

# How will COVID-19 persist in the future? Simulating future dynamics of COVID-19 using an agent-based network model

Ethan Roubenoff<sup>1</sup>

<sup>1</sup>*Department of Demography, University of California, Berkeley*

## Abstract

Despite the United States Center for Disease Control (CDC)'s May 2023 expiration of the declared public health emergency pertaining to the COVID-19 pandemic (Silk 2023), approximately 3 years after the first cases of SARS-CoV-2 appeared in the United States, thousands of new cases persist daily. Many questions persist about the future dynamics of SARS-CoV-2's in the United States, including: will COVID continue to circulate as a seasonal disease like influenza, and will annual vaccinations be required to prevent outbreaks? In response, we present an Agent Based Networked Simulation of COVID-19 transmission to evaluate recurrent future outbreaks of the disease, accounting for contact heterogeneity and waning vaccine-derived and natural immunity. Our model is parameterized with data collected as part of the Berkeley Interpersonal Contact Survey (BICS; Feehan and Mahmud 2021) and is used to simulate time series of confirmed cases and deaths due to SARS-CoV-2, paying special attention to seasonal forces and waning immunity (Kronfeld-Schor et al. 2021; X. Liu et al. 2021; Nichols et al. 2021). From the BICS ABM model we simulate SARS-CoV-2 dynamics over the 10-year period beginning in 2021 with waning immunity and inclusion of annual booster doses under a variety of transmission scenarios. We find that SARS-CoV-2 outbreaks are likely to occur frequently, and that distribution of booster doses during certain times of the year—notably in the late winter/early spring—may reduce the severity of a wintertime outbreak depending on the seasonal epidemiology of the pathogen.

## 1 Introduction

Three years after the first cases of SARS-CoV-2—the pathogen responsible for the COVID-19 pandemic—appeared in the United States, many control measures put in place during the early phase of the pandemic have been eliminated (Silk 2023) in favor of a desire to return to ‘business as usual’. This includes mask mandates, shelter-in-place and work from home ordinances, physical distancing guidelines, and recommendations to isolate symptomatic cases. While vaccines for COVID-19 were a source of optimism through early 2021, it became clear over the subsequent months that waning natural and vaccine-derived immunity and the pathogen’s immunity-evading mutations rendered the vaccines less effective at ending the pandemic than initially hoped (Levin et al. 2021, Centers for Disease Control and Prevention 2021). Additionally, uptake of booster doses has lagged far behind targets (National Center for Immunization and Respiratory Diseases (NCIRD) 2022a; National Center for Immunization and Respiratory Diseases (NCIRD) 2022b). Over time, ‘pandemic fatigue’ has set in as compliance with disease-preventing behavioral mandates, especially mask usage and contact limitation, has slipped and such ordinances have been lifted (Reicher and Drury 2021). The current phase of the pandemic is substantially different from the initial phase, characterized by a highly

36 transmissible but less severe form of the illness owing in part to many factors acting in different directions,  
37 including: highly transmissible but less severe later variants (Davies et al. 2021; Strasser et al. 2022; Yang  
38 et al. 2022), higher levels of partial or full immunity from vaccine or prior infection (Clarke 2022), higher  
39 levels of social contacts approaching pre-pandemic levels, low or absent rates of mask usage and physical  
40 distancing (Crane et al. 2021), and better treatments reducing the probability of death or severe illness after  
41 infection (National Institutes of Health 2022). While better treatments, milder variants, and prior immunity  
42 has resulted in a far lower case fatality ratio than the early days of the pandemic, avoiding the illness is still  
43 a persistent challenge for those who remain at elevated risk of severe illness and death due to COVID-19,  
44 including the elderly and people with chronic health conditions.

45 Understanding the various drivers of future SARS-CoV-2 outbreaks can help to plan for future inter-  
46 ventional strategies, including vaccine distribution, school or work closure, and other non-pharmaceutical  
47 interventions. Infectious disease models can help to plan for these future outbreak scenarios by helping  
48 understand how SARS-CoV-2 will exist in our medium to long-term future. Here, we consider the effect  
49 of recurrent outbreaks, annual distribution of booster doses, and seasonal change in transmission of SARS-  
50 CoV-2. It is presently unknown if SARS-CoV-2 will demonstrate a strong seasonal pattern; however, it  
51 is hypothesized to follow the seasonal patterns of influenza and other coronaviruses (Kronfeld-Schor et al.  
52 2021).

53 To answer these questions, we use a stochastic Agent-Based Network Model parameterized with contact  
54 data from the Berkeley Interpersonal Contact Survey (BICS; Feehan and Mahmud 2021). Using BICS data  
55 allows us to consider how contact heterogeneity, household structure, and other network dynamics play  
56 into the periodicity and size of future outbreaks. Our model also includes seasonal forcing of transmission  
57 parameters, waning immunity from vaccines and prior infection, and variable-rate case importation to capture  
58 interaction with counter-seasonal populations (i.e., travel between the hemispheres experiencing opposite  
59 seasons).

60 Agent-Based Models are alternative to compartmental models and allow for more flexible and dynamic  
61 transmission dynamics, including network structure (Ajelli et al. 2010; Bansal, Read, et al. 2010). Roubenoff,  
62 Feehan, and Mahmud 2023 utilized a compartmental model for analyzing SARS-CoV-2 transmission. While  
63 these types of models are heavily utilized for analyzing disease transmission, one particular limitation of  
64 these models with contact data is their requirement for relatively few and well-defined categorizations. ABMs  
65 are more flexible and contrast with compartmental models by keeping track of the disease status for each  
66 individual in the simulation, rather than the tally of individuals in a particular compartment. In place of  
67 differential equations describing flows between compartments, ABMs use highly explicit (either deterministic  
68 or stochastic) rules governing interaction (Bonabeau 2002; Bruch and Atwell 2015; J. D. Baker et al. 2013; He,  
69 Ionides, and King 2010). Interactions are governed by some aspect of the simulated population contained  
70 within an objective function, such that interactions between nodes of certain values are more likely than  
71 others. As a group, ABMs are free of many of the analytic requirements of compartmental models—especially  
72 the need for of explicit transition properties between states, only an objective function for optimization—  
73 earning them the descriptor ‘plug and play’ (He, Ionides, and King 2010). Importantly for our purposes,  
74 ABMs allow for flexibility in how the population mixes, allowing for contact inequality between simulated  
75 agents through either a spatial or network component. Network models are a particular type of agent-based  
76 model that assume an explicit network structure for disease-transmitting contacts. In network models,  
77 instead of a homogenous or even matrix-structured contact pattern employed by compartmental models,  
78 disease transmission is simulated as occurring over a graph representing network connections (Bansal, Bryan

79 T Grenfell, and Meyers 2007; Danon et al. 2011; Matt J Keeling and Eames 2005).

80 A plethora of agent-based and network simulations of the COVID-19 pandemic have been published and  
81 tools released. The flexible yet highly specific ways that ABMs can be used to model social interactions is  
82 ideal for testing network and behavioral interventions for COVID-19. These models include a wide set of  
83 techniques, including models that explicitly account for how individuals navigate geographic space (Cuevas  
84 2020) and social networks (Hunter and J. D. Kelleher 2021). Agent-based models have used to estimate  
85 parameters for the COVID-19 outbreak in France (Hoertel et al. 2020), Ireland (Hunter and J. D. Kelleher  
86 2021), and Colombia (Gomez et al. 2021). Models can utilize existing contact data, such as Moghadas et al.  
87 2021 and Sah et al. 2021, who use data from the POLYMOD survey (Mossong et al. 2008) as inputs to an  
88 ABM to evaluate willingness to vaccinate. Holmdahl et al. 2020 test a series of behavioral interventions in  
89 nursing homes using a two-cohort ABM (patients and caregivers), finding that testing frequency and isolation  
90 are the most effective ways to limit the spread of disease. We draw on a number of general ABMs developed  
91 for COVID-19, including Covasim (Kerr et al. 2021), an aspatial model that combines Erdos-Reyni Poisson  
92 random networks with SynthPops networks that are generated from empirical contact data, and OpenABM  
93 (Hinch et al. 2021), that simulates social network interaction through stochastic network simulation at the  
94 household, occupational, and random connectivity additively.

95 Although it remains to be seen, research has suggested that SARS-CoV-2 may exhibit seasonality similarly  
96 to influenza and other coronaviruses, which exhibit higher incidence in the colder months (Nichols et al.  
97 2021). Indeed, in the United States, the highest number of cases were observed in winters 2020-2021 and  
98 2021-2022. The periodicity and severity of future SARS-CoV-2 outbreaks is currently unknown, largely  
99 since the rate of mutations and long-term vaccine-derived and natural immunity is unknown, but many  
100 mechanisms are theorized (Kronfeld-Schor et al. 2021). Seasonal forcing of respiratory diseases involves a  
101 consideration of multiple temporal factors relevant to modeling the transmission of SARS-CoV-2 including  
102 seasonal changes in host behavior and immune function (Altizer et al. 2006; Grassly and Fraser 2006).  
103 Although modeling studies suggest that climate may mediate the timing and peak incidence of SARS-CoV-2  
104 outbreaks, susceptible supply driven by population immunity is the primary driver of such dynamics (R. E.  
105 Baker et al. 2020). Many childhood diseases, namely measles, exhibit seasonal cycles driven by the birth rate  
106 and increased contact between non-immune children during the school year (Metcalf et al. 2009). In some  
107 situations, it can be assumed that the pathogen is circulating at a very low level locally in between outbreaks;  
108 in others, like influenza, seasonal human-animal interactions and case importation between populations in  
109 opposite hemispheres experiencing counter-cyclical temperature-forced outbreaks may drive timing (Lofgren  
110 et al. 2007; Lowen and Steel 2014).

111 We use a stochastic agent-based network simulation of SARS-CoV-2 transmission parameterized with  
112 data from the Berkeley Interpersonal Contact Survey (Feehan and Mahmud 2021) and include seasonality,  
113 annual vaccination, waning immunity, and demography. To our knowledge, this represents the first agent-  
114 based model for examining COVID-19 endemic outbreak cycles and seasonality. We find that outbreaks are  
115 likely to occur regularly and that annually-distributed booster doses can be an effective tool to eliminate  
116 regular outbreaks. Depending on seasonal epidemiology of the pathogen, booster doses are most effective  
117 when distributed at certain times of year; in the absence of seasonality, booster doses are most effective when  
118 distributed in the first half of the year, but in a seasonally-forced transmission scenario distributing vaccines  
119 in early fall is more successful at eliminating major annual outbreaks.

## 120 2 Methods

### 121 2.1 Data

122 Like Roubenoff, Feehan, and Mahmud 2023, we also utilize here contact survey data collected by Feehan  
123 and Mahmud 2021 as part of the ongoing Berkeley Interdisciplinary Contact Survey (BICS), which captures  
124 disease-transmitting behavior during the COVID-19 pandemic. The BICS survey, collected in several waves  
125 beginning in March 2020, is an online survey aimed at capturing the frequency and nature of respondents’  
126 face-to-face contact over a 24-hour period. Respondents to the BICS survey are recruited through a quota  
127 sample using an online survey panel provider, Lucid. Respondents are asked to report the total number of  
128 close, face-to-face contacts they had over the previous 24 hours, and to elaborate on three such contacts in  
129 detail. Respondents are also asked to report information regarding their demographic information, household  
130 structure, and other questions regarding their behavior. We utilize responses from Wave 6 (n = 5418, 12  
131 May - 25 May 2021) of the BICS national (U.S.) survey to capture post vaccination contact patterns.

### 132 2.2 Model: BICS ABM

133 Using Hunter, Mac Namee, and J. D. Kelleher 2017’s taxonomy for categorizing Agent Based Models, the  
134 simulation model used here is disease-specific to COVID-19 and society-specific to the behaviours captured  
135 from respondents in the BICS sample frame. Behavior is modeled on networks and is without transportation  
136 and without environment. The BICS ABM simulation population is constructed of individuals (also referred  
137 to as agents, nodes, or vertices) within households. We simulate interaction and disease spread among  
138 a population of 1000 households (approx. 3200 individuals) representative of the U.S. according to the  
139 procedure described below and in the model supplement. Each agent in the simulation directly corresponds  
140 to a respondent in the BICS or POLYMOD surveys sampled with survey weights to match the distribution  
141 of age and sex of the US population, and the agents’ demographic and behavioral data is derived from the  
142 corresponding survey respondent. The simulation includes three types of social contacts: household contacts  
143 with household members, school contacts for children below the age of 18, and non-household ‘random’  
144 contacts. As employment data are not available for this wave of the survey, ‘random’ contacts are designed  
145 to include employment contacts for adults. Household contacts and school contacts are drawn randomly  
146 at the start of the simulation according to the procedure described below and are maintained throughout  
147 the simulation; random draws of graphs representing random non-household contacts are taken during each  
148 daytime time step. In this way, the total network is *dynamic* as it changes throughout the course of the  
149 simulation.

150 The graph of household contacts is drawn according to the procedure described in the model supplement,  
151 which is similar to the SynthPops procedure utilized in COVASIM (Kerr et al. 2021; Mistry et al. 2021).  
152 Briefly, a supplied number of households are created with the following two-step procedure. First, BICS  
153 survey respondents are repeatedly sampled with replacement and adjustment for survey weights to be heads  
154 of household. Heads of household are chosen to match the age- and sex-distribution of adults in the United  
155 States using 2021 American Community Survey estimates (US Census Bureau 2022). Then, households are  
156 filled by sampling BICS respondents (again, with replacement and adjustment for survey weights) who match  
157 the household head’s reported household members’ age and gender, until each household is the proper size.  
158 Respondents under the age of 18 were not ascertained in the BICS survey; instead, they are sampled uniformly  
159 from the POLYMOD UK survey (Mosson et al. 2008). Throughout the simulation, each node’s behavior

160 is derived from the corresponding BICS survey respondent’s responses; nodes derived from POLYMOD  
161 respondents are derived from the corresponding fields in the POLYMOD survey<sup>1</sup>.

162 The model progresses in hourly steps through simulation time. During morning and evening hours  
163 (6pm-8am), agents have contact with all members of their household. During daytime hours, random  
164 connections occur between members of the simulation population governed by the degree (number of non-  
165 household contacts) supplied by each survey respondent. A daily contact network is drawn using the Network  
166 Configuration Model, described below. Each such contact is designated to begin at a random time during  
167 the day chosen uniformly between 8am and 6pm. Each contact has a randomly chosen duration sampled  
168 according to the following probabilities: respondents to wave 6 of the BICS survey indicated that 17.1% of  
169 contacts were less than one minute, 45.2% were between 1 minute and 15 minutes, 18.7% were less than  
170 one hour, and 18.9% were more than 1 hour. Here, we choose to use the marginal distribution rather  
171 than individual-level responses due to computational limitations. During the duration of each contact,  
172 respondents are disconnected from other members of their household and reconnected after the conclusion of  
173 the random contact. If a node is clinically infectious, they may enter isolation for the duration of symptoms;  
174 if asymptomatic, they continue to mix as before. Isolation is incorporated by a parameter representing the  
175 multiplicative reduction in random daily contacts: for example, an isolation parameter of 0.1 means that a  
176 node normally with 10 daily non-household contacts with would have 1 such contact while in isolation.

177 Random contacts are drawn using the Network Configuration Model, which generates a random graph of  
178 contacts that preserves each node’s degree—here, the number of daily non-household contacts. The network  
179 configuration model creates random networks using only a provided degree distribution as an input. The  
180 configuration model works through a two-step procedure (Albert-László Barabási 2021):

- 181 1. First, assign a degree to each node in the network such that the distribution of degrees matches the  
182 desired distribution. For each node, assign a number of ‘stubs’ or half-edges equal to the degree of that  
183 node.
- 184 2. Second, randomly and uniformly join stubs to create edges until there are no stubs remaining in the  
185 network.

186 Without alteration, the model may produce self-edges (a node connected to itself), or multi-edges (multiple  
187 edges connecting a pair of nodes). Sampling ‘simple’ graphs that lack self- or multi-edges is a computationally  
188 intensive procedure and non-uniform in its graph-generating process. In our application, we continue to  
189 sample graphs uniformly and remove self-edges, but maintain multi-edges. Realizations of this type are  
190 likely to have total degree less than would be implied by the supplied degree distribution.

## 191 **2.3 Transmission of SARS-CoV-2**

192 All agents begin susceptible and vaccine roll-out begins at the beginning of simulation time. At a given time  
193  $T_0$  a supplied number of index cases are chosen randomly to be exposed to SARS-CoV-2. In addition, a  
194 vector representing the number of cases imported daily is provided as input to the simulation—in the present  
195 application, one case weekly is imported to ensure that SARS-CoV-2 is constantly circulating at a low level.  
196 At exposure, each agent is assigned a randomly drawn number of hours spent as exposed and infectious;  
197 they then proceed to either symptomatic or asymptomatic infection with a supplied probability. Baseline

---

<sup>1</sup>Some fields collected as part of the BICS survey are not available in the POLYMOD data and are imputed. For the present application, only mask/NPI usage is unavailable, and is taken to be the False.

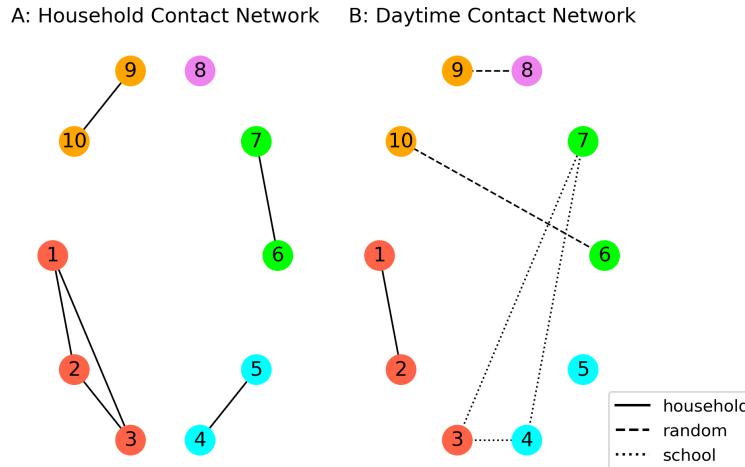


Figure 1: Illustration of network structures used during simulation. (A): household contact network representing evening and morning contacts, and (B): daytime contact network, consisting of school contacts and randomly drawn contacts. While school contacts are maintained throughout the simulation (with the exception of summertime school closures), random contacts are re-drawn hourly.

198 probability of transmission—before considering vaccine efficacy, contact duration, non-pharmaceutical inter-  
 199 ventions, and asymptomatic reduction in transmission probability—from an infected node to a susceptible  
 200 node occurs with probability  $\beta(t)$ , where  $t$  is the simulation’s  $t$ th day in the year. The value of  $\beta(t)$  thus  
 201 represents the probability of transmission during an hour-long contact between one clinically symptomatic  
 202 node and one susceptible, unvaccinated node without mask usage.

203 Various factors multiplicatively reduce the probability of transmission. First, transmission is reduced  
 204 proportional to the duration of contact in fractions of an hour; a 15-minute contact is 1/4-th as likely to result  
 205 in transmission as an hour-long contact. As well, transmission from an asymptomatic node to a susceptible  
 206 node occurs at a reduced probability  $\alpha$  relative to symptomatic nodes. The susceptible node’s vaccination  
 207 status reduces the probability of transmission by the corresponding vaccine efficacy; the infectious node’s  
 208 vaccination status is not assumed to affect transmission probability. As detailed further below, we allow for  
 209 reinfection after a set amount of time; previous infection offers protection for the recovered node as a reduction  
 210 in the transmission probability. Finally, the model includes a single Non-Pharmaceutical Intervention (NPI),  
 211 designed to capture the combined disease-blocking effectiveness of masks, physical distancing, and other  
 212 preventative measures. If BICS respondents corresponding to both nodes in a random contact report any  
 213 mask usage, the probability of transmission is proportionally reduced by a supplied effectiveness. If NPI  
 214 effectiveness is set to 0, the simulation is effectively in the absence of NPI usage. We assume that NPIs  
 215 are not used among household contacts. At the conclusion of the infectious period, asymptomatic nodes all  
 216 recover; symptomatic nodes die with a supplied age-based probability.

217 Unlike compartmental models that often hinge on parameter  $R_0$ —the basic reproduction ratio, repre-  
 218 senting the average number of secondary cases caused by an index case in a fully susceptible population,  
 219 estimated as the product of the transmission probability, contact rate, and duration of infectiousness—no  
 220 such closed form solution for  $R_0$  in an ABM necessarily exists. Although the ABM’s ability to model con-  
 221 tact and transmission through separate processes and objective functions allows for for increased flexibility,  
 222 including time-variable and stochastic transmission probability, heterogeneous contact rates and network

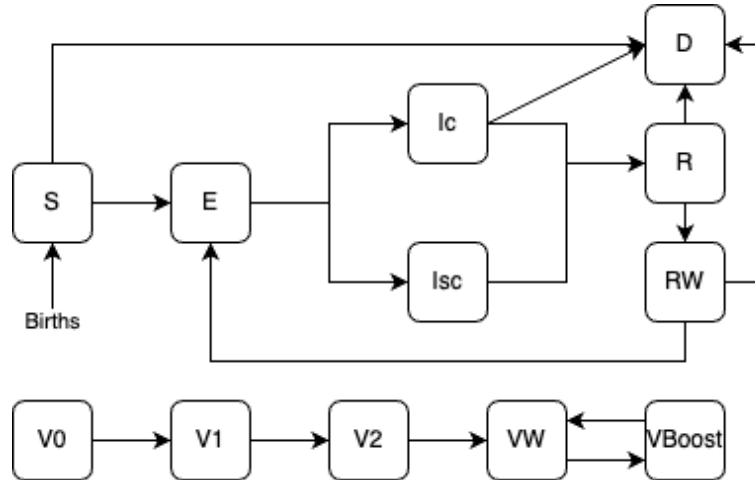


Figure 2: Schematic for disease states in the Agent-Based Model used in the present simulation, including disease status (top) and vaccination status (bottom). Disease states include susceptible (S), exposed/pre-infectious (E), clinically infectious (Ic), subclinically infectious (Isc), recovered (R), recovered with waned immunity (RW), and deceased (D). Vaccination states include unvaccinated (V0), first and second primary doses (V1, V2), waned immunity (VW), and boosted (VBoost).

223 structure, variable duration of contact, isolation of infectious cases, and uneven NPI and vaccination usage,  
 224 the parameters that govern the overall transmission dynamics are not well-defined in closed form. Instead,  
 225 the  $R_0$  must be estimated from the the contact rate or incidence curve generated by the simulation itself  
 226 (Hunter, Namee, and J. Kelleher 2018; Venkatramanan et al. 2018; Hunter and J. D. Kelleher 2021; Hoertel  
 227 et al. 2020). We estimate  $R_0$  by observing the hourly contact rates  $\hat{c}_h$  in the initial 7 days of the simulation,  
 228 using the expression:

$$\hat{c}_h = \sum_{e \in E_h} (Duration_e \cdot NPI_e) \quad (1)$$

229 which estimates the average hourly number of edges weighted by the duration and effectiveness of non-  
 230 pharmaceutical interventions, if used during each contact. Then, the average contact rate  $\bar{c}$  is taken to be  
 231 the average of the hourly contact rates  $\hat{c}_h$ . Finally,  $R_0$  is taken to be:

$$R_0 = \bar{\beta} \cdot \bar{d} \cdot \frac{\bar{c}}{N} \cdot [\rho + (1 - \rho)\alpha] \quad (2)$$

232 where parameter  $\bar{\beta}$  is the average transmission probability over the course of the simulation period,  $\bar{d}$  is the  
 233 average duration of infectiousness,  $N$  is the size of the population, and the expression  $\rho + (1 - \rho)\alpha$  represents  
 234 the reduction in infectiousness among subclinical cases. Since  $R_0$  is not necessarily known until the conclusion  
 235 of the simulation, we instead determine the overall transmission rate by varying the transmission probably  
 236  $\beta$ , then estimating  $R_0$  post-facto.

## 237 2.4 Vaccine effectiveness, waned immunity, and reinfection

238 An important feature of the model is waning natural and vaccine-derived immunity. Vaccination occurs  
 239 uniformly in the population assuming that all eligible members of the population are equally able to access  
 240 the vaccine (differing from the prioritization procedure taken by Roubenoff, Feehan, and Mahmud 2023), at  
 241 a baseline 70% uptake rate. A set number of vaccine doses are available daily, and are distributed at 8am

242 each day according to optional priority rules. Nodes are eligible for a second dose of the vaccine after 25  
243 days. After a supplied amount of time, immunity wanes, and nodes are eligible for booster doses; booster  
244 doses are made available at a set date every year and are distributed with the same rate and priority schedule  
245 as primary doses. Only nodes with waned immunity are eligible for booster doses. All vaccination statuses  
246 (1st dose, 2nd dose, waned, and booster) have fixed proportional reductions in transmission probability.

247 As well, we include reinfection in the model with a similar procedure. Nodes that have recovered from  
248 infection remain completely protected from infection for a fixed amount of time; after this, nodes are assigned  
249 disease status ‘Recovered-Waned’ indicating that they may be re-infected, yet have some protection against  
250 future infection. Waned immunity is assumed to be the same for clinical and subclinical infections, and the  
251 protection offered does not depend on the number of previous infections.

252 At present, the duration of immunity after infection and vaccination is not known, but is estimated to  
253 be approximately 6 months of near-complete immunity followed by a steady decrease over time (Centers for  
254 Disease Control and Prevention 2021). This is implemented in our model with a pair of parameters: first, the  
255 duration of complete immunity, governing the time after infection or vaccination that an individual experi-  
256 ences the full effect of vaccine-derived or natural immunity; second, the waned immunity effectiveness. For  
257 simplicity, these parameters are held to be the same for both vaccine-derived and post-infectious immunity.

## 258 2.5 Incorporation of Seasonality

259 Seasonal environmental changes are known to affect infectious disease transmission in predictable, annually-  
260 recurrent cycles. Although seasonality is well documented in many infectious diseases, the underlying mech-  
261 anisms are frequently poorly understood or difficult to tease out from other compounding effects (Fisman  
262 2012). For respiratory infections transmitted between humans via the airborne pathway such as SARS-CoV-  
263 2 and influenza, seasonal effects can be grouped in three broad areas: environmentally-driven changes in  
264 host behavior, such as summertime school closings or increased wintertime indoor gatherings; the pathogen’s  
265 ability to survive outside of the human host adapted to certain climatic conditions, in turn affecting fitness  
266 for transmission; and seasonal changes in the host’s immune response, possibly due to changes in temperature  
267 or sunlight exposure (Altizer et al. 2006; Grassly and Fraser 2006; Dowell 2001; Kronfeld-Schor et al. 2021;  
268 Buonomo, Chitnis, and d’Onofrio 2018; Held and Paul 2012). Additionally, seasonal migration—especially  
269 temporary domestic migration with an annual cyclical pattern—may fundamentally change the landscape  
270 of interactions and population at risk (Buckee, Tatem, and Metcalf 2017).

271 Incorporating seasonality into a compartmental model is typically done by adding sinusoidal temporal  
272 forcing to the transmission parameter  $\beta$  as  $\beta(t) = \beta_0(1 + \beta_1 \cos(2\pi t))$ , through a binary indicator in the case  
273 of seasonal school closings as  $\beta(t) = \beta_0(1 + \beta_1 \text{term}(t))$ , or other time-dependent functional form (Grassly and  
274 Fraser 2006; Matt J. Keeling, Rohani, and Bryan T. Grenfell 2001). Here, the basic reproductive number  $R_0$   
275 represents the average number of secondary cases from a single index case introduced at a random time of the  
276 year, and is defined as  $\hat{R}_0 = D \int \beta(t) dt$  where  $D$  is the average duration of infection. Forcing functions can  
277 be easily extended to include age-structured contact (Bolker and B. Grenfell 1993), and time-series methods  
278 allow for modeling of outbreak dynamics without fitting a functional form to the transmission parameter  
279 (Metcalf et al. 2009; Finkenstädt and B. T. Grenfell 2000). Extending beyond compartmental models,  
280 seasonality can be incorporated into modeling of incidence data; Held and Paul (2012) demonstrate how  
281 seasonal incidence can be decomposed into an endemic and epidemic component with independent temporal  
282 structure.

283 The long-term seasonal patterns and drivers of COVID-19 are still unknown and not necessarily possible



284 to disentangle from control efforts, especially noting non-pharmaceutical interventions like shelter-in-place  
285 ordinances and mask usage. Weaver et al. 2022 note in a review that COVID-19 may be more stable and more  
286 transmissible in cooler environments, consistent with influenza<sup>2</sup>, although both stability in high humidity  
287 and low humidity have been observed (Morris et al. 2021; Marr et al. 2019; Matson et al. 2020; Dabisch  
288 et al. 2021). SARS-CoV-2’s preference for colder, drier conditions is consistent with climatic effects observed  
289 with influenza (Lofgren et al. 2007; Lowen and Steel 2014; Shaman and Kohn 2009). Another review article  
290 by Mecenas et al. 2020 finds a similar conclusion. While climate may affect SARS-CoV-2’s transmissibility  
291 directly, the indirect effect of climate’s effect on human behaviors has been demonstrated to be a much  
292 stronger effect (Susswein, Rest, and Bansal 2023; Damette, Mathonnat, and Goutte 2021; Weaver et al.  
293 2022). Indeed, research on contact patterns that relate to COVID-19 have are known to be a substantial  
294 driver of outbreak dynamics (Feehan and Mahmud 2021).

295 While many applications of ABMs to infectious disease focus on investigating the interaction-level, net-  
296 work, or transportation aspects of infectious disease, few have focused directly on seasonality. Arduin et al.  
297 2017 incorporate seasonal forcing of pneumococcal infections linked to influenza infection using a fixed mul-  
298 tiplication of the transmission probability during the flu season, similar to the school-term forcing of the  
299 transmission probability used in compartmental models by Matt J. Keeling, Rohani, and Bryan T. Grenfell  
300 2001. Similarly, Williams et al. 2022 incorporate seasonal forcing to an ABM used to study influenza by  
301 adding a multiplicative effect to the transmission parameter related to the temporal distance of each time  
302 period from the winter solstice, which is ‘intended to account for factors that may influence transmissibility  
303 across a range of seasons due to variability in factors such as temperature, humidity, and changes in contact  
304 rates.’ In an application of ABMs to COVID-19, Krivorotko et al. 2022 use an time-series model to decom-  
305 pose incidence counts into a time series effect, a seasonal effect, and a noise component; the seasonal effect  
306 and the time-series effects are specified to have a temporally autocorrelative function. ABMs have also been  
307 used to study seasonality in non-human diseases (Dawson et al. 2018; Oraby et al. 2014).

308 We incorporate seasonality in two ways: in the transmission probability  $\beta(t)$  and in the number of  
309 nonhousehold contacts. We allow for seasonal forcing of the transmission probability to capture how the  
310 transmissibility of the pathogen may change with weather, modeled as:

$$\beta(t) = \beta_0(1 + \beta_1 * \cos(2\pi/365 * t)) \quad (3)$$

311 where  $\beta_0$  represents the average transmission probability and  $\beta_1$  represents the amplitude of seasonal forcing  
312 (X. Liu et al. 2021; Grassly and Fraser 2006). Second, we include school contacts between children under  
313 18, which are derived from the POLYMOD survey (Mossong et al. 2008). School contacts are drawn once at  
314 the start of the simulation and maintained through simulation time. Schoolchildren are taken to have school  
315 contacts during the same business hours as random contacts between September 1st and June 1st annually;  
316 children who are clinically infectious with SARS-CoV-2 are kept home from school until they recover.

## 317 2.6 Incorporation of Demography

318 Optional in the model is the inclusion of basic demographic vital rates in the form of age-specific fertility  
319 and mortality data. At baseline, we draw from the CDC’s 2021 estimates (Martin, Hamilton, and Osterman  
320 2022; Xu et al. 2022) summarized to the age categories used in the model (0-18, 18-25, then in 10 year

---

<sup>2</sup>See Weaver et al’s review of the following studies, among others: Chin et al. 2020; J. Liu et al. 2020; Ma et al. 2021; Matson et al. 2020; Morris et al. 2021; Nottmeyer and Sera 2021; Raiteux et al. 2021; Riddell et al. 2020; Sera et al. 2021; Smith et al. 2021; Wu et al. 2020

321 increments through age 85). The rates used at baseline are shown in figure 12. These rates are used to  
322 randomly introduce new susceptibles into the population and randomly remove members of the population,  
323 representing deaths due to non-SARS-CoV-2 causes. Each birth represents a new, fully susceptible and  
324 unvaccinated child in the population in the household of the birthing parent; they are assumed to have the  
325 number of non-household and school contacts equal to the population average. Demography is incorporated  
326 into the model once monthly as a Bernoulli random draw for each member of the population with rate equal  
327 to the supplied rate, divided by 12 (for males, fertility rate is 0).

## 328 2.7 Simulation Procedure and Parameters

329 We identify a set of baseline transmission parameters in line with those used by Roubenoff, Feehan, and  
330 Mahmud 2023, adapted to fit the parametric needs of our Agent Based Model. For all simulations, the number  
331 of households is fixed at 1,000, producing approximately 3,200 individuals at the start of the simulation.  
332 Simulations are run for 10 years and are seeded with 5 index cases at time  $t = 0$ , intended to mimic the  
333 wintertime outbreak of early January 2021. During the course of the simulation, one case is imported weekly  
334 to ensure that all SARS-CoV-2 is constantly circulating at a low level. To account for a wide range of  
335 transmission scenarios, we consider three levels of transmissibility: low, with  $\beta_0 = 0.01$  (implying  $R_0 \approx 1.3$ );  
336 moderate, with  $\beta_0 = 0.025$  ( $R_0 \approx 3.4$ ); and high, with  $\beta_0 = 0.05$  ( $R_0 \approx 6.5$ ). Baseline simulations are without  
337 seasonal forcing of transmission or contact but with school contacts included during school-year weekdays  
338 (Monday-Friday, 9am-3pm). Seasonal forcing of both transmission and contact parameters is introduced  
339 with low (10%), moderate (25%), or extreme (50%) seasonal amplitude as described above. To introduce  
340 stochasticity into the model, each infected case in the simulation contains a randomly drawn duration of  
341 latent period of between 48 and 96 hours after transmission; this is followed by a randomly drawn infectious  
342 period of between 72 and 120 hours. These distributions are held constant across all simulations. We assume  
343 that each case has a 20% chance of being subclinical—fewer than used by Roubenoff, Feehan, and Mahmud  
344 2023 (derived from Johansson et al. 2021), but in line with recent meta-review estimates by Buitrago-Garcia  
345 et al. 2022. That same analysis identified seven studies comparing the secondary attack rate of asymptomatic  
346 cases and symptomatic cases with an average ratio of 32%. At baseline we assume that symptomatic cases  
347 have all of their normal random contacts and do not isolate, for a ‘business as usual’ scenario; we test the  
348 effect of isolation in sensitivity analysis. However, children are assumed to always be kept at home when ill.  
349 NPI effectiveness is set to zero, equivalent to the absence of NPIs or masks, but is varied during sensitivity  
350 analysis.

351 Vaccine effectiveness is taken to be 80% after one, dose, 90% after two doses, and 80% after three doses,  
352 consistent with estimates published in 2021 and 2022 (Tenforde 2021, Thompson 2021; Thompson 2022) and  
353 values used by Roubenoff, Feehan, and Mahmud 2023. Unclear at the present moment is the duration  
354 of immunity after infection and vaccination and the effectiveness of waned immunity; a 2021 CDC brief  
355 (updated 2022) estimates 6 months of nearly complete immunity that diminishes over time (Centers for  
356 Disease Control and Prevention 2021). We take 6 months to be the baseline assumption but vary this  
357 duration in monthly increments from 6 months to two years in a sensitivity test. We assume a pessimistic  
358 25% efficacy of waned natural- and vaccine-derived immunity.

359 All main simulations are run for 10 years in replicates of 10, and we report a number of summary values  
360 for all simulations. These include the total number of clinical and subclinical cases, deaths due to SARS-  
361 CoV-2, and the timing and size of all outbreaks after year 5. All estimates are standardized to the population  
362 size to account for populations that vary randomly in size. Outbreaks are found using the `Python` library

363 `scipy`'s `find_peaks` function on the daily sequence of clinical cases, for a minimum incidence threshold of  
364 5% of the population infected over a 30-day window.

### 365 **3 Results**

366 To elucidate future outbreaks of SARS-CoV-2, we simulate outbreaks at various levels of transmissibility,  
367 and test the distribution of annual booster doses in the absence of and presence of seasonality. We find that  
368 the optimal date of booster dose distribution for reducing the number of clinical infections is different for  
369 the simulations with and without seasonality; in the absence of seasonality booster doses in the first half of  
370 the year are most effective at eliminating a large annual outbreak, but with seasonality booster doses are  
371 most effective when distributed in early fall.

372 Infectiousness of COVID-19 and the duration of immunity after infection and vaccination have a strong  
373 effect on the dynamics of outbreaks. In a moderate transmission scenario, where the base probability of  
374 transmission for an hour-long contact the absence of NPIs or vaccination  $\beta_0 = 0.025$  (corresponding to  
375 an  $R_0$  of approximately 3.2), an average of 6.16 clinical infections occur per capita over a 10-year period.  
376 Over this period, an average of 33.6 outbreaks occur, each infecting an average of 17.7% of the population.  
377 However, when  $\beta_0$  is raised to 0.05 (corresponding  $R_0 \approx 6.5$ ), outbreaks are fewer (an average of 16.0 over  
378 the 10-year period) but more severe, with an average outbreak size of 59.63% and about 9.75 infections  
379 per capita—nearly one per person per year. These simulations are summarized in figure 3 and trajectories  
380 are shown in figure 4. We observe this dynamic throughout many simulations: when SARS-CoV-2 is more  
381 transmissible or less mitigated, outbreaks are fewer but more severe. Mortality in the high-transmission  
382 scenario is higher, proportional to the number of clinical infections—2.63% on average compared to 1.67%  
383 in the moderate transmission scenario and 1.1% in the low-transmission scenario. These simulations suggest  
384 that the public health planning and response for future variants may differ based on their epidemiology. More  
385 transmissible variants are likely to have fewer, larger outbreaks that may overwhelm the healthcare system  
386 capacity, but show few cases outside of the season; less transmissible variants may require a year-round  
387 response, but with less severe outbreaks.

388 Distributing annual booster shots consistently lowers the rates of SARS-CoV-2 for all simulations, but  
389 may independently induce a seasonal pattern in outbreaks. The ability of booster doses to successfully limit  
390 SARS-CoV-2 outbreaks is dependent on the timing of their distribution and whether or not seasonal forcing  
391 of transmission is included in the model. In the absence of seasonal forcing of transmission and contact  
392 (figures 5 and 6), distributing booster doses earlier in the year is most effective at reducing the size of  
393 outbreaks: assuming a 75% update of vaccinations distributed annually between January and May, about  
394 1.68 – 2.35 clinical infections occur per capita (average outbreak size ranging from 4.25% to 6.13% of the  
395 population); when distributed after July 1st, this rises to 4.34 – 4.76 clinical infections per capita (average  
396 outbreak size ranging from 13.21% to 15.61%). In a high-vaccine uptake scenario (90% uptake, shown in  
397 the model supplement), the overall rates of SARS-CoV-2 are lower only when vaccines are distributed in the  
398 first half of the year; when vaccines distributed in the latter half of the year, the total cases and severity of  
399 outbreaks is comparable to the regular-uptake scenario. Outbreaks are observed to generally occur around 6  
400 months—which is also the duration of full immunity after vaccination—after the date that booster doses are  
401 made available, with the strongest seasonal patterns observed with June-September distribution inducing  
402 a strong wintertime and October - December inducing an early spring outbreak. Indeed, as observed in  
403 figure 6, the peak out the outbreak appears to be 'shifted' approximately 6 months after the date of booster

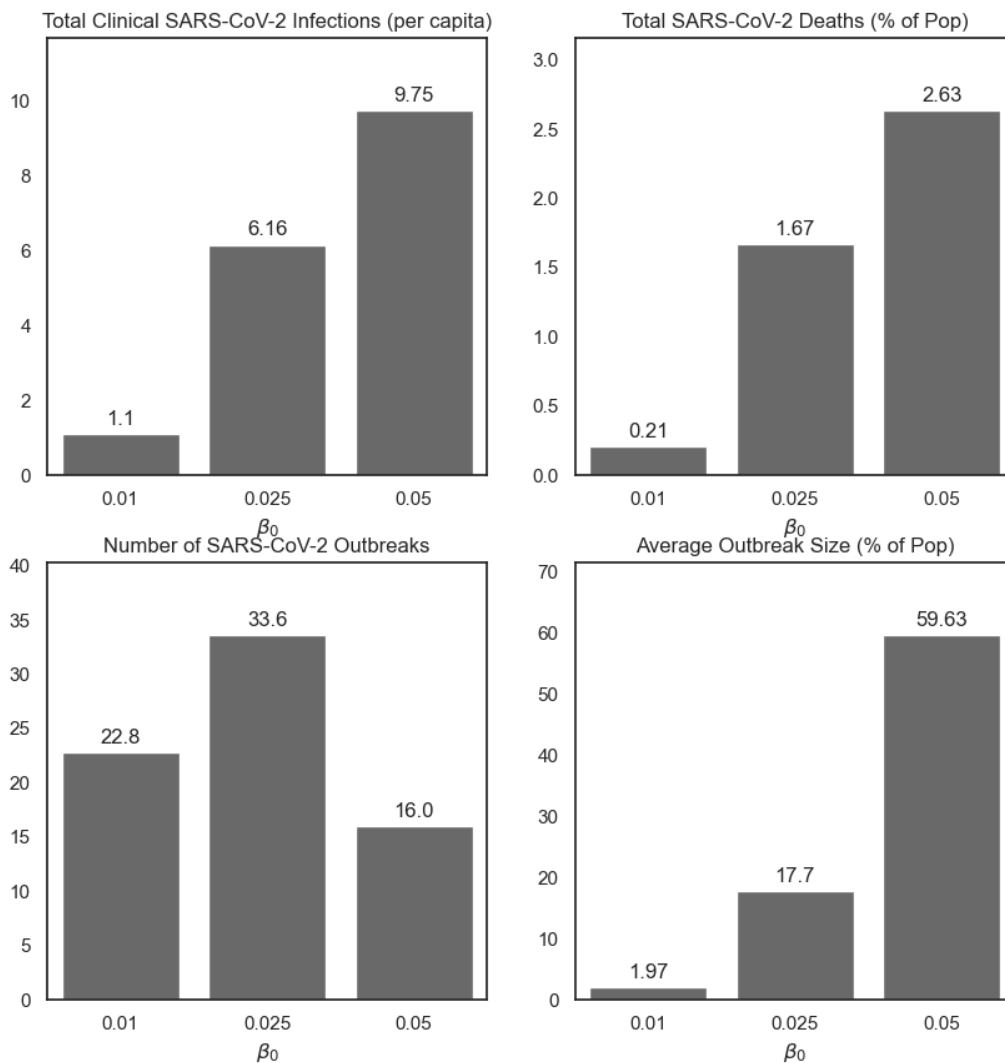


Figure 3: Summary of SARS-CoV-2 clinical infections, deaths, number of outbreaks, and average outbreak size for different values of  $\beta_0$ , the average baseline transmission probability, in the absence of seasonal forcing, isolation, or vaccine distribution. Simulations are run for 10 years in replicates of 10. Approximate corresponding values of  $R_0$  are:  $\beta_0 = 0.01$ ,  $R_0 \approx 1.3$ ;  $\beta_0 = 0.025$ ,  $R_0 \approx 3.4$ ;  $\beta_0 = 0.05$ ,  $R_0 \approx 6.5$ .

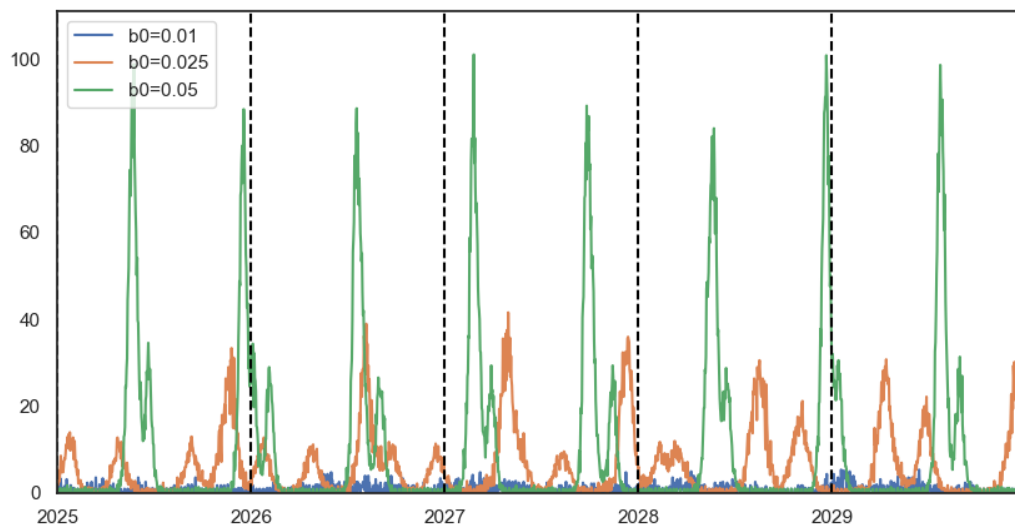


Figure 4: 5-year trajectory of SARS-CoV-2 clinical infections for different values of  $\beta_0$ , the average baseline transmission probability, averaged across 10 simulations each. When  $\beta_0$  is high, there are generally 1-2 large outbreaks per year; when lower, outbreaks are smaller and more frequent. Approximate corresponding values of  $R_0$  are:  $\beta_0 = 0.01$ ,  $R_0 \approx 1.3$ ;  $\beta_0 = 0.025$ ,  $R_0 \approx 3.4$ ;  $\beta_0 = 0.05$ ,  $R_0 \approx 6.5$ .

404 dose distribution, the duration of of full immunity after vaccination. This phenomenon—whereby January-  
405 May boosters nearly eliminate annual outbreaks in the steady state but later-year boosters fail to do so,  
406 albeit shift the outbreak timing—is likely driven by the initial outbreak (set to occur in January of 2021 to  
407 capture the large winter outbreak in the United States), which establishes the clock for a sufficient number  
408 of individuals with waned immunity to appear for an outbreak to occur predictably after. These simulations  
409 indicate that, in the absence of seasonality, the timing of booster dose distribution may have the power to  
410 govern the timing of the primary annual outbreak.

411 We also tested the distribution of vaccines in the presence of seasonal forcing of the transmission param-  
412 eters ( $\beta_1 = 0.5$ ), shown in figure 7 and 8. Unlike when distributing booster doses in the absence of seasonal  
413 forcing, in which the timing of booster dose distribution shifts the timing of the main outbreak, in these  
414 simulations with seasonal forcing a substantial wintertime outbreak occurs at nearly the same time every  
415 year. However, the size of this outbreak, as well as the presence of secondary outbreaks throughout the  
416 year, depends on the timing of booster doses. When boosters are distributed in the first half of the year—  
417 January 1st through May 1st—a moderate-sized fall outbreak occurs. When boosters are distributed by July  
418 1st, this fall outbreak nearly doubles in size; a September 1st distribution day results in a less predictable  
419 situation, with multiple (2-3) smaller outbreaks throughout the year. However, distributing doses too late  
420 (November 1st) results in a large summertime outbreak, despite the relatively lowered transmission rate  
421 during the summer months. These dynamics are similar when seasonal forcing is present in the transmission  
422 parameters and the contact rate, shown in the supplementary material. Overall, simulations where vaccines  
423 are distributed between January and June resulted in 2.76 – 3.36 infections per capita—1-1.5 fewer than  
424 when vaccines are distributed in the highest months of July or December (4.82, 5.19 respectively). However,  
425 distributing booster doses in September or October results in fewer infections, comparable with simulations

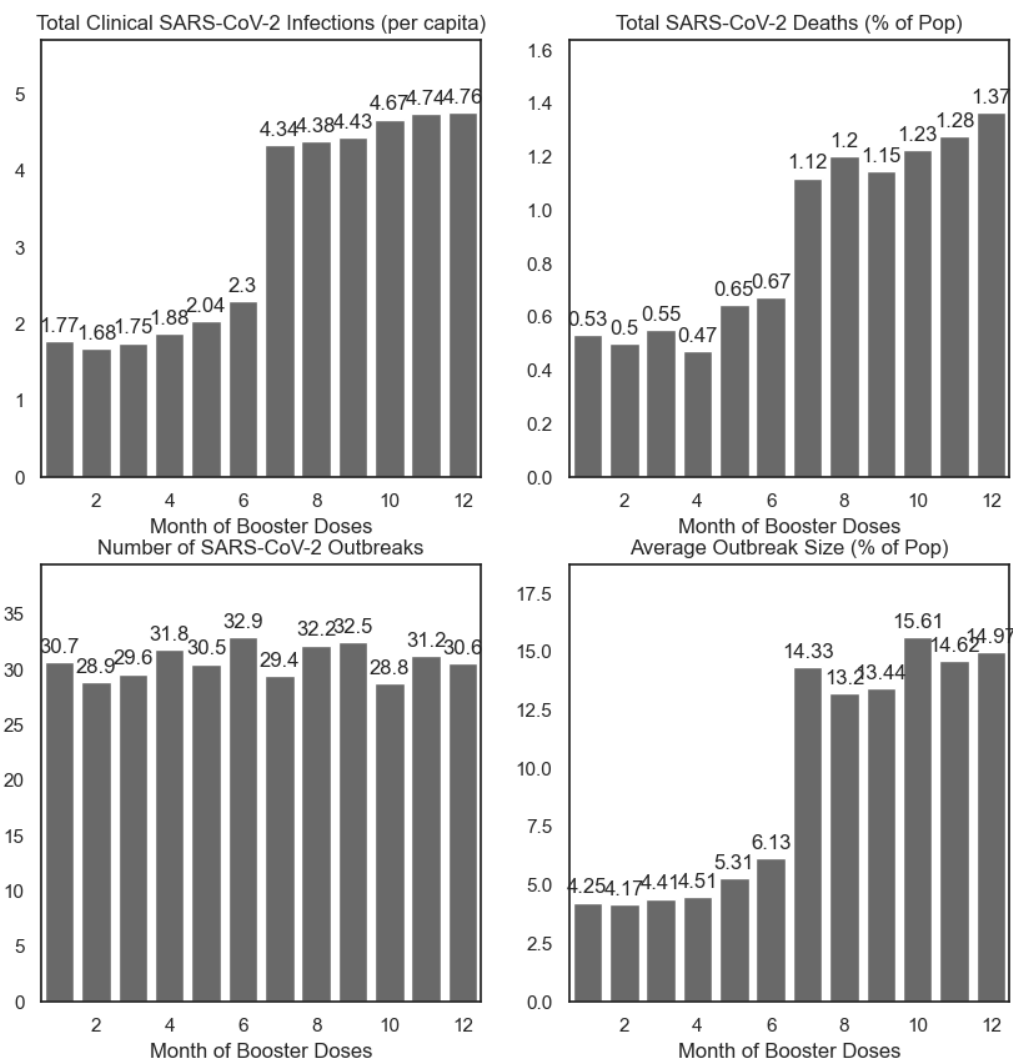


Figure 5: Summary of simulations by day of booster dose distribution, varied as the first of each month, in the absence of seasonal forcing of the transmission parameter  $\beta$ . Simulations are run for 10 years in replicates of 10.

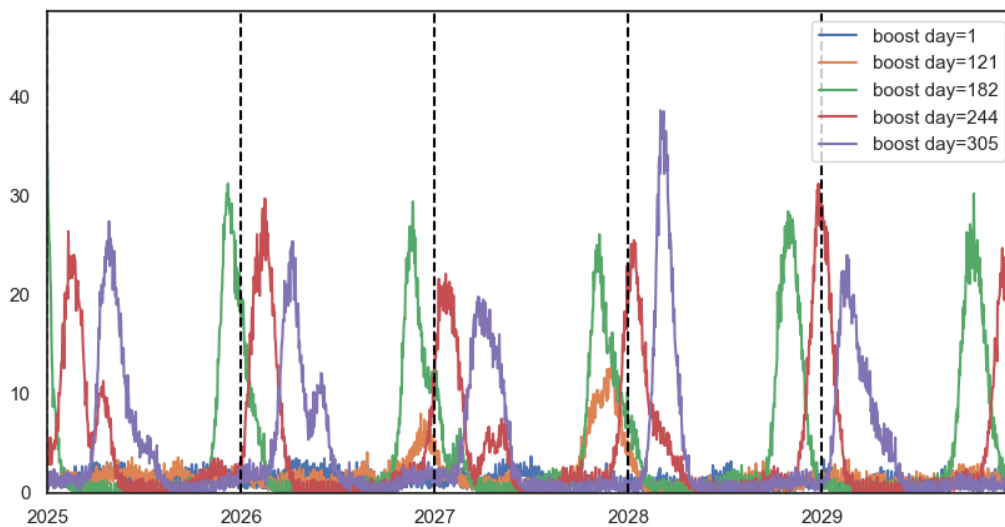


Figure 6: 5-year trajectory of simulations by day of booster dose distribution, in the absence of seasonal forcing, for selected distribution days: Jan 1st (day 1), May 1st (day 121), July 1st (day 182), Sept 1st (day 244), and Nov 1st (day 305), averaged across 10 replications. When doses are distributed earlier in the year—Jan 1st–May 1st—the major outbreaks are largely averted, but persist when doses are distributed too late in the year.

426 when vaccines distributed earlier in the year, and smaller outbreaks (8.69%–9.59% of the population infected  
427 on average per outbreak).

428 As a sensitivity test, we varied the duration of immunity after infection and vaccination, and find that  
429 this parameter has a substantial effect on the timing and size of outbreaks. As this parameter governs the  
430 rate and which susceptibles are effectively re-introduced into the population, our results are consistent with  
431 M. G. Baker, Peckham, and Seixas 2020. Recurrent outbreaks of SARS-CoV-2 are driven by the seasonality  
432 included in the model but also by the effect of waning natural and vaccine-derived immunity, such that even  
433 models without seasonal forcing and booster dose distribution may exhibit predictable outbreaks (figure 9).  
434 Rather than these outbreaks occurring at specific times throughout the year, they occur a certain amount  
435 of time after the previous outbreak—generally equal to the duration of complete immunity. At 6 month  
436 immunity, generally two outbreaks occur per year, spaced slightly more than 6 months apart, with periodic  
437 secondary outbreaks between; at one year of full immunity, large outbreaks occur slightly more than one  
438 year apart, without secondary outbreaks. These simulations indicate that preparations for outbreaks should  
439 include evaluation of the previous major outbreak.

440 We also explored the possibility of isolation as a means to control SARS-CoV-2, despite it being unlikely  
441 to be used as a general control strategy in the future. Isolation remains an effective way to limit the spread  
442 of SARS-CoV-2 in the steady state, however only the higher degrees of isolation—reducing non-household  
443 contacts among clinically infectious individuals to less than 50% of their normal amounts as compared to a  
444 ‘business as usual’ scenario—has a substantial effect on transmission dynamics (figure 10). At 50% isolation,  
445 an average of 2.72 clinical infections occur per capita, down from 4.84 when clinically infectious nodes have  
446 75% of their normal random contacts and 6.22 in the complete absence of isolation. With 50% isolation, the

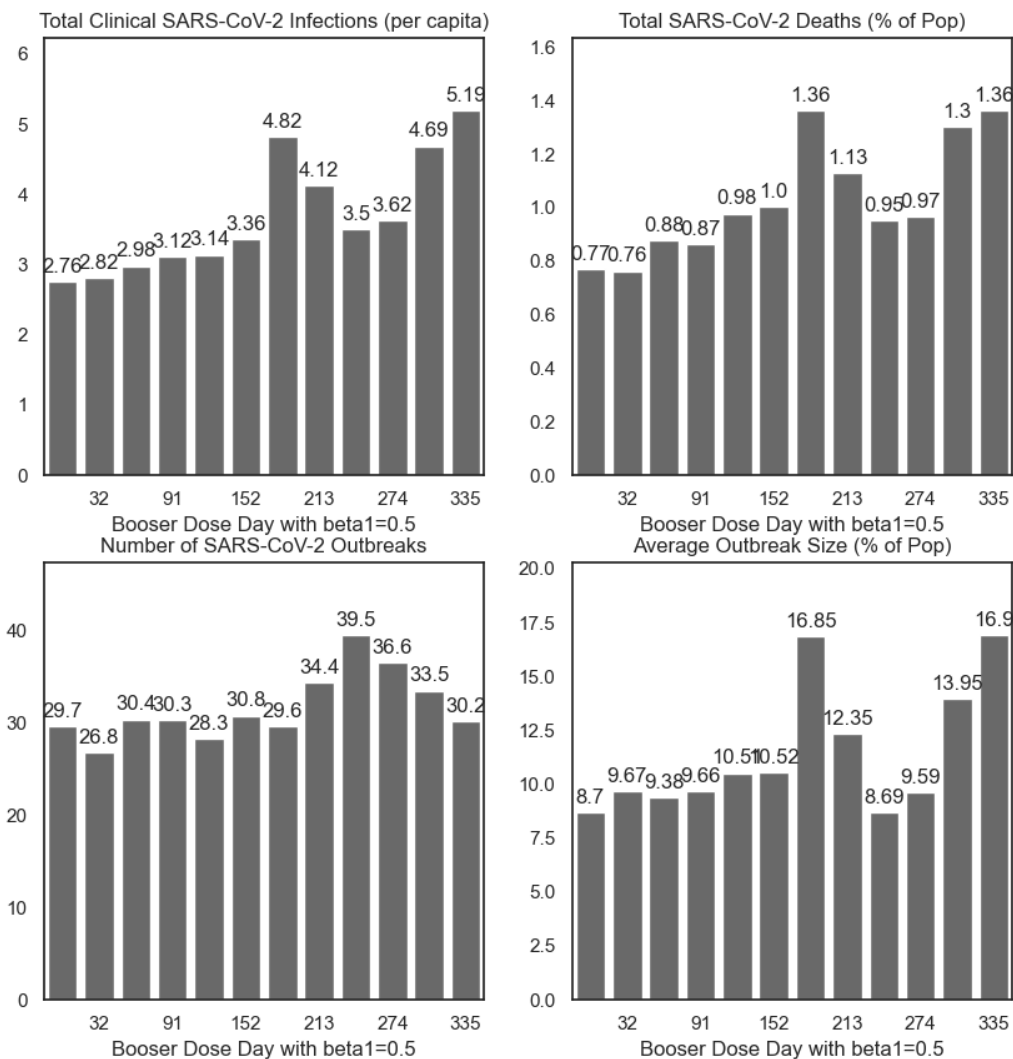


Figure 7: Summary of simulations by day of booster dose distribution, varied as the first of each month, in the presence of seasonal forcing of the transmission parameter  $\beta$ . Simulations are run for 10 years in replicates of 10.



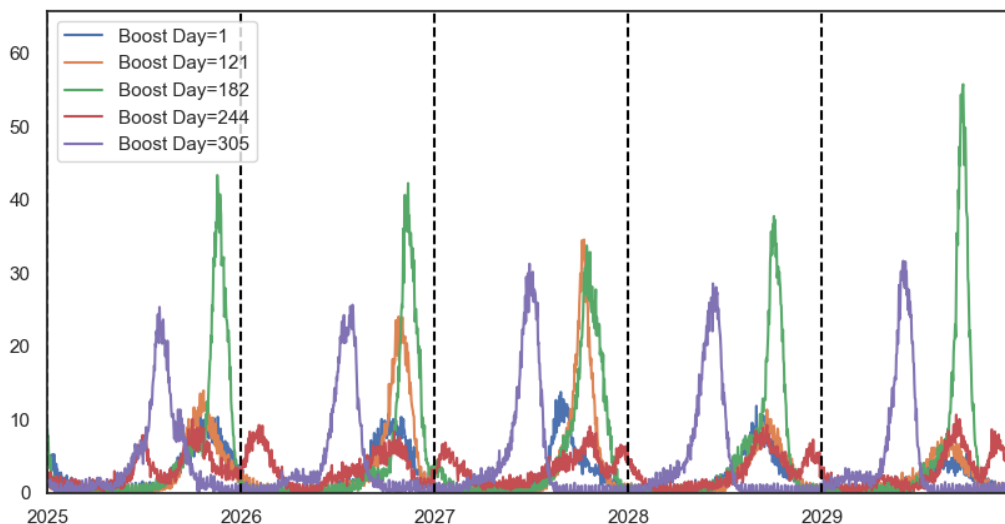


Figure 8: 5-year trajectory of simulations by day of booster dose distribution, for selected distribution days: Jan 1st (day 1), May 1st (day 121), July 1st (day 182), Sept 1st (day 244), and Nov 1st (day 305), in the presence of seasonal forcing of the transmission parameter  $\beta$ , averaged across 10 replications.

447 outbreak size drops dramatically to 6.05% of the population infected during an average of 40.8 outbreaks,  
448 down from from 18.7% of population infected in an average of 32.2 outbreaks in the absence of isolation.  
449 More extreme isolation reduces the severity of outbreaks even further: at 25% of normal contacts, clinical  
450 infections average one per capita; with perfect isolation, clinical infections are less than 0.5 per capita.

## 451 4 Discussion

452 Across all simulations, we observed frequent and predictable SARS-CoV-2 outbreaks over a 10-year period,  
453 even with the annual distribution of booster doses as the primary disease-averting intervention. Depend-  
454 ing on the epidemiology of the pathogen—namely, should SARS-CoV-2 exhibit seasonality in transmission  
455 probability and contact—outbreaks may occur at predictable times of the year, and distribution of booster  
456 doses may be able to mitigate the worst of seasonal outbreaks. Our results are consistent with R. E. Baker  
457 et al. 2020, who find that outbreak cycles are primarily determined by the levels of susceptibility in the  
458 population, although seasonality is an important moderator in outbreak dynamics. Different vaccination  
459 campaigns may be needed in areas that exhibit stronger transmission seasonality. We find that distributing  
460 booster doses in the first half of the year—January through May—may be an effective strategy at limiting  
461 recurrent outbreaks depending on the seasonality exhibited by the pathogen. We find that in simulations  
462 without seasonal forcing, distributing booster doses in the first half of the year is most effective at limiting  
463 outbreaks; however, with the inclusion of seasonal forcing of transmission and contact, distributing booster  
464 doses in September or October may limit the average outbreak size the most. In these simulations, distribut-  
465 ing booster doses in the late fall ‘shifts’ the outbreak to the summer, when transmissibility is lower. Since  
466 influenza vaccines are distributed in the fall, including booster doses for SARS-CoV-2 at the same time may  
467 be easiest in implementation, but less successful than Springtime distribution in limiting outbreaks should

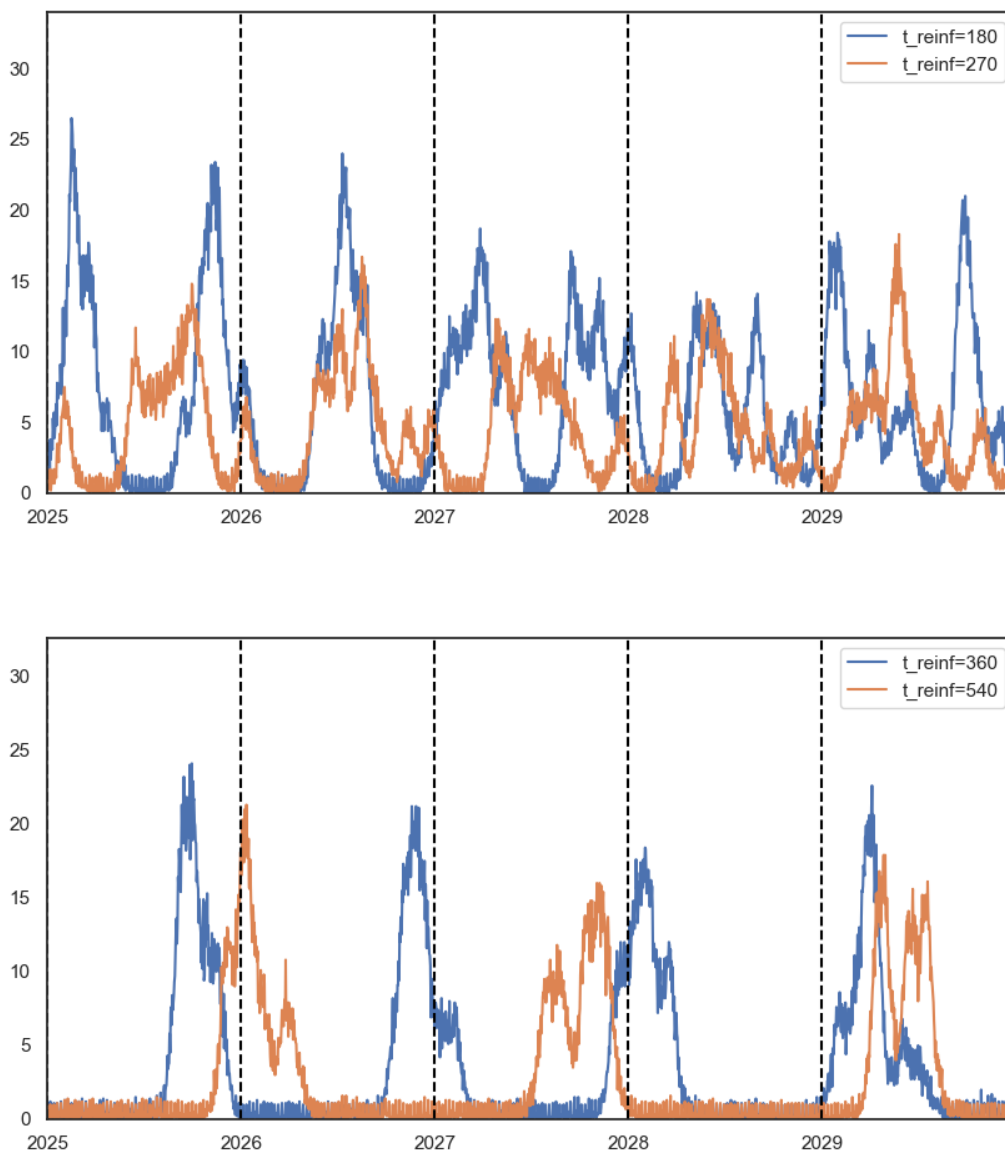


Figure 9: Trajectory of SARS-CoV-2 clinical infections with varied duration of complete immunity after infection and vaccination, illustrating how outbreak timing can be affected by immunity. Top: full immunity lasting for 180 days (6 months) and 270 days (9 months); bottom: full immunity lasting for 360 days (12 months) and 540 days (18 months). Averaged across 10 replications.

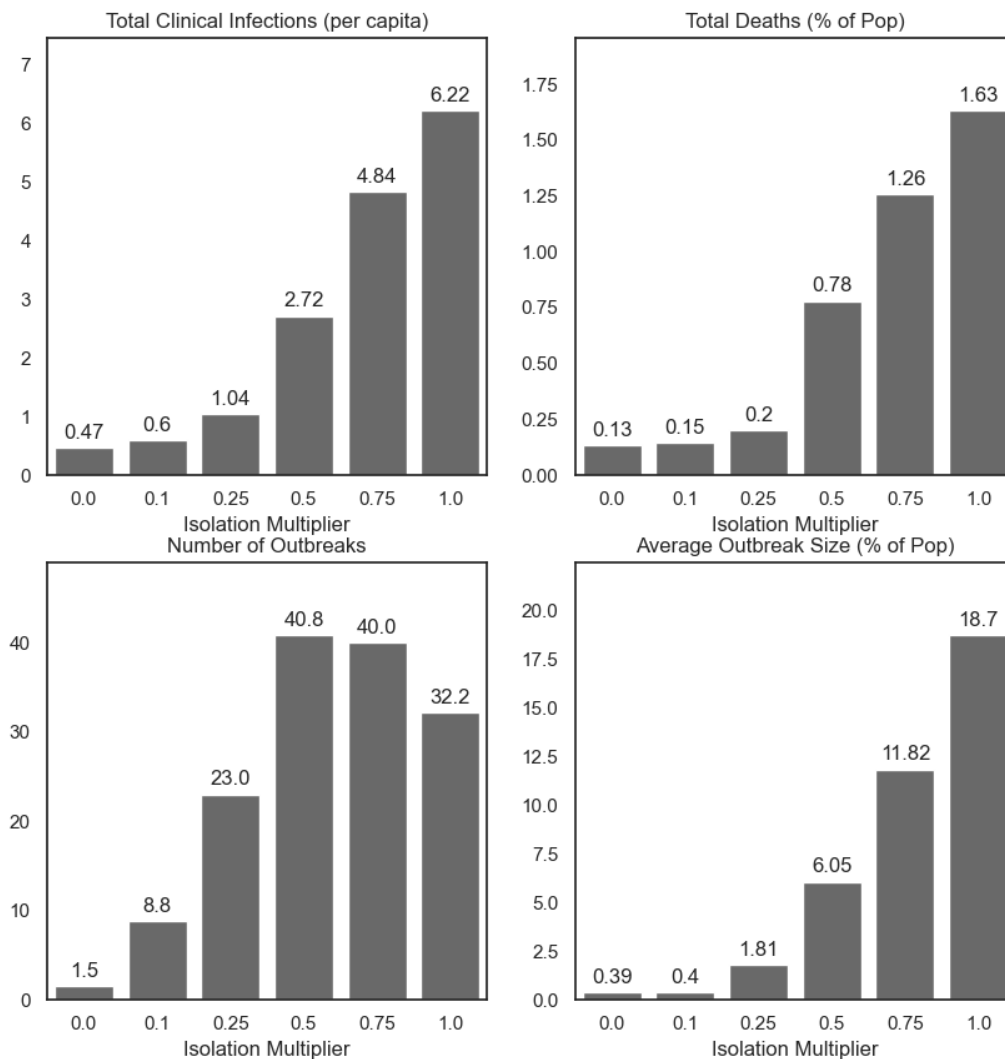


Figure 10: Summary of SARS-CoV-2 clinical infections, deaths, number of outbreaks, and average outbreak size for different levels of isolation, in the absence of seasonal forcing or vaccine distribution. Isolation multiplier is a factor used to scale a clinically infectious node's random, non-household contacts; 1.0 indicates business as usual and 0 indicates perfect isolation.

468 SARS-CoV-2 fail to exhibit transmission seasonality.

469 In addition to illustrating how vaccination interventions can avert the burden of illness and death due to  
470 SARS-CoV-2, our simulations further understanding of how to prepare for future SARS-CoV-2 outbreaks.  
471 Although distributing booster doses in early fall—like annual vaccinations for influenza—avoids a large  
472 wintertime outbreak in a seasonally forced environment, multiple smaller outbreaks may occur throughout  
473 the year. This may be preferable to avoid exceeding the treatment capacity of the health system. However,  
474 this strategy may not be effective in a less seasonally-variable climate, where booster dose timing merely  
475 shifts the main outbreak to later in the year after immunity wanes. Overall, we generally observe variance  
476 in mortality proportional to the number of clinical infections that occur in the simulations, indicating both  
477 that the most effective strategies for limiting clinical infections also limit deaths. Although we focus on the  
478 number of infections as the primary outcome, reducing the number of deaths due to SARS-CoV-2 may be  
479 possible with the interventional strategies outlined here.

480 While agent-based models have been used in a variety of applications for COVID-19, ours represents one  
481 of the first to examine long-term dynamics using real population contact data. Our results hint at a rather  
482 bleak outlook for the future of SARS-CoV-2: that outbreaks are extremely likely to continue given that  
483 natural and vaccine-derived immunity wanes over time. However, annual booster doses—especially when  
484 timed properly—and isolation of infectious cases may be effective control strategies. The investigation above  
485 tells us that outbreaks can be expected to occur more frequently than annually, and the epidemiology of  
486 the pathogen—namely, the base transmission probability and the duration of immunity after infection and  
487 vaccination—determines the frequency and severity of outbreaks more than vaccination timing and within a  
488 reasonable range of effectiveness. As our approach to SARS-CoV-2 shifts from eradication to management,  
489 the strategies presented here show how we can time the distribution of doses to minimize strain on the  
490 healthcare system and limit chronic complications from SARS-CoV-2 infections.

491 Our model has a number of shortcomings that limit its generalizability. First, we are limited by comput-  
492 ing resources to a population of 1000 households (approximately 3,200 individuals). During development,  
493 we found that using too few households resulted in simulations that were highly unstable; with a larger  
494 population, simulation results were more consistent between runs but took much longer to complete. As  
495 a result, we chose the number of households to balance numerical stability while maintaining a reasonable  
496 run time of approximately 4 minutes per simulation. Larger populations may exhibit different dynamics as  
497 the spread of infection may take longer; as such, it is not known presently if this chosen population size is  
498 representative of a larger population or is limited in generalizability to smaller communities. As well, like  
499 Roubenoff, Feehan, and Mahmud (2023), we borrow contact patterns for the youngest age group from the  
500 POLYMOD UK survey (Mossong et al. 2008), which may not be representative of that age group in the US  
501 during the COVID-19 pandemic. However, since our application here is in consideration of the long-term  
502 patterns of SARS-CoV-2, we believe that these contacts represent a return to ‘business as usual’ for school  
503 children.

504 A key feature of our model is in the random network generation, that produces daily draws of random  
505 contacts according to the network configuration model parameterized with degree and duration from the  
506 BICS survey. Like any random network generation, the network structure of contacts produced by this  
507 model may affect transmission dynamics. Future work should draw inspiration from the COVASIM agent  
508 based model (Kerr et al. 2021), which allows for comparison of outbreak dynamics between simulations  
509 with Poisson random networks, Hybrid networks, or SynthPops networks depending on the needs of the  
510 simulation and data available. As well, we do not include any assortative mixing as emphasized by the

511 model used by Roubenoff, Feehan, and Mahmud 2023 or serial (repeat) contacts, instead choosing a network  
512 generation function that maintains the degree of each node. Employment contacts, like school contacts for  
513 children under the age of 18, provide a consistent set of individuals having contact most days; inclusion of  
514 these such contacts may affect outbreak in unpredictable ways. Future development of the model should  
515 include associativity by age and employment status; however, doing so was outside of the scope of the present  
516 analysis.

## 517 **Implementation Overview**

518 The BICS ABM model is implemented in the C++ language and with use of the `iGraph-C` library (G. Csárdi  
519 and Nepusz, T. 2006; Gábor Csárdi et al. 2023), and a Python 3.8 API. The core C++ implementation was  
520 chosen over other languages, like Python or R, to maximize speed. Full implementation details are given in  
521 the supplementary material. Each simulation of 1000 households for 10 years completes in approximately  
522 4 minutes on an Apple MacBook Air M1, and the entire suite of simulations completes in approximately 8  
523 hours. Replication code is publicly available at [https://github.com/eroubenoff/BICS\\_ABM](https://github.com/eroubenoff/BICS_ABM).

## 524 5 Model Supplement

525 The model described here is a stochastic Agent-Based Network Simulation for COVID-19 transmission that  
 526 utilizes data collected as part of the Berkeley Interpersonal Contact Survey to simulation population structure  
 527 and contact networks. The core algorithm is written in C++, compiled using Apple Clang++17, and utilizes  
 528 CMake 3.16 and utilizes the `igraph` 10.4 library (G. Csárdi and Nepusz, T. 2006; Gábor Csárdi et al.  
 529 2023), and includes a Python 3.8 user API. The BICS ABM C++ library is compiled to a dynamic library  
 530 for linkage to the Python API and can be used with an external C++ program.

531 The BICS ABM C++ library hinges on two input data structures and their Python equivalents: `Params`,  
 532 a C-struct that contains the input parameters, documented in table 1; and an array of the simulation popu-  
 533 lation. The input population requires a strict format: each row is an individual and each column represents  
 534 individual-level data passed to the simulation, flattened to one dimensional array of floats in column-major  
 535 (Fortran-style) order. Currently, 8 fields are required for each simulated node: the node’s household id; age,  
 536 represented as a categorical index 0-8; gender, where 0 corresponds to male and 1 corresponds to female;  
 537 number of non-household contacts; number of school contacts, which is taken to be zero for adults; number  
 538 of times left home (unused presently, but maintained for legacy purposes); vaccine priority (see 5.1.5); and a  
 539 boolean indicating if the node uses NPIs or not. Parameters must be supplied indicating the dimensionality  
 540 of the dataset. The simulation returns a `trajectory`, a C-struct containing an hourly time-series of all  
 541 disease states.

542 The Python API contains a class `BICS_ABM`, which is a wrapper around all of the above utilizing the  
 543 `ctypes` library for cross-language functionality. Parameters can be passed to the model through the Python  
 544 API identically as to the C++ library and we recommend interacting with the simulation through the Python  
 545 API. The class constructor for `BICS_ABM` takes any of the arguments to `Params`, runs the simulation, and  
 546 saves as fields the components of the resultant trajectory as a `numpy.ndarray`; as such, the trajectory of  
 547 clinical cases can be accessed as `BICS_ABM.Cc`. The population can be accessed through `BICS_ABM._pop`.

Parameter Name	Description	Default Value
<code>N_HH</code>	Number of simulation households	1000
<code>WAVE</code>	(Python only) BICS survey wave used to derive simulation population	6
<code>GAMMA_MIN,MAX</code>	Bounds for uniformly-sampled duration of latent period	2,4
<code>SIGMA_MIN,MAX</code>	Bounds for uniformly-sampled duration of infectious period	4,6
<code>BETA_VEC</code>	Array of length 365 indicating the daily baseline probability of infection	[0.025, ... 0.025]
<code>BETA0, BETA1</code>	(Python only) Average and amplitude of sinusoidal seasonal forcing of the baseline transmission probability; transformed into <code>BETA_VEC</code>	0.025, 0
<code>CONTACT_MULT_VEC</code>	Array of length 365 indicating daily multiplier for number of random contacts	[1, ... ,1]

C1	(Python only) Amplitude of sinusoidal seasonal forcing of contact multiplier; transformed into CONTACT_MULT_VEC	0
SCHOOL_CONTACTS	Whether to include school contacts for children during weekdays during the school year	True
MU_VEC	Vector of length 9 indicating the age-specific SARS-CoV-2 mortality for each of the 9 age categories	See main text
INDEX_CASES	Number of index cases	5
IMPORT_CASES_VEC	Array of length 365 indicating the number of imported cases each day of the year; only begins after T0	One Weekly
SEED	Random seed	None
N_VAX_DAILY	Number of vaccines distributed daily	100
VE1,2,BOOST	Efficacy of vaccines after first, second, and booster doses	0.8, 0.9, 0.8
VEW	Efficacy of waned immunity for both vaccines and infectious-derived immunity	0.5
ISOLATION_MULTIPLIER	Scaling factor for random contacts by clinically infectious nodes; 0 is full isolation and 1 is business as usual	0.5
T_REINFECTION	Duration (in hours) of full vaccine and infectious-derived immunity before waning	24*180
T0	Date of appearance of index cases	0
ALPHA	Relative infectiousness of subclinical cases	0.32
RHO	Proportion of subclinical cases	0.20
MAX_DAYS	Duration of simulation, in days	10 * 365
BOOSTER_DAY	Day of year that booster doses are made available	244
FERTILITY_VEC	Array of length 9 indicating the age-category specific fertility rate for females, adjusted for age group bin width	See main text
Mortality_VEC	Array of length 9 indicating the age-category specific mortality rate, adjusted for age group bin width	See main text

Table 1: Parameters used in the ABM model

## 548 5.1 Model Pseudocode

549 Pseudocode for the model is given below:

- 550 1. In Python API: Establish the following parameters governing the population structure: number of  
551 households  $n_{hh}$ , survey wave, and vaccine priority. Establish all other transmission parameters and  
552 store them in an object `params`.

- 553 2. Generate  $n_{hh}$  households from the corresponding survey wave using the procedure described below.  
554 Assign each household a unique household identifier.
- 555 (a) First, the distribution of adult ages and sex is derived from ACS data, and a set of  $N_{HH}$   
556 households are sampled from this distribution.
- 557 (b) A ‘head’ of household is randomly chosen from the BICS survey data. A respondent is eligible  
558 to be head of household  $h_i$  if they are the corresponding age and sex for the sampled household  
559 head. Eligible household heads are chosen with replacement and with probability adjusted for  
560 survey weights.
- 561 (c) Finally, households are filled by sampling (with replacement and adjustment for survey weights)  
562 from the set of BICS respondents who match each of  $h_i$ ’s reported household members’ age,  
563 gender, and household size. Children under 18 are not ascertained in the survey; children are  
564 instead sampled from the POLYMOD survey. The max size of a household is 6 as respondents  
565 were only asked to report 6 of their household members.
- 566 3. Assign vaccine priority to all nodes in the network based off of the rules provided as input, as elaborated  
567 in section 5.1.5.
- 568 4. Create and pass the `params` and population object to the C++ core algorithm.
- 569 5. In C++ core: Determine the household contact network assuming that all nodes have contact with all  
570 members of their household. Randomly draw a school contact network for children under the age of  
571 18.
- 572 6. Repeat the following procedure representing one ‘day’ of simulation time, where each day contains  
573 24 ‘hours’ of simulation time, until either no nodes are exposed or infectious OR the simulation has  
574 occurred for a supplied maximum number of days.
- 575 (a) If hour == 0 (midnight) and an index case is supplied to appear on the current simulation day,  
576 transition one node at random into ‘Exposed’ status.
- 577 (b) Each hour between midnight and 8am: assume that all nodes have contact with all members of  
578 their household. Execute `transmission` and `decrement` procedures for all nodes.
- 579 (c) 8am: Distribute vaccine doses to `n_vax` nodes awaiting any dose of the vaccine.
- 580 (d) 8am: generate a random graph of daily contacts using the procedure outlined in section 5.1.3;  
581 assign a random duration and start time for all contacts.
- 582 (e) Each hour between 8am and 6pm: connect all random contacts; disconnect each node having a  
583 random contact from their household nodes; `transmit`; and `decrement`. Reconnect nodes after  
584 termination of random contact.
- 585 (f) Each hour between 6pm and midnight: transmit within households assuming that all nodes have  
586 contact with all members of their household. Execute `transmit` and `decrement` procedures.
- 587 7. At the conclusion of the simulation, return trajectories of each disease and vaccination status.



### 588 **5.1.1 Update Handlers**

589 The C++ program contains a centralized method for handling and dispatching changes to nodes, edges,  
590 and the graph itself. Classes exist for five types of changes can be executed: `UpdateGraphAttribute`,  
591 `CreateEdge`, `DeleteEdge`, `UpdateEdgeAttribute`, and `UpdateVertexAttribute`. Vertices cannot be created  
592 or destroyed using the update handler. All updates are stored in wrapper class `UpdateList`, which contains an  
593 overloaded method `UpdateList.add_update()` to add an update of each type. Updates are then dispatched  
594 with the method `UpdateList.add_updates_to_graph(igraph_t*)`, which takes a reference to the graph  
595 object as an argument to perform the updates.

596 The centralized update handlers were developed to streamline the attribute interface. Although the  
597 `igraph` API contains methods for updating individual node or edge attributes, it is more computationally  
598 efficient to pull all of the attributes, make all changes, then push them back to the graph. Since the `igraph`  
599 API involves many dynamically-allocated objects, this meant keeping track of many pointers, being sure to  
600 free all used vectors of attributes. With a project of this size, adding a centralized way of dispatching updates  
601 helped with debugging many issues with memory management, at the cost of a slight function overhead.  
602 As well, the object-oriented interface allows for saving of all, say, household edges in a single object to be  
603 connected and disconnected as needed.

### 604 **5.1.2 Decrement procedure**

605 The `decrement` procedure is among the most important function in the C++ core for tracking the progression  
606 of nodes through simulation time. Each ‘event’ that can occur to a node (infection, development of clinical  
607 or subclinical infectiousness, recovery, waning immunity, becoming vaccinated, etc) is accompanied by a  
608 duration of the event. The `decrement` procedure decrements the time remaining at each status, and for  
609 some events (for example, recovery from infectiousness) will automatically trigger a status change; for other  
610 events (like eligibility for vaccination), eligible nodes are placed in a queue for the next event to occur.

### 611 **5.1.3 Random contacts**

612 Random contacts are drawn for the daytime hours, 8am-6pm, using the network configuration model (as  
613 described in the main text; Albert-László Barabási 2021). First, the number of daily-nonhousehold contacts  
614 is supplied for each simulation node; this number is first multiplied by the isolation multiplier if the node is  
615 clinically infectious and contact multiplier if included in the model, then taken to be a random draw from a  
616 Poisson distribution with rate parameter equal to the product of all three terms. This is done to allow for  
617 minor stochasticity in the model. Each node is assigned a number of stubs equal to this Poisson random  
618 draw; if the total number of stubs subs to an odd number, one stub is randomly deleted until the sum is an  
619 even number. Should this sum be zero stubs then the procedure aborts. The configuration model is drawn  
620 using the `igraph.degree_sequence_game` from the `igraph` library; the graph is then simplified to remove  
621 self-edges but not multi-edges.

### 622 **5.1.4 Non-Pharmaceutical Interventions**

623 We include a single generic Non-Pharmaceutical Intervention (NPI) intended to capture the combined  
624 transmission-preventing power of mask usage, gloves, and physical distancing. In wave 6, about 60% BICS  
625 respondents reported the usage of any of these possible NPIs in any of their reported contacts; any simula-  
626 tion nodes representing these respondents are given an NPI status of `True`. During the daytime simulation

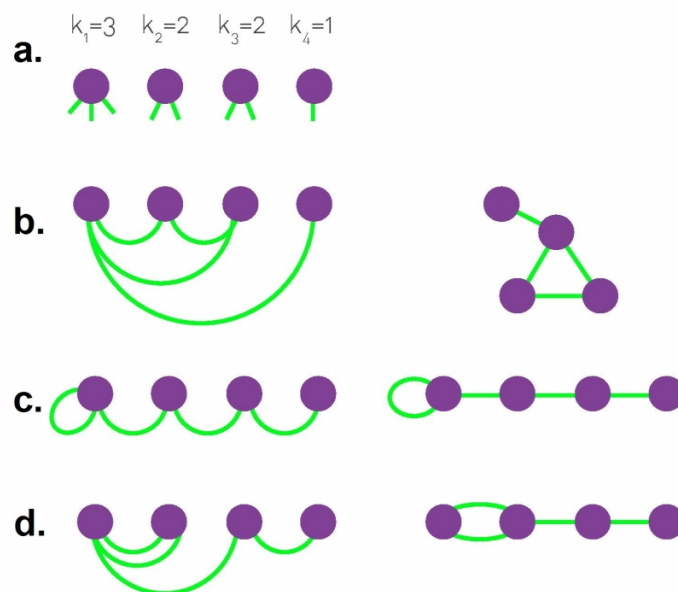


Figure 11: Albert-László Barabási 2021's diagram representing realizations of the Network Configuration Model showing multiple ways of connecting the four nodes in panel (a) with corresponding degree  $k$ . (b): no self- or multi-edges; (c): allowing self-edges but not multi-edges; (d): allowing multi-edges, but not self-edges. Our application would allow for configuration (d) but not (c).

627 procedure, if two non-household nodes are connected who both have an NPI status of `True`, then their  
 628 transmission probability is lowered by a supplied parameter representing the strength of these NPIs (see  
 629 below).

### 630 5.1.5 Vaccine Distribution

631 Nodes in the population are assigned a discrete priority level for vaccination before the simulation begins.  
 632 A set number of vaccines are distributed daily among nodes with the highest priority level until no nodes  
 633 in that priority level remain; remaining doses are distributed among nodes with the next highest priority  
 634 level, repeating until the day's number of vaccines are exhausted. A priority level of  $-1$  indicates that the  
 635 node declines or is ineligible for vaccination. Nodes are eligible to receive the second dose of the vaccine 25  
 636 days after they received first dose. After a period of time, vaccine efficacy is assumed to wane; at this point,  
 637 nodes are eligible to receive an additional booster dose.

638 **5.1.6 Demographic Rates**

639 Fertility and all-cause mortality are incorporated in the simulation according to published age-specific rates,  
640 aggregated for each age group in the simulation:

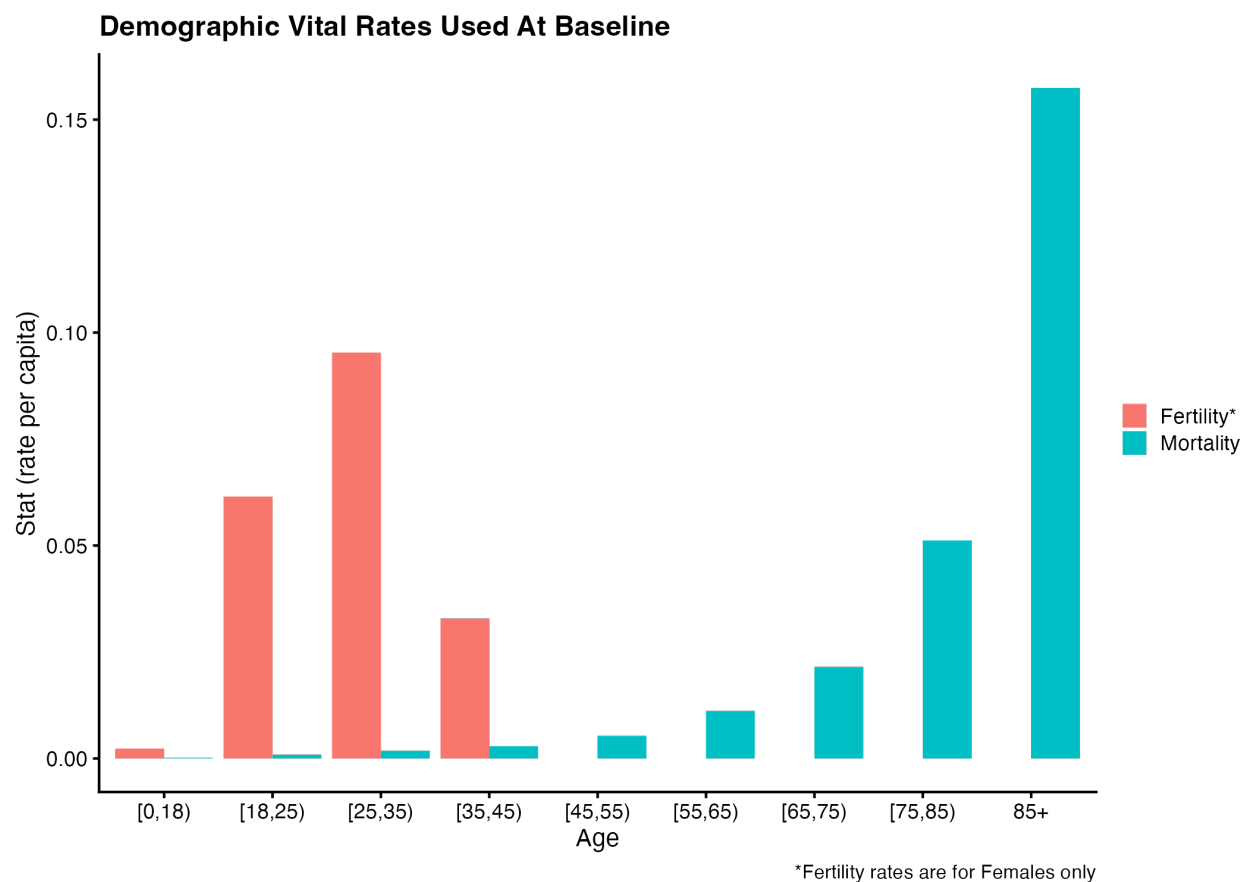


Figure 12: Baseline demographic vital rates used in the simulation.

641 **5.2 Supplementary Figures**

642 **5.2.1 Booster Dose Distribution Day, High Uptake**

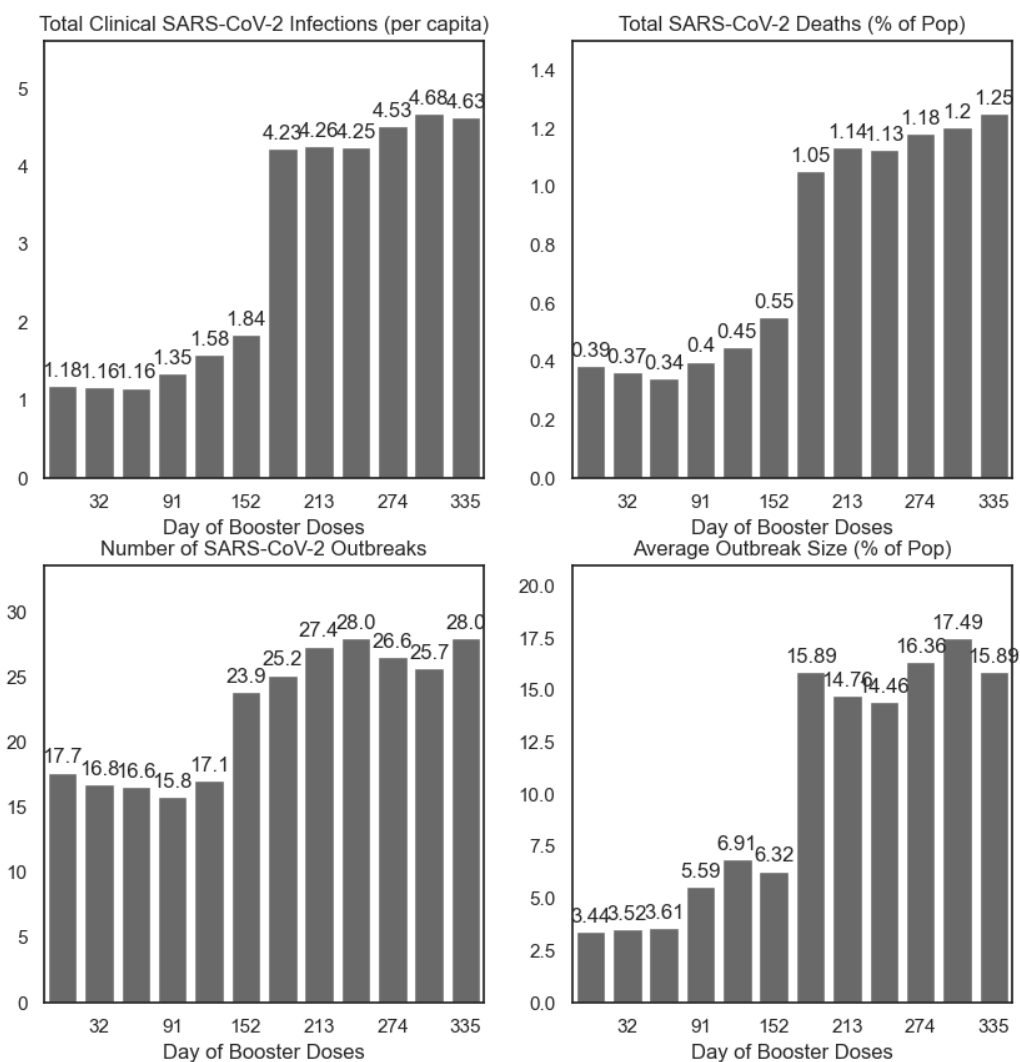


Figure 13: Summary of simulations by day of booster dose distribution, varied as the first of each month, in the absence of seasonal forcing of the transmission parameter  $\beta$ , with 90% vaccine uptake.

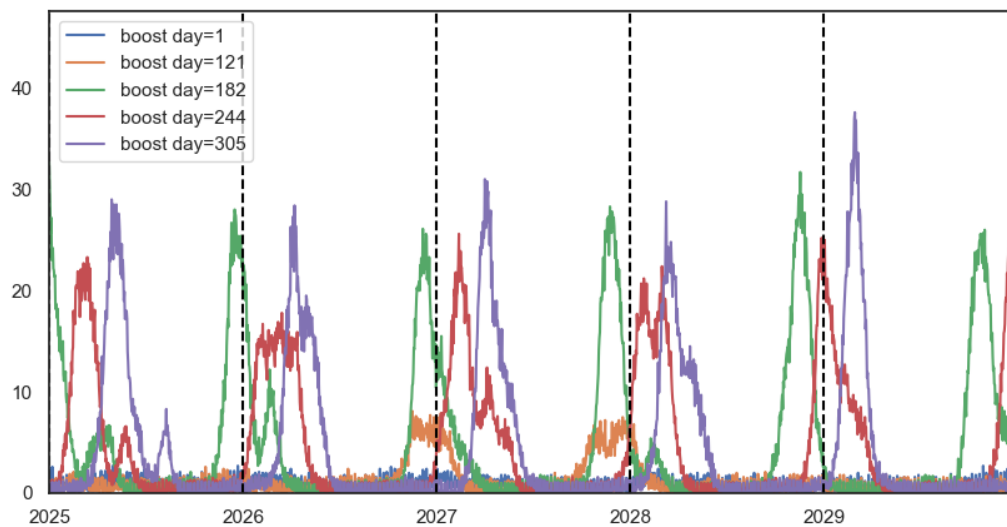


Figure 14: Trajectory of simulations by day of booster dose distribution, without seasonal forcing, for selected distribution days: Jan 1st (day 1), May 1st (day 121), July 1st (day 182), Sept 1st (day 244), and Nov 1st (day 305), with 90% uptake.

643 **5.2.2 Seasonality in contact rates**

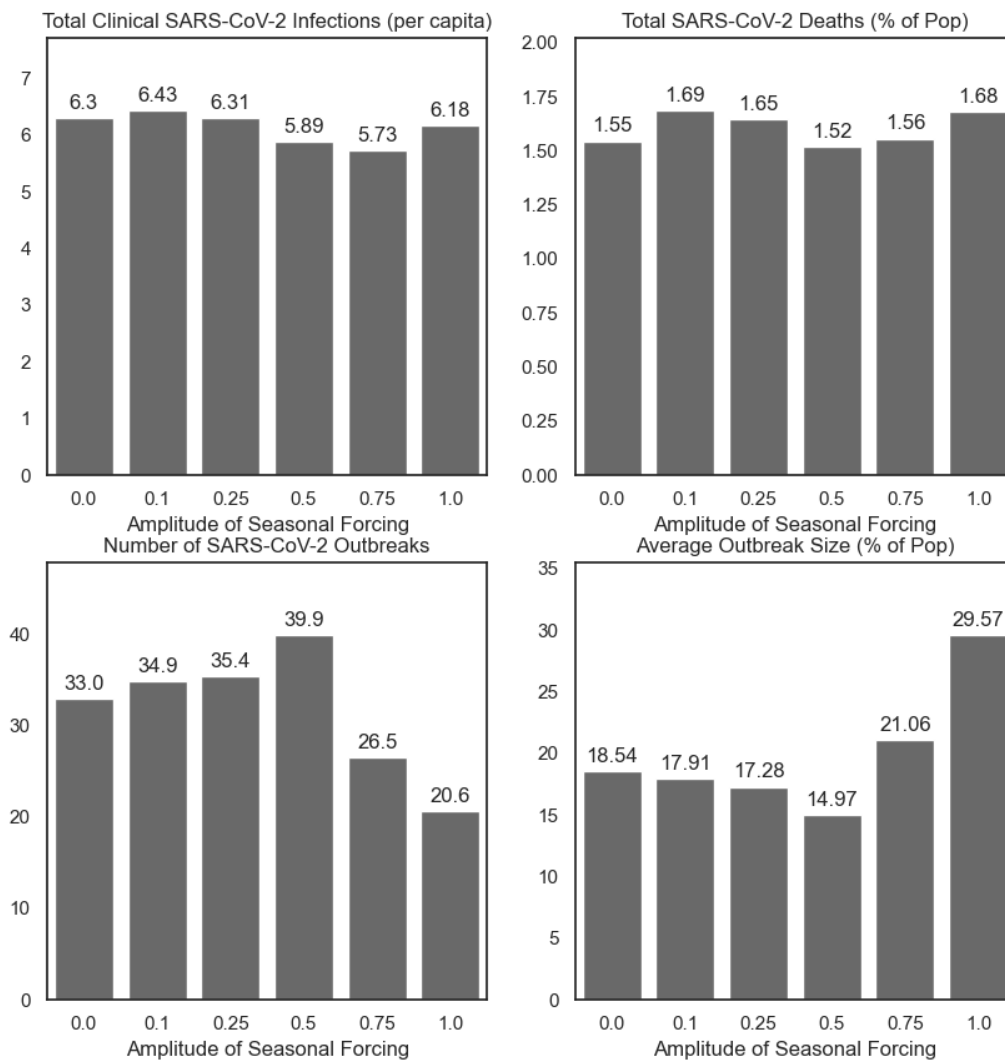


Figure 15: Summary of simulations at selected levels of  $c_1$ , the amplitude of seasonal forcing of the contact parameter.

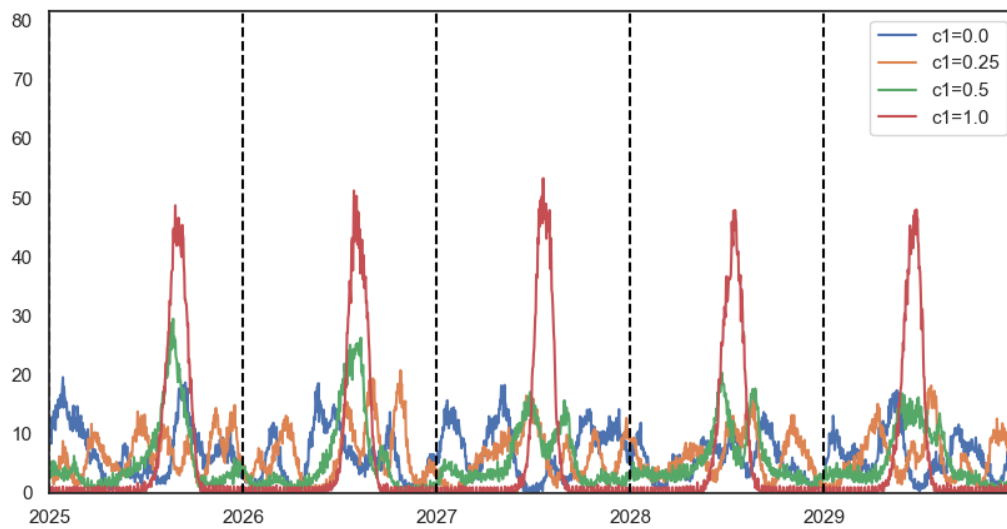


Figure 16: Trajectories at selected levels of  $c_1$ , the amplitude of seasonal forcing of the contact parameter.

644 **5.2.3 Isolation of infectious cases**

Average Outbreaks by Month

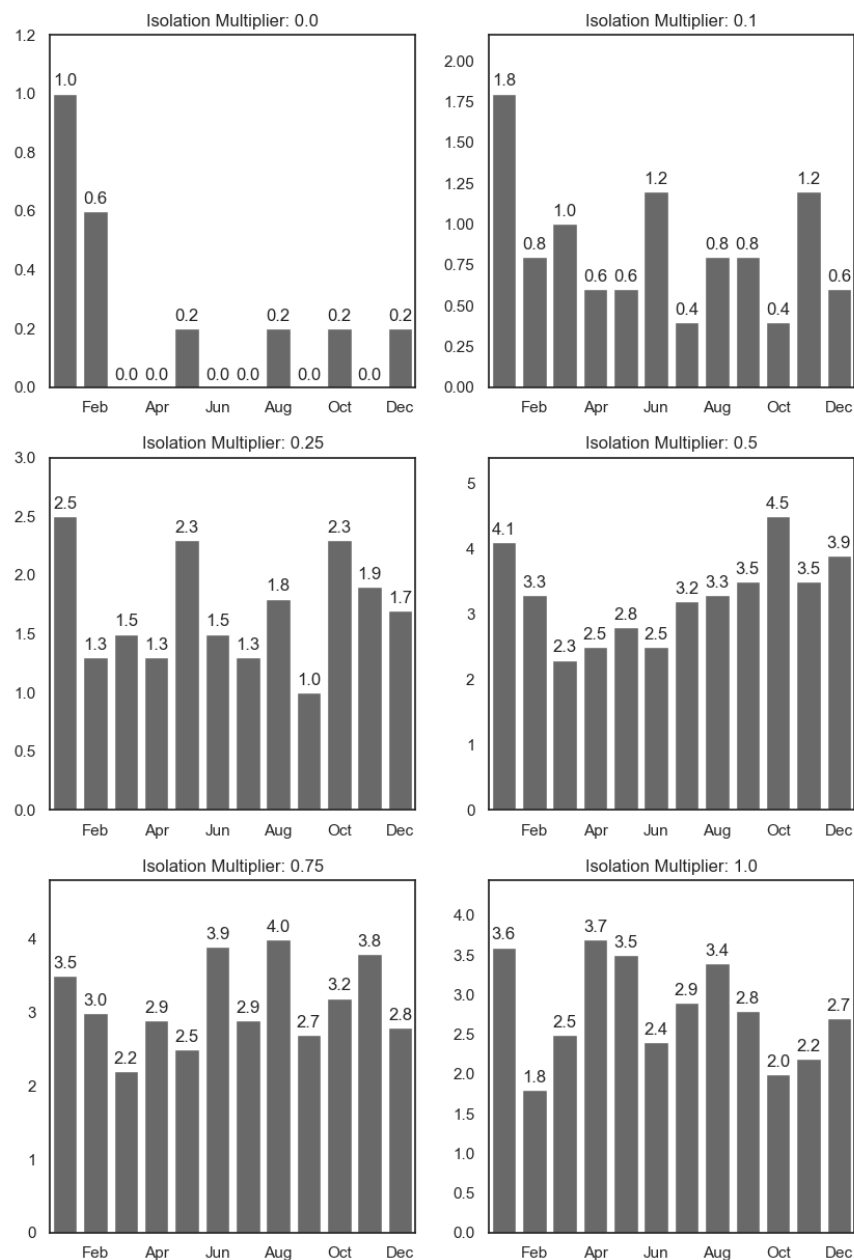


Figure 17: Outbreak seasonality at selected levels of isolation



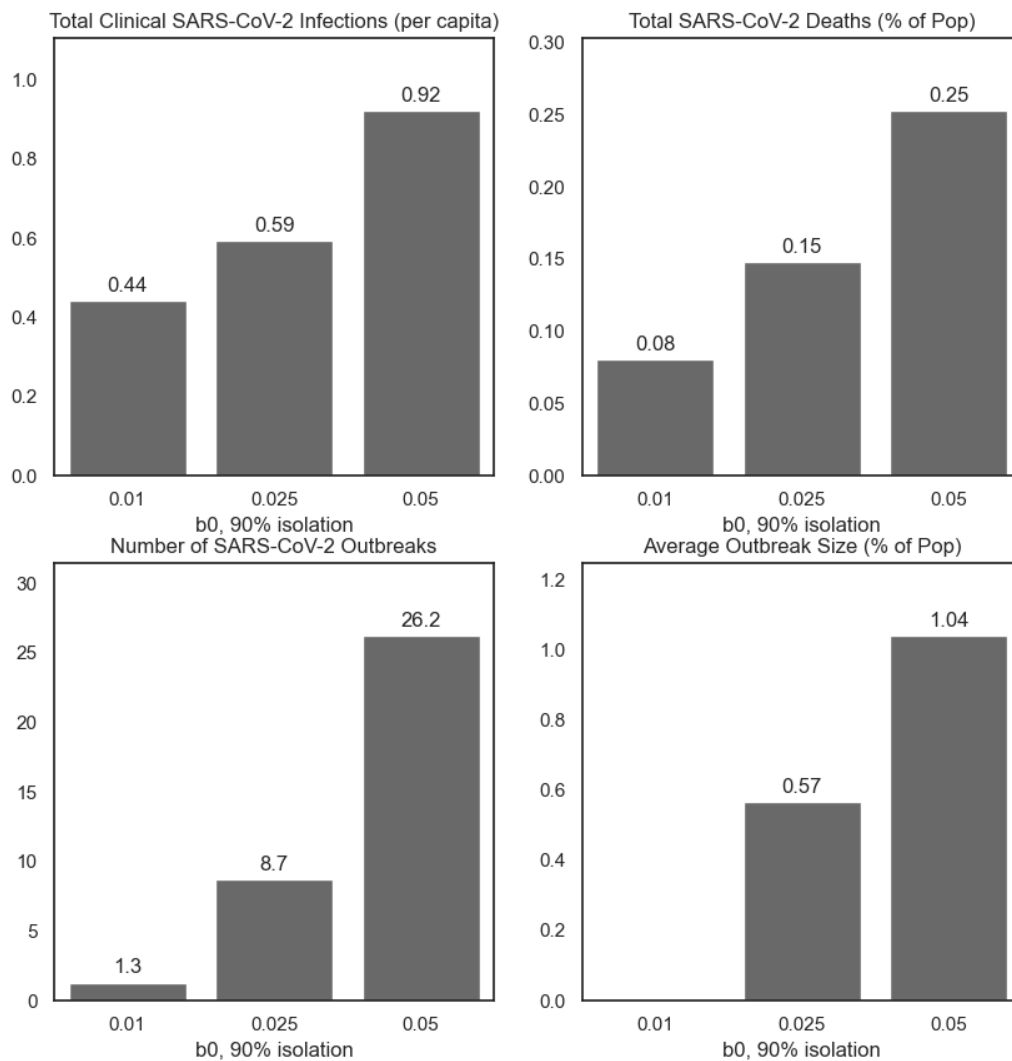


Figure 18: Summary of simulations at selected levels of  $\beta_0$  with 90% isolation of clinically infectious cases (corresponding to an isolation multiplier of 10%).

## References

- 645
- 646 Ajelli, Marco et al. (June 2010). “Comparing large-scale computational approaches to epidemic modeling:  
647 Agent-based versus structured metapopulation models”. In: *BMC Infectious Diseases* 10.1, p. 190. ISSN:  
648 1471-2334. DOI: 10.1186/1471-2334-10-190. URL: <https://doi.org/10.1186/1471-2334-10-190>  
649 (visited on 09/01/2021).
- 650 Albert-László Barabási (2021). *Network Science by Albert-László Barabási*. URL: [http://networksciencebook.](http://networksciencebook.com/)  
651 [com/](http://networksciencebook.com/) (visited on 10/06/2021).
- 652 Altizer, Sonia et al. (Apr. 2006). “Seasonality and the dynamics of infectious diseases”. eng. In: *Ecology*  
653 *Letters* 9.4, pp. 467–484. ISSN: 1461-0248. DOI: 10.1111/j.1461-0248.2005.00879.x.
- 654 Arduin, Hélène et al. (June 2017). “An agent-based model simulation of influenza interactions at the host  
655 level: insight into the influenza-related burden of pneumococcal infections”. en. In: *BMC Infectious Dis-*  
656 *eases* 17.1, p. 382. ISSN: 1471-2334. DOI: 10.1186/s12879-017-2464-z. URL: [https://doi.org/10.](https://doi.org/10.1186/s12879-017-2464-z)  
657 [1186/s12879-017-2464-z](https://doi.org/10.1186/s12879-017-2464-z) (visited on 06/05/2023).
- 658 Baker, Jack D. et al. (Dec. 2013). “An evaluation of the accuracy of small-area demographic estimates of  
659 population at risk and its effect on prevalence statistics”. In: *Population Health Metrics* 11.1, p. 24. ISSN:  
660 1478-7954. DOI: 10.1186/1478-7954-11-24. URL: <https://doi.org/10.1186/1478-7954-11-24>  
661 (visited on 04/01/2020).
- 662 Baker, Marissa G., Trevor K. Peckham, and Noah S. Seixas (2020). “Estimating the burden of United States  
663 workers exposed to infection or disease: A key factor in containing risk of COVID-19 infection”. eng. In:  
664 *PloS One* 15.4, e0232452. ISSN: 1932-6203. DOI: 10.1371/journal.pone.0232452.
- 665 Baker, Rachel E. et al. (July 2020). “Susceptible supply limits the role of climate in the early SARS-CoV-  
666 2 pandemic”. In: *Science* 369.6501. Publisher: American Association for the Advancement of Science,  
667 pp. 315–319. DOI: 10.1126/science.abc2535. URL: [https://www.science.org/doi/10.1126/](https://www.science.org/doi/10.1126/science.abc2535)  
668 [science.abc2535](https://www.science.org/doi/10.1126/science.abc2535) (visited on 08/01/2023).
- 669 Bansal, Shweta, Bryan T Grenfell, and Lauren Ancel Meyers (Oct. 2007). “When individual behaviour  
670 matters: homogeneous and network models in epidemiology”. In: *Journal of the Royal Society Interface*  
671 4.16, pp. 879–891. ISSN: 1742-5689. DOI: 10.1098/rsif.2007.1100. URL: [https://www.ncbi.nlm.nih.](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2394553/)  
672 [gov/pmc/articles/PMC2394553/](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2394553/) (visited on 04/01/2020).
- 673 Bansal, Shweta, Jonathan Read, et al. (Sept. 2010). “The dynamic nature of contact networks in infec-  
674 tious disease epidemiology”. In: *Journal of Biological Dynamics* 4.5. Publisher: Taylor & Francis eprint:  
675 <https://doi.org/10.1080/17513758.2010.503376>, pp. 478–489. ISSN: 1751-3758. DOI: 10.1080/17513758.  
676 2010.503376. URL: <https://doi.org/10.1080/17513758.2010.503376> (visited on 08/01/2023).
- 677 Bolker, BM and BT Grenfell (Jan. 1993). “Chaos and biological complexity in measles dynamics”. en. In:  
678 *Proceedings of the Royal Society of London. Series B: Biological Sciences* 251.1330, pp. 75–81. ISSN: 0962-  
679 8452, 1471-2954. DOI: 10.1098/rspb.1993.0011. URL: [https://royalsocietypublishing.org/doi/](https://royalsocietypublishing.org/doi/10.1098/rspb.1993.0011)  
680 [10.1098/rspb.1993.0011](https://royalsocietypublishing.org/doi/10.1098/rspb.1993.0011) (visited on 06/05/2023).
- 681 Bonabeau, Eric (May 2002). “Agent-based modeling: Methods and techniques for simulating human sys-  
682 tems”. en. In: *Proceedings of the National Academy of Sciences* 99.suppl 3. Publisher: National Academy  
683 of Sciences Section: Colloquium Paper, pp. 7280–7287. ISSN: 0027-8424, 1091-6490. DOI: 10.1073/pnas.  
684 082080899. URL: [https://www.pnas.org/content/99/suppl\\_3/7280](https://www.pnas.org/content/99/suppl_3/7280) (visited on 09/01/2021).
- 685 Bruch, Elizabeth and Jon Atwell (May 2015). “Agent-Based Models in Empirical Social Research”. In:  
686 *Sociological Methods & Research* 44.2. Publisher: SAGE Publications Inc, pp. 186–221. ISSN: 0049-1241.

- 687 DOI: 10.1177/0049124113506405. URL: <https://doi.org/10.1177/0049124113506405> (visited on  
688 09/01/2021).
- 689 Buckee, Caroline O., Andrew J. Tatem, and C. Jessica E. Metcalf (Jan. 2017). “Seasonal Population Move-  
690 ments and the Surveillance and Control of Infectious Diseases”. en. In: *Trends in Parasitology* 33.1,  
691 pp. 10–20. ISSN: 1471-4922. DOI: 10.1016/j.pt.2016.10.006. URL: [https://www.sciencedirect.com/  
692 science/article/pii/S1471492216301891](https://www.sciencedirect.com/science/article/pii/S1471492216301891) (visited on 06/05/2023).
- 693 Buitrago-Garcia, Diana et al. (May 2022). “Occurrence and transmission potential of asymptomatic and  
694 presymptomatic SARS-CoV-2 infections: Update of a living systematic review and meta-analysis”. en.  
695 In: *PLOS Medicine* 19.5. Publisher: Public Library of Science, e1003987. ISSN: 1549-1676. DOI: 10.1371/  
696 journal.pmed.1003987. URL: [https://journals.plos.org/plosmedicine/article?id=10.1371/  
697 journal.pmed.1003987](https://journals.plos.org/plosmedicine/article?id=10.1371/journal.pmed.1003987) (visited on 07/04/2023).
- 698 Buonomo, B., N. Chitnis, and A. d’Onofrio (June 2018). “Seasonality in epidemic models: a literature review”.  
699 en. In: *Ricerche di Matematica* 67.1, pp. 7–25. ISSN: 1827-3491. DOI: 10.1007/s11587-017-0348-6. URL:  
700 <https://doi.org/10.1007/s11587-017-0348-6> (visited on 05/22/2023).
- 701 Centers for Disease Control and Prevention (Oct. 2021). *Science Brief: SARS-CoV-2 Infection-induced and*  
702 *Vaccine-induced Immunity*. en-us. URL: [https://www.cdc.gov/coronavirus/2019-ncov/science/  
703 science-briefs/vaccine-induced-immunity.html](https://www.cdc.gov/coronavirus/2019-ncov/science/science-briefs/vaccine-induced-immunity.html) (visited on 07/04/2023).
- 704 Chin, Alex W H et al. (May 2020). “Stability of SARS-CoV-2 in different environmental conditions”. en.  
705 In: *The Lancet Microbe* 1.1, e10. ISSN: 2666-5247. DOI: 10.1016/S2666-5247(20)30003-3. URL: [https:  
706 //www.sciencedirect.com/science/article/pii/S2666524720300033](https://www.sciencedirect.com/science/article/pii/S2666524720300033) (visited on 06/05/2023).
- 707 Clarke, Kristie E. N. (2022). “Seroprevalence of Infection-Induced SARS-CoV-2 Antibodies — United States,  
708 September 2021–February 2022”. en-us. In: *MMWR. Morbidity and Mortality Weekly Report* 71. ISSN:  
709 0149-21951545-861X. DOI: 10.15585/mmwr.mm7117e3. URL: [https://www.cdc.gov/mmwr/volumes/71/  
710 wr/mm7117e3.htm](https://www.cdc.gov/mmwr/volumes/71/wr/mm7117e3.htm) (visited on 02/27/2023).
- 711 Crane, Matthew A. et al. (Mar. 2021). “Change in Reported Adherence to Nonpharmaceutical Interventions  
712 During the COVID-19 Pandemic, April–November 2020”. In: *JAMA* 325.9, pp. 883–885. ISSN: 0098-7484.  
713 DOI: 10.1001/jama.2021.0286. URL: <https://doi.org/10.1001/jama.2021.0286> (visited on  
714 02/27/2023).
- 715 Csárdi, G. and Nepusz, T. (2006). “The igraph software package for complex network research”. In: *Inter-  
716 Journal Complex Systems*, p. 1695. URL: <https://cir.nii.ac.jp/crid/1370294643763138711> (visited  
717 on 07/06/2023).
- 718 Csárdi, Gábor et al. (June 2023). *igraph*. DOI: 10.5281/ZENODO.3630268. URL: [https://zenodo.org/  
719 record/3630268](https://zenodo.org/record/3630268) (visited on 07/06/2023).
- 720 Cuevas, Erik (June 2020). “An agent-based model to evaluate the COVID-19 transmission risks in facilities”.  
721 en. In: *Computers in Biology and Medicine* 121, p. 103827. ISSN: 0010-4825. DOI: 10.1016/j.compbiomed.  
722 2020.103827. URL: [https://www.sciencedirect.com/science/article/pii/S001048252030192X  
723](https://www.sciencedirect.com/science/article/pii/S001048252030192X) (visited on 11/16/2022).
- 724 Dabisch, Paul et al. (Feb. 2021). “The influence of temperature, humidity, and simulated sunlight on the  
725 infectivity of SARS-CoV-2 in aerosols”. In: *Aerosol Science and Technology* 55.2. Publisher: Taylor &  
726 Francis \_eprint: <https://doi.org/10.1080/02786826.2020.1829536>, pp. 142–153. ISSN: 0278-6826. DOI: 10.  
727 1080/02786826.2020.1829536. URL: <https://doi.org/10.1080/02786826.2020.1829536> (visited on  
728 06/05/2023).

- 729 Damette, Olivier, Clément Mathonnat, and Stéphane Goutte (June 2021). “Meteorological factors against  
730 COVID-19 and the role of human mobility”. en. In: *PLOS ONE* 16.6. Publisher: Public Library of Science,  
731 e0252405. ISSN: 1932-6203. DOI: 10.1371/journal.pone.0252405. URL: [https://journals.plos.org/  
732 plosone/article?id=10.1371/journal.pone.0252405](https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0252405) (visited on 06/05/2023).
- 733 Danon, Leon et al. (Mar. 2011). “Networks and the Epidemiology of Infectious Disease”. en. In: *Interdis-*  
734 *ciplinary Perspectives on Infectious Diseases* 2011. Publisher: Hindawi, e284909. ISSN: 1687-708X. DOI:  
735 10.1155/2011/284909. URL: <https://www.hindawi.com/journals/ipid/2011/284909/> (visited on  
736 09/01/2021).
- 737 Davies, Nicholas G. et al. (Apr. 2021). “Estimated transmissibility and impact of SARS-CoV-2 lineage  
738 B.1.1.7 in England”. en. In: *Science* 372.6538. Publisher: American Association for the Advancement of  
739 Science Section: Research Article. ISSN: 0036-8075, 1095-9203. DOI: 10.1126/science.abg3055. URL:  
740 <https://science.sciencemag.org/content/372/6538/eabg3055> (visited on 05/05/2021).
- 741 Dawson, Daniel E. et al. (Oct. 2018). “Investigating behavioral drivers of seasonal Shiga-Toxigenic Escherichia  
742 Coli (STEC) patterns in grazing cattle using an agent-based model”. en. In: *PLOS ONE* 13.10. Publisher:  
743 Public Library of Science, e0205418. ISSN: 1932-6203. DOI: 10.1371/journal.pone.0205418. URL:  
744 <https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0205418> (visited on  
745 06/05/2023).
- 746 Dowell, S F (2001). “Seasonal variation in host susceptibility and cycles of certain infectious diseases.” In:  
747 *Emerging Infectious Diseases* 7.3, pp. 369–374. ISSN: 1080-6040. URL: [https://www.ncbi.nlm.nih.gov/  
748 pmc/articles/PMC2631809/](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2631809/) (visited on 05/22/2023).
- 749 Feehan, Dennis M. and Ayesha S. Mahmud (Feb. 2021). “Quantifying population contact patterns in the  
750 United States during the COVID-19 pandemic”. en. In: *Nature Communications* 12.1. Number: 1 Pub-  
751 lisher: Nature Publishing Group, p. 893. ISSN: 2041-1723. DOI: 10.1038/s41467-021-20990-2. URL:  
752 <https://www.nature.com/articles/s41467-021-20990-2> (visited on 05/06/2021).
- 753 Finkenstädt, B. F. and B. T. Grenfell (2000). “Time series modelling of childhood diseases: a dynamical  
754 systems approach”. en. In: *Journal of the Royal Statistical Society: Series C (Applied Statistics)* 49.2.  
755 eprint: <https://rss.onlinelibrary.wiley.com/doi/pdf/10.1111/1467-9876.00187>, pp. 187–205. ISSN: 1467-  
756 9876. DOI: 10.1111/1467-9876.00187. URL: [https://rss.onlinelibrary.wiley.com/doi/abs/10.  
757 1111/1467-9876.00187](https://rss.onlinelibrary.wiley.com/doi/abs/10.1111/1467-9876.00187) (visited on 06/15/2020).
- 758 Fisman, D. (Oct. 2012). “Seasonality of viral infections: mechanisms and unknowns”. en. In: *Clinical Micro-*  
759 *biology and Infection* 18.10, pp. 946–954. ISSN: 1198-743X. DOI: 10.1111/j.1469-0691.2012.03968.x.  
760 URL: <https://www.sciencedirect.com/science/article/pii/S1198743X14610910> (visited on  
761 06/28/2023).
- 762 Gomez, Jonatan et al. (Feb. 2021). “INFEKTA—An agent-based model for transmission of infectious diseases:  
763 The COVID-19 case in Bogotá, Colombia”. en. In: *PLOS ONE* 16.2. Publisher: Public Library of Science,  
764 e0245787. ISSN: 1932-6203. DOI: 10.1371/journal.pone.0245787. URL: [https://journals.plos.org/  
765 plosone/article?id=10.1371/journal.pone.0245787](https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0245787) (visited on 09/01/2021).
- 766 Grassly, Nicholas C and Christophe Fraser (Oct. 2006). “Seasonal infectious disease epidemiology”. In: *Pro-*  
767 *ceedings of the Royal Society B: Biological Sciences* 273.1600, pp. 2541–2550. ISSN: 0962-8452. DOI:  
768 10.1098/rspb.2006.3604. URL: [https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1634916/  
769](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1634916/) (visited on 06/15/2020).
- 770 He, Daihai, Edward L. Ionides, and Aaron A. King (Feb. 2010). “Plug-and-play inference for disease dynam-  
771 ics: measles in large and small populations as a case study”. In: *Journal of the Royal Society Interface*

- 772 7.43, pp. 271–283. ISSN: 1742-5689. DOI: 10.1098/rsif.2009.0151. URL: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2842609/> (visited on 08/16/2020).
- 773
- 774 Held, Leonhard and Michaela Paul (2012). “Modeling seasonality in space-time infectious disease surveillance data”. en. In: *Biometrical Journal* 54.6. eprint: <https://onlinelibrary.wiley.com/doi/pdf/10.1002/bimj.201200037>,  
775 pp. 824–843. ISSN: 1521-4036. DOI: 10.1002/bimj.201200037. URL: <https://onlinelibrary.wiley.com/doi/abs/10.1002/bimj.201200037> (visited on 05/23/2023).
- 776
- 777
- 778 Hinch, Robert et al. (July 2021). “OpenABM-Covid19—An agent-based model for non-pharmaceutical interventions against COVID-19 including contact tracing”. en. In: *PLOS Computational Biology* 17.7. Publisher: Public Library of Science, e1009146. ISSN: 1553-7358. DOI: 10.1371/journal.pcbi.1009146.  
779 URL: <https://journals.plos.org/ploscompbiol/article?id=10.1371/journal.pcbi.1009146>  
780 (visited on 09/01/2021).
- 781
- 782
- 783 Hoertel, Nicolas et al. (Sept. 2020). “A stochastic agent-based model of the SARS-CoV-2 epidemic in France”. en. In: *Nature Medicine* 26.9. Bandiera\_abtest: a Cg\_type: Nature Research Journals Number: 9 Primary\_atype: Research Publisher: Nature Publishing Group Subject\_term: Diseases;Health care Subject\_term\_id: diseases;health-care, pp. 1417–1421. ISSN: 1546-170X. DOI: 10.1038/s41591-020-1001-6.  
784 URL: <https://www.nature.com/articles/s41591-020-1001-6> (visited on 09/01/2021).
- 785
- 786
- 787
- 788 Holmdahl, Inga et al. (Nov. 2020). *Frequent testing and immunity-based staffing will help mitigate outbreaks in nursing home settings*. en. Tech. rep. Type: article. medRxiv, p. 2020.11.04.20224758. DOI: 10.1101/2020.11.04.20224758. URL: <https://www.medrxiv.org/content/10.1101/2020.11.04.20224758v2>  
789 (visited on 03/04/2022).
- 790
- 791
- 792 Hunter, Elizabeth and John D. Kelleher (June 2021). “Adapting an Agent-Based Model of Infectious Disease Spread in an Irish County to COVID-19”. en. In: *Systems* 9.2, p. 41. ISSN: 2079-8954. DOI: 10.3390/systems9020041. URL: <https://www.mdpi.com/2079-8954/9/2/41> (visited on 09/01/2021).
- 793
- 794
- 795 Hunter, Elizabeth, Brian Mac Namee, and John D. Kelleher (2017). “A Taxonomy for Agent-Based Models in Human Infectious Disease Epidemiology”. In: *Journal of Artificial Societies and Social Simulation* 20.3, p. 2. ISSN: 1460-7425.
- 796
- 797
- 798 Hunter, Elizabeth, Brian Mac Namee, and John Kelleher (Dec. 2018). “An open-data-driven agent-based model to simulate infectious disease outbreaks”. en. In: *PLOS ONE* 13.12. Publisher: Public Library of Science, e0208775. ISSN: 1932-6203. DOI: 10.1371/journal.pone.0208775. URL: <https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0208775> (visited on 09/01/2021).
- 799
- 800
- 801
- 802 Johansson, Michael A. et al. (Jan. 2021). “SARS-CoV-2 Transmission From People Without COVID-19 Symptoms”. In: *JAMA Network Open* 4.1, e2035057. ISSN: 2574-3805. DOI: 10.1001/jamanetworkopen.2020.35057. URL: <https://doi.org/10.1001/jamanetworkopen.2020.35057> (visited on 11/28/2022).
- 803
- 804
- 805 Keeling, Matt J and Ken T.D Eames (Sept. 2005). “Networks and epidemic models”. In: *Journal of The Royal Society Interface* 2.4. Publisher: Royal Society, pp. 295–307. DOI: 10.1098/rsif.2005.0051. URL: <https://royalsocietypublishing.org/doi/10.1098/rsif.2005.0051> (visited on 09/01/2021).
- 806
- 807
- 808 Keeling, Matt J., Pejman Rohani, and Bryan T. Grenfell (Jan. 2001). “Seasonally forced disease dynamics explored as switching between attractors”. en. In: *Physica D: Nonlinear Phenomena* 148.3-4, pp. 317–335. ISSN: 01672789. DOI: 10.1016/S0167-2789(00)00187-1. URL: <https://linkinghub.elsevier.com/retrieve/pii/S0167278900001871> (visited on 06/05/2023).
- 809
- 810
- 811
- 812 Kerr, Cliff C. et al. (Apr. 2021). “Covasim: an agent-based model of COVID-19 dynamics and interventions”. en. In: Company: Cold Spring Harbor Laboratory Press Distributor: Cold Spring Harbor Laboratory Press Label: Cold Spring Harbor Laboratory Press Type: article, p. 2020.05.10.20097469. DOI: 10.1101/2020.
- 813
- 814

- 815 05.10.20097469. URL: <https://www.medrxiv.org/content/10.1101/2020.05.10.20097469v3>  
816 (visited on 09/01/2021).
- 817 Krivorotko, Olga et al. (Mar. 2022). “Agent-based modeling of COVID-19 outbreaks for New York state  
818 and UK: Parameter identification algorithm”. en. In: *Infectious Disease Modelling* 7.1, pp. 30–44. ISSN:  
819 2468-0427. DOI: 10.1016/j.idm.2021.11.004. URL: [https://www.sciencedirect.com/science/  
820 article/pii/S2468042721000798](https://www.sciencedirect.com/science/article/pii/S2468042721000798) (visited on 06/05/2023).
- 821 Kronfeld-Schor, N. et al. (Feb. 2021). “Drivers of Infectious Disease Seasonality: Potential Implications for  
822 COVID-19”. en. In: *Journal of Biological Rhythms* 36.1. Publisher: SAGE Publications Inc, pp. 35–54.  
823 ISSN: 0748-7304. DOI: 10.1177/0748730420987322. URL: <https://doi.org/10.1177/0748730420987322>  
824 (visited on 05/22/2023).
- 825 Levin, Einav G. et al. (Dec. 2021). “Waning Immune Humoral Response to BNT162b2 Covid-19 Vaccine  
826 over 6 Months”. In: *New England Journal of Medicine* 385.24. Publisher: Massachusetts Medical Society  
827 eprint: <https://doi.org/10.1056/NEJMoa2114583>, e84. ISSN: 0028-4793. DOI: 10.1056/NEJMoa2114583.  
828 URL: <https://doi.org/10.1056/NEJMoa2114583> (visited on 07/06/2023).
- 829 Liu, Jiangtao et al. (July 2020). “Impact of meteorological factors on the COVID-19 transmission: A multi-  
830 city study in China”. en. In: *Science of The Total Environment* 726, p. 138513. ISSN: 0048-9697. DOI:  
831 10.1016/j.scitotenv.2020.138513. URL: [https://www.sciencedirect.com/science/article/pii/  
832 S004896972032026X](https://www.sciencedirect.com/science/article/pii/S004896972032026X) (visited on 06/05/2023).
- 833 Liu, Xiaoyue et al. (Apr. 2021). “The role of seasonality in the spread of COVID-19 pandemic”. In: *En-  
834 vironmental Research* 195, p. 110874. ISSN: 0013-9351. DOI: 10.1016/j.envres.2021.110874. URL:  
835 <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7892320/> (visited on 03/03/2023).
- 836 Lofgren, Eric et al. (June 2007). “Influenza Seasonality: Underlying Causes and Modeling Theories”. In: *Jour-  
837 nal of Virology* 81.11. Publisher: American Society for Microbiology, pp. 5429–5436. DOI: 10.1128/JVI.  
838 01680-06. URL: <https://journals.asm.org/doi/10.1128/JVI.01680-06> (visited on 02/01/2023).
- 839 Lowen, Anice C. and John Steel (July 2014). “Roles of Humidity and Temperature in Shaping Influenza  
840 Seasonality”. In: *Journal of Virology* 88.14, pp. 7692–7695. ISSN: 0022-538X. DOI: 10.1128/JVI.03544-  
841 13. URL: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4097773/> (visited on 02/01/2023).
- 842 Ma, Yiqun et al. (June 2021). “Role of meteorological factors in the transmission of SARS-CoV-2 in the  
843 United States”. en. In: *Nature Communications* 12.1. Number: 1 Publisher: Nature Publishing Group,  
844 p. 3602. ISSN: 2041-1723. DOI: 10.1038/s41467-021-23866-7. URL: [https://www.nature.com/  
845 articles/s41467-021-23866-7](https://www.nature.com/articles/s41467-021-23866-7) (visited on 06/05/2023).
- 846 Marr, Linsey C. et al. (Jan. 2019). “Mechanistic insights into the effect of humidity on airborne influenza  
847 virus survival, transmission and incidence”. In: *Journal of The Royal Society Interface* 16.150. Publisher:  
848 Royal Society, p. 20180298. DOI: 10.1098/rsif.2018.0298. URL: [https://royalsocietypublishing.  
849 org/doi/10.1098/rsif.2018.0298](https://royalsocietypublishing.org/doi/10.1098/rsif.2018.0298) (visited on 06/05/2023).
- 850 Martin, Joyce, Brady Hamilton, and Michelle Osterman (Aug. 2022). *Births in the United States, 2021*.  
851 Tech. rep. National Center for Health Statistics (U.S.) DOI: 10.15620/cdc:119632. URL: [https://  
852 stacks.cdc.gov/view/cdc/119632](https://stacks.cdc.gov/view/cdc/119632) (visited on 07/01/2023).
- 853 Matson, M. Jeremiah et al. (2020). “Effect of Environmental Conditions on SARS-CoV-2 Stability in Human  
854 Nasal Mucus and Sputum - Volume 26, Number 9—September 2020 - Emerging Infectious Diseases journal  
855 - CDC”. en-us. In: DOI: 10.3201/eid2609.202267. URL: [https://wwwnc.cdc.gov/eid/article/26/9/  
856 20-2267\\_article](https://wwwnc.cdc.gov/eid/article/26/9/20-2267_article) (visited on 06/05/2023).

- 857 Mecenas, Paulo et al. (Sept. 2020). “Effects of temperature and humidity on the spread of COVID-19: A  
858 systematic review”. en. In: *PLOS ONE* 15.9. Publisher: Public Library of Science, e0238339. ISSN: 1932-  
859 6203. DOI: 10.1371/journal.pone.0238339. URL: [https://journals.plos.org/plosone/article?  
860 id=10.1371/journal.pone.0238339](https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0238339) (visited on 06/05/2023).
- 861 Metcalf, C. Jessica E. et al. (Dec. 2009). “Seasonality and comparative dynamics of six childhood infections  
862 in pre-vaccination Copenhagen”. In: *Proceedings of the Royal Society B: Biological Sciences* 276.1676,  
863 pp. 4111–4118. ISSN: 0962-8452. DOI: 10.1098/rspb.2009.1058. URL: [https://www.ncbi.nlm.nih.  
864 gov/pmc/articles/PMC2821338/](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2821338/) (visited on 06/15/2020).
- 865 Mistry, D et al. (2021). *SynthPops: a generative model of human contact networks*. original-date: 2020-  
866 03-22T19:40:51Z. URL: <https://github.com/InstituteForDiseaseModeling/synthpops> (visited on  
867 11/30/2022).
- 868 Moghadas, Seyed M et al. (Dec. 2021). “The Impact of Vaccination on Coronavirus Disease 2019 (COVID-19)  
869 Outbreaks in the United States”. In: *Clinical Infectious Diseases* 73.12, pp. 2257–2264. ISSN: 1058-4838.  
870 DOI: 10.1093/cid/ciab079. URL: <https://doi.org/10.1093/cid/ciab079> (visited on 11/16/2022).
- 871 Morris, Dylan H et al. (Apr. 2021). “Mechanistic theory predicts the effects of temperature and humidity  
872 on inactivation of SARS-CoV-2 and other enveloped viruses”. In: *eLife* 10. Ed. by Wendy S Garrett,  
873 C Brandon Ogbunugafor, and Andreas Handel. Publisher: eLife Sciences Publications, Ltd, e65902. ISSN:  
874 2050-084X. DOI: 10.7554/eLife.65902. URL: <https://doi.org/10.7554/eLife.65902> (visited on  
875 06/05/2023).
- 876 Mossong, Joël et al. (Mar. 2008). “Social Contacts and Mixing Patterns Relevant to the Spread of Infectious  
877 Diseases”. en. In: *PLOS Medicine* 5.3. Publisher: Public Library of Science, e74. ISSN: 1549-1676. DOI:  
878 10.1371/journal.pmed.0050074. URL: [https://journals.plos.org/plosmedicine/article?id=10.  
879 1371/journal.pmed.0050074](https://journals.plos.org/plosmedicine/article?id=10.1371/journal.pmed.0050074) (visited on 04/01/2020).
- 880 National Center for Immunization and Respiratory Diseases (NCIRD) (2022a). *National Immunization Sur-  
881 vey Adult COVID Module (NIS-ACM)*. URL: [https://data.cdc.gov/Vaccinations/National-  
882 Immunization-Survey-Adult-COVID-Module-NI/udsf-9v7b](https://data.cdc.gov/Vaccinations/National-Immunization-Survey-Adult-COVID-Module-NI/udsf-9v7b) (visited on 02/02/2022).
- 883 — (2022b). *National Immunization Survey Child COVID Module (NIS-CCM)*. URL: [https://data.cdc.  
884 gov/Vaccinations/National-Immunization-Survey-Child-COVID-Module-NI/uny6-e3dx](https://data.cdc.gov/Vaccinations/National-Immunization-Survey-Child-COVID-Module-NI/uny6-e3dx) (visited  
885 on 02/02/2022).
- 886 National Institutes of Health (2022). *COVID-19 Treatment Guidelines Panel: Clinical Spectrum of SARS-  
887 CoV-2 Infection. Coronavirus Disease 2019 (COVID-19) Treatment Guidelines*. en. URL: [https://www.  
888 covid19treatmentguidelines.nih.gov/overview/clinical-spectrum/](https://www.covid19treatmentguidelines.nih.gov/overview/clinical-spectrum/) (visited on 02/27/2023).
- 889 Nichols, G. L. et al. (Oct. 2021). “Coronavirus seasonality, respiratory infections and weather”. In: *BMC  
890 Infectious Diseases* 21.1, p. 1101. ISSN: 1471-2334. DOI: 10.1186/s12879-021-06785-2. URL: [https:  
891 //doi.org/10.1186/s12879-021-06785-2](https://doi.org/10.1186/s12879-021-06785-2) (visited on 02/01/2023).
- 892 Nottmeyer, Luise N. and Francesco Sera (May 2021). “Influence of temperature, and of relative and absolute  
893 humidity on COVID-19 incidence in England - A multi-city time-series study”. en. In: *Environmental  
894 Research* 196, p. 110977. ISSN: 0013-9351. DOI: 10.1016/j.envres.2021.110977. URL: [https://www.  
895 sciencedirect.com/science/article/pii/S0013935121002711](https://www.sciencedirect.com/science/article/pii/S0013935121002711) (visited on 06/05/2023).
- 896 Oraby, Tamer et al. (Jan. 2014). “Modeling seasonal behavior changes and disease transmission with appli-  
897 cation to chronic wasting disease”. en. In: *Journal of Theoretical Biology* 340, pp. 50–59. ISSN: 0022-5193.  
898 DOI: 10.1016/j.jtbi.2013.09.003. URL: [https://www.sciencedirect.com/science/article/pii/  
899 S0022519313004244](https://www.sciencedirect.com/science/article/pii/S0022519313004244) (visited on 06/05/2023).

- 900 Raiteux, Jérémy et al. (July 2021). “Inactivation of SARS-CoV-2 by Simulated Sunlight on Contaminated  
901 Surfaces”. In: *Microbiology Spectrum* 9.1. Publisher: American Society for Microbiology, 10.1128/spec-  
902 trum.00333–21. DOI: 10.1128/spectrum.00333–21. URL: [https://journals.asm.org/doi/10.1128/  
903 Spectrum.00333–21](https://journals.asm.org/doi/10.1128/Spectrum.00333-21) (visited on 06/05/2023).
- 904 Reicher, Stephen and John Drury (Jan. 2021). “Pandemic fatigue? How adherence to covid-19 regulations  
905 has been misrepresented and why it matters”. en. In: *BMJ* 372. Publisher: British Medical Journal  
906 Publishing Group Section: Views & Reviews, n137. ISSN: 1756-1833. DOI: 10.1136/bmj.n137. URL:  
907 <https://www.bmj.com/content/372/bmj.n137> (visited on 02/27/2023).
- 908 Riddell, Shane et al. (Oct. 2020). “The effect of temperature on persistence of SARS-CoV-2 on common  
909 surfaces”. In: *Virology Journal* 17.1, p. 145. ISSN: 1743-422X. DOI: 10.1186/s12985-020-01418-7. URL:  
910 <https://doi.org/10.1186/s12985-020-01418-7> (visited on 06/05/2023).
- 911 Roubenoff, Ethan, Dennis M. Feehan, and Ayesha S. Mahmud (June 2023). “Evaluating primary and booster  
912 vaccination prioritization strategies for COVID-19 by age and high-contact employment status using data  
913 from contact surveys”. en. In: *Epidemics* 43, p. 100686. ISSN: 1755-4365. DOI: 10.1016/j.epidem.2023.  
914 100686. URL: <https://www.sciencedirect.com/science/article/pii/S1755436523000221> (visited  
915 on 06/27/2023).
- 916 Sah, Pratha et al. (May 2021). “Accelerated vaccine rollout is imperative to mitigate highly transmissible  
917 COVID-19 variants”. eng. In: *EClinicalMedicine* 35, p. 100865. ISSN: 2589-5370. DOI: 10.1016/j.eclinm.  
918 2021.100865.
- 919 Sera, Francesco et al. (Oct. 2021). “A cross-sectional analysis of meteorological factors and SARS-CoV-2  
920 transmission in 409 cities across 26 countries”. en. In: *Nature Communications* 12.1. Number: 1 Publisher:  
921 Nature Publishing Group, p. 5968. ISSN: 2041-1723. DOI: 10.1038/s41467-021-25914-8. URL: [https:  
922 //www.nature.com/articles/s41467-021-25914-8](https://www.nature.com/articles/s41467-021-25914-8) (visited on 06/05/2023).
- 923 Shaman, Jeffrey and Melvin Kohn (Mar. 2009). “Absolute humidity modulates influenza survival, trans-  
924 mission, and seasonality”. In: *Proceedings of the National Academy of Sciences* 106.9. Publisher: Pro-  
925 ceedings of the National Academy of Sciences, pp. 3243–3248. DOI: 10.1073/pnas.0806852106. URL:  
926 <https://www.pnas.org/doi/full/10.1073/pnas.0806852106> (visited on 06/05/2023).
- 927 Silk, Benjamin J. (2023). “COVID-19 Surveillance After Expiration of the Public Health Emergency Dec-  
928 laration — United States, May 11, 2023”. en-us. In: *MMWR. Morbidity and Mortality Weekly Report*  
929 72. ISSN: 0149-21951545-861X. DOI: 10.15585/mmwr.mm7219e1. URL: [https://www.cdc.gov/mmwr/  
930 volumes/72/wr/mm7219e1.htm](https://www.cdc.gov/mmwr/volumes/72/wr/mm7219e1.htm) (visited on 06/30/2023).
- 931 Smith, Thomas P. et al. (June 2021). “Temperature and population density influence SARS-CoV-2 trans-  
932 mission in the absence of nonpharmaceutical interventions”. In: *Proceedings of the National Academy  
933 of Sciences* 118.25. Publisher: Proceedings of the National Academy of Sciences, e2019284118. DOI:  
934 10.1073/pnas.2019284118. URL: [https://www.pnas.org/doi/full/10.1073/pnas.2019284118  
935](https://www.pnas.org/doi/full/10.1073/pnas.2019284118) (visited on 06/05/2023).
- 936 Strasser, Zachary H. et al. (Oct. 2022). “Estimates of SARS-CoV-2 Omicron BA.2 Subvariant Severity in New  
937 England”. In: *JAMA Network Open* 5.10, e2238354. ISSN: 2574-3805. DOI: 10.1001/jamanetworkopen.  
938 2022.38354. URL: <https://doi.org/10.1001/jamanetworkopen.2022.38354> (visited on 02/27/2023).
- 939 Susswein, Zachary, Eva C Rest, and Shweta Bansal (Apr. 2023). “Disentangling the rhythms of human ac-  
940 tivity in the built environment for airborne transmission risk: An analysis of large-scale mobility data”.  
941 In: *eLife* 12. Ed. by Niel Hens, Diane M Harper, and Guillaume Béraud. Publisher: eLife Sciences Pub-



- 942 lications, Ltd, e80466. ISSN: 2050-084X. DOI: 10.7554/eLife.80466. URL: <https://doi.org/10.7554/eLife.80466> (visited on 08/01/2023).
- 943
- 944 Tenforde, Mark W. (2021). “Effectiveness of Pfizer-BioNTech and Moderna Vaccines Against COVID-19  
945 Among Hospitalized Adults Aged  $\geq 65$  Years — United States, January–March 2021”. en-us. In: *MMWR.  
946 Morbidity and Mortality Weekly Report* 70. ISSN: 0149-21951545-861X. DOI: 10.15585/mmwr.mm7018e1.  
947 URL: <https://www.cdc.gov/mmwr/volumes/70/wr/mm7018e1.htm> (visited on 09/07/2021).
- 948 Thompson, Mark G. (2021). “Interim Estimates of Vaccine Effectiveness of BNT162b2 and mRNA-1273  
949 COVID-19 Vaccines in Preventing SARS-CoV-2 Infection Among Health Care Personnel, First Re-  
950 sponders, and Other Essential and Frontline Workers — Eight U.S. Locations, December 2020–March  
951 2021”. en-us. In: *MMWR. Morbidity and Mortality Weekly Report* 70. ISSN: 0149-21951545-861X. DOI:  
952 10.15585/mmwr.mm7013e3. URL: <https://www.cdc.gov/mmwr/volumes/70/wr/mm7013e3.htm> (visited  
953 on 09/07/2021).
- 954 — (2022). “Effectiveness of a Third Dose of mRNA Vaccines Against COVID-19–Associated Emergency  
955 Department and Urgent Care Encounters and Hospitalizations Among Adults During Periods of Delta  
956 and Omicron Variant Predominance — VISION Network, 10 States, August 2021–January 2022”. en-us.  
957 In: *MMWR. Morbidity and Mortality Weekly Report* 71. ISSN: 0149-21951545-861X. DOI: 10.15585/mmwr.  
958 mm7104e3. URL: <https://www.cdc.gov/mmwr/volumes/71/wr/mm7104e3.htm> (visited on 03/02/2022).
- 959 US Census Bureau (2022). *Table B01001: Sex by Age, 2021. American Community Survey 1-year Estimates.*  
960 URL: <https://data.census.gov/table?q=B01001:+SEX+BY+AGE&tid=ACSDT1Y2021.B01001> (visited  
961 on 07/06/2023).
- 962 Venkatramanan, Srinivasan et al. (Mar. 2018). “Using data-driven agent-based models for forecasting emerg-  
963 ing infectious diseases”. en. In: *Epidemics. The RAPIDD Ebola Forecasting Challenge* 22, pp. 43–49. ISSN:  
964 1755-4365. DOI: 10.1016/j.epidem.2017.02.010. URL: [https://www.sciencedirect.com/science/  
965 article/pii/S1755436517300221](https://www.sciencedirect.com/science/article/pii/S1755436517300221) (visited on 09/01/2021).
- 966 Weaver, Amanda K. et al. (2022). “Environmental Factors Influencing COVID-19 Incidence and Severity”.  
967 In: *Annual Review of Public Health* 43.1. eprint: [https://doi.org/10.1146/annurev-publhealth-052120-  
968 101420](https://doi.org/10.1146/annurev-publhealth-052120-101420), pp. 271–291. DOI: 10.1146/annurev-publhealth-052120-101420. URL: [https://doi.org/10.  
969 1146/annurev-publhealth-052120-101420](https://doi.org/10.1146/annurev-publhealth-052120-101420) (visited on 06/05/2023).
- 970 Williams, Katherine V. et al. (Nov. 2022). “Can a Two-Dose Influenza Vaccine Regimen Better Protect  
971 Older Adults? An Agent-Based Modeling Study”. en. In: *Vaccines* 10.11. Number: 11 Publisher: Mul-  
972 tidisciplinary Digital Publishing Institute, p. 1799. ISSN: 2076-393X. DOI: 10.3390/vaccines10111799.  
973 URL: <https://www.mdpi.com/2076-393X/10/11/1799> (visited on 06/05/2023).
- 974 Wu, Yu et al. (Aug. 2020). “Effects of temperature and humidity on the daily new cases and new deaths of  
975 COVID-19 in 166 countries”. en. In: *Science of The Total Environment* 729, p. 139051. ISSN: 0048-9697.  
976 DOI: 10.1016/j.scitotenv.2020.139051. URL: [https://www.sciencedirect.com/science/article/  
977 pii/S0048969720325687](https://www.sciencedirect.com/science/article/pii/S0048969720325687) (visited on 06/05/2023).
- 978 Xu, Jiaquan et al. (Dec. 2022). *Mortality in the United States, 2021*. Tech. rep. National Center for Health  
979 Statistics (U.S.) DOI: 10.15620/cdc:122516. URL: <https://stacks.cdc.gov/view/cdc/122516> (visited  
980 on 07/01/2023).
- 981 Yang, Zhen et al. (Apr. 2022). “Clinical Characteristics, Transmissibility, Pathogenicity, Susceptible Popu-  
982 lations, and Re-infectivity of Prominent COVID-19 Variants”. In: *Aging and Disease* 13.2, pp. 402–422.  
983 ISSN: 2152-5250. DOI: 10.14336/AD.2021.1210. URL: [https://www.ncbi.nlm.nih.gov/pmc/articles/  
984 PMC8947836/](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8947836/) (visited on 02/27/2023).