect. Measures to slow or prevent transmission, such as social distancing and mask wearing, can reduce the reproduction number. Immunity acquired either through past infection or vaccination can also reduce the reproduction number. If $R(t)$ is greater than one, then an epidemic will continue to grow; if $R(t)$ is less than one, it will begin to subside. By tracking $R(t)$, we can detect whether policies and individual-level behaviors are having the desired impact and project cases, hospitalizations and deaths into the future.

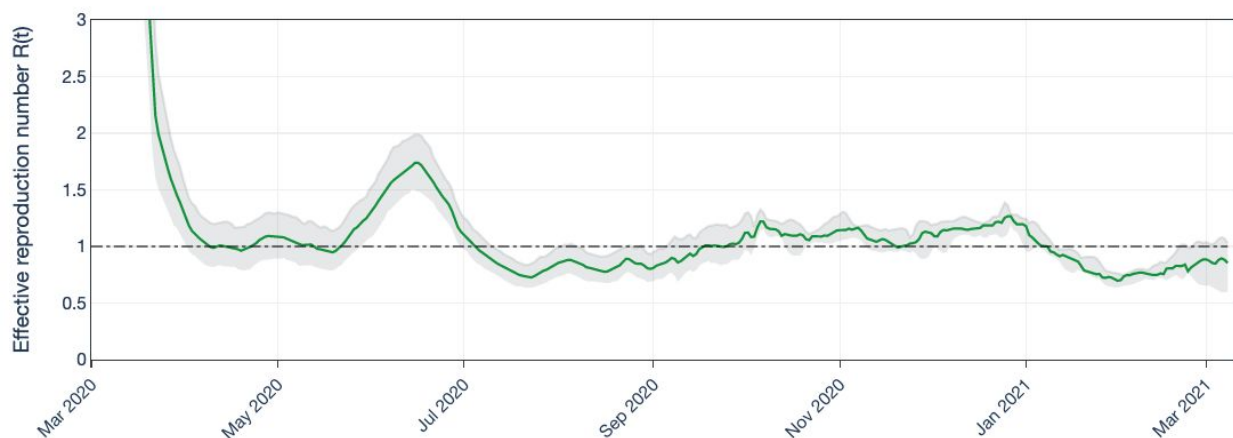


Figure 1: The 7-day average effective reproduction number, $R(t)$, of the COVID-19 pandemic in Austin from February 17, 2020 to March 9, 2021. $R(t)$ is an epidemiological quantity used to describe the contagiousness of a disease. An epidemic is expected to continue if $R(t)$ is greater than one and to end if $R(t)$ is less than one. This *epidemic threshold* of $R(t) = 1$ is indicated by a horizontal dashed line. $R(t)$ can be interpreted as the average number of people that an infected case will infect. The value of $R(t)$ depends on the basic infectiousness of the disease, the number of people that are susceptible to infection, and the impact of social distancing, mask wearing and other measures to slow transmission. The solid line gives the mean daily estimate and the shaded ribbon indicates the 95% credible interval.

To model the possible impacts of the recent statewide policy changes and increased mixing and travel during spring break, we estimate the impacts of similar events earlier in the pandemic. Specifically, the relaxation of strict COVID-19 measures led to an approximately 60% increase in the transmission rate on two occasions—immediately following the Phase 2 of Open Texas [5,6] in May and following the start of the school

year and state orders GA-30 and GA-31 in September [10] (Figure 1). We also estimate that, following Thanksgiving, transmission immediately spiked by roughly 30%. Thus, in the reopening + spring break scenarios below we assume that (1) transmission increases by 60% over the two weeks starting on March 6, 2021 and (2) spring break increases the transmission rate additional 30% for exactly one week starting on March 12, 2020.

COVID-19 healthcare projections for spring 2021 in Austin under four scenarios

We consider the following four scenarios:

- **Status quo:** The reproduction number of 0.73 (95%CrI 0.46-0.96) estimated on March 5 does not change.
- **Variant:**
 - We assume that the transmission rate of the B.1.1.7 variant is 60% higher than the original (wildtype) variant [3].
 - We assume that B.1.1.7 is spreading at 50% prevalence in Austin on March 5 and the proportion of cases caused by B.1.1.7 grows according to a logistic curve until it causes 99.7% of all infections by June 1 [2,3,11].
- **Reopening + Spring Break:**
 - To model the statewide relaxation of NPI's, we assume the transmission rate increases linearly between March 6 and March 20, reaching a rate that is 60% higher than estimated on March 5th.
 - To model spring break, we assume that the transmission rate increases by 30% between March 12 and 19.
- **Reopening + Spring Break + Variant:** We combine the two scenarios above.

For each scenario, we project COVID-19 hospitalizations, ICU patients, and deaths through June 1, assuming no other policy or behavioral changes during this period and that vaccinations continue in Austin at a rate of 35,000 per week (Figures 2-4). We also estimate the probability that Austin will reach the threshold for entering [Stage 5](#) (7-day rolling average of COVID-19 hospital admissions above 50) or surpass the estimated COVID-19 ICU capacity of 200 patients for the Austin-Round Rock MSA (Table 1).

Table 1: Projected impact of statewide policy change, spring break and the B.1.1.7 variant on pandemic burden in the Austin-Round Rock MSA from March 6 to June 1, 2021. Numbers are median values with 80% prediction intervals in parenthesis.

Scenario	Total COVID-19 hospitalizations	Total COVID-19 mortality	Chance of triggering Stage 5	Chance of exceeding 200 ICU patients
Status quo	550 (350-950)	100 (75-155)	<1%	<1%
Variant	650 (350-1,300)	110 (80-190)	<1%	<1%
Reopening + spring break	1,750 (550-7,200)	240 (110-810)	35%	22%
Reopening + spring break + variant	3,550 (700-15,300)	415 (125-1,600)	54%	47%

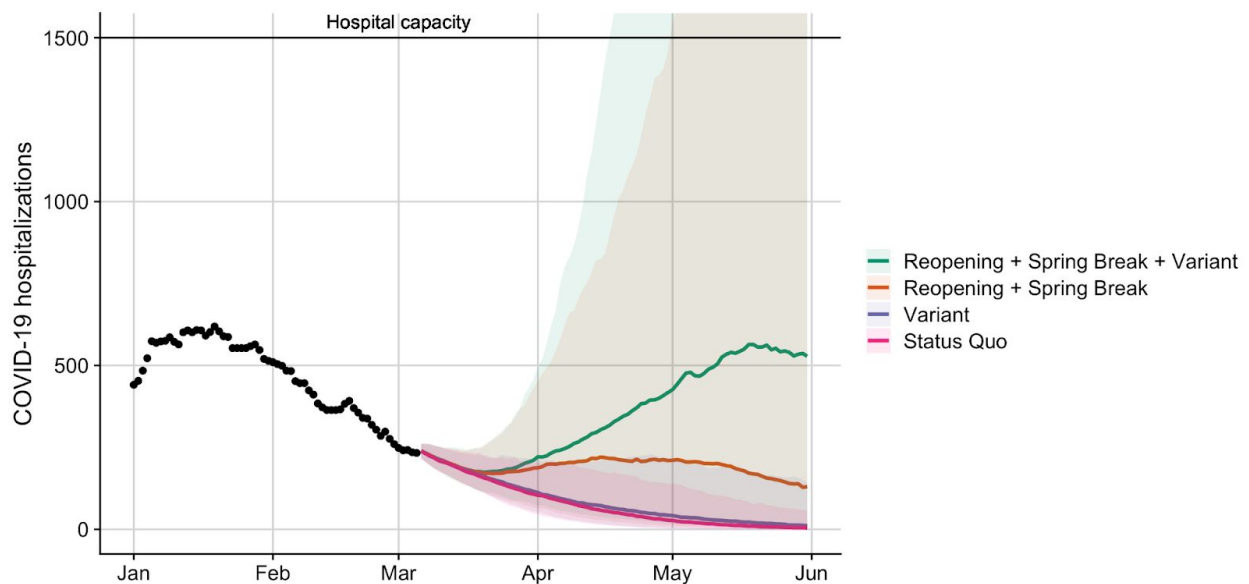


Figure 2: Projected COVID-19 hospitalizations in the Austin-Round Rock MSA from March 6 to June 1, 2021. Black points represent the reported daily COVID-19 patients in all Austin area hospitals. Colored lines represent median projections and shading indicates the 95% prediction interval for each scenario, across 500 stochastic simulations. The horizontal black line indicates the estimated COVID-19 hospital capacity of 1,500 patients in the MSA.

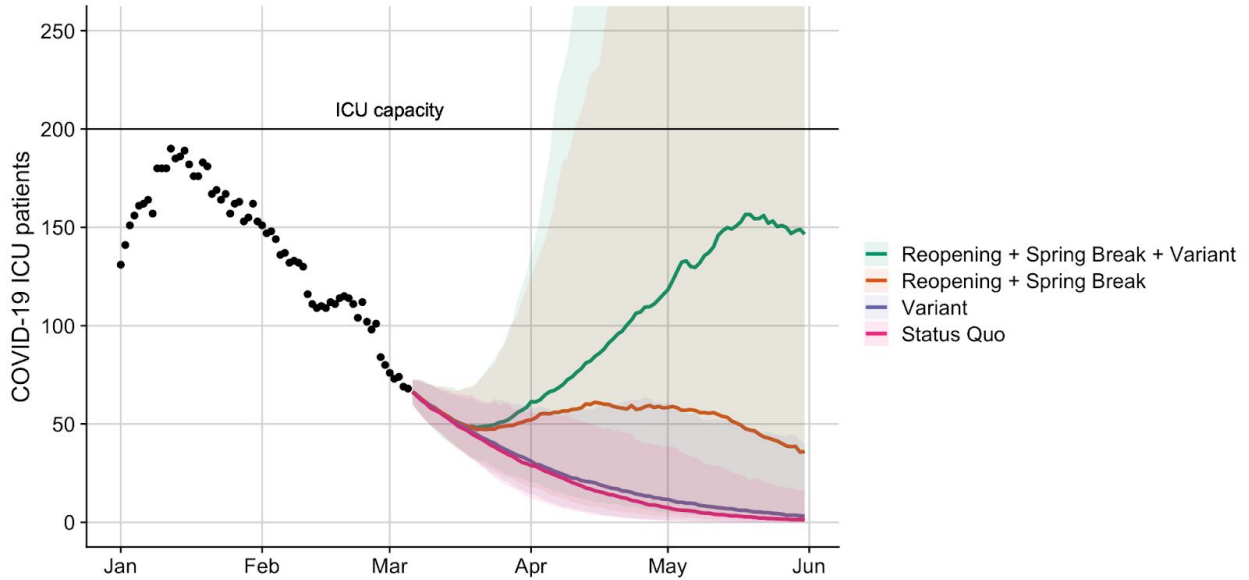


Figure 3: Projected COVID-19 ICU patients in the Austin-Round Rock MSA from March 6 to June 1, 2021. Black points represent the reported daily COVID-19 ICU patients in all Austin area hospitals. Colored lines represent median projections and shading indicates the 95% prediction interval for each scenario, across 500 stochastic simulations. The horizontal black line indicates the estimated ICU capacity of 200 COVID-19 patients in the MSA.

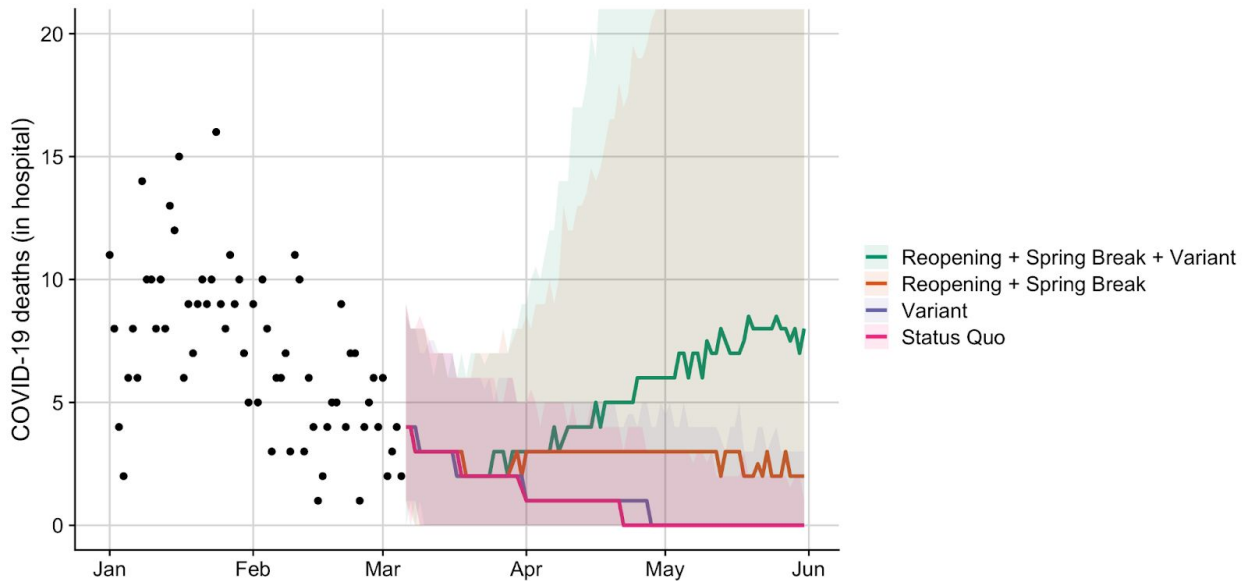


Figure 4: Projected daily COVID-19 hospital mortality in the Austin-Round Rock MSA from March 6 to June 1, 2021. Black points represent the daily number of COVID-19 deaths reported by all Austin area hospitals. COVID-19 deaths occurring outside of hospitals are not included in these projections. Colored lines represent median projections and shading indicates the 95% prediction interval for each scenario, across 500 stochastic simulations.

Appendix

COVID-19 Epidemic Model Structure and Parameters

The model structure is diagrammed in Figure A1 and described in the equations below. For each age and risk group, we build a separate set of compartments to model the transitions between the states: susceptible (S), exposed (E), pre-symptomatic infectious (P^Y), pre-asymptomatic infectious (P^A), symptomatic infectious (I^Y), asymptomatic infectious (I^A), symptomatic infectious that are hospitalized (I^H), recovered (R), and deceased (D). The symbols S, E, P^Y , P^A , I^Y , I^A , I^H , R, and D denote the number of people in that state in the given age/risk group and the total size of the age/risk group is

$$N = S + E + P^Y + P^A + I^Y + I^A + I^H + R + D.$$

The deterministic model for individuals in age group a and risk group r is given by:

$$\begin{aligned} \frac{dS_{a,r}}{dt} &= -S_{a,r} \cdot \sum_{i \in A} \sum_{j \in K} (I_{i,j}^Y \omega^Y + I_{i,j}^A \omega^A + P_{i,j}^Y \omega^{PY} + P_{i,j}^A \omega^{PA}) \beta(t) \phi_{a,i} / N_i \\ \frac{dE_{a,r}}{dt} &= S_{a,r} \cdot \sum_{i \in A} \sum_{j \in K} (I_{i,j}^Y \omega^Y + I_{i,j}^A \omega^A + P_{i,j}^Y \omega^{PY} + P_{i,j}^A \omega^{PA}) \beta(t) \phi_{a,i} / N_i - \sigma E_{a,r} \\ \frac{dP_{a,r}^A}{dt} &= (1 - \tau) \sigma E_{a,r} - \rho^A P_{a,r}^A \\ \frac{dP_{a,r}^Y}{dt} &= \tau \sigma E_{a,r} - \rho^Y P_{a,r}^Y \\ \frac{dI_{a,r}^A}{dt} &= \rho^A P_{a,r}^A - \gamma^A I_{a,r}^A \\ \frac{dI_{a,r}^Y}{dt} &= \rho^Y P_{a,r}^Y - (1 - \pi) \gamma^Y I_{a,r}^Y - \pi \eta I_{a,r}^Y \\ \frac{dI_{a,r}^H}{dt} &= \pi \eta I_{a,r}^Y - (1 - \nu) \gamma^H(t) I_{a,r}^H - \nu \mu(t) I_{a,r}^H \\ \frac{dR_{a,r}}{dt} &= \gamma^A I_{a,r}^A + (1 - \pi) \gamma^Y I_{a,r}^Y + (1 - \nu) \gamma^H(t) I_{a,r}^H \\ \frac{dD_{a,r}}{dt} &= \nu \mu(t) I_{a,r}^H \end{aligned}$$

where A and K are all possible age and risk groups, $\omega^A, \omega^Y, \omega^{PA}, \omega^{PY}$ are the relative infectiousness of the I^A, I^Y, I^{PA}, I^{PY} compartments, respectively, β is transmission rate, $\phi_{a,i}$ is the mixing rate between age group $a, i \in A$, and $\gamma^A, \gamma^Y, \gamma^H(t)$ are the recovery rates for the

I^A , I^Y , I^H compartments, respectively, σ is the exposed rate, ρ^A, ρ^Y are the pre-(a)symptomatic rates, τ is the symptomatic ratio, π is the proportion of symptomatic individuals requiring hospitalization, η is rate at which hospitalized cases enter the hospital following symptom onset, ν is mortality rate for hospitalized cases, and $\mu(t)$ is daily instantaneous rate at which terminal patients die.

We simulate the model using a stochastic implementation of the deterministic equations. Transitions between compartments are governed using the τ -leap method [12,13] with key parameters given in Table A1-2. We simulate the model according to the following equations:

$$\begin{aligned}
S_{a,r}(t+1) - S_{a,r}(t) &= -P_1 \\
E_{a,r}(t+1) - E_{a,r}(t) &= P_1 - P_2 \\
P_{a,r}^A(t+1) - P_{a,r}^A(t) &= (1 - \tau)P_2 - P_3 \\
P_{a,r}^Y(t+1) - P_{a,r}^Y(t) &= \tau P_2 - P_4 \\
I_{a,r}^A(t+1) - I_{a,r}^A(t) &= P_3 - P_5 \\
I_{a,r}^Y(t+1) - I_{a,r}^Y(t) &= P_4 - P_6 - P_7 \\
I_{a,r}^H(t+1) - I_{a,r}^H(t) &= P_7 - P_8 - P_9 \\
R_{a,r}(t+1) - R_{a,r}(t) &= P_5 + P_6 + P_8
\end{aligned}$$

with

$$\begin{aligned}
P_1 &\sim B(n = S_{a,r}(t), p = 1 - e^{-(F_{a,r}(t)) \cdot dt}) \\
P_2 &\sim B(n = E_{a,r}(t), p = 1 - e^{-(\sigma) \cdot dt}) \\
P_3 &\sim B(n = P_{a,r}^A(t), p = 1 - e^{-(\rho^A) \cdot dt}) \\
P_4 &\sim B(n = P_{a,r}^Y(t), p = 1 - e^{-(\rho^Y) \cdot dt}) \\
P_5 &\sim B(n = I_{a,r}^A(t), p = 1 - e^{-(\gamma^A) \cdot dt}) \\
P_6 &\sim B(n = I_{a,r}^Y(t), p = 1 - e^{-((1-\pi)\gamma^Y) \cdot dt}) \\
P_7 &\sim B(n = I_{a,r}^Y(t), p = 1 - e^{-(\pi\eta) \cdot dt}) \\
P_8 &\sim B(n = I_{a,r}^H, p = 1 - e^{-((1-\nu)\gamma^H(t)) \cdot dt}) \\
P_9 &\sim B(n = I_{a,r}^H(t), p = 1 - e^{-(\nu\mu(t)) \cdot dt})
\end{aligned}$$

where $B(n,p)$ denotes a binomial distribution with n trials each with probability of success p . $F_{a,r}^I$ denotes the force of infection for individuals in age group a and risk group r and is given by

$$F_{a,r}(t) = \sum_{i \in A} \sum_{j \in K} (I_{i,r}^Y(t)\omega^Y + I_{i,r}^A(t)\omega^A + P_{i,j}^Y(t)\omega^{PY} + P_{i,j}^A(t)\omega^{PA})\beta(t)\phi_{a,i}/N_i$$

with

$$\beta(t) = e^{\log(\beta(0)) + b_1(t) \cdot PC1 + b_2(t) \cdot PC2 + Z(t) + AZ(t)}$$

$$b_1(t) \sim N(b_1(t-1), \sigma_{b_1})$$

$$b_2(t) \sim N(b_2(t-1), \sigma_{b_2})$$

$$Z(t) \sim N(0.97 \cdot Z(t-1), \sigma_Z), Z(0) = 0.$$

where PC1 and PC2 describe the first and second principal components from our mobility data as described below. The adjustment $AZ(t)$ modifies $\beta(t)$ to model the impacts of reduced mask wearing and opening of businesses, spring break, rise of B.1.1.7 variant, and increased level of vaccination:

$$AZ(t) = A_m(t) + A_{sb}(t) + A_{B.1.1.7}(t) + A_v(t)$$

where $A_m(t)$ indicates the impact of the reopening and mask order, $A_{sb}(t)$ indicates the impact of spring break, $A_{B.1.1.7}(t)$ indicates the impact of the B.1.1.7 variant, and $A_v(t)$ indicates the impact of vaccine rollout.

Finally,

$$\mu(t) = e^{\log(\mu(0)) + Z_\mu} \text{ where } Z_\mu(t) \sim N(\psi_\mu \cdot Z_\mu(t-1), \sigma_\mu), Z_\mu(0) = 0 \text{ and}$$

$$\gamma^H(t) = e^{\log(\gamma^H(0)) + Z_\gamma} \text{ where } Z_\gamma(t) \sim N(0.99 \cdot Z_\gamma(t-1), \sigma_\gamma), Z_\gamma(0) = 0.$$

We estimate $\beta(t)$, k , σ_Z , $b_1(t)$, $b_2(t)$, σ_{b_1} , σ_{b_2} , ψ_μ , σ_μ , and σ_γ as described in the model fitting section below.

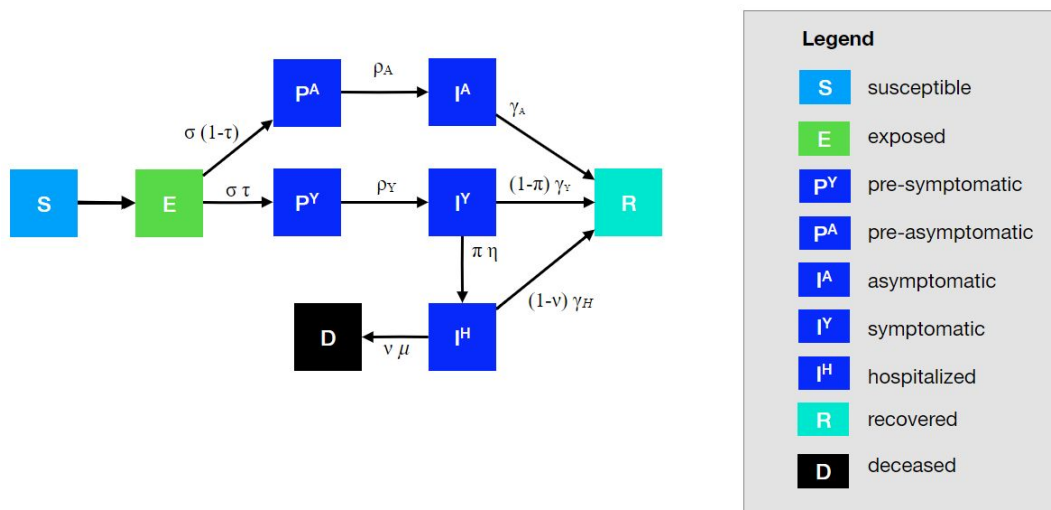


Figure A1. Compartmental model of COVID-19 transmission in the Austin MSA. Each subgroup (defined by age and risk) is modeled with a separate set of compartments. Upon infection, susceptible individuals (S) progress to exposed (E) and then to either pre-symptomatic infectious (P^Y) or pre-asymptomatic infectious (P^A) from which they move to symptomatic infectious (I^Y) and asymptomatic infectious (I^A) respectively. 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.

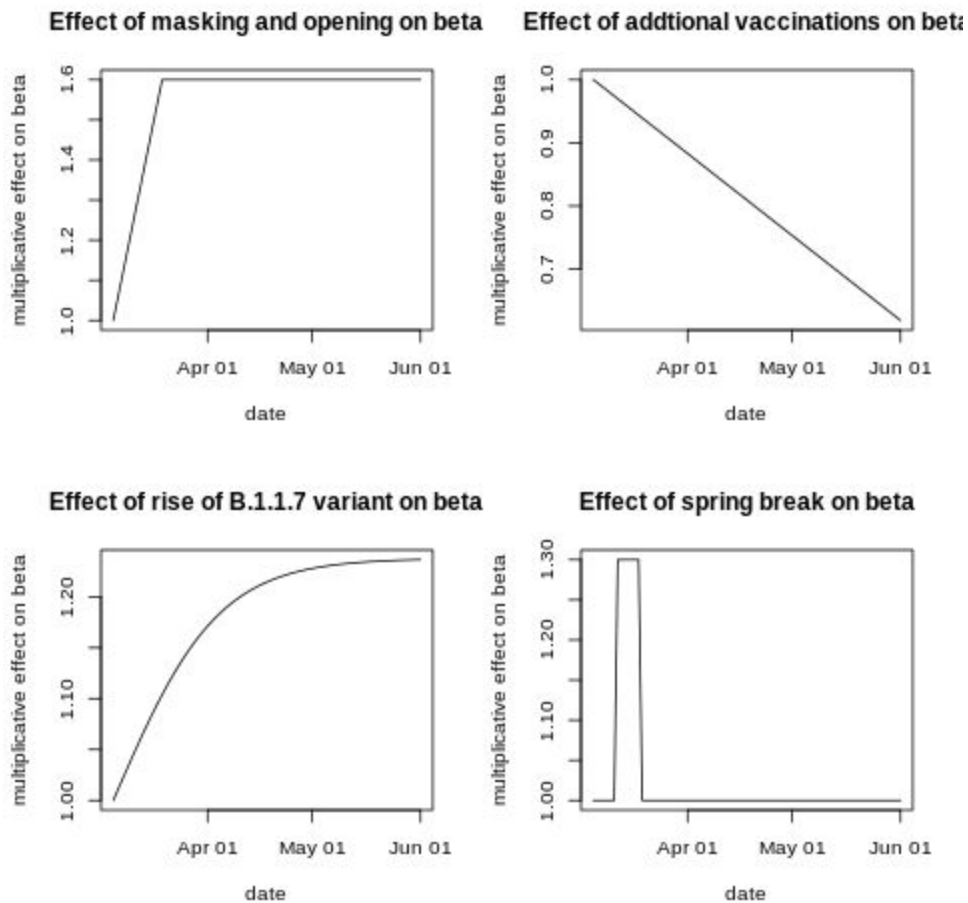


Figure A2. Components of the transmission modifier ($AZ(t)$) modeling changing risks in the Austin MSA. The four scenario projections adjust $\beta(t)$ to model the statewide reopening and removal of mask mandates, additional vaccinations, the rise of the B.1.1.7 variant, and spring break. The graphs show the multiplicative adjustment of $\beta(t)$ over time. The components of $AZ(t)$ are the logarithms of these values.

Mobility trends

We used mobility trends data from the Austin MSA to inform the transmission rate in our model. Specifically, we ran a principal component analysis (PCA) on seven independent mobility variables provided by SafeGraph, including home dwell time and visits to universities, bars, grocery stores, museums and parks, schools, and restaurants [8]. We regressed the transmission rate on the first two principal components from the mobility data as described in the modeling equations for $\beta(t)$.

Epidemic starting conditions

We could not estimate the epidemic start date directly using our model, because the transmission rate flexibility gave rise to similarly good fits within a wide-range of potential values for t_0 . We therefore conducted an independent estimation procedure to obtain reasonable epidemic start dates for Austin. We then used our best guess parameters as described in Table A2 and chose $\beta(0) = 0.67$ as it produced three-day doubling rate in cumulative cases and gave $R_t(0) = 4$ which are consistent with observations for the Austin early outbreak dynamics [14]. We ran 1,000 stochastic simulations with these initial conditions, and identified the wait time for when there was 1 admit for Austin. We estimated the start time from the resulting distribution of wait times for Austin as February 17, 2020 (IQR = February 11 - February 23), and chose February 17th, 2020 as the start date for the model.

Model likelihood

We obtained daily hospital admit ($H_A(t)$), discharge data ($H_L(t)$), total hospitalizations ($H(t)$), and death data ($H_D(t)$) for the Austin MSA. In this model we estimated $\beta(t)$, k , σ_Z , $b_1(t)$, $b_2(t)$, σ_{b_1} , σ_{b_2} , ψ_μ , σ_μ , σ_γ and fixed the remaining parameters as described in Table A1-2. We assumed all sources of data were negative binomially distributed around their predicted values from the SEIR stochastic model, and chose informative, but relatively dispersed priors for certain parameters for stability in parameter estimation and to prevent the model from overfitting data through large perturbations to time-dependent variables.

Following all of these considerations, the likelihood for our stochastic model was:

$$p(Y(t), b_1(0), \sigma_{b_1}, b_2(0), \sigma_{b_2}, k | \theta) = p(Y(t) | \theta, b_1(0), \sigma_{b_1}, b_2(0), \sigma_{b_2}, k) \cdot p(\theta, b_1(0), \sigma_{b_1}, b_2(0), \sigma_{b_2}, k)$$

where $Y(t)$ refers to the four types of data from hospitals, θ contains all parameters from Table A1 not explicitly listed, and where

$$p(Y(t) | \theta, b_1(0), \sigma_{b_1}, b_2(0), \sigma_{b_2}, k) = p(H_A(t) | \hat{H}_A(t)) p(H_L(t) | \hat{H}_L(t)) p(H_D(t) | \hat{H}_D(t)) p(H(t) | \hat{H}(t))$$

$$p(\theta, b_1(0), \sigma_{b_1}, b_2(0), \sigma_{b_2}, k) = p(b_1(0)) \cdot p(\sigma_{b_1}) \cdot p(b_2(0)) \cdot p(\sigma_{b_2}) \cdot p(k)$$

with

$$p(H_A(t) | \hat{H}_A(t)) = \binom{k + H_A(t) - 1}{H_A(t)} \cdot p^k (1 - p)^{H_A(t)}, \text{ and } p = \frac{k}{k + \hat{H}_A(t)}$$

$$p(H_L(t) | \hat{H}_L(t)) = \binom{k + H_L(t) - 1}{H_L(t)} \cdot p^k (1 - p)^{H_L(t)}, \text{ and } p = \frac{k}{k + \hat{H}_L(t)}$$

$$p(H_D(t) | \hat{H}_D(t)) = \binom{k + H_D(t) - 1}{H_D(t)} \cdot p^k (1 - p)^{H_D(t)}, \text{ and } p = \frac{k}{k + \hat{H}_D(t)}$$

$$p(H(t)|\hat{H}(t)) = \binom{k + H(t) - 1}{H(t)} \cdot p^k (1 - p)^{H(t)}, \text{ and } p = \frac{k}{k + \hat{H}(t)}$$

$$p(b_1(0)) \cdot t_d = \frac{1}{\sqrt{2}} e^{-\frac{1}{2}(\hat{b}_1(0))^2}$$

$$p(b_2(0)) \cdot t_d = \frac{1}{\sqrt{2}} e^{-\frac{1}{2}(\hat{b}_2(0))^2}$$

$$p(\sigma_{b_1}) \cdot t_d = \frac{1}{\Gamma(1.1) \cdot \frac{1}{1.1}} \hat{\sigma}_{b_1}^{1.1-1} e^{-1.1 \cdot \hat{\sigma}_{b_1}}$$

$$p(\sigma_{b_2}) \cdot t_d = \frac{1}{\Gamma(1.1) \cdot \frac{1}{1.1}} \hat{\sigma}_{b_2}^{1.1-1} e^{-1.1 \cdot \hat{\sigma}_{b_2}}$$

$$p(k) \cdot t_d = e^{\hat{k}}$$

and t_d is the number of days in the fitting time period.

Fitting method

In this model we estimated $\beta(t)$, k , σ_Z , $b_1(t)$, $b_2(t)$, σ_{b_1} , σ_{b_2} , ψ_μ , σ_μ , σ_γ and fixed the remaining parameters as described in Table A1. Fitting was carried out using the iterated filtering algorithm made available through the `mif2` function in the `pomp` package in R [15,16]. This algorithm is a stochastic optimization procedure; it performs maximum likelihood estimation using a particle filter to provide a noisy estimate of the likelihood for a given combination of the parameters. For each parameter combination we ran 1,000 iterations of iterated filtering, each with 10,000 particles. We calculated smoothed posterior estimates for all of the states within the model through time (including $\beta(t)$ and other time-dependent parameters which are technically state variables in our model formulation, as it changes through time according to a stochastic process). We calculated these smoothed posteriors as follows:

1. We ran 1,000 independent particle filters at the MLE, each with 10,000 particles. For each run, l , of particle filtering, we kept track of the complete trajectory of each particle, as well as the filtered estimate of the likelihood, L_l .
2. For each of the 1,000 particle filtering runs, we randomly sampled a single complete particle trajectory, giving us 1,000 separate trajectories for all state variables.
3. We resampled from these trajectories with probabilities proportional to L_l to give a distribution of state trajectories

The result can be thought of as an empirical-Bayes posterior distribution: that is, a set of 1,000 smoothed posterior draws from all state variables, conditional on the maximum likelihood estimates for the model's free parameters. This smoothed posterior distribution is how we calculate means and credible intervals for $\beta(t)$ in addition to all other time-varying state variables.

Table A1. Model parameters^a

Parameters	Value	Source
Start date	February 17, 2020	Estimated
Initial infections	1 symptomatic case age 18-49y	Assumption
$\beta(t)$: daily transmission rate	N/A	Estimated
γ^A : recovery rate on asymptomatic compartment	Equal to γ^Y	Assumption
γ^Y : recovery rate on symptomatic non-treated compartment	0.25	He et al. [17]
τ : symptomatic proportion (%)	57	Fox et al. [18]
σ : exposed rate	1/2.9	Zhang et al. [19]; He et al. [17]
ρ^A : pre-asymptomatic rate	Equal to ρ^Y	
ρ^Y : pre-symptomatic rate	$\frac{1}{2.3}$	He et al. [17]
P : proportion of pre-symptomatic transmission	44%	He et al. [17]
ω^P : relative infectiousness of pre-symptomatic individuals	$\omega^P = \frac{P}{1-P} \frac{\tau \omega^Y [YHR/\eta + (1-YHR)/\gamma^Y] + (1-\tau)\omega^A/\rho^A}{\tau \omega^Y/\rho^Y + (1-\tau)\omega^A/\rho^A}$ $\omega^{PY} = \omega^P \omega^Y, \omega^{PA} = \omega^P \omega^A$	
ω^A : relative infectiousness of infectious individuals in compartment I ^A	$\frac{2}{3}$	He et al. [20]
IFR : infected fatality ratio, age specific (%)	Low risk: [0.0009, 0.0022, 0.0339, 0.2520, 0.6440] High risk: [0.0092, 0.0218, 0.3388, 2.5197, 6.4402]	Age adjusted from Verity et al. [21]
YFR : symptomatic fatality ratio, age specific (%)	Low risk: [0.001608, 0.003823, 0.05943, 0.4420, 1.130] High risk: [0.01608, 0.03823, 0.5943, 4.420, 11.30]	$YFR = \frac{IFR}{\tau}$

h : high-risk proportion, age specific (%)	[8.2825, 14.1121, 16.5298, 32.9912, 47.0568]	Estimated using 2015-2016 Behavioral Risk Factor Surveillance System (BRFSS) data with multilevel regression and poststratification using CDC's list of conditions that may increase the risk of serious complications from influenza [22–24]
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^aValues 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.

Table A2 Hospitalization parameters

Parameters	Value	Source
$\gamma^H(t)$: recovery rate in hospitalized compartment	Fitted	
YHR : symptomatic case hospitalization rate (%)	Low risk: [0.04021, 0.03091, 1.903, 4.114, 4.879] High risk: [0.4021, 0.3091, 19.03, 41.14, 48.79]	Age adjusted from Verity et al. [21]
π : rate of symptomatic individuals go to hospital, age-specific	$\pi = \frac{\gamma^Y * YHR}{\eta + (\gamma^Y - \eta)YHR}$	
η : rate from symptom onset to hospitalized	0.1695	5.9 day average from symptom onset to hospital admission Tindale et al. [25]
$\mu(t)$: rate from hospitalized to death	Fitted	
HFR : hospitalized fatality ratio, age specific (%)	[4, 12.365, 3.122, 10.745, 23.158]	$HFR = \frac{IFR}{\tau}$
ν : death rate on hospitalized individuals, age specific	$\nu = \frac{\gamma^H HFR}{\mu + (\gamma^H - \mu)HFR}$	
ICU : proportion hospitalized people in ICU	0.35	Estimated from Austin COVID-19 hospitalization data

Table A3 Contact matrix. Daily number contacts by age group on an average day.

	0-4y	5-17y	18-49y	50-64y	65y+
0-4y	1.88	2.02	4.01	0.79	0.28
5-17y	0.55	7.06	5.02	0.70	0.22
18-49y	0.37	2.19	8.72	1.45	0.21
50-64y	0.33	1.62	5.79	2.79	0.50
65y+	0.19	0.88	2.36	1.19	1.22

Estimation of age-stratified proportion of population at high-risk for COVID-19 complications

We estimate age-specific proportions of the population at high risk of complications from COVID-19 based on data for Austin, TX and Round-Rock, TX from the CDC’s 500 cities project (Figure A2) [26]. We assume that high risk conditions for COVID-19 are the same as those specified for influenza by the CDC [22]. The CDC’s 500 cities project provides city-specific estimates of prevalence for several of these conditions among adults [27]. The estimates were obtained from the 2015-2016 Behavioral Risk Factor Surveillance System (BRFSS) data using a small-area estimation methodology called multi-level regression and poststratification [23,24]. It links geocoded health surveys to high spatial resolution population demographic and socioeconomic data [24].

Estimating high-risk proportions for adults. To estimate the proportion of adults at high risk for complications, we use the CDC’s 500 cities data, as well as data on the prevalence of HIV/AIDS, obesity and pregnancy among adults (Table A6).

The CDC 500 cities dataset includes the prevalence of each condition on its own, rather than the prevalence of multiple conditions (e.g., dyads or triads). Thus, we use separate co-morbidity estimates to determine overlap. Reference about chronic conditions [28] gives US estimates for the proportion of the adult population with 0, 1 or 2+ chronic conditions, per age group. Using this and the 500 cities data we can estimate the proportion of the population p_{HR} in each age group in each city with at least one chronic condition listed in the CDC 500 cities data (Table A6) putting them at high-risk for flu complications.

HIV: We use the data from table 20a in CDC HIV surveillance report [29] to estimate the population in each risk group living with HIV in the US (last column, 2015 data). Assuming independence between HIV and other chronic conditions, we increase the proportion of the population at high-risk for influenza to account for individuals with HIV but no other underlying conditions.

Morbid obesity: A BMI over 40kg/m² indicates morbid obesity, and is considered high risk for influenza. The 500 Cities Project reports the prevalence of obese people in each city with BMI over 30kg/m² (not necessarily morbid obesity). We use the data from table 1 in Sturm and Hattori [30] to estimate the proportion of people with BMI>30 that actually have BMI>40 (across the US); we then apply this to the 500 Cities obesity data to estimate the proportion of people who are morbidly obese in each city. Table 1 of Morgan et al. [31] suggests that 51.2% of morbidly obese adults have at least one other high risk chronic condition, and update our high-risk population estimates accordingly to account for overlap.

Pregnancy: We separately estimate the number of pregnant women in each age group and each city, following the methodology in CDC reproductive health report [32]. We assume independence between any of the high-risk factors and pregnancy, and further assume that half the population are women.

Estimating high-risk proportions for children. Since the 500 Cities Project only reports data for adults 18 years and older, we take a different approach to estimating the proportion of children at high risk for severe influenza. The two most prevalent risk factors for children are asthma and obesity; we also account for childhood diabetes, HIV and cancer. From Miller et al. [33], we obtain national estimates of chronic conditions in children. For asthma, we assume that variation among cities will be similar for children and adults. Thus, we use the relative prevalences of asthma in adults to scale our estimates for children in each city. The prevalence of HIV and cancer in children are taken from CDC HIV surveillance report [29] and cancer research report [34], respectively.

We first estimate the proportion of children having either asthma, diabetes, cancer or HIV (assuming no overlap in these conditions). We estimate city-level morbid obesity in children using the estimated morbid obesity in adults multiplied by a national constant ratio for each age group estimated from Hales et al. [35], this ratio represents the prevalence in morbid obesity in children given the one observed in adults. From Morgan et al. [31], we estimate that 25% of morbidly obese children have another high-risk condition and adjust our final estimates accordingly.

Resulting estimates. We compare our estimates for the Austin-Round Rock Metropolitan Area to published national-level estimates [36] of the proportion of each age group with underlying high risk conditions (Table A6). The biggest difference is observed in older adults, with Austin having a lower proportion at risk for complications for COVID-19 than the national average; for 25-39 year olds the high risk proportion is slightly higher than the national average.

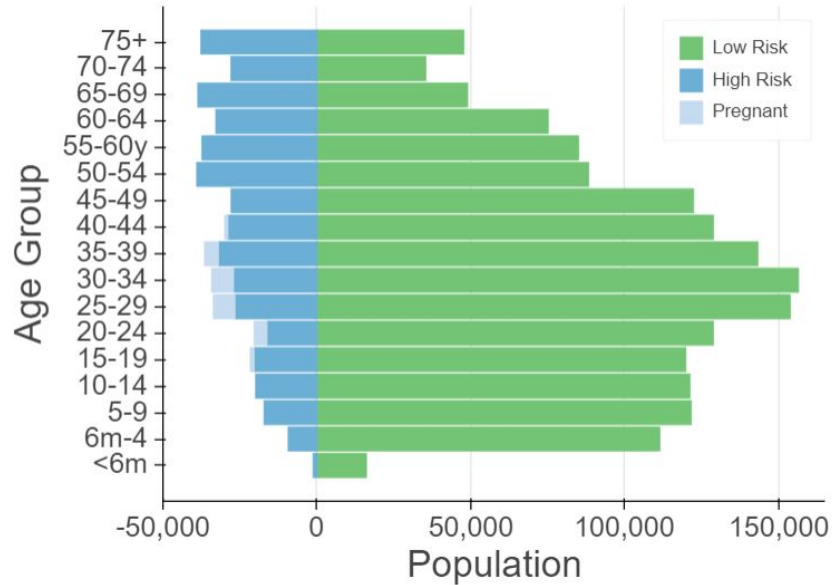


Figure A2. Demographic and risk composition of the Austin-Round Rock MSA. Bars indicate age-specific population sizes, separated by low risk, high risk, and pregnant. High risk is defined as individuals with cancer, chronic kidney disease, COPD, heart disease, stroke, asthma, diabetes, HIV/AIDS, and morbid obesity, as estimated from the CDC 500 Cities Project [26], reported HIV prevalence [29] and reported morbid obesity prevalence [30,31], corrected for multiple conditions. The population of pregnant women is derived using the CDC’s method combining fertility, abortion and fetal loss rates [37–39].

Table A4. High-risk conditions for influenza and data sources for prevalence estimation

Condition	Data source
Cancer (except skin), chronic kidney disease, COPD, coronary heart disease, stroke, asthma, diabetes	CDC 500 cities [26]
HIV/AIDS	CDC HIV Surveillance report [29]
Obesity	CDC 500 cities [26], Sturm and Hattori [30], Morgan et al. [31]
Pregnancy	National Vital Statistics Reports [37] and abortion data [38]

Table A5. Comparison between published national estimates and Austin-Round Rock MSA estimates of the percent of the population at high-risk of influenza/COVID-19 complications.

Age Group	National estimates [35]	Austin-Round Rock (excluding pregnancy)	Pregnant women (proportion of age group)
0 to 6 months	NA	8.1	-
6 months to 4 years	6.8	9.0	-
5 to 9 years	11.7	14.6	-
10 to 14 years	11.7	16.7	-
15 to 19 years	11.8	17.0	3.2
20 to 24 years	12.4	13.2	10.6
25 to 34 years	15.7	17.4	9.6
35 to 39 years	15.7	22.1	3.7
40 to 44 years	15.7	22.5	0.6
45 to 49 years	15.7	22.7	-
50 to 54 years	30.6	37.5	-
55 to 60 years	30.6	37.4	-
60 to 64 years	30.6	37.3	-
65 to 69 years	47.0	53.2	-
70 to 74 years	47.0	53.2	-
75 years and older	47.0	53.2	-

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