How will mass-vaccination change COVID-19 lockdown requirements in Australia?

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Abstract

To prevent future outbreaks of COVID-19, Australia is pursuing a mass-vaccination approach in which a targeted group of the population comprising healthcare workers, aged-care residents and other individuals at increased risk of exposure will receive a highly effective priority vaccine. The rest of the population will instead have access to a less effective vaccine. We apply a large-scale agent-based model of COVID-19 in Australia to investigate the possible implications of this hybrid approach to mass-vaccination. The model is calibrated to recent epidemiological and demographic data available in Australia, and accounts for several components of vaccine efficacy. Within a feasible range of vaccine efficacy values, our model supports the assertion that complete herd immunity due to vaccination is not likely in the Australian context. For realistic scenarios in which herd immunity is not achieved, we simulate the effects of mass-vaccination on epidemic growth rate, and investigate the requirements of lockdown measures applied to curb subsequent outbreaks. In our simulations, Australia's vaccination strategy can feasibly reduce required lockdown intensity and initial epidemic growth rate by 43% and 52%, respectively. The severity of epidemics, as measured by the peak number of daily new cases, decreases by up to two orders of magnitude under plausible mass-vaccination and lockdown strategies. The study presents a strong argument for a large-scale vaccination campaign, which would significantly reduce the intensity of non-pharmaceutical interventions in Australia and curb future outbreaks.

I. INTRODUCTION

The Australian response to the COVID-19 pandemic has been very effective to date. Strict control measures, including travel restrictions and social distancing, successfully suppressed the initial pandemic wave in Australia (March – June 2020) [1], as well as several secondary outbreaks across the states, most notably in Victoria (June – September 2020) [2]. However, as vaccines become available a more refined response is needed, given the need to balance population health against the high socio-economic impacts of local, regional and nation-wide lockdowns.

The national COVID-19 vaccine rollout strategy developed by the Australian Government commenced in late February 2021, aiming to vaccinate a significant portion of the Australian population (the majority of the adult population) by the end of October 2021 [3]. The first phase of the strategy targets priority groups with the BNT162b2 (Pfizer/BioNTech) vaccine, while the remainder of the population will receive the ChAdOx1 nCoV-19 (Oxford/AstraZeneca) vaccine during phases two and three. Both of these vaccines have demonstrated high clinical efficacy [4, 5]. Thus, many questions remain. Is herd immunity achievable with current vaccination approaches? To what extent can the strict lockdown rules be relaxed with a partial mass vaccination? Is there an optimal but feasible balance between the vaccination efforts and social distancing practice? In this work, we approach these questions with a large-scale agent-based

model (ABM) of COVID-19 transmission, case-targeted non-pharmaceutical interventions, lockdowns, and mass-vaccination in the context of Australia.

There are several specific challenges in modelling COVID-19 vaccination campaigns: the complexity and burden of non-pharmaceutical interventions (NPIs); the heterogeneity of the population; country-specific demographics; logistical and supply constraints; as well as unknown vaccine characteristics. The heterogeneity of the Australian population has been shown to unevenly affect the spread of respiratory diseases across different social contexts and wider jurisdictions [1, 6]. We can therefore expect complex trade-offs between NPIs and vaccination interventions, covering overlapping but not identical parts of the population. These effects may be difficult to predict for situations in which the vaccine efficacy differs with respect to reducing susceptibility, preventing symptoms of infection, and limiting further transmission of the virus. Some of the available vaccines, most notably BNT162b2 (Pfizer/BioNTech), have shown a high efficacy against documented infection, as well as symptomatic and severe disease [4]. However, comprehensive results across multiple efficacy components are still lacking. In this work, we account for differences in vaccine efficacy for the two distinct vaccine types approved for distribution in Australia: a *priority* vaccine, (e.g.., BNT162b2), and a *general* vaccine, (e.g., ChAdOx1 nCoV-19).

In order to capture population heterogeneity, we adapted a previously developed and validated high-resolution ABM of mitigation and control of the COVID-19 pandemic in Australia [1, 6]. This model included a range of dynamically adjustable NPIs, such as travel restrictions, case isolation, home quarantine and mandated social distancing (lockdown). Here, we extended the ABM to include several detailed vaccination measures. These extensions included an explicit account of separate components of vaccination efficacy (susceptibility, disease, and infectiousness), and changeable levels of age-stratified mass-vaccination coverage with the general and priority vaccines.

This paper addresses several open questions surrounding vaccination in Australia. Firstly, we investigate the feasibility of herd immunity following a mass-vaccination campaign. This is addressed by varying vaccination coverage with different vaccine efficacy combinations. Secondly, we quantify the benefit of the general vaccine in scenarios where all priority vaccine supplies are consumed by considering different levels of general vaccination distributed in addition to a fixed realistic priority vaccination coverage. Finally, we quantify to what extent mass-vaccination can reduce or eliminate the need for lockdowns by varying lockdown compliance levels for various extents of vaccination coverage.

II. METHODS

A. Simulating COVID-19 in Australia

Our approach to simulating COVID-19 in Australia follows that of our previous work [1], with the following modifications to our model of COVID-19 disease natural history and case ascertainment:

- Infectious incubation times (T_{inc}) calibrated to the findings of Lauer et al. [7] who inferred log-normally distributed incubation times with mean 5.5 days ($\mu = 1.62,\ \sigma = 0.418$).
- An infectious asymptomatic or symptomatic period (T_{symp}), following incubation, lasting between 7 days and 14 days (uniformly distributed), based on estimates of the replication-competent viral shedding period used to support guidance on case isolation periods published by the United States Centre for Disease Control and Prevention (CDC, see [8] and references therein [9]).
- Differentiation between “asymptomatic infectivity" and “pre-symptomatic infectivity". In the previous iteration of the model used in Chang et al. [1], asymptomatic and pre-symptomatic individuals had reduced infectivity to contacts. That assumption was modified in this work, for which pre-symptomatic cases are assumed to be as infectious as symptomatic cases (with respect to viral load), while those who remain asymptomatic throughout the course of disease have reduced infectivity (a factor of 0.5 is applied to the force of infection exerted on contacts). This change reflects the general finding that pre-symptomatic transmission is responsible for a substantial amount of COVID-19 spread (up to 50% of transmission), and allows a parsimonious calibration of disease natural history, reproductive ratio, and generation interval [10, 11].
- To simulate the imperfect detection of cases in a scenario with high levels of voluntary population screening, we introduce two case detection probabilities, one for symptomatic case detection and the other for pre-symptomatic and asymptomatic detection. For symptomatic cases, the probability of detection per day is set to 0.23, while for pre-symptomatic and asymptomatic cases, the probability of detection per day is 0.01.

B. Model calibration

Because the models of disease natural history and case ascertainment were modified, we re-calibrated the model used by Chang et al. [1] to approximately match the case incidence

data recorded during the first and second waves of COVID-19 in Australia and the global reproduction number of $R_0 \approx 2.9$ 95% CI $[2.39, 3.44]$ [12] (See Supporting Information).

C. Mass-vaccination simulations

In our mass-vaccination scenarios, we used an age-stratified vaccine allocation scheme. Starting with no individuals vaccinated, the algorithm allocates new immunisations randomly according to the following ratio: 100/10/1, which correspond to [age $\geq$ 65] / [18 $\leq$ age $<$ 65] / [age $<$ 18]. That is, for every 100 individuals aged over 64 years, 10 individuals aged between 18 and 64 years are immunised and one individual under the age of 18 years is immunised. This allocation ratio applies unless there are no remaining unvaccinated individuals in an age category, in which case vaccines are allocated to the remaining age categories according to the same specified proportions until the specified number of immunisations is depleted. The priority vaccines are distributed first, followed by the general vaccines. In practice, due to the age distribution in our model of the Australian population (based on the 2016 ABS Census) this means that all individuals aged over 65 years are immunised unless there are fewer than 3.9×10^6 total immunisations. In our hybrid mass-vaccination scenarios, we assume at least 5×10^6 priority immunisations, so the entire population over the age of 64 years is immunised with the priority vaccine, while the remaining immunisations are distributed between children and adults under 65 in a ratio of 10/1 (adults/children). All immunisations are allocated on day 0 of each outbreak simulation.

D. Growth rate estimation

Table II, Table S4, and Figure S3 report growth rates estimated from incidence data produce by the ABM or collected from government case reports. To estimate growth rates, we fit each case incidence timeseries to a delayed exponential function:

$$I(t) = \exp(\lambda(t - \Delta t)) \,, \tag{1}$$

where λ is the exponential growth rate of case incidence, and the delay Δt accounts for transient stochastic effects during the early stages of outbreaks as well as delays in detection of new cases.

III. RESULTS

We present our results in three sections, first covering questions related to herd immunity and vaccine efficacy, then the effect of mass-vaccination on epidemic growth rate, and lastly the effects of future lockdowns in conjunction with prepandemic mass-vaccination. Section III A investigates the feasibility of achieving herd immunity given the existing known and unknown aspects of COVID-19 vaccine efficacy. For clinical efficacy values of 0.9 and 0.6 (corresponding to conservative estimates for the priority and the general vaccine, respectively), we use a homogeneous approximation to calculate the coverage required to achieve herd immunity as a function of vaccine efficacy against susceptibility, symptom expression, and onward transmission. We then compare the results of our ABM to the homogeneous approximation for a subset of vaccine efficacy values. Section III B describes the effects of realistic simulated mass vaccination regimes on epidemic growth rate. In scenarios where herd immunity is not achieved, we compute the growth rate of cumulative incidence for different levels of vaccination coverage, in combination with case-targeted NPIs. Section III C investigates outbreak suppression in mass-vaccination scenarios and shows how vaccination can reduce the fraction of the population required to be in lockdown to achieve suppression of case incidence.

A. Herd immunity requirements: coverage and efficacy

For the purposes of modelling the effects of vaccination on the spread of COVID-19 and evaluating the requirements for herd immunity, three main components of vaccine efficacy constitute important unknown factors:

- Efficacy for susceptibility (VEs) determines the level of immunity vaccination imparts to those susceptible to the virus. In the ABM this parameter reduces the probability of becoming infected if exposed.

- Efficacy for disease (VEd) determines the expression of illness in those who are vaccinated and subsequently become infected. In the ABM this parameter reduces the probability of expressing symptoms if infected.

- Efficacy for infectiousness (VEi) reduces the potential for vaccinated individuals to transmit the virus if infected. In the ABM, this parameter reduces the force of infection produced by infected individuals who are vaccinated.

The practical bounds of the efficacy terms (VEs, VEi, and VEd) are only partially constrained by the clinical efficacy (VEc) reported in clinical trials, since:

$$\mathrm{VEc} = \mathrm{VEd} + \mathrm{VEs} - \mathrm{VEsVEd}. \tag{2}$$

The clinical efficacy, VEc, is defined as the reduction in presentation of clinical disease in the vaccine group relative to the control. The vaccine efficacy for an individual's susceptibility and disease, VEs and VEd, are constrained by the clinical efficacy, VEc, through Eq. (2); however, the vaccine efficacy for infectiousness, VEi, is left undefined by clinical trial data. Although unreported, it is crucial for VEi to be defined in order to compute the population-level vaccine efficacy, VE, for a given proportion of the population vaccinated. For defined values of the input parameters (coverage, VEi, VEs, and VEd), VE can be estimated from a homogeneous approximation of population mixing (see Supporting Information) and the necessary vaccine coverage threshold for herd immunity can then be estimated by computing the effective reproductive number, R, after vaccination:

$$R = R_0(1 - \mathrm{VE}), \tag{3}$$

where R_0 is the basic reproductive ratio in a completely susceptible population, and herd immunity is achieved when $R < 1$. In the Supporting Information Fig. S1, we provide herd immunity thresholds in the VEi $\times$ coverage plane for different combinations of VEd and VEs constrained through Eq. (2), with $R_0 = 2.75$ to match the basic reproductive ratio used in our ABM.

These results show that, for herd immunity in a homogeneous system, the general vaccine (with VEc = 0.6) must produce a substantial reduction in the transmission potential of infected individuals, with an efficacy for infectiousness (VEi) on the same order of efficacy for susceptibility (VEs) and disease (VEd). On the other hand, Fig. S1b illustrates that a priority vaccine with clinical efficacy of 90% (VEc = 0.9) could produce herd immunity with vaccination coverage between 64% and 86%. Moreover, as long as VEs $\geq$ VEd, this result can be achieved without a substantial contribution from the unknown effect of the vaccine on reducing infectiousness (i.e., VEi).

Of course, Australia is not a homogeneous system with respect to population mixing patterns, and the true vaccine allocation strategy is intentionally heterogeneous, first prioritising those in high-risk age groups and prioritising children last. For these reasons, we expected our ABM to produce results differing from those estimated by the homogeneous approximation. To match our scenarios to the Australian context, which has consistently maintained case-targeted non-

pharmaceutical interventions, we performed a systematic scan of efficacy parameters both with and without combinations of detected case isolation, home quarantine of household contacts, and international travel restrictions. While the proposed vaccination regime in Australia involves both priority and general vaccines, initially we treat each separately in order to reduce the complexity of the parameter space. Therefore, the results presented in this section are not directly applicable to proposed vaccination levels in Australia, but can be used to guide intuition with respect to the influence of different efficacy combinations and potential deviation from estimates based on homogeneous approximations.

For a given mass-vaccination scenario, the ABM mimics the current government roll-out policy by allocating immunisations using an age-stratified system. In this system, individuals aged 65 and older are preferentially vaccinated, with second preference for those aged 18 - 64 years, and third preference for those under the age of 18 (see Methods). We do not explicitly simulate partial vaccination (i.e., using one dose only), with the number of individuals immunised corresponding to the prospective number of completed vaccine treatment schedules.

Our full results are given in the Supporting Information Tables S5 through S12, and are summarised here below. Note that in the ABM, due to continuous introductions of cases from overseas, the clustering of children into school contact networks, and age-stratified vaccine roll-out, formal herd immunity is not possible (finite epidemic growth is observed for all parameter combinations). However, a nonlinear reduction in epidemic severity with increasing vaccine coverage is observed, with a distinct threshold effect that agrees qualitatively with the predictions of the homogeneous model (Figure 1). In the below, we refer to this nonlinear reduction as herd immunity.

Our results show that the general vaccine (VEc = 0.6) alone cannot feasibly induce herd immunity, even when combined with targeted non-pharmaceutical interventions. Specifically, the general vaccine by itself (without case-targeted NPIs) can only produce the desired nonlinear drop in peak case prevalence with an unrealistically high efficacy for infectiousness (VEi > 0.5) and a vaccine coverage greater than 80% (Fig. S2a). However, there are still significant benefits for feasible ranges of vaccine coverage and efficacy. Notably, peak prevalence can be reduced by a factor of two without the need for case-targeted interventions, with central values of VEd and VEs (i.e., when VEd = VEs $= 1 - \sqrt{1 - \text{VEc}} = 0.368$), a reasonable value of VEi ≈ 0.5, and only 40% population coverage (Tab. S5). Referring to Fig. S2a, increasing this vaccine coverage to approximately 80% reduces peak prevalence by 83–88% (Tab. S5) and delays the peak by several weeks (Tab. S6). Finally, by combining case-targeted interventions with this 80% coverage, the general vaccine can dramatically slow the spread of the virus (peak prevalence

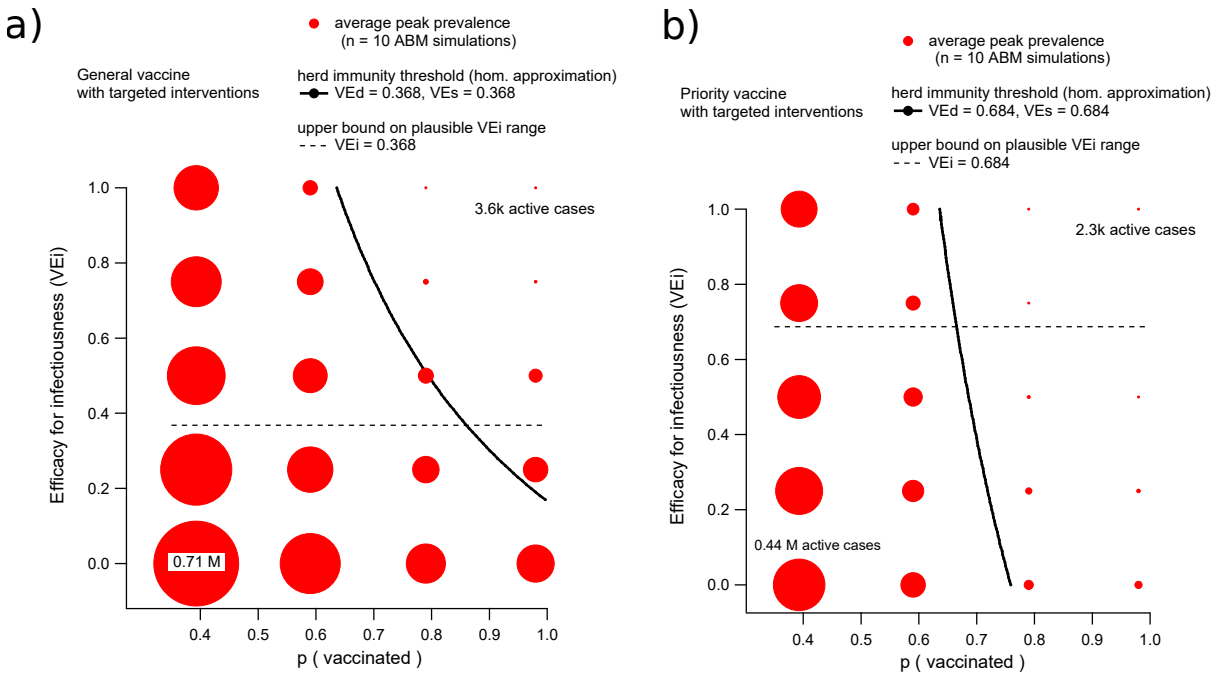

FIG. 1. Simulations suggest that herd immunity is unlikely to be attained by either the general or the priority vaccine alone. The epidemic severity shown here is simulated by the ABM over a range of values for coverage and vaccine efficacy against infectiousness (VEi), for the general vaccine (a, VEc = 0.6) and the priority vaccine (b, VEc = 0.9). Red circles correspond to mean peak prevalence levels produced by ABM simulations. The solid black lines give the coverage thresholds for herd immunity estimated by the homogeneous approximation, and the dashed lines illustrate conservative practical upper bounds on VEi. Here, central values of efficacy against disease and susceptibility were used (VEd = VEs = $1-\sqrt{1-\text{VEc}}$), and case-targeted NPIs were applied in addition to vaccination.

delayed by approximately 60 days, Tab. S10), and reduce peak prevalence by 92% (Fig. 1a, Tab. S9). As a baseline value, note that our simulations show case-targeted interventions alone decrease peak prevalence by 32%.

The priority vaccine (VEc = 0.9), produces similar benefits with a vaccine coverage as low as 60% and central efficacy values of VEs = VEd = 0.684, VEi $\in [0.5, 0.75]$ (Fig. S2b). However, the current proposal for vaccination rollout in Australia constrains the maximum coverage of the priority vaccine to around 40%. Therefore, substantial uptake of the less-effective general vaccine will be required if the benefits of the priority vaccine are to be realised for the larger population. In combination with targeted interventions, our simulations suggest that the priority vaccine could produce herd immunity with a vaccine coverage of above 70%. For the more feasible coverage (of 40%), the priority vaccine reduces the peak prevalence by 80% and delays the peak by approximately 40 days (Fig. 1b, Tab. S12).

scenario	targeted NPIs	priority immunisations	general immunisations
no intervention	nil	nil	nil
targeted NPIs only	TR, CI, HQ*	nil	nil
priority vaccine (5M)	”	5×10^6	nil
general vaccine (11.5M)	”	nil	11.5×10^6
priority vaccine (10M)	”	10^7	nil
priority 10M, general 2.5M	”	10^7	2.5×10^6
priority 10M, general 6.1M	”	10^7	6.1×10^6
priority 10M, general 10.7M	”	10^7	10.7×10^6

TABLE I. Selected vaccination scenarios simulated with the ABM. The numbers under “priority immunisations" and “general immunisations" correspond to the number of individuals who have undergone a full vaccination regime (i.e., a two-dose regime for the priority vaccine). *TR: travel restrictions (ban on international travel), CI: case isolation (in-home isolation of detected cases), HQ: home quarantine (in-home isolation for household contacts of detected cases).

B. Effects of mass-vaccination on epidemic growth

To investigate the possible effects of realistic mass-vaccination strategies on epidemic growth dynamics, we selected central values of efficacy for the priority vaccine (VEi = VEs = VEd = 0.684) and general vaccine (VEi = VEs = VEd = 0.368), and simulated initial epidemic growth in eight different scenarios (Tab. I). We find that realistic hybrid vaccination campaigns systematically reduce epidemic growth rate with increasing coverage (by up to a factor of two). However, we do not identify a distinct coverage level beyond which growth rate decreases sharply (i.e., a herd immunity threshold).

To improve the realism of our model for simulating hybrid vaccination scenarios, we use the latest estimates of coverage with the priority vaccine (enough has been ordered at the time of writing to vaccinate up to 10M individuals, or approximately 40% of the population), and the general vaccine. We assume that the general vaccine will not be subject to supply constraints, with coverage limited instead by uptake. To reflect the current situation in Australia where the disease is currently controlled, we do not simulate a progressive rollout during the outbreak. Instead, we explore different coverage levels as a proxy for timing of the next epidemic wave between the beginning and end of the vaccination campaign. We compare scenarios ranging from a relatively small number of priority vaccine immunisations (5M two-dose vaccinations), to a realistic endpoint scenario with 10M priority (two-dose) immunisations and an additional 10.7M general immunisations, for coverage on the order of 90%.

For each scenario, we simulated 110 realisations of outbreaks and estimated the expected growth rate of cumulative incidence for the first 2 000 cases (Fig. 2). To do so, we computed the

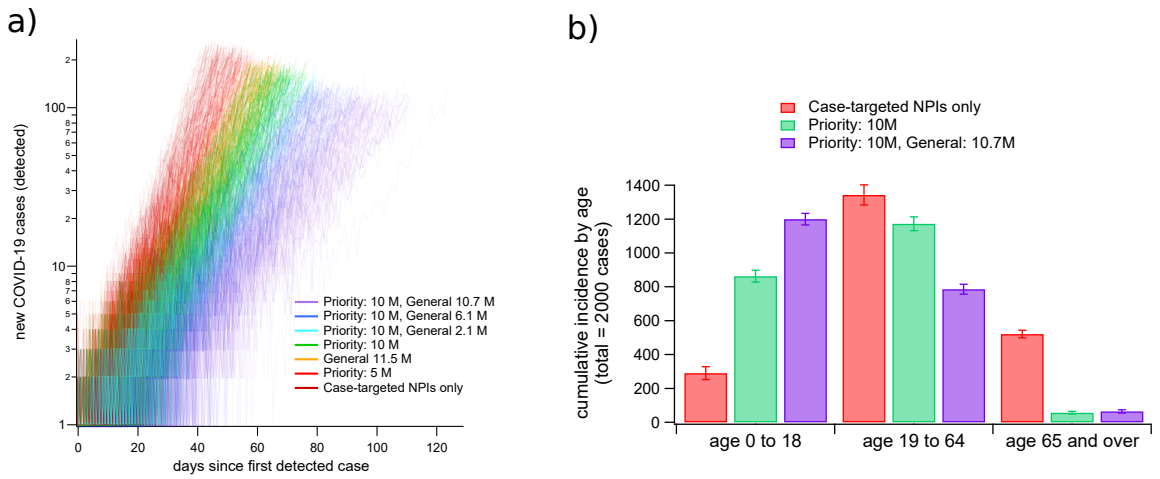

FIG. 2. Hybrid vaccination programs produce up to a two-fold reduction of the epidemic growth rate. Individual incidence trajectories are color-coded by mass-vaccination scenario (110 trajectories are shown for each). Each trajectory ends at the time the lockdown trigger condition was reached (cumulative incidence exceeding 2000 cases). Subplot (a) shows log-scaled incidence trajectories for each vaccination scenario. Subplot (b) shows the distribution of the first 2000 cases in the three age groups used to prioritise vaccination in three representative scenarios (error bars show standard deviations over 110 realisations). Summary growth rate statistics for each scenario are given in Tab. II.

incidence growth rate of each realisation (see Methods) and estimated the mean growth rate over all realisations of each mass-vaccination scenario (Tab. II). Initial growth of case incidence decreases gradually as vaccination levels increase (Fig. 2). With 10M priority immunisations (maximum projected supply), the growth rate decreased by 28% relative to the rate computed with targeted NPIs only. At a feasible endpoint condition with 88% of the population vaccinated (10M priority and 10.7M general immunisations), the average growth rate decreases by 52% (from 0.118 d^{-1} with targeted NPIs only, to 0.057 d^{-1} with vaccination). Log-scaled plots of initial case incidence (Figure 2a) clearly demonstrate the lack of a defined herd immunity threshold within the set of plausible scenarios we investigated. This is surprising given the high levels of vaccine coverage reached. The distribution of the first 2000 cases between age groups suggests that systematically placing children at low priority for immunisation may increase the required threshold for nonlinear reductions in epidemic growth rate (Figure 2b). Based on the risk-averse precedent for COVID-19 response in Australia, it is plausible that even the relatively slow spreading rates we calculate for the endpoint vaccination scenario would lead to some level of lockdown imposition, which we address in the following section.

scenario	mean growth rate	quantiles [5%, 95%]	95% CI (mean, bootstrap)
no intervention	0.137	[0.128, 0.146]	[0.1356, 0.1376]
targeted NPIs only	0.118	[0.110, 0.127]	[0.1171, 0.1189]
priority vaccine (5M)	0.104	[0.097, 0.112]	[0.1034, 0.1052]
general vaccine (11.5M)	0.091	[0.083 0.099]	[0.0901, 0.0917]
priority vaccine (10M)	0.085	[0.076, 0.092]	[0.0843, 0.0859]
priority 10M, general 2.5M	0.078	[0.072, 0.087]	[0.0771, 0.0787]
priority 10M, general 6.1M	0.067	[0.061, 0.072]	[0.0666, 0.0679]
priority 10M, general 10.7M	0.057	[0.052, 0.062]	[0.0562, 0.0573]

TABLE II. Growth rates of daily incidence for eight different intervention scenarios. For each scenario, growth rates were computed for 110 realisations. The values shown here are ensemble means from each scenario, as well as the 5% and 95% quantiles of the growth rate distribution from each set of realisations and the 95% bootstrap confidence interval for the mean.

C. Effects of mass-vaccination on lockdown requirements

For the realistic vaccination scenarios given in Tab. I, we estimated the level of lockdown compliance required to suppress epidemic growth. In each scenario, the population-scale physical distancing (lockdown) measures were enacted when the epidemic reached cumulative incidence of 2 000 cases. Figure 3 illustrates how epidemic dynamics depend on vaccination levels, and how these dynamics respond to the implementation of lockdown restrictions. Incidence initially increases exponentially, and the growth rate is reduced after the imposition of lockdown restrictions (occurring at approximately day 50 in Fig. 3a, and at day 120 in Fig. 3b). If enough of the population complies with physical distancing measures, the growth rate becomes negative and the conditions for eventual suppression are met. In concordance with our previous work [1], this threshold lies between 60% and 70% compliance when only NPIs are considered. In these simulations, vaccination systematically decreased the fraction of the population required to maintain physical distancing restrictions in order to suppress epidemic spread. Summary results shown in Fig. 4 demonstrate how this compliance threshold depends on the level of vaccination. At the endpoint condition of 10M priority immunisations and 10.7M general immunisations, the lockdown compliance threshold drops to approximately 40% (Fig. 3b, Fig. 4). In the Supplementary Material, we show results obtained for higher general vaccine efficacy (raised from VEc = 0.6 to VEc = 0.75, Figure S5), demonstrating that this approximate threshold was not sensitive within this range to the precise efficacy value used for the general vaccine.

In addition to reducing the lockdown compliance threshold, vaccination reduces the growth rate of the epidemic even in the absence of lockdown restrictions, so incidence levels during outbreaks decrease dramatically for the same proportion of the population complying with

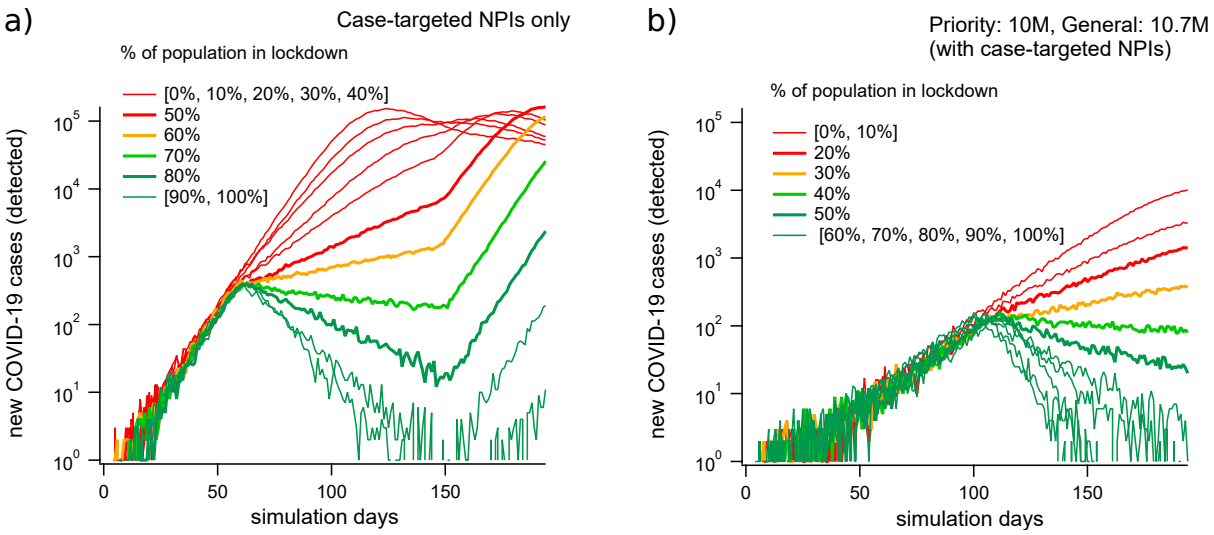

FIG. 3. Our simulations suggest that realistic hybrid vaccinations strategies reduce the required intensity of lockdowns (mandated physical distancing) by a factor of two. Timeseries plots of representative case incidence trajectories for the scenario with targeted NPIs only (a) demonstrate a lockdown compliance threshold for elimination lying between 60% and 70%. Similar plots for the vaccination scenario with 10M priority vaccinations (VEc = 0.9; VEi = VEs = VEd = 0.684) and 10.7M general vaccinations (VEc = 0.6; VEi = VEs = VEd = 0.368), in addition to case-targeted NPIs, (b) show a lockdown compliance threshold for elimination lying between 30% and 40%.

physical distancing. For example, Figure 4 demonstrates that, 60 days after the beginning of lockdown, the complete vaccination program consistently decreases case incidence by almost two orders of magnitude, independently to the proportion of the population complying. Such a difference in case incidence could be expected to have a dramatic impact on the projected strain to medical infrastructure, even without considering vaccine efficacy for severe disease. Additionally, Figure 2 and Figure 4 demonstrate that, due to slower epidemic growth with vaccination, case incidence at the onset of lockdown decreases from approximately 250 cases per day (targeted NPIs only), down to 100 cases per day (completed vaccination program). Taken together, these results indicate that vaccination would allow for shorter, less restrictive physical distancing mandates, if such measures were required in order to suppress subsequent outbreaks.

IV. DISCUSSION

Stochastic agent-based models have been established as robust tools for tracing fine-grained effects of complex intervention policies in diverse epidemic and pandemic settings [13, 14]. ABM studies have produced policy recommendations developed for the control of COVID-19 outbreaks, which have been adopted broadly by the WHO [15].

Typically, ABMs simulate each individual separately, aiming to account for heterogeneity

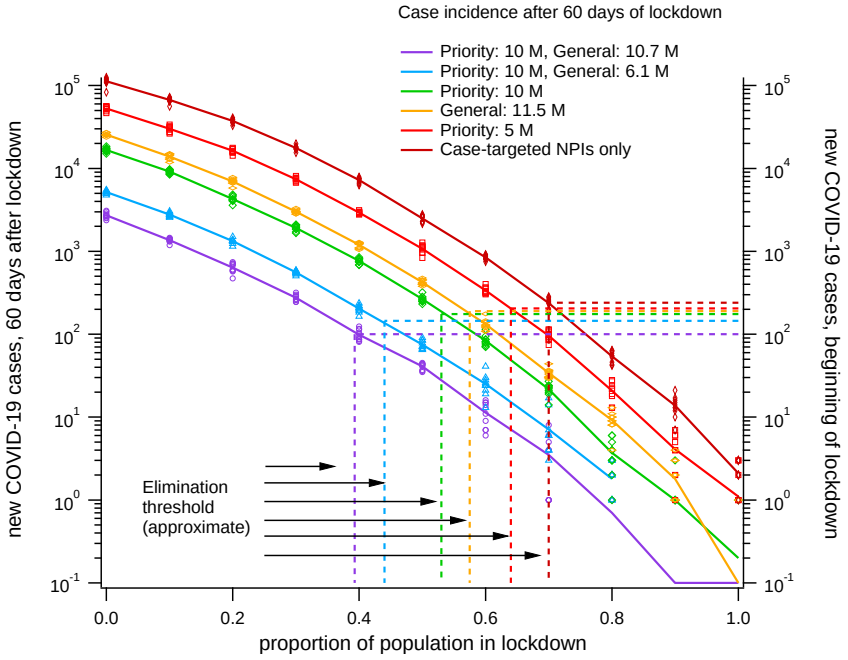

FIG. 4. The intensity of lockdown required for gradual elimination of the virus steadily decreases with increasing vaccination levels. Solid lines connect ensemble averages of case incidence 60 days into the lockdown period for each scenario (left y-axis) while the values recorded from each individual simulation are shown as symbols. Each horizontal dashed line corresponds to the average incidence at the onset of lockdown (right y axis) for the vaccination scenario labelled with the same color. The vertical dashed lines correspond to the approximate proportion of the population in lockdown required for case incidence to decrease, in each vaccination scenario.

of demographic and epidemic conditions, as well as details of social interactions and mobility patterns. This approach has a relatively high computational cost, driven by calibration of numerous internal ABM parameters [1, 16] and reconstruction of mobility patterns. However, ABMs offer an important advantage, combining both behavioural and mechanical adequacy of the model mechanism. Behavioural adequacy is verified by comparing simulated and actual epidemic patterns. Mechanical adequacy ensures, in addition, that the natural history of the disease progresses in concordance with the known estimates of incubation periods, serial and generation intervals, and other key parameters. Two ABMs, verified with respect to both behavioural and mechanical adequacy, provided a strong foundation for our study: ACEMod developed to simulate influenza pandemics [17], and AMTraC-19 created to simulate COVID-19 [1].

Our early COVID-19 study [1] compared several non-pharmaceutical intervention strategies and identified the minimal levels of social distancing required to control the pandemic. A compliance rate below 70% was found to be inadequate for any duration of social distancing, while a compliance at the 90% level was shown to control the disease within 13-14 weeks, when coupled with other restrictions. In another study we modelled pre-pandemic vaccination

and targeted antiviral prophylaxis for influenza pandemics in Australia [18]. However, the COVID-19 pandemic demands new intervention protocols, optimised for given vaccine efficacy and coverage [19, 20], and accounting for logistical constraints, limited supply and hesitancy.

An early investigation by [19] modelled trade-offs between the vaccine efficacy and vaccination coverage, before and during an epidemic in the USA. The work showed that in order to prevent an epidemic, the efficacy has to be at least 60% when vaccination coverage is perfect (100%), and when the latter reduces to 60%, the necessary efficacy threshold increases to 80%. During an ongoing epidemic, higher efficacy thresholds were found to be needed to significantly reduce peak severity. The study of [20] modelled several vaccine components, in particular distinguishing between the vaccine types that may reduce susceptibility by inhibiting viral transmission (thus, indirectly protecting the individual) and the vaccine types that directly protect only the vaccinated individual by reducing the probability of developing severe symptoms. Importantly, this study suggested that vaccinating older age groups initially may prevent a second wave within the UK, but only if the vaccine reduces both transmission and disease. Even in this optimistic scenario, only a highly efficacious vaccine, delivered to at least 70% of the population, was shown to succeed without NPIs. A high level of vaccination was also shown to be required in France, based on the ABM developed by [21] who reported that vaccinating only priority groups (e.g., older adults) would be insufficient to lift NPIs.

Thus, while it is clear that vaccination cannot provide herd immunity without mass population coverage, to what extent coverage requirements can be alleviated by maintaining some (relaxed) level of social distancing and other suppression measures in a highly heterogeneous demographic setting remains an open and country-specific question. This question becomes more complex when several vaccines are considered, each with a different combination of efficacy against susceptibility, infectiousness and disease.

In attempting to reduce uncertainty of this complex space, we considered different mass vaccination scenarios tailored to the current situation in Australia, while varying the key intervention parameters. As a result, we identified several salient trade-offs between the (pre-pandemic) vaccination and (future) lockdown requirements. Qualitatively, these trade-offs are not dissimilar to those reported in [19], however, the thresholds which we quantified for Australia are specific for a more refined, hybrid, mass vaccination campaign (with priority and general vaccines). We also reinforce the findings of [20] with respect to separate components of the vaccine efficacy: future outbreaks become preventable only when the distributed vaccines avert transmission as well as disease. Crucially, no combination of realistic vaccine efficacy values was found to completely eliminate the pandemic threat in Australia without population-scale NPIs, under the

feasible vaccination coverage extents that we simulated. This is somewhat different from the results of [20], where such an outcome was shown to be theoretically possible in UK, but only by adopting a highly efficacious vaccine. The necessity of partial lockdowns is in concordance with the analysis of [21] which highlighted the difficulties in lifting NPIs under realistic vaccination strategies considered in France.

There is a remaining uncertainty about the clinical efficacy of ChAdOx1 nCoV-19 (Oxford/AstraZeneca) vaccine. An interim analysis of four randomised controlled trials carried out in three countries (Brazil, South Africa, and the UK) between April and November 2020 suggested a vaccine efficacy of 62.1% (95% CI: 41.0–75.7) in participants who received two standard doses separated by four weeks [22]. When two standard doses were separated by 12 weeks or longer, the vaccine efficacy was observed to be higher at 81.3% (95% CI: 60.3–91.2) [23]. However, the follow-up study did not explicitly account for seasonal effects and variation between the trial countries in terms of the epidemic intensity (as well as circulating viral variants). In addition, these trials were not designed to discriminate between vaccine efficacies by dose interval, and therefore, these encouraging "post-hoc exploratory findings could be biased" [24]. Given the reported wide confidence intervals and a possible bias, we adopted a conservative estimate for efficacy of the general vaccine (i.e., 60%), while varying different components of the efficacy in a broader range. Furthermore, we found that our results regarding the lockdown compliance rate required for elimination did not change when we increased the efficacy of the general vaccine from VEc = 0.6 to VEc = 0.75 (Figure S5).

Our finding, that herd immunity is not attained even when a large proportion (88%) of the population is vaccinated, can be explained as a consequence of two correlated sources of heterogeneity. The first is structural, and occurs due to the unavoidable clustering of children in schools and classrooms. The second is imposed by the choice to place children at low priority for immunisation (due to typically low disease severity in this cohort). In Australia, school-aged children (aged from 5 to 18 years), comprise approximately 15% of the total population (with about 5% composed of children under the age of 5). Therefore, even with 90% of the population vaccinated, roughly half of the child population will remain fully susceptible to the virus. Of those who are vaccinated, very few (if any) will receive the priority vaccine. In our ABM, the result is an interconnected subpopulation with high susceptibility. During outbreaks, this produces deviations from the homogeneous approximation in the form of heterogeneous epidemic spread strongly biased towards school-aged children.

It has been widely established that children are at much lower risk of severe COVID-19 disease. However, estimates of transmission rates among children are made difficult by the

relatively low rate of symptom expression in young cohorts [25]. The current advice from the United States CDC suggests an emerging consensus that transmission rates in young people are similar to those in the adult population [26]In our model of COVID-19 transmission, we did not truncate the susceptibility or infectiousness of children but we did assume a lower rate of symptom expression, in line with the available evidence [1].

Taken together, these factors mean that age-stratified vaccine prioritisation represents a trade-off: by comprehensively vaccinating older adults with the priority vaccine, the system ensures lower levels of severe disease in the highest-risk cohort, even with low levels of coverage. However, our results demonstrate that this may come at the cost of precluding eventual herd immunity as the virus continues to spread slowly among the (largely asymptomatic) young cohort. Meehan *et al.* simulated optimised vaccination strategies and demonstrated that in order to achieve herd immunity, those at highest risk of *transmission* must be targeted for immunisation [27]. In our model, infections in children produce a high transmission risk, despite presenting a low risk of symptomatic disease. In this context, and given the current uncertainty with respect to efficacy of COVID-19 vaccines on transmission, our results demonstrate that the Australian strategy trades *possible* herd immunity for a reduction in severe case load potential.

A. Limitations and future work

In comparison with [1], our model included a more refined natural history of the disease, calibrated to recent outbreaks which occurred after the first wave in Australia. Nevertheless, the model does not explicitly capture in-hotel quarantine, hospitalisations, and in-hospital transmissions. This limitation is offset by the fact that in Australia's vaccination plan, healthcare and border control professionals are included in the priority vaccination phase, carried out in a pre-pandemic mode. We also do not systematically quantify mortality rates, and do not elaborate on the expanding disease surveillance capacity and standard clinical pathways in Australia [28].

As we pointed out, no herd immunity is attainable under realistic and feasible conditions. This outcome has several ramifications beyond the direct consequences of future partial lockdowns. On the one hand, the main reason for the inadequate collective immunity is the existence of highly clustered, networked communities (e.g., educational, religious and community groups, etc.). This highlights the need for a more sophisticated simulation of contact networks , in addition to the workplace/school environments generated from the census data. On the other hand, the lack of herd immunity may affect the behaviour of the "free-riders" who typically exploit the collective protection of mass immunisation while not committing to the vaccination themselves. This may create a feedback loop, reducing vaccine hesitancy in the near- to

mid-term, and generating long-term oscillatory dynamics in vaccine adoption [29]. Another caveat is that our ABM population is matched to the latest Australian Census data, which was collected in 2016. This produces a model population of 23.4M individuals, which is smaller than the current Australian population by approximately 2M people. Due to this discrepancy, the coverage proportions defined for fixed numbers of vaccines are slightly inflated in our simulations (by about 8%), relative to what would be achieved with the current population count. Because we did not identify a threshold in epidemic severity as a function of vaccine coverage in our hybrid scenarios, we do not expect this discrepancy to qualitatively alter our results.

V. CONCLUSION

In this work, we extended a high-resolution agent-based model of COVID-19 mitigation and control to simulate the effects of coupled vaccination and non-pharmaceutical strategies on future outbreaks in Australia. We found that, combined with case-targeted interventions, a completed mass-vaccination campaign using both a 90% effective vaccine for priority populations and a 60% effective vaccine for the general population would dramatically slow viral spread but would not produce herd immunity. If a community transmission outbreak were to occur during or after the vaccination campaign, population-scale non-pharmaceutical interventions (i.e., lockdown) would be necessary to curb transmission. In our simulations, the required extent and duration of these measures decreased gradually with the level of vaccination coverage obtained by the time of the outbreak. For realistic endpoint conditions, with 90% of the population vaccinated, the required lockdown intensity and initial epidemic growth rate decreased by 43% and 52%, respectively. Due to coupling between these two factors (broadening of the epidemic curve and increased effectiveness of nonpharmaceutical interventions), the severity of epidemics as measured by the peak number of new cases over a 24hr period decreased by up to two orders of magnitude under plausible mass-vaccination campaign endpoint conditions.

With respect to the prognosis for future outbreaks of COVID-19 in Australia, several important questions remain unaddressed by our study. Because we did not model the effect of vaccination on hospital case load, medical infrastructure, and mortality, our results do not directly inform estimates of the trade-off between the socioeconomic costs of lockdown and the human cost of allowing a low level of COVID-19 transmission. Finally, at the time of writing, several SARS-CoV-2 variants of concern have been identified. Among them, variant B.1.1.7, which emerged in the United Kingdom and has spread globally, was associated with increased transmission potential: specifically, its R_0 was estimated to be 43-90% (95% CrI: 38–130%) higher than reproduction number of preexisting variants [30]. Variant B.1.351 (originally iden-

tified in South Africa) has been provisionally associated with lower rates of neutralisation by polyclonal antibodies, and variant P.1 (thought to have originated in Brazil) has been associated with a major outbreak in a population thought to be effectively immune [31, 32]. Given the currently low level of understanding about the implications of these variants in future outbreak scenarios, our results should be viewed as optimistic guidelines that assume the continuing vaccination efforts can keep pace with the evolution of SARS-CoV-2.

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S1. SUPPORTING INFORMATION

A. Constraints on vaccine efficacy

The efficacy of a vaccine can be decomposed into three components:

- the efficacy for susceptibility (VEs) which determines the level of immunity induced in susceptible individuals
- the efficacy for disease (VEd) which determines protection against symptomatic illness if a vaccinated individual is infected
- efficacy against infectiousness (VEi) which determines how contagious a vaccinated individual will be if they become infected

The clinical efficacy (VEc), which is measured in stage-3 clinical trials and corresponds to the reported efficacy numbers for both vaccines, is a function of VEs and VEd, neither of which are directly measured:

$$\text{VEc} = \text{VEd} + \text{VEs} - \text{VEsVEd}, \tag{S1}$$

while the efficacy for transmission, VE, which is the value of interest for computing herd immunity is a function of vaccine coverage (c), VEi, VEs, and VEd. Because VEi is not constrained by VEc through Equation S1, the population-level effectiveness is currently uncertain for both vaccines.

In the worst-case scenario, the factor VEd would account for 100% of the clinical efficacy (VEd = VEc, VEs = 0), and the efficacy against transmission would be negligible (VEi = 0), which would leave all unvaccinated individuals unprotected. In this scenario, herd immunity is not possible per se (though complete coverage would provide clinical protection to the entire population). In the best-case scenario, efficacy against symptoms would be maximised (VEd = VEc), protecting all vaccinated individuals regardless of their infection status, while the unknown parameter VEi would take a value of 1, and all transmission from vaccinated individuals would cease, protecting both vaccinated and unvaccinated subpopulations. In reality, the true efficacy values will lie between these extremes, and may be correlated. For example, because symptoms may increase the contagiousness of those infected, VEd can positively influence VEi. On the other hand, symptoms can also lead to case detection and behavioural change, which may produce a negative relationship between VEd and VEi.

B. Vaccine efficacy: homogeneous approximation

Here, we give a homogeneous approximation for vaccine efficacy and the associated herd immunity thresholds as functions of VEs, VEi, and VEd. We derive an expression for overall reduction to force of infection provided by a vaccination program with coverage $c \in [0, 1]$ as follows:

$$\text{VE} = 1 - F_{vax}/F_o \,, \tag{S2}$$

where F_o is proportional to the overall force of infection when none of the population is vaccinated, and F_{vax} is the relative reduction to force of infection produced through vaccination. For a given prevalence of infection $p(\text{infected}) = N_{\text{infected}}/N_{\text{tot}} << 1 = 1 - p(\text{susceptible})$, and assuming a negligible recovered population, we compute F_o as:

$$F_o = \beta_o^2 \; p(\text{infected}) \; p(\text{susceptible}) \,, \tag{S3}$$

in which β_o is the average force of infection produced by an infected individual who is unvaccinated:

$$\beta_o = \beta(p_s + a(1 - p_s)) \tag{S4}$$

in which β is the force of infection from a symptomatic individual, p_s is the probability of expressing symptoms if infected and unvaccinated (in this work we assumed a symptomatic fraction of $2/3$) and a is the factor by which force of infection is reduced if an infected agent is asymptomatic (for this work $a = 0.3$). The $p(\text{infected})p(\text{susceptible})$ term in Eq. (S3) captures the potential for interaction between susceptible and infected individuals, the potential for transmission given interaction is given by one of the β_o terms, and the infectious potential of the newly infected individual, should transmission take place, is given by the second β_o term.

The relative force of infection with vaccination, F_{vax}, can be found similarly by summing the potential for contact and transmission between individuals with different vaccination status:

$$F_{\text{vax}} = F_{\text{unvax}\to\text{unvax}} + F_{\text{unvax}\to\text{vax}} + F_{\text{vax}\to\text{unvax}} + F_{\text{vax}\to\text{vax.}} \,, \tag{S5}$$

where

$$F_{\text{unvax}\to\text{vax}} = \beta\beta_{\text{vax}}(1 - \text{VEs}) \; p(\text{infected, unvaccinated}) \; p(\text{susceptible, vaccinated}) \,, \tag{S6}$$

in which

$$\beta_{\text{vax}} = \beta(1 - \text{VEi})\ [p_s(1 - \text{VEd}) + a(1 - p_s(1 - \text{VEd}))]\,, \tag{S7}$$

is the average force of infection from a vaccinated individual who becomes infected. Note that both VEi and VEd play a role in computing β_{vax}. The remaining terms of Eq. (S5) are:

$$F_{\text{vax}\to\text{unvax}} = \beta_{\text{vax}}\ \beta_o\ p(\text{infected, vaccinated})\ p(\text{susceptible, unvaccinated})\,, \tag{S8}$$

and

$$F_{\text{vax}\to\text{vax}} = \beta_{\text{vax}}^2\ (1 - \text{VEs})\ p(\text{infected, vaccinated})\ p(\text{susceptible, vaccinated})\,. \tag{S9}$$

The joint probabilities

- p(infected, unvaccinated),
- p(infected, vaccinated),
- p(susceptible, unvaccinated), and
- p(susceptible, vaccinated)

can be represented as products of the conditional and independent probabilities of infection, vaccination, and infection given vaccination:

$$p(\text{infected, unvaccinated}) = p(\text{infected} \mid \text{unvaccinated})\ p(\text{unvaccinated})\,, \tag{S10}$$

$$p(\text{susceptible, unvaccinated}) = p(\text{susceptible} \mid \text{unvaccinated})\ p(\text{unvaccinated})\,, \tag{S11}$$

$$p(\text{infected, vaccinated}) = p(\text{infected} \mid \text{vaccinated})\ p(\text{vaccinated})\,, \tag{S12}$$

$$p(\text{susceptible, vaccinated}) = p(\text{susceptible} \mid \text{vaccinated})\ p(\text{vaccinated})\,, \tag{S13}$$

where

$$p(\text{vaccinated}) = c\,, \tag{S14}$$

$$p(\text{unvaccinated}) = 1 - c\,, \tag{S15}$$

$$p(\text{susceptible} \mid \text{unvaccinated}) = 1 - p(\text{infected} \mid \text{unvaccinated})\,, \tag{S16}$$

$$p(\text{susceptible} \mid \text{vaccinated}) = 1 - p(\text{infected} \mid \text{vaccinated})\,, \tag{S17}$$

$$p(\text{infected} \mid \text{unvaccinated}) = \frac{p(\text{infected}) - p(\text{infected} \mid \text{vaccinated})\ p(\text{vaccinated})}{1 - p(\text{vaccinated})}\,, \tag{S18}$$

and

$$p(\text{infected} \mid \text{vaccinated}) = p(\text{infected})\ (1 - \text{VEs})\ \frac{\beta_{\text{vax}}}{\beta}\,, \tag{S19}$$

where the ratio β_{vax}/β gives the effect of VEd and VEi on infectiousness of vaccinated individuals (Eq. (S7)). Here, the state denoted “infected" would more precisely be described as “infectious", because VEi and VEd reduce transmission potential without reducing the infection probability of vaccinated individuals.

Assuming that the effective reproductive ratio R is proportional to the force of infection F_{vax}, it can be computed from the total efficacy, VE, as

$$R = R_0(1 - \text{VE}), \tag{S20}$$

where R_0 is the basic reproductive number for the epidemic, which we set to $R_0 = 2.75$ in the present work. Using $R_0 = 2.75$, Fig. S1 demonstrates the coverage threshold for herd immunity as a function of VEi, VEs, and VEd given VEc = 0.6 (Fig. S1a), and VEc = 0.9 (Fig.S1b).

The homogeneous approximation corresponds qualitatively to the results of the ABM which demonstrates a dramatic decrease in peak prevalence after increasing coverage crosses the $R = 1$ boundary estimated by evaluating Eq. (S20) for $R_0 = 2.75$ (Fig. S2). However, while prevalence is dramatically decreased, we still observed positive growth rates for parameter combinations which correspond to $R < 1$. Furthermore, for the less-effective general vaccine, the boundary computed through the homogeneous approximation does not correspond to a large decrease in peak prevalence when VEi ≤ 0.5 (Fig. S2a and S2c). For the priority vaccine (VEc = 0.9), the threshold computed through the homogeneous approximation matched the observed nonlinear

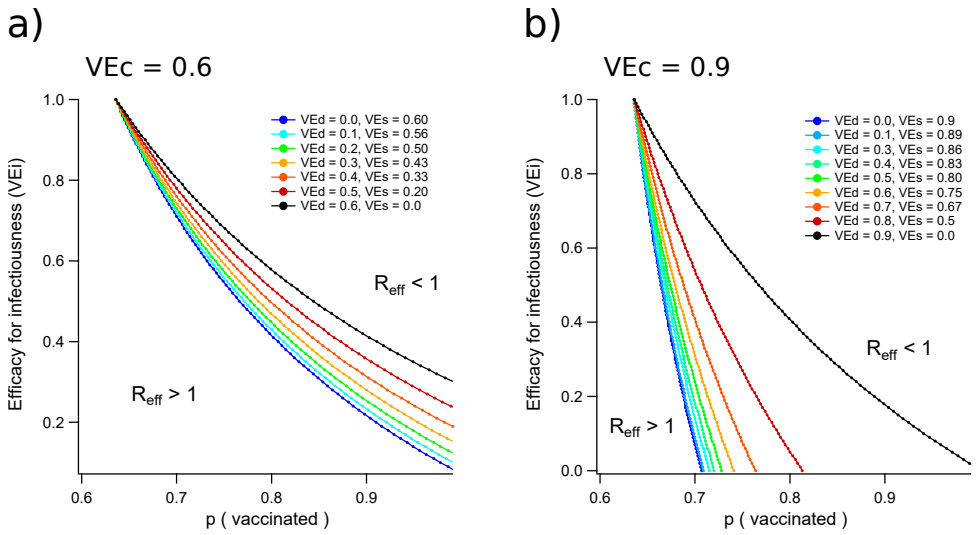

FIG. S1. Herd immunity thresholds as functions of efficacy for infectiousness (VEi) and vaccine coverage (p(vaccinated)) computed through a homogeneous approximation of vaccine effectiveness taking into account three forms of vaccine efficacy (VEs, VEd, and VEi). Subplot (a) shows thresholds for the general vaccine, with clinical efficacy VEc = 0.6, while subplot (a) shows thresholds for the priority vaccine with clinical efficacy VEc = 0.9.

decrease in peak prevalence more closely (Fig. S2b, and Fig. S2), but finite epidemics were still observed for all cases. The boundary computed by the homogeneous approximation should therefore be viewed as an optimistic estimate of coverage threshold.

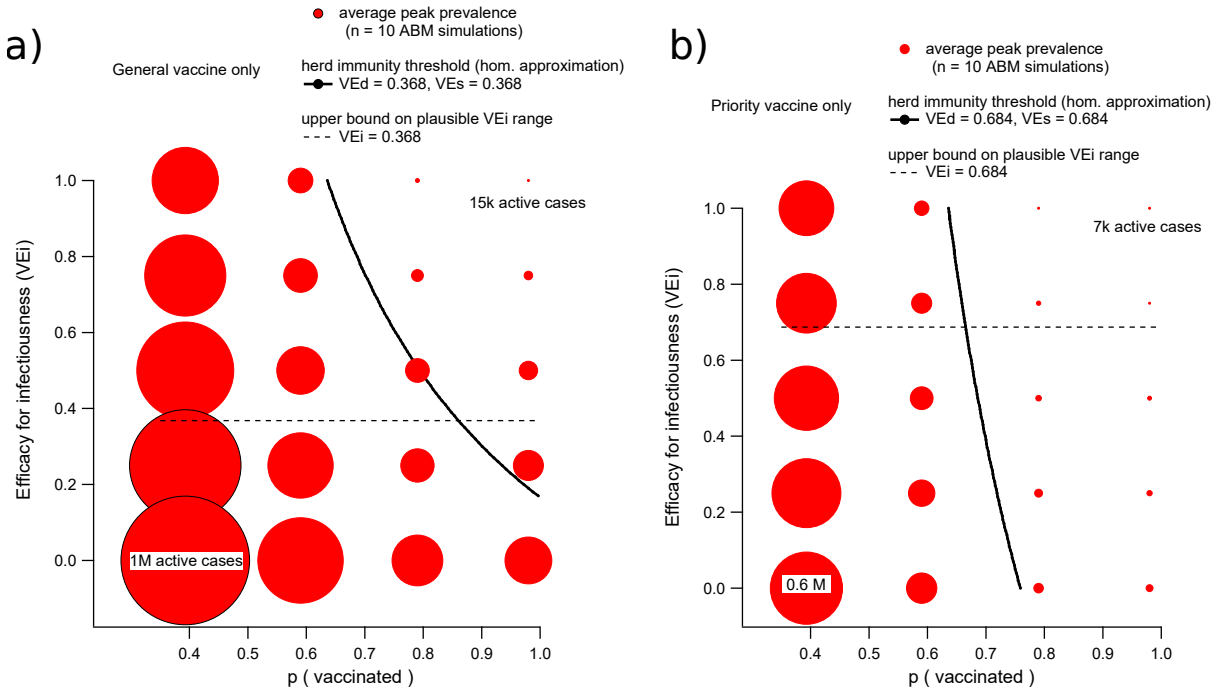

FIG. S2. Alignment of ABM results with homogeneous approximation of herd immunity thresholds for the general (a) and priority (b) vaccines. The peak prevalence values shown here (red circles) generated by the ABM over a range of values for coverage and vaccine efficacy against infectiousness. The solid black lines give the coverage thresholds for herd immunity estimated by the homogeneous approximation, and the dashed lines illustrate conservative practical upper bounds on VEi. Here, central values of efficacy against disease and susceptibility were used (VEd = VEs = $1 - \sqrt{1 - \text{VEc}}$).

C. Model calibration

Primary calibration was performed by first tuning disease parameters κ, and the bounds of T_{symp} for a basic reproductive number falling within the range specified in literature reports [12]. To compute R_0 for a given set of parameters, we performed an age-group biased micro-simulation Monte Carlo estimate of secondary cases produced by a typical index case as described in our previous studies [1, 18].

We then tuned the parameters defining case-targeted NPIs to match the early incidence data issued in Australia during the initial wave of COVID-19 in March 2020. The resulting incidence growth rates generated by our ABM (growth rate ≈ 0.118) lie between the plausible values estimated for the first wave of COVID-19 (growth rate $\approx [0.1, 0.2]$ Fig. S3a). If the first three cases are ignored when determining the growth rate for the first wave, the calculated rate doubles (Fig. S3a), but provides a better fit to the remaining case data. It is unclear whether this discrepancy occurred due to ineffective case ascertainment (due to e.g., delays to initial surveillance efforts), or to stochastic die-out of the outbreak that produced the first recorded cases.

On the other hand, the growth rate produced by our ABM closely matches the rate estimated

parameter	value	distribution	notes
κ	2.4	NA	global transmission scalar
T_{inc}	5.5 days (mean)	lognormal($\mu = 1.62,\ \sigma = 0.418$)	incubation period
T_{symp}	10.5 days (mean)	uniform [7, 14]	symptomatic (or asymptomatic) period
a	0.3	NA	asymptomatic transmission scalar
$p_{\text{ symptomatic}} \mid$ adult	0.67	NA	probability of symptoms (age $<$ 18)
$p_{\text{ symptomatic}} \mid$ child	0.134	NA	probability of symptoms (age 18+)
$p_{\text{ detect}} \mid$ symptomatic	0.227	NA	daily case detection prob. (symptomatic)
$p_{\text{ detect}} \mid$ asymptomatic	0.01	NA	daily case detection prob. (asymptomatic)

TABLE S1. Key control parameters for COVID-19 transmission model.

for the second wave, which began in early June, 2020, was confined to the state of Victoria, and occurred mostly within the urban area of Greater Melbourne [2] (growth rate ≈ 0.123, Fig. S3b). While our ABM simulates the entirety of Australia, early cases mostly arise in urban centres which contain the international airports from which importations are generated [17]. Therefore, a close match between the growth rates generated by our model and the rate observed during the beginning of the second wave in Greater Melbourne indicates that our simulations produce reasonable approximations to the early disease dynamics of an Australian outbreak.

The parameters defining population-scale NPIs (lockdown) were chosen to match the peak incidence and prevalence data produced during the first wave, which was suppressed with a national-scale lockdown (Fig. S4). While the initial incidence growth dynamics of our model are qualitatively similar to observations from the 1st wave (Fig. S3b, Fig. S4a), and well-matched to those observed during the second wave (Fig. S3b), there are some aspects of the observed data from the first wave that are not reproduced well by our model. In particular, simulated case prevalence is substantially lower than the number of active cases reported during the first wave. This is a direct consequence of our decision to model the infectious period after symptom onset in accordance to the period of replication competent viral shedding rather than the period over which a case may test positive (which can be longer by 1-2 weeks [8]). Therefore, in our model a case recovers and is no longer included in the prevalence count substantially earlier than it would be removed from an active case count ascertained through PCR tests. In addition, the cumulative incidence produced by our model is higher than what was observed in the first wave during March 2020 (Fig. S4b). However, this discrepancy only becomes substantial during the period after lockdown was implemented, so it can be interpreted as the consequence of our conservative estimates in the efficacy of lockdown on transmission in various contexts (Table S3). We chose conservative values for these parameters, so that our estimates of lockdown thresholds would err on the side of caution.

parameter	value	distribution	notes
p_{CI}	0.7	NA	case isolation compliance rate
p_{HQ}	0.5	NA	home quarantine compliance rate
T_{HQ}	14 d	NA	home quarantine duration
$f_{\mathrm{home}}(\mathrm{HQ})$	2	NA	NPI transmission scalar (HQ, home)
$f_{\mathrm{community}}(\mathrm{HQ})$	0.25	NA	NPI transmission scalar (HQ, community)
$f_{\mathrm{workplace}}(\mathrm{HQ})$	0.25	NA	NPI transmission scalar (HQ, workplace)
$f_{\mathrm{home}}(\mathrm{CI})$	1	NA	NPI transmission scalar (CI, home)
$f_{\mathrm{community}}(\mathrm{CI})$	0.25	NA	NPI transmission scalar (CI, community)
$f_{\mathrm{workplace}}(\mathrm{CI})$	0.25	NA	NPI transmission scalar (CI, workplace)

TABLE S2. Key control parameters for targeted NPIs. NPI transmission scalars multiply the force of infection produced by infected individuals in the specified contexts. Compliance rates determine the proportion of individuals who act in accordance with the specified measures (case isolation, CI; home-quarantine of household contacts, HQ).

parameter	value	distribution	notes
p_{LD}	variable	[0, 1]	lockdown compliance rate
T_{LD}	91 d	NA	lockdown duration
$f_{\mathrm{home}}(\mathrm{LD})$	1	NA	NPI transmission scalar factor (LD, home)
$f_{\mathrm{community}}(\mathrm{LD})$	0.25	NA	NPI transmission scalar factor (LD, community)
$f_{\mathrm{workplace}}(\mathrm{LD})$	0.1	NA	NPI transmission scalar (LD, workplace)
$\mathrm{AR}_{\mathrm{trigger}}(\mathrm{LD})$	2000	NA	cum. incidence triggering population-level NPIs

TABLE S3. Key control parameters for population-level NPIs (lockdown, LD).

parameter	value from ABM	target value	notes
R_0	2.75 [2.71, 2.80]	2.9 [2.39, 3.44]	basic reproductive ratio [12]
T_{gen}	7.14 [7.05, 7.23]	7.0 days [5.8, 8.1]	generation/serial interval [11]
growth rate	0.118 [0.110, 0.127]	0.10 [0.097, 0.103]	growth rate of case incidence (1st wave)
growth rate	0.118 [0.110, 0.127]	0.201 [0.170, 0.233]	growth rate of case incidence (1st wave, from day 21)
growth rate	0.118 [0.110, 0.127]	0.123 [0.102, 0.143]	growth rate of case incidence (2nd wave, VIC)
peak prevalence	2790, range [2623, 3059]	4935	peak case prevalence (1st wave)
peak incidence	377, range [336, 415]	497	peak case incidence (1st wave)

TABLE S4. Calibration targets for key model outputs.

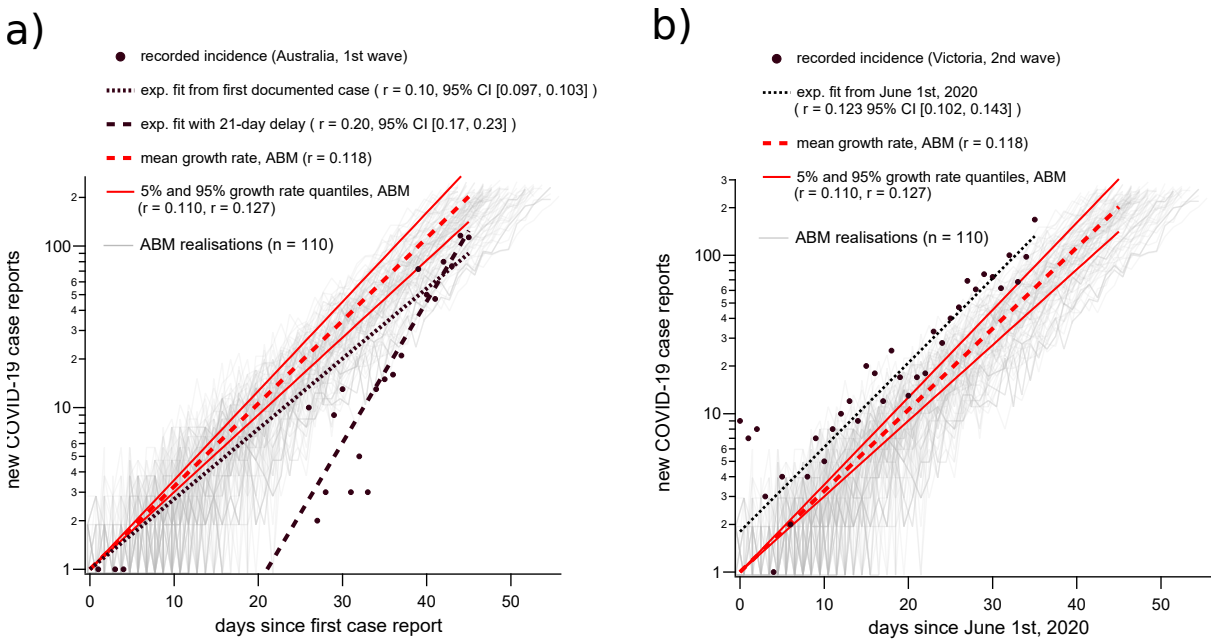

FIG. S3. The growth rate computed by the ABM with case-targeted NPIs approximately matches the rate computed for the 2nd wave of COVID-19 in Victoria. Incidence growth rates computed by the model are compared to case data recorded by the Australian Department of Health for the 1st wave of COVID-19 (beginning on Feb. 3rd, 2020) (a), and case data recorded by the Victorian Department of Health and Human Services for the 2nd wave of COVID-19 (b).

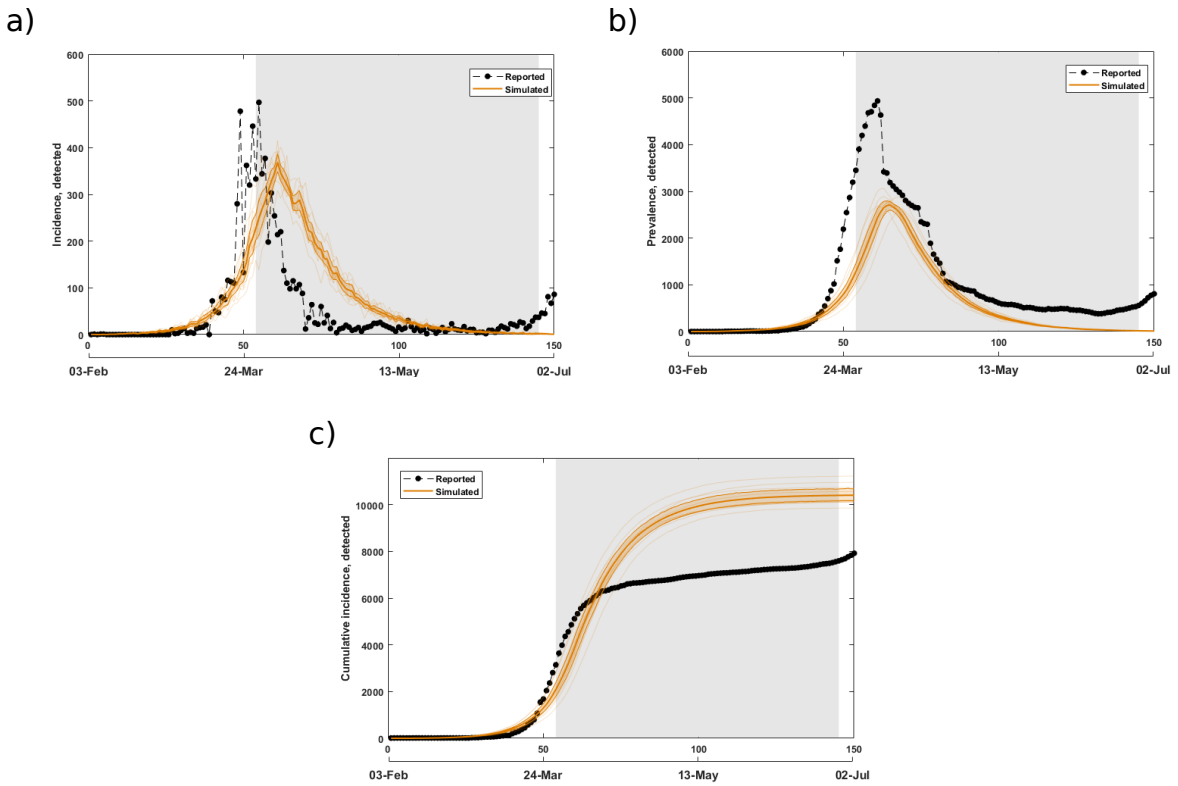

FIG. S4. Alignment of model results with recorded case data from the first wave of COVID-19 in Australia. Daily case incidence is shown in (a), with reported case data shown as black dots connected by dashed lines and mean case incidence produced by the ABM shown as a solid yellow line (shaded bands represent 95% bootstrap CI bounds). The output of individual instances of the ABM (n = 10) are shown as transparent yellow traces. Case prevalence (active case counts) are shown in (b), and cumulative case incidence is shown in (c). The grey shaded region corresponds to the lockdown period used in the ABM, which utilised a lockdown compliance level of 90% to match the conditions used in our previous work [1].

D. Sensitivity of lockdown threshold to efficacy of general vaccine

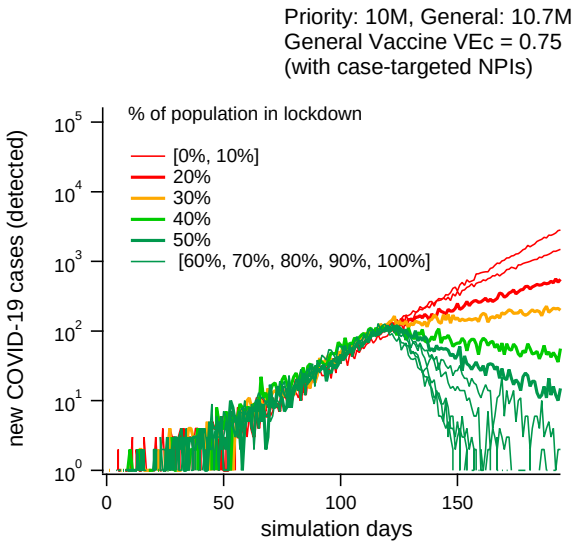

FIG. S5. Increasing the efficacy of the general vaccine does not substantially change the lockdown compliance threshold for elimination. Representative incidence trajectories for different lockdown compliance rates demonstrate an elimination threshold between 30% and 40%, for a vaccination program consisting of 10M priority vaccinations (VEc = 0.9), and 10.7M general vaccinations (VEc = 0.75; VEs = VEd = VEi = 0.5).

E. Epidemic severity as a function of vaccine efficacy and coverage

Here we provide full ABM results for epidemic severity as measured by peak prevalence and the timing of the prevalence peak from the start of the epidemic as functions of VEi, VEs, VEd, and coverage.

General Vaccination Only (no case-targeted NPIs), VEc = 0.6

population vaccinated: 2.3 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.6	1694634.5	114685.7	1650773.5	94762.76	1629223.3	55700.21	1614394.2	70262.99	1541667.8	68177.1
0.15	0.529	1724598.1	83620.22	1579926.6	137695.8	1640835	123713.3	1609051.1	71148.02	1481871.2	49215.28
0.368	0.368	1662242.1	107523	1686555	88769.91	1544641.2	70688.95	1551058.8	107626.6	1524159.7	98149.77
0.529	0.15	1697362.2	102395.5	1648734.5	127794	1574838.6	103819.1	1558597.3	113511	1534359.6	76555.07
0.6	0	1708183.7	97094.04	1594540.6	106683.9	1662164.7	104520.5	1554586.1	85596.02	1549125.5	116230.9

population vaccinated: 4.6 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.6	1505237.1	116179.2	1368045.3	79225.84	1302649.1	86563.75	1251543.1	75210.86	1168139.9	64141.94
0.15	0.529	1520296.4	47294.24	1410805	48832.03	1362716.4	88023.49	1235611.4	66532.53	1202481	48954.23
0.368	0.368	1454273.1	126355.4	1413590.2	59406.03	1343273.5	72415.24	1267068.4	84236.9	1176053.8	43491.7
0.529	0.15	1482833.2	71794.3	1370923.3	123898.6	1376196.7	55845.56	1290088.8	35683.09	1215891.7	56502.73
0.6	0	1482247.7	89800.27	1420829.8	85168.87	1327845.5	58239.02	1295320.7	88357.57	1216827.8	63557.39

population vaccinated: 9.2 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.6	1124721.8	49589.55	991879.3	29638.47	827919.5	39855.84	672680.2	25322.33	529775.9	35912.35
0.15	0.529	1128452.5	64588.07	946942.9	33731.32	822498.2	45921.16	677136.3	37302.91	518185.2	45857.21
0.368	0.368	1073843.2	58058.14	929609.9	21919.62	809352.1	34127.85	672488.6	43365.24	556715.8	65395.3
0.529	0.15	1033206.6	53213.15	907490.2	56951.44	803713.4	47486.77	650176.8	50225.4	568369.5	26118.64
0.6	0	1031877.7	33762.65	920601.3	30211.08	785933.5	37198.69	676706.8	50226.87	561132	38072.92

population vaccinated: 13.8 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.6	782430.4	46236.26	594283.7	33036.28	455563.2	16972.36	293817.3	25011.03	200549.8	20679.61
0.15	0.529	756981.4	28561.98	583797.2	41563.08	416508.9	38520.82	298144.4	12048.88	201873.5	21393.7
0.368	0.368	714819.4	47401.9	545324.5	20709.08	401734	23505.81	292381.2	24072.38	210428.5	21119.93
0.529	0.15	667972.2	33470	537972.8	24879.08	394888	26854.35	290691.9	25181.04	219565.6	18712.55
0.6	0	651102.9	31715.12	492315.1	44784.82	395277	18130.56	282486.3	27010.69	204984.3	21105.98

population vaccinated: 18.4 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.6	492845	33562.6	340379.4	16286.79	211716.2	19105.15	120268.4	9358.623	33475.3	10663.73
0.15	0.529	481244.6	33810.64	308188	35561.87	205059.2	12192.24	111171.6	12510.2	29190.7	11807.57
0.368	0.368	428070.1	12791.47	282926.7	24922.09	201250.8	12007.94	103354.3	18109.07	37918.3	12170.94
0.529	0.15	394573.2	15254.97	260931.3	16337.89	174307.8	12003.48	102516.4	17463.32	30722.7	10838.78
0.6	0	363117.6	17322.2	262074.6	15641.98	157372.2	17642.05	110274.8	9080.909	33057.3	8625.228

population vaccinated: 23 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.6	455460.9	28731.91	298658.9	11463.31	185596	11739.9	87004.4	15612.86	14853.8	6745.719
0.15	0.529	418211.5	28782.53	284518.3	20471.06	178650.7	9734.197	83356.6	9373.866	17043	7654.132
0.368	0.368	389640.8	23500.62	255471	13673.92	159463.3	13301.35	78322.4	11855.75	16497.8	6088.752
0.529	0.15	326542.5	27672.59	221718	23301.02	136824	18657.04	79826.2	14042.95	18215.6	7081.891
0.6	0	322056.4	19072.94	221617.7	17825.68	147733.7	8628.58	70438.5	12883.24	15481.4	6302.058

TABLE S5. Mean peak prevalence values and standard deviations produced by the ABM for various combinations of vaccine efficacy parameters and coverage levels, assuming a clinical efficacy of VEc = 0.6 and no case-targeted NPIs (n = 10 instances per scenario).

General Vaccination Only (no case-targeted NPIs), VEc = 0.6

population vaccinated: 2.3 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.6	120.5	1.649916	121.1	1.37032	123.3	3.12872	124	4.546061	122.8	2.820559
0.15	0.529	119.6	2.716207	120.7	4.347413	121.5	1.354006	122.1	2.601282	124.4	3.438346
0.368	0.368	121	1.333333	121.7	2.750757	120.7	1.05935	124	3.800585	123.8	3.705851
0.529	0.15	121.4	2.716207	122.1	2.558211	123.9	2.998148	124.6	3.306559	124.3	2.750757
0.6	0	121	1.154701	124	4.966555	123.2	2.973961	122.6	2.366432	125	4.027682

population vaccinated: 4.6 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.6	123.8	2.820559	125.5	3.308239	128.3	4.217688	129.5	0.971825	131.7	3.12872
0.15	0.529	124.2	3.155243	126.2	3.705851	125.9	3.17805	127.5	2.460804	128.6	3.806427
0.368	0.368	124	4.109609	124.8	3.32666	127.8	3.119829	127.3	4.831609	129.6	2.221111
0.529	0.15	124.7	3.164034	126.5	3.503966	128.9	3.314949	128.6	3.339993	131	2.44949
0.6	0	123.8	2.529822	126.4	3.470511	128.2	4.022161	130.4	3.893014	130.4	3.405877

population vaccinated: 9.2 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.6	126.6	3.204164	133	4.320494	137.5	4.719934	144.8	5.593647	152.3	6.272515
0.15	0.529	130.2	4.391912	133.5	3.778595	139.2	4.565572	145	3.620927	150.1	3.813718
0.368	0.368	134	3.018462	136.9	0.994429	140.8	3.457681	146.9	2.998148	151.7	8.152028
0.529	0.15	135	4.714045	140.6	4.299871	142.8	3.614784	149.5	7.61942	150.6	3.977716
0.6	0	136.4	2.319004	137.5	3.597839	143.8	4.54117	146.9	3.034981	154.1	5.646041

population vaccinated: 13.8 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.6	133.4	6.239658	140.8	3.675746	148.6	4.033196	161.4	6.131884	180	10.64581
0.15	0.529	136.5	2.953341	144.3	2.359378	154.3	6.733828	167.4	8.707596	184.7	7.103207
0.368	0.368	144	4.21637	152.6	5.232378	156.2	4.022161	167.2	8.879439	181.5	6.883959
0.529	0.15	147.8	3.675746	151.2	3.645393	159.7	7.211873	167.2	6.442912	178.9	8.319322
0.6	0	146.2	2.440401	152.5	4.034573	161.5	0.527046	169.4	9.72054	182.2	5.573748

population vaccinated: 18.4 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.6	137.8	5.116422	152.5	5.967505	168.7	11.33382	186.2	6.178817	195	0
0.15	0.529	145.4	3.134042	155.5	3.836955	168.9	5.586691	190.2	4.077036	195	0
0.368	0.368	152.6	5.561774	165.3	4.137901	176.5	4.428443	193.4	2.674987	195	0
0.529	0.15	159.7	4.831609	173.4	6.552353	181.3	8.393781	194.6	0.516398	195	0
0.6	0	162.9	6.244108	169.4	5.232378	187.8	6.8443	193.6	2.716207	195	0

population vaccinated: 23 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.6	139.8	3.392803	150.8	5.028806	171	6.377042	194.6	0.699206	195	0
0.15	0.529	143.5	3.24037	158	5.120764	174.8	6.425643	193.8	3.119829	195	0
0.368	0.368	150.8	5.223877	168.4	7.662318	182.3	7.860591	195	0	195	0
0.529	0.15	164.6	6.686637	171.8	9.750214	186.9	7.06242	194.8	0.421637	195	0
0.6	0	164.6	6.686637	174.1	5.3427	188.7	4.164666	194.9	0.316228	195	0

TABLE S6. Prevalence peak times (means and standard deviations) produced by the ABM for various combinations of vaccine efficacy parameters and coverage levels, assuming a clinical efficacy of VEc = 0.6 and no case-targeted NPIs (n = 10 instances per scenario).

Priority Vaccination Only (no case-targeted NPIs), VEc = 0.9

population vaccinated: 2.3 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.9	1629065.7	118132.4	1558927.8	84303.1	1513835.4	59419.12	1552729.2	70771.27	1507688.9	58007.78
0.225	0.871	1615009.9	91163.03	1565056.7	78768.3	1538809.8	105203.4	1506884	128797.5	1484369.8	147577.9
0.684	0.684	1601246.1	65987.7	1572014.1	96047.59	1540017.3	97252.37	1519191.9	97681.67	1456609.4	89988.75
0.871	0.225	1547327.2	97824.33	1525381.4	105564.3	1446064.6	104216.6	1516560.3	93471.38	1470250.9	106956.8
0.9	0	1547096.5	61128.53	1532795.5	67554.32	1467230	72887.83	1493777.1	52880.25	1511284.3	83797.52

population vaccinated: 4.6 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.9	1316015.6	43693.05	1271002.5	119080.1	1193446.9	93584.92	1129114.7	77343.94	1012743.8	61268.1
0.225	0.871	1254482.9	53773.69	1232219.5	74260.9	1188284.9	63330.25	1148032.6	48003.87	1104527.1	78093.73
0.684	0.684	1253338.8	57691.84	1162648.3	58968.75	1167649.6	79240.56	1111467	62381.44	1058284.7	57091.85
0.871	0.225	1156430.3	84833.06	1133062	71424.78	1116377	75612.76	1133710.4	35693.84	1042650.4	129981.6
0.9	0	1188472.4	48574.04	1150525	82061.35	1189090.5	36449.38	1128339.3	78125.2	1119165.9	68528.22

population vaccinated: 9.2 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.9	785889.1	51156.26	675623.9	38661.52	589043.7	67383.19	498393.8	45668.48	450494.3	28648.95
0.225	0.871	738497.3	34135.75	635593.1	41766.42	616823.8	25531.99	527691.8	24908.78	429800.7	24720.67
0.684	0.684	605547	36111.95	578563	36259.45	537648.3	33615.18	500529.8	31666.51	459785.2	30153.37
0.871	0.225	563114.5	27517.63	540163.2	33222.46	509098.5	40592.38	485696.6	45628.59	472644.4	29588.81
0.9	0	559101.5	21303.74	533271.2	43615.63	506275	39206.24	478572.1	42094.62	473178.9	31547.24

population vaccinated: 13.8 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.9	397363.5	24330.41	313491.7	22589.47	253450.2	18009.29	191671.4	13430.72	137890	15179.74
0.225	0.871	365332.3	18390.4	292478.8	8246.286	239912	18723.9	185624.9	10953.72	129741.2	18029.72
0.684	0.684	256899.2	16007.49	225031.1	18316.68	194371.7	14202.9	172948.1	12767.79	127799.6	14211.65
0.871	0.225	209773.5	16926.97	192410.8	13302.96	169416.3	12076.64	173010.6	10550.52	139962.3	9600.772
0.9	0	201652.1	14098.89	193539.6	15888.58	179255.5	11082.19	159967.9	17345.61	148767.6	14406.14

population vaccinated: 18.4 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.9	147325.2	11561.39	112112.6	7923.788	82079.7	6742.019	55088.6	7576.666	18783.9	8424.104
0.225	0.871	131306.2	6567.362	99277.4	11099.41	75408.9	6814.06	53842.6	3747.046	16543.8	3916.69
0.684	0.684	85748	5697.925	69852.8	6336.83	52995.8	4231.045	41969.2	8916.66	21246.5	7851.876
0.871	0.225	67441.2	6170.463	56792.8	4767.832	45490.7	10137.06	33887.8	7233.689	21113	7980.867
0.9	0	60703.4	4397.363	54339.2	4363.054	46486.5	5919.98	32434.9	6012.33	17744.5	5052.494

population vaccinated: 23 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.9	117739.4	7885.719	85525.9	8267.443	63316.5	7301.414	34653.5	6634.042	8620.1	2654.243
0.225	0.871	100089.8	12422.69	76300.6	8757.573	55531.8	9586.911	34958	5518.997	10057.8	2170.623
0.684	0.684	63370.4	7490.222	51529.8	5862.205	38204.4	5717.474	16616.4	5998.315	7073.2	3514.551
0.871	0.225	46563.9	4712.144	36837.6	3878.499	23336.4	6127.356	14895.1	5990.814	7619.5	3763.814
0.9	0	44418.5	4034.131	36728.5	7117.539	26205.2	4300.458	16642.2	3811.702	9455.1	1685.901

TABLE S7. Mean peak prevalence values and standard deviations produced by the ABM for various combinations of vaccine efficacy parameters and coverage levels, assuming a clinical efficacy of VEc = 0.9 and no case-targeted NPIs (n = 10 instances per scenario).

Priority Vaccination Only (no case-targeted NPIs), VEc = 0.9

population vaccinated: 2.3 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.9	120.6	1.837873	122.3	2.710064	124.1	2.884826	122.7	2.668749	123	2.211083
0.225	0.871	120.7	4.137901	122	2.828427	122.5	2.013841	124.2	5.181162	122.6	3.565265
0.684	0.684	122.3	1.766981	121.9	2.469818	121.7	3.368151	122.7	2.359378	124.5	3.566822
0.871	0.225	122.4	3.339993	124	2.828427	126.7	2.983287	124.3	4.498148	123.9	4.175324
0.9	0	122.7	2.406011	121.3	3.683296	123.6	3.657564	123.7	3.020302	125.3	3.267687

population vaccinated: 4.6 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.9	123.3	2.869379	125.2	4.104198	128	2.211083	129.3	3.560587	130.7	4.191261
0.225	0.871	124.5	3.100179	126.4	4.550946	126.5	3.566822	131.5	3.308239	130	2.581989
0.684	0.684	126.6	2.951459	128.6	0.843274	130.2	4.417138	130.1	3.28126	129.4	3.405877
0.871	0.225	128.2	3.852849	127.5	3.27448	129.9	3.928528	130.4	2.412928	128.7	3.917199
0.9	0	128.9	1.286684	129.7	3.433495	128.6	1.173788	130.1	3.754997	132.5	3.439961

population vaccinated: 9.2 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.9	132.9	4.931757	135.9	4.605552	140.1	3.665151	150.1	8.332667	151.3	4.967673
0.225	0.871	134.3	3.772709	139.8	3.705851	141.2	4.049691	146.1	5.743595	154	6.63325
0.684	0.684	142.6	4.115013	142.6	5.146736	145.9	4.383048	149.7	3.917199	152.4	5.966574
0.871	0.225	142.9	4.909175	144.9	3.813718	150.1	7.202623	147	4.618802	151.1	3.842742
0.9	0	143.9	4.748099	143.6	3.025815	148.4	3.565265	151.9	7.385421	150.3	3.973523

population vaccinated: 13.8 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.9	141.1	4.148628	147.4	5.146736	155.7	6.360468	164.5	6.363961	178.2	8.573862
0.225	0.871	146.9	6.951419	150.2	5.245104	158.2	6.8443	167.1	6.539623	182.3	8.512083
0.684	0.684	160.1	3.95671	164.9	7.40045	163.9	5.3427	175.5	6.222718	179.9	7.781031
0.871	0.225	162.8	8.954204	166.1	4.012481	169.7	8.446564	174.8	5.533735	180.6	7.676805
0.9	0	161.4	3.627059	164.6	8.342661	166.1	6.919377	175.7	8.340663	181.1	5.743595

population vaccinated: 18.4 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.9	147.9	6.261878	158.9	5.087021	173.9	7.894442	190.5	5.582711	195	0
0.225	0.871	154.2	4.491968	165.1	6.62403	179.1	6.0452	192.5	3.503966	194.9	0.316228
0.684	0.684	181.4	6.686637	186.9	4.976612	193.3	3.020302	194.8	0.421637	194.9	0.316228
0.871	0.225	192.8	2.780887	193.9	0.737865	194.2	0.632456	194.8	0.421637	195	0
0.9	0	191.3	4.164666	194	0.666667	194.5	0.527046	194.9	0.316228	195	0

population vaccinated: 23 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.9	146	5.577734	160.6	5.521674	180	8.51143	194.5	0.707107	195	0
0.225	0.871	159.1	9.243015	168.5	9.617692	183.1	7.549099	194.6	0.516398	195	0
0.684	0.684	182	6.463573	191.9	3.414023	194.1	0.875595	195	0	195	0
0.871	0.225	194.3	0.823273	194.6	0.699206	194.8	0.421637	194.9	0.316228	195	0
0.9	0	194.3	0.483046	194.7	0.483046	194.7	0.483046	194.9	0.316228	195	0

TABLE S8. Prevalence peak times (means and standard deviations) produced by the ABM for various combinations of vaccine efficacy parameters and coverage levels, assuming a clinical efficacy of VEc = 0.9 and no case-targeted NPIs (n = 10 instances per scenario).

General Vaccination with Case-Targeted NPIs, VEc = 0.6

population vaccinated: 2.3 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.6	1113051.2	60664.25	1079826	55995.69	1074758.1	45207.73	1014519.5	46393.39	985386.7	63393.86
0.15	0.529	1103756.7	54021.73	1091477.2	42361.65	1024048.3	64749.58	1000782.7	67769.31	962575.8	61778.18
0.368	0.368	1083806.7	49127.63	1057838.9	46109.82	1028498.4	45318.65	989216.4	52658.29	977531.6	30026.45
0.529	0.15	1081031.8	57337.99	1064064.2	36637.05	1001492.8	68246.37	1012250.6	50735.45	955420.2	24936.49
0.6	0	1067999.1	69809.29	1058287.9	48887.51	999627.8	63958.35	961113.4	48361.51	965021.2	44014.73

population vaccinated: 4.6 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.6	1025677.7	54690.74	936910.2	54584.45	885363.9	18324.13	813439.6	42875.69	757145.2	41965.16
0.15	0.529	973756.7	55094.83	959809.9	39744.55	861309.6	33682.4	786742.9	51363.3	716046.1	81924.8
0.368	0.368	936673	58725.81	919686.2	51559.13	844202.4	45537.43	789903	52008.84	745124.2	63176.07
0.529	0.15	931522.6	64024.41	878363.2	54853.85	807370.3	58907.39	761069.1	43778.51	752118.1	42142.34
0.6	0	912361.7	42640.34	859472.1	51068.59	830125.5	32412.18	780857.5	27292.05	734086.8	55126.59

population vaccinated: 9.2 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.6	845419.6	40563.83	697710.9	38958.54	561352.9	35756.62	479467.5	24046.08	358142.8	36509.29
0.15	0.529	781175.3	49602.95	670209.2	46151.6	528564.1	48536.74	448719.1	32054.54	352477.9	27744.6
0.368	0.368	715839.8	32035.17	601371	31468.07	490079.2	44454.85	425935.2	22686.69	378427.9	22203.19
0.529	0.15	669504	19392.47	580119.1	32623.35	489424.9	33239.25	417896.2	39176.22	360534.2	33177.56
0.6	0	638651.4	24022.73	548672.4	41779.15	486535	30413.5	428034.6	16035.04	367413.1	36723.12

population vaccinated: 13.8 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.6	649241.7	38376.56	481211.5	45229.99	358196.2	20149.03	253789.1	18893.47	140313.7	21920.3
0.15	0.529	598214.6	21613.18	452772.7	22541.9	332679.9	17785.86	237691.2	14346.11	127228.2	31439.23
0.368	0.368	508853	28770.99	385809.8	22758.99	290232.5	29325.04	221024.9	17205.76	126232.7	18514.99
0.529	0.15	420308.2	23145.83	339910.5	17893.81	257984.2	33778.42	202051.4	19410.84	130640.3	24199.2
0.6	0	395432.7	18894.12	312421.2	35383.36	251914.6	19649.08	194329.4	17788.94	134565.4	34257.66

population vaccinated: 18.4 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.6	504527.6	29175.68	353858.8	15427.77	213013.1	14053.28	65885.1	19224.41	5782.2	3725.691
0.15	0.529	428940	48423.61	294286.8	19433.57	184468.4	15411.88	64213.5	17065.8	7791.1	4056.096
0.368	0.368	331919.5	18598.22	228494.8	9453.499	130188	17802.13	46043.6	21071.76	7074.6	2639.598
0.529	0.15	273853.7	15544.15	196870.2	10531.21	114850.8	7150.97	33497.3	12365.15	8752.7	4517.487
0.6	0	236792.5	14609.4	164832.4	17455.76	90335.7	16517.35	31287.2	10296.76	7615.1	5185.758

population vaccinated: 23 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.6	481768	38162.51	320659.9	14579.02	180345.2	20400.94	51290.5	17471.15	3591.1	2174.384
0.15	0.529	407284.8	39451.75	277480	6298.987	156268.5	23465.22	40570.6	12750.37	3564.2	1427.981
0.368	0.368	318953.1	10855.92	210331.6	13341.43	115279.6	15073.09	27482.6	11159.76	3554.4	1760.959
0.529	0.15	244327.8	16617.74	155280.4	24028	73360.3	20247.86	20954.2	7426.625	3597.1	1883.115
0.6	0	212925.5	12623.49	142729.6	10414.58	62763.9	18131.34	17826.7	4490.861	2896.1	1919.26

TABLE S9. Mean peak prevalence values and standard deviations produced by the ABM for various combinations of vaccine efficacy parameters and coverage levels, assuming a clinical efficacy of VEc = 0.6 and active case-targeted NPIs consisting of case isolation, home-quarantine of household contacts of detected cases, and international travel restrictions (n = 10 instances per scenario).

General Vaccination with Case-Targeted NPIs, VEc = 0.6

population vaccinated: 2.3 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.6	133.9	2.514403	133.4	3.806427	136.1	3.28126	136.4	2.170509	137.1	2.884826
0.15	0.529	134.3	4.715224	134.5	2.798809	134.7	2.750757	136	3.800585	138.7	3.917199
0.368	0.368	136.8	2.699794	135.2	2.347576	136.6	3.238655	135.9	0.994429	137.3	3.164034
0.529	0.15	134.8	3.966527	134.7	2.710064	135.4	2.875181	136	3.333333	137	2.494438
0.6	0	135.7	1.159502	135.8	2.440401	137	3.464102	136.3	2.057507	136.3	4.24395

population vaccinated: 4.6 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.6	134.7	845419.6	138.1	3.665151	139.5	3.205897	142.1	2.233582	145.1	3.695342
0.15	0.529	137.8	781175.3	137.1	4.175324	140.9	3.60401	143.3	5.755191	148.1	6.505553
0.368	0.368	136.5	715839.8	138.3	3.335	141.6	3.025815	143.8	5.223877	142.1	4.121758
0.529	0.15	138.2	669504	139.6	2.91357	142.5	3.778595	142.6	1.505545	145	3.651484
0.6	0	138.5	638651.4	139.6	3.627059	141.4	3.747592	145.9	4.976612	145.1	4.306326

population vaccinated: 9.2 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.6	138	2.981424	145.5	4.062019	153.3	4.24395	158	3.197221	171.6	12.02959
0.15	0.529	140.4	2.674987	146.5	3.689324	154.4	5.420127	162.9	5.801341	169.4	6.25744
0.368	0.368	146.5	3.27448	151.4	4.623611	155.1	4.012481	160	5.981453	166.4	6.963396
0.529	0.15	148.4	4.221637	153.5	3.472111	159.2	6.941021	164.3	6.219146	167	9.225568
0.6	0	149.8	4.366539	155.9	4.840799	158.2	4.315347	161.6	3.204164	167.7	4.191261

population vaccinated: 13.8 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.6	143.8	5.287301	157.4	8.275801	167	3.711843	181.8	5.750362	193.8	3.794733
0.15	0.529	148	3.711843	157.8	2.699794	169.1	6.349978	183.4	8.221922	193.9	3.478505
0.368	0.368	156	3.620927	160.8	4.756282	176.6	7.471427	188.4	5.680376	194.7	0.948683
0.529	0.15	165	6.815016	170.4	4.168666	180.8	7.083627	192.3	4.522782	194.7	0.674949
0.6	0	165	4.853407	172.8	4.779586	181.9	6.190495	191.7	2.58414	194.7	0.674949

population vaccinated: 18.4 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.6	145.4	2.674987	160.5	4.994441	183.4	4.835057	195	0	195	0
0.15	0.529	154	4.666667	166.9	3.573047	191.5	3.778595	195	0	195	0
0.368	0.368	166.4	3.470511	180.3	6.896859	194.9	0.316228	195	0	195	0
0.529	0.15	178.8	4.825856	186.8	4.638007	194.7	0.948683	195	0	195	0
0.6	0	180.1	5.839521	194.4	1.264911	195	0	195	0	195	0

population vaccinated: 23 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.6	147.7	5.292552	164.6	4.526465	187.1	6.9033	195	0	195	0
0.15	0.529	154.9	4.408325	170.3	5.012207	193.1	3.414023	195	0	195	0
0.368	0.368	167.1	3.573047	184	4.807402	194.7	0.948683	195	0	195	0
0.529	0.15	179	5.792716	193.6	2.065591	195	0	195	0	195	0
0.6	0	183.7	5.716448	195	0	195	0	195	0	195	0

TABLE S10. Prevalence peak times (means and standard deviations) produced by the ABM for various combinations of vaccine efficacy parameters and coverage levels, assuming a clinical efficacy of VEc = 0.6 and active case-targeted NPIs consisting of case isolation, home-quarantine of household contacts of detected cases, and international travel restrictions (n = 10 instances per scenario).

Priority Vaccination with Case-Targeted NPIs, VEc = 0.9

population vaccinated: 2.3 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.9	1051743.5	63317.38	1057650.9	51286.25	1010179.2	71510.13	977927.9	60337.52	926397.7	81184.61
0.225	0.871	1030125.9	61916.41	1001845.5	52651.18	998986.2	47807.15	938575.1	61106.76	957048	44542.66
0.684	0.684	995824.4	22616.14	985706.7	46142.78	966397.8	57614.38	935862.2	56592.6	915874.3	69476.01
0.871	0.225	990080.5	53986.17	933987.8	49137.36	913596.5	64794.04	916175.6	39270.72	954710.6	27399.29
0.9	0	951071.2	71087.83	960617.4	47586.25	921967	49646.15	922040	34846.65	923791.5	49533.33

population vaccinated: 4.6 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.9	914219.7	50639.01	897505.5	35916.01	791166.2	44124.79	757750.5	40031.17	711349.3	31122.42
0.225	0.871	867342.4	60439.48	814683.5	58677.88	800068.7	34521.81	763880.6	29210.58	721389.1	17284.03
0.684	0.684	774125.9	52855.1	760709.6	42760.01	746806.8	48905.38	728887.7	42255.63	711026.9	36202.02
0.871	0.225	716828.5	52936.15	705732.4	48848.86	709034.4	38968.92	677431.4	41430.63	689777.1	38106.62
0.9	0	724564.5	27908.02	733091.5	35924.97	716175.1	24501.16	694378.2	42112.63	685770	26585.9

population vaccinated: 9.2 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.9	648378.7	29273.9	565461.6	26295.8	489218.7	18941.81	408263.8	33258.87	324865.2	35443.53
0.225	0.871	598804.1	32563.13	502990.7	21178.74	442792.7	27128.86	385424.1	15527.46	321500.4	16956.29
0.684	0.684	437357	28322.62	398257.9	22027.51	363415.7	24498.95	315360.2	27049.78	307086.1	20104.82
0.871	0.225	358295.6	25336.58	367286.2	17042.53	337421	9592.737	310218.4	20025.17	304483.8	16107.64
0.9	0	357604.1	8047.304	347697.8	20811.92	336234.2	24049.94	320398.3	16587.88	300442.3	22132.38

population vaccinated: 13.8 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.9	439183.3	25972	334772.3	28392.63	263076.5	11418.5	185688.7	19062.73	115913.3	18642.68
0.225	0.871	362355.9	24773.27	280590.7	27317.39	221842.4	25139.23	158886	23808.07	106186.9	16301.5
0.684	0.684	208399.2	12846.05	183061.7	12276.39	156684.1	15824.4	122523.2	21783.72	106321.8	22444.46
0.871	0.225	152516.9	8796.435	142644.9	8257.227	132220	12987.14	104919.6	18888.01	95117.7	13737.44
0.9	0	146360.6	8989.45	140100.9	13144.24	128913.5	9753.468	115373.4	9042.581	91831.8	9944.103

population vaccinated: 18.4 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.9	285944.3	13547.91	201497.7	12776.98	134175.3	9041.692	45575.2	13212.14	5989.5	2600.225
0.225	0.871	217278.4	10551.26	151968.9	17399.66	102044.4	13321.98	30908.4	9537.389	6375.5	3706.461
0.684	0.684	81415.4	8924.646	57520.5	6020.975	31756.3	9403.057	12547.6	3997.243	6950.6	2740.178
0.871	0.225	36130	9858.871	23746.8	4772.083	14857.4	5940.658	8967.8	1956.474	4822.3	2295.82
0.9	0	27585.2	7408.469	22635.1	3410.01	16004.6	4002.377	9104	2907.633	5131.5	1382.24

population vaccinated: 23 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd	prevalence	sd
0	0.9	250656.7	19854.06	183386.7	15484.43	114695.3	12987.93	31319.7	4416.007	3294.5	1138.83
0.225	0.871	192504.3	10360.06	133458.2	8835.682	75185.1	15131.4	18340.4	5132.497	2659.4	1562.184
0.684	0.684	65175.9	7744.362	37409.5	7900.39	16720.5	8642.882	7203.6	3522.024	2376.4	1100.971
0.871	0.225	20195.5	5969.454	12711.4	2318.5	7255.3	2048.643	5387.9	1698.449	1868.2	1163.189
0.9	0	17052.6	2035.515	11468.9	2527.022	7412.8	1791.776	4714.2	2141.233	2139.1	855.1846

TABLE S11. Mean peak prevalence values and standard deviations produced by the ABM for various combinations of vaccine efficacy parameters and coverage levels, assuming a clinical efficacy of VEc = 0.9 and active case-targeted NPIs consisting of case isolation, home-quarantine of household contacts of detected cases, and international travel restrictions (n = 10 instances per scenario).

Priority Vaccination with Case-Targeted NPIs, VEc = 0.9

population vaccinated: 2.3 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.9	134	1.632993	135.3	3.497618	135.3	3.40098	136.8	2.936362	137.8	4.104198
0.225	0.871	135.4	3.777124	136.8	3.645393	136.4	3.169297	136.3	2.002776	137.4	3.134042
0.684	0.684	135.4	1.429841	135.3	3.772709	135.2	1.873796	138.1	5.087021	138.4	3.596294
0.871	0.225	135.4	1.505545	138.2	3.583915	137.7	4.270051	137.1	2.806738	138.1	3.3483
0.9	0	136.9	3.813718	137.3	4.029061	136.7	3.743142	138.6	4.221637	136.4	2.75681

population vaccinated: 4.6 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.9	136.8	3.190263	138.5	5.212165	140.8	3.155243	144.1	5.237684	146.9	3.928528
0.225	0.871	139	3.197221	141.2	5.411921	142.2	6.696599	142.7	3.860052	143.7	2.790858
0.684	0.684	141.1	2.726414	140.6	4.087923	141.5	3.24037	143.2	2.820559	144.9	4.45845
0.871	0.225	143.5	5.275731	140.7	3.301515	142.4	3.627059	144.1	5.40473	144.6	4.273952
0.9	0	138	2.666667	144.4	4.880801	143.2	3.705851	144.2	3.823901	145	5.163978

population vaccinated: 9.2 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.9	142.6	4.427189	148.7	3.622461	151.6	5.189733	160.8	6.729702	168.2	7.330302
0.225	0.871	145.4	4.273952	152.4	4.452215	157	3.265986	161.6	4.788876	170	6.666667
0.684	0.684	153.8	4.565572	160.5	4.527693	163.4	3.339993	165.9	3.984693	168.2	4.104198
0.871	0.225	161.5	6.240548	163.2	6.285786	163.1	5.15213	165.2	5.865151	167.4	4.599517
0.9	0	157.3	4.24395	162.4	6.449806	160	5.077182	163.9	4.012481	166	4.496913

population vaccinated: 13.8 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.9	146.9	3.60401	158	5.354126	169.2	9.919677	185.2	6.356099	195	0
0.225	0.871	155.1	3.212822	164.7	5.869885	175.1	8.157478	191.3	4.295993	194.8	0.632456
0.684	0.684	175.3	4.808557	181.4	4.623611	186.4	7.121173	192.5	4.326918	194.8	0.632456
0.871	0.225	182.4	5.966574	187.8	7.375636	190.3	4.667857	194.4	1.577621	193.8	3.794733
0.9	0	184.7	4.595892	188.8	4.211096	189.1	6.063552	191.7	3.12872	195	0

population vaccinated: 18.4 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.9	152.9	5.15213	169.5	5.854723	186.6	4.880801	195	0	195	0
0.225	0.871	164	5.477226	180.1	7.218033	195	0	195	0	195	0
0.684	0.684	194.8	0.421637	195	0	195	0	195	0	195	0
0.871	0.225	195	0	195	0	195	0	195	0	195	0
0.9	0	195	0	195	0	195	0	195	0	195	0

population vaccinated: 23 M

		VEi									
VEs	VEd	0		0.25		0.5		0.75		1	
		day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd	day of peak	sd
0	0.9	153.5	5.104464	171.7	4.347413	192.1	3.573047	195	0	195	0
0.225	0.871	166.3	6.429965	182.3	5.850926	195	0	195	0	195	0
0.684	0.684	195	0	195	0	195	0	195	0	195	0
0.871	0.225	195	0	195	0	195	0	195	0	195	0
0.9	0	195	0	195	0	195	0	195	0	195	0

TABLE S12. Prevalence peak times (means and standard deviations) produced by the ABM for various combinations of vaccine efficacy parameters and coverage levels, assuming a clinical efficacy of VEc = 0.9 and active case-targeted NPIs consisting of case isolation, home-quarantine of household contacts of detected cases, and international travel restrictions (n = 10 instances per scenario).