

Potential Health Impacts from the COVID-19 Pandemic for New Zealand if Eradication Fails: Report to the NZ Ministry of Health

Prepared for the Ministry of Health

by

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23 March 2020



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<https://www.otago.ac.nz/wellington/departments/publichealth/research/heiru/index.html>
<https://www.otago.ac.nz/wellington/departments/publichealth/research/bode3/index.html>

NOTE

Although this report was correct at the time of writing, the information it presents may no longer be current because of continuing evolution of the COVID-19 pandemic and our understanding of it.

Unless otherwise indicated, peer review and full consultation with relevant agencies was not always possible in the timeframe available for producing this report.

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Abstract

Aims: While New Zealand may hopefully succeed with its current eradication strategy for COVID-19, this modelling aimed to consider the health outcomes if this strategy fails.

Methods: A SEIR model designed for COVID-19 (CovidSIM) was adapted and utilised. It was populated with NZ demographic data and relevant parameters sourced from the international literature. Different scenarios varied the basic reproduction number (R_0) and levels of disease control, and involved threshold analyses for controls to push the epidemic peak into the next year (ie, a point where a vaccine might become available).

Results: We found that it would be relatively easy to push some epidemic curves into the subsequent year (ie, for $R_0 = 1.5$). This would be harder for the more plausible $R_0 = 2.5$ scenarios, but still potentially feasible eg, via over 41% of “general contact reduction” for nine months, combined with over 50% of cases having hospital isolation for nine months.

When considering the two highest levels of R_0 (2.5 and 3.5), and the two levels of “general contact reduction” (at 25% for six months and 50% for nine months), the estimated health impacts were: 44% to 64% of the population sick; 18% to 26% seeking a medical consultation (including Healthline and internet consultations); 22,200 to 32,000 people needing to be hospitalised; 5,540 to 8,000 people needing critical care (in an intensive care unit [ICU]); 2,770 to 4000 requiring ventilators; and 8560 to 14,400 dying (0.17% to 0.29% of the population). On the worst day for the worst scenario ($R_0 = 3.5$, 25% contact reduction), there would be 11,200 people needing to be hospitalised and 2,800 needing to be admitted to critical care. For this scenario ICU capacity would be full on day 92, at a very early point in the epidemic curve. Such demands are unprecedented in New Zealand’s history and unless there was a major reconfiguration of services, would be overwhelming, with a risk of this pushing up the case fatality rate. The mortality burden would almost certainly be much higher in older age-groups (eg, 89% of the deaths estimated to be in the 60+ age-group), and also would probably be patterned by ethnicity (higher for Māori and Pacific peoples), and for people living in deprived areas.

Conclusions: If New Zealand fails with its current eradication strategy toward COVID-19, then health outcomes for New Zealand could be very severe. If interventions were intense enough however, in some scenarios the epidemic peak could still be suppressed or pushed out to the following year (at which time a vaccine may be available). Due to the high levels of uncertainty with some of the parameters used in this modelling work, it should be regularly repeated as new information on the epidemiological characteristics of COVID-19 become available.

Introduction

There is pandemic spread of the new coronavirus “SARS-Cov-2”, causing the disease “COVID-19”, with the World Health Organization (WHO) reporting over 200,000 cases and over 8000 deaths on 19 March 2020 [1]. One approach to informing the potential health burden and relevant control measures for a new pandemic is to study its dynamics using mathematical models. Recently published mathematical modelling work on COVID-19 has reported that “in most scenarios, highly effective contact tracing and case isolation is enough to control a new outbreak of COVID-19 within 3 months” [2]. Another modelling study found that “combining all four interventions (social distancing of the entire population, case isolation, household quarantine and school and university closure) is predicted to have the largest impact, short of a complete lockdown which additionally prevents people going to work” [3]. Other such models have been used to estimate the impact of disease control measures in China [4, 5].

Given this background, we explore the potential health impact of the spread of the COVID-19 pandemic in the New Zealand, particularly if the Government’s current strategy of eradication of COVID-19 fails.

Methods

In this modelling, we took the standard approach of using a deterministic SEIR model ie, key compartments for: susceptible [S], exposed [E], infected [I], and recovered/removed [R]. This model was developed specifically for COVID-19 by our German collaborators (see Acknowledgements), with various adaptations suggested by the New Zealand authors. This model is freely available online with a dashboard display to facilitate user interaction (<http://covidsim.eu>; version 1.0, 19 March). For additional quality tests we subjected the CovidSIM model to extreme value testing and conducted a head-to-head comparison with a completely independently developed SEIR model produced by Australian colleagues (McVernon et al, University of Melbourne). The comparison results (when accounting for minor differences in model structure) were very similar and gave us additional confidence in the quality of the CovidSIM model.

The Appendix details the parameters, derived variables and differential equations used in the CovidSIM model. Table A1 in the Appendix also provides the input parameters used in the modelling, as based on available publications and best estimates used in the modelling work on COVID-19 to date (as known to us on 21 March 2020).

Results

It is to be hoped that New Zealand can succeed with its current eradication strategy. But this modelling consider the potential outcomes if this strategy fails.

Baseline and threshold analyses: To provide a baseline, Figure 1 shows the epidemic curves for three values of the reproduction number (R_0) – all with the unrealistic scenario of no changes in normal behaviour in response to the pandemic. In an attempt to push the peaks of these epidemic curves into the subsequent year (when a vaccine might become available), we adjusted various interventions (Table 1). The results suggest that pushing the peak of the epidemic into the next year, when assuming a low basic reproduction number (R_0) of only 1.5, was achievable with “general contact reduction” at levels of over 16% to over 21% (for nine and six month time periods

respectively) (Table 1). Similarly, it was achievable for probabilities of isolating symptomatic cases in hospital in the range of over 30% to over 35% (Table 1). Using a possibly more realistic value of 2.5 for R_0 , the most achievable way of shifting the epidemic peak into the next year was if both contact reduction and case isolation were used (at levels of over 41% and over 50% respectively for nine months). The equivalent values for when $R_0 = 3.5$, was over 64% and over 50% for nine months.

Figure 2 shows the impact of “25% general contact reduction” for a six-month intervention period. The epidemic curves for the $R_0 = 3.5$ and $R_0 = 2.5$ epidemic scenarios still occurred in the intervention period, albeit partly suppressed compared to the baseline in Figure 1. But the $R_0 = 1.5$ epidemic scenario was very largely suppressed with no peak.

Figure 3 shows the impact of “50% general contact reduction” for a nine-month period. The epidemic curve for the $R_0 = 3.5$ epidemic scenario still occurred in the intervention period, albeit partly suppressed compared to the baseline in Figure 1. But the $R_0 = 2.5$ epidemic scenario was suppressed until the intervention period ended and then the epidemic accelerated again, peaking at the end of the year. Similar to the results in Figure 2, the $R_0 = 1.5$ epidemic was almost extinguished.

Health impacts: When considering the two highest levels of R_0 , and the two levels of “general contact reduction” (at 25% for six months and 50% for nine months), the estimated ranges for health impacts were: 44% to 64% of the population sick; 18% to 26% seeking a medical consultation (including Healthline and internet consultations); 22,200 to 32,000 people needing to be hospitalised; 5,540 to 8,000 people needing critical care (in an ICU); 2,770 to 4000 requiring ventilators; and 8560 to 14,400 dying (0.17% to 0.29% of the population) (Table 2).

In terms of timing, on the worst day for the worst scenario studied ($R_0 = 3.5$, 25% contact reduction), there would be 11,200 people needing to be hospitalised and 2,800 needing to be admitted to critical care. In this particular scenario, New Zealand’s 221 ICU beds would be filled up with COVID-19 patients on day 92, at a very early point of the epidemic curve (Table 2, Figure 2). If ICU capacity was doubled, then all these additional beds would be filled up with COVID-19 patients just six days later (day 98).

Age distribution of health impacts: Based on the age distribution data from China [6], the hospitalisations and deaths from COVID-19 are known to particularly effect older age-groups. But using New Zealand population distribution data and age-specific case fatality estimates for a more similar country (UK, [3]), we generated the results shown in Figure 4. This suggested that 88.9% of the deaths would occur in the 60+ age group. Indeed, nearly a third (32.9) occurred in the 80+ age group.

Ethnic and socio-economic distribution of health impacts: There is no robust basis for estimating impacts by ethnic group in New Zealand from international COVID-19 data. But we note that in the 2009 Influenza A(H1N1) pandemic in New Zealand, the risk of hospitalisation was five times higher for Māori and seven times higher for Pacific peoples than for New Zealand European/Other [7]. Similarly, the risk of death was 2.6 times higher for Māori (95%CI: 1.3 – 5.3) than for NZ European/Other [8]. Indeed, there is evidence for relatively higher Māori mortality in both the 1957 and 1918 influenza pandemics as well [8].

Furthermore, there was also some evidence of a socioeconomic gradient in the 2009 influenza pandemic with 39% of those dying having an area deprivation score of either 9 or 10 (the most

deprived two deciles), compared with the expected 20% of the population. Of those dying, 86% had at least one comorbid or associated condition [8].

Seasonality impacts: Figure 5 shows that increased seasonal variation in the R_0 value resulted in winter acceleration of the epidemic and a higher peak to the epidemic curve. However, as discussed in the parameter table (Table A1) there is substantial uncertainty about the role of seasonality in the epidemiology of COVID-19.

Table 1: Threshold analyses for pushing the peak of the spread of the COVID-19 pandemic in New Zealand into the next year if the current eradication strategy fails (ie, pushing the peak to after day 365 of the simulation with the start of the simulation on 1 April 2020, the date we assumed that uncontrolled spread began; see Table A1 for input parameters)

Intervention settings	Assumed basic reproduction number (with $R_0=2.5$ being the most plausible value; Table A1)		
	$R_0=1.5$	$R_0=2.5$	$R_0=3.5$
<i>Intensity and length of “general contact reduction” starting on day 1 of the simulation</i>			
Level of general contact reduction for 6 months needed to push the epidemic into the next year	>21%	Not possible	Not possible
– for 9 month intervention period (274 days)	>16%	>53%	>71%
<i>Proportion of symptomatic cases in hospital isolation (with home isolation at 50% effectiveness when hospital capacity is exceeded; beginning on day 1 of the simulation)</i>			
Probability of case isolation in hospital needed to push the epidemic into the next year (for a 6 month intervention period)	>35%	Not possible	Not possible
– for 9 month intervention period (274 days)	>30%	>91%	Not possible
<i>Intensity and length of “general contact reduction” in the context of 50% of symptomatic cases being identified and having hospital isolation for a six month period (with home isolation at 50% effectiveness when hospital capacity is exceeded)</i>			
Level of general contact reduction for 6 months needed to push the epidemic into the next year	$\geq 0\%$	Not possible	Not possible
– for 9 month intervention period (274 days)	$\geq 0\%$	>41%	>64%

Table 2: Potential health impacts of the spread of the COVID-19 pandemic in New Zealand if the current eradication strategy fails (for a range of basic reproduction number (R_0) values and differing intensity of “general contact reduction” as the control measure; see Table A1 for input parameters)

Key results	$R_0=1.5$		$R_0=2.5$ (the most plausible value)		$R_0=3.5$	
	25% control for 6 months*	50% control for 9 months*	25% control for 6 months	50% control for 9 months*	25% control for 6 months	50% control for 9 months
General pattern seen for symptomatic cases	Highly suppressed, peak in next year	Highly suppressed, peak in next year	Peak in intervention period	Peak after intervention period (truncated)	Peak in intervention period	Peak in intervention period
Symptomatic cases (which are 67% of all infected cases)						
Total	617,000	1,770	2,830,000	2,220,000	3,200,000	2,620,000
Proportion of population (%)**	12.3%	0.04%	56.6%	44.3%	64.0%	52.4%
Peak week for incidence	Next year	Next year	23	50	17	24
Peak month for incidence	Next year	Next year	5	12	4	6
Number of sick people on the worst day of the simulated year	57,800	110	660,000	375,000	1,120,000	550,000
Proportion of population sick on the worst day (%)**	1.2%	0.0%	13.2%	7.5%	22.4%	11.0%
Consultations (40% of symptomatic cases seek consultations, possibly mainly telephone/internet)						
Total	247,000	706	1,132,000	887,000	1,280,000	1,050,000
Proportion of population (%)**	4.9%	0.0%	22.6%	17.7%	25.6%	21.0%
Severe cases likely to require hospitalisation (1.0% of symptomatic cases)						
Total	6,170	18	28,300	22,200	32,000	26,200
Proportion of population (%)**	0.12%	0.00%	0.57%	0.44%	0.64%	0.52%
Number of people in hospital on the worst day (if capacity existed)	578	223	6,600	3,750	11,200	5,500
Proportion of population in hospital on the worst day (%)**	0.01%	<0.01%	0.13%	0.07%	0.22%	0.11%
Cases likely to require ICU (25% of hospitalised cases)						
Total	1,540	4	7,070	5,540	8,000	6,550
People in ICU on the peak day (if capacities exist)	145	2	1,650	937	2,800	1,380
Day when the current 221 ICU beds are all full	Not reached	Not reached	123	305	92	132
As above, but assuming bed capacity is doubled to 442	Not reached	Not reached	131	321	98	142
Cases likely to require ventilation in ICU (50% of those in ICU)						
Total**	771	2	3,540	2,770	4,000	3,280
Deaths (case fatality risk amongst symptomatic cases of 0.45%)						
Total	2,520	7	12,700	8,560	14,400	11,800
Proportion of population (%)**	0.05%	<0.01%	0.25%	0.17%	0.29%	0.24%

* Results are shaded as they were right truncated (ie, only the results for the first 365 days of the simulation are reported) as the epidemic peak was pushed into the following year.

** Results in these rows were not standard outputs for the CovidSIM model but were based on further Excel-based calculations from the CovidSIM output.

All numbers are rounded to three meaningful digits.

Figure 1: Epidemic curves for the uncontrolled spread of the COVID-19 pandemic in New Zealand for three different reproduction numbers (R_0) and with no changes in human behaviour or interventions assumed (ie, which is unrealistic but is shown here to demonstrate uncontrolled epidemic patterns)

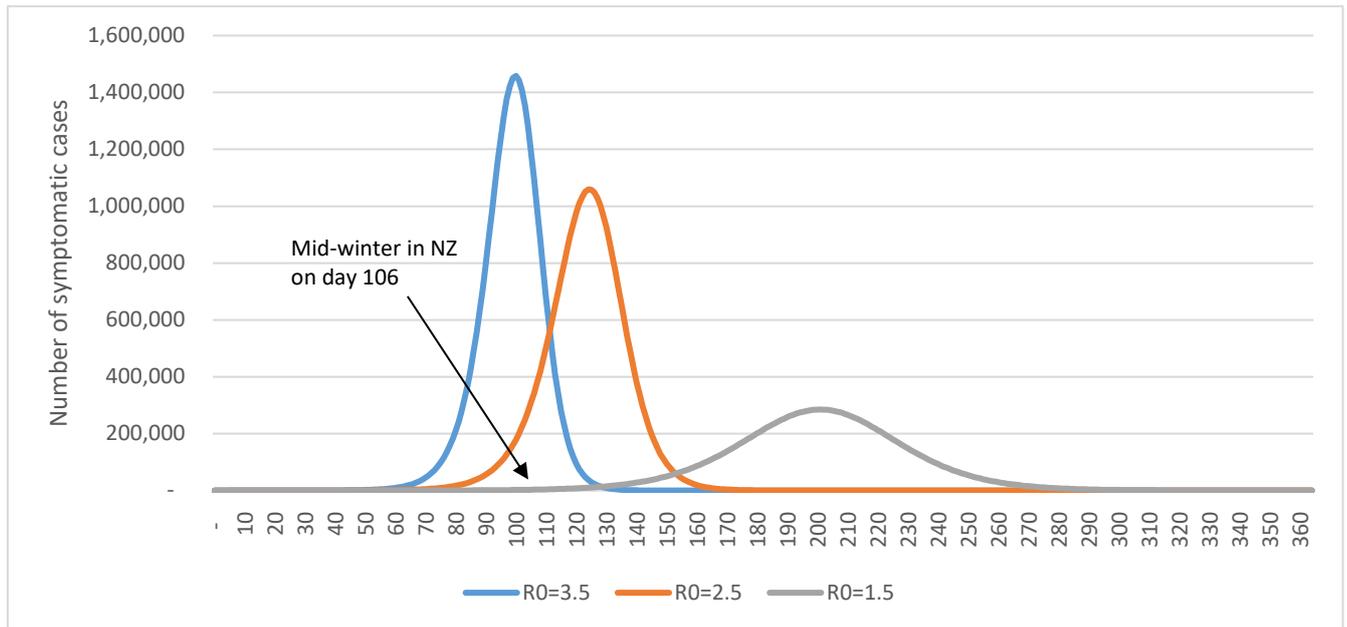


Figure 2: Epidemic curves for the spread of the COVID-19 pandemic in New Zealand for three different reproduction numbers and at 25% “general contact reduction” intervention for six months (albeit the epidemic curve for $R_0 = 1.5$ is largely suppressed)

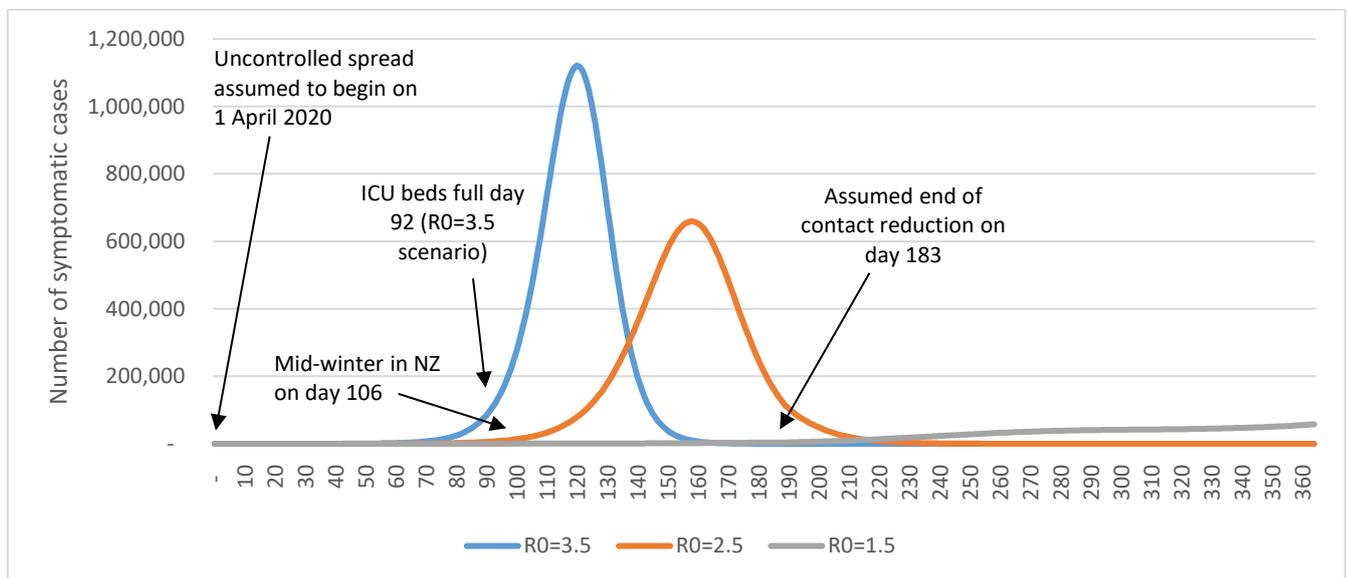


Figure 3: Epidemic curves for the spread of the COVID-19 pandemic in New Zealand for three different reproduction numbers and with the 50% “general contact reduction” intervention for nine months (albeit the epidemic curve for $R_0 = 1.5$ is nearly completely suppressed)

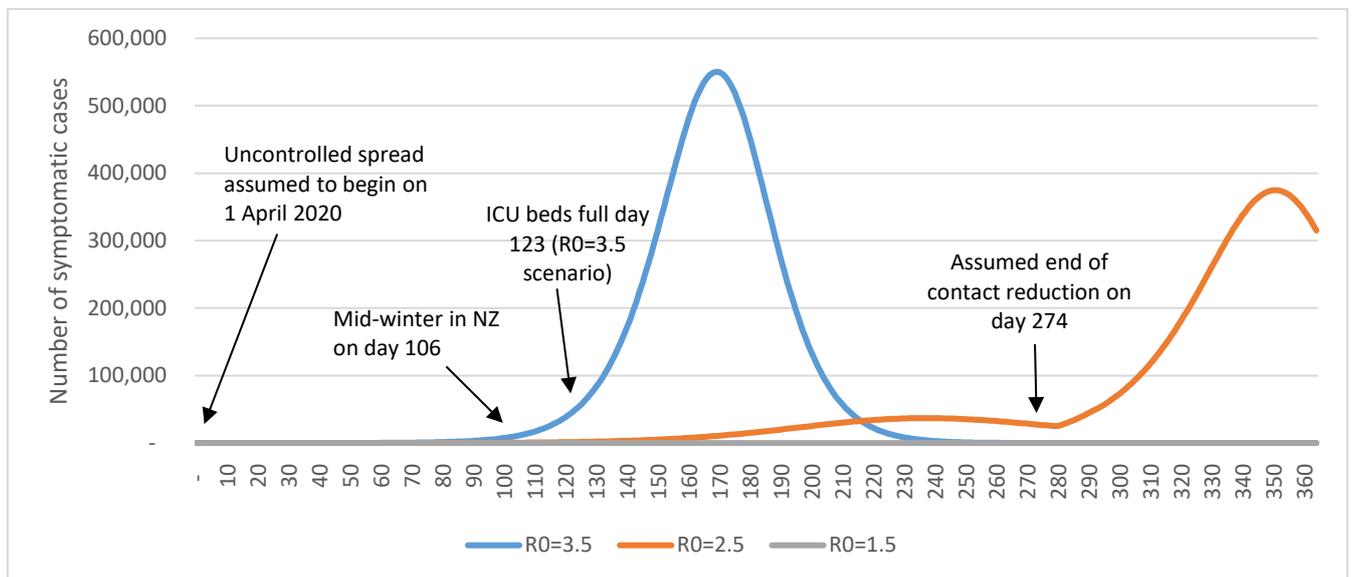


Figure 4: Distribution of deaths by age-group for the $R_0 = 2.5$ epidemic scenario (with 25% “general contact reduction” for six months; using NZ population age structure from the 2018 Census and age-specific case-fatality rates as per Imperial College modelling [3])

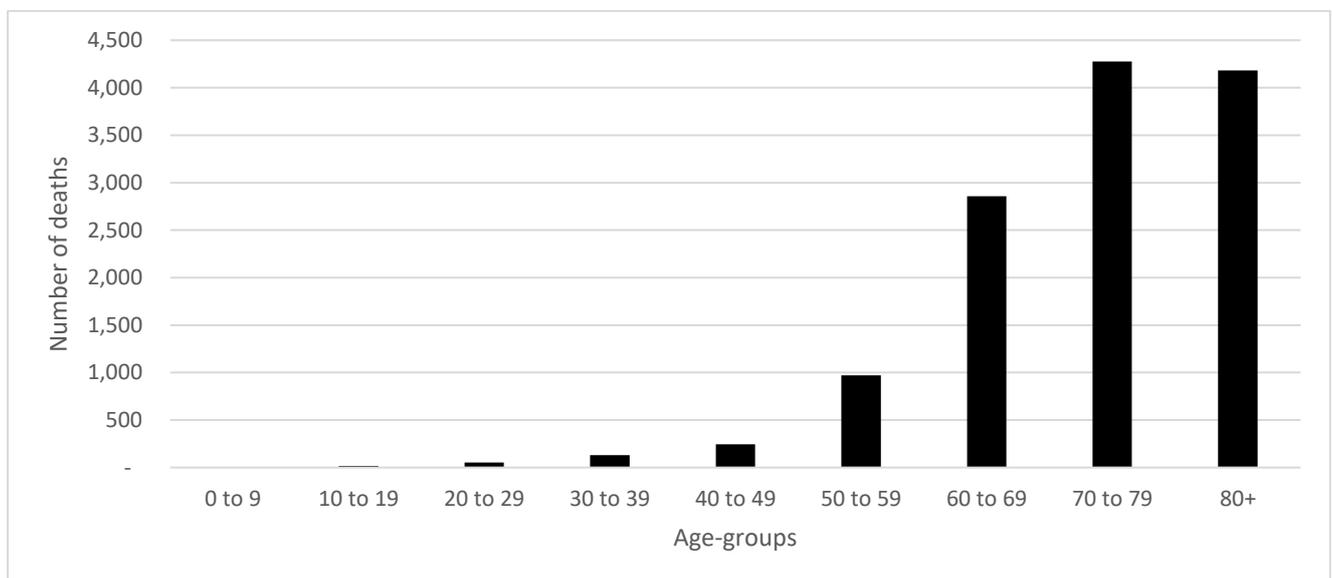
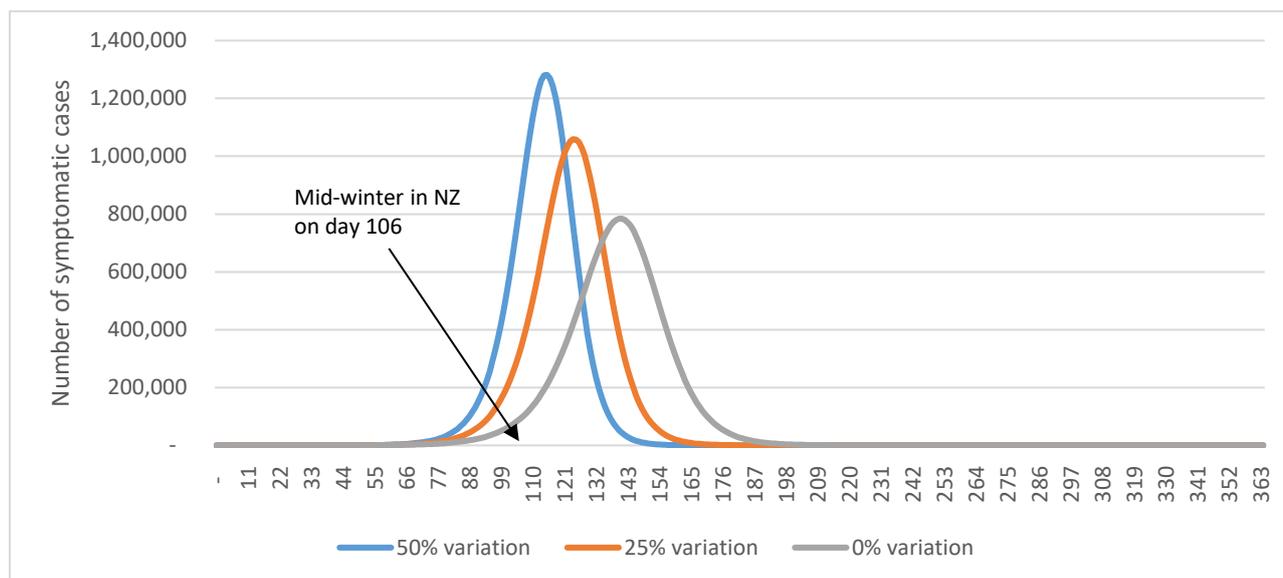


Figure 5: Impact of seasonality via sinusoidal variation throughout the year of the R_0 value for the $R_0 = 2.5$ epidemic scenario (with 25% “general contact reduction” continuously throughout the simulated year)



Discussion

Main findings and interpretation

It is obvious that achieving eradication of COVID-19 in New Zealand would be the optimal outcome from a health sector perspective. But if this fails then in some scenarios it is still possible to nearly completely suppress the epidemic (as per the $R_0 = 1.5$ scenarios). But such suppression becomes more difficult for the more plausible $R_0 = 2.5$ epidemic scenarios where even nine months of “50% general contact reduction” still doesn’t quite push the epidemic peak into the subsequent year. Even so, the delay from this intervention could still allow for time for improved health system organisation and treatments to be identified – as so could allow for reductions in the hospitalisation and mortality burdens.

The most concerning aspect of the results is the mortality burden for the two highest R_0 values at 8,560 to 14,400 deaths. Nevertheless, at 0.17% to 0.29% of the population dying, this is still not as severe as the 0.8% seen for the 1918 influenza pandemic in New Zealand (ie, 9,000 deaths [9] out of 1.149 million people at that time). Another unfortunate comparison with the 1918 pandemic is the potential increased risk of death for Māori and Pacific populations (albeit based on the H1N1 influenza pandemic experience in 2009 [7, 8]).

When focusing on the $R_0 = 2.5$ scenario with 25% control for six months, the modelling indicates very major impacts on health services. These levels of demand would be completely unprecedented for New Zealand, which has a secondary care sector with very little surge capacity. The filling up of all ICU beds in the country at a very early stage of the epidemic curve (Figures 2 and 3), would mean that the case fatality risk would probably increase (with some signs of this occurring in Italy in March 2020 from the pandemic there).

Study strengths and limitations

This is one of the first SEIR modelling studies of this new pandemic agent COVID-19 and the associated online model has advanced dashboard features and graphic visualisation of results that

facilitate user engagement. Nevertheless, the following are the major limitations of this work, with more minor ones alluded to in the table on the input parameters (Table A1).

- There is still a high degree of uncertainty around many aspects of COVID-19 epidemiology. For example, the R_0 could conceivably be higher than the highest level in the scenarios we modelled (at 3.5, Table 2). Similarly, the CFR could be overestimated (due to missing mild cases in the denominator) or underestimated (if hospitals and ICUs become overloaded and if there are shortages of ventilators during an epidemic peak). Pre-symptomatic spread might also be more important than we have assumed.
- The model was not stochastic, though we largely offset this issue with modelling a wide range of scenarios. The lack of stochastic elements mainly translates into increased uncertainty in the very early stages of epidemic spread, which then impacts on the timing of the peak. But as soon as there are some hundreds of infections in the population, the time course becomes highly predictable (ie, “deterministic”), particularly for the period from “1% of the population have been infected” to the peak.
- The model neither considers any long-term health damage to survivors (especially among risk patients) nor does it consider the hard-to-estimate health loss arising from untreated other health conditions as a result of having an overburdened health system. Likewise we do not consider the additional health harm to the health workers involved (eg, adverse mental health impacts arising from working during a pandemic [10, 11]).

Potential research and policy implications

Clearly, given the uncertainty with many of the input parameters, there will be an on-going need to further revise this type of modelling work. In particular, a more accurate estimate of the R_0 is critical, but even so, this will vary by setting (and New Zealand might be expected to have a relatively low value given that it has comparatively low population density and low use of mass transit compared to many other countries). The uncertainty around this key input parameter further highlights the importance of intensive case identification and contact tracing in the New Zealand population, not only as a control measure *per se*, but also to generate a rapid understanding of local transmission dynamics to ensure that potentially costly population control measures are appropriate and proportionate. Firmer data are also needed on the CFR and the hospitalisation rate from the international literature (ie, the rate when excluding where mild cases are hospitalised just to isolate them and also the rates when a health system becomes overloaded).

Another critical research need is around the feasibility of achieving high levels of contact reduction and case isolation and how long these can be sustained for. At least in the short-term, China has used intensive containment measures successfully as per the findings of the WHO-China Joint Mission Report [12]. This report stated that: “China has rolled out perhaps the most ambitious, agile, and aggressive disease containment effort in history.” Since this report was published it appears that China may have succeeded with eradication (ie, in late March it was only reporting imported cases). While it is an open question around the generalisability of the Chinese approach to other jurisdictions [13], there is also evidence of containment success (as of late March 2020) outside mainland China, from Singapore, Hong Kong and Taiwan [14].

But if the current eradication strategy fails in New Zealand, it is important to also consider how to reduce the load on the health system – which if overloaded may fail to prevent severe outcomes such as death. Potential options are:

- Continue with major investment in prevention and intense containment to slow disease spread (eg, identification and isolation/quarantine of cases and contacts; promoting hygiene measures and physical distancing measures). If strong enough, these measures may still allow

for epidemic peak suppression and displacement into the future until a vaccine becomes available.

- Consideration around investing in website-based educational information for home care for mild/moderately severe cases and capacity for online consultations with health workers (to reduce the demand on the Healthline, other primary care services and hospital services).
- Consideration of specific programmes to protect highly vulnerable groups from infection. The data from China shows older age-groups and those with co-morbidities are at relatively much greater risk of death than younger and healthier groups [6]. Previous New Zealand research on influenza has identified markedly higher rates of hospitalisation for those living with long-term conditions [15]. Also if COVID-19 follows the same patterns as previous pandemics, we would expect a relatively high and heavily unequal hospitalisation and mortality burden on Māori and Pacific populations. Such a protection programme could aim to ensure vulnerable people have the option of moving to or living in “safe havens” for the duration of the pandemic, or for periods when it is at its most intense. Options could include a range of scales from: specific measures for those living in their own homes and well managed institutions to voluntary relocation to specific places that can be protected.
- Continue with investment in planning by hospitals and ICUs (eg, updating triage processes and planning around when to suspend elective surgery and annual leave for public sector health workers etc). For example 40% of admissions to the ICU in Wellington are following elective major surgery [16]. One ICU expert has suggested that ICU bed capacity could potentially be doubled in New Zealand [17]. Fortunately, as of late March 2020, there was already much District Health Board attention being paid to addressing these issues.

Many of these interventions require substantial resources and indeed these are being mobilised by the New Zealand Government with substantial additional health sector funding announced in March 2020. But it will be up to political leaders to continue to balance the potential health benefits of various pandemic control interventions with their downsides. These include the psychological, social and economic costs that may arise from any closing of schools, closing of venues, restricting mass transit and restricting internal travel.

Acknowledgements: We thank the New Zealand Ministry of Health for funding support and for providing feedback on an earlier manuscript draft which used more provisional parameters: particularly the Chief Science Advisor Dr Ian Town; and Dr Richard Jaine. Nevertheless, we note that this work is that of the named authors alone and does not necessarily represent the views of the New Zealand Ministry of Health or any other parties. We thank our German colleagues for their developmental work on the CovidSIM model and their rapid responses around improvements to it: Prof Martin Eichner (University of Tübingen, Germany, and Epimos GmbH), Stefan Brockmann (Landesgesundheitsamt Baden-Württemberg, Stuttgart, Germany), and Dr Markus Schwehm (ExploSYS GmbH, Germany). We also thank our Australian colleagues (Prof Jodie McVernon et al of the University of Melbourne) for providing modelled results from their SEIR model (as used in the head-to-head model comparison). Prof Wilson is supported by the Health Research Council and Ministry of Business Innovation and Employment (MBIE) funding of the BODE³ Programme.

Appendix 1: Parameters, derived variables and differential equations used in the CovidSIM model

Model Description of CovidSIM

Model dynamics

$$\begin{aligned}
 \text{Number of susceptible individuals} \quad & \frac{dS}{dt} = -\frac{S}{N} \left(\beta_P(t) \sum_{k=1}^{n_P} P_k(t) + \beta_I(t) \left(\sum_{k=1}^{n_I} I_k(t) - I_{Iso}(t) - I_{Home}(t) c_{Home} \right) + \psi \right) (1 - c_{Cont}(t)) \\
 \text{Number of individuals in the latent period} \quad & \frac{dE_1}{dt} = \frac{S}{N} \left(\beta_P(t) \sum_{k=1}^{n_P} P_k(t) + \beta_I(t) \left(\sum_{k=1}^{n_I} I_k(t) - I_{Iso}(t) - I_{Home}(t) c_{Home} \right) + \psi \right) (1 - c_{Cont}(t)) - \varepsilon E_1 \\
 & \frac{dE_k}{dt} = \varepsilon E_{k-1} - \varepsilon E_k \quad (1 < k \leq n_E) \\
 \text{Number of individuals in the prodromal period} \quad & \frac{dP_1}{dt} = \varepsilon E_{n_E} - \varphi P_1 \\
 & \frac{dP_k}{dt} = \varphi P_{k-1} - \varphi P_k \quad (1 < k \leq n_P) \\
 \text{Number of individuals in the symptomatic period} \quad & \frac{dI_1}{dt} = \pi P_{n_P} - \gamma I_1 \\
 & \frac{dI_k}{dt} = \gamma I_{k-1} - \gamma I_k \quad (1 < k \leq n_I) \\
 \text{Number of removed individuals} \quad & \frac{dR}{dt} = \gamma (1 - p_{Sick} p_{Death}) I_{n_I} \\
 \text{Number of dead individuals} \quad & \frac{dD}{dt} = \mathcal{P}_{Sick} p_{Death} I_{n_I} \\
 \text{Number of isolated cases at time } t \quad & I_{Iso}(t) = \begin{cases} \sum_{k=1}^{n_I} p_{Sick} I_k(t) & \text{if } t_{Iso_1} \leq t \leq t_{Iso_2} \text{ and } \sum_{k=1}^{n_I} p_{Sick} I_k(t) \leq Q_{max} \\ Q_{max} & \text{if } t_{Iso_1} \leq t \leq t_{Iso_2} \text{ and } \sum_{k=1}^{n_I} p_{Sick} I_k(t) > Q_{max} \\ 0 & \text{if } t < t_{Iso_1} \text{ or } t > t_{Iso_2} \end{cases}
 \end{aligned}$$

Number of fully isolated cases at time t :

$$I_{Iso}(t) = \begin{cases} \sum_{k=1}^{n_I} p_{Sick} I_k(t) & \text{if } t_{Iso_1} \leq t \leq t_{Iso_2} \text{ and } \sum_{k=1}^{n_I} p_{Sick} I_k(t) \leq Q_{max} \\ Q_{max} & \text{if } t_{Iso_1} \leq t \leq t_{Iso_2} \text{ and } \sum_{k=1}^{n_I} p_{Sick} I_k(t) > Q_{max} \\ 0 & \text{if } t < t_{Iso_1} \text{ or } t > t_{Iso_2} \end{cases}$$

Number of home isolated cases at time t :

$$I_{Home}(t) = \begin{cases} \sum_{k=1}^{n_I} p_{Sick} I_k(t) - Q_{max} & \text{if } t_{Iso_1} \leq t \leq t_{Iso_2} \text{ and } \sum_{k=1}^{n_I} p_{Sick} I_k(t) > Q_{max} \\ 0 & \text{otherwise} \end{cases}$$

Initial values

Number of susceptible individuals	$S(0) = N - X$	
Number of individuals in the latent period	$E_1(0) = X$	
	$E_k(0) = 0$	$(1 < k \leq n_E)$
Number of individuals in the prodromal period	$P_k(0) = 0$	$(1 \leq k \leq n_P)$
Number of individuals in the symptomatic period	$I_k(0) = 0$	$(1 \leq k \leq n_I)$
Number of immune individuals	$R(0) = 0$	
Number of dead individuals	$D(0) = 0$	

Parameters

N	Population size
X	Number of initial infections
t_{\max}	Day after introduction of the infection when the transmission potential is highest
Q_{\max}	Maximum isolation capacity
t_{Iso_1}	Time at which isolation measures start
t_{Iso_2}	Time at which isolation measures end
c_{Home}	Fraction of contacts which are prevented for cases who are in home isolation
c_{Cont}	Fraction of contacts which are prevented
t_{Cont_1}	Time at which contact reduction starts
t_{Cont_2}	Time at which contact reduction ends
$c(t)$	Fraction of contacts which are reduced at time t
ψ	Force of infection which originates from outside of the population (e.g. via travellers)
R_0	Average value of the basic reproduction number
a	Amplitude of the seasonal fluctuation of R_0
D_E	Average duration of the latent period
n_E	Number of stages for the latent period
ε	Stage transition rate in the latent period ($\varepsilon = n_E / D_E$)
D_P	Average duration of the prodromal period
n_P	Number of stages for the prodromal period
φ	Stage transition rate in the prodromal period ($\varphi = n_P / D_P$)
i_P	Relative infectiousness during prodromal period
D_I	Average duration of the symptomatic period
n_I	Number of stages for the symptomatic period
γ	Stage transition rate in the symptomatic period ($\gamma = n_I / D_I$)
$\beta_I(t)$	Effective contact rate of individuals in the symptomatic period at time t $\beta_I(t) = R_0 / (i_P D_P + D_I) \cdot (1 + a \cos(t / 365))$
$\beta_P(t)$	Effective contact rate of individuals in the prodromal period at time t ($\beta_P(t) = \beta_I(t) i_P$)
p_{Sick}	Fraction of infected individuals who become sick
$p_{Consult}$	Fraction of sick individuals who seek medical help
p_{Hosp}	Fraction of sick individuals who are hospitalized
p_{ICU}	Fraction of hospitalized individuals who are admitted to the ICU
p_{Death}	Fraction of sick individuals who die from the disease

Derived variables

$$\begin{aligned}
 \text{Symptomatic cases at time } t &= p_{Sick} \sum_{k=1}^{n_I} I_k(t) \\
 \text{Asymptomatic cases at time } t &= (1 - p_{Sick}) \sum_{k=1}^{n_I} I_k(t) \\
 \text{Hospitalized cases at time } t &= p_{Sick} p_{Hosp} \sum_{k=1}^{n_I} I_k(t) \\
 \text{Cases in ICU at time } t &= p_{Sick} p_{Hosp} p_{ICU} \sum_{k=1}^{n_I} I_k(t) \\
 \text{New infections in interval } [t_1, t_2] &= \int_{t_1}^{t_2} \frac{S(t)}{N} \left(\beta_P(t) \sum_{k=1}^{n_P} P_k(t) + \beta_I(t) \sum_{k=1}^{n_I} I_k(t) \right) dt \\
 \text{New sick individuals in interval } [t_1, t_2] &= \int_{t_1}^{t_2} p_{Sick} \varphi P_{n_p}(t) dt \\
 \text{New consultations in interval } [t_1, t_2] &= \int_{t_1}^{t_2} p_{Sick} p_{Consult} \varphi P_{n_p}(t) dt \\
 \text{New hospitalizations in interval } [t_1, t_2] &= \int_{t_1}^{t_2} p_{Sick} p_{Hosp} \varphi P_{n_p}(t) dt \\
 \text{New ICU admissions in interval } [t_1, t_2] &= \int_{t_1}^{t_2} p_{Sick} p_{Hosp} p_{ICU} \varphi P_{n_p}(t) dt \\
 \text{New deaths in interval } [t_1, t_2] &= \int_{t_1}^{t_2} p_{Sick} p_{Death} \gamma I_{nI}(t) dt
 \end{aligned}$$

Detection probability

SARS-CoV-2 infections which are brought into the country may not be detected and may spread without being noticed because the symptoms of COVID-19 may easily be confused with other influenza-like illnesses (ILI). Few practitioners may decide to order a SARS-CoV-2 test for what they regard a normal ILI patient while no community-transmitted cases in the population have been reported. If we assume that fraction of p_{Test} ILI patients who (a) seek medical help or who (b) are hospitalized or who (c) die from the disease are tested for SARS-Cov-2, then the probability that *not one single test* has been performed on a COVID-19 patient by time t despite the ongoing transmission in the population is given by:

$$(1 - p_{Test}) \int_0^t P_{nP}(\tau) \varphi P_{Sick} (p_{Consult} + p_{Hosp} + p_{Death}) d\tau .$$

The probability that at least one test has been performed (and has returned a positive result) is then

$$1 - (1 - p_{Test}) \int_0^t P_{nP}(\tau) \varphi P_{Sick} (p_{Consult} + p_{Hosp} + p_{Death}) d\tau .$$

Table A1: Input parameters for modelling the potential health impacts of the COVID-19 pandemic in the New Zealand setting if eradication fails

Parameter	Value/s used	Further details for inputs into the CovidSIM model and additional Excel-based analyses
Population size	5 million	NZ population as per December 2019 was rounded up from 4,951,500. ^[18] Indeed, the 5 million figure is probably more accurate as per March 2020 due to both population growth and the return of New Zealanders from overseas.
Incoming infected people from outside of NZ	1 per day (from 1 April)	To simulate the start of uncontrolled silent spread in the modelling, we assumed that this began on 1 April 2020 as a result of an asymptomatic traveller entering the country. For the remaining course of the one year simulation, we assumed that this level of introduction persisted (given NZ's commitment to allow its citizen's to return and the potential for home isolation to fail).
Infections that lead to sickness	67%	This figure is still uncertain but we used the same estimate as per modelling by Imperial College at "two thirds of cases being sufficiently symptomatic to self-isolate" [3]. Of note is that another modelling study used a 50% value [19]. Nevertheless, some proportion of asymptomatic cases is consistent with the findings of a very large Chinese study [6], where 81% of cases of COVID-19 did not involve severe illness.
Sick people seek medical help (including telephone and internet consultations)	40%	We used the default value in the CovidSIM model, which is based on medical consultations for influenza-like illness (ILI). During a pandemic there might be a shift away from face-to-face consultations with health workers, so that some of these consultations may be either telephone or internet-based. This parameter is not used for determining subsequent outcomes like hospitalisations and deaths. We further assume that cases only seek medical help once.
Sick people need hospitalisation	1%	This estimate is highly uncertain. We have multiplied by 5 the percentage which has been observed for seasonal influenza and is the default setting in the CovidSIM model (ie, 0.2%), to account for the apparent increased severity of COVID-19. The high uncertainty for this parameter is due to the likely under-diagnosis of mild cases in many settings (impacting the size of the denominator). It also may vary between countries given the use of hospital facilities to isolate mild cases. Modellers in the United Kingdom (UK) have used 4.4% (of all infected cases) [3], and for modelling in the United States 3%, 5% and 12% have been proposed [20]. However, we consider these to be potential over-estimates in the NZ setting where homecare for mild to moderate pneumonia may be promoted in the community in pandemic circumstances. The length of hospitalisation was assumed to be 10 days which is similar to other modelling work eg, 10.4 days for the UK [3].
Hospitalised cases need intensive care (ICU admission)	25%	We used the data from a very large Chinese study for the ratio of "critical" to "severe" cases (ie, $4.7\% / (13.8\% + 4.7\%) = 25.4\%$) [6]. This is similar to the Chinese case series reported by Wang et al at 26.1% [21]. Nevertheless, it is higher than reported in a smaller case series from Singapore at 11% (2/18) [22]. A UK modelling study used a proportion of 30% "based on early reports from COVID-19 cases in the UK" [3]. Of note is that this value is also higher than the NZ experience for the 2009 influenza pandemic at 9.1% (102/1122) [23]. ICU bed capacity: We used the reported number of ICU ventilated beds in NZ at 221 and an estimate from an ICU expert that these could be doubled (ie, to 442) in "extreme circumstances" [17].
Intensive care cases requiring	50%	We use the same value as per a US model of 50% [24] for additional calculations outside of CovidSIM. This proportion is around that reported in a Chinese study of 47% (17/36 ICU admissions) [21], but is less than in

Parameter	Value/s used	Further details for inputs into the CovidSIM model and additional Excel-based analyses
mechanical ventilation		another Chinese study at 71% (37/52) [25].
Sick people die from the disease (case fatality risk)	0.45%	Given the relatively high quality of the healthcare systems in NZ, we considered the lower end of the range reported by the WHO for the infection fatality risk (IFR) of 0.3% to 1% (based on 3 publications) [26]. This IFR was then adjusted by the proportion assumed to be symptomatic (at 67%, as above) to give a case fatality risk (CFR) of 0.45% (ie, $0.3\% \times 100\%/67\% = 0.45\%$). Nevertheless, we note that higher estimates exist, including a CFR for “China outside of Hubei Province” of 0.81% (95%CI: 0.67 to 0.98; and adjusted for the time delay in reporting deaths) [27]. Another CFR for “China outside of Hubei Province” was similar, at 0.9% (95% credible interval: 0.6-1.3%; also adjusted for the time delay in reporting deaths) [28]. A value used in UK modelling was an IFR of 0.9% [3], equivalent to a CFR of 1.3% (assuming 67% of cases are symptomatic).
Basic reproduction number (R_0)	1.5, 2.5 and 3.5 (3 scenario analyses)	On 6 March 2020, the WHO reported that this number was likely to be in the range of 2.0 to 2.5 [29]. But given persisting uncertainty, we used the same three values as in the modelling work by Hellewell et al [2]. Of note is that an earlier review of 12 studies [30], suggested estimates that ranged from 1.4 to 6.49, with a mean of 3.28, a median of 2.79 and interquartile range of 1.16. But this review also noted that in more recent studies, R_0 estimates seem to have stabilised at around 2–3. Recent UK modelling used an estimate of 2.4 (range: 2.0 to 2.6) [3]. Of note is that in the NZ setting R_0 values may be lower than estimated in other settings. This is because relative to many other countries population density is relatively low, mass transit use is low (especially crowded mass transit such as subways), and susceptibility to respiratory viruses might also be reduced (due to relatively low smoking prevalence and low air pollution exposure in NZ).
Relative contagiousness in the prodromal period	50%	There is uncertainty around this value but we used the same estimate as in recent UK modelling [3]. This has biological plausibility as while there is similarity in viral loads between asymptomatic and symptomatic COVID-19 patients [31], it would be expected that those who are fully symptomatic (with a cough etc.) would be more likely to transmit infection. Of note is an estimate from the Diamond Princess cruise ship outbreak, that 17.9% of COVID-19 infections were from asymptomatic individuals (95% credible interval 15.5-20.2%) [32]. But it is unclear how generalisable this finding is given the crowded cruise ship conditions and the typically elderly nature of the passengers.
Latency period	4 days	We used an average duration of 4 days as per Read et al [33], with a standard deviation of 25% (calculated using 16 stages; Erlang distribution). This is similar to the estimate in a Chinese study which reported a median latent period of 3.69 days [34].
Prodromal period	1 day	There is as yet insufficient data on this for COVID-19, so we used an assumed value for influenza (SD = 25%, Erlang distribution).
Symptomatic period	10 days	The WHO-China Joint Mission report stated that “the median time from onset to clinical recovery for mild cases is approximately 2 weeks and is 3-6 weeks for patients with severe or critical disease” [12]. But given that mild cases may have been missed in this particular assessment, we used a slightly shorter time period of 10 days (SD = 25%, Erlang distribution). During this symptomatic period, cases were considered infectious. We note that there is evidence from COVID-19 cases of shedding of viral RNA from sputum that has outlasted the end of symptoms [35]. However, the significance of this for disease transmission is unknown.
Interventions		
General contact reduction	Two scenarios	This variable covers the summated impact of a potentially wide variety of different interventions: people may adopt enhanced personal hygiene

Parameter	Value/s used	Further details for inputs into the CovidSIM model and additional Excel-based analyses
	(25%, 50%) and threshold analyses	<p>measures (hand washing, cough etiquette etc); they may decide to have fewer contacts (physical distancing); and governments may close venues and schools, restrict mass transit, curtail mass gatherings, and restrict travel (within and between countries).</p> <p>Scenario “25%”: This scenario is our approximation of a modest level of the above listed interventions.</p> <p>Scenario “50%”: This scenario assumed an intensification of the measures being adopted (relative to the above scenario).</p> <p>Threshold analyses: This was where we increased the level of “general contact reduction” to a level which pushed the epidemic peak into the following year (ie, past day 365 after the first day of assumed uncontrolled spread of COVID-19 in NZ on 1 April).</p>
Contact reduction begins	1 April 2020	For the purposes of this modelling we assumed that the cases of COVID-19 detected in NZ during March 2020 triggered the process of contact reduction so that this was in place by the time the simulation of uncontrolled spread began on 1 April (see above). Indeed, during March 2020 there was NZ Government advice on hygiene promotion, physical distancing and constraints imposed on the upper size of mass gatherings etc. Multiple organisations also increased provision of hand sanitisers and local government closed some venues.
Contact reduction duration	6 months (9 months and “rest of year” in scenario analyses)	This 6 month period was selected for demonstration purposes and was varied in threshold analyses (Table 1). As further discussed in the main text the feasibility of such sustained interventions for any country is highly uncertain and may not be realistic at high levels for long periods given the adverse social and economic implications.
Seasonality effect	Variation in R_0 of 25%	Winter conditions are known to accelerate transmission of influenza and also the other coronaviruses which cause common cold like symptoms [36]. Enveloped viruses show strong seasonality with winter peaks [37], and SARS-Cov-2 is an enveloped virus. Even though there are many uncertainties relating to seasonality and this novel coronavirus [38], it seems prudent to assume some seasonal fluctuation so we increased the average by 25% in winter and reduced it by 25% in summer (with a sinusoidal variation throughout the simulated year), using a mid-winter peak for NZ of 15 July (ie, day 106 of the simulation).
Case isolation (only used in the threshold analyses)	Varied in threshold analyses (Table 1)	<p>We set the following values in threshold analyses (while setting 0% for “general contact reduction” – see above):</p> <ul style="list-style-type: none"> • Probability that a sick person is isolated = varied in threshold analyses • Maximum capacity of isolation wards = 3 per 10,000 population (ie, 1,500 in total in NZ, see below). • Contact reduction for cases in home isolation = 50% (this occurs when hospital isolation capacity is exceeded) • Beginning of case isolation measures = the 1 April date used for the start of the simulation (ie, assuming increased clinician awareness from the cases in NZ detected during March 2020). • Duration of case isolation measures = 6 months (183 days), or 9 months (274 days) or the rest of the simulated year. <p>For isolation capacity in NZ hospitals we assumed that 10% of hospital beds could be converted for this use during the pandemic, with NZ having 2.61 hospital beds per 1000 population in 2018 [39]. If 10% of these were used for isolation purposes, then this is 2.6 per 10,000 (rounded to 3 per 10,000 for use in CovidSIM, or 1,500 beds in total).</p>

References

1. World Health Organization. Coronavirus disease 2019 (COVID-19) Situation Report – 59. 2020;(19 March). https://www.who.int/docs/default-source/coronaviruse/situation-reports/20200319-sitrep-59-covid-19.pdf?sfvrsn=c3dcdef9_2.
2. Hellewell J, Abbott S, Gimma A, Bosse NI, Jarvis CI, Russell TW, et al. Feasibility of controlling COVID-19 outbreaks by isolation of cases and contacts. *Lancet Glob Health*. 2020.
3. Ferguson N, Laydon D, Nedjati-Gilani G, Imai N, Ainslie K, Baguelin M, et al. Impact of non-pharmaceutical interventions (NPIs) to reduce COVID-19 mortality and healthcare demand. Imperial College 2020;(16 March):1-20.
4. Wang C, Liu L, Hao X, Guo H, Wang Q, Huang J, et al. Evolving epidemiology and impact of non-pharmaceutical interventions on the outbreak of coronavirus disease 2019 in Wuhan, China. *MedRxiv* 2020;(6 March). <https://www.medrxiv.org/content/10.1101/2020.03.03.20030593v1>.
5. Lai S, Ruktanonchai N, Zhou L, Prosper O, Luo W, Floyd J, et al. Effect of non-pharmaceutical interventions for containing the COVID-19 outbreak: an observational and modelling study. *MedRxiv* 2020;(9 March). <https://www.medrxiv.org/content/10.1101/2020.03.03.20029843v2.full.pdf>.
6. The Novel Coronavirus Pneumonia Emergency Response Epidemiology Team. The epidemiological characteristics of an outbreak of 2019 novel coronavirus diseases (COVID-19) — China, 2020. *China CDC Weekly* 2020. <http://weekly.chinacdc.cn/en/article/id/e53946e2-c6c4-41e9-9a9b-fea8db1a8f51>.
7. Verrall A, Norton K, Rooker S, Dee S, Olsen L, Tan CE, et al. Hospitalizations for pandemic (H1N1) 2009 among Maori and Pacific Islanders, New Zealand. *Emerg Infect Dis*. 2010;16(1):100-102.
8. Wilson N, Telfar Barnard L, Summers J, Shanks G, Baker M. Differential mortality by ethnicity in 3 influenza pandemics over a century, New Zealand. *Emerg Infect Dis*. 2012;18:71-77.
9. Rice GW. *Black Flu 1918: The Story of New Zealand's Worst Public Health Disaster*. Christchurch: Canterbury University Press; 2017.
10. Reynolds DL, Garay JR, Deamond SL, Moran MK, Gold W, Styra R. Understanding, compliance and psychological impact of the SARS quarantine experience. *Epidemiol Infect*. 2008;136(7):997-1007.
11. Wu P, Fang Y, Guan Z, Fan B, Kong J, Yao Z, et al. The psychological impact of the SARS epidemic on hospital employees in China: exposure, risk perception, and altruistic acceptance of risk. *Can J Psychiatry*. 2009;54(5):302-311.
12. WHO-China Joint Mission. Report of the WHO-China Joint Mission on Coronavirus Disease 2019 (COVID-19). 2020;(16-24 February). <https://www.who.int/docs/default-source/coronaviruse/who-china-joint-mission-on-covid-19-final-report.pdf>.
13. Kupferschmidt K, Cohen J. China's aggressive measures have slowed the coronavirus. They may not work in other countries. *Science* 2020;(2 March). <https://www.sciencemag.org/news/2020/03/china-s-aggressive-measures-have-slowed-coronavirus-they-may-not-work-other-countries>.
14. Cowling B, Lim W. They've contained the coronavirus. Here's how. *New York Times* 2020;(13 March). <https://www.nytimes.com/2020/03/13/opinion/coronavirus-best-response.html>.
15. Walker TA, Waite B, Thompson MG, McArthur C, Wong C, Baker MG, et al. Risk of Severe Influenza Among Adults With Chronic Medical Conditions. *J Infect Dis*. 2020;221(2):183-190.

16. Capital & Coast DHB. Capital & Coast DHB Intensive Care Unit (ICU). (Updated 11 February 2020). <https://www.healthpoint.co.nz/public/intensive-care/capital-coast-dhb-intensive-care-unit-icu/>.
17. Lewis O. Coronavirus: ICU expert says NZ could double bed numbers in 'exceptional circumstances'. Stuff 2020;(19 March). <https://i.stuff.co.nz/national/health/coronavirus/120398253/coronavirus-icu-expert-says-nz-could-double-bed-numbers-in-exceptional-circumstances>.
18. Statistics New Zealand. Population (December 2019). Statistics New Zealand. <https://www.stats.govt.nz/topics/population> <https://www.stats.govt.nz/topics/population>.
19. Wu J, Leung K, Bushman M, Kishore N, Niehus R, de Salazar P, et al. Estimating clinical severity of COVID-19 from the transmission dynamics in Wuhan, China. *Nature Med.* 2020;(E-publication 19 March).
20. Fink S. Worst-case estimates for U.S. coronavirus deaths. *New York Times* 2020;(Updated 14 March). <https://www.nytimes.com/2020/03/13/us/coronavirus-deaths-estimate.html>.
21. Wang D, Hu B, Hu C, Zhu F, Liu X, Zhang J, et al. Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus-infected pneumonia in Wuhan, China. *JAMA.* 2020;(E-publication 8 February).
22. Young BE, Ong SWX, Kalimuddin S, Low JG, Tan SY, Loh J, et al. Epidemiologic Features and Clinical Course of Patients Infected With SARS-CoV-2 in Singapore. *JAMA.* 2020.
23. Wilson N, Summers JA, Baker MG. The 2009 influenza pandemic: a review of the strengths and weaknesses of the health sector response in New Zealand. *N Z Med J.* 2012;125(1365):54-66.
24. Predictive Healthcare team at Penn Medicine. COVID-19 Hospital Impact Model for Epidemics. University of Pennsylvania, 2020. <http://penn-chime.phl.io/>.
25. Yang X, Yu Y, Xu J, Shu H, Xia J, Liu H, et al. Clinical course and outcomes of critically ill patients with SARS-CoV-2 pneumonia in Wuhan, China: a single-centered, retrospective, observational study. *Lancet Respir Med.* 2020;Published Online (21 February). [https://doi.org/10.1016/S2213-2600\(20\)30079-5](https://doi.org/10.1016/S2213-2600(20)30079-5).
26. World Health Organization. Coronavirus disease 2019 (COVID-19) Situation Report – 30. 2020;(19 February). https://www.who.int/docs/default-source/coronaviruse/situation-reports/20200219-sitrep-30-covid-19.pdf?sfvrsn=3346b04f_2.
27. Wilson N, Kvalsvig A, Telfar Barnard L, Baker M. Case-fatality estimates for COVID-19 calculated by using a lag time for fatality. *Emerg Infect Dis.* 2020 [Early release 13 March]. <https://doi.org/10.3201/eid2606.200320>.
28. Mizumoto K, Chowell G. Estimating the risk of 2019 novel coronavirus death during the course of the outbreak in China, 2020. *MedRxiv* 2020;(23 February). <https://www.medrxiv.org/content/10.1101/2020.02.19.20025163v1>.
29. World Health Organization. Coronavirus disease 2019 (COVID-19) Situation Report – 46. 2020;(6 March). https://www.who.int/docs/default-source/coronaviruse/situation-reports/20200306-sitrep-46-covid-19.pdf?sfvrsn=96b04adf_4.
30. Liu Y, Gayle AA, Wilder-Smith A, Rocklöv J. The reproductive number of COVID-19 is higher compared to SARS coronavirus. *J Travel Med.* 2020.
31. Zou L, Ruan F, Huang M, Liang L, Huang H, Hong Z, et al. SARS-CoV-2 Viral Load in Upper Respiratory Specimens of Infected Patients. *New Engl J Med.* 2020.
32. Mizumoto K, Kagaya K, Zarebski A, Chowell G. Estimating the asymptomatic proportion of coronavirus disease 2019 (COVID-19) cases on board the Diamond Princess cruise ship, Yokohama, Japan, 2020. *Euro Surveill.* 2020;25:pil=2000180. <https://doi.org/2000110.2002807/2001560-2007917>.

33. Read J, Bridgen J, Cummings D, Ho A, Jewell C. Novel coronavirus 2019-nCoV: early estimation of epidemiological parameters and epidemic predictions. MedRxiv 2020. doi: <https://doi.org/10.1101/2020.01.23.20018549>.
34. Li R, Pei S, Chen B, Song Y, Zhang T, Yang W, et al. Substantial undocumented infection facilitates the rapid dissemination of novel coronavirus (SARS-CoV2). Science (New York, NY). 2020.
35. Woelfel R, Corman V, Guggemos W, Seilmaier M, Zange S, Mueller M, et al. Clinical presentation and virological assessment of hospitalized cases of coronavirus disease 2019 in a travel-associated transmission cluster. MedRxiv 2020;(8 March). <https://www.medrxiv.org/content/10.1101/2020.03.05.20030502v1>.
36. Killerby ME, Biggs HM, Haynes A, Dahl RM, Mustaquim D, Gerber SI, et al. Human coronavirus circulation in the United States 2014-2017. J Clin Virol. 2018;101:52-56.
37. Price RHM, Graham C, Ramalingam S. Association between viral seasonality and meteorological factors. Sci Rep. 2019;9:929.
38. Cohen J. Why do dozens of diseases wax and wane with the seasons—and will COVID-19? Science 2020;(13 March). <https://www.sciencemag.org/news/2020/03/why-do-dozens-diseases-wax-and-wane-seasons-and-will-covid-19>.
39. OECD.Stat. Health care resources (2017 and 2018 data for hospital beds per 1000 population). https://stats.oecd.org/index.aspx?DataSetCode=HEALTH_REAC#.