

# Near-term forecasting of Covid-19 cases and hospitalisations in Aotearoa New Zealand

Michael J. Plank<sup>1</sup>, Leighton Watson<sup>1</sup>, Oliver J. Maclaren<sup>2</sup>

1. School of Mathematics and Statistics, University of Canterbury, Christchurch, New Zealand.
2. Department of Engineering Science, University of Auckland, Auckland, New Zealand.

## Abstract

Near-term forecasting of infectious disease incidence and consequent demand for acute healthcare services can support capacity planning and public health responses. Despite well-developed scenario modelling to support the Covid-19 response, Aotearoa New Zealand lacks advanced infectious disease forecasting capacity. We develop a model using Aotearoa New Zealand's unique Covid-19 data streams to predict reported Covid-19 cases, hospital admissions and hospital occupancy. The method combines a semi-mechanistic model for disease transmission to predict cases with Gaussian process regression models to predict the fraction of reported cases that will require hospital treatment. We evaluate forecast performance against out-of-sample data over the period from 2 October 2022 to 23 July 2023. Our results show that forecast performance is reasonably good over a 1-3 week time horizon, although generally deteriorates as the time horizon is lengthened. The model has been operationalised to provide weekly national and regional forecasts in real-time. This study is an important step towards development of more sophisticated situational awareness and infectious disease forecasting tools in Aotearoa New Zealand.

Keywords: disease surveillance; epidemic forecast; renewal equation; semi-mechanistic model; SARS-CoV-2.

## 19 **Author summary**

20 The emergency phase of the Covid-19 pandemic has ended, but Covid-19 continues to put  
21 significant additional load on stretched healthcare systems. Forecasting the number of hos-  
22 pital cases caused an infectious disease like Covid-19 over the next few weeks can help with  
23 effective planning and response. The ability to forecast reliably requires timely, high-quality  
24 data and accurate mathematical models. We have developed a model for forecasting the  
25 number of Covid-19 cases and hospitalisations in Aotearoa New Zealand. The model works  
26 in two stages: firstly predicting the number of new cases and secondly estimating the pro-  
27 portion of those cases that will need hospital treatment. The model produces a range of  
28 likely values, which is important because is impossible to predict with 100% accuracy. We  
29 show that the model does a reasonably good job of predicting hospitalisations up to 3 weeks  
30 ahead. The model has been used by public health agencies in Aotearoa New Zealand to help  
31 with healthcare capacity planning.

## 32 Introduction

33 New Zealand used a combination of border controls and public health and social measures  
34 to suppress or eliminate transmission of SARS-CoV-2 in 2020 and 2021. By the end of 2021,  
35 high vaccine coverage had been achieved, with around 90% of those aged over 12 years having  
36 had at least two doses of the Pfizer/BioNTech BNT162b2 vaccine. Up to this time, there  
37 had been only around 3 confirmed cases of Covid-19 per 1,000 people and 0.01 Covid-19  
38 deaths per 1,000 people. In February 2022, the B.1.1.529 (Omicron) variant began to spread  
39 in the community and subsequently caused a series of large waves dominated by a series of  
40 subvariants [1, 2] .

41 A range of epidemiological models have been used to provide situational awareness and  
42 policy advice to inform the New Zealand Government’s pandemic response. These have  
43 primarily consisted of increasingly complex mechanistic models of transmission dynamics,  
44 including factors such as age structure, vaccination status [3], social contact networks [4],  
45 waning immunity [5], reinfection [6], dynamic behavioural change, new variants [2]. This  
46 level of detail requires making relatively strong assumptions on the mechanisms underlying  
47 observed dynamics and is hence most appropriate for scenario analysis, which does not aim  
48 to make accurate long-term predictions but rather to deliver insights into key mechanisms  
49 affecting epidemic dynamics and a systematic approach to exploring the likely consequences  
50 of alternative strategies or policy decisions.

51 Near-term forecasting is another use of epidemiological modelling, distinct from medium-  
52 term or long-term scenario analysis. Here the focus is on accurately predicting epidemic  
53 dynamics and consequent demand for acute healthcare services over a time horizon of a  
54 few weeks [7]. Assuming no dramatic changes in the mechanisms driving observed epidemic  
55 dynamics over the short term, higher-level models can be used, which summarise the com-  
56 bined effects of underlying transmission mechanisms in terms of coarse-grained parameters  
57 that can be empirically estimated. This class of model includes so-called ‘semi-mechanistic’  
58 models, typically based on the renewal equation [8–11]. These models require fewer detailed  
59 assumptions and are less sensitive to parameter uncertainty and model mis-specification.  
60 On the other hand, they aim to maintain sufficient mechanism and flexibility to respond  
61 realistically to changing trends in epidemiological data and be fitted, evaluated, interpreted

62 and updated in real-time. They also account for known lags affecting epidemiological data  
63 streams, such as delays from infection to symptom onset, testing, hospital admission or death  
64 [12, 13].

65 Some approaches to epidemic forecasting incorporate more mechanistic assumptions about  
66 transmission based on the standard susceptible-exposed-infectious-recovered (SEIR) epi-  
67 demiological modelling framework [14, 15]. This allows the effect of immunity in reducing  
68 transmission rates to be explicitly accounted for, which may improve forecast performance.  
69 However, the downside of this is that it typically requires additional data or assumptions  
70 about, for example, case ascertainment rates, effectiveness of vaccine-derived and infection-  
71 derived immunity, and waning immunity [16, 17]. An advantage of a simpler approach is that  
72 the combined effect of immunity and other factors affecting the time-varying reproduction  
73 number, such as contact patterns and population heterogeneity, is inferred empirically from  
74 the data in real-time.

75 Some forecasting frameworks incorporate independent data, for example from behavioural  
76 surveys, about changes in the average number of contacts per person [17–19] or contact rates  
77 between different age groups [11, 20]. Such data can allow the effects of potential behavioural  
78 change and age structure to be built into forecasts and their associated uncertainty. However,  
79 this type of data is not available in Aotearoa New Zealand.

80 Aotearoa New Zealand currently lacks dedicated forecasting tools for Covid-19 and other  
81 infectious diseases [21]. In this study, we present a method for forecasting Covid-19 cases,  
82 hospital admissions and hospital occupancy in Aotearoa New Zealand. The model has been  
83 developed specifically for New Zealand’s Covid-19 surveillance systems and data collection  
84 and reporting standards. The method is being used operationally by Te Whatu Ora (Health  
85 New Zealand) with support from Precision Driven Health to provide intelligence to health  
86 planners around the country. The model is a semi-mechanistic model for disease transmission  
87 based on the renewal equation [8, 10]. We use a Bayesian particle filter approach [12] to  
88 estimate the time-varying reproduction number and forecast the number of reported cases.  
89 This is coupled with Gaussian process regression models for the distribution of cases across  
90 age groups and the age-specific case hospitalisation ratio to forecast hospital admissions.  
91 Hospital occupancy is estimated using empirical data on age-specific length of hospital stay.  
92 We evaluate model performance by comparing forecasts generated from data supplied on a

93 specific date to subsequently reported data.

## 94 **Methods**

### 95 **Data**

96 We used Ministry of Health data on reported cases of Covid-19 in New Zealand between  
97 25 January 2022 and 24 July 2023. The dataset contained unit record data on age, report  
98 date and, for a subset of cases, self-reported symptom onset date. For hospitalised cases,  
99 data was available on the admission date and the number of days for which the patient was  
100 receiving hospital treatment for Covid-19, referred to as length-of-stay. This dataset was  
101 generated by the Ministry of Health by linking self-reported positive test results (mostly  
102 from self-administered rapid antigen tests) with hospital data from the National Minimum  
103 Dataset (NMDS) and Inpatient Admissions (IP) database based on national health index  
104 (NHI) number. In this dataset, hospital admissions are categorised by the Ministry of Health  
105 as either Covid-19-related or incidental (i.e. those who had tested positive but were not being  
106 treated for Covid-19), using clinical codes (for NMDS) or health specialty (for IP). There  
107 are significant time lags in reporting this information and as a result the number of Covid-  
108 19-related admissions recorded on a given day can vary from one update to another, and is  
109 typically incomplete for the most recent 1-2 weeks of data (see Supplementary Figure S1).

110 We also accessed Ministry of Health data on the total number of confirmed Covid-19 patients  
111 occupying an admitted bed (hospital occupancy). These data are from the Daily Hospital  
112 Capacity survey, which provides a count of the total number of Covid-19 patients in hospital  
113 each day and as such does not suffer from any significant reporting lag or revisions to  
114 historical data. These data are publicly available at [https://github.com/minhealthnz/  
115 nz-covid-data/tree/main/cases](https://github.com/minhealthnz/nz-covid-data/tree/main/cases) and updated daily.

116 From the unit record data, we calculated the number of daily reported cases and number of  
117 new daily hospital admissions in 10-year age bands (Supplementary Figure S1). Hospitalisa-  
118 tions that were classified as “not Covid-19-related” were excluded. We estimated the number  
119 of daily discharges from hospital by assigning each hospitalised case a pseudo-discharge date

120 using their admission date and Covid-19-related length-of-stay. This is an approximation  
121 because some patients may have been admitted for non-Covid-19-related treatment and were  
122 only treated for Covid-19 later during they stay. However, we only use discharge data for  
123 visual comparison of model outputs, not for model fitting or validation.

124 We calculated the onset-to-report distribution for cases that were reported in the 70 days  
125 prior to the date the data was supplied and had an onset date recorded (Supplementary  
126 Figure S2a). Cases with report date more than 7 days prior or more than 14 days after onset  
127 date ( $< 0.2\%$  of the cases that had onset date recorded) were excluded.

128 We calculated the Covid-19-related length-of-stay distribution in each 10-year age band  
129 and the report-to-admission distribution for cases reported between 56 and 126 days prior  
130 to the date the data was supplied (Supplementary Figure S2b-c). We did not include cases  
131 reported less than 56 days prior to this date in these calculations because of lags in recording  
132 hospitalisation data and right-censoring of patients who had not yet been discharged. Cases  
133 with Covid-19-related length-of-stay longer than 56 days (approximately  $0.5\%$  of admissions)  
134 were excluded. Cases with admission date more than 7 days prior to report date were  
135 excluded. This was a significant proportion (approximately  $7\%$ ) of admissions but it is likely  
136 that many of these were initially admitted for non-Covid-related treatment and were only  
137 later treated for Covid.

## 138 **Model**

139 We used a model consisting of two components. The first was a semi-mechanistic disease  
140 transmission model that was fitted to data on reported daily cases. We used this to produce  
141 simulated time series for cases, which can be projected forwards in time. The second com-  
142 ponent was a hospitalisation model that we used to estimate the time-varying, age-specific  
143 case hospitalisation ratio (CHR). We then applied this to the simulated time series for cases  
144 to produce simulated time series for admissions and hospital occupancy.

145 Reported cases represent only a fraction of all infections due to the fact that the majority  
146 of cases are self-reported and intensive case finding and contact tracing programmes had  
147 been wound down by the time of the study period in 2022-23. It is likely that high rates

148 of mild and asymptomatic infection and high levels of population immunity during the  
149 study period further reduced case ascertainment. We did not attempt to estimate the total  
150 number of infections, which is difficult to do without serological data or regular testing of a  
151 representative cohort [22, 23]. Instead, we estimated the case hospitalisation ratio directly  
152 based on the proportion of cases in each age group that were hospitalised. This variable will  
153 be influenced both by disease severity and by case ascertainment rates. However, the key  
154 output of interest (near-term forecast hospitalisations) is insensitive to these factors once  
155 the number of cases and the case hospitalisation ratio are known.

## 156 **Transmission submodel**

157 We modelled the number of cases  $I_t$  infected on day  $t$  using a semi-mechanistic framework  
158 based on the renewal equation [8]

$$I_t \sim \text{Poisson} \left( R_t \sum_{s=1}^n I_{t-s} u_s \right), \quad (1)$$

159 where  $R_t$  is the time-varying instantaneous reproduction number and  $u_t$  is the probability  
160 mass function for the generation time distribution, assumed to be a discretised Weibull  
161 distribution with mean 3.3 days and standard deviation 1.3 days [24, 25]. The reproduction  
162 number was modelled as a Gaussian random walk

$$R_t \sim N(R_{t-1}, \sigma_R). \quad (2)$$

163 Reporting lags were accounted for via a distribution  $v_t$  of times from infection date to report  
164 date. This was the convolution of the incubation period distribution, assumed to be a  
165 discretised Weibull distribution with mean 3.2 days and standard deviation 2.2 days [26–  
166 28], and the empirical onset-to-report distribution (see Data section above). The expected  
167 number of cases reported on day  $t$  was therefore

$$Z_t = \sum_{s=1}^n I_{t-s} v_s. \quad (3)$$

168 The number of observed cases on day  $t$  was modelled as

$$C_t \sim \text{NegBin}(\mu = \omega_{i[t]} Z_t, k_c), \quad (4)$$

169 where  $\omega_{i[t]}$  is an empirical day-of-the-week effect ( $i = t \bmod 7$ ) and  $k_c$  is a dispersion factor.  
170 The day-of-the week effect was estimated directly from the data as the relative difference  
171 between daily reported cases  $\hat{C}_t$  and the seven-day rolling average over a 15 week period:

$$\omega_i = \frac{1}{N} \sum_{t \bmod 7=i} \frac{\hat{C}_t}{\sum_{s=t-3}^{t+3} \hat{C}_s} \quad (5)$$

172 We fitted the model to the time series of reported daily cases  $\hat{C}_t$  using a bootstrap filter  
173 as follows [29]. We simulated  $M$  realisations (or particles) of the stochastic model defined  
174 by Eqs. (1)–(3) (i.e.  $M$  particles), with each particle consisting of time series for  $I_t$ ,  $R_t$   
175 and  $Z_t$ . At each time step, particle  $j$  was assigned a weight  $j$  using the likelihood of the  
176 observed value of  $\hat{C}_t$  under the distribution in Eq. (4). The population of  $M$  particles was  
177 then resampled by drawing, with replacement, from the full set of particles with weights  $j$ .  
178 For time steps after the last available data point (i.e. the prediction period), each particle  
179 was simply simulated forwards in time according to Eqs. (1)–(3) with no filtering.

180 We initialised the model over a period of  $t_{\text{init}} = 20$  days by drawing  $I_t$  from a Poisson  
181 distribution with mean equal to the number of observed cases  $\hat{C}_{t+m}$ , where  $m$  is the mean  
182 infection to report time. The value of  $R_t$  at the end of the initialisation period ( $t = t_{\text{init}}$ )  
183 was drawn from the estimated posterior for  $R_t$  based on the values of  $I_s$  for  $s < t$  using the  
184 method of [8]. Model results were not sensitive to the initialisation period because all model  
185 simulations were initialised a minimum of 88 days prior to the forecast date.

## 186 Hospitalisation submodel

187 To estimate hospitalisations, we fitted models for the distribution of new cases by age and  
188 for the CHR in each 10-year age band. We fitted the log-transformed ratio  $r_{it} = \hat{C}_{it}/\hat{C}_{i't}$  of  
189 cases in age group  $i$  to cases in a reference age group  $i'$  (arbitrarily set to be the 40-49-year  
190 group), and the logit-transformed CHR in age group  $i$  as independent Gaussian process over  
191 time:

$$\log(r_{it}) \sim GP(\mu(t), K(t, t')), \quad (6)$$

$$\text{logit CHR}_{it} \sim GP(\mu(t), K(t, t')), \quad (7)$$



192 where  $\text{CHR}_{it}$  was defined as the proportion of cases reported on day  $t$  that were hospitalised  
193 for Covid-19 using a 7-day centred rolling average. These models were trained using the *fitrgp*  
194 package in Matlab2022b with a squared exponential kernel  $K$  and default hyperparameter  
195 settings. We fitted the age distribution model to data in the 56 days prior to the most recent  
196 available data. To allow for reporting lags in hospitalisation data, we fitted the CHR model  
197 to data between 84 and 21 days prior to the most recent available data.

198 We then used the fitted models to predict the overall CHR on day  $t$  as

$$\text{CHR}_t = \frac{\sum_i r_{it} \text{CHR}_{it}}{\sum_i r_{it}} \quad (8)$$

199 We included model uncertainty in  $r_{it}$  and  $\text{CHR}_{it}$  by independently sampling different trajec-  
200 tories from the fitted Gaussian processes for each particle  $j$ .

201 We then simulated the number of new admissions  $A_t$  on day  $t$  by applying the predicted  
202 CHR from Eq. (8) to the output  $I_t$  of the particle filter:

$$A_t \sim \text{NegBin}(\mu = A_t^* \text{CHR}_t, k_h), \quad (9)$$

203 where  $k_h$  is a dispersion factor,  $A_t^* = \sum_s I_{t-s} w_s$  and  $w_s$  is the probability mass function  
204 for the distribution of time from infection to admission. This distribution was estimated as  
205 the convolution of the assumed distribution for the time from infection to report  $v_t$  and the  
206 empirical distribution for the time from report to admission.

207 In order to predict hospital occupancy, we also needed to model hospital discharges. We  
208 modelled the distribution of Covid-19-related length-of-stay for cases admitted on day  $t$   
209 by combining the empirical age-specific length-of-stay distributions with the modelled age  
210 distribution of hospitalised cases. Specifically, the probability  $l_{st}$  that an admission on day  
211  $t$  will have length-of-stay  $s$  days was calculated as

$$l_{st} = \frac{\sum_i l_{si}^{(\text{age})} r_{it} \text{CHR}_{it}}{\sum_i r_{it} \text{CHR}_{it}}, \quad (10)$$

212 where  $l_{si}^{(\text{age})}$  is the probability that an admission in age group  $i$  will have length-of-stay  $s$   
213 days.

214 We calculated the number of discharges  $D_t$  on day  $t$  by summing over day of admission  $t'$ :

$$D_t = \sum_{t'} A_{t'} N_{t-t',t}, \quad (11)$$

215 where  $N_{st} \sim \text{Multinomial}(A_t, l_{st})$  is the number of admissions on day  $t$  that have Covid-19-  
216 related length-of-stay  $s$ . We calculated net change in hospital occupancy since day  $t_0$  as the  
217 cumulative number of admissions minus the cumulative number of discharges since day  $t_0$ .  
218 Hospital occupancy at time  $t = t_0$  was set so that the mean and standard deviation of the  
219 particles' hospital occupancy were equal to the observed hospital occupancy on day  $t_0$  and  
220 the standard deviation in observed hospital occupancy in the week prior to  $t_0$  respectively.

## 221 Forecast generation and evaluation

222 In order to test the performance of the model against out-of-sample data, we generated  
223 forecasts using data supplied on one of a series of dates spaced at one-week intervals from  
224 2 October 2022 to 23 July 2023. This ensured that forecasts were based only on the data  
225 that was available at a given time point, at which recent hospitalisation data was typically  
226 incomplete (see Supplementary Figure S1b). We then compared forecasts generated at time  
227  $t_f$  with subsequently observed data at times  $[t_f - 6, t_f]$  (nowcast),  $[t_f + 1, t_f + 7]$  (7-day  
228 forecast),  $[t_f + 8, t_f + 14]$  (14-day forecast), and  $[t_f + 15, t_f + 21]$  (21-day forecast).

229 We quantified forecast skill by calculating the continuous ranked probability score (CRPS)  
230 (see e.g. [30]) and bias. For a forecast specified by cumulative distribution function  $F(x)$   
231 and data  $\hat{x}$ , the CRPS is defined as

$$\text{CRPS}(\hat{x}) = \int_{-\infty}^{\infty} (F(x) - I(x \geq \hat{x}))^2 dx \quad (12)$$

232 where  $I(\cdot)$  is the indicator function. Bias is defined as  $\text{bias}(\hat{x}) = 1 - 2F(\hat{x})$ . This metric lies  
233 between  $-1$  and  $1$  and is equal to zero if the data coincides with the median of the forecast  
234 distribution.

235 We calculated the CRPS on log transformed data using the transformation  $\tilde{x} = \ln(x + 1)$ .  
236 This better reflects the exponential nature of epidemic growth and decay, and leads to CRPS  
237 values that are independent of the magnitude of the observed quantity [31], which will be  
238 very different for cases compared to admissions for example. It also means the CRPS values  
239 can be interpreted as a probabilistic measure of relative error [31]. For example, for a point  
240 forecast  $x_f$ , it follows from Eq. (12) that

$$\exp(\text{CRPS}) - 1 = \frac{|x_f - \hat{x}|}{\min(x_f, \hat{x}) + 1} \quad (13)$$

Parameter	Value
Generation time mean (s.d.)	3.3 (1.3) days
Incubation mean (s.d.)	3.2 (2.2) days
Std. dev. in daily random walk step for $R_t$	$\sigma_R = 0.025$
Dispersion factor for daily cases	$k_c = 100$
Dispersion factor for daily admissions	$k_h = 100$
Number of particles	$M = 10^4$
Initialisation period for renewal equation model	$t_{\text{init}} = 20$ days

Table 1: Parameter values used in the model.

241 which is an approximation to the relative difference between the forecast  $x_f$  and the data  $\hat{x}$ .

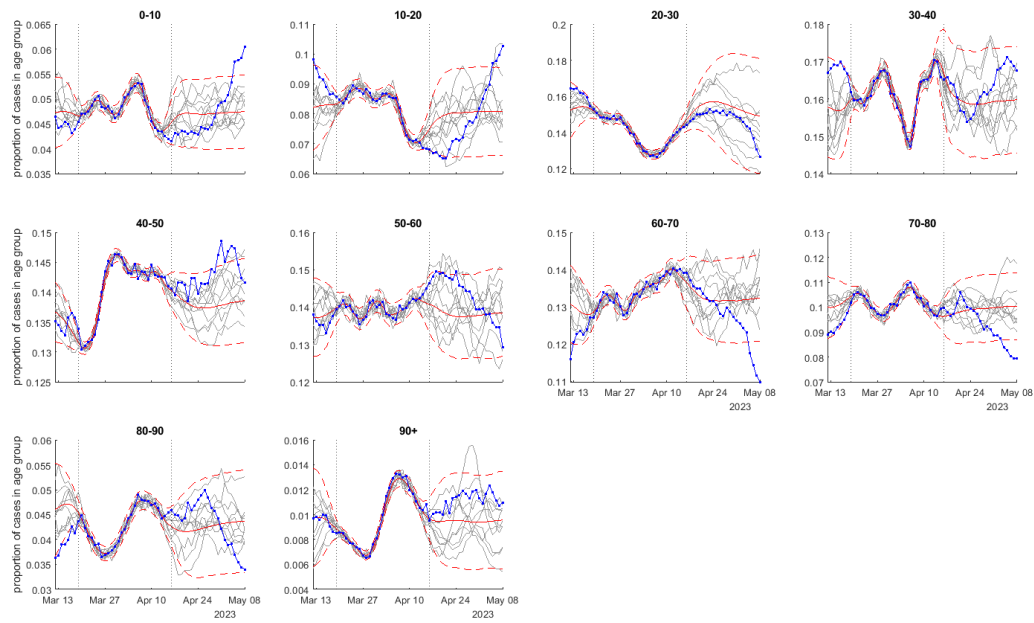
242 Model parameters are shown in Table 1. Model results were not highly sensitive to the  
243 random walk standard deviation  $\sigma_R$  and dispersion factors  $k_c$  and  $k_h$ ; the selected values were  
244 found to give good coverage properties and a reasonable balance between being responsive  
245 to changes in trends while avoiding overfitting. Data and documented code to reproduce  
246 the results are available at [https://github.com/michaelplanknz/covid19\\_forecasting\\_](https://github.com/michaelplanknz/covid19_forecasting_public)  
247 `public`.

## 248 Results

249 Figure 1 shows the fitted Gaussian process regression models for the age distribution of  
250 reported cases and the age-specific case hospitalisation ratio (CHR). The models were fitted  
251 to data supplied on an example forecast date (16 April 2023) and then projected forwards  
252 in time and compared to subsequently available data up to 7 May 2023. Overall, the fitted  
253 models made good predictions for future, out-of-sample data, which generally fell within the  
254 95% prediction intervals and visually exhibited a similar level of temporal autocorrelation  
255 as simulated model trajectories. There were some notable exceptions. For example the  
256 proportion of cases in age bands in the under 20 years and 60 to 80 years range started to  
257 track outside the predicted intervals 2-3 weeks after the forecast date (Figure 1(a)). The  
258 CHR in the 0-10 years age group deviated outside the prediction interval for a period of time  
259 around 1-2 weeks after the forecast date (Figure 1(b)).

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(a)



(b)

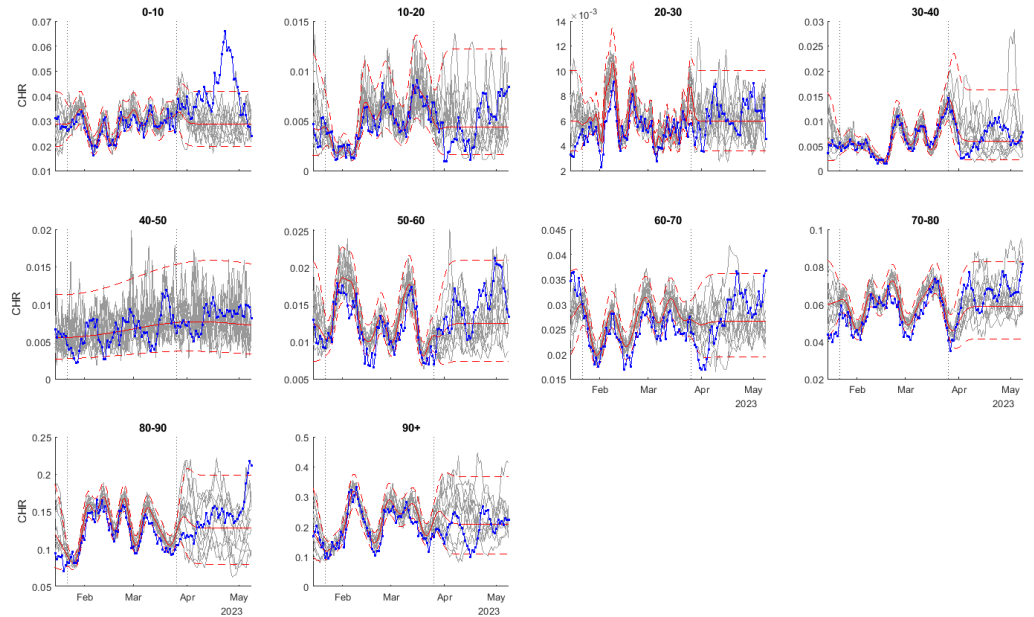


Figure 1: Fitted Gaussian process regression models for: (a) the proportion of cases in each age group; (b) the case hospitalisation ratio (CHR) in each age group. Models fitted to data supplied on 16 April 2023. Dotted vertical lines shown the fitting window (19 March to 16 April 2023 for proportion of cases in each age group, 22 January to 26 March 2023 for CHR). Each panel shows the mean (solid red) and 95% prediction interval (dashed red) of the fitted model, ten example simulated trajectories from the fitted model (grey), and comparison to subsequently observed data up to 7 May 2023 (blue).

260 Figure 2 shows the forecast for cases, new hospital admissions and hospital occupancy, gen-  
261 erated from data supplied on 16 April 2023. The forecast performed reasonably well when  
262 compared against subsequently available data up to 7 May 2023. Daily cases and daily ad-  
263 missions were almost entirely within the 90% prediction interval, although predominantly  
264 below the predicted median, indicating that epidemic growth slowed in the 3 weeks following  
265 the forecast date. Note that the historic modelled levels of hospital occupancy (to the left  
266 of the dotted vertical line in Figure 2(d)) may deviate from the data because the model  
267 occupancy was not produced by fitting directly to hospital occupancy data, but by calcu-  
268 lating net change in hospital occupancy relative to the forecast date from simulated daily  
269 admissions and discharges. Therefore, unlike cases and admissions, accuracy in modelled  
270 occupancy tends to decrease the further backwards in time you go relative to the forecast  
271 date. However, this is not important for the purposes of forecasting.

272 In order to assess forecast performance over time, Figure 3 shows the full time series of data  
273 alongside the results of the forecast that was generated between 15 and 21 days previously  
274 (data available on the third Sunday prior). The accuracy of the forecast 15-21 days ahead  
275 was variable but the large majority of data points fell within the 90% prediction interval.  
276 The most notable deviation is that the forecast overestimated the height of the peak that  
277 occurred in December 2022. This may be partly explained by a drop-off in testing and  
278 reporting of cases during the Christmas summer holiday period, as indicated by wastewater  
279 surveillance [32], and other holiday-related effects on transmission rates. The forecast also  
280 overestimated hospitalisations during this period, but to a lesser extent than it overestimated  
281 cases. Accurately predicting the peak of a wave is known to be a difficult problem in epidemic  
282 forecasting [33], and other models have suffered from similar problems [15, 17].

283 Forecast skill was generally higher for a shorter time horizon. For example, in the 7-day  
284 ahead forecast (see Supplementary Figures S3–S4), the prediction intervals were more tightly  
285 focused around the subsequent data in most cases compared to the 21-day ahead forecast.  
286 In general, the CRPS increased with time (Figure 4a), indicating that forecast accuracy  
287 deteriorated as the time horizon was extended. For a 3-week time horizon, the mean CRPS  
288 on log transformed data was approximately 0.25 for cases and admissions and around 0.17  
289 for occupancy. The admissions forecast was positively biased, particularly at short time  
290 horizons, whereas the occupancy forecast was negatively biased although less strongly (Figure  
291 4b). The cases forecast was close to unbiased.

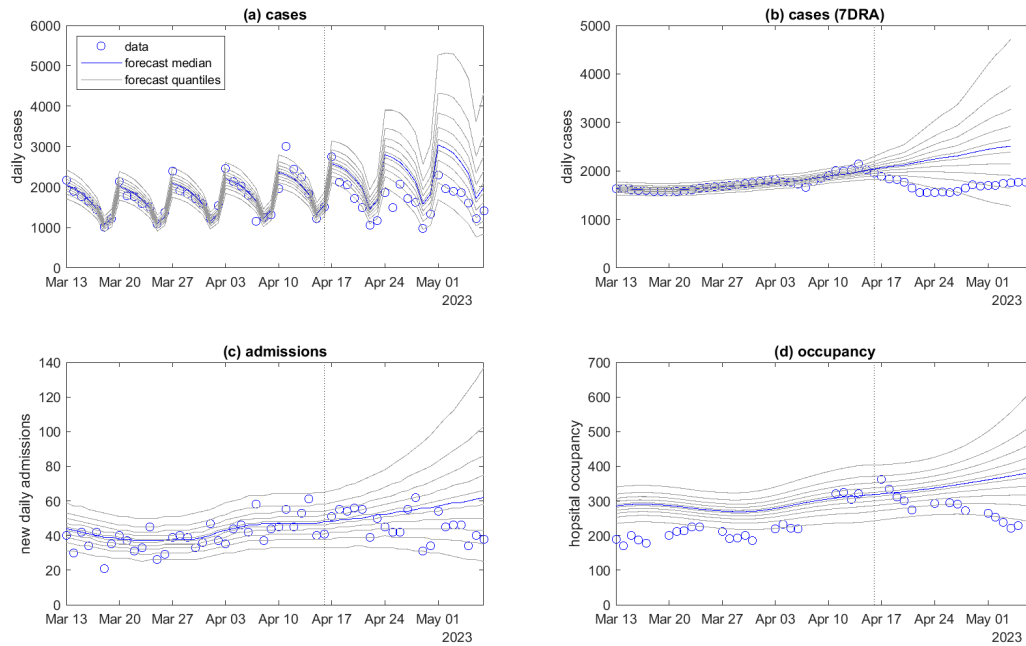


Figure 2: Model fitted to data up to 16 April 2023 (vertical dotted line), projected forwards in time for 21 days and compared to subsequently available data available up to 7 May 2023 for: (a) new daily cases; (b) smoothed daily cases (seven-day rolling average); (c) new daily hospital admissions; (d) hospital occupancy. The day-of-the-week effect is visible in panel (a) for reported daily cases. Blue curve is the median and grey curves are the 5<sup>th</sup>, 15<sup>th</sup>, ..., 85<sup>th</sup>, 95<sup>th</sup> percentiles of  $M = 10^5$  particles.

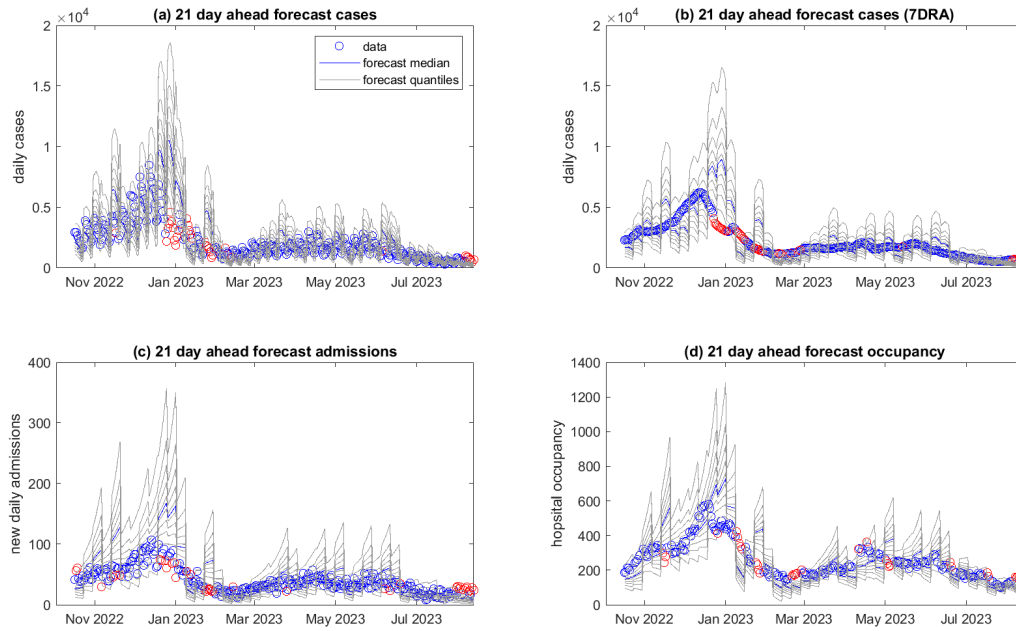


Figure 3: 21-day ahead forecast performance. Model results generated from data supplied at one of a series of weekly time points from 2 October 2022 to 23 July 2023, compared to actual data for the period 15-21 days subsequent to the date the data was supplied for: (a) new daily cases; (b) smoothed daily cases (seven-day rolling average); (c) new daily hospital admissions; (d) hospital occupancy. Testing data was supplied on 20 August 2023 (i.e. 4 weeks subsequent to the last forecast). Weekly discontinuities in the forecasts are because each 7-day block represents a forecast generated from data supplied on a different date. Blue curve is the median and grey curves are the 5<sup>th</sup>, 15<sup>th</sup>, ..., 85<sup>th</sup>, 95<sup>th</sup> percentiles of  $M = 10^5$  particles. Data points outside the 5<sup>th</sup>–95<sup>th</sup> percentile range of the forecast are shown in red.

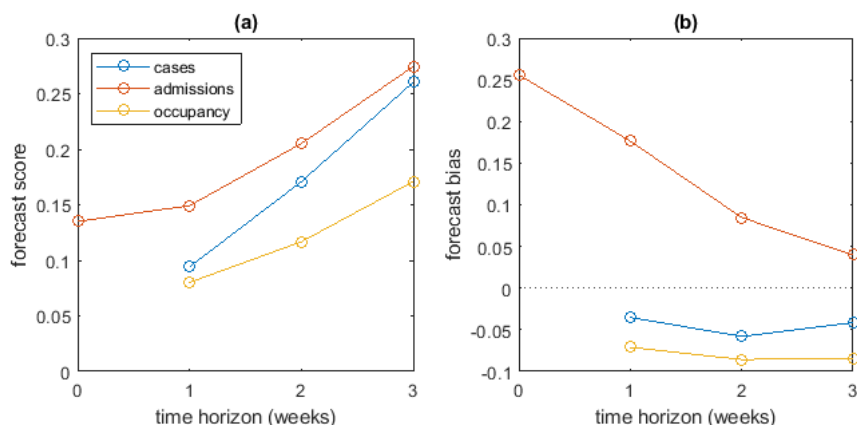


Figure 4: Forecast performance quantified by (a) continuous ranked probability scores (CRPS), and (b) bias, for forecasts up to 0, 1, 2 and 3 weeks ahead. Model results generated from data supplied at one of a series of weekly time points from 2 October 2022 to 23 July 2023, and tested against data supplied on 20 August 2023. Smaller scores indicate more accurate forecasts, and values of bias closer to zero indicated less biased forecasts.

## Discussion

292

293 Near-term forecasting of infectious disease activity and consequent demand for acute health-  
294 care can support situational awareness, planning and public health response [7]. We have  
295 developed a method for forecasting Covid-19 cases, hospital admissions and hospital oc-  
296 cupancy based on Aotearoa New Zealand’s unique disease surveillance and data collection  
297 systems. The method couples a semi-mechanistic model for disease transmission to forecast  
298 cases with Gaussian process regression models for the time-varying case hospitalisation ratio.

299 We have demonstrated that the model provides useful forecasts by benchmarking against  
300 subsequently observed data up to 21 days ahead. The forecasting tool has been opera-  
301 tionalised by Te Whatu Ora Health New Zealand in 2023 to provide weekly national and  
302 regional level forecasts in real-time. Our method is a useful component of health system  
303 capacity planning and response to Covid-19. It is also an important step towards devel-  
304 opment of more sophisticated situational awareness and forecasting capability in Aotearoa  
305 New Zealand for other infectious diseases and future pandemic threats.

306 Strengths of our model include that it is specifically designed to use New Zealand’s unique



307 Covid-19 data streams, including linked unit record data on date of symptom onset and case  
308 report and, where applicable, date of hospital admission and length of stay. This data allowed  
309 us to empirically estimate the distribution of onset-to-report time, report-to-admission time  
310 and age-specific length of hospital stay. The model accounts for known lags in the reporting  
311 of hospital admissions for Covid-19, but uses up-to-date data on reported cases in each age  
312 group to improve accuracy of hospitalisation forecasts. Forecasts performed reasonably well  
313 when benchmarked against subsequently observed, out-of-sample data.

314 Numerous variables affect the age-specific case hospitalisation ratio (CHR), such as vaccine  
315 coverage, rates of prior infection, comorbidities and case ascertainment (which affects the  
316 denominator of the ratio) [16]. Our method avoids the need for assumptions about the  
317 effects of these variables by taking an empirical approach to estimating the age-specific case  
318 hospitalisation ratio from recent data. This is reasonable because, although the variables  
319 affecting CHR will vary over time, they will generally vary slowly relative to the typical  
320 forecasting time horizon of 1-3 weeks.

321 The model has several important limitations. It assumes that, over the forecasting time  
322 horizon, the effective reproduction number follows a simple random walk. This ignores  
323 mechanisms that may systematically affect transmission dynamics (e.g. depletion of the  
324 susceptible population, changes in contact patterns) meaning it is not suitable for forecasting  
325 more than a few weeks ahead, and cannot provide any insight into the reasons for changes  
326 in transmission patterns or the effects of possible interventions.

327 Abrupt changes in case ascertainment, for example as the result of a policy change or a  
328 change in access to testing, would invalidate the forecast for a period of time until the time  
329 window used for estimating the CHR falls inside the new case ascertainment regime. The  
330 same would apply if there was an abrupt change in clinical severity, for example due to rapid  
331 takeover of a new variant. Other than a temporary drop in case ascertainment during the  
332 2022-2023 holiday period [32], there is no evidence of these issues arising during the study  
333 period of October 2022 to July 2023. However, there was subsequently an abrupt drop in  
334 case ascertainment following the lifting of the government isolation mandate for confirmed  
335 cases of Covid-19 on 14 August 2023.

336 We have applied and tested the model in Aotearoa New Zealand during a period in which

337 the Omicron variant of SARS-CoV-2 was dominant, there were limited non-pharmaceutical  
338 interventions, and increasing levels of hybrid immunity [1, 2, 34]. Application of the model  
339 in other contexts, such as in an immune naive population or during periods of intense non-  
340 pharmaceutical interventions or behavioural change, would likely require significant model  
341 adaptation and recalibration.

342 Future improvements of the model could incorporate wastewater surveillance data as an  
343 independent measure of prevalence [32] and more mechanistic transmission assumptions, for  
344 example to account for the accumulation of population immunity during a wave or following  
345 a vaccine rollout.

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