# Projections for COVID-19 omicron wave in Florida

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## Key findings

- Because there is substantial uncertainty in key properties of omicron infections, we consider four different scenarios in this report based on how infectious omicron SARS-CoV-2 is, how effective it is at evading acquired immunity, and how severe infections tend to be.
- We consistently find that the omicron wave in Florida is likely to cause many more infections than occurred during the delta wave.
- Preliminary data suggest that omicron infections may be less severe than those caused by delta. This means that despite causing more infections, it is possible that substantially fewer deaths will result from the omicron wave.
- Because detection of infections depends in part on whether symptoms are present (and their severity), the detected and reported size of the omicron wave may be similar to delta, or much larger.
- Across all scenarios, we consistently find that the omicron wave is likely to grow slowly through December 2021, rapidly through January 2022, and peak in February 2022.
- Preliminary data suggest that boosting may dramatically increase protection against disease caused by omicron infections [1]. It takes 10-14 days for protection to develop post-vaccination. Because relatively few Floridians have received booster doses at this point, we do not consider their effect in these results. Nonetheless, we recommend eligible people receive boosters, and we expect that an increase in booster uptake will result in more optimistic trajectories for the omicron wave in Florida.

## Introduction

The first known case of the omicron variant of concern (VOC) of SARS-CoV-2 in Florida was reported on December 7, 2021 [2]. First detected in southern Africa, the omicron variant has been associated with rapid increases in reported cases in southern Africa, Europe, and North America [3]. Although quantitative evidence exists regarding this variant's transmissibility, ability to evade acquired immunity, and severity, there is substantial uncertainty in exactly how severe omicron infections tend to be, and what the trade off is between inherent infectiousness and ability to evade acquired immunity [4]. Because of these parameter uncertainties, we consider four scenarios that span the range of likely epidemiological characteristics:

Scenario 1. Moderate transmission advantage, high immune escape; low severity

Scenario 2. Moderate transmission advantage, high immune escape; moderate severity

Scenario 3. High transmission advantage, moderate immune escape; low severity

Scenario 4. High transmission advantage, moderate immune escape; moderate severity

For these projections, we describe omicron's assumed properties relative to the previously dominant VOC in Florida, delta (Table 1). Increases in transmissibility for omicron are represented as a transmission advantage, calculated as the ratio of the basic reproduction numbers of omicron and delta (i.e., $R_0^o/R_0^\delta$). Omicron's immune escape capability is modeled as a reduction in the probability that existing immune protection, whether infection- or vaccine-derived, will prevent infection. For comparison, we assume that delta's immune escape probability is 15%. "Severity" is the probability that a person with symptoms will develop severe disease, a precondition we assume for hospitalization, ICU admission, or death. We consider two different omicron severity levels, 0.25 and 0.5 times that of delta.

| **Parameter** | **Lower estimate** | **Upper estimate** |
|---|---|---|
| Relative transmission advantage | 1.5 | 2.0 |
| Immune escape | 50% | 70% |
| Relative severity | 0.25 | 0.5 |

**Table 1:** Lower and upper omicron parameter assumptions.

Although it is not possible to know how individuals will respond in reaction to the spread of omicron, we expect the behavioral response to reflect prior trends. In our projections, we make the simplifying assumption that the change in personal protective behaviors will mirror the response that occurred during Florida's delta wave. In some of our scenarios, omicron causes more reported cases but fewer deaths than delta did. It is not clear whether such an outcome would result in a stronger or weaker individual-level response. As the state government has not indicated a plan to substantially change policies in response to omicron, we do not consider the possibility here.

Booster vaccine doses may substantially increase vaccine protection against disease caused by omicron [1]. At this time, 15.7% of Florida's population has received a booster dose [5]. In these projections, we do not include potential effects that boosting may have on omicron dynamics given the low booster prevalence at this time. Nonetheless, we recommend eligible people receive boosters, and we expect that an increase in booster uptake will result in more optimistic trajectories for the omicron wave in Florida.

## Results

The following multi-panel figures follow a consistent format (from top to bottom): simulated reported cases (yellow) are compared to empirical reported cases (black); simulated reported deaths (red) are compared to empirical reported deaths (black); simulated viral strain prevalence over time; total simulated infections (including asymptomatic infections and both reported and unreported cases); and time-varying reproduction number ($R_t$) measured from the simulation. For the forecasted period (December 2021 onward) in each panel, we show 100 realizations of the model, with the mean trajectory overlaid as a bold line.

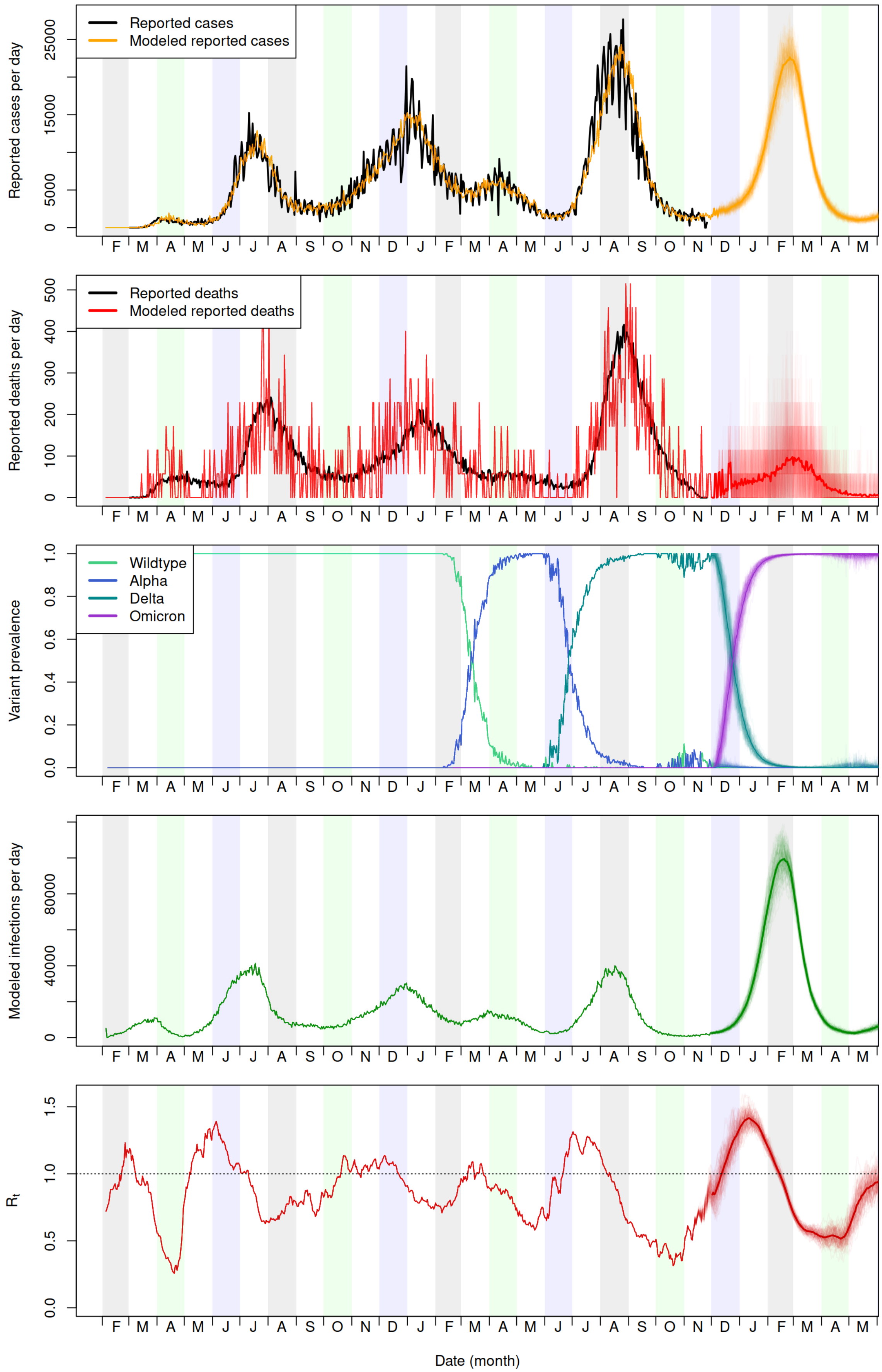


**Figure 1:** Omicron projection scenario 1: moderate transmission advantage, higher immune escape, low severity (see Table 1).

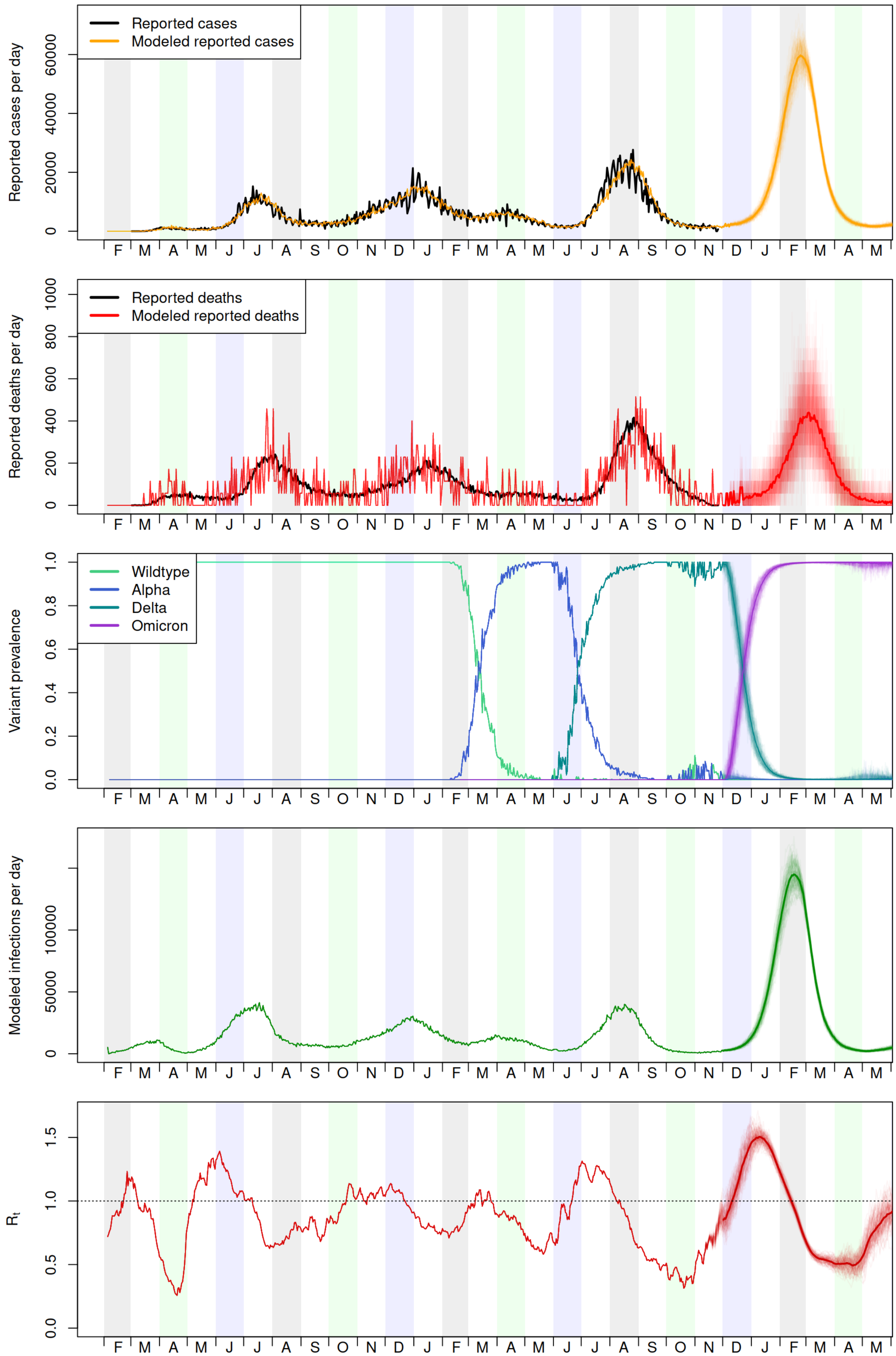


**Figure 2:** Omicron projection scenario 2: moderate transmission advantage, higher immune escape, moderate severity (see Table 1).

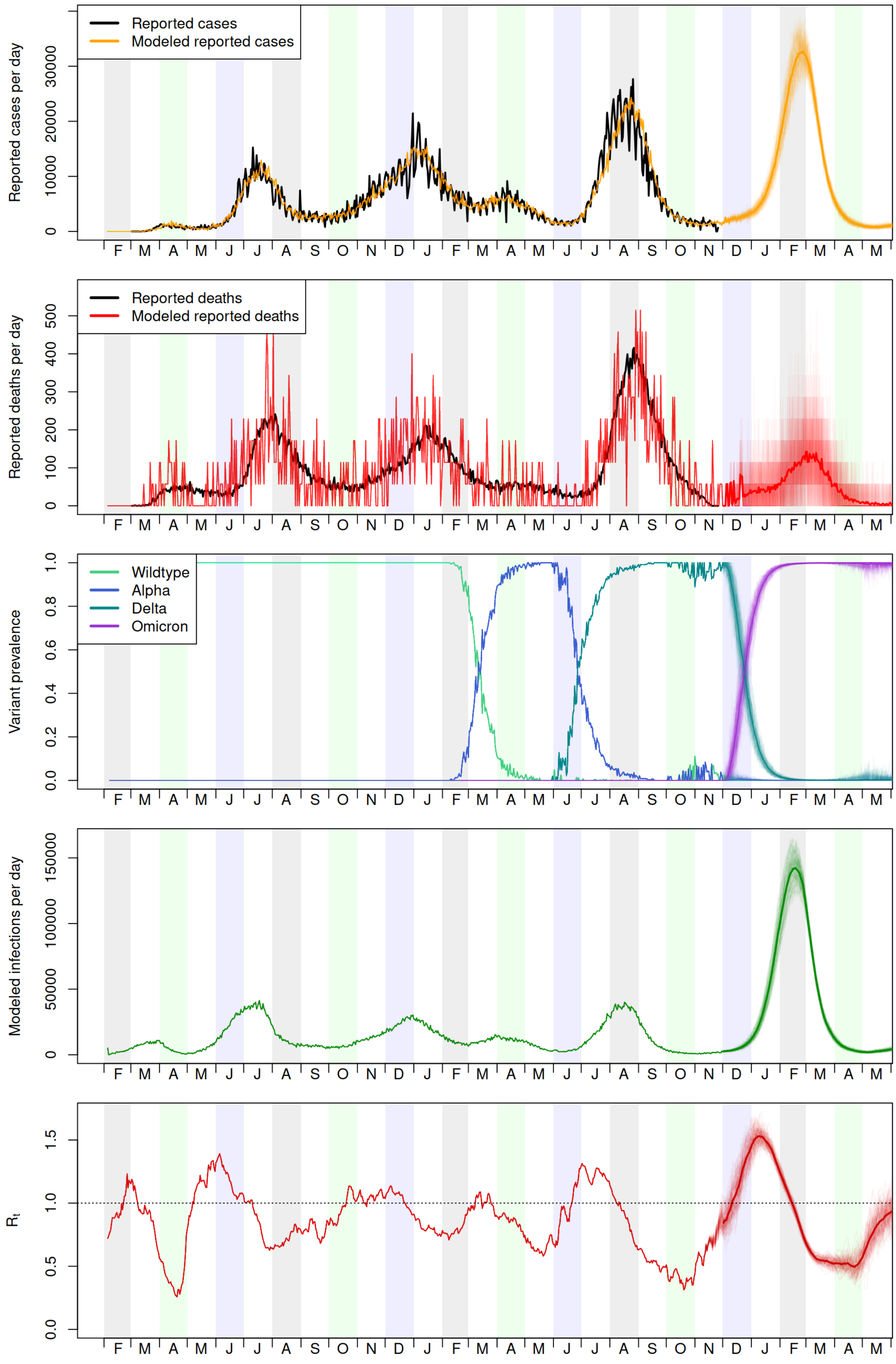


**Figure 3:** Omicron projection scenario 3: higher transmission advantage, moderate immune escape, low severity) (see Table 1).

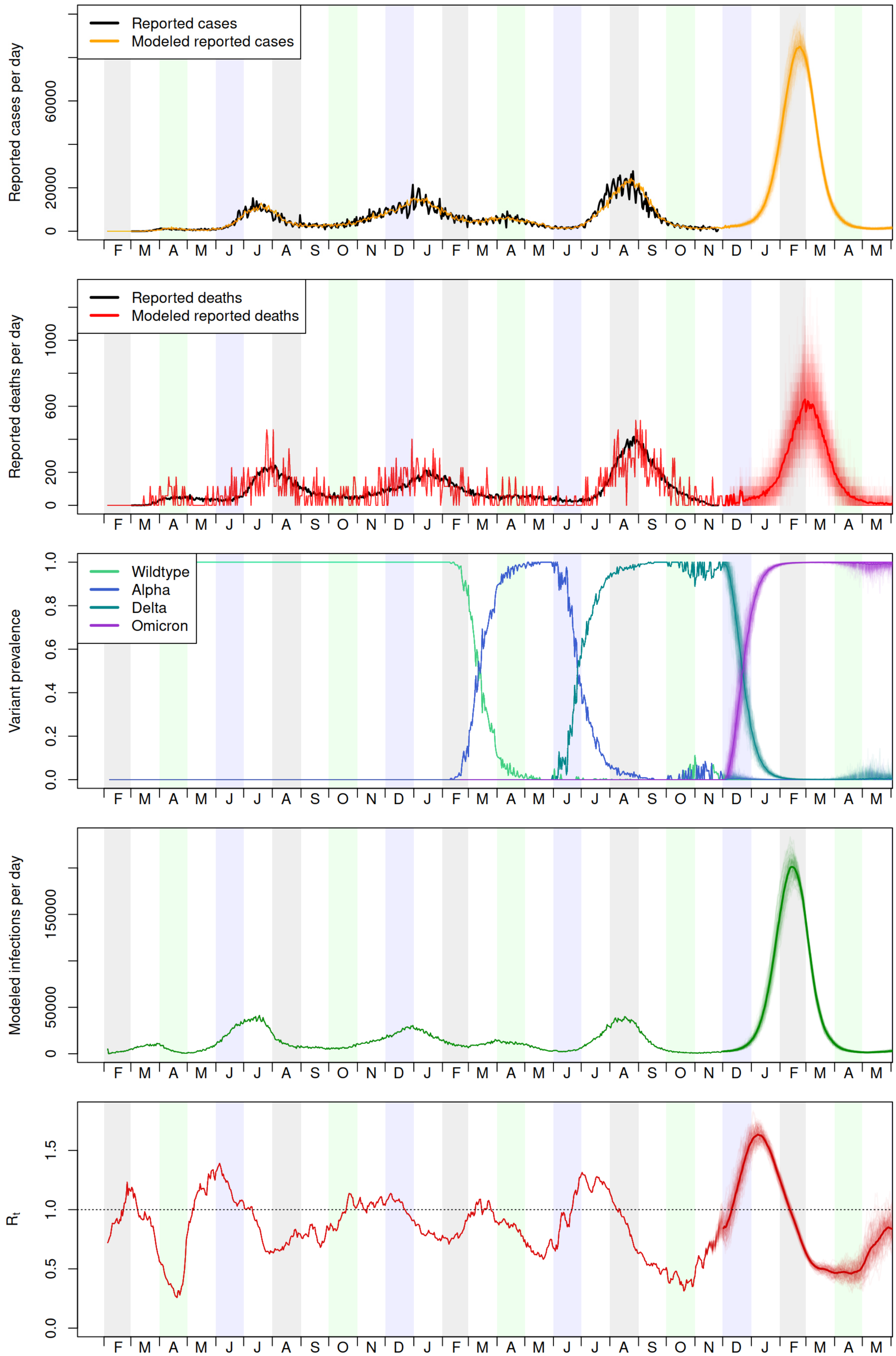


**Figure 4:** Omicron projection scenario 4: higher transmission advantage, moderate immune escape, moderate severity (see Table 1).

# Methods

We have developed a detailed simulation model to serve as a tool for assessing the COVID-19 epidemic in Florida. The model is a data-driven, stochastic, discrete-time, agent based model with an explicit representation of people and places [6]. Households in the model are sampled from census and survey data in order to establish a realistic distribution of age, sex, comorbidity, employment and school-attendance status. Activities and interaction patterns affect how likely someone is to be exposed in the model, and age, health status, and healthcare seeking behavior affect how severe a person's infection is likely to be. People go to work or school, visit friends, and patronize businesses in the model. The simulation includes closure of non-essential businesses, reduced school attendance, and changes in behaviors during the course of the pandemic. Our full Florida model represents 20.6 million people residing in 11.2 million households and 3.8 thousand long-term care facilities and who work in 2.3 million workplaces and attend 7.6 thousand schools. However, for this simulation study, we created a smaller, representative sample of the entire synthetic population totalling 375,000 people. We rescale the output from the model in order to estimate the cases and deaths for the entire state.

During each simulated day, infectious and susceptible individuals can aggregate in households, workplaces (both as employees and as customers), schools, long-term care facilities, and hospitals at different times in the day (Fig. 5). When susceptible and infectious people come together at the same location, there are new opportunities for the transmission of the virus.

If an individual becomes infected, the progression of the infection follows an $SEIRD$ model where people progress through susceptible ($S$), exposed ($E$), infected ($I$), recovered ($R$), and dead ($D$) states. Additionally, infected individuals can develop mild ($I_A$), severe ($I_M$), or critical ($I_C$) symptoms (Fig. 5. People who become ill can may seek healthcare, resulting in that individual receiving hospital care (for severe symptoms) or ICU care (for critical symptoms), which in turn lowers the risk of death.

Beyond non-pharmaceutical interventions (e.g. business or school closures, social distancing, stay-at-home orders), the model also represents vaccination of the synthetic population. In our model, we simulate a generalized mRNA vaccine (Table. 2) that performs similarly to the BioNTech and Moderna mRNA vaccines that have been used in Florida [7]. We simulate a rollout of vaccines that begins in January, 2021, with vaccine availability and campaign phases reflecting the vaccine rollout that has occurred in Florida (i.e. starting with healthcare workers and older members of the population and progressively widening eligibility to younger age groups).

Since our last report, we have revised our model of immunity to account for new data on immune dynamics and the effects of new variants. For vaccine-derived immunity, all people start with the same initial efficacy, whereas infections generate variable initial protection against reinfection. Both infection- and vaccine-derived immunity is modeled as leaky (in which every exposure has some chance of causing infection). We assume that efficacy against susceptibility ($VE_S$) does not inherently wane, but does decrease due to changes in the circulating variants [8]. Efficacy against pathology ($VE_P$) and against severe outcomes ($VE_H$) remain constant over time. To calculate the current protection an individual has against infection due to vaccination ($VE_S$) or infection ($IE_S$), we use Equation 1 where $E_S^i$ is the initial level of protection from either vaccination or infection, and $\Omega$ is the variant's immune escape probability. In Table 2, we document our modeled vaccine efficacy values given the assumption that delta is a 15% immune escape mutant. Similar calculations are performed to determine a simulation's $VE_S$ or $IE_S$ for omicron using immune escape assumptions.

$$E_S^{VOC} = E_S^i * (1 - \Omega) \tag{1}$$

|  | Wildtype |  | Alpha |  | Delta |  | Omicron |  |
|---|---|---|---|---|---|---|---|---|
|  | Dose 1 | Dose 2 | Dose 1 | Dose 2 | Dose 1 | Dose 2 | Dose 1 | Dose 2 |
| $VE_S$ | 0.4 | 0.8 | 0.4 | 0.8 | 0.34 | 0.68 | [0.12, 0.24] | [0.2,0.4] |
| $VE_P$ | 0.67 | 0.75 | 0.67 | 0.75 | 0.67 | 0.75 | 0.67 | 0.75 |
| $VE_H$ | 0.9 | 1.0 | 0.9 | 1.0 | 0.9 | 1.0 | 0.35 | 0.7 |
| $VE_I$ | 0.4 | 0.8 | 0.4 | 0.8 | 0.4 | 0.8 | 0.4 | 0.8 |

**Table 2:** Vaccine efficacy ($VE$) values assumed in our model, based on estimates from multiple Phase III trials and other published sources [7]. Delta $VE_S$ values assume 15% immune escape. Omicron $VE_S$ were calculated using Equation 1 with Wildtype as $VE_S^i$ and are reported above for the lower and upper immune escape assumptions in Table 1. Other $VE$ parameters for omicron will match those for other VOCs. $VE_S$ and $VE_P$ estimates have been revised since the October 08 report. Note on $VE$ details: $VE_S$ refers to vaccine efficacy against infection. $VE_P$ refers to vaccine efficacy against symptoms given infection. $VE_H$ refers to vaccine efficacy against severity given symptoms. $VE_I$ refers to vaccine efficacy against onward transmission.

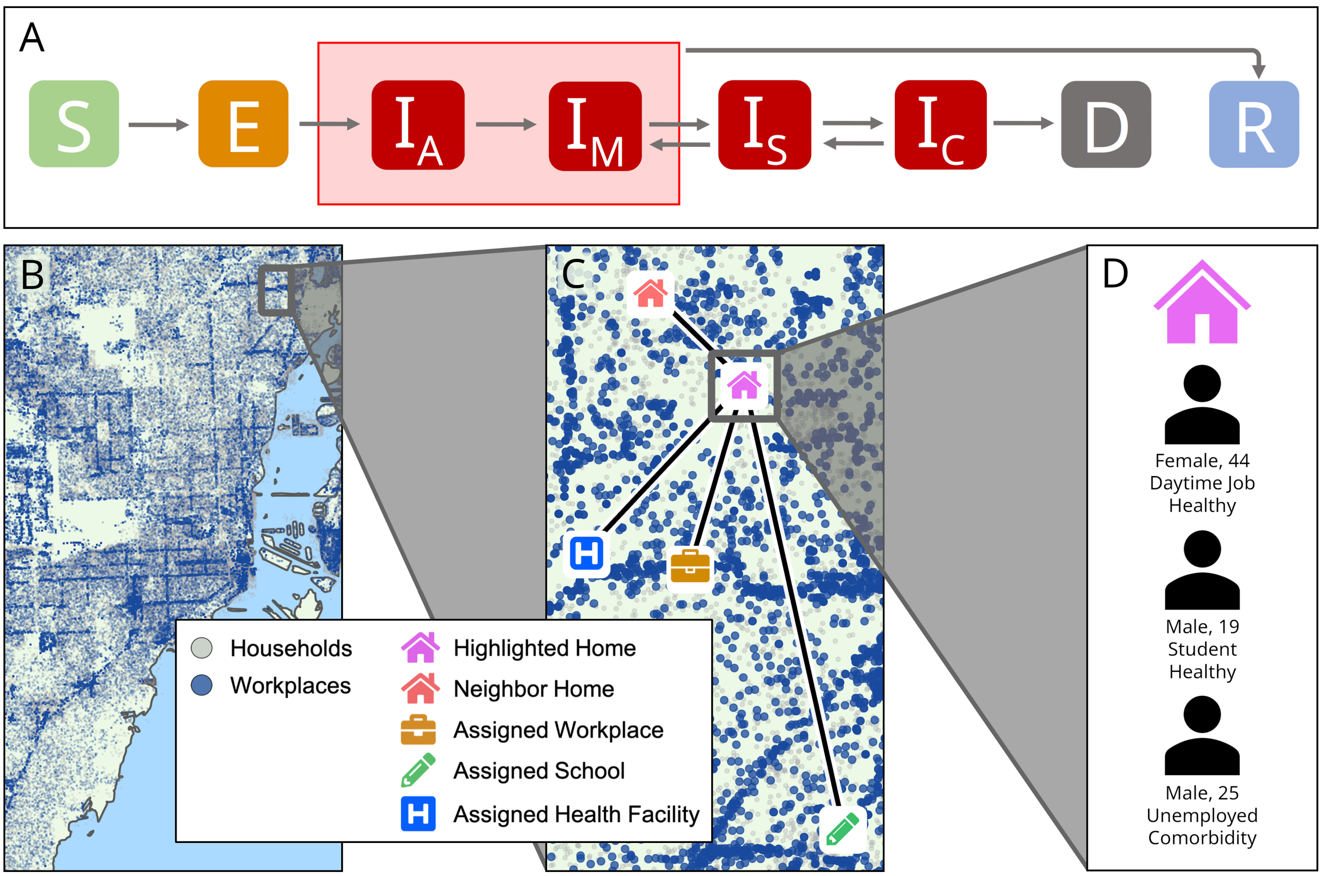


**Figure 5:** (A) Progression of the disease states in the model: susceptible ($S$) individuals may become exposed ($E$) to the virus, then progress to being infected (initially asymptomatic [$I_A$], possibly progressing to mild [$I_M$], severe [$I_S$] or critical [$I_C$]), and finally recovering ($R$) or dying ($D$). (B) Model locations of households and workplaces in an urban region (Miami, FL). (C) An example household. People may contact others by socializing with other households, by going to work or school, by going to the hospital, or by patronizing nearby businesses (not shown). (D) Attributes of the people in this household.

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