Te Pūnaha Matatini
Data ▪ Knowledge ▪ Insight

Note: This paper has not yet undergone formal peer review

A COVID-19 Vaccination Model for Aotearoa New Zealand

Supplementary Information

Contents

1. Full Model Specifications

Deterministic SEIR Implementation

Transmission Model

The SEIR model consists of five non-vaccinated compartments for each age group i: susceptible (S_i), exposed (E_i), clinical infectious (I_i), subclinical infectious (A_i), and recovered (R_i). The model also includes the same five compartments for vaccinated age groups, denoted with a superscript v. Finally, an additional compartment for vaccinated infection-immune individuals Imm_i is included. (Figure S1, Equations S1 and S2).

The model is initialised with a proportion, v_i, of each age group vaccinated. Of these, $(1 - e_I)v_i$ are assigned to the susceptible vaccinated compartment S_i^v, and the remaining $e_I v_i$ are assigned to the immune vaccinated compartment Imm_i. That is, we are assuming the infection blocking aspect of the vaccine acts in an "all-or-nothing" fashion. This is elaborated on in the *Sensitivity to Vaccine Infection Blocking Assumptions* in Supplementary Information Section 2.

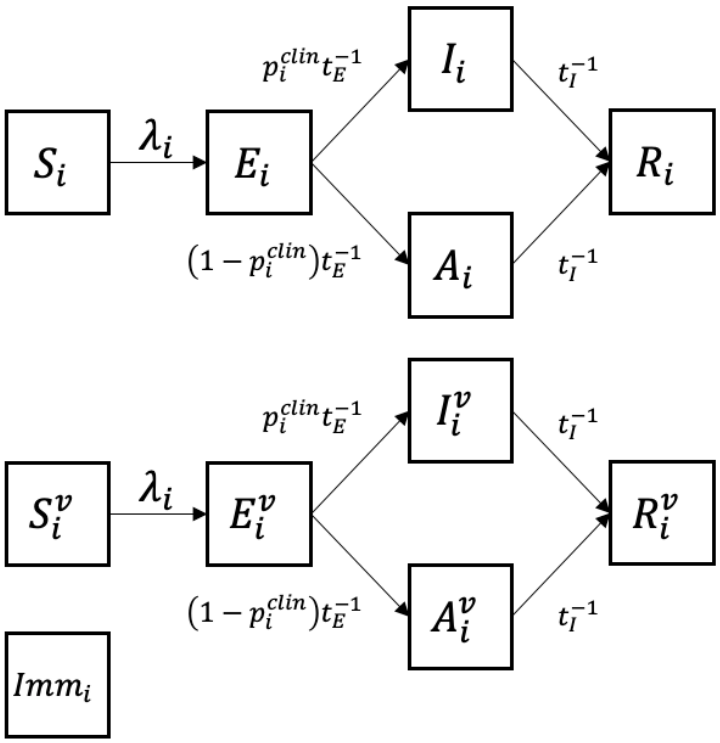

Figure S1. Structure diagram of the deterministic SEIR implementation. S_i, E_i, I_i, A_i, R_i represent the number of susceptible, exposed, clinical infectious, subclinical infectious and recovered individuals respectively in age group i. Superscript v's indicate vaccinated compartments and Imm_i consists of individuals that are vaccinated and immune to infection. The model consists of 10 ordinary differential equations for each age group which are outlined in equations (S1).

$$\frac{dS_i}{dt} = -\lambda_i S_i \qquad \frac{dS_i^v}{dt} = -\lambda_i S_i^v$$

$$\frac{dE_i}{dt} = \lambda_i S_i - t_E^{-1} E_i \qquad \frac{dE_i^v}{dt} = \lambda_i S_i^v - t_E^{-1} E_i^v$$

$$\frac{dI_i}{dt} = p_i^{clin} t_E^{-1} E_i - t_I^{-1} I_i \qquad \frac{dI_i^v}{dt} = p_i^{clin} t_E^{-1} E_i^v - t_I^{-1} I_i^v$$

$$\frac{dA_i}{dt} = (1 - p_i^{clin}) t_E^{-1} E_i - t_I^{-1} A_i \qquad \frac{dA_i^v}{dt} = (1 - p_i^{clin}) t_E^{-1} E_i^v - t_I^{-1} A_i^v$$

$$\frac{dR_i}{dt} = t_I^{-1}(I_i + A_i) \qquad \frac{dR_i^v}{dt} = t_I^{-1}(I_i^v + A_i^v)$$

Equations S1. Equations are identical for the vaccinated and non-vaccinated groups. The model is typically solved with initial conditions $S_i = N_i(1 - v_i)$, $S_i^v = N_i v_i(1 - e_I)$, and $Imm_i = N_i v_i e_I$ (although Imm_i is fixed so doesn't feature in these equations).

$$\lambda_i = U\frac{u_i}{N_i}\sum_j \left(I_j + \tau A_j + (1 - e_T)\left(I_j^v + \tau A_j^v\right) + t_I m_j\right)C_{j,i} \qquad (S2)$$

In equations (S1), t_E and t_I is the mean time spent in the exposed compartment (i.e. the latent period) and mean time spent in the infectious compartment respectively. The proportion of infections in age group i that are clinical is given by p_i^{clin}, and λ_i is the infection force acting on age group i, defined in equation (S2).

In equation (S2), N_i is the total number of individuals in age group i, u_i is the relative susceptibility to infection of age group i, and U is a constant chosen so the model is run with the desired value of R_0. Together Uu_i can be thought of as the probability of an individuals in age group i becoming infected given contact with an "average" infectious individual. The number of imported cases per day in age group j is given by m_j – these are assumed to be clinical, not vaccinated, and spend their entire infectious period in the country. τ is the relative infectiousness of subclinical individuals. These cases are not assigned to a compartment so are not counted towards total cases, hospitalisations, and fatalities.

The average infectious period, t_I, is assumed to be 5 days [1]. While the cited paper uses a latent period of 3 days, we use $t_E = 2.55$ days for consistency with the branching process implementation. This ensures the mean of the implied generation time distribution matches that used in the branching process. Sensitivity analysis performed on t_E shows it only affects the timing of epidemic peaks, rather than the final size or health outcomes.

Hospitalisation and Fatality Subroutine

The number of hospitalisations and fatalities are also tracked through a hospitalisation subroutine. This consists of an additional three compartments: H_i, F_i, and $Disch_i$. These represent individuals in hospital, fatalities, and those discharged from hospital. The cumulative

number of hospitalisations is given by the sum in F_i and $Disch_i$. The same three compartments are repeated for vaccinated individuals. (Figure S2, Equations S3).

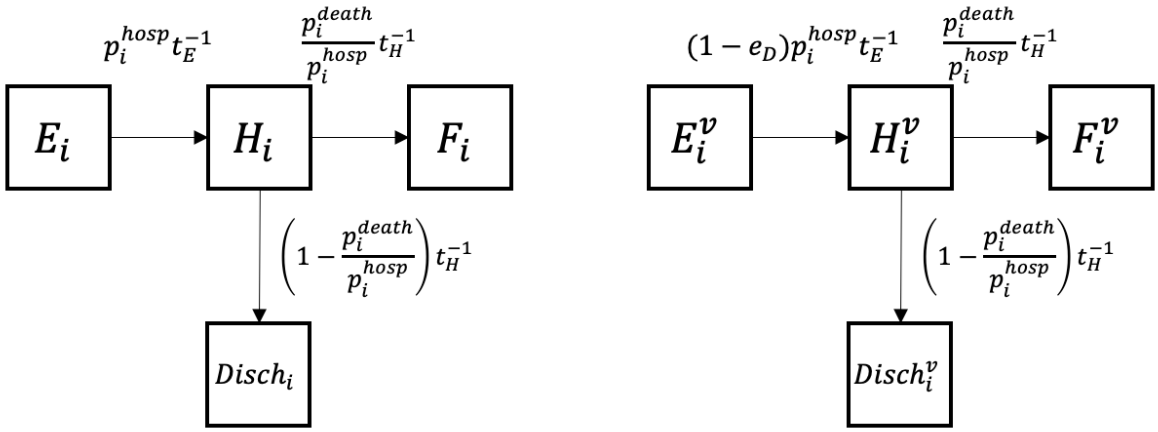

Figure S2. Structure diagram of the hospitalisation and fatality subroutine. E_i, H_i, $Disch_i$, and F_i represent the number of exposed individuals (from the primary transmission model), number of current hospitalisations, number of discharged, and number of fatalities respectively.

$$\frac{dH_i}{dt} = p_i^{hosp} t_E^{-1} E_i - t_H^{-1} H_i \qquad \frac{dH_i^v}{dt} = (1 - e_D) p_i^{hosp} t_E^{-1} E_i^v - t_H^{-1} H_i^v$$

$$\frac{dF_i}{dt} = \frac{p_i^{death}}{p_i^{hosp}} t_H^{-1} H_i \qquad \frac{dF_i^v}{dt} = \frac{p_i^{death}}{p_i^{hosp}} t_H^{-1} H_i^v$$

$$\frac{dDisch_i}{dt} = \left(1 - \frac{p_i^{death}}{p_i^{hosp}}\right) t_H^{-1} H_i \qquad \frac{dDisch_i^v}{dt} = \left(1 - \frac{p_i^{death}}{p_i^{hosp}}\right) t_H^{-1} H_i^v$$

Equations S3.

In these equations, p_i^{hosp} and p_i^{death} are the probability of being hospitalised and dying conditional on being infected. t_H is the mean time spent in hospital. For simplicity, we assume that exposed individuals are hospitalised with the same average delay as they become infectious, and that hospitalised individuals die with the same average delay as they are discharged. This only has a minor effect on the timing of the peak hospital occupancy estimates.

Stochastic Branching Process Implementation

Evidence suggests overdispersion in the distribution of the number of secondary cases in SARS-CoV-2 transmission: a few individuals typically account for a large amount of transmission ("superspreading") [2]. One estimate suggests a negative binomial distribution with $r = k = 0.5$ is an appropriate model for the distribution of the number of secondary cases [3]. To include this in the branching process, each clinical infected individual l is assigned a reproduction number R_l^{clin} given by:

$$R_l^{clin} = Ut_l Y_l (1 - V_l e_T) \sum_j u_j C_{a_l,j}$$

where Y_l is drawn independently from a gamma distribution with shape k and scale $1/k$, a_l is the age group of individual l, and V_l is an indicator variable that equals 1 when individual l is vaccinated and 0 otherwise. If the individual is subclinical then $R_l^{sub} = \tau R_l^{clin}$.

The number of secondary cases that would be generated by individual l at timestep t in a non-vaccinated population is then given by:

$$S_l^t \sim Poisson\left(F^{ctrl}(t) F_l^{isol}(t) R_l \int_t^{t+\Delta t} w(\tau - t_{inf,l})\, d\tau\right)$$

Assuming $F^{ctrl(t)} = F_l^{isol(t)} = 1$, the distribution of $\sum_t S_l^t$ is negative binomial with mean R_l and overdispersion $k = 0.5$. $F_l^{ctrl}(t)$ represents the reduction in transmission as a result of any population-level control measures and is equal to 1 if there are no control measures in place at time t. $F_l^{isol}(t)$ represents the reduction in transmission as a result of case isolation and contact tracing and is equal to 1 before individual l has been isolated, and equal to 0 after. $t_{inf,l}$ is the time that individual l was infected, and $w(t)$ is the probability density function for the generation time distribution, which is assumed to be a Weibull distribution with mean 5 days and standard deviation 1.9 days [4].

Each would-be secondary infection is randomly assigned an age group, with probability of being assigned to age group j of $\frac{u_j C_{a_l,j}}{\sum_j u_j C_{a_l,j}}$, and is then assigned to the vaccinated class with probability v_j and to the clinical class with probability p_j^{clin}. Would-be secondary infections in the vaccinated class have probability e_I of being prevented (i.e. not infected). Clinical

individuals have onset dates drawn from a gamma distribution with mean 5.51 days and standard deviation 2.29 days [4]. Clinical non-vaccinated individuals are assigned to the hospitalised class with probability $\frac{p_j^{hosp}}{p_j^{clin}}$, and similarly clinical vaccinated individuals are assigned to the hospitalised class with probability $(1-e_D)\frac{p_j^{hosp}}{p_j^{clin}}$. Finally, hospitalised individuals are assigned to the fatality class with probability $\frac{p_j^{died}}{p_j^{hosp}}$.

The branching process is simulated in time steps of $\Delta t = 1\ day$.

Case Detection, Isolation and Controls

A simple case detection and contact tracing model is also implemented. Before an outbreak is detected, symptomatic individuals are assumed to have a probability p_{detect}^{pre} of getting a test and being detected and subsequently isolated. The time from symptom onset to detection is drawn from an exponential distribution with mean t_{detect} and we assume individuals are immediately isolated on detection. We assume there is no testing of asymptomatic individuals in the period before an outbreak is detected.

Once an outbreak is detected, contact tracing begins and all existing and future infections are assumed to be detected by contract tracing with probability p_{trace}. For simplicity, we assume this probability is the same for symptomatic and asymptomatic individuals and independent of other cases. Traced individuals are isolated with an exponentially distributed delay with mean t_{trace} from the time of infection (or from the time the outbreak was first detected if this was later than the time of infection). Non-traced clinical cases are also detected with probability p_{detect}^{post} and isolated with mean delay of t_{detect} days from symptom onset. This models symptom-triggered testing for individuals that are missed by contact tracing. Typically we assume $p_{detect}^{post} > p_{detect}^{pre}$ to model greater symptom awareness and higher testing rates once an outbreak is detected. There is no testing of untraced asymptomatic individuals.

The effect of case isolation and contact tracing on R after outbreak detection in the model can be calculated analytically. Individuals that are contact traced (with probability p_{trace}) spend an average of $P(T_G < T_{trace})$ of their infectious period in the community prior to isolation, where T_G is a random variable representing the generation time. The probability that a randomly chosen individual in age group j is not traced but is detected through symptom-triggered testing is $(1 - p_{trace})p_{detect}^{post}p_j^{clin}$ and they spend an average of $P(T_G < T_{onset} + T_{detect})$ of their infectious period in the community. As the branching process is simulated in time steps of 1 day, the random variable representing the generation time T_G is the ceiling of the Weibull random variable described in Table 3.

This implies the elements of the post-detection next generation matrix are:

$$NGM_{i,j}^{trace} = \alpha_j NGM_{i.j} \tag{S4}$$

where:

$$\begin{aligned}\alpha_j = 1 - p_{trace}\big(1 - p_{detect}^{post}p_j^{clin}\big)P(T_G > T_{trace}) \\ - (1 - p_{trace})p_{detect}^{post}P(T_G > T_{ons} + T_{detect}) \\ - p_{trace}p_{detect}^{post}p_j^{clin}P(T_G > \min(T_{trace}, T_{ons} + T_{detect}))\end{aligned}$$

and the implied reduction in R from case isolation and contact tracing is:

$$1 - \frac{\rho(NGM^{trace})}{\rho(NGM)}$$

Baseline parameter values for testing and tracing are $p_{trace} = 0.7, t_{trace} = 6\ days, p_{detect}^{post} = 0.4, t_{detect} = 4\ days$. The individual choices for the parameter values are less important than their combined effect on the reproduction number R. For these values, $P(T_G > T_{trace}) = 0.583$, $P(T_G > T_{ons} + T_{detect}) = 0.206$, and $P(T_G > \min(T_{trace}, T_{ons} + T_{detect})) = 0.648$, leading to a reduction in R from contact tracing of 43.7% in a non-vaccinated population.

Under current assumptions, aside from a very small effect due to age-structured clinical rates, the effectiveness of contact tracing and case isolation (measured by a percentage reduction in R without contact tracing) is largely invariant to vaccination levels. Future work will be required to consider how vaccination may change this. In such a case it may be useful to define different probabilities of detection and tracing for vaccinated and non-vaccinated individuals.

Contact Matrix

In the absence of New Zealand specific contact data, we use a synthetic contact matrix C by Prem, Cook [5] for both model implementations. This matrix is constructed from international POLYMOD data [6], fit to the New Zealand age, household, and work structures. The elements of this matrix $C_{i,j}$ give the average daily number of contacts that an individual in group i has with individuals in group j. This implies that he total number of daily contacts that occur between an individual in group i and an individual in group j is $N_i C_{i,j}$ where N_i is the size of group i. Therefore by symmetry, the contact matrix should satisfy the detailed balance condition $N_i C_{i,j} = N_j C_{j,i}$ but it does not satisfy this condition, which has implications on its use in modelling.

The contact matrices by Prem et al and POLYMOD [5, 6] have been used in various modelling studies. Some define the SEIR infection pressure on age group i as $\lambda_i \propto \sum_j C_{j,i} I_j$ [6, 7], whereas others define the infection pressure as $\lambda_i \propto \sum_j \frac{C_{i,j} I_j}{N_j}$ [5, 8, 9]. If the contact matrix does not satisfy the detailed balance condition, these give different results.

It is not obvious which method is more correct, so we impose detailed balance by using the modified contact matrix defined by:

$$\widehat{C_{i,j}} = \frac{1}{2}\left(C_{i,j} + \frac{N_j}{N_i} C_{j,i}\right)$$

This ensures both expressions of the infection pressure produce identical results and can be thought of as "averaging" over both methods. We test the implications of using each method in Supplementary Information section 2.

2. Sensitivity Analysis

Sensitivity to Contact Structure Assumptions

We begin by considering six next-generation-matrices:

$$NGM_{i,j} \propto \frac{1}{2}\left(C_{j,i} + \frac{N_i}{N_j}C_{i,j}\right) u_i [p_j^{clin} + \tau(1 - p_j^{clin})] \quad (1)$$

$$NGM_{i,j} \propto C_{j,i}\, u_i [p_j^{clin} + \tau(1 - p_j^{clin})] \quad (2)$$

$$NGM_{i,j} \propto \frac{N_i}{N_j} C_{i,j} u_i \, [p_j^{clin} + \tau(1 - p_j^{clin})] \quad (3)$$

$$NGM_{i,j} \propto \frac{1}{2}\left(C_{j,i} + \frac{N_i}{N_j}C_{i,j}\right) \quad (4)$$

$$NGM_{i,j} \propto N_i \, [p_j^{clin} + \tau(1 - p_j^{clin})] \quad (5)$$

$$NGM_{i,j} \propto \frac{1}{2}\left(C_{j,i} + \frac{N_i}{N_j}C_{i,j}\right) \left[p_j^{clin} + \tau\left(1 - p_j^{clin}\right)\right] \quad (6)$$

The first four are based on the Prem, Cook [5] contact matrix C. Our implementation is given in (1), which can be thought of as an "average" of (2) and (3) that ensures the detailed balance condition holds. (2) uses the matrix in the same way as [6, 7] while (3) uses the matrix in the same way as [5, 9]. (4) assumes that clinical and subclinical individuals have the same infectiousness. (5) assumes proportional mixing, where individuals interact with other age groups proportional to their size. Finally, (6) reproduces our implementation of (1) without age-based susceptibility.

The dominant eigenvectors of these next-generation-matrices give the pseudo-steady age distribution of infections assuming a fully susceptible population (Figure S3). They can be thought of as representing the expected age distribution of cases before any significant immunity has accumulated.

Critically our implementation in (1) places slightly more weighting on older age groups than (2) and slightly less than (3). Thus, with respect to negative health outcomes, it is more pessimistic than (2) but more optimistic than (3). The similarity between (1) and (4) suggests the assumption that subclinical individuals are less infectious doesn't significantly alter the transmission dynamics.

Proportional mixing (5) assumes significantly higher contact between older age groups and the rest of the population, hence health outcomes from models that use this are expected to be significantly worse. Finally, removing age-based susceptibility in (6) results in under-20-year-olds, who typically have more contacts, contributing to a large amount of spread.

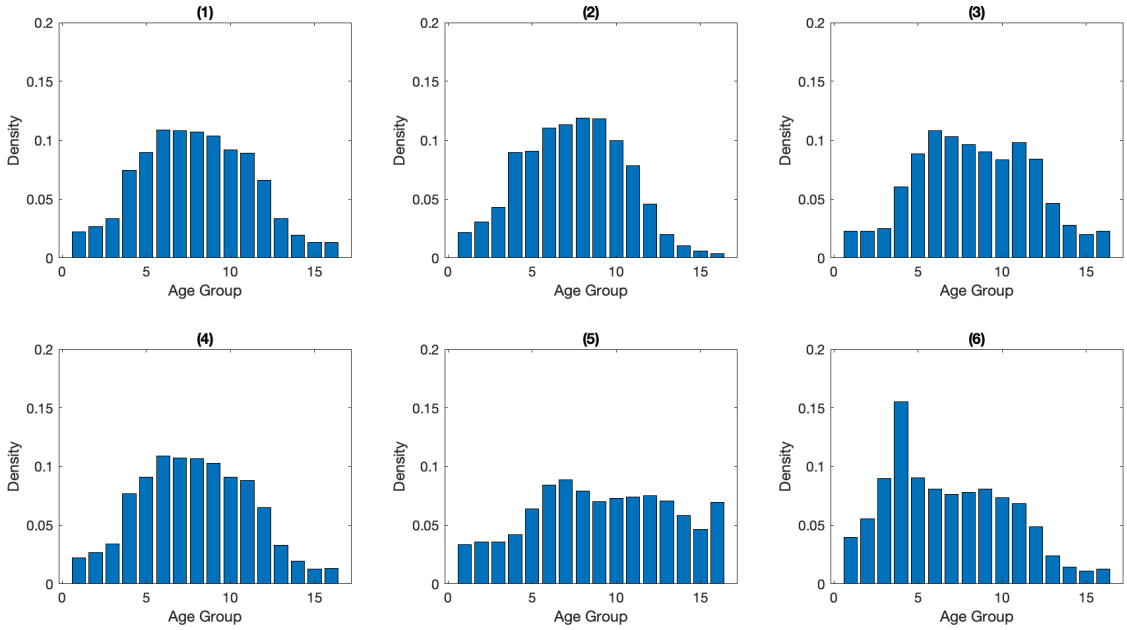

Figure S3. Dominant eigenvector of the four next-generation-matrices defined in equations (1-6) above.

Effect of Different Contact Assumptions on the Vaccinated Reproduction Number

Varying the relative importance of age groups on transmission has implications for the effect of vaccination on the reproduction number. We reproduce the results from Figure 2 under the same vaccine roll-out assumptions and baseline parameters for three of the contact matrices described above: (1) standard, (5) proportional mixing, and (6) no age based susceptibility (Figure S4). Since the relative importance of children in (6) is increased, in scenarios like this where older individuals are vaccinated first, more vaccinations are required to reach the population immunity threshold.

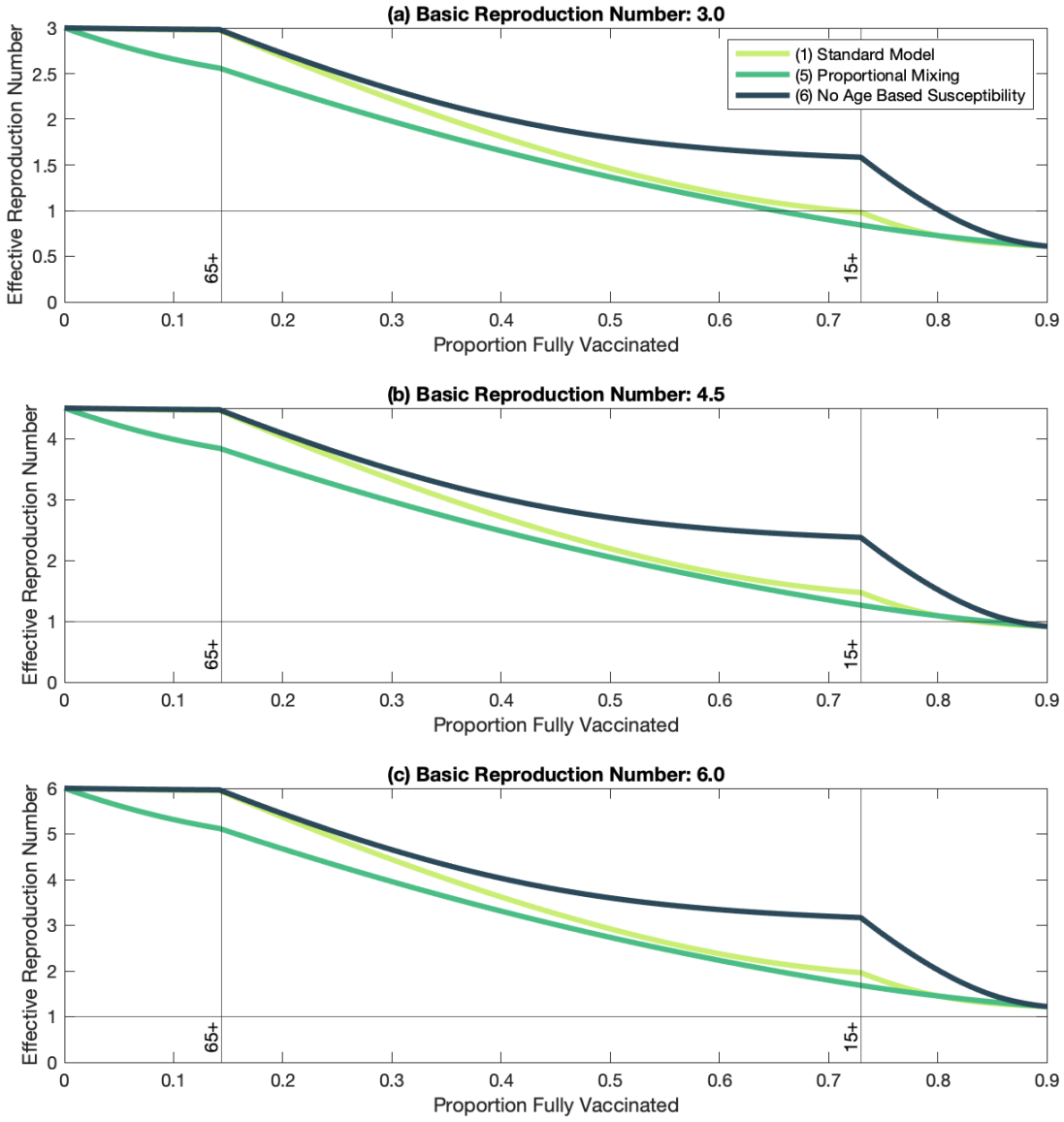

Figure S4. Vaccinated reproduction number under three different contact assumptions: the standard model, proportional mixing, and no age based susceptibility. Baseline vaccine effectiveness is assumed for all scenarios.

Sensitivity analysis of results on Relaxation of Border Restrictions

Assuming baseline vaccine effectiveness, we reproduce the results from Tables 5 and 6 in the main text under three contact assumptions: the standard model, proportional mixing, and no age based susceptibility (Tables S1 and S2).

$R_0 = 3.0$	Standard	Proportional Mixing	No Age Based Susceptibility
R_v	0.98	0.84	1.58
Infections	150,000 (44%)	60,000 (55%)	990,000 (31%)
Hospitalisations	2,000 (35%)	1,400 (35%)	8,900 (35%)
Fatalities	230 (35%)	240 (35%)	1,100 (35%)
Peak in hospital	N/A	N/A	1000 (after 140 days)
$R_0 = 4.5$	**Standard**	**Proportional Mixing**	**No Age Based Susceptibility**
R_v	1.47	1.26	2.38
Infections	1,300,000 (43%)	1,200,000 (53%)	1,600,000 (36%)
Hospitalisations	17,000 (35%)	25,000 (35%)	18,000 (35%)
Fatalities	2,200 (35%)	4,400 (35%)	2,400 (35%)
Peak in hospital	2,000 (after 140 days)	2,300 (after 160 days)	3,500 (after 80 days)
$R_0 = 6.0$	**Standard**	**Proportional Mixing**	**No Age Based Susceptibility**
R_v	1.96	1.68	3.17
Infections	1,800,000 (44%)	1,800,000 (50%)	1,900,000 (39%)
Hospitalisations	25,000 (35%)	35,000 (35%)	24,000 (35%)
Fatalities	3,400 (35%)	6,100 (35%)	3,300 (35%)
Peak in hospital	4,700 (after 90 days)	6,000 (after 100 days)	5,900 (after 60 days)

Table S1. Results from an unmitigated epidemic with 90% vaccine coverage of the over 15-year-old age groups. Values in parenthesis give percentage of infections/hospitalisations/fatalities that occur in vaccinated individuals.

$R_0 = 3.0$	Standard	Proportional Mixing	No Age Based Susceptibility
R_v	0.61	0.61	0.61
Infections	13,000 (73%)	16,000 (73%)	13,000 (73%)
Hospitalisations	240 (35%)	420 (35%)	380 (35%)
Fatalities	26 (35%)	75 (35%)	21 (35%)
$R_0 = 4.5$	**Standard**	**Proportional Mixing**	**No Age Based Susceptibility**
R_v	0.92	0.92	0.92

Infections	210,000 (73%)	260,000 (73%)	190,000 (73%)
Hospitalisations	3,800 (35%)	6,900 (35%)	2,800 (35%)
Fatalities	410 (35%)	1,200 (35%)	310 (35%)
$\boldsymbol{R_0 = 6.0}$	**Standard**	**Proportional Mixing**	**No Age Based Susceptibility**
R_v	1.22	1.22	1.22
Infections	770,000 (73%)	920,000 (73%)	760,000 (73%)
Hospitalisations	14,000 (35%)	24,000 (35%)	12,000 (35%)
Fatalities	1,700 (35%)	4,200 (35%)	1,400 (35%)
Peak in hospital	1,300 (after 150 days)	2,200 (after 150 days)	1,100 (after 150 days)

Table S2. Results from an unmitigated epidemic with 90% vaccine coverage of the total population. Values in parenthesis give percentage of infections/hospitalisations/fatalities that occur in vaccinated individuals.

Sensitivity to Vaccine Roll-out Structure

In the main paper we assume that the population is separated into three groups: 65+ year-olds, 15-64-year-olds, and under-15-year-olds. In mid-June 2020 it was announced that the general roll-out would be further age-structured. We re-calculate the results presented in Figure 2 and Figures 4-6 under a more structured roll-out.

We assume, as before, that over 65-year-olds receive the vaccine first. Once 90% of this group is vaccinated, we assume that each 5-year age band is vaccinated successively up to 90%, until the under-15-year-old group is reached. The effect of this on the reproduction number is presented in Figure S5. The effect of this on an unmitigated outbreak (see the Open Borders Scenario section in the main text) is presented in Figures S6-S8.

The more structured roll-out implies a slower reduction in R due to vaccination. This occurs as groups with higher contact are vaccinated later in the roll-out. However, the health implications of any widespread outbreak decrease faster under this assumption, as older individuals that are more at risk are vaccinated earlier in the roll-out.

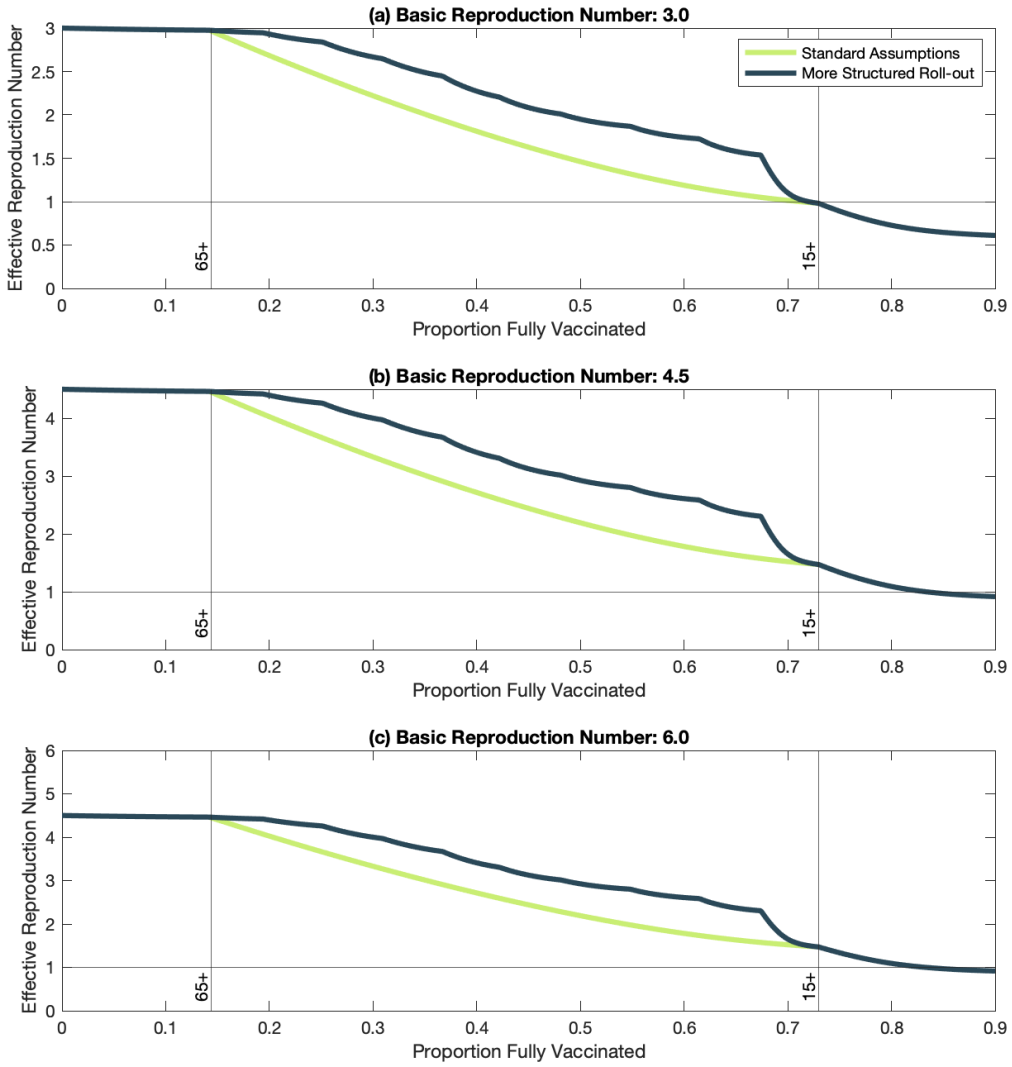

Figure S5. Effective reproduction number R_v after vaccination as a function of total vaccine courses administered, with a maximum of 90% coverage in any age group, for (a) $R_0 = 3$, (b) $R_0 = 4.5$, and (c) $R_0 = 6.0$. Default vaccine effectiveness parameters are used ($e_I = 70\%$, $e_T = 50\%$). Standard assumptions represent the roll-out outlined in Figure 2 of the main text, with the 15-64 year old age groups being targeted together. The more structured scenario assumes vaccination of this group occurs in successive 5-year groups (see paragraph above).

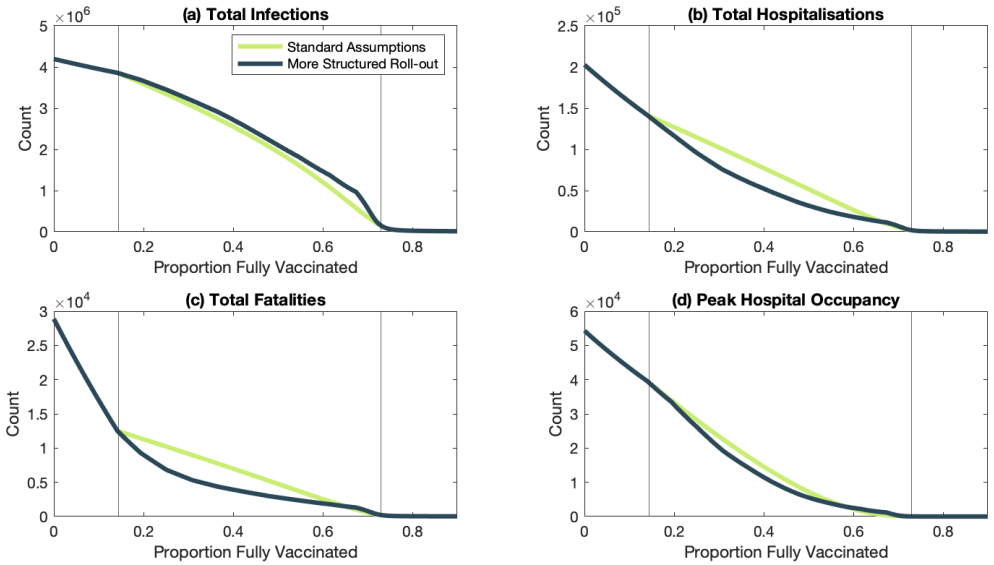

Figure S6. Total infections (a), hospitalisations (b), fatalities (c), and peak hospital occupancy (d) at the two vaccine rollouts described in the caption of Figure S5 and $R_0 = 3.0$. Results are from a 2-year simulation, assuming there is no further vaccination after the outbreak begins.

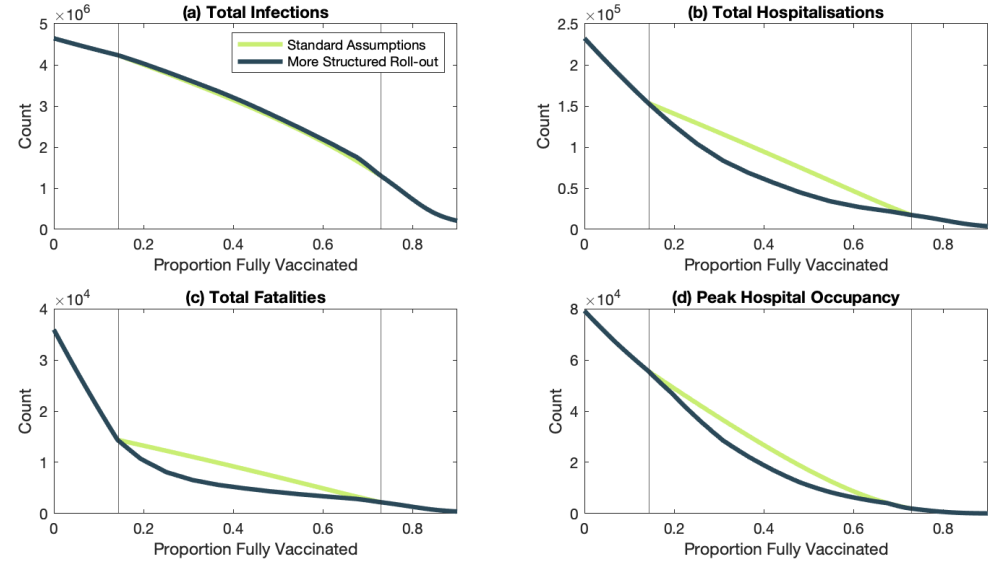

Figure S7. Total infections (a), hospitalisations (b), fatalities (c), and peak hospital occupancy (d) at the two vaccine rollouts described in the caption of Figure S5 and $R_0 = 4.5$. Results are from a 2-year simulation, assuming there is no further vaccination after the outbreak begins.

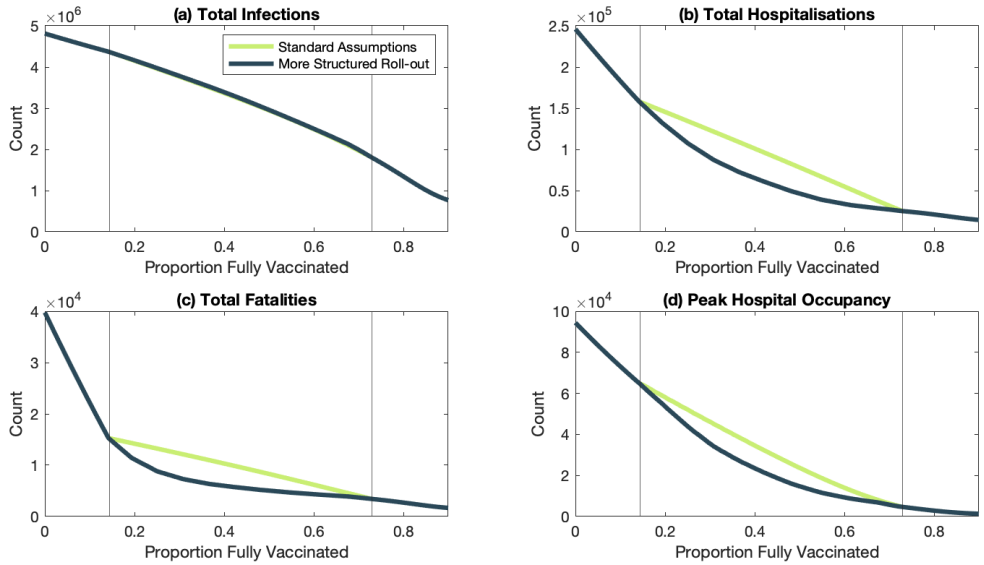

Figure S8. Total infections (a), hospitalisations (b), fatalities (c), and peak hospital occupancy (d) at the two vaccine rollouts described in the caption of Figure S5 and $R_0 = 6.0$. Results are from a 2-year simulation, assuming there is no further vaccination after the outbreak begins.

Sensitivity to Vaccine Infection Blocking Assumptions

There are two ways in which a vaccine that prevents a proportion e_I of infections is typically modelled. The first, often described as an "all-or-nothing" vaccine, is where a proportion e_I of vaccinated individuals are completely immune to any infection and a proportion $1 - e_I$ are completely susceptible. The second, often described as a "leaky" vaccine, assumes all vaccinated individuals have some likelihood of being infected given exposure, but that likelihood is reduced by a factor of e_I relative to non-vaccinated individuals. There is, at present, limited evidence as to which of these assumptions is more realistic and it is possible that reality lies somewhere between the two (i.e. individuals are distributed along a spectrum of vaccine effectiveness). The results in the main text assume an "all-or-nothing" vaccine. Here, we investigate model outputs under a leaky vaccine assumption.

The choice of an all-or-nothing or a leaky vaccine assumption only affects how the accumulation of infection-acquired immunity changes the dynamics of an epidemic. The results on the effective reproduction number in the absence of any infection-acquired immunity (i.e. Figures 2-3 of the main text) are invariant to this assumption. The results from the

stochastic implementation are also invariant to this assumption as infection-acquired immunity is ignored in this model. Previous modelling work also suggests that the choice of implementation has little effect on optimal vaccine roll-out strategies [1].

The choice of vaccine assumption only significantly affects the counterfactual scenarios where an epidemic wave occurs, ending due to the build-up of infection-acquired immunity in the population (e.g. Tables 5 and 6 and Figures 4, 5, and 6 in the main text). For comparison, we reproduce the results from Tables 5 and 6 "Open Borders Scenario". Recall this scenario assumes five non-vaccinated imported cases per day. Vaccine effectiveness against infection and transmission are given in the table, with all scenarios assuming that e_D is set so the overall effectiveness against severe disease is 95%.

$R_0 = 4.5$	Baseline	Lower Effectiveness	Higher Effectiveness
R_v	0.92	1.58	0.47
Infections	320,000	2,500,000	8,700
Hospitalisations	5,700	36,000	240
Fatalities	610	4,500	26

Table S3. Results from a 2-year unmitigated epidemic **with a leaky vaccine** and 90% coverage of the total population (as per Table 3).

$R_0 = 4.5$	Baseline	Lower Effectiveness	Higher Effectiveness
R_v	1.47	1.86	1.35
Infections	1,800,000	3,300,00	760,000
Hospitalisations	24,000	43,000	9,700
Fatalities	3,000	5,600	1,200

Table S4. Results from a 2-year unmitigated epidemic **with a leaky vaccine** and 90% coverage of over 15-year-olds (as per Table 4).

In scenarios where R_v is high (e.g. low vaccine effectiveness, low vaccination coverage, or high R_0), the leaky vaccine assumption leads to substantially higher numbers of infections, hospitalisations, and deaths than the all-or-nothing vaccine assumption does. When R_v is low the results are very similar [10].

Approval for 12+ Year-Olds Only

Some countries have approved the Pfizer-BioNTech vaccine for use in children 12 years old and above. As an approximation, vaccinating 90% of 12-14-year-olds is similar to vaccinating 54% of 10-14-year-olds (90% of three out of five of the ages). We reproduce Table 5 with these additional vaccines in Table S5.

When $R_0 = 3.0$, these additional vaccinations have a particularly large effect in the baseline vaccine effectiveness scenario. When only 15+ year-olds are vaccinated, R_v is estimated to be 0.98, or fairly close to 1. The additional vaccines administered lower this to 0.82, significantly decreasing the number of infections from 150,000 to 34,000.

When $R_0 = 4.5$, these additional vaccinations are still insufficient to reach the population immunity threshold, although in the higher effectiveness scenario $R_v = 1.03$ is fairly close (compared to $R_v = 1.35$ when only 15+ year-olds are vaccinated). Minor social distancing and/or testing and tracing measures would be sufficient to bring R below 1 in this scenario.

$R_0 = 3.0$	Baseline	Lower Effectiveness	Higher Effectiveness
Vaccine Effectiveness	$e_I = 70\%, e_T = 50\%$	$e_I = 50\%, e_T = 40\%$	$e_I = 90\%, e_T = 50\%$
R_v	0.82	1.16	0.69
Infections	34,000 (53%)	880,000 (67%)	9,500 (26%)
Hospitalisations	500 (35%)	12,000 (47%)	160 (15%)
Fatalities	56 (35%)	1,400 (47%)	18 (15%)
Peak in hospital	N/A	630 (after 240 days)	N/A
$R_0 = 4.5$	**Baseline**	**Lower Effectiveness**	**Higher Effectiveness**
R_v	1.22	1.74	1.03
Infections	960,000 (50%)	2,000,000 (64%)	280,000 (22%)
Hospitalisations	14,000 (35%)	29,000 (47%)	4,000 (15%)
Fatalities	1,700 (35%)	3,700 (47%)	480 (15%)
Peak in hospital	1,100 (after 180 days)	4,500 (after 100 days)	120 (after 310 days)
$R_0 = 6.0$	**Baseline**	**Lower Effectiveness**	**Higher Effectiveness**
R_v	1.63	2.32	1.38
Infections	1,600,000 (49%)	2,500,000 (63%)	800,000 (22%)

Hospitalisations	23,000 (35%)	36,000 (47%)	12,000 (15%)
Fatalities	3,000 (35%)	5,100 (47%)	1,500 (15%)
Peak in hospital	3,500 (after 110 days)	8,100 (after 70 days)	1,300 (after 150 days)

Table S5. Results from an unmitigated epidemic and 90% coverage of over 12-year-olds. This is modelled by assuming 54% coverage of 10-14-year-olds.

Deterministic SEIR Sensitivity to Other Epidemiological Parameters

We consider the sensitivity of various outputs of the deterministic SEIR implementation and vaccinated reproduction number to other epidemiological parameters. $R_0 = 4.5$ unless otherwise stated.

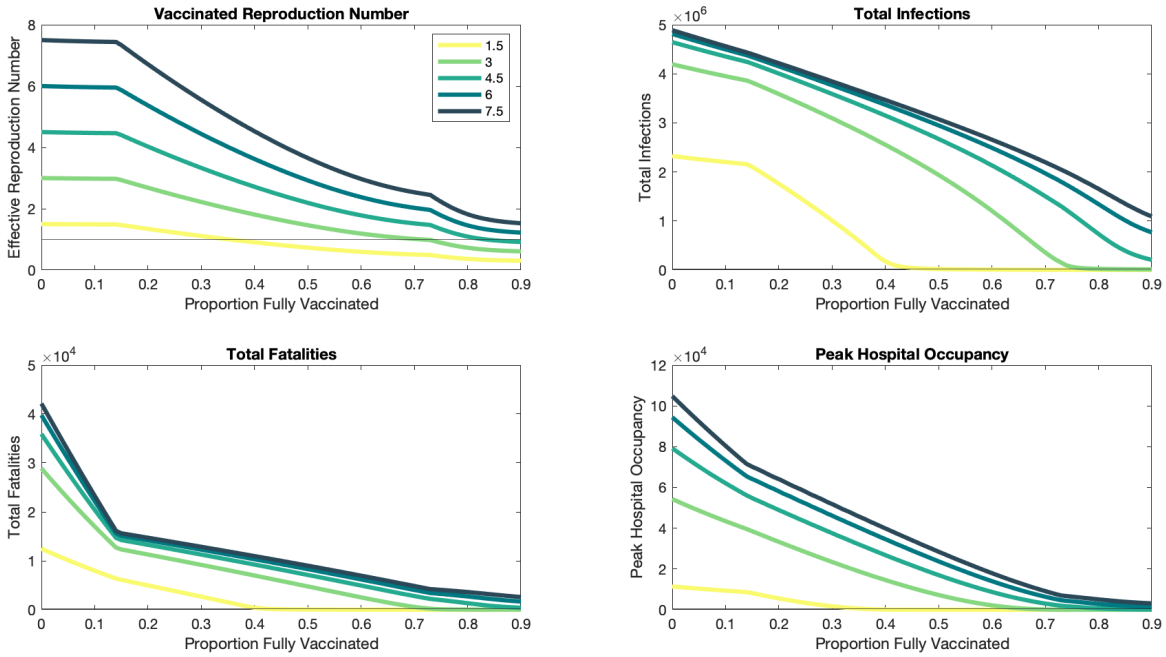

Figure S9. Sensitivity to R_0, testing values between 1.5 and 7.5 in increments of 1.5. When $R_0 = 1.5$ and 3.0, the population immunity threshold can be reached without vaccinating under 15-year-olds. When $R_0 = 4.5$ vaccination of under-15-year-olds is required. When $R_0 = 6.0$ and 7.5 the population immunity threshold cannot be reached without greater than 90% population coverage. Furthermore, as R_0 increases, the number of infections, fatalities, and peak hospital occupancy also increase.

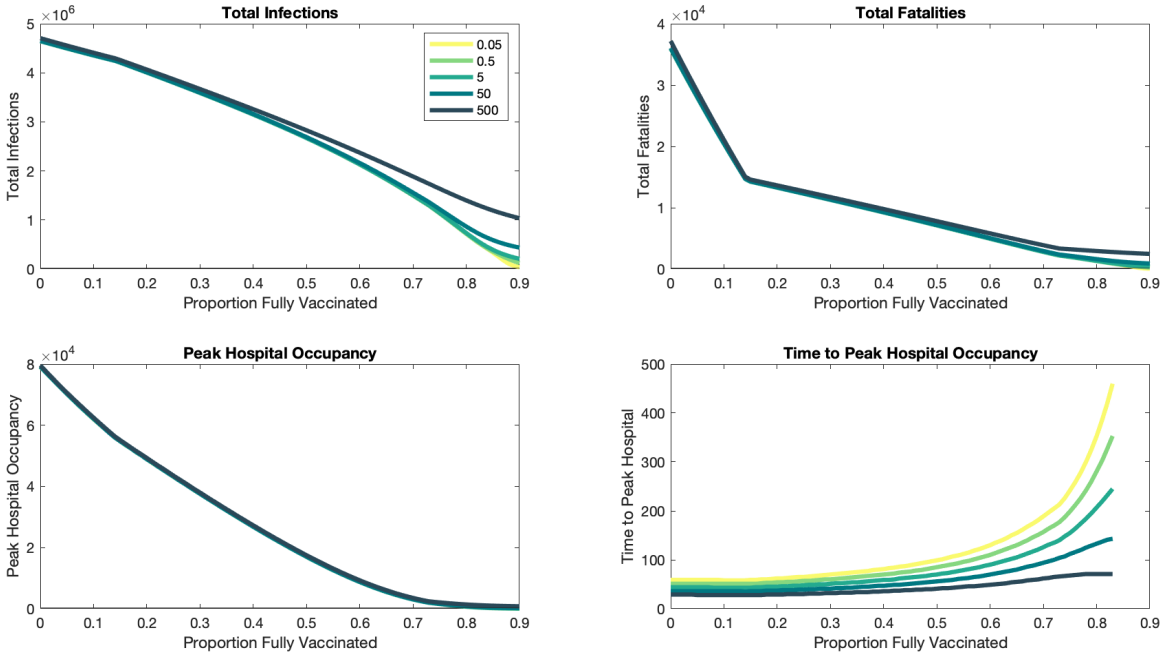

Figure S10. Sensitivity to the rate of imported cases, testing values between 0.05 and 500 imported infectious cases per day. Until high levels of vaccination are reached ($R_v < 1$), differences in rates of imported cases makes little difference to the overall results, except in extremely high infected arrival rates (500 per day). The timing of the epidemic peak is more sensitive, with higher arrival rates resulting in an earlier peak. This scenario assumes $R_0 = 4.5$.

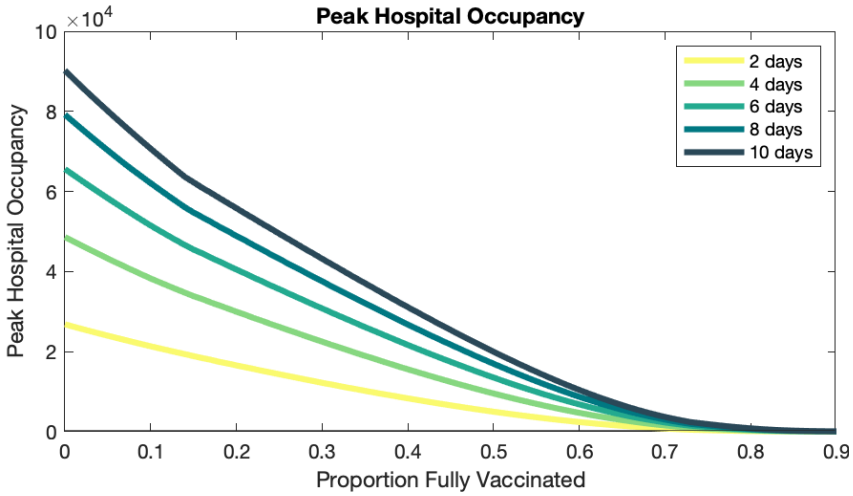

Figure S11. Sensitivity to the mean length of hospital stay, testing values between 2 and 10 days. This parameter only has implications on the peak hospital occupancy, with longer times in hospital resulting in a larger peak occupancy. In reality this likely also varies with

vaccination coverage as different age groups have different expected hospitalisation durations, although this is not considered in our modelling. This scenario assumes $R_0 = 4.5$.

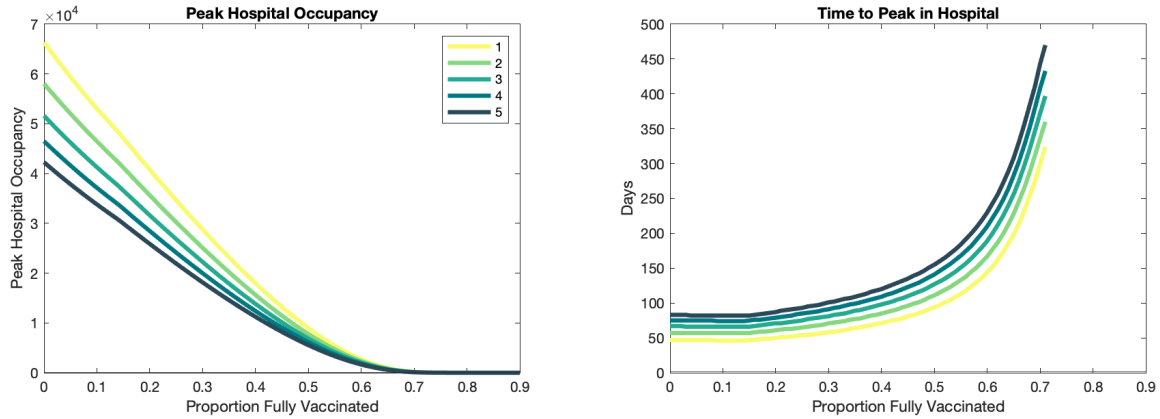

Figure S12. Sensitivity to the incubation period (mean time in "exposed" compartment), testing values between 1 and 5 days. This parameter only affects the timing and size of the epidemic peak, with longer incubation periods implying lower peak hospital occupancy and longer time to the peak. This scenario assumes $R_0 = 4.5$.

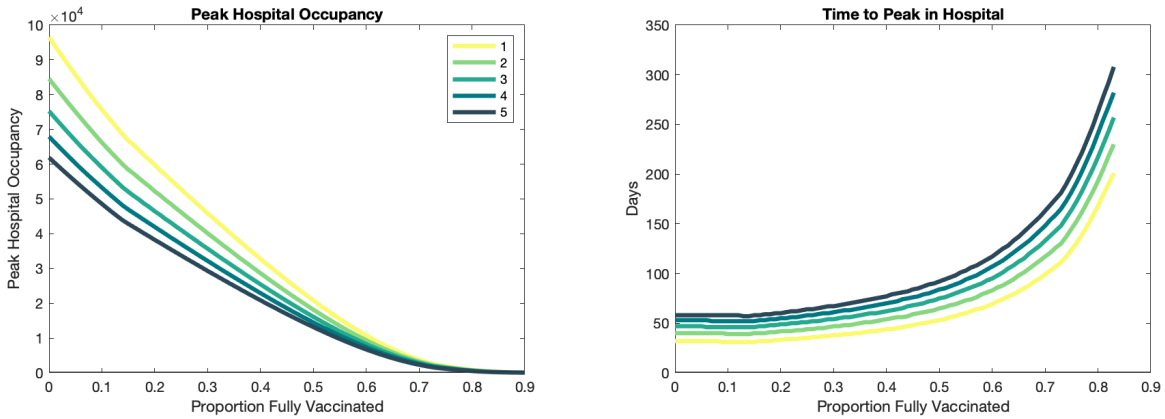

Figure S13. Sensitivity to the duration of the infectious period, testing values between 3 and 7 days. There is a similar effect in varying this as with the incubation period. This scenario assumes $R_0 = 4.5$.

Effect of Testing and Case Isolation on Reproduction Number

Results in the main paper (Table 4) on the level of vaccine coverage required to reach the population immunity threshold assume there are no non-pharmaceutical interventions.

Results from the stochastic implementation in the main paper assume that case isolation and contact tracing begin once a new outbreak is detected. This is reasonable under an elimination strategy coupled with strong border measures designed to keep COVID-19 out of the community. However, due to capacity constraints, the impact contact tracing system on transmission would be far smaller if there were regular imported cases triggering multiple outbreaks simultaneously.

An intermediate situation between: (i) no non-pharmaceutical interventions and (ii) intensive contact tracing for small, sporadic outbreaks is where there some baseline control measures remain in place. To investigate this, we calculated the model-implied reduction in effective reproduction number with: $p_{Trace} = 0$ (no contact tracing), $p_{detect}^{pre} = p_{detect}^{post} = 70\%$ (significantly increased detection rates, regardless of whether or not there is a current outbreak), and $t_{trace} = 2\ days$ (decreased mean time to detection and isolation). Assuming case isolation is 100% effective in preventing transmission, this reduces the reproduction number by an estimated 14% (as per equation S4 in Supplementary Sec 1) compared to no control. If case isolation is imperfect and reduces transmission by 80%, this reduces the reproduction number by an estimated 11%. This is a much smaller reduction in R than can be achieved by intensive contact tracing of small outbreaks, but it does mean that effective population immunity ($R < 1$ with widespread testing and case isolation but without mass restrictions) can be achieved with a slightly lower vaccine coverage (Table S6).

	Baseline	**Lower Effectiveness**	**Higher Effectiveness**
Vaccine Effectiveness	$e_I = 70\%, e_T = 50\%$	$e_I = 50\%, e_T = 40\%$	$e_I = 90\%, e_T = 50\%$
$R_0 = 3.0$	63%	80%	55%
$R_0 = 4.5$	79%	-	75%
$R_0 = 6.0$	93%*	-	79%

Table S6. Vaccine coverage required to achieve effective population immunity ($R < 1$ with case-targeted control such that 70% of cases are isolated an average of 2 days after symptom onset, with subsequent transmission reduced by 80%) for each vaccine effectiveness scenario at three different values of R_0. Estimates assume a structured roll-out, beginning in 65+ year-olds, then 15-64 year-olds, and finally under 15-year olds, with up to 90% of each group

vaccinated. Estimates with an asterisk are greater than 90%, so this is assumed to be equal coverage of the entire population.

Stochastic Branching Process Sensitivity to Vaccine Effectiveness

As we do not consider varying vaccine effectiveness in the main paper, we consider lower and higher effectiveness vaccines. Other parameters and assumptions are the same as in the "Other Outbreaks and Control" section of the main paper – these include contact tracing and case isolation amounting to a 43% reduction in the reproduction number. Population coverage is assumed to be 90% of over 15-year-olds.

$R_0 = 3.0$	Baseline	Lower Effectiveness	Higher Effectiveness
Vaccine Effectiveness	$e_I = 70\%, e_T = 50\%$	$e_I = 50\%, e_T = 40\%$	$e_I = 90\%, e_T = 50\%$
R_v	0.98	1.24	0.90
Infections at detection	6 (1, 58)	12 (1, 77)	3 (1, 45)
P(elim before 1000 infs)	100%	100%	100%
Time to elimination	12 (0, 26)	15 (0, 31)	10 (0, 23)
Total hospitalisations	0 (0, 2)	0 (0, 2)	0 (0, 1)
$R_0 = 4.5$	**Baseline**	**Lower Effectiveness**	**Higher Effectiveness**
R_v	1.47	1.86	1.35
Infections at detection	14 (1, 97)	22 (1, 142)	8 (1, 83)
P(elim before 1000 infs)	99.7%	82.7%	99.98%
Time to elimination	17 (0, 34)	22 (2, 42)	13 (0, 32)
Total hospitalisations	0 (0, 2)	0 (0, 4)	0 (0, 2)
$R_0 = 4.5$	**Baseline**	**Lower Effectiveness**	**Higher Effectiveness**
R_v	1.96	2.48	1.80
Infections at detection	22 (1, 154)	34 (1, 217)	15 (1, 126)
P(elim before 1000 infs)	81.4%	61.2%	93.1%
Time to elimination	22 (1, 44)	29 (3, 56)	18 (0, 40)
Total hospitalisations	0 (0, ,4)	1 (0, 6)	0 (0, 3)

Table S7. Branching process sensitivity to vaccine effectiveness against infection and transmission with 90% of 15+ year-olds vaccinated. Default values are used for all parameters (see Table 3). Median values from 10,000 trials reported with 95% confidence intervals in parenthesis. Probability of elimination before 1,000 infections assumes effective and scalable

contact tracing and case isolation takes place after detection. Time to elimination and total hospitalisations assume population-level controls are used in addition to contact tracing and case isolation.

Stochastic Branching Process Sensitivity to Other Epidemiological Parameters

We also test sensitivity to R_0, t_{detect}, p_{detect}^{pre}, t_{trace}, and p_{trace} in Tables S8 to S12. Two stages of vaccination are now considered: no vaccination and 90% coverage of 15+ year-olds. Once an outbreak is detected, population level controls are implemented so there is an additional 2/3 reduction in R, in addition to contact tracing and case isolation (which provides a 43.7% reduction in R under default parameters). Reported values for infections at detection, time to elimination, and total hospitalisations are medians. P(elim) is the proportion of simulations that resulted in elimination before 1,000 infections, with the aforementioned contact tracing and case isolation operating.

R_0	No Vaccination					90% coverage of 15+				
	1.5	2.5	3.5	4.5	5.5	1.5	2.5	3.5	4.5	5.5
Infections at det	13	38	66	102	138	2	7	15	21	31
P(elim) (%)	100	61	48	41	36	100	100	100	82	65
Time to elimination	17	33	70	N/A	N/A	8	12	17	21	27
Total hosps	1	3	16	N/A	N/A	0	0	0	0	1

Table S8. Branching process sensitivity to varying values of R_0. As R_0 increases the infections at detection, time to elimination, and total hospitalisations from a mitigated outbreak increase and probability of elimination decreases. In the no vaccination scenario with $R_0 = 5.5$, contact tracing and default population level controls are not sufficient to control an outbreak, with $R_{eff} > 1$.

t_{detect} (days)	No Vaccination					90% coverage of 15+				
	1	2	3	4	5	1	2	3	4	5
Infections at det	41	51	58	70	77	11	12	13	14	15
P(elim) (%)	49	49	49	49	47	100	100	100	100	99
Time to elimination	50	58	65	72	77	16	17	17	17	17
Total hosps	7	10	12	16	20	0	0	0	0	0

Table S9. Branching process sensitivity to varying values of mean delay from symptom onset to case detection. As the delay from onset to detection increases, the number of infections at detection increase substantially, particularly in the no vaccination scenario.

p_{detect}^{pre}	No Vaccination					90% coverage of 15+				
	5%	10%	20%	50%	80%	5%	10%	20%	50%	80%
Infections at det	175	85	38	11	6	34	17	8	3	2
P(elim) (%)	47	47	49	52	56	99	99	100	100	100
Time to elimination	97	78	56	30	21	21	18	14	11	10
Total hosps	45	20	8	1	1	0	0	0	0	0

Table S10. Branching process sensitivity to varying values of probability of detecting a symptomatic case before an outbreak is detected. As probability of detection increases, the number of infections at detection increases, as does the probability of elimination. In scenarios where p_{detect}^{pre} is greater than the default value of $p_{detect}^{post} = 40\%$, we increase p_{detect}^{post} to match.

t_{trace} (days)	No Vaccination					90% coverage of 15+				
	1	3	5	7	9	1	3	5	7	9
P(elim) (%)	52	49	48	48	48	100	100	100	99	96
Time to elimination	35	44	61	85	141	16	17	17	17	17
Total hosps	6	8	13	20	45	0	0	0	0	0

Table S11. Branching process sensitivity to varying values of mean delay from exposure to detection via contact tracing. As the delay in tracing increases, the probability of elimination before 1000 cases decreases.

p_{trace}	No Vaccination					90% coverage of 15+				
	50%	60%	70%	80%	90%	50%	60%	70%	80%	90%
P(elim) (%)	48	48	48	49	48	95	98	100	100	100
Time to elimination	N/A	98	72	57	48	18	17	17	17	17
Total hosps	N/A	24	16	12	10	0	0	0	0	0

Table S12. Branching process sensitivity to varying values of probability of detecting an infected individual by contact tracing. As this increases, the probability of elimination increases.

3. Effectiveness of the Pfizer-BioNTech Vaccine

In March 2020, New Zealand confirmed the purchase of sufficient doses of the Pfizer-BioNTech BNT162b2 mRNA vaccine to vaccinate the entire population. In this section, we first briefly justify our assumptions for vaccine effectiveness parameters. In the subsections below, we summarise published results on the efficacy and effectiveness of the Pfizer vaccine in the context of our model.

Lipsitch and Kahn [11] argue that vaccine efficacy against viral RT-PCR positivity is a plausible lower bound on the vaccine's efficacy against transmission. The studies discussed in the "effectiveness against infection" subsection below are all variations on effectiveness against viral positivity studies, suggesting a lower bound on the overall reduction of transmission of around 90%. However, early results suggest there may be decreased effectiveness against variants of concern [12, 13], and evidence is limited for the effect in young and old people, so we use $e_I = 70\%$ effectiveness against infection and $e_T = 50\%$ effectiveness against onwards transmission conditional on breakthrough infection. This gives an overall implied transmission reduction from vaccination of $1 - (1 - e_I)(1 - e_T) = 85\%$ and is in-line with modelling from the UK [14].

The effectiveness against disease appears to be as high as 95% across many studies. We use 80% as our baseline estimate for e_D, implying overall 94% effectiveness against severe disease. Some studies have estimated the effectiveness against severe disease and death to be higher than effectiveness against mild disease (see below). However, because these outcomes are rarer, there is less data and therefore more uncertainty around this conclusion, particularly for specific age groups.

Effectiveness against SARS-CoV-2 Infection

Weekes, Jones [15] present evidence from healthcare workers in the UK for effectiveness against asymptomatic viral positivity. Data was analysed over two weeks from 18th to 31st January 2021 and included 4,408 PCR test-results in the first week and 4,411 in the second. The results found that 26/3,252 (0.8%) of tests from non-vaccinated HCWs were positive, 13/3,535 (0.37%) of tests from those <12 days post-vaccination were positive, and 4/1,989

(0.20%) of tests from those >12 days post-vaccination were positive. This suggests an approximate 75% effectiveness against viral positivity following a single-dose. Similar results were found when symptomatic individuals were included. All results were for a single-dose. This may be evidence of decreased infection duration or decreased overall susceptibility or a combination of both. In any case, the vaccine effectiveness against viral positivity (or equivalently, documented infection), is a plausible lower bound on effectiveness against transmission [11].

Dagan, Barda [16] in Israel found an effectiveness against documented infection of 46% (40%, 51%) between days 14 and 20 following the first dose, and 92% (88%, 95%) more than 7 days after the second. This is more pessimistic than [15] for single-dose effectiveness, but more optimistic long-term. This study is also based on a much larger sample size (>1m participants).

Chodick, Tene [17], also in Israel, found an effectiveness against documented infection of 51.4% in days 13-24 following the first dose. This was estimated by comparing individuals >13 days after their first dose with those after 1-12 days. As such, we place less weighting on this study.

Moustsen-Helms, Emborg [18], in Denmark, found an effectiveness against viral positivity of 64% (14%, 84%) in long-term care facility residents and 90% (92%, 95%) in healthcare workers. There are many caveats, but these results may suggest that reduced effectiveness in older individuals is plausible.

Thompson, Burgess [19], in the US, found an effectiveness against infection of 80% (59%, 90%) >= 14 days after the first dose, and an effectiveness against infection of 90% (68%, 97%) >= 14 days after the second dose.

Abu-Raddad, Chemaitelly [13], in Qatar, found an effectiveness against viral positivity for B.1.1.7 of 89.5% (85.9%, 92.3%) and an effectiveness against viral positivity for B.1.351 of 75.0% (70.5%, 78.9%) (both > 14 days after second dose). This is strong evidence for reduced effectiveness against B.1.351.

Effectiveness against Transmission given Breakthrough Infection

Harris, Hall [20], in the UK, provide early evidence for additional prevention against onward transmission given breakthrough infection. The adjusted odds ratio for onward transmission conditional on being vaccinated with BNT162b2 was 0.51 (0.44, 0.59). Individuals in this study were considered vaccinated if they received their first dose at least 21 days prior to testing positive.

Effectiveness against Symptomatic COVID-19

Polack, Thomas [21] present the stage 2/3 clinical trial results. The primary endpoint was COVID-19 disease, to which the vaccine efficacy was found to be 95.0% (90.3%, 97.6%).

Dagan, Barda [16] in Israel found an effectiveness against symptomatic COVID-19 of 57% (50%, 63%) between days 14 and 20 after the first dose, and 94% (87%, 98%) more than 7 days after the second.

Effectiveness against Severe Disease/Hospitalisation

Dagan, Barda [16] in Israel found an effectiveness against hospitalisation of 74% (56%, 86%) between 14 and 20 days after the first dose, and 87% (55%, 100%) at least a week after the second. Similarly, they found an effectiveness against severe disease of 62% (39%, 80%) and 92% (75%, 100%).

Abu-Raddad, Chemaitelly [13] found effectiveness against severe (or worse) disease of 100% for both B.1.1.7 and B.1.351 with confidence intervals of (81.7%, 100%) and (73.7%, 100%) respectively. Against all SARS-CoV-2 they found an effectiveness of 97.4% (99.2%, 99.5%).

Effectiveness against Death

Dagan, Barda [16] in Israel found an effectiveness against death of 84% (44%, 100%) in individuals 21 to 27 days after their first dose. There was insufficient data to estimate this for individuals with multiple doses.

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