

1 Supplementary Material: Modelling of the COVID-19 protection
2 framework to manage the Delta variant of SARS-CoV-2 in Aotearoa New
3 Zealand
4

5 This appendix includes a detailed description of the model used for the B.1.617.2 (Delta) variant of
6 SARS-CoV-2 in the New Zealand population. We model transmission of SARS-CoV-2 in the community
7 using a stochastic age-structured branching process model (Steyn et al., 2022) in a partially vaccinated
8 population. The model is parameterised to represent the Delta variant, which at the time the
9 modelling was undertaken was the dominant variant globally and in New Zealand. Infected individuals
10 are categorised as either clinical or subclinical, with the clinical fraction increasing with age. Subclinical
11 individuals are assumed to be $\tau = 50\%$ as infectious as clinical individuals (Byambasuren et al., 2020;
12 Davies et al., 2020). Clinical individuals are assigned a symptom onset time which is Gamma distributed
13 from exposure time with mean 5.5 days and s.d. 2.3 days (Lauer et al., 2020). In the absence of
14 interventions, we assume generation times follow a Weibull distribution with mean 5.05 days and s.d.
15 1.9 days (Ferretti et al., 2020). All parameter values used in our model can be found in Supp. Tables
16 S1, S2 and S3.

17 This appendix also includes an extensive sensitivity analysis on several of the assumed model
18 parameters. Results of the sensitivity analysis can be found in Supp. Table S6 and Supp. Figure S2.

19

20 *Test-trace-isolate-quarantine system model*

21 A test, trace, isolate, quarantine (TTIQ) system provides an additional reduction in transmission. We
22 assume that, independently of contact tracing, the probability that an infected individual is confirmed
23 as a case a result of symptom-triggered testing and test sensitivity is 45% for clinical individuals and
24 0% for subclinical individuals. There is a delay between symptom onset and detection that is assumed
25 to be exponentially distributed with mean 4 days. We assume that the detection rate for clinical
26 individuals is the same for vaccinated and non-vaccinated individuals and across all age groups. Once
27 an infection is detected, the individual is assumed to be immediately isolated, resulting in an 80%
28 transmission reduction. Some transmission may still happen within the household and isolation
29 compliance is not perfect, hence we don't model isolation as 100% effective in reducing onward
30 transmission. Contact tracing parameters are dependent on the number of daily cases. If the seven-
31 day rolling average number of daily detected cases remains below 100 cases per day (contact tracing
32 capacity) for 12 consecutive days, a proportion $p_{trace} = 0.7$ of secondary infections of a confirmed

33 case are identified via contact tracing and quarantined with a mean of 3 days from detection of the
 34 index case. This applies to clinical and subclinical contacts. If the number of daily detected cases
 35 exceed the contact tracing capacity, no secondary infections can be traced and quarantined (although
 36 they may still be detected as a result of symptom-triggered testing). Individuals in quarantine (i.e.
 37 asymptomatic or pre-symptomatic traced contacts) are assumed to have a 50% reduction in
 38 transmission. Individuals in isolation (i.e. confirmed cases and symptomatic traced contacts) are
 39 assumed to have an 80% reduction in transmission.

40 In our results, we report the percentage reduction in transmission as a result of TTIQ. We calculate
 41 this as the relative reduction in the reproduction number of individual i as a result of quarantine and
 42 isolation:

$$TTIQ_{eff,i} = 1 - (1 - c_{quar})w_{quar,i} + (1 - c_{iso})w_{iso,i}$$

43 averaged over all infected individuals, where $w_{quar,i}$ and $w_{iso,i}$ are the fraction of the transmission
 44 kernel (the probability density function of the number of infection events required to link a pair of
 45 cases) that falls in the quarantine and isolation period respectively for individual i .

46

47 *Age-structured transmission model*

48 The stochastic model tracks the number of infections in the community. The population is divided into
 49 15 five-year age bands, plus an over-75-year-old age band. The relative contact rate within each and
 50 between age groups are defined by a matrix \hat{C} as in (Steyn et al., 2022). A next-generation matrix
 51 ($NGM_{i,j}$) defines the average number of individuals in group i that will be infected by a single
 52 infectious individual in group j over their whole infectious period given a fully susceptible population:

$$NGM_{i,j} = Uu_i M_{ji} [p_{clin,j} + \tau(1 - p_{clin,j})]$$

53 where M is the contact matrix describing mixing rates between age groups (Steyn et al., 2022), u_i
 54 is the relative susceptibility to infection of age group i , $p_{clin,j}$ is the fraction of infections in age group j
 55 that are clinical, and τ is the relative infectiousness of subclinical individuals. The basic reproduction
 56 number of the age-structured model is the dominant eigenvalue of the next generation matrix,
 57 denoted $R_0 = \rho(NGM)$. In model simulations, the value of the constant U is chosen to give the
 58 desired value of R_0 . We assume $R_0 = 6.0$, approximately representing the Delta variant of SARS-
 59 CoV-2 (Kang et al., 2021; Zhang et al., 2021).

60 The number $\lambda_{i,j}^u(t)$ of unvaccinated people in age group j and the number $\lambda_{i,j}^v(t)$ of vaccinated people
 61 in age group j who are infected by clinical individual l between time t and $t + \delta t$ are a Poisson
 62 distributed random variables with respective means:

$$\lambda_{i,j}^u(t) = Y_l F_l(t) \left(\int_t^{t+\delta t} w(t' - t_{inf,l}) dt' \right) NGM_{j,a_l}^{clin} (1 - V_l e_T) s_j^u(t) \quad (1)$$

$$\lambda_{i,j}^v(t) = Y_l F_l(t) \left(\int_t^{t+\delta t} w(t' - t_{inf,l}) dt' \right) NGM_{j,a_l}^{clin} (1 - V_l e_T) (1 - e_i) s_j^v(t)$$

65

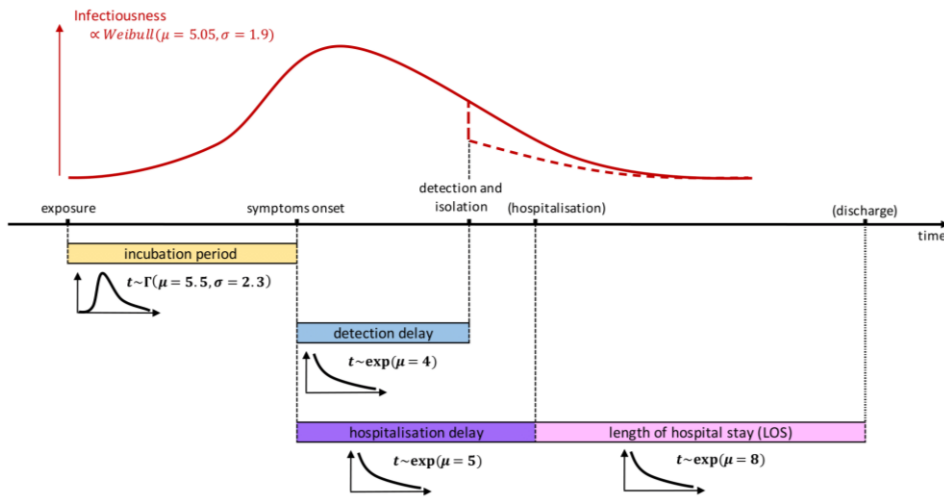
66 where:

- 67 • Y_l is a gamma distributed random variable with mean 1 and variance $1/k$ representing
68 individual heterogeneity in transmission (Lloyd-Smith et al., 2005). We set $k = 0.5$ which
69 represents a moderate level of over-dispersion consistent with estimates for SARS-CoV-2
70 transmission patterns (James et al., 2021; Riou & Althaus, 2020).
- 71 • $F_l(t)$ represents the effect of quarantine or isolation on the transmission rate of individual
72 l at time t , and is equal to 1 if individual l is not in quarantine/isolation at time t , equal to
73 $c_{quar} = 0.5$ if individual l is in quarantine, and equal to $c_{isol} = 0.2$ if individual l is in
74 isolation.
- 75 • $w(\tau)$ is the probability density function of the assumed generation time distribution and $t_{inf,l}$
76 is the time individual l was infected.
- 77 • $NGM_{j,a_l}^{clin} = U u_j M_{a_l,j}$ is the next generation matrix for clinical individuals and a_l is the age
78 group of individual l .
- 79 • V_l is an indicator variable that is equal to 1 if individual l is fully vaccinated at the time they
80 became infected and 0 otherwise. $s_j^u(t)$ and $s_j^v(t)$ are the fractions of age group j that are
81 unvaccinated and fully vaccinated respectively and have not previously been infected at time
82 t .
- 83 • e_i and e_T are vaccine effectiveness against infection and against transmission given infection
84 parameters, presented in Supplementary Table S1

85

86 The expressions for $\lambda_{i,j}(t)$ above are multiplied by τ if individual l is subclinical. Note that the factor
87 Y_l means that, in the absence of control measures, the total number of people infected by a randomly
88 selected individual has a negative binomial distribution with mean R_0 and variance $R_0(1 + R_0/k)$
89 (Lloyd-Smith et al., 2005)

90 At each daily time step, the susceptible compartments $s_j^u(t)$ and $s_j^v(t)$ are depleted according to the
91 number of new infections that occurred in that compartment. Prior infection is assumed to provide
92 complete immunity against re-infection for the duration of the simulation.



93

94 **Supplementary Figure S1** Timeline showing infectiousness of a case (i.e. probability of transmitting
 95 the virus to a contact) as a function of time since infection. Infectiousness is modelled using a
 96 Weibull distribution (see Equation (1) and Supp. Table S2) with mean 5.05 days and standard
 97 deviation 1.9 days. Case detection through a positive test and isolation happen at the same time.
 98 After isolation, infectiousness is reduced to a lower level (red dashed curve, see Supp. Table S2).
 99 Subclinical infections are not isolated and follow the shape of the blue curve throughout, but with a
 100 lower overall probability of transmission. Note that this diagram does not show the possibility of
 101 quarantine through contact tracing, which would also reduce infectiousness.

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104 *Hospitalisation and fatality model*

105 Age-stratified hospitalisation rates are as in (Herrera-Espósito & de los Campos, 2021) with an
 106 additional hazard ratio of 2.26 applied to represent the increased severity of the Delta variant relative
 107 to the ancestral strain of SARS-CoV-2 (Twohig et al., 2022). Fatality rates are based on those of
 108 (Herrera-Espósito & de los Campos, 2021), adjusted by an odds ratio of 2.32 for Delta (Fisman & Tuite,
 109 2021) (Supp. Table S3). Clinical individuals in age group i with 2 doses of the vaccine are assumed to
 110 require hospitalisation with probability $(1 - e_D) p_{hosp,i} / p_{clin,i}$ where e_D is the vaccine effectiveness
 111 against severe disease in breakthrough infections (Supp. Table S1), $p_{hosp,i}$ is the infection to
 112 hospitalisation ratio for unvaccinated people in age group i (Supp. Table S3), and $p_{clin,i}$ is the fraction
 113 of infections in age group i that are clinical. The time between symptom onset and hospitalisation is
 114 assumed to be exponentially distributed with mean 5 days. The length of hospital stay is assumed to

115 be exponentially distributed with mean 8 days. Hospitalised cases in age group i die with probability
 116 $IFR_i/p_{hosp,i}$ where IFR_i is the infection fatality ratio for unvaccinated cases in age group i .

117

118 *Vaccination coverage and effectiveness*

119 Vaccine effectiveness assumptions are as shown in Supp. Table S1. All vaccinated individuals have an
 120 overall transmission reduced by $1 - (1 - e_I)(1 - e_T) = 85\%$ and an overall probability of developing
 121 severe disease reduced by $1 - (1 - e_I)(1 - e_D) = 94\%$. We use a leaky vaccine model as opposed
 122 to an all-or-nothing vaccine model, where a proportion e_I of vaccinated individuals are completely
 123 immunised and a proportion $1 - e_I$ are completely susceptible (Moore et al., 2021). Reality may be
 124 somewhere between these idealised models (i.e. there may be some individual heterogeneity in the
 125 level of protection provided by the vaccine but not as extreme as all-or-nothing). The all-or-nothing
 126 and the leaky vaccine model behave similarly when the proportion of the population with immunity
 127 from prior infection is relatively small. Waning of immunity from prior infection is ignored.

128

129 **Supplementary Table S1.** Vaccine effectiveness parameters against Delta for
 130 the Pfizer-BioNTech vaccine after 2 doses. Source: (*Public Health England,*
 131 *2021*)

Parameter	Value
Effectiveness against infection (e_I)	70%
Effectiveness against transmission given infection (e_T)	50%
Effectiveness against severe disease given infection (e_D)	80%
Implied overall transmission reduction	85%
Implied overall protection against severe disease	94%

132

133

134

135 **Supplementary Table S2.** Other parameter values used in the “baseline” scenario of our model.

Parameter	Value
Reproduction number excluding effects of immunity	$R_0 = 6.0$
Incubation period	Mean 5.5 days, s.d. 2.3 days

Generation interval	Mean 5.05 days, s.d. 1.9 days
Relative infectiousness of subclinical individuals	$\tau = 0.5$
Heterogeneity in individual reproduction number	$k = 0.5$
Probability of detection for clinical individuals	$p_{test} = 0.45$
Probability of a contact of a confirmed case being traced	$p_{trace} = 0.7$
Relative transmission rate for individuals in quarantine	$c_{quar} = 0.5$
Relative transmission rate for individuals in isolation	$c_{isol} = 0.2$
Time from symptom onset to isolation	Mean 4.0 days, s.d. 4.0 days
Time from case detection to quarantine of contacts	Mean 2.0 days, s.d. 1.2 days
Time from symptom onset to hospital admission	Mean 5.0 days, s.d. 5.0 days
Length of hospital stay	Mean 8.0 days, s.d. 8.0 days

136

137

138 **Supplementary Table S3.** Age-specific parameter values used in the “baseline” scenario of our
 139 model.

Age (yrs)	0-4	5-9	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75+
2 nd dose vax cov* (%)	0	0	62	88	83	84	90	91	93	90	93	92	94	95	96	96
Pr(clinical) (%)	54.4	55.5	57.7	59.9	62	64	65.9	67.7	69.5	71.2	72.7	74.2	75.5	76.8	78	80.1
Pr(hosp) (%)	0.19	0.29	0.41	0.61	0.88	1.26	1.84	2.69	3.8	5.56	8.17	11.37	16.15	22.17	30	48.97
Pr(death) (%)	8E-04	0.002	0.003	0.01	0.01	0.02	0.05	0.09	0.17	0.35	0.67	1.29	2.52	4.74	8.81	26.65
Susceptibility**	0.46	0.46	0.45	0.56	0.8	0.93	0.97	0.98	0.94	0.93	0.94	0.97	1	0.98	0.9	0.86
Popn (1000s)	306	327	335	315	337	378	380	338	311	328	329	326	295	251	217	339

* New Zealand’s 1st dose vaccination coverage as of 3rd November 2021, scaled up to obtain 90% national coverage

**Susceptibility for age group i is stated relative to susceptibility for age 60-64 years (Davies et al., 2020).

Age-dependent rates of clinical disease are based on (Hinch et al., 2021).

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144 *Traffic lights trigger points*

145 Supp. Table S4 presents the average number of hospital beds occupied when a given trigger point for
 146 the number of cases is met can be calculated as a model output, which enables the trigger points for
 147 the low, medium and high tolerance scenarios to be directly compared.

148

149 **Supplementary Table S4.** Trigger criteria used to raise/lower traffic light settings for low, medium,
 150 and high tolerance outbreak management responses (black text), together with the average model
 151 output number of hospital beds occupied (red text) at the time when the corresponding trigger for
 152 the number of cases was met. The high tolerance response uses hospitalisations as the trigger to move
 153 between traffic settings, whereas the other responses use reported cases. These results are provided
 154 to enable direct comparison of the criteria for moving between traffic light settings,
 155 *The “very low tolerance” triggers were only used for the border and community seed sensitivity
 156 analysis.

157

			Very low tolerance*	Low tolerance	Medium tolerance	High tolerance
escalation criteria	→ O	cases	10	50	200	
		hosp beds	4	20	70	100
	→ R	cases	25	100	400	
		hosp beds	9	40	140	200
	→ E	cases	50	300	1200	
		hosp beds	20	110	430	600
relaxation criteria	→ G	cases	0	0	100	
		hosp beds	0	0	40	50
	→ O	cases	10	75	300	
		hosp beds	4	30	110	150
	→ R	cases	30	200	800	
		hosp beds	10	70	290	400

158

159 **Derivation of “toy border model”**

160

161 Suppose there is a pre-defined tolerance y^* for prevalence $y(t)$ of active community infections. When
 162 $y(t)$ rises above y^* , stringent control measures are imposed resulting in an effective reproduction
 163 number of $R_2 < 1$. When $y(t)$ falls below some proportion α of the tolerance y^* , control measures
 164 are relaxed and the effective reproduction number is $R_1 > 1$. The parameter $\alpha < 1$ is needed to avoid
 165 instantaneous alternation between escalation and relaxation of control measures; however we will
 166 take the limit $\alpha \rightarrow 1$ to derive an idealised expression for the average proportion of time spent with
 167 control measures imposed.

168 In a standard SIR modelling framework, the effective reproduction number R is related to the epidemic
 169 growth rate r via

170
$$R = 1 + rg, \quad (2)$$

171 where g is the mean generation interval (Wallinga & Lipsitch, 2007).

172 If there are b additional infections per unit time introduced into the community via the border, then
 173 prevalence $y(t)$ is governed by the differential equation

174
$$\frac{dy}{dt} = ry + b. \quad (3)$$

175 During periods when control measures are relaxed, the prevalence at the start of the period is αy^* by
 176 definition, and subsequently grows according to

177
$$y(t) = (\alpha y^* + b/r_1) e^{r_1 t} - b/r_1 \quad (4)$$

178 The time taken for prevalence to rise above the threshold y^* for imposition of control measures is
 179 therefore

180
$$t_1 = \frac{1}{r_1} \ln \left(\frac{y^* + b/r_1}{\alpha y^* + b/r_1} \right).$$

181 During periods when control measures are imposed, the prevalence at the start of the period is y^* by
 182 definition, and subsequently declines according to

183
$$i(t) = (y^* + b/r_2) e^{r_2 t} - b/r_2. \quad (5)$$

184 The time taken for prevalence to fall below the threshold αy^* for relaxation of control measures is
 185 therefore

186
$$t_2 = \frac{1}{r_2} \ln \left(\frac{\alpha y^* + b/r_2}{y^* + b/r_2} \right)$$

187 If α is close to 1, the above expressions for t_1 and t_2 may be written as a Taylor series in $1 - \alpha$:

188
$$t_1 = \frac{(1-\alpha)y^*}{r_1 y^* + b} + O((1-\alpha)^2), \quad (6)$$

189
$$t_2 = -\frac{(1-\alpha)y^*}{r_2 y^* + b} + O((1-\alpha)^2) \quad (7)$$

190

191 Over a sufficiently long time window, the approximate average proportion of time p_{int} spent with
192 control measures in places is therefore

$$193 \quad p_{int} = \frac{t_2}{t_1+t_2} = \frac{r_1+b/y^*}{r_1-r_2}, \quad (8)$$

194 where we have neglected terms of order $(1-\alpha)^2$ and higher.

195 Writing this in terms of reproduction numbers R_1 and R_2 instead of growth rates r_1 and r_2 gives

$$196 \quad p_{int} = \frac{R_1-1+bg/y^*}{R_1-R_2}. \quad (9)$$

197 Note that for this result to be valid requires that $b < (1-R_2)y^*/g$. If $b > (1-R_2)y^*/g$, the
198 large number of border cases means that prevalence will continue to increase above y^* even with
199 control measures in place 100% of the time.

200 Converting the threshold for prevalence y^* to an approximate corresponding threshold i^* for
201 incidence of new infections per day via $y^* = gi^*$ yields the equation in the main text for p_{int} .

202

203 **Sensitivity analysis**

204
 205 In addition to the scenarios presented in the main text, we explore the effects of changing model
 206 assumptions for community case isolation, the probability of case detection, the effectiveness of
 207 control measures, vaccine effectiveness, the capacity of the contact tracing system, and the risk of
 208 hospitalisation (Supp. Table S5).

209 From each simulation, we output the number of infections, detected cases, hospital admissions and
 210 deaths, and the time spent under different traffic light settings. All simulations were run for a one year
 211 period and results were averaged over 50 independent simulations of the stochastic model for each
 212 set of parameters.

213
 214 **Supplementary Table S5** Parameters used in the sensitivity analysis

Parameters	Baseline values	Scenarios tested
Comm. cases isolation effectiveness (%)	100	50
Probability of case detection	0.45	0.30
Reduction in transmission (%) at G/O/R/E ¹	10/20/30/60	0/10/20/60
Vaccine effectiveness ($e_i/e_i/e_d^2$)(%)	70/50/80	50/40/80
National vaccination coverage (%)	90	95
Contact tracing	Capacity ³ = 100 cases per day pTrace ⁴ =70	1.No cap, pTrace=70 2.Cap=250 cases per day, pTrace=70 3.Cap=100 cases per day, pTrace=30 4.No contact tracing

215
 216 ¹reduction in transmission a G/O/R/E – Green/Orange/Red and Emergency setting
 217 ² $e_i/e_i/e_d$ – Effectiveness of vaccine against infection/transmission given infection/disease given infection
 218 ³ Capacity – Contact tracing capacity above which no infections are found by contact tracing compared to
 219 70% before capacity is reached.
 220 ⁴ pTrace – percentage of infections found by contact tracing before capacity is reached

221
 222 *Sensitivity analysis of community case isolation effectiveness*
 223 Reducing the effectiveness of case isolation in the community from 100% to 50% reduces the
 224 effectiveness of TTIQ and leads to increased transmission. As a result, the trigger points for escalating
 225 control measures are met sooner (and those for relaxing are met later), increasing the amount of time
 226 spent under more stringent settings (Supp. Fig. S2c, Supp. Table S6.C). For example, in a low tolerance

227 response, the time spent in the emergency setting increases from 15% to 21% (one extra month in the
228 emergency setting) relative to the baseline scenario. The increase in time in the emergency setting is
229 not as profound for the medium and high tolerance response. The number of infections,
230 hospitalisations and deaths are higher than in the baseline scenario. However, this increase is offset
231 to a large extent by the more stringent public health response described above, keeping the epidemic
232 to pre-defined tolerances.

233

234 *Sensitivity analysis of contact tracing system capacity*

235 Under a low tolerance response, increasing contact tracing capacity from 100 cases to 250 cases per
236 day increases the effect of TTIQ on transmission from 8% to 16%, which leads to fewer infections and
237 hospitalisations and much less time spent in emergency setting (Supp. Table S6.G2). However, it has
238 almost no effect on health outcomes or time spent in lockdown under a medium or high tolerance
239 response. This is because the number of cases is almost always above 250 cases per day, so contact
240 tracing is always performing at the reduced level in these scenarios. With no assumed limit to contact
241 tracing capacity, there is a clear decrease in the number of infections and hospitalisations. Under a
242 low and medium tolerance response, the time spent in red and emergency slightly increases relative
243 to the baseline scenario as a higher proportion of infections are detected (about 50% as compared to
244 30% under the baseline setting) (Supp. Fig. S2g, Supp. Table S6.G1). Under a high tolerance response,
245 the time spent in emergency decreases, but the time spent in red setting increase.

246 Reducing the proportion of contacts of a confirmed case who are via contact tracing from 70% to 30%
247 has almost no effect on infections, hospitalisations or time spent in emergency setting relative to the
248 baseline scenario (Supp. Table S6.G3) under the low and medium response. This is because the
249 number of cases quickly exceeds the contact tracing capacity (set to 100 cases) in all scenarios. It
250 resulted in more infections, cases, and hospitalisations, but had no effect on time spent in emergency
251 setting under a high tolerance response.

252

253 *Sensitivity analysis of probability of case detection*

254 For the low and medium tolerance scenarios, a reduction in the probability of case detection, i.e. the
255 probability of individuals seeking a test and testing positive, from 45% to 30% corresponds to a slower
256 response to the increase in cases and a delayed move to higher traffic lights, leading to more
257 infections, hospitalisations and deaths over the year (Supp. Fig. S2d, Supp. Table S6.D).

258 Interestingly, reducing the probability of case detection resulted in fewer infections and deaths under
259 a high tolerance strategy than under the medium tolerance strategy. Essentially, the high tolerance
260 strategy became more effective at controlling the spread of COVID-19 because the higher tolerance

261 scenario uses hospital beds as a metric of demand on the healthcare system (as opposed to case
262 numbers in the low and medium tolerance response), which is not as affected by the lower probability
263 of case detection. This suggests that the low and medium tolerance scenario display a higher
264 sensitivity to the probability of case detection.

265

266 *Sensitivity analysis of traffic light control effectiveness*

267 Reducing the effectiveness of public health measures under the different traffic light settings in
268 reducing transmission of the virus leads to a large increase in infections, hospitalisations and deaths
269 (Supp. Fig. S2e, Supp. Table S6.E). The time spent in emergency setting is doubled under a low
270 tolerance response (about one third of the year in emergency setting) and almost tripled under a
271 medium and high tolerance response (2 months in emergency) relative to the baseline parameter
272 settings.

273

274 *Sensitivity analysis of vaccine effectiveness*

275 Reducing vaccine effectiveness against infection from 70% to 50% and against transmission from 50%
276 to 40% causes a more than a threefold increase in hospitalisations and about a twofold increase in the
277 number of deaths (Supp. Fig. S2f, Supp. Table S6.F). The time spent in the emergency setting increases
278 to about half of the year under a medium or high tolerance response, and about two thirds of the year
279 under a low tolerance response.

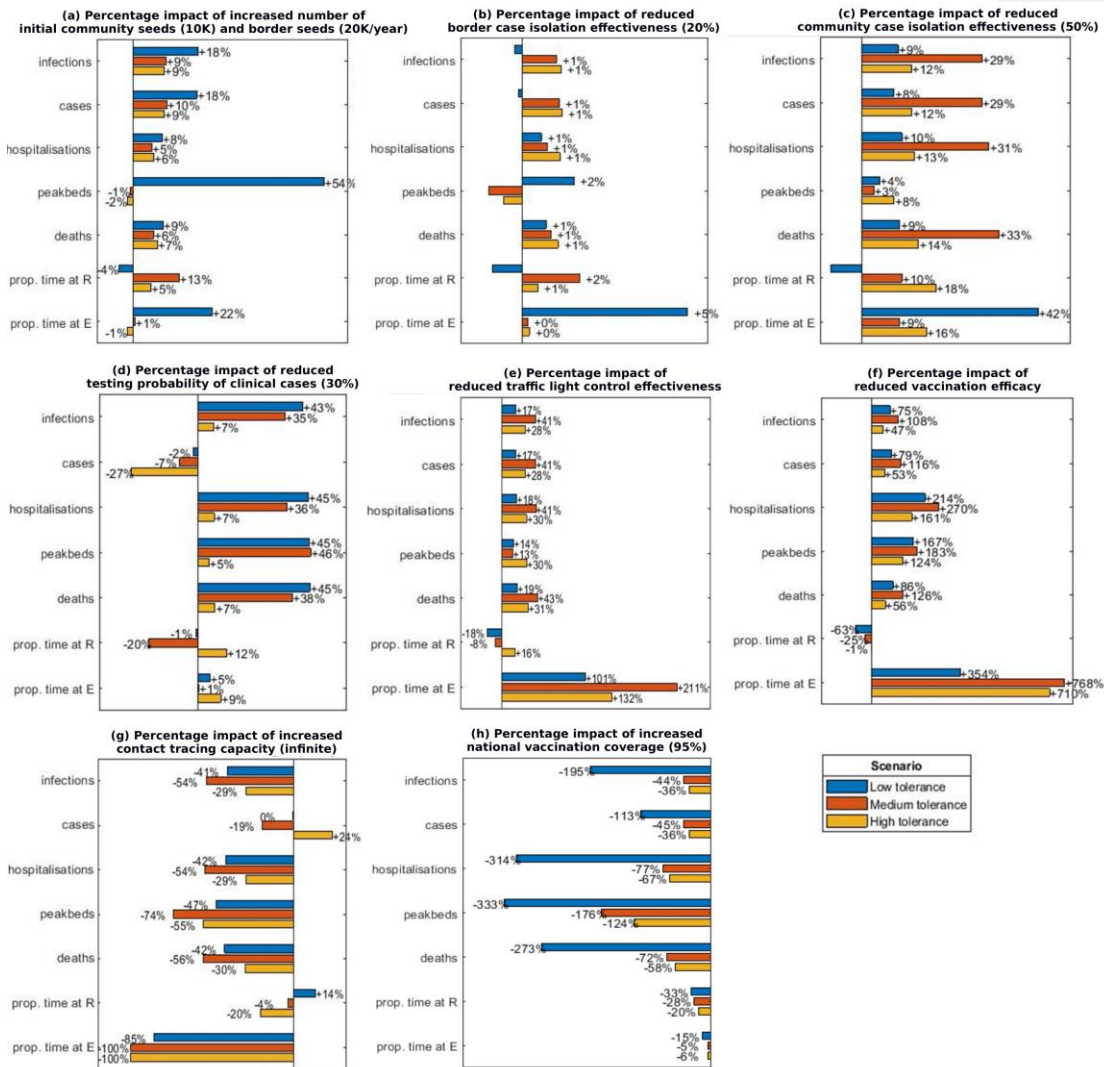
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281 *Sensitivity analysis of vaccination coverage*

282 Increasing the national vaccination coverage from 90% to 95% results in a significant drop in all public
283 health outcomes and in the time spent in the red and emergency settings, with a near two-fold
284 reduction in the number of hospitalisations and deaths (Supp. Fig. S2h, Supp. Table S6.H).

285

286



287
 288 **Supplementary Figure S2** Percentage impact of different model parameter settings compared to the
 289 baseline (Table 2), for the low (blue), medium (red) and high (yellow) tolerance scenarios.

290
 291
 292

293 **Supplementary Table S6:** Median number of infections, detected cases, hospitalisations, peak hospital
 294 occupancy and deaths over a year under the low, medium and high tolerance outbreak management
 295 response for all scenarios tested. The G, O, R, and E columns indicate the median percentage time
 296 spent in each of the traffic light setting (green, orange, red and emergency). The TTIQeff indicate the
 297 reduction in transmission as a result of the test, trace, isolate, quarantine system.

scenario	infections	cases	hospitalisations	peak beds	deaths	G	Y	R	E	TTIQeff
<i>BASELINE SCENARIO</i>										
Very low tolerance	32,000	13,000	400	40	50	0%	3%	82%	15%	12%
Low tolerance	215,000	66,000	2,900	130	410	0%	0%	85%	15%	8%
Medium tolerance	553,000	165,000	7,600	470	1,100	0%	23%	72%	5%	8%
High tolerance	684,000	204,000	9,500	650	1,390	7%	38%	49%	6%	8%
<i>A1. Border cases = 10K</i>										
Very low tolerance	37,000	15,000	400	40	50	0%	0%	78%	22%	12%
Low tolerance	217,000	66,000	2,900	130	400	0%	0%	84%	16%	8%
Medium tolerance	551,000	165,000	7,500	470	1,090	0%	21%	75%	5%	8%
High tolerance	690,000	206,000	9,500	650	1,390	6%	38%	50%	6%	8%
<i>A2. Border cases = 20K</i>										
Very low tolerance	46,000	17,000	400	40	50	0%	0%	61%	39%	11%
Low tolerance	223,000	67,000	2,800	130	400	0%	0%	84%	16%	8%
Medium tolerance	554,000	166,000	7,400	460	1,080	0%	19%	76%	5%	8%
High tolerance	703,000	210,000	9,600	650	1,410	6%	38%	50%	6%	8%
<i>A3. Community seed cases = 10K, border cases = 10K</i>										
Very low tolerance	93,000	32,000	1,100	200	150	0%	0%	75%	25%	10%
Low tolerance	245,000	75,000	3,200	210	450	0%	0%	82%	18%	8%
Medium tolerance	588,000	176,000	8,000	470	1,170	0%	12%	83%	5%	8%
High tolerance	725,000	217,000	9,900	640	1,460	0%	43%	51%	6%	8%
<i>A4. Community seed cases = 10K, border cases 20K</i>										
Very low tolerance	103,000	34,000	1,100	210	150	0%	0%	60%	40%	9%
Low tolerance	257,000	78,000	3,200	200	450	0%	0%	82%	18%	8%
Medium tolerance	604,000	181,000	8,100	470	1,170	0%	12%	83%	5%	8%
High tolerance	747,000	224,000	10,100	640	1,490	0%	44%	51%	6%	8%
<i>B. Low border cases isolation effectiveness (20%)</i>										
Low tolerance	216,000	66,000	2,900	130	420	0%	0%	84%	16%	8%
Medium tolerance	557,000	167,000	7,700	470	1,110	0%	20%	75%	5%	8%
High tolerance	689,000	206,000	9,600	650	1,400	6%	38%	50%	6%	8%

C. Low community cases isolation effectiveness (50%)

Low tolerance	235,000	71,000	3,200	140	450	0%	0%	79%	21%	4%
Medium tolerance	710,000	212,000	9,900	490	1,460	0%	14%	81%	5%	4%
High tolerance	762,000	228,000	10,700	700	1,570	6%	29%	58%	7%	4%

D. Low testing probability (30%)

Low tolerance	308,000	65,000	4,200	190	600	0%	0%	84%	16%	5%
Medium tolerance	745,000	152,000	10,400	690	1,520	2%	35%	59%	5%	5%
High tolerance	725,000	148,000	10,100	680	1,480	6%	32%	55%	7%	5%

E. Low traffic light control effectiveness

Low tolerance	252,000	77,000	3,400	150	490	0%	0%	70%	30%	8%
Medium tolerance	774,000	231,000	10,700	540	1,570	0%	17%	67%	15%	8%
High tolerance	873,000	261,000	12,300	850	1,820	4%	25%	57%	14%	8%

F. Low vaccination effectiveness (50/40/80)

Low tolerance	386,000	118,000	5,300	200	780	0%	0%	32%	68%	8%
Medium tolerance	1,000,000	305,000	14,100	770	2,190	0%	2%	60%	38%	8%
High tolerance	1,000,000	305,000	14,100	1,370	2,220	0%	4%	71%	25%	8%

G1. Unlimited contact tracing capacity

Low tolerance	128,000	66,000	1,700	70	240	0%	1%	97%	2%	17%
Medium tolerance	256,000	133,000	3,500	120	490	0%	29%	71%	0%	18%
High tolerance	481,000	252,000	6,700	290	970	8%	53%	39%	0%	18%

G2. High contact tracing capacity (250)

Low tolerance	136,000	66,000	1,800	80	250	0%	0%	97%	3%	16%
Medium tolerance	555,000	170,000	7,700	470	1,110	0%	24%	72%	5%	8%
High tolerance	679,000	206,000	9,400	650	1,380	8%	39%	48%	6%	8%

G3. Low proportion of contacts traced (30%)

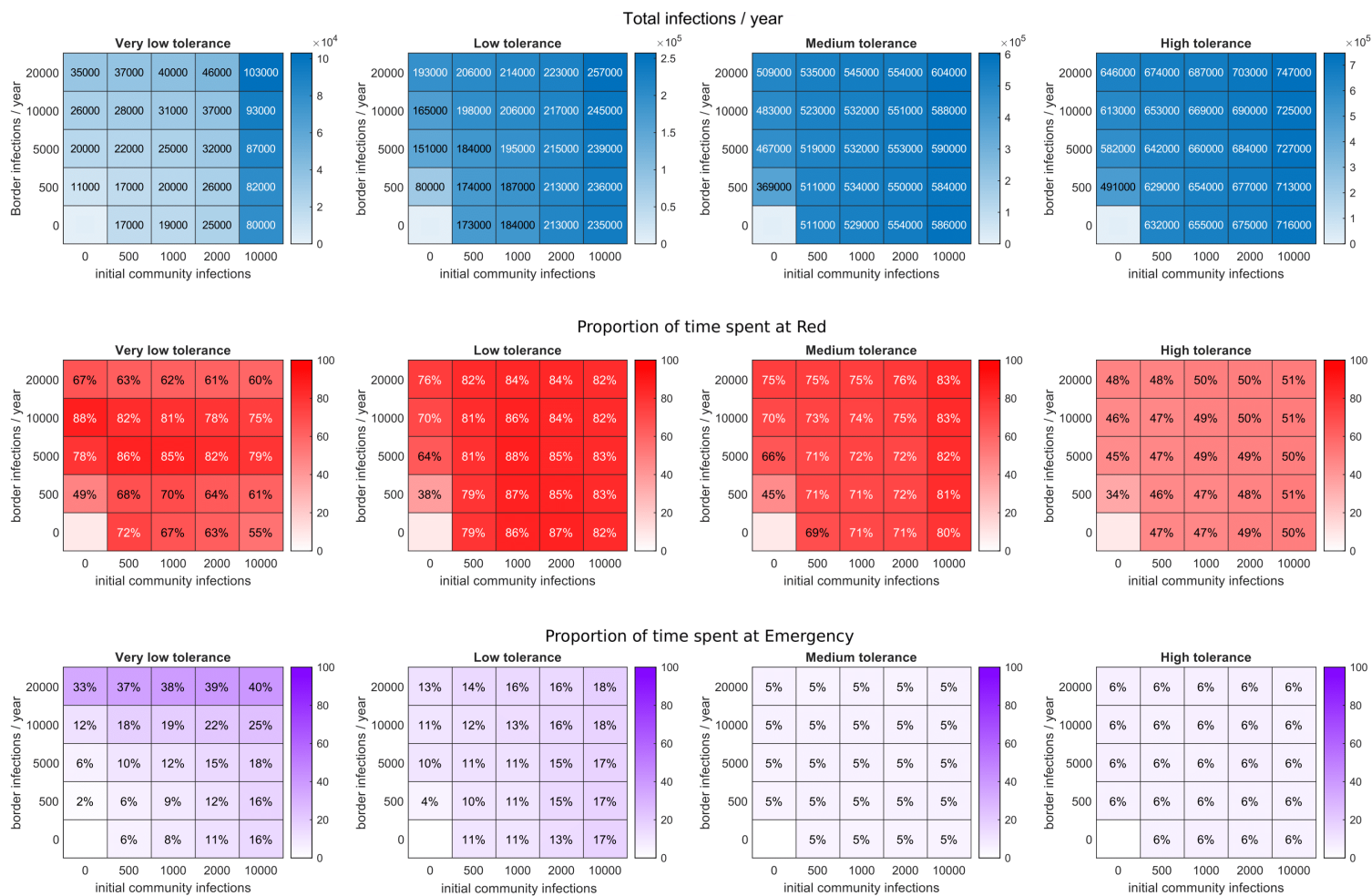
Low tolerance	219,000	66,000	2,900	130	410	0%	0%	84%	16%	8%
Medium tolerance	557,000	166,000	7,700	470	1,110	0%	20%	75%	5%	8%
High tolerance	690,000	206,000	9,600	650	1,410	6%	38%	50%	6%	8%

G4. No contact tracing

Low tolerance	222,000	66,000	3,000	130	420	0%	0%	84%	16%	7%
Medium tolerance	558,000	166,000	7,700	470	1,100	0%	20%	76%	5%	7%
High tolerance	690,000	206,000	9,600	650	1,400	6%	38%	50%	6%	7%

H. High vaccination coverage (95%)

Low tolerance	73,000	31,000	700	30	110	0%	48%	52%	0%	8%
Medium tolerance	383,000	114,000	4,300	170	640	9%	47%	44%	0%	8%
High tolerance	504,000	150,000	5,700	290	880	14%	57%	29%	0%	8%



Supplementary Figure S3 Heatmaps of total infections per year (top row), proportion of time spent in the Red setting (middle row), and proportion of time spent in the Emergency setting (bottom row), for the very low (first column), low (second column), medium (third column), and high (fourth column) tolerance scenarios. Each heatmap was produced through different combinations of initial community seed infections and border infections per year, as described in Table 2.

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