Modelling Estimates of Expected Size: the August 2021 COVID-19 Outbreak in Aotearoa

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EXECUTIVE SUMMARY

This report, produced near the start of the August 2021 COVID-19 outbreak, was written to provide advice to officials on the likely number of cases that might have existed in the community when the first case was detected on August 17th; the likely size and date of the peak number of cases; and the likely overall size of the outbreak over a period of up to two months at Alert Level four. The report uses case data, as at August 26th, to inform these estimates. As the outbreak had not reached its peak at the time of writing, the report does not attempt to estimate the effect of Alert Level four interventions on the effective reproduction number, $R_{eff}$. However, estimates of $R_{eff}$ were calculated in subsequent updates to this report, employing the same methodology as presented here.

We use both an Aotearoa-specific Contagion Network Model (CNM) and a simplified Branching Process Model (BPM) to simulate the spread of COVID-19 in the community, with parameters comparable for those observed for the Delta variant. A simple Gaussian random field/Gaussian Process (GP) methodology is applied to the outputs of both the BP and NC simulations in order to condition the models to real-world data from the current (August 2021) outbreak. We use these conditioned models to provide separate estimates and predictions of a range of metrics related to the outbreak.

Key findings:

- Combined, these models indicate a range of approximately 150–250 cases at detection, with the conditioned branching process model providing a lower range than the conditioned network model. We note for comparisons to the other branching process model reports that our estimates are relatively narrow/less uncertain due to us explicitly conditioning simulation trajectories on the observed data history to date.

- The network model provides a rough estimate of around 2000 total (detected and undetected) cases by October 26, with closer to 1800 detected cases by that date.

- In contrast, the simplified branching process model provides significantly more pessimistic estimates of final case numbers, predicting around 5000 total (detected and undetected) cases by October 26 and closer to 3000 detected cases by that date.

- Both models roughly agree that daily cases are expected to fall between late August and early September. The network model predicts a faster post-peak drop-off in cases, with AL4 spread confined primarily to households and workplaces that remain open.

- The branching process predicts higher peak cases, with a median peak of about 80 daily new cases and an upper limit exceeding 100 daily new cases. In contrast, the median peak of the daily confirmed cases in the network model is around 65 daily cases, with an the upper limit (97.5% quantile) of around 90. We note that daily cases can be expected to be more volatile than cumulative cases, and are also more dependent on testing and tracing assumptions.

These results indicate that it may still be several days (as of August 27) before policy interventions lead to daily case numbers dropping, and that the final size of the present outbreak is expected to be significantly larger than the August 2020 outbreak, with a high probability of exceeding 1000 cases even with Alert Level 4 interventions.

We caution that, due to un-modelled factors, we would not expect predictions to be as reliable as the uncertainty bands indicate beyond a few weeks. The hybrid epidemic model-Gaussian process methodology used here does, however, allow forecasts to be efficiently updated as new data arrives. In future we plan to include both model types into a combined ensemble to condition on data and use to provide consensus forecasts.
Summary of technical findings by model

Network model

- For the network model, there was a median of 244 total cases at detection, with an IQR of 225 to 275 cases. We estimate that there will be approximately 2071 total cases (detected and undetected) by October 26th with an IQR of 1668 and 2534 cases. We estimate that there will be approximately 1823 detected cases by October 26th with an IQR of 1486 and 2220 cases.

- Based on the network model we estimate that case numbers will on average begin to drop 13 days post-intervention, i.e. after August 29th 2021 with a median peak of about 65 cases and upper limit around 90. This is likely to occur between August 27th and September 1st 2021.

- Post-intervention, the majority of transmission occurs via dwellings in the network model, with the rest of transmission primarily in workplaces that remain open. This likely explains the faster drop-off in cases than in the branching process model, as spread saturates within these contexts but does not spread much between contexts.

Simplified branching process model

- For the simplified branching process model, there was a median of 155 total cases at detection, with an IQR of 145 to 164 cases. We estimate that there will be approximately 5260 total cases (detected and undetected) by October 26th with an IQR of 3750 and 7250 cases. We estimate that there will be approximately 3040 detected cases by October 26th with an IQR of 2250 and 4100 cases.

- Based on the simplified branching process model we estimate that case numbers will on average begin to drop 14 days post-intervention, i.e. after August 31st 2021, with a median peak of about 80 cases, with an IQR of 69 to 93 cases. This is likely to occur between August 31st and September 3rd 2021.

- Based on the simplified branching process model, it will take until September 26 to have a 0.7 probability of less than 70 cases being detected at any date afterwards. On average branching process model simulations predict 50 daily cases at this time.
1 Introduction

Here we provide approximate model-based, data-calibrated forecasts of the recent 17 August 2021 outbreak. We consider both a detailed individual-based contagion network model (CNM) that explicitly represents ~5 million individuals along with the contexts in which they interact, as well as a simplified branching process model (BPM) without spatial or demographic structure. The network and associated contagion process are broadly similar to those in\(^1\),\(^2\), while the branching process model is an adapted and simplified version of presented in\(^3\).

The use of two different types of model allows us to compare and cross-compare and validate results produced by the two different modelling approaches. Both models rely on a number of input parameters which must be either estimated from real-world data during the outbreak or inferred from previous outbreaks or from epidemiological literature. Since the models require different types of input parameters comparing their output behaviour gives us a further opportunity to check the consistency of those assumptions. For example the branching process model requires estimates of the effective reproduction number, \(R_{\text{eff}}\), as an input parameter, while the contagion network model requires estimates of parameters related to the reproduction of the SARS-CoV-2 pathogen, the interaction patterns of (potentially infected) individuals. Outputs such as numbers of cases over time can be used to compare the quality of these input assumptions. This is particularly useful in the early stages of an outbreak where case data may be limited or imprecise.

The low computational cost of the BPM also makes it possible to quickly generate large numbers of simulations over a range of parameters, which can be verified against the more computationally expensive CNM. From these BPM ensembles it is possible to produce probability estimates over a large parameter space; while from the more detailed CNM it is possible to look at finer-grained aspects of contagion, such as the interaction context through which infection spreads, under different interventions.

In the early stages of the outbreak, before the peak number of cases has been reached, it is difficult to make accurate estimates of the expected rate of decrease in the number of daily cases in the later part of a (controlled) outbreak. In the case of the BPM, this corresponds to uncertainty in the final value of \(R_{\text{eff}}\). In this report we take a conservative estimate of the final \(R_{\text{eff}}\) value, of between 0.7 and 1.2. This corresponds to a proportional effect of Alert Level 4 on \(R_{\text{eff}}\) post-intervention approximately the same as that estimated for the March/April 2020 Outbreak\(^4\). This can result in a long tail for the duration of outbreaks. If the effect of interventions are closer to absolute than relative, for example if AL4 reduces the present outbreak to a similar \(R_{\text{eff}}\) as in 2020, then this may overestimate the time to elimination and final case numbers (for the BPM in particular). We hence present numbers for the eventual size of the outbreak as estimates for total cases as at October 26th for both the BPM and the CNM.

We use a simple Gaussian random field (GRF)/Gaussian-process (GP) methodology (we treat GPs as finely discretised GRFs for simplicity) developed in\(^5\) to provide conditional prediction bands for both the BPM and the CNM on data from the outbreak over the period 17 August to 26 August 2021. This methodology is described in more detail in the Appendix, along with validation on August 2020 data. Briefly, we fit an empirical mean and covariance to unconditioned model simulations of cumulative (observed and unobserved) cases. These define a GP approximation to the simulation model of interest. This GP then provides easy-to-compute pointwise and curvewise prediction bands and can be efficiently updated conditional on observed data. In addition, we use our conditioned GP approximation to filter our simulation realisations, retaining those that remain within specified curvewise quantile bands. This filtering will retain simulations lying within the future quantile bands while approximately conditioned on the data to date, though it may retain additional simulations giving consistent predictions but less consistent histories (see the Appendix for more). Together, this provides hybrid epidemic-statistical models informed by the more epidemiologically sound models but adaptable to the data without re-simulation.

Using our hybrid contagion-GP methodology, we present a range of projections, including updated model estimates and projections for confirmed and actual cases, beginning from the first confirmed case on 17 August 2021. For the network model we also present infections by context.
2 Simulation settings

2.1 Network model

To provide input into our method for efficiently constructing real-time estimates and projections of case numbers starting from detection to the present, we first use our network contagion model to simulate contagion over a range of parameters centered on our best estimates of real-world values. A detailed description of model parameter is included in Appendix B, but important assumptions and parameters include:

• Disease progression for infected individuals proceeds through a sequence of states. Initially, exposed individuals are infected but not yet infectious, they transition to either pre-symptomatic or asymptomatic states (both infectious). Cases that develop symptoms can become hospitalised and can die while the remainder of the infected cases recover. The parameters controlling transitions between these states are based on international literature — primarily from the OpenABM model\(^6\).

• People can only infect people who they interact with through contexts specified in the interaction network. These contexts are broken into the four categories: Dwelling, Work, School, and Community, with different context types having different levels of infection risk. These contexts can be either ‘close’ or ‘casual’. Interactions through casual contexts have a much lower infection risk than interactions through close contexts.

• Cases that develop symptoms have a propensity that they would seek testing for COVID-19, this level of testing is low pre-detection, and high after the first case has been detected. The time from symptom onset to seeking a test also varies pre- and post-detection.

• The proportion of infections that are asymptomatic varies with age, and equates to about 16% over the whole population, in line with findings from PCR based studies with inclusive symptom case definitions\(^7,8\). Asymptomatic cases are assuming to have zero chance of being tested for COVID-19 unless they have been identified as a casual or close contact of a confirmed case.

• All cases that are detected (confirmed cases) initiate a contact tracing process for all close contacts. The time from positive test to isolation of close contacts differs by context, with dwelling contacts traced faster, then work and school, then community. Casual contacts of detected cases do not isolate, but have a higher propensity to seek a test regardless of symptoms once they find out that they are casual contacts.\(^8\)

• The Alert Level is raised one day after the first case is detected, but contact tracing starts straight away.

• At Alert Level 4 we have two key parameters that we vary for every interaction type in the network. The first is whether each interaction would still occur or not, this represents places that are closed and events that are cancelled. The second is, given that the interaction occurs, what is the level of transmission reduction compared to baseline due to transmission reduction measures such as masks, physical distancing, and rostering changes. For these simulations, we have had to estimate the impact of the current Alert Level 4 setting, we expect to get better estimates of these reductions as the outbreak proceeds.

• Vaccination is allocated randomly in the model using the number of people within each age band, sex, and DHB that had been fully vaccinated (two doses) by 27th July 2021, allowing for 2-3 weeks before it would be fully effective.

The network results are based on an ensemble of simulations that vary in their ‘casual contact tracing’ settings, representing the mechanisms through which casual contacts seek a test. Specifically, we vary two sets of casual contact tracing parameters across three levels: test seeking delay ‘Slow’, ‘Medium’ and ‘Fast’, and test seeking propensity levels ‘Low’, ‘Moderate’ and ‘High’. We parameterise these two sets of three levels as:

\(^*\)We only consider our original test-trace-isolate (TTI) mechanism used previously\(^4\) — representing the TTI policies and procedures used in previous outbreaks. Specific differences in TTI policies that we do not yet include are: the broader definition of close contacts, e.g. everyone at the same school being classed as close, when previously they were previously defined as casual contacts; the requirement for household contacts of close contacts to isolate as well, until the day 5 test result is returned. We are in the process of updating this to reflect the strengthened TTI policy changes for new variants, but have not incorporated this yet.
• ‘Slow’: mean test seeking delay of 5.2 days (∼ Beta(3, 5) × 4.5 + 3.5).
• ‘Medium’: mean test seeking delay of 4.2 days (∼ Beta(3, 5) × 4.5 + 2.5).
• ‘Fast’: mean test seeking delay of 2.6 days (∼ Beta(3, 5) × 3 + 1.5).

and

• ‘Low’: test seeking propensity of 50% in home, school and work casual contacts, 25% in casual community contacts
• ‘Moderate’: test seeking propensity of 75% in home, school and work casual contacts, 50% in casual community contacts
• ‘High’: test seeking propensity of 90% in home, school and work casual contacts, 75% in casual community contacts.

We run 100 realisations for each of the 9 possible combinations, except for (Medium, Moderate) which we run 300 times. We then pool the outputs to provide an empirical prior predictive distribution and fit a GP to this distribution. The GP is then conditioned on observed (cumulative) cases to produce conditional prediction bands. Finally, we also retain and plot simulations giving predictions consistent with these bands. This process is described further in the Appendix.

This ‘hybrid’ GP approximation to the network model can then be efficiently conditioned to match confirmed case numbers as they arrive, without re-running the network model. This hybrid model can adapt to the data to some extent if the model is initially mis-calibrated but is still constrained to follow the same qualitatively expected trajectories from the network model. We found this approach provided bands which could confidently forecast previous outbreaks\(^5\). However, as always, short term forecasts can be expected to be better than the longer term projections.

The results presented in this report use a value of the transmissibility parameter $\beta$ of 2.55. This has been revised upwards from the value of 1.7 that was used in the earlier reports\(^9,10\) on the basis of data on the observed growth rate in the initial period of the current outbreak for the Delta variant.

2.2 Branching process

In addition to the contagion network model, we also consider a simplified branching process model using a broad ensemble of parameters across 8000 model realisations. We validate (and centre) these parameters on 2020 data in the Appendix, before using them (updated for Delta) on 2021 data. The parameters used can be seen in Table 1. Our $R_{eff}$ is increased for the 2021 outbreak due to the higher infectivity of the COVID-19 Delta variant\(^11\). As in the network model, we use our branching model simulations as an empirical prior for a Gaussian process (GP). We then condition the GP on confirmed case numbers to get prediction bands for our forecasts, as well as retain simulation realisations lying within these bands.

The parameters chosen for post-intervention measures correspond to an estimated final $R_{eff}$ value of between 0.7 and 1.2. This causes an estimated proportional effect of Alert Level 4 measures on $R_{eff}$ of between 0.14 and 0.24 ($[0.7, 1.2] \div 5$). This gives a proportional effect of Alert Level 4 on $R_{eff}$ post-intervention that is roughly the same as that estimated for Alert Level 4 on the March/April 2020 Outbreak of between 0.15 and 0.24 ($[0.28, 0.43] \div 1.8$)\(^4\).

We note that the mean times from symptom onset to test result given in Table 1 are very short. This is to account for the lack of contact tracing built into the model; in reality some people will test positive when asymptomatic or pre-symptomatic (due to contact tracing). This is why a low number is used within this simulation. Future models will incorporate a contract tracing mechanism that allows this parameter to adopt a more realistic value.

Based on experience with the network model, post-intervention $R$ scaling is initially $0.8 \times$ the pre-intervention $R$ value, before linearly reducing to the values given in Table 1 over an average 6 days. This linear decrease is intended to capture the effect of a post-intervention lag in reduction of $R$, with infections occurring within households of infectious individuals for a period after the initial intervention. This effect decreases with time, based on the assumption that all the members of a dwelling will eventually either be already infected or will isolate away from the infected individual(s). It should be noted that the R scaling parameter decreases $R_{eff}$, but is not the only thing that affects $R_{eff}$ post-intervention (for example $R_{eff}$ is also a function of the testing rate).
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Minimum Value</th>
<th>Maximum Value</th>
<th>Average Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basic reproduction number $R$ (2020)</td>
<td>3.5</td>
<td>4.5</td>
<td>4</td>
</tr>
<tr>
<td>Resultant pre-intervention $R_{\text{eff}}$ (2020)</td>
<td>2.9</td>
<td>3.75</td>
<td>3.33</td>
</tr>
<tr>
<td>Post-intervention $R$ scaling lower bound (2020)</td>
<td>$0.20 \times R$</td>
<td>$0.30 \times R$</td>
<td>$0.25 \times R$</td>
</tr>
<tr>
<td>Basic reproduction number $R$ (2021)</td>
<td>5.5</td>
<td>6.5</td>
<td>6</td>
</tr>
<tr>
<td>Resultant pre-intervention $R_{\text{eff}}$ (2021)</td>
<td>4.6</td>
<td>5.4</td>
<td>$5^{11}$</td>
</tr>
<tr>
<td>Post-intervention $R$ scaling lower bound (2021)</td>
<td>$0.20 \times R$</td>
<td>$0.25 \times R$</td>
<td>$0.225 \times R$</td>
</tr>
<tr>
<td>Speed of linear decrease to $R$ scaling post-intervention</td>
<td>2 days</td>
<td>10 days</td>
<td>6 days</td>
</tr>
<tr>
<td>Pre-intervention symptomatic testing rate</td>
<td>0.05</td>
<td>0.10</td>
<td>0.75</td>
</tr>
<tr>
<td>Post-intervention symptomatic testing rate</td>
<td>0.8</td>
<td>1.0</td>
<td>0.9</td>
</tr>
<tr>
<td>Pre/post-intervention asymptomatic testing rate</td>
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<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Pre-intervention mean time from symptom onset to test result</td>
<td>1.0 days</td>
<td>3.0 days</td>
<td>2.0 days</td>
</tr>
<tr>
<td>Post-intervention mean time from symptom onset to test result</td>
<td>0.5 days</td>
<td>1.5 days</td>
<td>0.75 days</td>
</tr>
<tr>
<td>Number of seed cases</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Number of cases detected before ‘detection’</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

**Table 1.** Parameter settings for Branching Process Model Ensemble. Parameters are uniformly distributed between their minimum and maximum values. Symptom onset to test result is exponentially distributed with the mean value given in this table. Additional parameter values not mentioned can be found in previous work$^{12}$. 


3 Results

As the branching process model is simpler and has less detailed statistics to report, we first present the results of conditioning this model on data using our Gaussian process methodology.

3.1 Branching process/Gaussian process projections

Figure 1 estimates that there was a median of 155 total cases at detection, with an interquartile range (IQR) of 145 to 164 cases. We estimate a median of 5,260 (IQR=[3750,7250]) total cases by October 26th; of these, we expect a median of 3,040 (IQR=[2250,4100]) detected cases. It shows both the unconditioned realisations of the model ensemble, and the tighter predictions of the model ensemble conditioned on the (cumulative) case data.

Figure 1. August 2021 outbreak using a wide ensemble of parameters centred around the August 2020 outbreak, with increased $R_{\text{eff}}$ for Delta, conditioned to current case data. Cumulative case numbers post-intervention.

Figure 2. August 2021 outbreak using a wide ensemble of parameters centred around the August 2020 outbreak, with increased $R_{\text{eff}}$ for Delta variant, conditioned to current case data. Daily reported case numbers post-intervention.

Figure 2 presents an estimate of daily case numbers reported on each day post-intervention. The estimates in this
figure are constructed by first conditioning the GP approximation on the cumulative data to date and then retaining simulations within 95% curvewise bands for the predictions of future cumulative cases. Due to the approximate nature of the GP, as well as the filtering method used, the model simulations retained will not follow the reported (cumulative or daily) data exactly, even for the data observed to date. However, the bands provide approximate prediction intervals of given levels of coverage, and we expect the general trend of reported daily cases to follow the median approximately.

We caution that the branching process has no knowledge of case ring-fencing or saturation of infections within households. This means that we would likely expect cases to start dropping at a faster rate than predicted after a longer period of time, e.g. more like that seen in the network model below. This could potentially be accounted for with a more complex, time-varying $R_{eff}$.

We estimate that case numbers will, on average, begin to drop 14 days post-intervention (after August 31st 2021), with a median peak of about 80 (IQR = [69, 93]) cases per day. This is likely to occur between August 31st and September 3rd 2021. Table 2 records these estimates based on Figure 2.

<table>
<thead>
<tr>
<th>Data Quartiles</th>
<th>Max Daily Case Number</th>
<th>Peak Position (Days since Intervention)</th>
<th>Peak Position (Date)</th>
</tr>
</thead>
<tbody>
<tr>
<td>25% Quartile</td>
<td>69</td>
<td>16</td>
<td>Sep 2</td>
</tr>
<tr>
<td>Median</td>
<td>78</td>
<td>14</td>
<td>Aug 31</td>
</tr>
<tr>
<td>75% Quartile</td>
<td>93</td>
<td>17</td>
<td>Sep 3</td>
</tr>
</tbody>
</table>

**Table 2.** Estimated dates and size of peak daily cases.

Figure 3 presents a weakened and more nuanced version of ‘probability of elimination’ after a given date based on the case numbers simulated in Figure 2. Instead of elimination, it presents the probability of never again detecting $N$ cases per day after a given date (for the present outbreak). Thus, for example, it estimates that it will take until September 26 to have a 0.7 probability of fewer than 70 cases being detected at any date afterwards. On average, the model predicts around 50 daily cases at this time, as seen in Figure 2.

**Figure 3.** Probability of never again seeing more than $N$ cases, after some specified date in the future given current case data to date. Probability contours are calculated by using multiple realisations of the BPM. **How to read this plot:** Select a number of cases on the y axis. Then using this case number look across the x axis for the probability of never again seeing this number of cases by a given date.
3.2 Contagion network/Gaussian process projections

Here we present results from running our contagion network model and then conditioning this on data from the current outbreak using our GP approximation.

Figure 5 shows the daily new confirmed infections from simulations of the contagion network model, and Figure 4 shows the cumulative confirmed and total case counts, including 95% curvewise quantile bands. Even with our increased transmission rate ($\beta = 2.55$), and the conditioning on recent data pulling our ensemble upwards, our network model appears to be relatively optimistic compared to the branching process. The network model predicts both a lower peak, as well as a steeper drop-off in daily cases. We note, however, that predicting daily case numbers is typically more difficult than predicting cumulative numbers, as the former are more volatile and more dependent on contact tracing and testing assumptions. [Note also that an earlier version of this figure was incorrect due to not plotting all confirmed case categories. Including all confirmed categories provides a much better fit to the data so far.]

Figure 4. (left) Cumulative confirmed cases and (right) cumulative total cases of the network simulations, conditioned on August 2021 outbreak case data.

As indicated in Figures 4 and 5, for the network model, there was a median of 244 total cases at detection, with an IQR of 225 to 275 cases. The model predicts approximately 2071 total cases (detected and undetected) by October 26th with an IQR of 1668 and 2534 cases and approximately 1823 detected cases by October 26th with an IQR of 1486 and 2220 cases. The network model predicts that case numbers will on average begin to drop around 13 days post-intervention, i.e. after August 29th 2021 with a median peak of about 65 cases and upper limit (97.5% quantile) of around 90. This is most likely to occur between August 27th and September 1st 2021.

Figure 6 shows the context in which infections occur, split as pre-intervention (AL1) and post-intervention (AL4). This reveals the role of AL4 in reducing community interactions (infections in EX and EV groups) and school interactions. We note that the (pre-intervention) low transmission in schools is due in part to an assumption about lower susceptibility to infection in children, from 2020 (wildtype) variants. We plan to test the effect of this age-based susceptibility in future, due to the observed infection rates of Delta variant in young people. Post-intervention, the majority of transmission occurs via dwellings in the network model, with the rest of transmission primarily in workplaces that remain open. Note:
Figure 5. Daily new confirmed infections for an ensemble of contagion network simulations conditioned on (cumulative) current outbreak data. Note that because we are approximately conditioning on cumulative cases we do not exactly match the (more volatile) daily cases, though the basic trend is reasonably well captured.

Pre-intervention, the relative infection risk in different interaction types are input parameters for the model. This is then mediated by the network structure, which bounds infections by the number of susceptible contacts in different contexts. We hope to be able to work with NCTS and ARPHS to better estimate both the relative risk in different contexts, and the distribution of number of events in different contexts and their size (number of close and casual contacts) in future.

References

Figure 6. Contexts through which infection occurs, pre- and post-intervention. Possible infection contexts are; dwellings (D), community interactions (EV - casual contacts, EX - close contacts), schools (S), and workplaces (W).


Appendix A: Conditioning methodology and validation on August 2020 Outbreak Results

Our conditioning approach consists of two separate components: a method of constructing conditional prediction bands and a subsequent step of retaining simulations consistent with these bands. The first component of both components involves fitting the empirical mean and empirical covariance to the outputs of an ensemble of simulation realisations from a given model (here, network or branching process). These realisations vary across given parameter ranges and modelling assumptions, and we only fit the combined outputs, i.e. we fit the (prior) predictive distribution, marginalising over parameter settings. The empirical mean and covariance define an empirical predictive prior for a surrogate statistical (non-mechanistic) Gaussian random field model/Gaussian process (GP). Although discretised, for simplicity, we refer to this statistical model as a GP. Using the empirical mean and covariance corresponds to an unconstrained maximum likelihood estimate for our model-based prior predictive distribution. In particular, we do not use a kernel function to constrain our estimated simulation-based prior covariance. This approach follows the ‘Data Space Inversion’ (DSI) methodology of\textsuperscript{13}. DSI avoids any hyper-parameter tuning, other than possibly transforming model outputs to improve the normality assumptions, which would be used in either case. Using the unconstrained estimates is simple but will tend to make the individual elements of the covariance matrix less well-estimated than when using a kernel function. Our main interest is in the predictive performance of prediction bands based on the diagonal entries in covariance, however, rather than precise estimation of the full covariance matrix.

Once we have this surrogate GP over a prior model predictions (the so-called prior predictive distribution in Bayesian inference), we can efficiently condition this on data, using the standard formulae for conditioning multivariate Gaussian distributions. The conditional GP then provides posterior predictions given observations to date. In addition to this relatively standard method of constructing predictive bands, we also retain and plot the simulations lying within these predictive bands. Note, however, that by keeping models giving predictions consistent with the bands on future predictions, this filtering may include some simulations which give similar predictions but a less close match to the data observed so far. This issue is avoided somewhat by conditioning on cumulative case data, for which the future is more strongly dependent on the past. Still, we plan to implement an alternative simulation filtering step (e.g. Approximate Bayesian Computation) in the future and compare this to the current approach. Again, we emphasise that the predictive bands themselves are well-justified as long as the Gaussian approximation is adequate.

For the network simulations, we first fit the empirical mean and empirical covariance to 600 simulation runs of cumulative cases. These 600 runs are made up of 100 runs for 6 different simulation settings. We transform these cumulative case numbers using a power transformation before fitting to ensure a better approximation of normality. We obtain an appropriate power transformation using Tukey’s ladder of powers. We use the same approach for the 8000 model realisations from the branching process.

These means and covariances define empirical priors for corresponding GPs which we then condition on the observed data up to the 26th of August 2021.

To ensure that our methods are reasonable, we validate them against the outbreak data from the 2020 August outbreak. As with the main section of the report, we first present results from the simplified branch process model and then the full network model.
**Hybrid branching process/Gaussian process projections: Validation on 2020 data**

Here we condition the branching process model outputs on the first 10 days of data from the August 2020 outbreak, and produce plots of the predicted confirmed and cumulative (detected and undetected) cases (Figure 7) and daily new confirmed cases (Figure 8).

![Figure 7](image1.png)

**Figure 7.** (left) Conditioned cumulative confirmed cases and (right) conditioned total cases of the branching process model as conditioned on the first 10 days of case data from the August 2020 outbreak.

![Figure 8](image2.png)

**Figure 8.** Daily new case numbers of the branching process model as conditioned on the first 10 days of case data from the August 2020 outbreak.
Hybrid network model/Gaussian process projections: Validation on 2020 data

We also validate our approach by conditioning the network model outputs on the first 10 days of data from the August 2020 outbreak (Figure 9 and 10). We see that the conditioned median tracks the actual outbreak’s confirmed case count quite well, despite the low number of realisations (approximately 100) that were used for conditioning.

Figure 9. Network model conditioned on the first 10 days of the August 2020 outbreak: predictions of (left) cumulative confirmed cases and (right) cumulative total cases.

Figure 10. Daily new case numbers of the network model as conditioned on the first 10 days of case data from the August 2020 outbreak.
Appendix B: Network model parameters

Individuals in the network, with the exception of the seed case(s), are assumed to start in the Susceptible state (though they may possibly be vaccinated). The full set of states that individuals can move through is shown in Figure 11.

Figure 11. State and Transition Diagram for Network Contagion Model. We note that there is no transition from IU→DX, IC→DX, or HQ→DX due to the assumption that all life-threatening cases would be hospitalised first, and all would require critical care before death.

Transmission parameters

Transmissibility We model infection as a transmission process from infected individuals to susceptible individuals that is mediated through the groups, or interaction contexts, that they share. The transmission is modelled as a Markovian process, which occurs with an exponentially distributed interval between successive infection events. The overall infectiousness ‘budget’ that people have is represented by the parameter $\beta$. This $\beta = \beta_c + \beta_0$, where $\beta_c$, the rate of risky contacts/events occurring for ‘close contacts’ is 10 times $\beta_0$, the rate of risky contacts/events occurring for ‘casual contacts’. Based on simulations with uncontrolled spread, and early estimates of the growth rate in the initial stages of the August 2021 outbreak, we use $\beta_c = 2.55$, $\beta_0 = 0.255$, which produces an overall $\beta = \beta_c + \beta_0 = 2.805$.

We conceptualise transmission budget as being a split up based on the ‘time’ that an infectious individual spends in a context with susceptibles, and the ‘riskiness of contact’. We use parameters to split up this transmission budget between three categories of mixing context: home, work or school, and the community. When individuals have more than one mixing context within a category, we assume that individuals spend an even amount of time in each mixing context in each of the categories. For example, if an individual may spend 4 ‘hours’ in community mixing contexts, and is linked to 4 different community mixing contexts, then we assume they spend 1 hour in each mixing context. We make a well-mixed assumption within each mixing context, where individuals spend equal amounts of time with each other individual in that mixing context. The parameters used to split the transmission budgets $\beta_c$ and $\beta_0$ are key input
parameters to the model, and determine the relative spread through different interaction contexts. For close contact events, we split infections up between (Dwellings):(Work or School):(Community) according to the ratios 2.5 : 1 : 0.66. For casual contact events, we split infections up between (Dwellings):(Work or School):(Community) according to the ratios 1 : 2 : 1.33.

Infection events are mediated by the attributes of the individuals involved, as well as the group that the infection event happens in. When a transmission occurs, it has a chance of being rejected, or failing, which is a function of the individuals and group involved. This is to model heterogeneities between individuals. Currently, transmission is automatically denied if both individuals are self-isolating and do not share a dwelling; down-weighted (to represent rule-breaching) if either is in a ‘Confirmed’ state, or is in ‘Self-isolating’ state and the individuals do not share a dwelling. The likelihood of transmission is also down-weighted by the age and vaccination status of the susceptible individual, as well as the current Alert Level policy’s effect on transmission in the group that event is occurring in. Infection events are also rejected in groups that are ‘closed’, due to Alert Level policies.

Finally, infection events involve one infectious individual, but one or more susceptibles. The number of susceptibles that are infected in a single event is drawn from a log-normal distribution based on the size of the group. This currently is only applied to casual community mixing contexts.

**Infectiousness of different states** The states Pre-symptomatic, Symptomatic, and Asymptomatic are all infectious. Due to the state transition model being Markovian, we need to use a constant infectiousness rate for each state, rather than allowing it to vary through time. Using generation times from uncontrolled spread, and comparing to estimates in the literature, we find that setting pre-symptomatic cases and asymptomatic cases to have 70% the infectiousness of a symptomatic individual helps produce reasonable model outputs.

**Susceptibility** We assume age-varying susceptibility to infection, which works by increasing the chance of ‘rejecting’ an infection event, depending on the age of the infectee. For this we use parameters from the OpenABM model\(^6\) for susceptibility by 10 year age band. We then use the population structure by ethnicity to aggregate these values up to the four age bands used in our network. This means that the values differ for individuals in the same age band, depending on their ethnicity.

**Interaction network parameters** We build the interaction network for dwellings, schools, and workplaces, from linked microdata in the StatisticsNZ Integrated Data Infrastructure.

In small dwellings, small schools, and small workplaces, we assume that all contacts are both close and casual contacts. If groups are larger than some threshold, we create smaller groups within the large group which represent the smaller number of close contacts such as a class within a school or a team within a workplace. Note: these close contact group sizes are drawn from a distribution, so some groups will have more and some fewer than the mean sizes listed below, and people can be in more than one close contact group.

For large dwellings (over size 12), the close contact groups have a mean of size 10 and ‘overcover parameter’ of 0.2 which means that people living in dwellings over size 12 have on average 12 close contacts. For large workplaces (over size 10), the close contact groups have a mean of size 10 and ‘overcover parameter’ of 0.1 which means that people in workplaces over size 10 have on average 11 close contacts. For large schools (over size 40, so almost all schools except ECE centres), the close contact groups have a mean of size 30 and ‘overcover parameter’ of 1 which means that people in schools over size 40 have on average 60 close contacts.

For all other close contacts (‘community contexts’) we use a Poisson distribution for the number of ‘events’ per person, with a minimum of one per person. The mean number of events per person is selected to produce a mean number of contacts per person that is double the daily contact counts in ‘other’ contexts from Prem et al.\(^{14}\) ([10.5, 13.2, 8, 6.6] for the four age bands [0-14, 15-29, 30-59, 60+]). We select these close contact event sizes using a Power-law distribution with a minimum of 2, mean of 3.2, and maximum size of 100. We use age-weighted group allocation to produce age-contact matrices in ‘other’ contexts that match Prem et al.\(^{14}\). Overall this results in a mean of 9.6 close contacts per person, with median=5, and LQ=2, UQ=10.

For all other casual contacts (‘community contexts’) we again use a Poisson distribution for the number of ‘events’ per person, with a minimum of one per person and a mean of 3 per person. The casual contact event sizes are drawn from a Power-law distribution, with a mean size of 5.4. We enforce a minimum size of 2, and a maximum of 1000. Once we project this, we get a mean of almost 200 casual community contacts per person, but it is very heavy-tailed (median=50, LQ=14, UQ=207). We assume no age-structure to the casual contacts.

The majority of community interactions are well-mixed within a single Territorial Authority, but a small proportion...
of people are also linked to community events in other Territorial Authorities, with the density of these long-range links between different Territorial Authorities being based on cellphone movement data from 2017\textsuperscript{15}.

**Case progression parameters**

**Latent period** All infected individuals are initially in an Exposed state, where they are not infectious (and will not test positive), for an average of 2 days\textsuperscript{6} (latent period). At that point infections will either move to a pre-symptomatic or asymptomatic state.

**Asymptomatic proportion** Asymptomatic cases ‘recover’\textsuperscript{†} in an average of 10 days\textsuperscript{6}. We use NZ case data from the August Outbreak, fit with a logistic regression model depending only on age, to estimate the proportion of infections that will be asymptomatic. Despite the underlying proportion depending only on age, the proportion in our model vary by ethnicity once we aggregate it up to our four age bands due to the different age structure for different ethnic groups. At a population level, if we assumed all people had an equal infection risk, this comes out to \(\sim 18\%\), but with a strong dependence on age (\(\sim 34\%\) for 0–14 year olds down to \(\sim 6\%\) for 60 years and older).

**Pre-symptomatic and symptomatic states** Pre-symptomatic cases will be infectious and test positive if tested. Symptom onset will occur an average of 3.5 days\textsuperscript{6} after progression from Exposed to Pre-symptomatic. This gives an overall incubation period of 5.5 days. Once symptomatic, cases will either ‘recover’\textsuperscript{†} or be hospitalised. Those that ‘recover’ will take an average of 10 days\textsuperscript{6}. Note: this means that symptomatic cases are infectious for 13.5 days, compared to 10 days for asymptomatic cases. So even though we assume the same infectiousness per unit time, we would expect fewer infections from asymptomatic cases.

**Hospitalisation, critical care, and death** Cases that end up in hospital move from Symptomatic to Hospitalised states in an average of 5 days\textsuperscript{6} after symptom onset, and will then either ‘recover’\textsuperscript{†} or need critical care. For those that ‘recover’\textsuperscript{†}, it will take an average of 6 days\textsuperscript{6}, those that will need critical care will move into critical care in an average of 2 days after hospital admission\textsuperscript{6}. We use age-based estimates for the proportion of hospitalised cases that will need critical care from\textsuperscript{6}. We do not vary this by ethnicity or for the Delta variant. At a population level, if we assumed all people had an equal infection risk, then the overall infection hospitalisation rate for the delta variant would come out to \(\sim 12\%\), but with a strong dependence on age and ethnicity.

Cases that need critical care will either ‘recover’ or die. We use age-based estimates for the proportion of critical cases that will die from\textsuperscript{6}. For those that ‘recover’\textsuperscript{†} from critical care, it will take an average of 16 days, those that die in critical care will die in an average of 9 days\textsuperscript{6}. At a population level, if we assumed all people had an equal infection risk, then the overall infection fatality rate for the delta variant would come out to \(\sim 1.5\%\), but with a strong dependence on age and ethnicity.

**Note on timings** As our disease progression model is Markovian, the above average times represent the means of exponentially-distributed random durations in the corresponding states. This assumption can be relaxed by introducing delay or semi-Markov processes but is not done here.

**Vaccination parameters**

- We currently assume that vaccine effects are uniform over the population, and have no age-, ethnicity- or location-dependent effects. This is only a first approximation as there is e.g. documented age-dependency in vaccine efficacy (especially a significant efficacy drop off in older individuals).

- We assume that the vaccine is effective against both infection and onwards transmission given infection, and that these are affected differently. The reduction in susceptibility to infection is assumed to be 30\% while the typical reduction in onward transmission, given infection, is assumed to be 50\%.

\textsuperscript{†}‘Recover’ only means that they are no longer infectious, and won’t test positive if tested. It does not mean they are symptom-free or even that they are discharged from hospital.
• We assume that infected, vaccinated individuals are 25% less likely to be symptomatic.

• We assume that the vaccine reduces the likelihood of severe outcomes: we assume a 50% reduced chance of hospitalisation, critical care, and death of infected, symptomatic individuals.

• We assume that symptomatic, infectious, vaccinated individuals are 50% less likely to seek a test, compared to similar unvaccinated individuals.

• We assume that the duration of infectivity of vaccinated individuals is identical to unvaccinated individuals.

• We assume that interventions, testing policies, and isolation policies are applied identically to vaccinated and unvaccinated individuals.

Test/trace/isolate parameters

Symptomatic testing We have two different parameters to represent community testing. The first is the likelihood (probability) that someone with symptoms (but no known contact with a case) would seek a test, and test positive. The second is the time from symptom onset to the test result being returned. Before detection of a case, we set the likelihood of seeking a test as check: 10%, the test positivity as 90%, which gives an overall 9% of symptomatic cases that will be detected. The time from symptom onset to return of test follows an exponential distribution with a mean of 5 days. After detection of a case, we decrease the time from symptom onset to the return of test to a mean of 2 days. We do not increase the likelihood of unconnected symptomatic cases seeking testing.

Casual contact testing Casual contacts are not explicitly contact traced. We assume that a proportion of casual contacts would know they were contacts, seek a test, and test positive. This test-seeking is regardless of symptoms. We do not assume that casual contacts would isolate until a test result is returned. The delay between the confirmed case notification and those casual contacts testing positive is modelled as a scaled Beta distribution. Parameters used for the ensemble of simulations used are given in Section 2.1. We do not assume that people being tested because of symptoms or being casual contacts would isolate until a test result is returned.

Close contact contact tracing and self-isolation Time from positive test to contact isolation follows a Weibull distribution (scale = 1.5, shape=1.5). These parameters match the target for P002 of 80% contacted within 48hrs. Only close contacts, as defined in our network structure, are contact traced. ‘Known (traceable) proportions’ of close contacts are set to 100% dwellings, 98% work/school, 85% community. Close contacts in dwellings are contacted first, then those in workplaces, schools, and community. As well as priority in contact order, there is a different probability of a tracing attempt not being successful by group type. Is it 0% for dwellings, 10% for workplaces and schools, and 10% for community contacts. We set the maximum number of trace attempts at 5 for any given contact, and set no capacity limits for contact tracing.

We assume that identified close contacts will isolate at home, so can still transmit to dwelling members as the same rate as baseline. We do not assume that their household members will be told to isolate. We also allow for a 1% ‘leak’ rate. Identified close contacts will isolate for 14 days from notification, and will have multiple tests and symptom follow up that will result in: 95% of symptomatic cases testing positive and 90% of asymptomatic cases. Close contacts who are infected will test positive over time following an exponential distribution with a mean of 2 days after isolation.

Confirmed cases We assume that all confirmed active cases (AC, PC, IC) will be moved to MIQ or equivalent, so there will be no transmission to any of their contacts, positive in those in their dwelling, but we allow for a 1% ‘leak’ rate. Hospitalised cases (HQ and CQ) are assumed to be confirmed on admission, if not already confirmed, but hospitals are assumed to be perfect isolation (no transmission).

Historical cases We define historical cases to be recovered individuals that were not confirmed during their course of infection, corresponding to the RU state. We assume that historical cases will not seek tests spontaneously, but will test positive if they are tested. Historical cases that are casual contacts of a confirmed individual will test, for example. Historical cases that are close contacts of a confirmed individual will be assumed to test similarly to other self-isolated individuals, if they are notified by contact tracing. Historical cases that are identified through testing will induce contact tracing processes as if they were infected, but will not be required to isolate themselves (we assume that they have no infectivity, so their isolation does not particularly matter in our model). They will also be categorised as “known” as they move to a RT state.
Alert Level 4 parameters

In Alert Level 4 everyone is meant to stay at home in their bubble, except for essential personal movement, all schools are closed, only essential workplaces are open, and those workplaces that are open must follow strict infection prevention practices. For each interaction context type we are able to set a proportion of groups/events that are closed/cancelled, and a proportion transmission reduction for those remaining open.

- We assume that there is a 2% reduction in transmission risk for close contacts in dwellings. In large dwellings, e.g. boarding houses, defense force facilities, we assume that there would be a 5% reduction in transmission to casual contacts.

- We set 100% of schools to be closed.

- We represent the impact of Alert Level 4 on workplaces as 30% of workplaces being open (based on StatsNZ Household Labour Force Survey estimates of proportion of workers working on site under different Alert Levels in 2020\textsuperscript{17}), but with 82% reduction in casual contact workplace transmission risk and 57% reduction in close contact workplace transmission risk.

- For all other non-work, non-school interactions (community layer), we assume the restrictions mean that compared to baseline community close contact events 99% wouldn’t occur, this results in only 2% of people who have close contact with someone outside their dwelling, and there is a 25% reduction in transmission risk for those that do still occur. For casual contact community events (e.g. shopping, transport, etc.) we assume that 92% don’t occur and that for those that do, there is a 75% reduction in transmission risk for those that do due to physical distancing measures, mask wearing, etc. Finally, for the community events that do still occur, we enforce an event size limit of 10. We assume here that, if an event is larger than that, that it does not happen, as opposed to keeping the same number of events but reducing their size.