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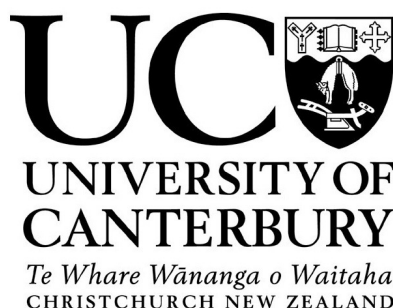
Opening the Borders With a Limited Vaccine

Modelling a Waning Immunity Vaccine for COVID-19 in New Zealand

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Executive Summary

We investigate the introduction of a vaccine to New Zealand that provides a limited immunity to COVID-19 of only 6 months and ask if such a vaccine would allow us to open our borders. Running 2-year *SIRV* simulations with the sporadic introduction of cases over the border, we find that both a constant and periodic vaccination strategy vaccinating up to 80% in the first year only reduce the total proportion of the population that contract the virus from 73.2% in a no vaccine scenario to $\sim 40\%$. The proportion of the population who need to contract the virus to obtain herd immunity is only reduced from 32.7% to $\sim 20\%$. And, the death rate is reduced from 1.0% to $\sim 0.5\%$. Therefore, it is our recommendation that under this scenario New Zealand does not initiate a plan to open the borders and instead focuses on developing methods for economic self-sufficiency.



MATH363 ASSIGNMENT 2

1 Introduction

As the advent of a successful vaccine for COVID-19 approaches, it is important for policy makers to anticipate and plan for non-permanent immunisation. Should a vaccine only provide only short term immunity, will it be economically and logistically feasible for New Zealand to open its borders?

To some extent, the impending introduction of a vaccine to COVID-19 has been hailed as a silver bullet that will return to the world to normalcy, and although it may be true that a vaccine will likely slow the progression of COVID-19, it will likely be no panacea. Almost all vaccines have a waning immunity. For most illnesses for which there is a vaccine available, immunity lasts between 10 and 20 years (Mumps, Polio, Rubella, etc.) [1]. But for some illnesses, the immune response triggered by a vaccine can die off in just a few years. Although it may be too soon to draw any firm conclusions, there has been evidence to suggest that immunity to COVID-19 may be similarly transient [2]. Therefore, we should, at the very least, prepare for the possibility that the silver bullet of long-term immunity may not come, at least for some time.

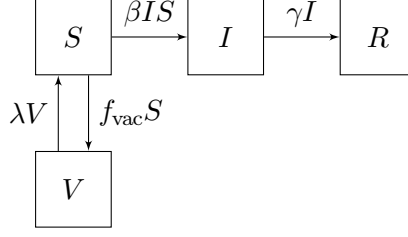
Opening New Zealand's borders, even partially, means accepting the introduction of COVID-19 into our communities. Although proper protections at the border will screen out most cases, some will inevitably slip through. If we decide to open our borders, which is a complex decision in its own right, would a vaccine providing only limited immunity allow us to remain for the most part unscathed, or would the cost to health, the economy, or both be too much to bear? Furthermore, we have seen that already marginalised communities are most adversely affected by COVID-19 [3] and so we should take extreme caution in recommending any strategy at the border.

We will look at a so-called *worst-case* vaccine, one that provides only 6 months of lasting immunity, and how different vaccination distribution strategies employing this vaccine deal with a constant, stochastic introduction of clusters across the border.

2 Model Description

We used the homogeneous compartmental SIR model as our base model. This means the New Zealand population is divided into a number of compartments and then move between those compartments at different rates. It is called homogeneous because it assumes perfect mixing of individuals in the population. The first compartment is *susceptible* and is where everyone starts out, the second is *infected*. People move from the susceptible compartment into the infected compartment at a rate proportional to the number of people already infected. The third is *removed* and is where people go after they've had the virus and have either recovered or passed away. We have added a fourth compartment *vaccinated*. People move into this compartment at a rate that we will specify, corresponding to a particular immunisation strategy.

We will investigate 2 immunisation strategies, a constant rate of vaccination and a 6-month periodic vaccine drive where we ramp up immunisations for 6 months in the year and scale back the other 6 months. Both strategies are normalised so that 80% of the population has been vaccinated by the end of the first year. We will also include a control with no vaccines. In all cases, vaccine-induced immunity only lasts 6 months after which people will be sent back into the susceptible category. Summarised in one picture our model looks like this:



To run the model, we start with everyone in the *susceptible* category and then set time moving. At random intervals of around 5 days, we stop time, introduce an infected cluster over the border by perturbing the number of *infected* cases very slightly (introducing an infectious person), and then start time again. This represents an open or partially-open border scenario.

Our model should represent the real world, and in the real world the rate of infection β and the rate of recovery/death γ would not remain constant, they would fluctuate from day to day due to innumerable compounding factors. Therefore, to ensure our models are robust, we let these parameters vary with the addition of Brownian noise. This means our parameters will fluctuate up and down slightly over time to represent the uncertainty in these rates.

2.1 Parameters

An important aspect to modelling the spread of COVID-19 is the parameter values used in the model. A value of $R_0 = 1.8$ was chosen for the basic reproductive number, the number of people an infected person will infect on average when total infections are low.¹ This was in accordance with a report from Te Pūnaha Matatini [4]. A value of 10 days was chosen as the average length of time that an infected person remains infectious for so that $\gamma = 1/10 \text{ days}^{-1}$. This was in accordance with research from Hu, Z., et al. [5], Liu, Y., et al. [6], and the CDC [7]. Then our rate of infection β is $\gamma * R_0 = 18 \text{ days}^{-1}$. The modelled cost of each vaccine was chosen to be \$30 (NZD). This was a "best guess" estimate, based on the cost of other comparable vaccines available today, and the fact that the US recently signed a \$2 billion (USD) contract with Pfitzer to provide 100 million vaccines. This is expected to set the baseline price for the COVID-19 vaccine internationally. Being an unprecedented event, and given that it is desirable for New Zealand to return to normalcy as soon as possible, a rate of vaccination in both the constant and the periodic immunisation strategies was chosen such that 80% of the population is vaccinated by the end of first year. Although this may be unrealistic, we consider this a *best-case* scenario. Finally the rate of lost immunity for our vaccine λ is just $1/(6 \text{ months})$.

3 Results

Running our simulations for our 3 vaccination options gives us the time evolution of the number of people in each of our 4 categories over a period of 2 years. Here are the results side-by-side:

¹The distinction between the basic and effective reproductive numbers R_0 and R_{eff} is that the first is the number of people someone infects *on average* in a fully susceptible population, while R_{eff} is the number of people someone infects *on average* accounting for the immunity of those who have already contracted the virus. As less than 0.03% of the New Zealand population have been confirmed to be infected at the time of writing, our population is essentially fully susceptible, meaning $R_0 \approx R_{\text{eff}}$.

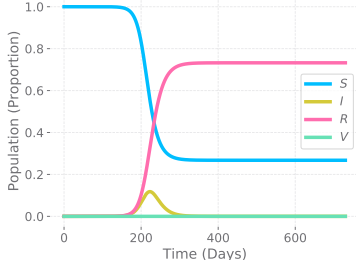


Fig 1: No vaccinations

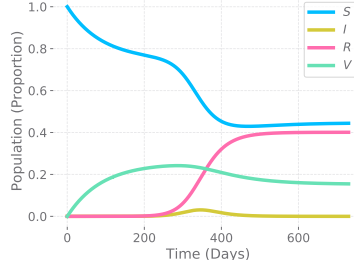


Fig 2: Constant vaccination

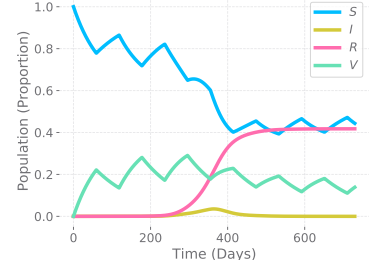


Fig 3: Periodic vaccination

At a first glance, we can see that vaccination in either form has a reasonable impact on the total number who contract the virus but a periodic strategy appears to have no benefit over a constant rate of vaccination. We can also see that vaccination in either form has the effect of '*flattening the curve*', extending and lowering the day of peak infection. With no vaccination, we reach peak infection on day 224 at 11.8%, 73.2% of the population contract the virus in the long term, and herd immunity, the proportion of the population with immunity required to halt the virus, is 32.7%. With constant vaccination, we reach peak infection on day 345 at 3.1%, 40.1% of the population contract the virus in the long term and herd immunity is 18.2%. And, with 6-month periodic vaccination, we reach peak infection on day 365 at 3.6%, 41.8% of the population contract the virus in the long-term and herd immunity is 20.9%.

The amount of vaccination we modelled was chosen to represent a physically feasible number of vaccinations able to be performed in New Zealand per year as this, rather than cost, will likely be the limiting factor. In both vaccination scenarios, spending was at its highest in the first year when the virus hit its peak with \$121.02 million and \$121.11 million being spent in the constant and periodic cases respectively, at \$30 per vaccine per person. This is well accounted for by the \$160 million increase to the Combined Pharmaceutical Budget [8] provided by the NZ government that will be used to cover the expense of the vaccines. In both cases, spending reduces by about 50% in the following year as vaccinations come into equilibrium with susceptibles. We can plot the cumulative vaccination spending against time to get a better idea of cost.

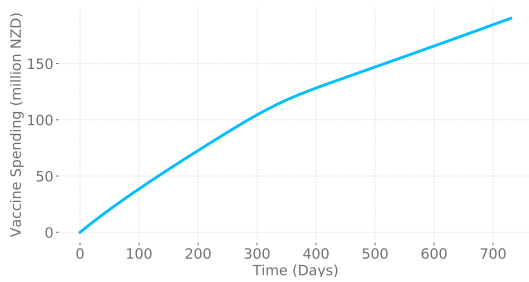


Fig 4: Constant vaccination cumulative spending

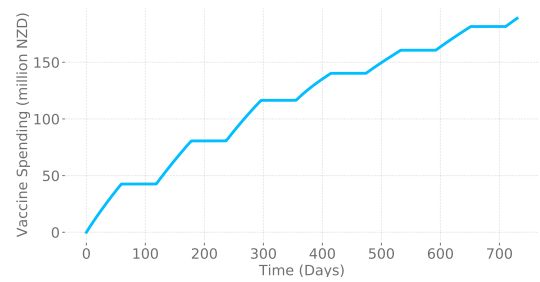


Fig 5: Periodic vaccination cumulative spending

So we see that after the first year, vaccination spending comes into equilibrium with loss of immunity.

4 Discussion

Our simulations tell us that while a vaccination of limited immunity may make a fairly significant difference to the rate of infection and overall number infected, the effect is nowhere near good enough to justify opening the borders regardless of whether a periodic or constant immunisation strategy is employed. Vaccination in either

form only reduces the total number infected from 73.2% to $\sim 40\%$ even with an aggressive vaccine quota.

With or without vaccination, New Zealand’s health system would not be prepared to deal with this many cases. It is estimated that on average 1 in 20 of those who contract the virus will become critical cases and require intensive care [9]. As New Zealand’s ICU capacity is 600 beds in a best case scenario, at this critical case rate we would blow past our hospital capacity at 12,000 cases. With a death rate of 1.4% [10] we would have 1.0% of the population dead with no vaccination or 0.5% with either of the vaccination strategies.

We should also take into account the fact that the *SIR* model is a homogeneous model and does not consider spatial variations in infection rate which is one way in which inequity manifests. We have already seen that the price paid during this pandemic is much higher for Māori, Pasifika, and other marginalised communities [3] and so accepting a death rate of 0.5% overall means accepting a much higher death rate for these communities. We have also assumed a vaccine effectiveness of 100% and so in reality the effects of a partial reopening under the scenario of limited immunisation may be even more bleak.

For that reason, it is our recommendation that in such a scenario, the government should not initiate an open or partially-open border plan. Especially as we have yet to confirm the long term effects of the virus, wilfully allowing $\sim 40\%$ of the population to contract the virus is likely not worth the short-term economic benefits of opening the border.

We suggest that it would be beneficial for New Zealand to explore methods for economic self-sufficiency such as boosting local tourism and initiating industry training programmes so that we may prepare ourselves for the prospect of further outbreaks or entirely new pandemics.

References

- [1] S. Plotkin, W. Orenstein, P. Offit, and K. Edwards, *Plotkin’s vaccines*, Vaccines (Plotkin) (Elsevier, 2017).
- [2] K. K.-W. To, I. F.-N. Hung, J. D. Ip, et al., “Covid-19 re-infection by a phylogenetically distinct sars-coronavirus-2 strain confirmed by whole genome sequencing”, *Clinical Infectious Diseases*, ciaa1275 (2020).
- [3] N. Steyn, R. N. Binny, K. Hannah, et al., “Estimated inequities in covid-19 infection fatality rates by ethnicity for aotearoa new zealand”, *medRxiv* (2020).
- [4] R. N. Binny, A. Lustig, A. Brower, et al., “Effective reproduction number for covid-19 in aotearoa new zealand”, *medRxiv* (2020).
- [5] Z. Hu, C. Song, C. Xu, et al., “Clinical characteristics of 24 asymptomatic infections with covid-19 screened among close contacts in nanjing, china”, *Science China Life Sciences* **63**, 706–711 (2020).
- [6] Y. Liu, L.-M. Yan, L. Wan, et al., “Viral dynamics in mild and severe cases of covid-19”, *The Lancet Infectious Diseases* (2020).
- [7] R. Wölfel, V. M. Corman, W. Guggemos, et al., “Virological assessment of hospitalized patients with covid-2019”, *Nature* **581**, 465–469 (2020).
- [8] N. Z. Treasury, “Summary of initiatives in the covid-19 response and recovery fund (crrf) foundational package”, 18–19 (2020).
- [9] Z. Wu and J. M. McGoogan, “Characteristics of and Important Lessons From the Coronavirus Disease 2019 (COVID-19) Outbreak in China: Summary of a Report of 72,314 Cases From the Chinese Center for Disease Control and Prevention”, *JAMA* **323**, 1239–1242 (2020).
- [10] J. T. Wu, K. Leung, M. Bushman, et al., “Estimating clinical severity of covid-19 from the transmission dynamics in wuhan, china”, *Nature Medicine* **26**, 506–510 (2020).

Appendix A: Model Details

We employed a standard compartmental homogeneous SIR model to which we added a compartment V to represent vaccinated individuals. Individuals are moved from susceptible to vaccinated at a rate proportional

to a distribution function of our choosing and moved back from vaccinated to susceptible after an average immune period of 6 months. We also add a compartment T to keep track of the total individuals vaccinated. Therefore, our modified SIR model becomes:

$$\begin{aligned}\dot{S} &= -\hat{\beta}(t)IS - f_{\text{vac}}(t)S + \lambda V \\ \dot{I} &= \hat{\beta}(t)IS - \hat{\gamma}(t)I \\ \dot{R} &= \hat{\gamma}(t)I \\ \dot{V} &= f_{\text{vac}}(t)S - \lambda V \\ \dot{T} &= P_0 f_{\text{vac}}(t)S\end{aligned}$$

where f_{vac} is our vaccination distribution function, λ is our rate of lost immunity, P_0 is the population size, and $\hat{\beta}(t)$ and $\hat{\gamma}(t)$ are the standard rate of infection and rate of recovery as stated in Parameters above, but with introduced Brownian noise to model the stochastic nature of these parameters. i.e.

$$\hat{\beta}(t) = \beta B_a(t) \quad \hat{\gamma}(t) = \gamma B_b(t)$$

where $B(t)$ is Brownian noise about 1. This set of ODEs was numerically solved over a time period of 2 years. However, to model the stochastic introduction of new cases, the integrator was stopped at random intervals, the number of infected individuals was perturbed very slightly (adding 1 infected individual), the other compartments were renormalised, and the integrator was restarted from that point. Therefore, we could use initial conditions, $S=1$, $I=0$, $R=0$, $V=0$.

Vaccination Distribution

We modelled 2 vaccination distributions both normalised to vaccinate 80% of the population in the first year and a control simulation with no vaccination. Both vaccination distributions have a dependence on S which has the effect of scaling the vaccination response according to the number of susceptible people (it also creates a fixed point which means our population can't become negative). The first vaccination response is constant with respect to time and so represents a strategy where we keep a steady rate of vaccination at all times (adjusting for number of susceptibles). The second response is a periodic response with a square wave f_{vac} oscillating off and on with a period of 6 months. This models periodic high intensity vaccine drives interspersed with periods of no vaccination. So our two f_{vac} look like:

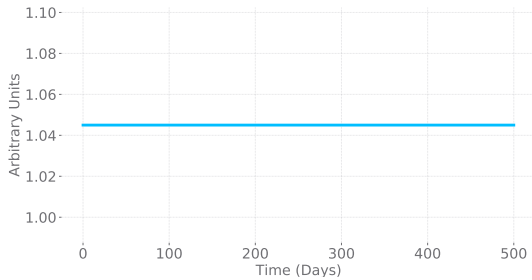


Fig 6: Constant f_{vac}

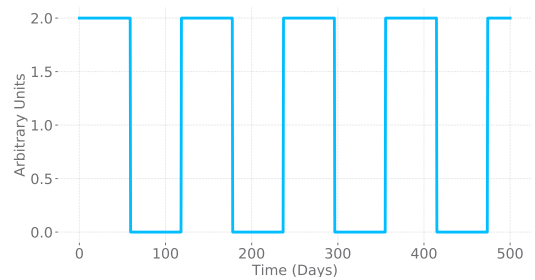


Fig 7: Periodic f_{vac}