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A COVID-19 Vaccination Model for Aotearoa New Zealand

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Executive Summary

1. We use a mathematical model to estimate the effect of New Zealand's vaccine rollout on the potential spread and health impacts of COVID-19 and the implications for controlling border-related outbreaks.
2. The model can be used to estimate the theoretical population immunity threshold, which represents a point in the vaccination rollout at which we could relax border restrictions with few or no controls in place and see only small occasional outbreaks.
3. While there are significant uncertainties in R_0 for new variants, for a variant that would have $R_0 = 4.5$ with no public health measures (e.g. the Alpha variant), the population immunity threshold is estimated to require 83% of the population to be vaccinated under baseline vaccine effectiveness assumptions. For a variant with $R_0 = 6.0$ (e.g. the Delta variant), this would need to be 97%.
4. While coverage is below this threshold, relaxing controls completely would risk serious health impacts, including thousands of fatalities.
5. Whether or not New Zealand reaches a theoretical population immunity threshold, the higher vaccination coverage is, the more collective protection the population has against adverse health outcomes from COVID-19, and the easier it will become to control outbreaks.
6. Reaching or getting as close as possible to the population immunity threshold is very likely to require vaccinating at least some under-16-year-olds, subject to official approval for the vaccine to be used in these age groups.
7. There remains considerable uncertainty in model outputs, in part because of the potential for the evolution of new variants. If new variants arise that are more transmissible or vaccine resistant, an increase in vaccine coverage will be needed to provide the same level of protection.
8. A second important source of uncertainty arises because not all parts of the population will have equal vaccine coverage. Even if population immunity is achieved at a national level, communities with relatively low vaccine coverage or high contact rates will remain vulnerable to major outbreaks. These thresholds may also vary seasonally.
9. Until the vaccine rollout is complete, retaining the elimination strategy will protect people who have not yet been vaccinated and, by keeping cases to a minimum, decrease the likelihood that the alert level system will be needed to control future outbreaks.

Abstract

We present two implementations of an age-structured model for COVID-19 spread in Aotearoa New Zealand with a partially vaccinated population. The first is a deterministic SEIR model, useful for considering population-level dynamics and questions about population immunity. The second is a stochastic branching process, useful for considering smaller community outbreaks seeded by individual border arrivals. This builds on an earlier model used to inform the response to outbreaks of COVID-19 in New Zealand. The main purpose of this paper is to develop a model that can be used as the basis for policy advice on border restrictions and control measures in response to outbreaks that may occur during the vaccination roll-out. We consider a range of scenarios at different stages in the vaccine roll-out, including an unmitigated epidemic and contained local outbreaks. This work is intended to form a foundation for further COVID-19 vaccination modelling in New Zealand that will account for additional demographic variables.

Introduction

COVID-19 was first detected in Wuhan, China in late 2019 [1], before spreading globally to become a pandemic in March 2020 [2]. New Zealand adopted an elimination approach early in the pandemic [3] and as of early May 2021, has experienced significantly lower cumulative incidence than many other countries [4]. Strict border controls have been implemented to keep the virus out, including complete closure to most non-residents, and mandatory quarantine on arrival for those allowed to enter. Although this has been largely successful in preventing community transmission of COVID-19, 10 border-related re-incursions have been detected in the period up to 31 March 2021 [5]. However, with the development of effective vaccines, widespread vaccination could allow these border restrictions to be safely relaxed.

New Zealand's vaccination programme began in February 2021 [6] with frontline border workers and their families receiving first vaccine doses. On the 10th of March 2021, the New Zealand government published their vaccine roll-out plan [7], which identified four successive groups for vaccination:

1. 50,000 border workers, managed isolation and quarantine (MIQ) staff, and their household contacts
2. 480,000 frontline health workers and people in high-risk settings
3. Approximately 1.7m priority individuals, primarily those in older age groups.
4. The remainder of the general population aged 16+ (approximately 2m people).

As of June 2021, vaccination of group 1 is largely complete, groups 2 and 3 are underway and group 4 is scheduled to start in late July following an age-based prioritisation. It has also been confirmed that the roll-out will consist entirely of the two-dose Pfizer/BioNTech BNT162b2 mRNA vaccine. Quantifying the effect of the vaccine roll-out on the population-level risk of severe health outcomes and on the degree of population immunity is critical for informing the response to future border-related outbreaks and decisions about when and how to relax border restrictions.

Current evidence suggests the Pfizer/BioNTech BNT162b2 mRNA vaccine is highly effective against mild and severe COVID-19, with efficacy against disease from stage three clinical trials reported to be 95% (90.3%, 97.6%) [8]. Data from Israel [9], a country with high vaccination

rates, suggests an effectiveness against mild disease of 94% (87%, 98%) and an effectiveness against severe disease of 92% (75%, 100%). The same study also found high effectiveness against documented infection with SARS-CoV-2 of 92% (88%, 95%), suggesting similar effectiveness against transmission [10]. For more details, see Supplementary Information sec 3.

Since late 2020, several SARS-CoV-2 variants of concern have been identified that are more transmissible and/or more resistant to vaccines [11]. The Alpha (B.1.1.7) variant, which became dominant in the UK in early 2021, is estimated to be 43-90% more transmissible than ancestral SARS-CoV-2 [12]. The Delta (B.1.617.2) variant, which was first identified in India and subsequently displaced the Alpha variant in the UK to become dominant in May/June 2021, has a secondary attack rate that is approximately 40% higher than Alpha [13]. The effectiveness of the Pfizer/BioNTech vaccine after 2 doses against symptomatic disease has been estimated to be around 88% for the Delta variant, compared to around 93% for Alpha [14]. This suggests that there is limited reduction in vaccine effectiveness for Delta after two doses (although the same study showed a bigger reduction in effectiveness after only one dose). While we do not consider specific variants in this work, we present results for three scenarios: $R_0 = 3.0$, $R_0 = 4.5$, and $R_0 = 6.0$. These are broadly reflective of spread of the original variant of SARS-CoV-2, the Alpha variant, and the Delta variant respectively. However, there is uncertainty as to the exact value of R_0 for the Delta variant in particular, and these three values should be interpreted as a range of low, medium and high transmission scenarios. While there is considerable uncertainty in vaccine effectiveness, our baseline assumptions are chosen to best represent the current understanding of the vaccine's effect on the Delta variant after two doses.

We develop an age-structured vaccination model that we implement in two ways: (1) a deterministic SEIR model and (2) a stochastic branching process model. The former is intended to analyse population-level dynamics and is similar to other SEIR models developed for COVID-19 [15, 16]. The latter is intended to analyse small-scale outbreaks where stochasticity plays an important role and is similar to our previous branching process model for COVID-19 spread [17, 18]. This builds on earlier models used to inform the response to previous outbreaks of COVID-19 in New Zealand [18, 19] by incorporating the effects of vaccination, age-structured contact rates, and contact tracing.

The primary objective of this paper is to describe the technical details of a model that can be used to answer a wide range of policy questions that may arise in the future, although some results are provided as examples. It is important to note that the model does not yet account for heterogeneity of vaccine coverage, inequities in health outcomes for different groups (for example, we know that Māori and Pacific peoples are more at risk of hospitalisation [20]), or any waning of immunity over time. As such, extensions to the model will be necessary in future work.

Methods

Model Description

We develop an age-structured model of SARS-CoV-2 transmission in New Zealand. The population is divided into 16 age groups: 0 to 4, 5 to 9, ..., 75+. The relative rates of contact within and between age groups is defined by a matrix $\boldsymbol{C}$ (Figure 1), which is a modified version of the New Zealand contact matrix in Prem, Cook [21]. The modification imposes the detailed balance condition, that the total number of contacts from group i to j is the same as the total number of contacts from group j to i, and is detailed in Supplementary information sec 1. 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.

We use the model to investigate transmission dynamics under different levels of vaccination coverage in each group. However, in each model scenario, the proportion of age group i that is vaccinated, denoted v_i, is assumed to be fixed. In other words, we investigate the effect of border incursions of COVID-19 at different stages in the vaccine roll-out, but we do not attempt to model simultaneous vaccination and transmission dynamics. The interplay between these dynamics is important in countries rolling out vaccination programmes at the same time as dealing with high rates of infection, but is less important for countries such as New Zealand where COVID-19 is currently eliminated. For simplicity, we also assume that all individuals are either not vaccinated, or fully vaccinated (i.e. more than 7 days after their second dose). The model ignores seasonal effects which, for temperate climates such as New Zealand's, likely mean the effective reproduction number is around 40% higher in winter than in summer [22].

Disease, Hospitalisation, and Fatality Rates

The proportion of infections (in non-vaccinated individuals) that result in symptomatic disease, hospitalisation, and fatality vary by age and are taken from international literature [23, 24] – see Table 3. This data is presented in 10-year age groups. We use linear interpolation matched to the mid-points of each age group to derive parameters in 5-year age groups. For the 75+ year age group, for symptomatic disease rates we use the reported symptomatic fraction for the 70+ year age group (the oldest group reported in [24]), and for hospitalisations and fatalities, a weighted average of the rates reported for the 75-79 year and the 80+ year age groups in [23].

We further assume that people with subclinical infection (those that do not develop clinical symptoms of COVID-19) have a transmission rate that is $\tau = 50\%$ lower than people with clinical infection [25], although the main results are not highly sensitive to the value of τ (given a fixed value of R_0). The relative transmission rate τ of subclinical infections is important for modelling contact tracing and case isolation as clinical infections are more likely to receive a test than subclinical infections. We assume that the fraction of infections that are clinical increases with age. A consequence of these assumptions is that infections in older age groups are more infectious on average than those in younger groups, although the difference is relatively small (see Supplementary Information sec 2).

Age Structured Susceptibility and Infectiousness

There is substantial evidence that susceptibility to infection varies by age [26]. Multiple studies suggest that young people (typically considered to be under 20 years old, although different studies have used different age bands) may be 50% less susceptible than adults [25, 27, 28]. We include this in the core transmission model using an age-structured relative susceptibility term estimated by Davies, Klepac [25].

There is less certainty around age-dependent infectiousness, with some studies suggesting younger individuals are more infectious [28], while others suggesting they are less infectious [27]. In our model, the youngest age group have an effective transmission rate that is 14% lower than the oldest age group as a consequence of the assumption that subclinical infections are more common in younger individuals and are less infectious than clinical infections.

Vaccine Effectiveness

The effectiveness of the vaccine is described by three parameters: effectiveness against infection (e_I), effectiveness against transmission conditional on breakthrough infection (e_T), and effectiveness against severe disease conditional on breakthrough infection (e_D). For simplicity, we do not directly model any additional reduction in mild disease beyond that provided by prevention of infection. We also assume that the vaccine is equally effective in all age groups and we do not consider the effect of any waning of immunity over time.

Multiple studies have found that the effectiveness of the Pfizer-BioNTech BNT162b2 mRNA vaccine against documented infection to be of the order 90% [9, 29, 30], although there is early evidence of reduced effectiveness in older individuals (64% (14%, 84%) in long-term care facility residents in Denmark) [31] and the Beta (B.1.351) variant of concern (75% (70.5%, 78.9%) in Qatar) [32]. Furthermore, it is unclear whether effectiveness against documented infection arises solely from infection prevention, or is due in part to lower infectiousness or shorter time windows of infectiousness. In either case, effectiveness against documented infection is expected to be a lower bound on overall effectiveness against transmission [10]. There is further evidence of transmission reduction in breakthrough infections, with an odds ratio of infection for household contacts of a vaccinated index case relative to an non-vaccinated index case estimated to be approximately 0.5 [33]. Based on the evidence available, we use effectiveness against infection of $e_I = 70\%$ and effectiveness against transmission given breakthrough infection of $e_T = 50\%$ as baseline assumptions. This is equivalent to an overall reduction in transmission of $1 - (1 - e_I)(1 - e_T) = 85\%$. These parameters are similar to those used in for the Delta variant in recent models of the UK government roadmap for relaxation of restrictions [34].

Efficacy against all disease from clinical trials has been found to be 95% (90.3%, 96.7%) [8] with similar effectiveness against severe disease [32]. For the baseline assumption, we set the effectiveness against severe disease given breakthrough infection to be $e_D = 80\%$, which gives an overall effectiveness against severe disease of $1 - (1 - e_I)(1 - e_D) = 94\%$. We assume that the vaccine effectiveness against fatality is the same as the effectiveness against severe disease.

To provide results over a plausible range for vaccine effectiveness, we consider two additional scenarios, one with lower effectiveness parameters, $e_I = 50\%$ and $e_T = 40\%$ (which gives a 70% reduction in transmission), and one with higher effectiveness parameters, $e_I = 90\%$ and $e_T = 50\%$ (which gives a 95% reduction in transmission). Although a 70% reduction in transmission is lower than published studies indicate, we include this scenario because there is evidence that existing vaccines are less effective against some SARS-CoV-2 variants of concern (e.g. the Beta variant) [35] and there is significant uncertainty about how effectiveness will change for future potential variants. In all cases e_D is set to 80%, implying overall effectiveness against severe disease of 90% and 98% in the lower and higher effectiveness scenarios respectively. Vaccine effectiveness parameters are shown in Table 2.

We assume the vaccine blocks infection in an "all-or-nothing" fashion. This means that the vaccine prevents any infection from occurring in a fixed proportion e_I of the vaccinated population, with the remaining vaccinated population being fully susceptible. An alternative assumption is the "leaky" vaccine, which assumes the vaccine prevents a proportion e_I of infections from occurring in 100% of the vaccinated population. There is at present insufficient empirical evidence to determine which of these assumptions is more appropriate. In scenarios with high R_0 and/or low vaccine effectiveness, the leaky vaccine assumption results in outbreaks with a larger final size than the all-or-nothing assumption [36]. This distinction is less important for determining optimal vaccine roll-out strategies [15], but we nonetheless test the effect of a "leaky vaccine" assumption in Supplementary Information sec 2.

The Next Generation Matrix

The elements of the next generation matrix, $NGM_{i,j}$, describe the average number of individuals in group i that will be infected by a single infectious individual in group j over their whole infectious period given a fully susceptible population:

$$NGM_{i,j} = Uu_i t_I \widehat{C_{j,i}}[p_j^{clin} + \tau(1 - p_j^{clin})]$$

where $\widehat{C}$ is the contact matrix, t_I is the mean duration of the infectious period in days, Uu_i describes the probability that a contact by an individual in age group i with an infectious individual results in transmission, p_j^{clin} is the fraction of infections in age group j that are clinical, and τ is the relative infectiousness of subclinical individuals. The basic reproduction

number of the age-structured model is the dominant eigenvalue of the next generation matrix, i.e.

$$R_0 = \rho(NGM)$$

In model simulations, the value of the constant U is chosen to give the desired value of R_0. We present model results for $R_0 = 3$, $R_0 = 4.5$, and $R_0 = 6.0$ with other values tested in the sensitivity analysis (see Supplementary Information sec. 2).

The next generation matrix can be modified to consider the effect of vaccination. We denote the fraction of age group i that is vaccinated by v_i, then the entries of the vaccinated next generation matrix NGM^v are given by:

$$NGM^v_{i,j} = (1 - e_I v_i)(1 - e_T v_j) NGM_{i,j}$$

This reflects the assumption that transmission from vaccinated individuals is reduced by e_T and infection of vaccinated individuals is reduced by e_I. The vaccinated reproduction number is $R_v = \rho(NGM^v)$.

To model different staging points in the vaccine roll-out, we assume that at most 90% of any age group can be vaccinated, with vaccinations beginning in the 65+ year age groups, then in the 15-64 years age groups, and finally in the 0-14 years age groups. We assume that, within each of these three priority age bands, vaccinations are distributed proportional to group size. This means that, at a given stage in the roll-out, the proportion of individuals that are vaccinated is the same for each five-year age group in the same priority age band.

Implementations

We envisage two distinct applications for a COVID-19 vaccination model in New Zealand. The first is to consider the effects of the vaccination programme on the degree of population immunity and potential for spread in the wider community. The second is to consider the effect of vaccination on relatively small community outbreaks seeded by border cases, like those that occurred in August 2020 and February 2021 [5].

To accomplish both goals, we propose two implementations of this model: (1) a deterministic SEIR model, and (2) a stochastic branching process. The former focusses on average behaviour, allowing it to efficiently model population-level dynamics. The latter considers

individual cases and stochasticity in transmission (e.g. superspreading), elements which are critical for analysing small clusters. Features specific to each implementation are described in Table 1. See Supplementary Information sec 1. for a full mathematical description of both implementations.

For the branching process implementation, we initialise each simulation with a single non-vaccinated seed case in the general community (that is, we do not model higher vaccination or surveillance rates for border workers, who are more likely to be a seed case). For each simulation, the seed case is randomly assigned to an age group with probability proportional to the size of that age group. While other initialisation assumptions are possible (e.g. an international arrival infected before travel), we do not consider these here. We simulate the outbreak dynamics under three different levels of community testing: low, moderate, and high, corresponding to the probability of detecting a symptomatic COVID-19 case of 5%, 15%, and 30% respectively during the period of time before the outbreak is first detected. We assume there is a delay from symptom onset to the return of a positive test result of 4 days on average. For simplicity we also assume the community detection rate is the same for vaccinated and non-vaccinated cases and across all age groups, although future work may be required to consider how varying this impacts the results.

Once an outbreak is detected, we assume that testing rates increase and case isolation and contact tracing begins. As a simplified representation of this, we assume that all infections have a probability of being detected by contact tracing of 70%. This occurs with a mean delay of 6 days since the exposure time. We further assume that, independently of contact tracing, the probability of detecting a clinical infection as a result of testing following symptom onset increases to 40%. This represents increased awareness of infection risk. Once an infection is detected, the individual is assumed to be immediately perfectly isolated so that there is no further transmission. Because the stochastic implementation of the model is used to represent relatively small outbreaks, we assume that the testing and contact tracing parameters are independent of the number of cases (i.e. we do not model a decrease in system performance as cases stretch case management capacity). Together, case isolation and contact tracing measures result in a reduction in the reproduction number of around 44% (see Supplementary Information sec. 1), which is consistent with estimates from empirical data from New Zealand's

March-April 2020 outbreak [19]. The individual values of the testing probabilities and delay times are less important than their combined effect on the reproduction number.

	Branching Process	Deterministic SEIR
Heterogeneity in transmission	Included. Increases the variance between individuals in the number of secondary infections, e.g. due to superspreading. This means that some outbreaks will naturally go extinct whereas others will grow very rapidly.	Not included. Deterministic models assume mean-field behaviour and do not allow for individual heterogeneity. These models cannot be used for estimating probability of elimination or dynamics of small outbreaks.
Infection-conferred immunity	Not included. The branching process is only used to consider small outbreaks where infection conferred immunity is expected to have negligible effect.	Included. The deterministic model is used to consider population-level outbreaks which could confer significant immunity.
Regular Importations	Not included. This implementation considers the downstream cases from a single seed-case. For a sufficiently small number of controlled outbreaks, the effect of regular importations can be considered by treating each importation independently.	Included.
Post-detection control measures	Included. Case isolation and contact tracing begin once an outbreak is detected.	Not included. The SEIR implementation models an epidemic in the absence of other interventions.

Table 1. Description of differences in assumptions between the stochastic branching process and deterministic SEIR implementations.

Effectiveness	Baseline	Lower Effectiveness	Higher Effectiveness
Against Infection (e_I)	70%	50%	90%
Against Transmission given infection (e_T)	50%	40%	50%
Against disease given infection (e_D)	80%	80%	80%
Implied overall transmission reduction	85%	70%	95%
Implied overall protection against severe disease	94%	90%	98%

Table 2. Vaccine effectiveness parameters. These are chosen to reflect estimates of the effectiveness of the Pfizer-BioNTech mRNA vaccine after 2 doses [34]. See Supplementary Information sec. 3 for more details. The overall reduction is transmission is given by $1-(1-e_I)(1-e_T)$, and the overall protection against severe disease is $1-(1-e_I)(1-e_D)$.

Parameter	Branching Implementation	SEIR Implementation	Source
Default R_0	3.0, 4.5, 6.0		
Relative infectiousness of subclinical individuals (τ)	50%		[25]
Latent period	N/A	2.55 days	Suppl. 1
Infectious period	N/A	5 days	[15]
Generation time distribution	Gamma(5.67, 2.83) *(mean = 5.05 days)*	N/A	[37]
Average hospital length of stay	8 days		[20]
Probability of detecting a case	Scenario dependent	N/A	
Incubation period	Gamma(5.8, 0.95) *(mean = 5.51 days)*	N/A	[19]
Delay from infection to contact tracing	Exponential(6 days)	N/A	Assumed
Delay from symptom onset to detection	Exponential(4 days)	N/A	Assumed

Age groups	[0-4, 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75+]		
Population size	N/A	5 million	
Population distribution	N/A	[5.98%, 6.39%, 6.56%, 6.17%, 6.59%, 7.40%, 7.44%, 6.62%, 6.08%, 6.41%, 6.43%, 6.38%, 5.77%, 4.90%, 4.24%, 6.64%]	[38]
Proportion of infections causing clinical disease by age group	[0.544, 0.555, 0.577, 0.5985, 0.6195, 0.6395, 0.6585, 0.6770, 0.6950, 0.7118, 0.7273, 0.7418, 0.7553, 0.768, 0.78, 0.8008]		Linear interpolation of [24]
Proportion of infections resulting in hospitalisation by age group	[0, 0.0001, 0.0003, 0.0029, 0.0079, 0.0164, 0.0283, 0.0364, 0.0405, 0.0523, 0.0718, 0.0907, 0.1089, 0.13, 0.154, 0.178]		Linear interpolation of [23]
Proportions of infections resulting in fatality by age group	[0, 0.00003, 0.00006, 0.0001, 0.0002, 0.0004, 0.0007, 0.001, 0.0014, 0.0027, 0.0049, 0.0093, 0.016, 0.0252, 0.0369, 0.0664]		Linear interpolation of [23]
Relative susceptibility by age group (compared to 60-64 year-olds)	[0.462, 0.457, 0.445, 0.558, 0.795, 0.934, 0.974, 0.977. 0.942, 0.931, 0.942, 0.965, 1.00, 0.977, 0.896, 0.856]		Linear interpolation of [25]

Table 3. Table of parameter values and data. Gamma distributions reported with shape parameter then scale parameter in parenthesis. Baseline values given in bold.

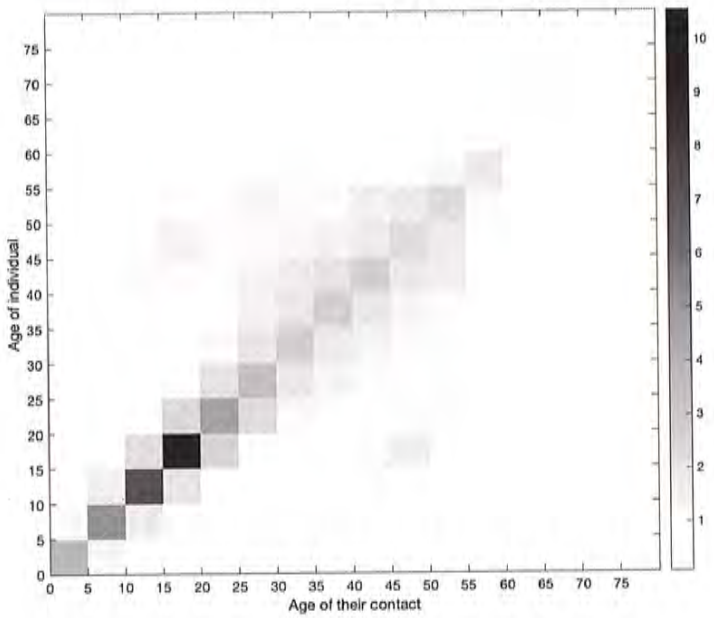

Figure 1. Visualisation of the modified contact matrix derived from [21] by imposing a symmetry condition described in the text. Darker colours indicate more contacts.

Results

The purpose of this paper is to develop a vaccination model that can be used as the basis for policy advice on border restrictions and control measures in response to outbreaks. Here we investigate some simple scenarios representing different stages in the vaccine roll-out and different border policies.

Effect of Vaccination on the Reproduction Number

A key aim of national vaccination programmes is to vaccinate enough people that the effective reproduction number falls below 1 without the need for other interventions. The proportion of the population that need to be vaccinated to achieve this is known as the herd immunity threshold or population immunity threshold. In this section, we use the vaccinated next generation matrix to estimate the reproduction number R_v at different staging points in the vaccine roll-out, and to predict the vaccination coverage required to reach the population immunity threshold.

With an assumed basic reproduction number of $R_0 = 3$ before vaccination, reaching the population immunity threshold ($R_v < 1$) requires vaccinating 90% of all over 15-year-olds, unless the vaccine meets the high effectiveness criteria (i.e. is 95% effective at reducing overall transmission) (Figure 2a). Under lower effectiveness parameters, the population immunity threshold cannot be reached. Similar results have been found for Australia [39] and the UK [40].

With an assumed basic reproduction number of $R_0 = 4.5$ before vaccination, reaching the population immunity threshold under baseline effectiveness assumptions requires vaccinating nearly 90% of the total population including children (Figure 2b). Vaccinating 90% of the population reduces R_v to 0.92. In such a scenario, it is important to realise that some population sub-groups will still be likely to have a reproduction number greater than 1 and so could still be vulnerable to a large outbreak. Even under the higher effectiveness assumption, vaccination of a significant proportion of under 15-year-olds is needed to reach the population immunity threshold. With an assumed basic reproduction number of $R_0 = 6.0$ before vaccination, the population immunity threshold can only be reached under the higher effectiveness scenario, and requires vaccination of under-15-year-olds (Figure 2c).

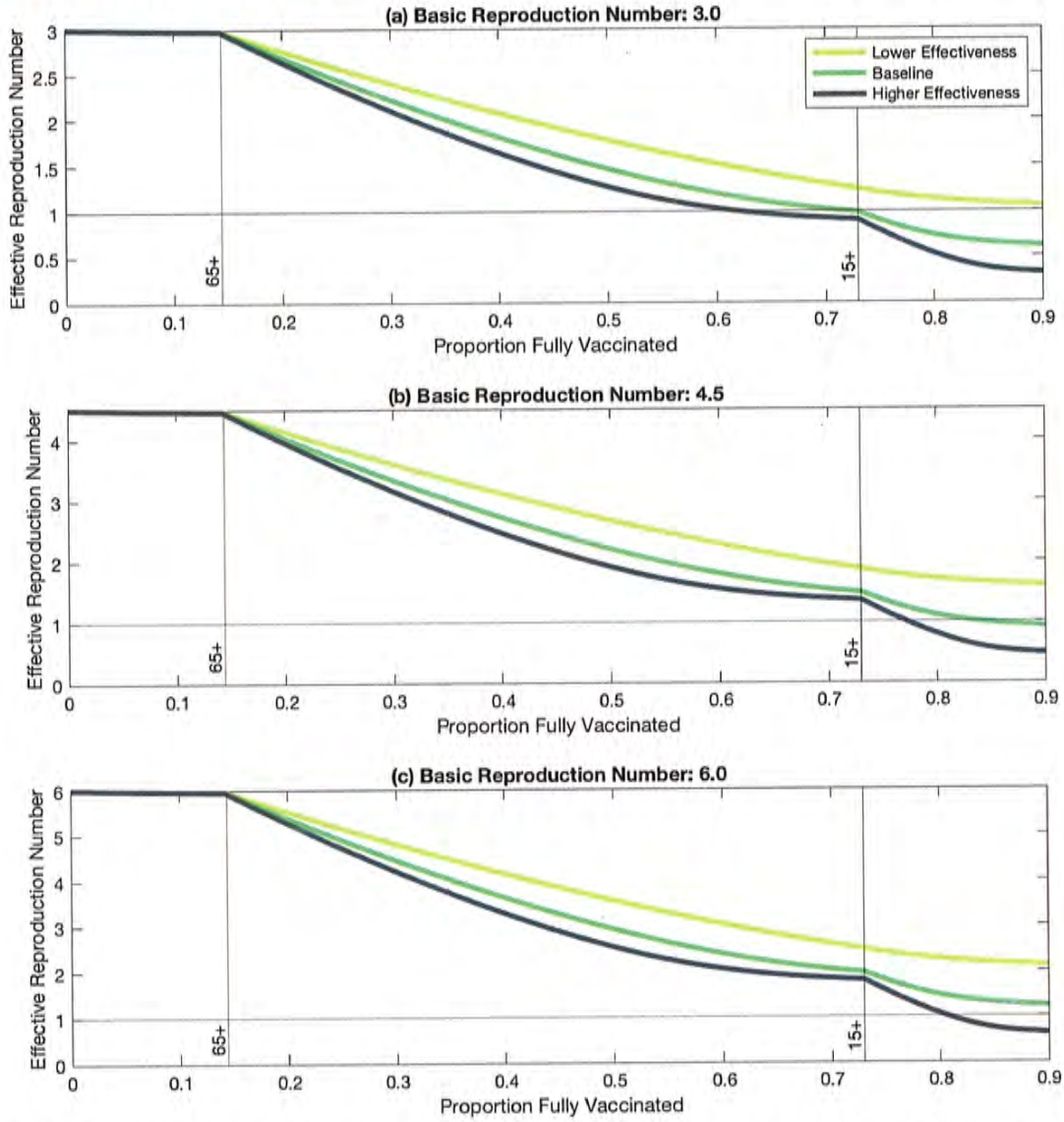

Figure 2. Effective reproduction number R_v after vaccination as a function of total vaccine courses administered, with a maximum of 90% coverage in any age group, for (a) $R_0 = 3$, (b) $R_0 = 4.5$, and (c) $R_0 = 6.0$. Along the x-axis, the roll-out begins in the 65+ year-old age group. Once 90% of the 65+ year old group is vaccinated, vaccination of the 15-64-year-old group begins. Once 90% of the 15-64-year-old group is vaccinated, vaccination of the under 15-year-old age group begins.

Figure 2 further shows that the first stage of the vaccine roll-out, where older age groups are vaccinated, does not substantially reduce the effective reproduction number at an overall

population level. These age groups have lower contact rates than other age groups, so contribute a relatively small amount to overall transmission. These groups are at greatest risk of severe health outcomes, however, so vaccinating them is expected to substantially reduce the potential morbidity and mortality if a large outbreak were to occur (see below).

The New Zealand government recently announced that the under 65 year old age group will be prioritised in 10-year age bands, although there is no end-date for the vaccination of a given age band and there is likely to be significant overlap between vaccination of different age bands. This is a more fine-grained age sequencing than has been modelled here. Results are presented in Supplementary Information sec. 2 under a more structured roll-out, starting in 65+ year-olds before progressing through the rest of the population in 5-year age bands, with the same assumed 90% maximum coverage in any one group. These results show that a more age-structured roll-out results in a slower decrease in the reproduction number, but a faster reduction in the expected health impacts of any outbreak.

Figure 3 shows the number of fully vaccinated individuals required to reach the population immunity threshold at varying levels of R_0 for each of the vaccine effectiveness scenarios. Under the baseline vaccine effectiveness assumptions, the maximum value of the basic reproduction number for which population immunity can be achieved with 90% coverage of over 15-year-olds is $R_0 = 3.0$. If R_0 is between 3.0 and 4.9, population immunity can be achieved with 90% coverage of the whole population. If R_0 is above 4.9 (which is possible for the Delta variant of concern [34]), population immunity cannot be achieved under baseline vaccine assumptions without a vaccine coverage of more than 90% of the whole population.

Under the high vaccine effectiveness assumptions, population immunity can be achieved with 90% coverage of over 15-year-olds provided R_0 is less than 3.3, and with 90% coverage of the whole population if R_0 is between 3.3 and 9.6. Under the low effectiveness assumptions (for example representing a variant of concern with significant immune escape), population immunity cannot be achieved for any value of R_0 greater than 2.8.

Table 4 gives the proportion requiring vaccination to reach the population immunity threshold under the three vaccine effectiveness scenarios outlined in Table 2 and under three values of

R_0. These values reflect the population immunity threshold under the roll-out assumptions described above. When $R_0 = 4.5$, under baseline vaccination effectiveness assumptions, the population immunity threshold is estimated to be 83%. This consists of 90% coverage of all over 15-year-olds, with additional coverage of 55% of the under 15-year old age groups. Values with an asterisk are greater than 90%, and assume equal coverage in all age groups.

	Baseline	**Lower Effectiveness**	**Higher Effectiveness**
Vaccine Effectiveness	$e_I = 70\%, e_T = 50\%$	$e_I = 50\%, e_T = 40\%$	$e_I = 90\%, e_T = 50\%$
$R_0 = 3.0$	71%	94%*	62%
$R_0 = 4.5$	83%	-	77%
$R_0 = 6.0$	97%*	-	81%

Table 4. Population immunity threshold estimates 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%and assume equal coverage in all age groups.

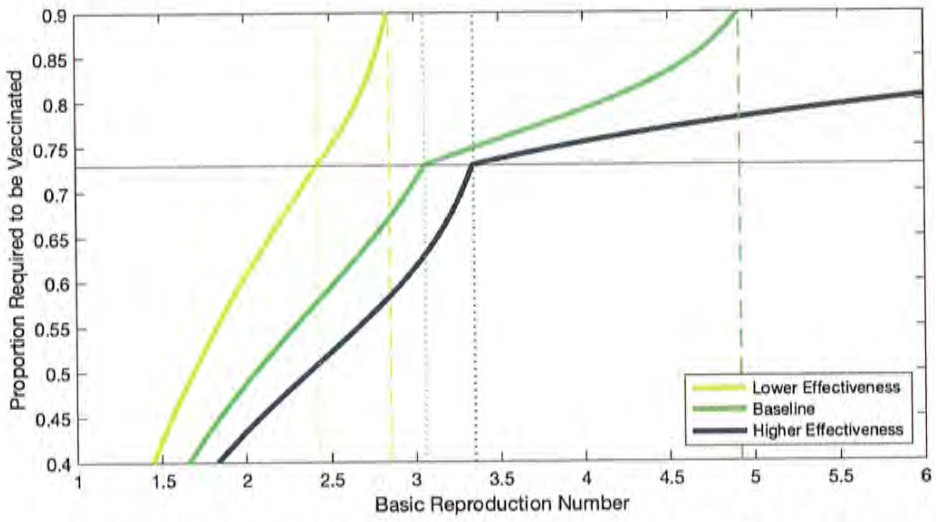

Figure 3. Number of vaccinated individuals required to reach the population immunity threshold (i.e. $R_v = 1$) for varying values of R_0 under the same age-prioritised roll-out sequence and maximum 90% coverage as in Figure 2. Points above the thin horizontal line require vaccination of under 15-year-olds. Vertical dotted lines indicate the largest value of R_0 for which the population immunity threshold can be reached with 90% coverage of over 15-

year-olds. Vertical dashed lines indicate the largest value of R_0 for which the population immunity threshold can be reached with 90% coverage of the total population.

Open Borders Scenario

To model the effect of relaxing border restrictions, which currently require 14 days of quarantine for all international arrivals from outside a safe travel zone, we assume there are five non-vaccinated imported cases per day. We assume imported cases are distributed over all age groups in proportion to the size of those groups, spend the full infectious period in the community, and have the same contact patterns as the general population. We run the deterministic SEIR implementation for two years (730 days) in a population with 90% vaccination coverage across all age groups, and 90% vaccination coverage across over 15-year-olds. Note that, if the model is run for a longer time period, more cases, hospitalisations, and fatalities occur, even if $R_v < 1$.

The choice of five imported non-vaccinated infected individuals per day is arbitrary and, over the two-year simulation, equates to approximately 2,500 symptomatic cases, 200 hospitalisations, and 35 fatalities among imported cases, independent of vaccine effectiveness and coverage, and the assumed basic reproduction number R_0. To focus on the effects of community transmission, we do not include imported cases in the results presented below. Other case importation rates are tested in Supplementary Information sec 2.

These are counterfactual scenarios in which no interventions are made to control the epidemic beyond vaccination. This does not mean that the number of hospitalisations and fatalities reported in Table 5 and 6 would be expected to occur, but it demonstrates that, under these scenarios, a significant public health response would still be needed to prevent a major epidemic and the outlined health impacts.

90% Coverage of 15+ year olds

With an assumed value of $R_0 = 3.0$ before vaccination, we found that $R_v = 0.98$ under baseline vaccine effectiveness parameters and with coverage of 90% of the over 15-year-old age groups. As this is only just below the population immunity threshold, each imported infection still leads to many local cases, so there are still substantial health outcomes in this scenario (150,000 local infections, 2,000 hospitalisations and 230 fatalities). Even under the

higher effectiveness vaccine scenario, R_v is only reduced to 0.9, leading to 25,000 local infections, 310 hospitalisations, and 37 fatalities.

The difference between scenarios with $R_v > 1$ and $R_v < 1$ is clearly visible in the results. When $R_0 = 3$, the lower effectiveness vaccine only brings R_v to 1.24, so this scenario exhibits a self-sustaining epidemic wave resulting in 1,100,000 local infections, 15,000 hospitalisations, and 1,800 fatalities. When $R_0 = 4.5$ and $R_0 = 6$, values that are more likely for the Delta variant, no scenario reaches the population immunity threshold, regardless of vaccine effectiveness. Under baseline effectiveness assumptions, $R_v = 1.47$ and $R_v = 1.96$ respectively, leading to 1,300,000 and 1,800,000 local infections, and 2,200 and 3,400 fatalities. These results are outlined in Table 5.

$R_0 = 3.0$	**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	150,000 (44%)	1,100,000 (61%)	25,000 (18%)
Hospitalisations	2,000 (35%)	15,000 (47%)	310 (15%)
Fatalities	230 (35%)	1,800 (47%)	37 (15%)
Peak in hospital	N/A	990 (after 210 days)	N/A
$R_0 = 4.5$	**Baseline**	**Lower Effectiveness**	**Higher Effectiveness**
R_v	1.47	1.86	1.35
Infections	1,300,000 (44%)	2,200,000 (58%)	690,000 (18%)
Hospitalisations	17,000 (35%)	30,000 (47%)	8,800 (15%)
Fatalities	2,200 (35%)	4,100 (47%)	1,100 (15%)
Peak in hospital	2,000 (after 140 days)	5,200 (after 100 days)	750 (after 174 days)
$R_0 = 6.0$	**Baseline**	**Lower Effectiveness**	**Higher Effectiveness**
R_v	1.96	2.48	1.80
Infections	1,800,000 (44%)	2,700,000 (58%)	1,100,000 (19%)
Hospitalisations	25,000 (35%)	37,000 (47%)	15,000 (15%)
Fatalities	3,400 (35%)	5,300 (47%)	2,000 (15%)
Peak in hospital	4,700 (after 90 days)	8,900 (after 70 days)	2,400 (after 110 days)

Table 5. Results from an unmitigated epidemic with $R_0 = 3$ (upper), $R_0 = 4.5$ (middle), and $R_0 = 6$ (lower) before vaccination and 90% vaccine coverage for over 15-year-olds. All results

are given to 2 significant figures and only include the locally acquired cases, not those imported from overseas. All scenarios assume protection against severe disease given infection of $e_D = 80\%$. Numbers in parentheses are the percentage of infections/hospitalisations/fatalities that occur in vaccinated individuals.

It is worth noting that in all scenarios, a significant proportion of infections, hospitalisations and deaths occur in vaccinated individuals, which is the expected outcome when vaccine coverage is high and effectiveness is less than 100%. This demonstrates the need for continued testing and other public health measures to be directed at vaccinated as well as non-vaccinated individuals.

90% Coverage of Entire Population

With an assumed value of $R_0 = 3.0$ before vaccination, we found that $R_v = 0.61$ under baseline vaccination effectiveness parameters and with 90% coverage across the entire population. In the lower effectiveness scenario, $R_v = 1.06$ and in the higher effectiveness scenario $R_v = 0.31$. Under the same vaccine coverage, but with $R_0 = 4.5$, we found $R_v = 0.92$ in the baseline scenario, with $R_v = 1.58$ and $R_v = 0.47$ in the lower and higher effectiveness scenarios respectively. Finally, with $R_0 = 6.0$, we found $R_v = 1.2\,2$ in the baseline scenario and $R_v = 2.11$ and $R_v = 0.63$ in the lower and higher effectiveness scenarios respectively.

The baseline effectiveness scenario does not completely suppress transmission but does prevent epidemic growth when $R_0 = 3$ and 4.5, resulting in much lower health impacts with 240 hospitalisations and 26 fatalities (when $R_0 = 3$), or 3,800 hospitalisations and 410 fatalities (when $R_0 = 4.5$). Baseline effectiveness is not sufficient to prevent epidemic growth when $R_0 = 6$, so the health impacts remain substantial (14,000 hospitalisations and 1,700 fatalities). The higher effectiveness scenario further reduces impacts to 100 hospitalisations and 11 fatalities (when $R_0 = 3$), or 240 hospitalisations and 26 fatalities (when $R_0 = 4.5$), and prevents epidemic growth even when $R_0 = 6$, resulting in 710 hospitalisations and 77 fatalities. This shows the value of increasing vaccination coverage even after the population immunity threshold is reached.

$R_0 = 3.0$	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	0.61	1.06	0.31
Infections	13,000 (73%)	440,000 (82%)	3,700 (47%)
Hospitalisations	240 (35%)	6,700 (47%)	100 (15%)
Fatalities	26 (35%)	730 (47%)	11 (15%)
Peak in hospital	N/A	210 (after 310 days)	N/A
$R_0 = 4.5$	Baseline	Lower Effectiveness	Higher Effectiveness
R_v	0.92	1.58	0.47
Infections	210,000 (73%)	1,500,000 (82%)	8,700 (47%)
Hospitalisations	3,800 (35%)	24,000 (47%)	240 (15%)
Fatalities	410 (35%)	2,900 (47%)	26 (15%)
Peak in hospital	N/A	3,200 (after 120 days)	N/A
$R_0 = 6.0$	Baseline	Lower Effectiveness	Higher Effectiveness
R_v	1.22	2.11	0.63
Infections	770,000 (73%)	2,000,000 (82%)	26,000 (47%)
Hospitalisations	14,000 (35%)	32,000 (47%)	710 (15%)
Fatalities	1,700 (35%)	4,300 (47%)	77 (15%)
Peak in hospital	1,300 (after 150 days)	6,600 (after 80 days)	N/A

Table 6. Results from an unmitigated epidemic with $R_0 = 3$ (upper), $R_0 = 4.5$ (middle) and $R_0 = 6$ (lower) before vaccination and 90% vaccine coverage for the entire population (including children). All results given to 2 significant figures and do not include imported cases, only the resulting community transmission cases. All scenarios assume protection against severe disease given infection of $e_D = 80\%$. Numbers in parentheses are the percentage of infections/hospitalisations/fatalities that occur in vaccinated individuals.

Other Stages in the Vaccine Roll-out

The results in Tables 5 and 6 can be reproduced for any level of vaccine coverage. Figure 4 and 5 presents the results from an unmitigated epidemic following the same vaccine roll-out sequence as in Figure 2. This shows that, although vaccinating older age groups first does not greatly reduce in the total number of infections, it does lead to a sharper reduction in hospitalisations and fatalities. These scenarios assume that the vaccination programme ceases once the outbreak begins, so these scenarios should be considered as indicative of the potential effect of opening the border once a certain number of individuals are fully vaccinated.

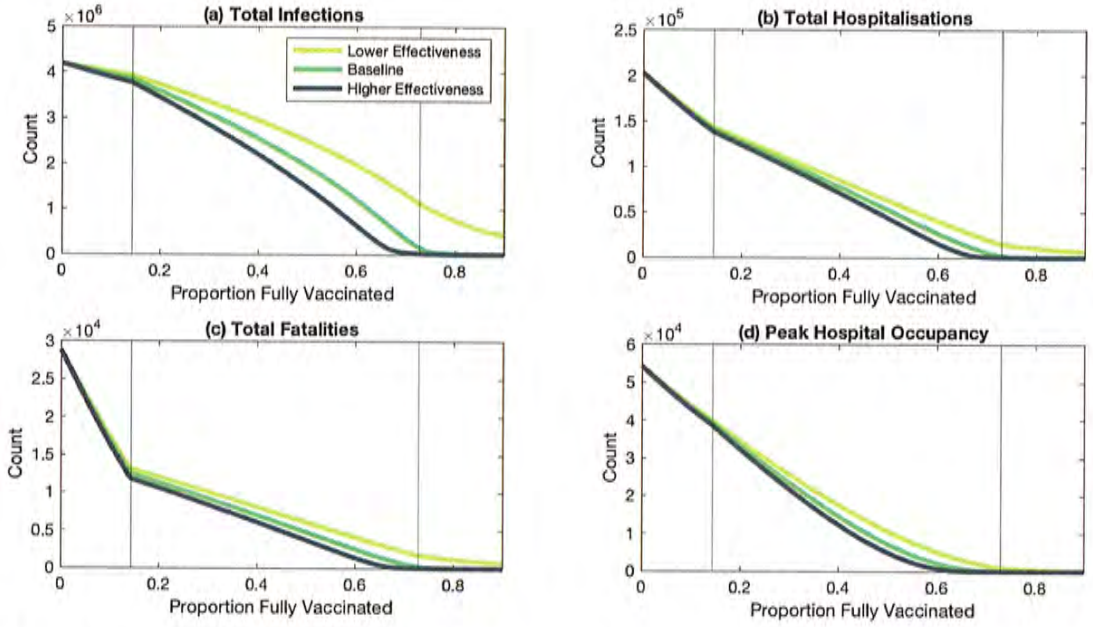

Figure 4. Total infections (a), hospitalisations (b), fatalities (c), and peak hospital occupancy (d) at the three vaccine effectiveness assumptions outlined in Table 2 and $R_0 = 3.0$. Results are from a 2-year simulation, assuming there is no further vaccination after the outbreak begins.

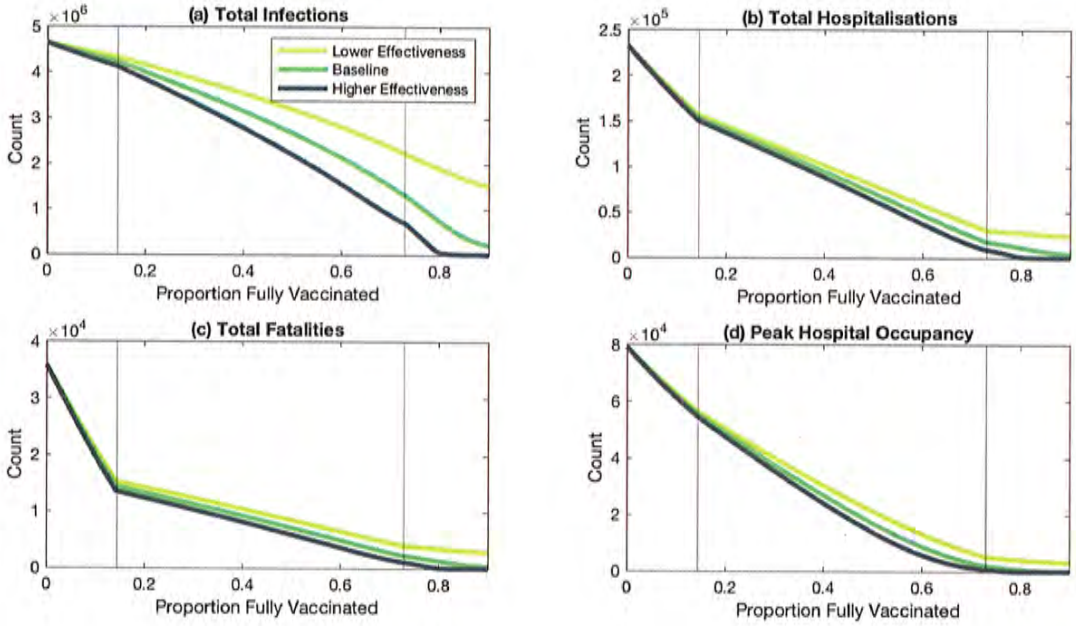

Figure 5. Total infections (a), hospitalisations (b), fatalities (c), and peak hospital occupancy (d) at the three vaccine effectiveness assumptions outlined in Table 2 and $R_0 = 4.5$. Results are from a 2-year simulation, assuming there is no further vaccination after the outbreak begins.

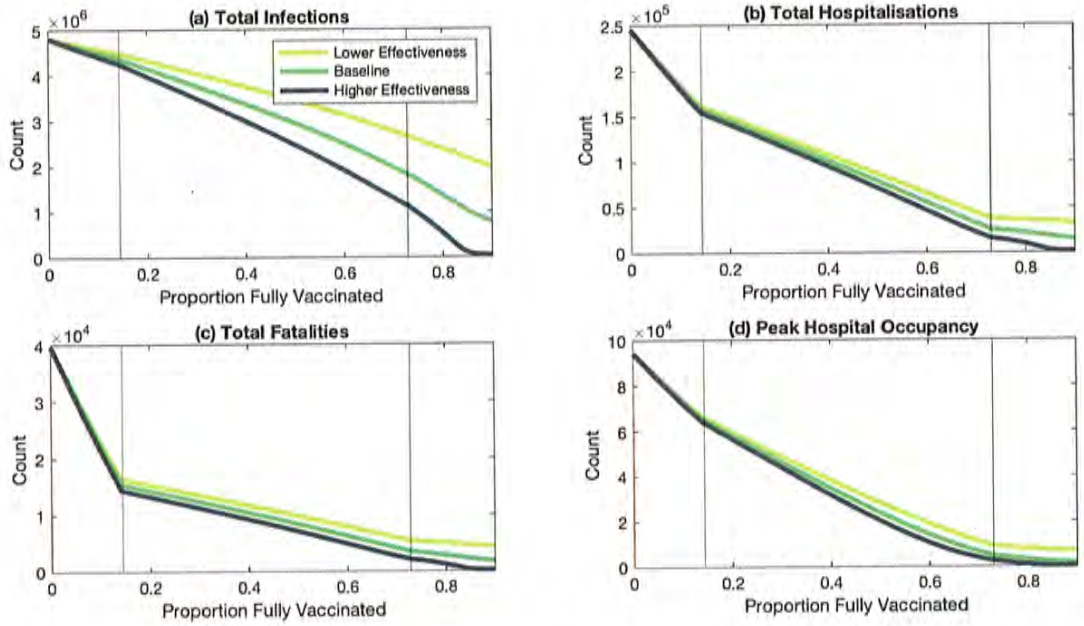

Figure 6. Total infections (a), hospitalisations (b), fatalities (c), and peak hospital occupancy (d) at the three vaccine effectiveness assumptions outlined in Table 2 and $R_0 = 6.0$. Results are from a 2-year simulation, assuming there is no further vaccination after the outbreak begins.

The model does not account for demographic changes over the two-year period we are considering, which may become important as part of a long-term strategy (e.g. births increase the number of susceptible individuals). Thus, the assumption that the proportion of people in each age group that are vaccinated remains constant may not be valid in the long run unless the vaccine is scheduled for people when they reach a prescribed age. Demographic changes, the possibility of waning immunity, and ongoing vaccination programmes will likely have an impact on these outcomes in the long term.

Outbreak Sizes and Control

Vaccination also changes the characteristics of border-related outbreaks. We use the stochastic branching process implementation to simulate community outbreaks seeded by a single infected case. The results from these simulations can be interpreted as the expected number of

community cases per fully infectious case arriving at the border. We consider four outputs: (1) the number of infections when an outbreak is first detected, (2) the probability that an outbreak eliminates before it reaches 1,000 cumulative infections without population-level interventions such as gathering size limits or business closures, (3) the time from detection until elimination, defined as 14 days since the last infection event, with fixed post-detection interventions, and (4) the number of hospitalisations that occur before elimination is achieved. As these outbreaks are all relatively small we do not model fatalities.

Results for output (1), the outbreak size at time of first detection, show that, until high levels of vaccination are achieved, probability of detection is the most important factor affecting the size of outbreaks at detection (Figure 6). This shows the ongoing importance of high rates of community testing for early outbreak detection.

Output (2), the probability of elimination, assumes case isolation and contact tracing are operating once the outbreak is detected, but no population-level interventions are taken. In a completely non-vaccinated population, the probability of elimination is around 60% when $R_0 = 3.0$, between 45% and 50% when $R_0 = 4.5$, and around 40% when $R_0 = 6$. (Figure 7). This is partly due to stochasticity of transmission, meaning that a relatively high proportion of infected individuals do not transmit the virus. Vaccination of the older age groups at the start of the roll-out does not substantially change the probability of elimination. However, once the roll-out in those under 65 years old begins (> 16% fully vaccinated), the probability of elimination steadily increases with vaccine coverage.

Once $R_v = 1.77$, case isolation and contact tracing are theoretically sufficient to bring the effective reproduction number below 1. When $R_0 = 3.0$ this occurs once approximately 41% of the population have been vaccinated, when $R_0 = 4.5$ this occurs once 60% of the population have been vaccinated, and when $R_0 = 6.0$ this occurs once 75% of the population have been vaccinated. Once coverage is greater than these values, assuming contact tracing and case isolation remain effective even in very large outbreaks, all outbreaks will eventually eliminate. In practice, however, the observed probability of elimination in Figure 8 is less than 1 because some outbreaks still reach 1,000 cases before elimination.

Heterogeneities in vaccine coverage, which are not modelled here, are also likely to have a significant effect on these results. For example, increased vaccination coverage in border workers and their families are likely to increase the probability of elimination for border-related outbreaks. However, if infection reaches a community with lower vaccine coverage, then the probability of elimination would be lower than results based on the average national vaccine coverage would suggest.

Outputs (3) and (4), the time to elimination and the number of hospitalisations, assume that, once a case is detected, population-level controls equivalent to an additional 67% reduction in the reproduction number are implemented. This corresponds to a combined reduction from case isolation, contact tracing and population-level controls of 81%, from $R = 3.0$, $R = 4.5$, and $R_0 = 6.0$, before control to $R = 0.56$, $R = 0.84$ and $R = 1.1$ respectively after control. This is consistent with estimates of the relative reduction in transmission from control measures in response to previous outbreaks in New Zealand and may be achieved via Alert Level changes or other public health interventions. When $R_0 = 6$, however, a reduction of 81% is not sufficient to control an outbreak, suggesting that stronger measures would be required for a fully non-vaccinated population. However, this result should be viewed with caution because we currently lack data on transmission of new variants under stay-at-home restrictions or other strong interventions. Indeed, the assumption we make that a given control measure leads to the same proportional reduction in transmission regardless of R_0 may not always be valid [41].

As the detection probability or vaccination coverage increases, the outbreak size at first detection decreases, so elimination can be achieved more rapidly (Figure 8). Vaccination also reduces the effective reproduction number after outbreak detection, so there will be less onward transmission. It may be possible to lift population-level restrictions before elimination is formally achieved, but we do not model this here. The number of hospitalisations decreases sharply with vaccination coverage (Figure 9), thanks to protection of high-risk groups in the early stages of the vaccine roll-out.

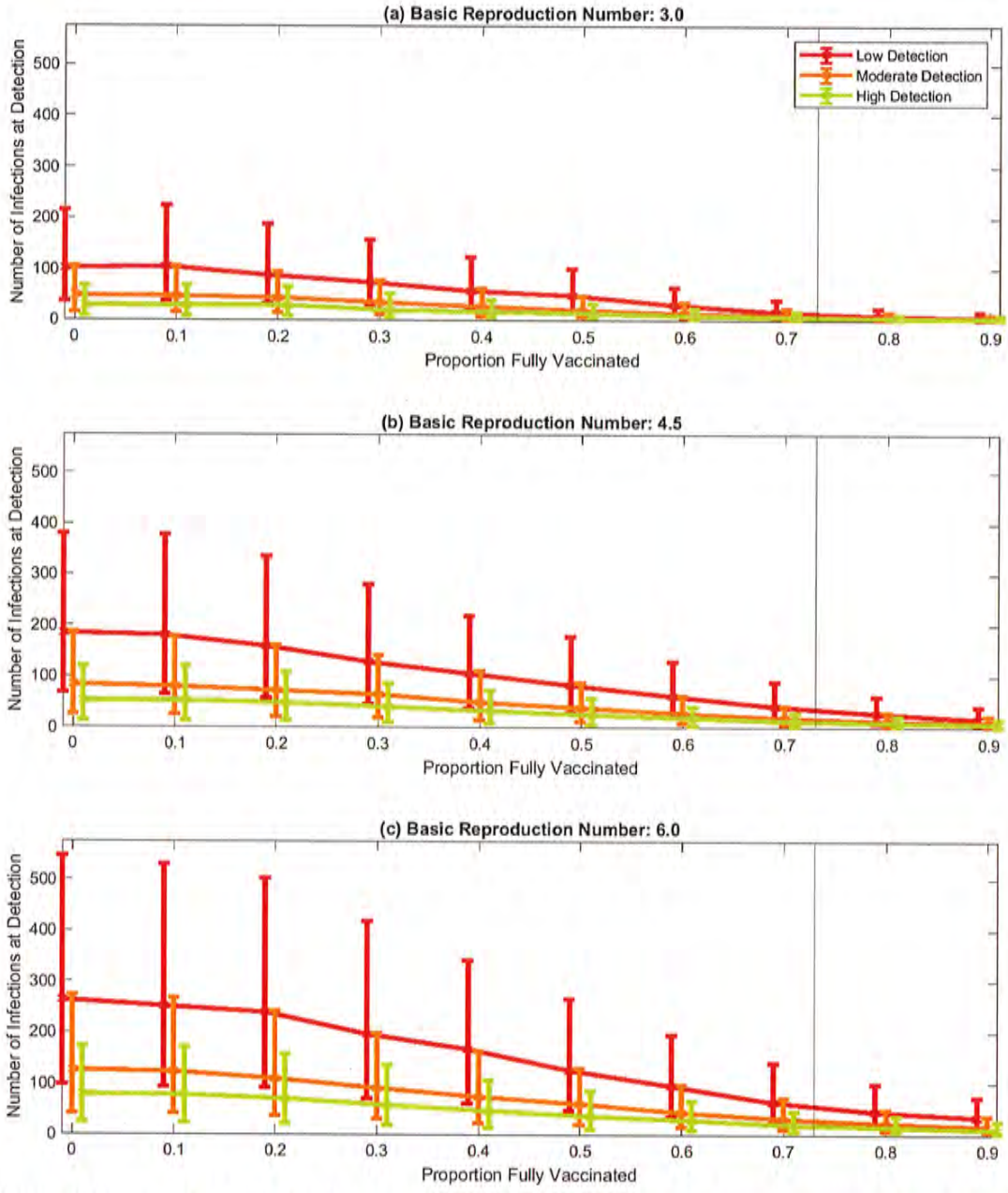

Figure 7. The number of infections when a community outbreak is first detected at three levels of testing. Points give the median and error bars represent the interquartile range. Low detection rates assume a symptomatic individual in the community has a 5% of being detected, moderate detection assumes 15%, and high assumes 30%. The solid line represents the threshold at which vaccination of the under 15-year-old age group begins. Results are from 10,000 independently initialised realisations of the stochastic model.

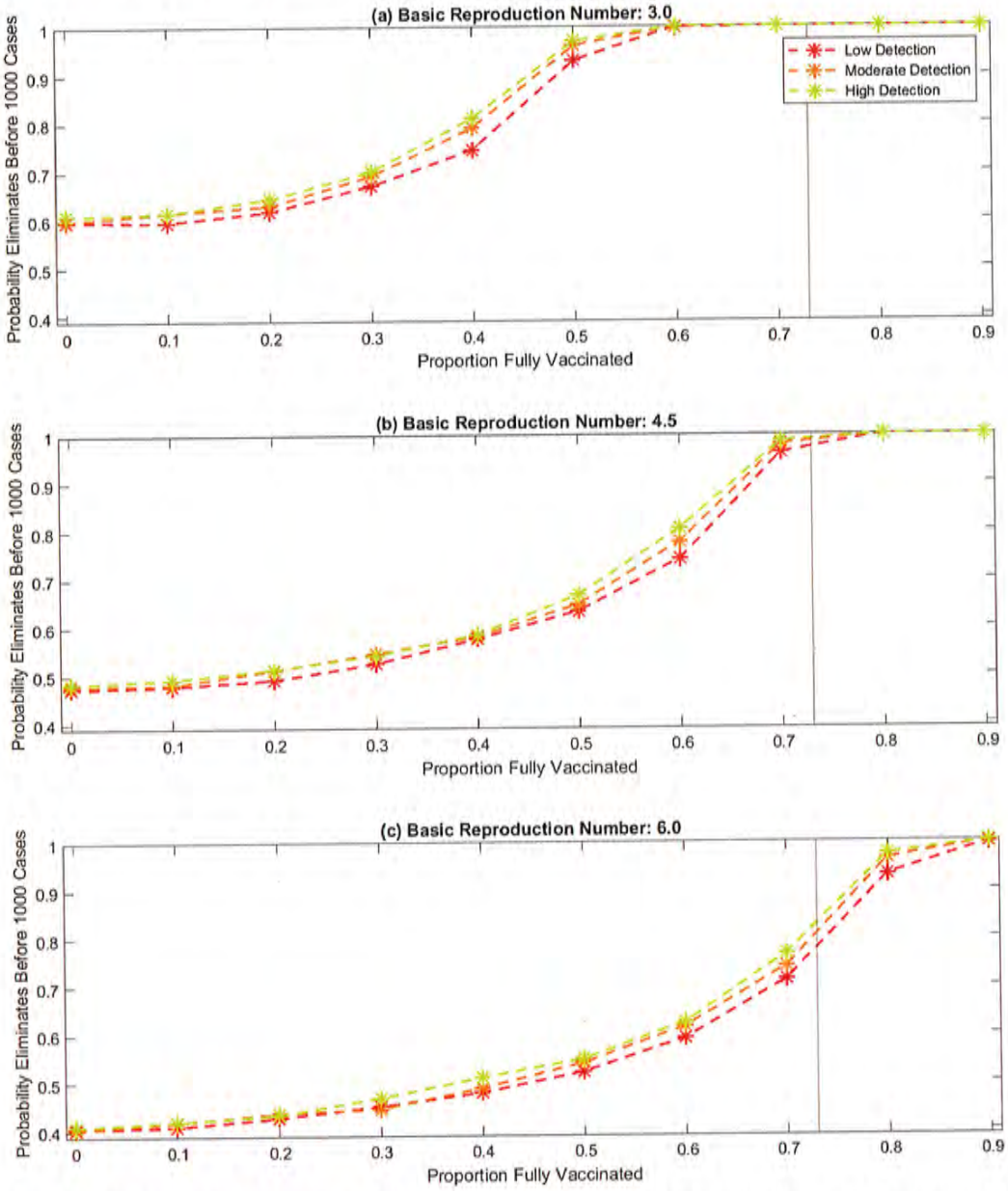

Figure 8. The probability that an outbreak is eliminated before reaching 1,000 cases without population-level interventions. Case isolation and contact tracing are assumed to be operating after the outbreak is detected, leading to a 43% reduction in R. The solid line represents the threshold at which vaccination of the under 15-year-old age group begins. Results are from 10,000 independently initialised realisations of the stochastic model.

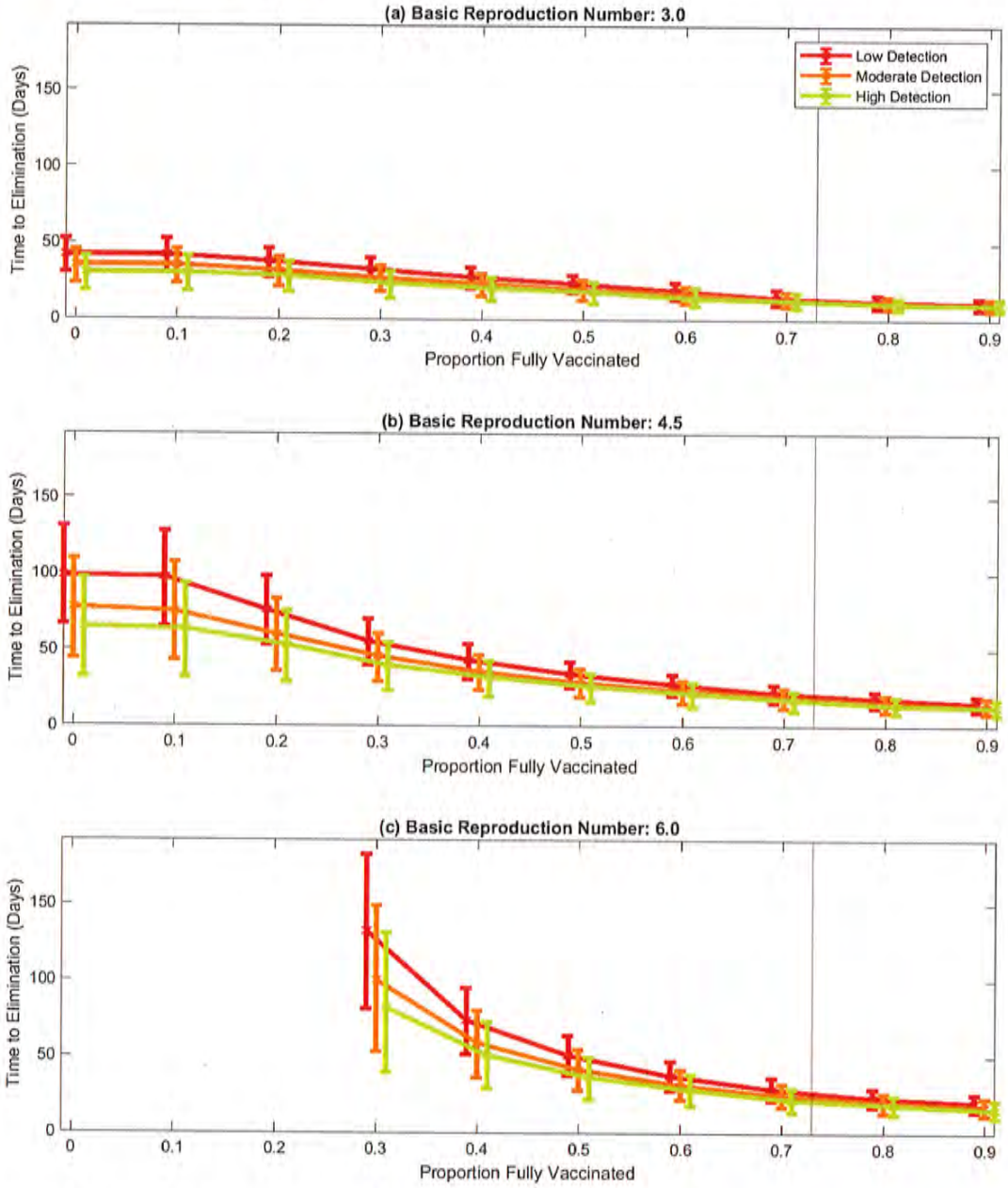

Figure 9. The time from detection to elimination assuming the introduction of case isolation, contact tracing and population-level controls after the outbreak is detected, leading to an aggregate 81.4% reduction in R. Elimination is defined as 14 days since the last infection event, but in some trials detection may occur after the last infection. %. The solid line represents the threshold at which vaccination of the under 15-year-old age group begins. When $R_0 = 6$, the 81% assumed reduction in R_{eff} after outbreak detection is insufficient to

guarantee elimination when vaccine coverage is less than 20%. Results are from 10,000 independently initialised realisations of the stochastic model.

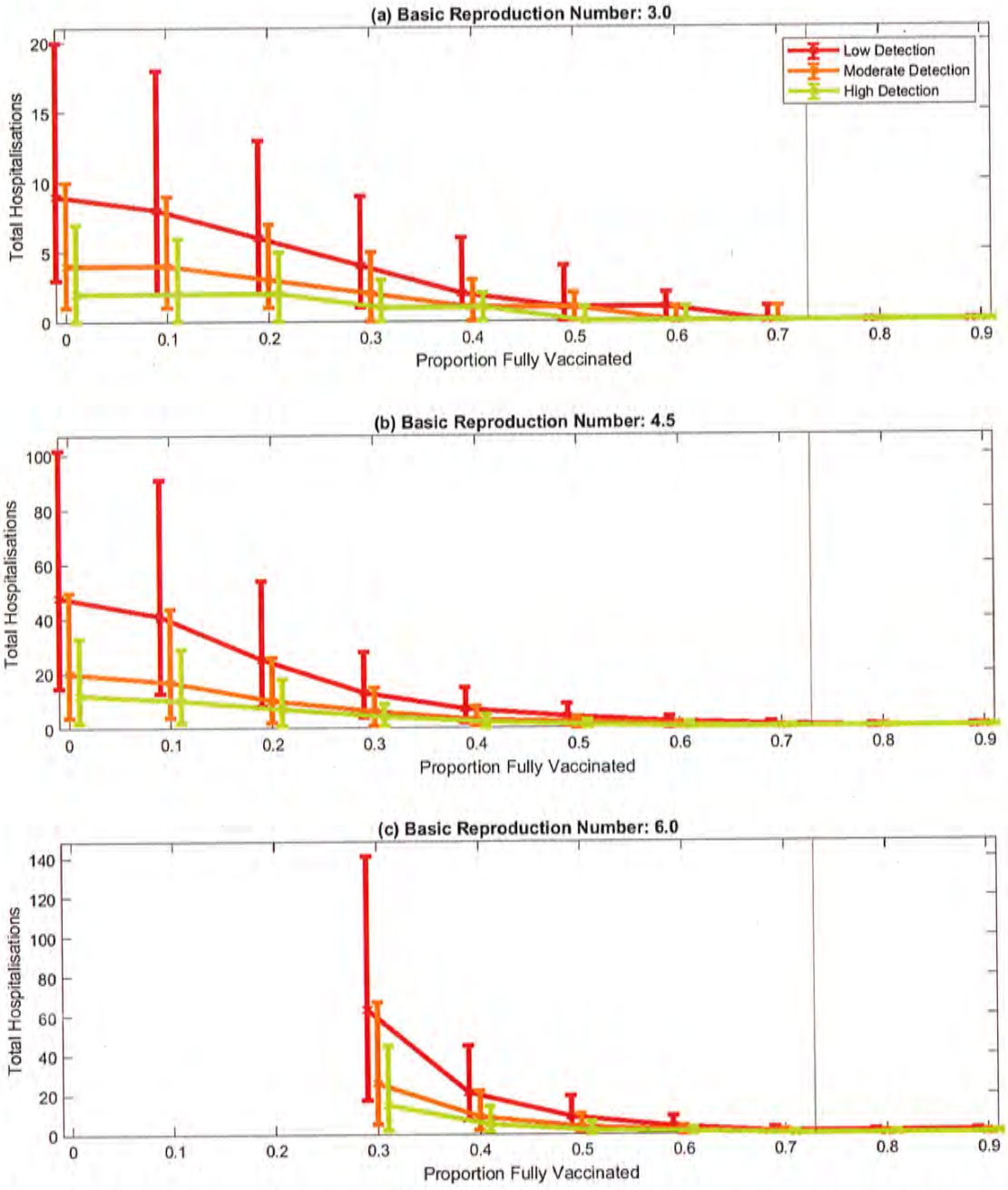

Figure 10. Total number of hospitalisations from a single-seed re-incursion, assuming the introduction of case isolation, contact tracing and population-level controls after the outbreak is detected, leading to an aggregate 81.4% reduction in R. Only outbreaks that are detected are included in these results. When $R_0 = 6$, the 81% assumed reduction in R_{eff} after outbreak

detection is insufficient to guarantee elimination when vaccine coverage is less than 20%. Results are from 10,000 independently initialised realisations of the stochastic model. Note the different vertical axes in the three panels.

Discussion

We have investigated an age-structured model for transmission of COVID-19 in a partially vaccinated population, parameterised to represent New Zealand's age-structure and age-specific contact rates. As of June 2021, in New Zealand the Pfizer/BioNTech vaccine is only approved for use in people aged over 16, although Medsafe has recently given regulatory approval for 12-15 year-olds. Our results show that, under baseline vaccine effectiveness assumptions and with $R_0 = 4.5$, vaccination of over 80% of the population will likely be necessary to reach the population immunity threshold, defined as a reproduction number that is less than 1 in the absence of other interventions. This would almost certainly require vaccination of at least some under 16 year olds. If $R_0 = 6$ (which could represent a highly transmissible SARS-CoV-2 variant such as Delta [13]), population immunity would require approximately 97% of the population, something which is unlikely to be achievable in practice.

The model also assumes that vaccine coverage is evenly spread throughout the population within each age group. In reality, we expect coverage to be heterogeneous. This means that, even if $R < 1$ at a national level, communities with relatively low vaccine coverage (or relatively high contact rates) could still have a local $R > 1$, which would expose them to major outbreaks. It will be important to pay attention to vaccine coverage in different geographic and socioeconomic groups to identify at-risk communities and prioritise them for vaccination.

Our results show that vaccination of the priority age group (over 65 years) at the beginning of the programme does not greatly reduce the potential for community transmission of the virus, but sharply reduces hospitalisations and fatalities by protecting the highest-risk groups in the model. However, there are other risk factors we have not accounted for here. Māori and Pacific peoples have previously been shown to be at greater risk of hospitalisation [20] and fatality [42] after accounting for age and reported comorbidities. Any strategy to minimise negative health outcomes from COVID-19 should prioritise these groups for vaccination. It will also be essential to ensure that Māori and Pacific communities can access the vaccine in a way that

meets their cultural, linguistic and social needs [43]. This includes removing costs and barriers, and ensuring that it is easy and convenient to access the vaccine. Once high-risk groups are vaccinated, extending high coverage levels to lower-risk groups including young people and, provided regulatory approval is obtained, children will be crucial to minimising the potential for transmission and reaching the population immunity threshold.

Whether or not New Zealand reaches the theoretical population immunity threshold, the higher vaccination coverage is, the more collective protection the population has against infections, hospitalisations and deaths from COVID-19. Conversely, even if population immunity is achieved, transmission can still occur. As a rule-of-thumb, for any $R < 1$, the average number of cases caused by a single re-introduction is given by $\frac{R}{1-R}$. If $R = 0.95$ for example, this means there will be an average of 19 community cases for every border re-introduction. This means that, regardless of the population immunity threshold, it will be essential to continue to vaccinate as many people as possible to minimise the combined potential for transmission and health impacts.

Our results also show that, until a high proportion of the population is vaccinated, the probability of case detection is still an important determinant of outbreak size. This demonstrates that high rates of community testing for surveillance purposes will remain crucial throughout the vaccine roll-out to minimise the risk of a large outbreak. Although the model ignores seasonal effects, this will be particularly important during the winter influenza season when the incidence of symptoms consistent with COVID-19 is relatively high and seasonality may increase the transmission potential of SARS-CoV-2 [22].

As vaccine coverage increases, the stringency and duration of population-level restrictions (e.g. via New Zealand's COVID-19 Alert Level system) required to eliminate an outbreak decreases. Under the baseline scenario with $R_0 = 4.5$, once approximately 60% of the population is fully vaccinated, the model estimates that it will be possible to eliminate any new outbreaks with case isolation and contact tracing alone. However, it is extremely unlikely that the contact tracing would perform at the level required if there were a large number of imported cases that triggered multiple concurrent outbreaks, which was the situation New Zealand faced in March 2020. In the medium term, a modest reduction in transmission could be achieved by baseline

measures such as mask use, widespread community testing, and isolation of symptomatic individuals. For example, if 70% of symptomatic cases are isolated (with 80% effectiveness) an average of 2 days after onset of symptoms, R is reduced by 11%. This would imply that effective population immunity could be achieved when vaccination reduces R to approximately 1.1. However, this will still require very high vaccine coverage and is subject to the caveats discussed above about low coverage and high contact groups remaining vulnerable.

Together these results suggest that, until we get close to the population immunity threshold, a major public health response that included significant interventions would still be required to control any resurgent outbreaks and prevent a major epidemic. Nonetheless, as vaccination coverage increases, it may be possible for the requirement for 14 day government-managed isolation period to be replaced with a combination of testing, shorter quarantine periods or home isolation for arrivals from low-risk countries. Future work will model the risk from different border policies and different traveller risk profiles. However, strong border restrictions designed to keep COVID-19 out of the community are likely to be required until very high vaccination coverage is achieved nationally.

We have investigated outcomes when the overall vaccine effectiveness against transmission is 70%, 85% and 95%. Published evidence shows the Pfizer-BioNTech mRNA vaccine is highly effective in preventing documented infection after two doses [9, 31, 44], thereby implying high effectiveness against transmission [10]. Despite this, there is still some uncertainty around the exact effectiveness of this vaccine in reducing transmission. Furthermore, the ongoing emergence of variants of concern that are potentially more transmissible [12] or less responsive to vaccines [35] could mean that, in future, R_0 is substantially higher or vaccine effectiveness is substantially lower than the values assumed in this paper [45]. Higher values of R_0 or lower values of vaccine effectiveness against transmission mean that higher vaccination coverage is required to obtain the same level of protection at a population level. Although the Pfizer vaccine has high effectiveness against the Delta variant after two doses, effectiveness after only one dose is significantly lower [13]. This means that administering the full course of two doses is essential to providing full immunity. Booster shots are being considered [46] and these may be required to reach the higher effectiveness scenario, or even the baseline scenario. Continued research into the pathogenicity, transmission characteristics, and responsiveness to vaccines of

emerging variants of SARS-CoV-2 is needed to evaluate their effect on population immunity levels and the risk they pose.

The model contains a number of simplifications, limitations, and sources of uncertainty. Firstly, it models transmission of SARS-CoV-2 at a national level and ignores geographic and socioeconomic heterogeneities that will affect transmission rates. It also does not consider differential transmission rates in specific settings (such as households, schools and workplaces). Secondly, there is still uncertainty about the intrinsic reproduction number in the absence of control and vaccine effectiveness, particularly for new and potential future variants of concern. Data is still being gathered and analysed on this internationally; model results will need to be updated as new data becomes available. Thirdly, the rates of contact between age groups are based on 2008 survey data from EU countries [21], mapped onto the current age structure of New Zealand's population. We have also assumed an age-dependent susceptibility profile based on modelled estimates [25], which are subject to uncertainty and are difficult to measure directly. If actual age-dependent contact rates or susceptibility have a significantly different structure than assumed, this would affect how cases are distributed across age groups, which would change model outputs for hospitalisations and deaths. Fourthly, the results describe model outcomes under specific assumptions about the interventions taken in response to cases. These are either counterfactuals modelling a completely unmitigated epidemic, or elimination scenarios that assume strong and effective interventions are implemented for as long as is needed to eliminate an outbreak. We do not attempt to predict the outcomes of other control responses or behavioural factors that may change the effectiveness of interventions such as contact tracing, quarantine or prolonged restrictions. Finally, the model also ignores the effects of seasonality, waning immunity and population dynamics (births, deaths, ageing and migration). These factors will introduce additional challenges in achieving and maintaining high levels of population immunity.

Until the vaccine rollout is complete, retaining the elimination strategy has two key benefits. Firstly, it protects people who have not yet been offered the vaccine and the minority of people for whom the vaccine is less effective, or who cannot be vaccinated for medical reasons. Secondly, by keeping cases to a minimum, it maximises the ability of the contact tracing system to reduce transmission. This decreases the likelihood of needing to use the alert level system

to control future outbreaks. Nonetheless this work provides insight into some of the opportunities to change policy settings that may arise as New Zealand's vaccination programme rolls out.

Acknowledgements

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