DOHERTY MODELLING –FINAL REPORT TO NATIONAL CABINET

5TH NOVEMBER 2021

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Attachments

Key Messages

Work Package 1 – Modelling to inform review and refinement of public health response measures

- Streamlined and focussed test-trace-isolate-quarantine (TTIQ) processes (supported by PHSMs) will be required for future public health responses to be effective and sustainable
- We have previously shown that case-initiated contact tracing can support timely quarantine in times of system stress
- Reduced contact tracing intensity and differential management of vaccinated individuals will help to ensure sustainable responses as caseloads increase
- Focussed TTIQ with wrap around support will be needed in communities that remain at risk of higher transmission and/or clinical impacts
- Ongoing data collection is advised to enable evaluation of TTIQ responses for situation assessment

Work Package 2 – Optimise vaccination at sub-jurisdictional level

1. First Nations Australians
    - High vaccine coverage can reduce transmission and health impacts in urban and remote communities
    - Reactive vaccination is a useful adjunct to community engaged and led outbreak response
    - Providing access to effective treatments will further promote health outcomes
2. Local Government and small area effects
    - Baseline transmission potential (TP) differs by small area, as do vaccine and PHSM impacts (ability to work from home)
    - Focussed TTIQ and wrap around supports will be needed to constrain TP in high-risk areas and may include additional measures in schools and workplaces
3. Schools
    - Early infection detection and high vaccine coverage markedly reduce outbreak risk
    - Allowing ongoing school attendance for class contacts of a case through a 'test to stay' strategy achieves equivalent outbreak containment to home quarantine and enables face to face learning
    - School based measures will have maximum utility in areas with higher than average transmission
    - Regular screening of students in areas at risk of outbreaks can result in even fewer infections and in-person teaching days lost

Work Package 3 – Review border measures and arrivals pathways

- Vaccination reduces the risk of infected people being released from quarantine into the community, mitigating against shorter duration
- These importations do not materially impact on established epidemics or lead to large outbreaks at the defined Phase C coverage threshold of 80%, when combined with 'low' PHSMs
- *These findings assume consistent vaccine protection and virus characteristics identical to those assumed for the Delta variant (ie no more transmissible, and equally preventable by vaccines)*

Revisions to parameter assumptions

- Mixing, vaccine effectiveness and clinical severity parameters have been updated for this phase of work, based on latest available evidence
- Previous recommendations of 70 and 80% vaccine coverage thresholds for National Plan transition phases remain robust

Background

On 30 July 2021, National Cabinet considered advice from the Doherty Institute and Commonwealth Treasury to inform the National Plan to Transition Australia's National COVID Response (National Plan). The combined modelling/Treasury conclusion was that where an outbreak occurs strict lockdowns were likely to be required to manage outbreaks until completed coverage of 70% or more had been achieved and that a 'low case' strategy was likely to be lower economic cost than managing higher transmission within the community. Additional recommendations of that work were that:

- Ongoing public health test, trace, isolate, quarantine (TTIQ) responses combined with public health and social measures (PHSMs) were critical interventions to achieve this low case strategy as vaccination alone would be insufficient;
- Achievement of vaccine coverage targets at small area level would be critical to ensure equity of program impact, as ongoing outbreaks in undervaccinated populations are reasonably anticipated from international experience;
- Ongoing situational assessment of measured transmission potential and circulating SARS-CoV-2 variants in the Australian population over coming months would allow benchmarking of these hypothetical scenarios to guide real time policy decision making about the transition to Phases B and C of the National Plan.

The consortium was subsequently tasked with a second phase of work to support implementation of the Plan, which was approved by National Cabinet on the 13th August 2021:

- Work Package 1: Modelling to inform review and refinement of public health response measures for optimal utility and sustainability in Phase B and beyond;
- Work Package 2: Optimise vaccination at sub-jurisdictional level, including attention to key populations and risk settings (First Nations, CALD and low SES communities, and schools);
- Work Package 3: Review border measures and arrivals pathways in context of revised risk tolerance.

Ongoing consultation has informed iterative revision of questions and outputs to inform key decisions as the local and international landscape changes. We have also revised several critical parameters as needed, based on emerging evidence from Australia and elsewhere.

Key Findings Work Package 1: Modelling to inform review and refinement of public health response measures for optimal utility and sustainability in Phase B and beyond

Key question: What are the most effective and sustainable strategies for test, trace, isolate, quarantine (TTIQ) to manage COVID as vaccination rates increase in Phase A then Phases B and C to achieve the aim of strong suppression and avoid lockdown requirement?

Through Department of Health-led consultations with the Communicable Diseases Network of Australia (CDNA) and Australian Health Protection Principal Committee (AHPPC), strategies have been identified to simplify and streamline TTIQ responses during the transition into a phase of established community transmission of COVID-19, with increasing caseloads and high vaccine coverage. Models were used to assess risks associated with proposed changes to measures, informing revisions of national guidelines. We have previously shown that case-initiated contact tracing can support timely quarantine in times of system stress. Reduced contact tracing intensity and differential management of vaccinated individuals will further help to ensure sustainable responses. Focussed TTIQ with wrap around support will be needed in communities that remain at risk of higher transmission and/or clinical impacts. Ongoing data collection is advised to enable evaluation of TTIQ responses for situational assessment of transmission potential.

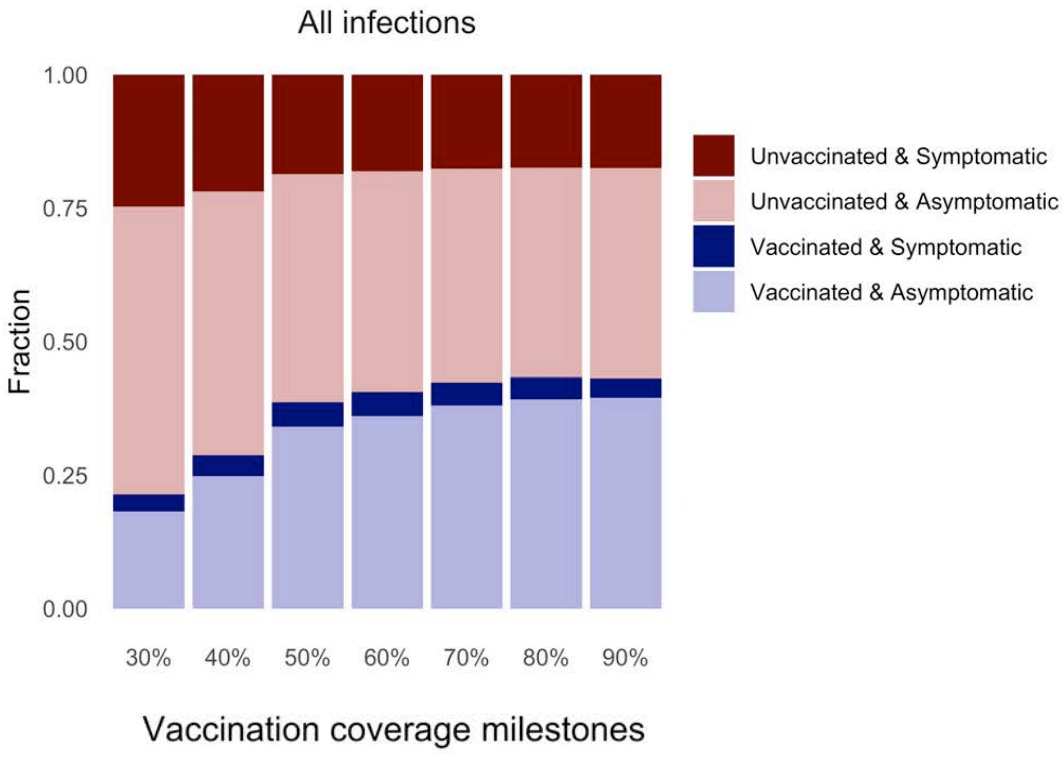
The current report focuses on some key findings and their implications for epidemic control, along with the importance of ongoing evaluation.

When considering streamlining of TTIQ processes for sustainable future responses, it is important to note that the impact of even minor changes in TP on the local epidemiology depends critically on how close TP is to the national strategic objective of maintaining a control threshold of 1. If TP at the population level is very close to 1, even a small change can be sufficient to enable escalation of the local epidemic.

Vaccine coverage and asymptomatic infections

As vaccine coverage increases, the proportion of all infections that occur in vaccinated people will increase, because they will represent a majority proportion of the population (Figure 1.1). Such cases are likely to be less symptomatic and infectious, supporting public health responses and limiting clinical impacts.

Figure 1.1: Proportion of all infections in vaccinated and unvaccinated individuals over time as coverage increase in the population, and related symptomatic fraction



Management of vaccinated cases and contacts

Doherty modelling is supporting risk appraisal to assess the impact on transmission potential (TP) of changing the management of cases and contacts in a highly vaccinated population. Understanding which measures can be safely altered as part of routine practice, and which are the most important to continue in times of system stress, will help to inform reduced intensity of case and contact management during the transition to living with COVID. Options include differential management of vaccinated individuals presenting either as index cases or contacts.

Figure 1.2.1 is an example of a change in routine management of vaccinated cases that has minimal impact on the overall impact of the public health response. Reducing the duration of isolation for vaccinated cases from the current guideline recommendation of 14 days to 7 days contributes only a 1% increase in TP, meaning that such a change poses a very low risk.

Figure 1.2.1 Reduced duration of isolation for vaccinated COVID positive cases. The left panel reports outputs from a simulation model estimating the reduction in transmission potential achieved by isolating fully vaccinated cases for 14 days (left) or 7 days (right). The small 'by eye' difference seen here is confirmed in the rightmost plot, which shows an overall increase in TP of approximately 1% for 7 days.

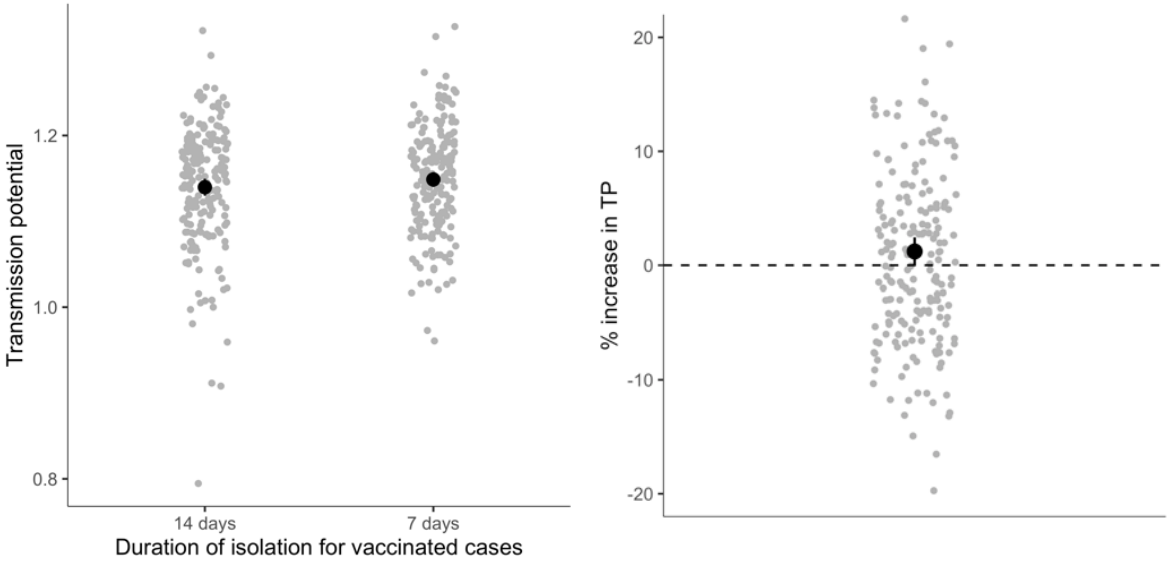


Figure 1.2.2 draws on the experience of NSW during the 2021 outbreak to demonstrate the utility of asking cases to notify their own primary close contacts (PCCs) and asking them to isolate, hastening the time to contact isolation as public health response efforts become less timely under system stress. We have developed a simulation approach to consider the likely impact of this strategy on transmission potential, given some assumptions about compliance and the proportion of contacts that can be ascertained by this means. The model was used to replicate three scenarios for NSW:

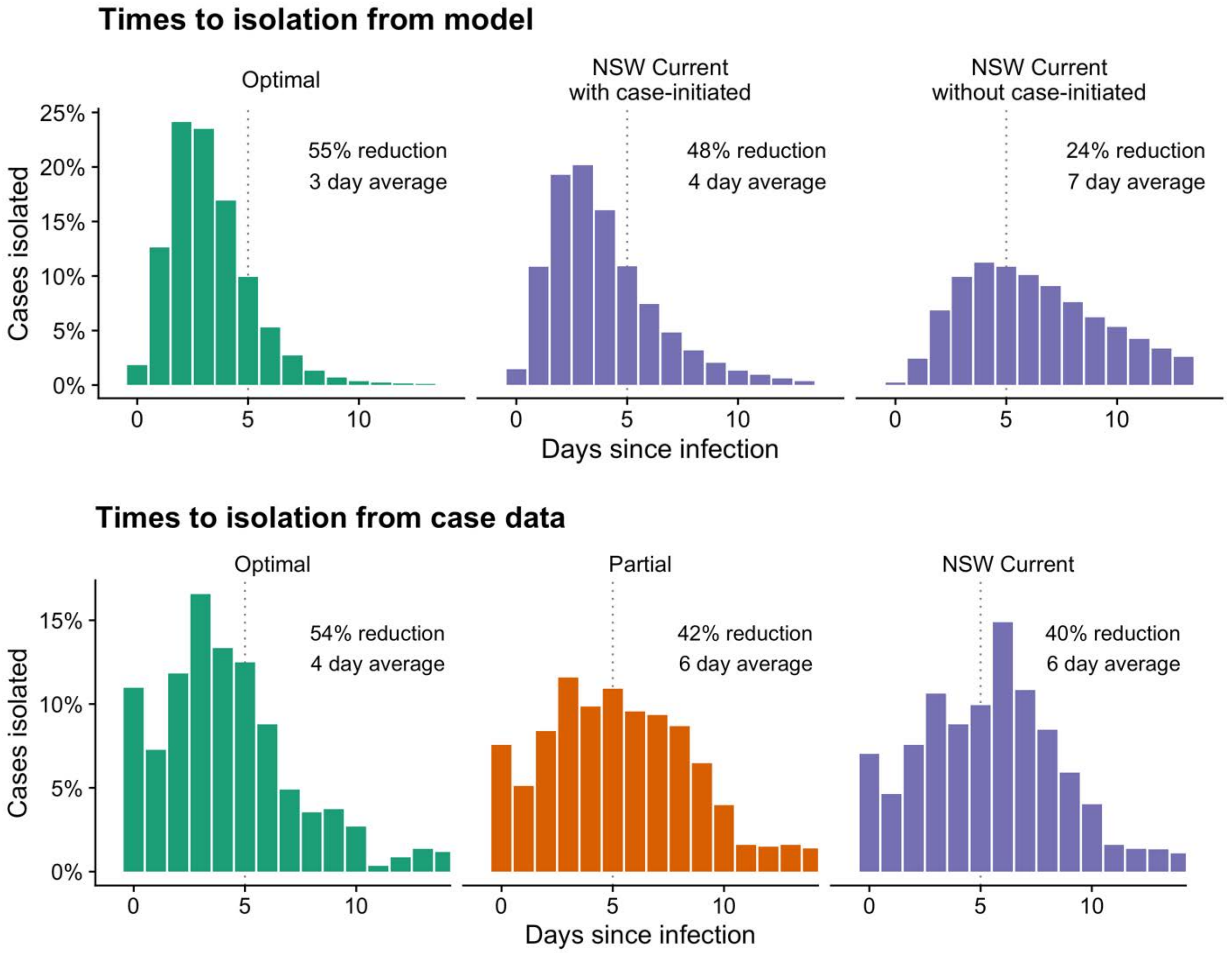
- Optimal TTIQ – the period from July 2020-February 2021 presented in our previous work;
- Current TTIQ – a four-week period commencing August 15 2021, without case initiated tracing;
- Current with case-initiated TTIQ – as above, but assuming 80% of contacts are case-notified.

Optimal and Current scenarios use assumptions about the time from case notification to interview based on data reported by public health units during the time periods above. The interview is the first timepoint at which public health units can identify contacts and ask them to test and quarantine. From these inputs, the model reports time delays to isolation of all cases, including those found through contact tracing.

The left most panel of the upper figure shows that our model can reproduce the TP reduction calculated from observed distributions of times from infection to isolation for 'optimal TTIQ' as estimated in Phase 1 of the National Plan Modelling (54% reduction). The middle panel shows that if we assume a high level of case-initiated contact tracing, NSW should still achieve similar reductions in TP to 'partial TTIQ' as estimated in Phase 1 of the National Plan Modelling (42% reduction). The right most panel shows that the

reported contact tracing delays in NSW would be predicted to result in a much smaller reduction in TP if case-initiated contact tracing (or other strategies to reduce times to isolation) were not in place.

Figure 1.2.2 Case initiated contact tracing – comparison of simulation model outputs (upper panel) with observed times to isolation from case duration during periods of Optimal and Partial TTIQ as defined in previous reporting, compared with NSW observations from mid-August to mid-September 2021



The lower panel of the figure displays estimates of times from infection to isolation from case data. The right most panel (NSW current) shows that between mid-August and mid-September 2021, TTIQ responses in NSW were reducing TP by 40%. This estimate is much higher than would be expected based on public health unit contact tracing alone (right most panel of the 'model' figure). It supports the hypothesis that a substantial proportion of cases did self-identify contacts, who complied with the recommendation to quarantine. *These findings further confirm the effectiveness of TTIQ responses to constrain Delta.*

Ongoing evaluation of the impacts of TTIQ on TP

Assessment of the continuing impact of TTIQ on transmission will be an important component of ongoing weekly situational assessment given its impact on TP in the population. A monitoring system needs to measure the overall impact of TTIQ, as well as the components that underpin system performance, to allow identification of reduced timeliness or completeness of response actions. The overall indicator of system performance is defined as the **TTIQ effect** as shown in Figure 1.3.1, which is the percentage reduction in transmission potential due to TTIQ. This effect is the product of two components: the impact on *detected* infections, and the overall proportion of infections detected (case ascertainment). Figure 1.3.2 demonstrates that our current estimates of TTIQ performance are based on a very high level of infection ascertainment, likely in the order of 90-100%. In future, if the proportion of all infections that can be identified falls substantially because of a higher asymptomatic fraction (as in Figure 1.1) or complacency, the impacts of TTIQ measures on TP will similarly decline.

Figure 1.3.1: Elements required to estimate the TTIQ effect (percentage reduction in TP due to TTIQ).

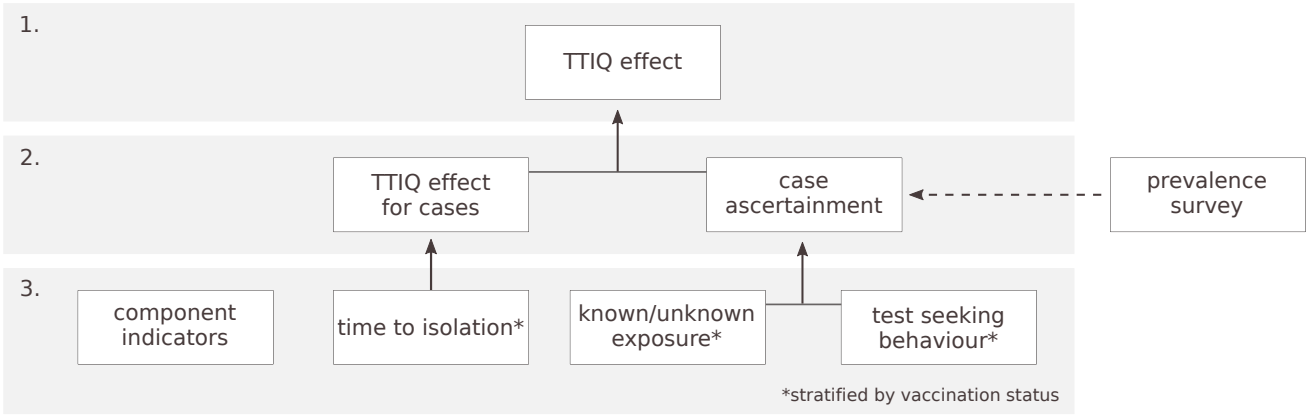
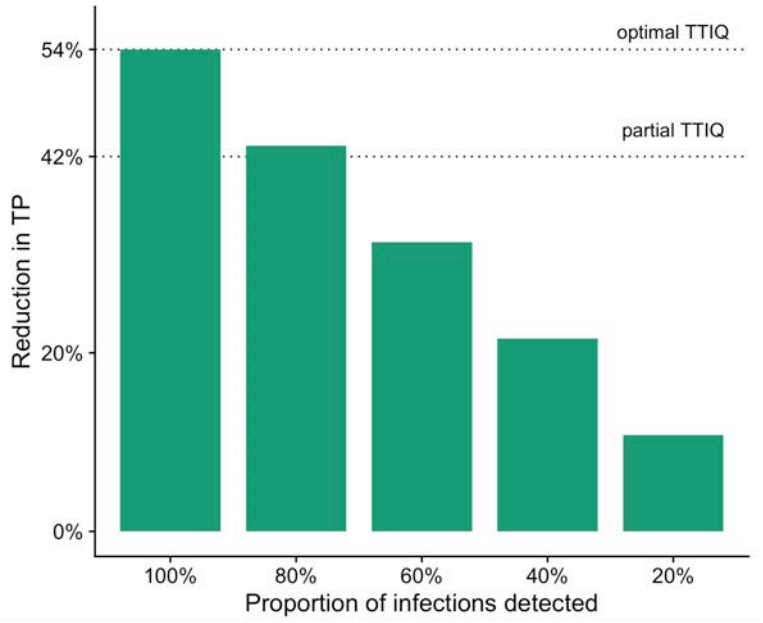


Figure 1.3.2: Relationship between case ascertainment and the proportional reduction in TP that has previously been observed in the Australian population (noting that historical ascertainment is likely somewhere between 80 and 100% of cases). Should ascertainment fall, these proportional reductions in TP will only apply to the proportion of infections that have been identified, initiating TTIQ responses.



Estimation of the proportion of all infections that are detected is critical to assessment of TTIQ impact and would ideally be informed by regular prevalence surveys. In their absence, modelling approaches may be used to infer the fraction of infections ascertained over time, but these estimates would be less accurate and difficult to validate.

Maintaining low case numbers through maintenance of ongoing PHSMs will further assist to constrain TP and support TTIQ.

Key Findings Work Package 2: Optimise vaccination at sub-jurisdictional level, including attention to key populations and risk settings

Key questions: What coverage targets are appropriate for populations at higher risk of transmission and disease impacts? What is the role of reactive vaccination in response should outbreaks occur in such localised groups and settings in the context of suboptimal coverage? What additional public health response measures will be most useful to regain control of transmission should outbreaks occur?

This report defines some of the key population characteristics, measurable in census and survey data, that allow identification of baseline increased risk of transmission in small areas and settings. The impacts on transmission potential (TP) of vaccination and public health and social measures may also be less than average in some of these groups. Enhanced public health focus including community engagement, strong TTIQ responses (including supports for isolation and quarantine) and heightened attention to transmission in schools will be required in such areas to improve health and social outcomes. Providing access to effective treatments will further promote health outcomes in populations at high risk of severe disease.

1. *First Nations Australians*

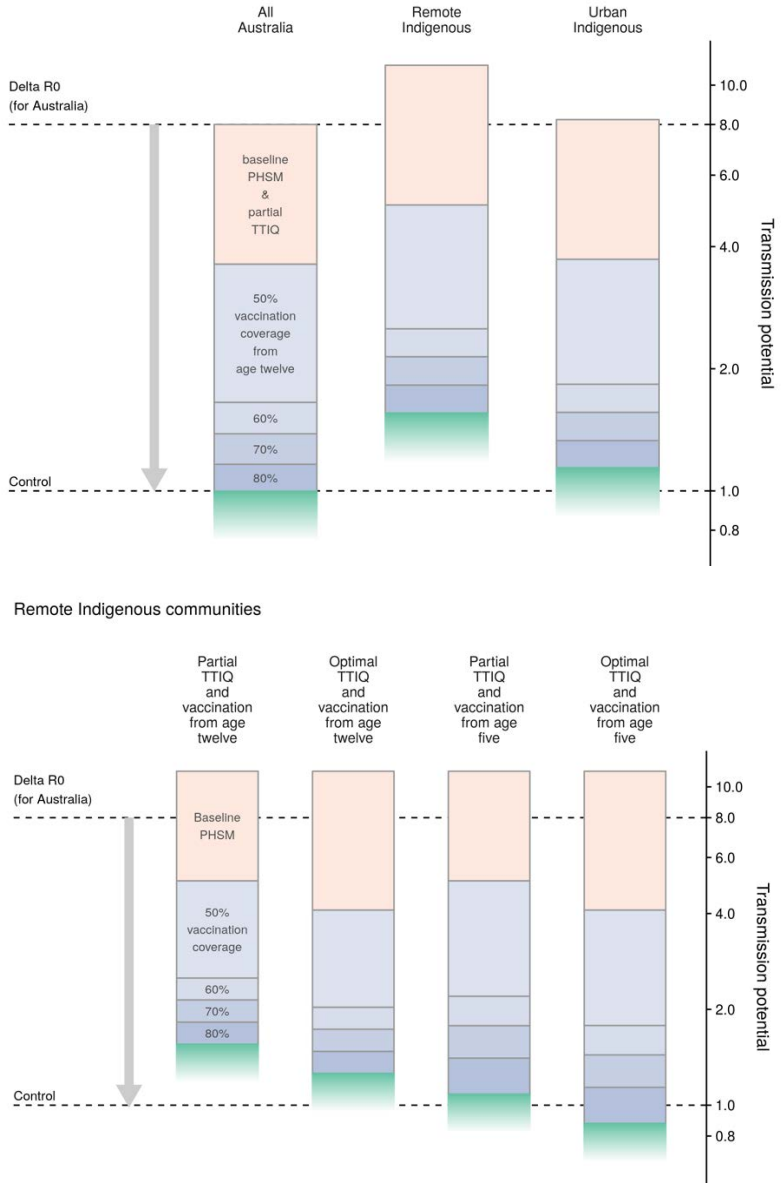
High vaccine coverage can reduce transmission and health impacts in urban and remote communities. Reactive vaccination is a useful adjunct to community engaged and led outbreak response, and can reduce health impacts, particularly in larger communities with low initial vaccine coverage. Providing access to effective treatments will further promote health outcomes, particularly where clinical access is limited.

Figure 2.1.1 indicates that 80% coverage of the 12+ population combined with PHSMs and partial TTIQ should be sufficient to reduce transmission potential (TP) to the control threshold of 1 for urban Indigenous communities. At this coverage level and even with optimal TTIQ, additional PHSMs will be needed to control outbreaks in remote settings. The figure illustrates how the baseline reproduction number (R_0) and the effects of vaccination coverage (for the population aged 12+ years) vary between population groups with different demographic profiles. Each column in the left panel of the figure represents a group with different age and household structure, estimated from population data and for the two rightmost columns, the Northern Territory Aboriginal Birth Cohort Study.

R_0 is increased when the proportion of adults aged 20-39 years is higher than the Australian average (29%). Indigenous populations in northern Australia also have a higher proportion of children less than 12 years and larger household sizes than the national average (3 x for remote, 1.7 times for urban). This intense household mixing drives the higher R_0 estimated for remote Indigenous communities. Vaccination of 12+ years has less effect on TP when children under 12 make up a larger proportion of the total population and live in larger households. Both factors increase their contribution to transmission despite lower susceptibility and infectiousness than adults. Lowering the age of immunisation to 5+ years is anticipated to substantially reduce TP in this context (right panel).

Note that the figure assumes baseline protective behaviours/PHSMs and TTIQ responses are equally achievable in all settings. The potential for responsive PHSMs to further reduce transmission in outbreak settings is indicated by the green shading in the Figure and would vary with measures employed. The effectiveness of community engaged and led responses to support TTIQ and distancing strategies has been clearly demonstrated in the recent Western NSW outbreaks. Note, however, that Figure 2.1.1 only considers impacts on transmission and not health outcomes, which are further mitigated by vaccine.

Figure 2.1.1: Transmission potential (TP) for Delta variant accounting for demography and household structure for remote living and urban Indigenous populations. 'Baseline' public health and social measures (PHSMs) and partial TTIQ public health responses are assumed. The figures report impacts on TP of 50-80% vaccine coverage* among individuals aged 12+ years (upper) and 5+ years (lower). Further TP reductions may be achievable in remote communities by vaccinating 5-11 year olds, even with partial TTIQ. Potential additional impacts of PHSMs are indicated by the green shading.



**Coverage at 70-80% includes some additional single doses at the two-dose threshold*

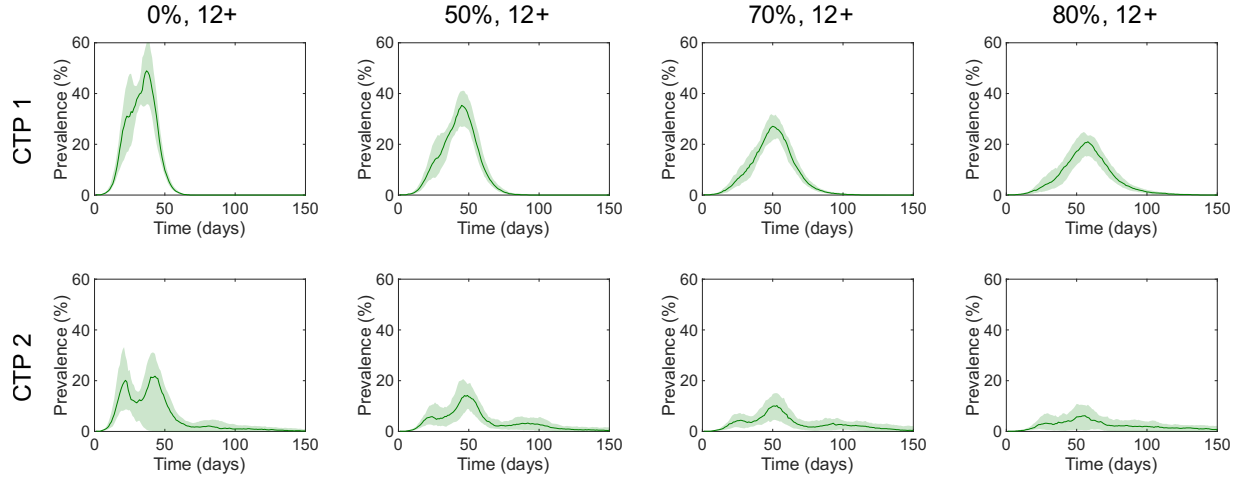
Attachment B reports findings from an agent-based model that captures key features of age structure, household composition and social connections in remote Aboriginal communities of different sizes. The model reports outbreak trajectories following silent introduction of infection in the context of different levels of prior vaccine coverage and given different response measures including reactive vaccination.

Modelled infections are translated into anticipated clinical outcomes using the clinical pathways model employed in our earlier phase work, with updated assumptions. Given the high prevalence of underlying health risk determinants in remote Indigenous communities we assume that the increasing likelihood of severe health outcomes by age commences from the age of 20 years and in each cohort thereafter maps to the non-Indigenous population 10 years older. This starting assumption has been approved by the Aboriginal and Torres Strait Islander Advisory group and benchmarked as reasonable against available data from NSW which demonstrates a higher prevalence of severe outcomes for Indigenous Australians.

Impact of pre-emptive vaccination on outbreak size and clinical outcomes

Figure 2.1.2 quantifies the impact of achieved uniform two-dose vaccine coverage of 50, 70 and 80% for ages 12+ on the magnitude and timing of an outbreak following silent introduction of infection into a remote community of size 1,000. Following identification of the first case, families are required to stay at home for 14 days, during which time all individuals are tested twice to enable case finding and household contact identification. All scenarios assume that cases, once identified, are isolated out of community. Upper and lower panels in the Figure compare currently recommended outbreak response strategies for management of contacts, quarantined either on (CTP1) or away from (CTP2) community. The lower panels show that the CTP2 strategy is demonstrably more effective at reducing outbreak size at all coverage levels.

Figure 2.1.2: Daily infection prevalence* in a remote Indigenous community of size 1,000 over time following initiation of an outbreak. Outputs compare different pre-emptive vaccine coverage levels and outbreak response management approaches. Results assume a starting TP of 10.7, and 90% compliance with stay at home orders implemented during the first 14 days following initial case detection.



**Solid lines: median prevalence, shaded regions: interquartile range of 100 simulations.*

Clinical outcomes of these observed infections will vary, depending on age and vaccination status of the individuals infected. Table 2.1.1 reports modelled health outcomes based on the clinical pathways model, noting the earlier assumption of a ten-year downward age shift in severity compared with the non-Indigenous population, based on the high prevalence of underlying clinical risk determinants in remotely living Indigenous Australians. Findings shown are for the less optimistic CTP1 strategy, reflecting feedback that quarantining of contacts away from community is noted to be challenging in many settings, but where it can be achieved, as in the Wilcannia outbreak, health outcomes are improved (the CTP2 strategy).

Table 2.1.1: Average cumulative symptomatic infections, ward admissions and ICU admissions over the course of outbreaks for achieved two dose vaccine threshold targets of 50, 70, and 80% for ages 12+. Results presented assume 90% level of compliance with lockdown and response policy CTP1, as in the top panel of Figure 2.1.2 above.

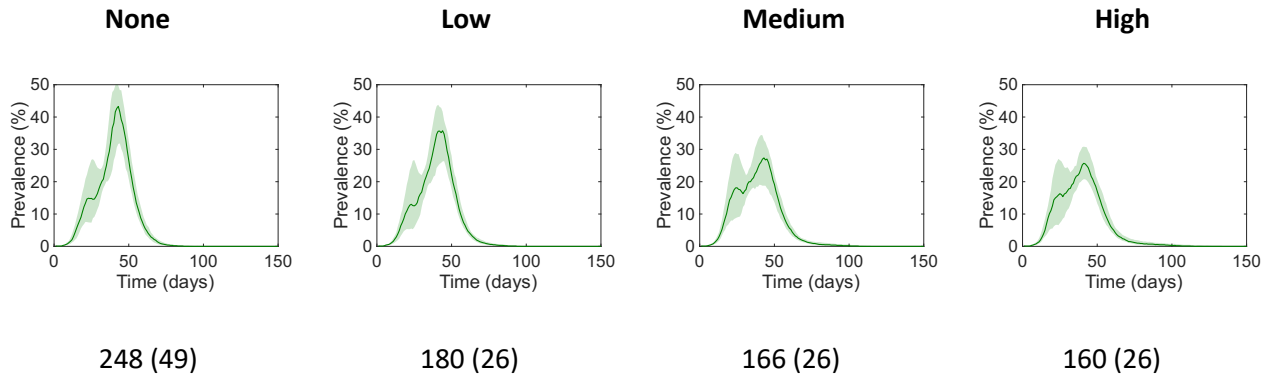
	Achieved coverage scenario	<15 yrs		15-39 yrs		40-59 yrs		60+ yrs	
		Vacc'd	Unvac	Vacc'd	Unvac	Vacc'd	Unvac	Vacc'd	Unvac
Symptomatic infections	50%	0	64	7	56	6	45	5	20
	70%	1	56	8	32	7	26	6	11
	80%	0	49	8	19	7	16	6	7
Ward admissions	50%	0	1	0	4	1	15	4	17
	70%	0	1	0	2	1	8	4	10
	80%	0	1	0	1	1	5	4	6
ICU admissions	50%	0	0	0	1	0	6	1	8
	70%	0	0	0	1	0	3	1	4
	80%	0	0	0	0	0	2	1	3

Impact of reactive vaccination in conjunction with other outbreak response measures

We further assessed the impact on transmission and clinical outcomes of a targeted immunisation program initiated as part of outbreak response. Outbreaks were simulated in communities of differing sizes and baseline coverage, based on case studies identified by the Aboriginal and Torres Strait Islander Advisory Group. The full set of outputs are included in Attachment B.

Findings for a community of size ~1,000 with low initial vaccine coverage are shown below and demonstrate the greatest benefits observed in the simulations. We consider how vaccines rolled out at different rates might augment the public health response in such a community. Rates of achievable delivery are based on advice from the Northern Territory, assuming different numbers of teams deployed for implementation. High acceptance is assumed among individuals not infected in the outbreak, with refusal of only 7%. This figure is concordant with recent experience from the Wilcannia outbreak.

Figure 2.1.3 Impact of reactive immunisation approaches as an adjunct to 'CTP1' response measures in a remote community of size 1,018. Baseline 2 dose immunisation coverage is ~50% for >50 years, ~25% for 40-49 years and <10% for eligible individuals <40 years. The reactive program is delivered at a low (30 doses/day for 13 days), medium (75 doses/day for 5 days) or high (120 doses/day for 3 days) rate. Clinical outcomes are reported below each panel as cumulative symptomatic infections over the course of the outbreak, with hospitalisations in parentheses.



In this scenario, reactive vaccination at any rate approximately halves severe outcomes, noting that vaccine protection against disease commences 2 weeks after a first dose, and increases further 5 days after a second dose.

Effects on transmission following the first dose begin a week later, rise over the subsequent fortnight and continue to increase after the second dose. In this example, lockdown measures are only maintained for 14 days. Differences in community size, baseline coverage and acceptance may increase the time taken to immunize communities effectively. In such cases it may be desirable to extend stay at home measures beyond the 14 days duration to slow down spread and maximise benefits of immunisation.

Beyond the National Cabinet reporting, we are continuing to work with the Advisory Group to develop extended narrative case studies of combined vaccine and other public health measures that may be feasible and implementable in remote settings with different starting vaccination coverage by age to maximise outbreak response impacts. Providing access to effective treatments will further promote health outcomes, particularly given limitations of clinical services in regional and remote Australia.

2. *Local Government and small area effects*

As shown for First Nations communities, demographic and social differences are anticipated to result in varying baseline transmission rates of COVID-19 across the Australian population more broadly. Drivers include larger mean household size (leading to more household contacts), larger working-age populations (leading to more workplace contacts) and social determinants such as housing quality and crowding. These factors tend to be geographically clustered and are often reported at the LGA-level. Such variation also influences likely vaccine impacts at subpopulation level as LGAs with a higher proportion of children will be more likely to observe ongoing transmission in those aged less than 12 years, who are currently ineligible for vaccination. In addition, inability to work from home reduces the impact of public health stay at home orders, and often correlates with higher baseline and post-vaccination transmission potential. Focussed TTIQ responses and augmented school and workplace measures will be needed in such areas, not lockdowns.

Figure 2.2.1 shows how population characteristics influence baseline transmission potential and vaccine impacts. Compared with the 'all Australian' population, small area TP and vaccine impacts will be heterogeneous, as demonstrated by five exemplar LGAs each for greater Melbourne and Sydney. Kingston (left panel) and Sutherland Shire (right panel) are most 'typical' of the national average. Affluent areas comprised of small households and a high proportion of working age adults (Port Phillip, Stonnington, North Sydney, Mosman) have an average baseline TP but larger than average vaccine change impacts. Areas like Greater Dandenong and Fairfield have a higher than average proportion of working age adults, which accounts for a higher starting TP but also marked reductions achieved following vaccination. Murrindindi and Oberon both have lower baseline transmission potential and vaccine impacts arising from higher proportions of children and older adults than the national average, respectively.

Experience has also shown that the ability of lockdowns to modify mixing and so reduce transmission are inequitable across geographical areas. While a number of behavioural changes result in PHSM impacts, the ability to work from home can be anticipated with reasonable certainty based on occupation and have been validated on the basis of survey data. In some LGAs, there is a high proportion of people whose work cannot be done remotely and are considered 'essential', who will continue to have workplace contacts even under the most restrictive of PHSMs. Varying ability to work from home is reflected in the differences between the green components of Figure 2.2.1. Port Phillip, Stonnington, North Sydney and Mosman have large population proportions in professional occupations that are amenable to stay at home working. Greater Dandenong and Oberon each have higher than the national average proportion of machinery operators and labourers, who cannot work from home. Murrindindi and Fairfield have a larger than average proportion of children who are not in employment, lessening the impact of work from home requirements on overall levels of mixing in these areas under public health orders.

Even within LGAs, the ability to work from home may be heterogeneous, resulting in subpopulation 'pockets' in which heightened transmission can occur. Such effects were notable in Western and South-West Sydney during the 2021 outbreak response. Figure 2.2.2 reports variation at SA2 level in the ability to work from home within different LGAs in NSW and VIC.

Figure 2.2.3 maps geographical variation in these described measures of baseline transmission potential, vaccine change impacts and overlaid work from home measures for Melbourne and Sydney. These figures show how the distribution of relative risk of transmission may change following vaccination due to variable vaccine change impacts. Anticipation of such shifts should guide enhanced surveillance and response efforts through the transition phase.

Full outputs for this work package are included in Attachment C. The main conclusion of this work is that stay at home orders will not necessarily mitigate importation and outbreak risks in many LGAs that would be anticipated to have higher than average ongoing risks of transmission, even with high 12+ vaccine coverage. Focussed TTIQ responses, wrap around supports and school and workplace measures are more likely to effectively reduce transmission and disease impacts in these settings.

Figure 2.2.1: Baseline transmission potential (TP) for the Delta variant accounting for demography and household structure across exemplar Melbourne and Sydney LGAs with differing population characteristics. The figure reports the impact on TP of 50-80% vaccine coverage for the 12+ years population. It further shows variables reductions in transmission achievable through work from home requirements under stay at home orders, based on predominant occupations with each LGA.

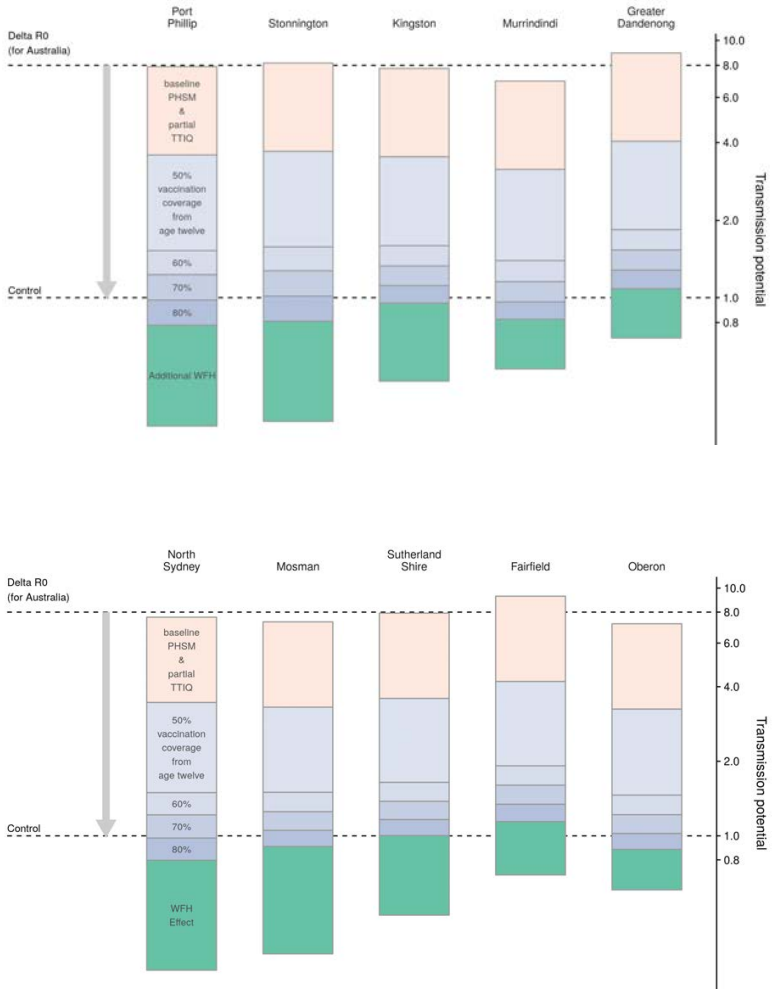


Figure 2.2.2 Proportion (with 95% Confidence Intervals) of residents with the ability to work from home based on ABS occupations data, calculated for each SA2 within listed LGAs

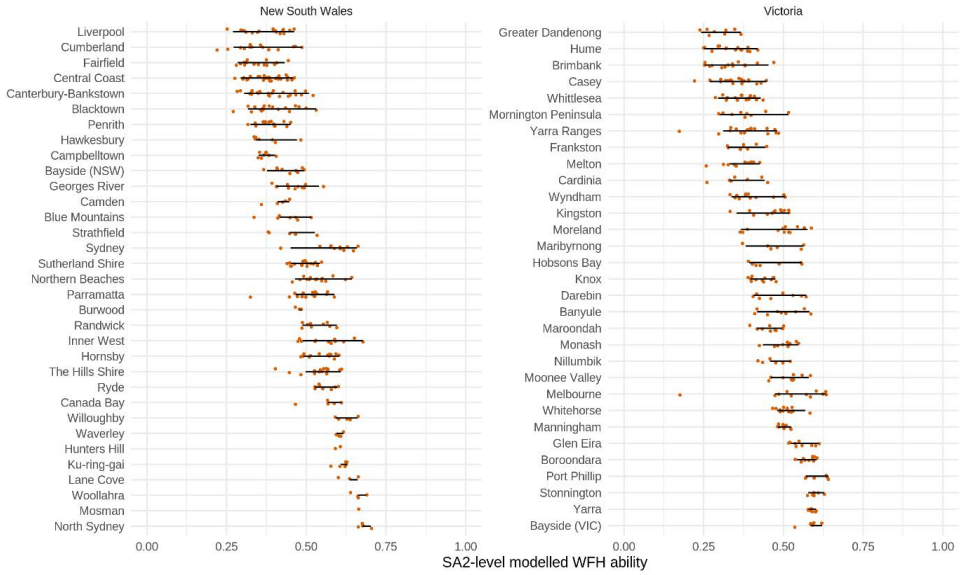
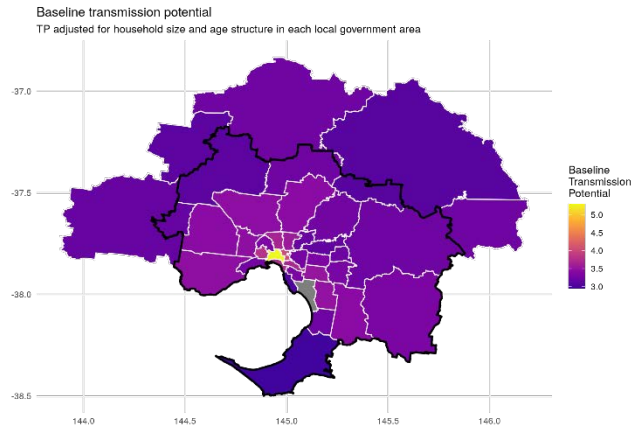
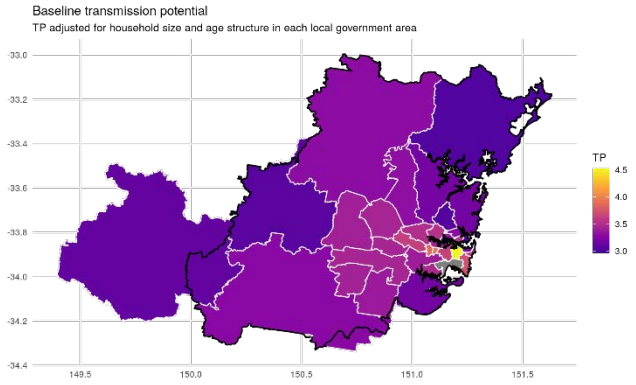
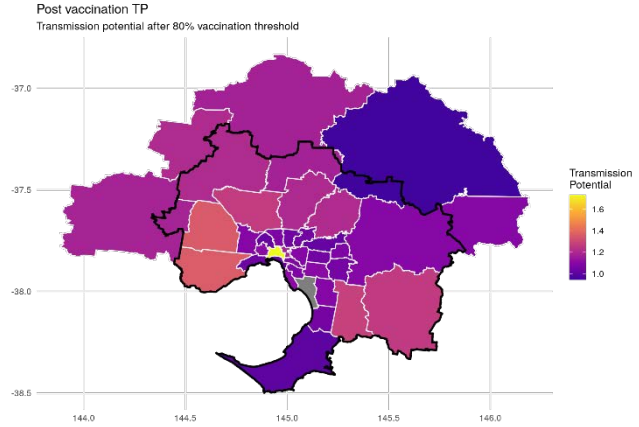
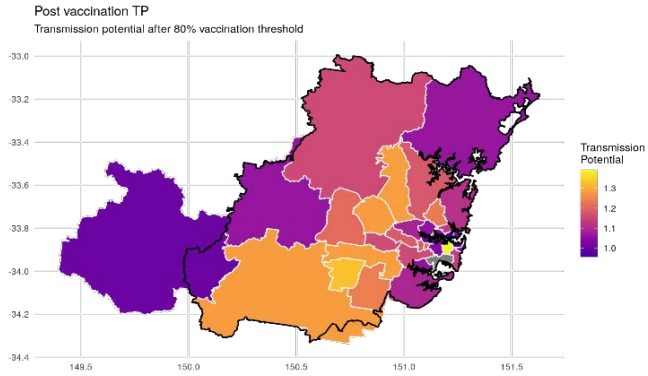


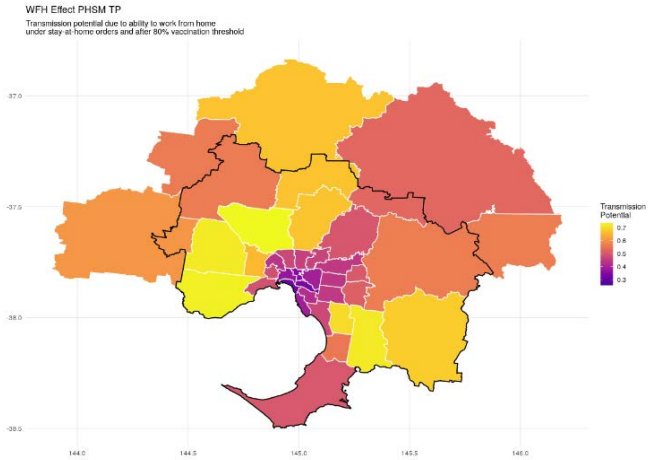
Figure 2.2.3 Mapped estimates of baseline TP, absolute reduction post vaccination, and absolute reduction following vaccination and overlaid work from home orders, by LGA, for Melbourne and Sydney

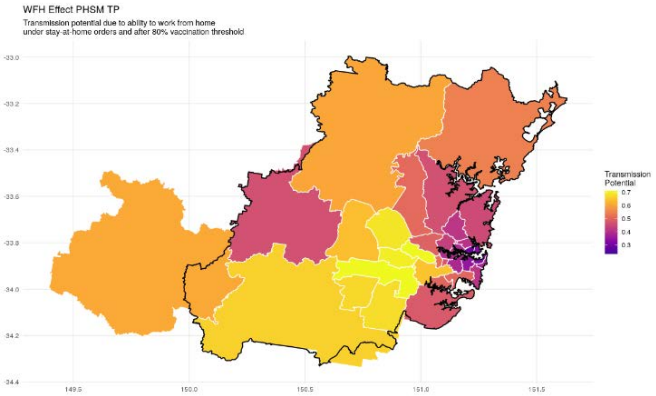












3. Schools

As community transmission becomes established, incursions into school settings will be inevitable. Returning students to in-person learning and keeping schools open safely during this phase has been identified as a national priority. Early detection of infections through surveillance testing substantially reduces the risk that incursions will lead to outbreaks and if feasible, may be an appropriate strategy in areas with high levels of ongoing transmission. Daily rapid antigen testing of contacts, with exclusion only if positive, is as effective for outbreak prevention as 14-day contact quarantine and dramatically reduces days of missed face to face learning.

We have used a model of primary and secondary schools within the context of a community to consider the likely consequences of incursions, for different screening and testing strategies, vaccination coverage and contact management approaches. As Figure 2.3.1 demonstrates, in the absence of screening or any form of contact tracing or management, between 37-47% of incursions will 'die out' given the heterogeneous nature of COVID transmission. But between one third to one half of introductions will result in 20 or more infections and sometimes as many as 50. These figures show the case for both high schools, where we assume that 80% of students and staff are vaccinated and primary schools where children are too young to be immunized. Our sensitivity analysis confirms that higher student vaccine coverage in high schools substantially reduces the risk of large outbreaks. Teacher vaccination has less influence on transmission within the school, even at 100% uptake, but would be anticipated to materially impact on importation risk.

Figure 2.3.1 further explores the ability of different routine surveillance strategies to minimize spread and days of face to face learning lost in schools. If symptomatic students are diagnosed and sent home early, on average only tens of teaching days will be lost per incursion over the reporting period. Screening teachers twice weekly regardless of symptoms with rapid antigen (RAT) testing makes little difference to school-based outbreaks, as there are relatively few teachers in the school. Twice weekly testing of students markedly increases the chances of nipping an outbreak in the bud. There is a small increase in average school days lost because we are looking harder for infections and so detect asymptomatic individuals, but far fewer large outbreaks. These findings are for a single infection introduction – as shown in Attachment D, the relative utility of this approach increases with the number of incursions over time.

Figure 2.3.1 Impact of twice weekly rapid antigen testing (RAT) surveillance of teachers and students on size of outbreaks following incursion and days of face to face learning lost. No contact tracing is assumed. All outputs are the results of 1,000 simulations.

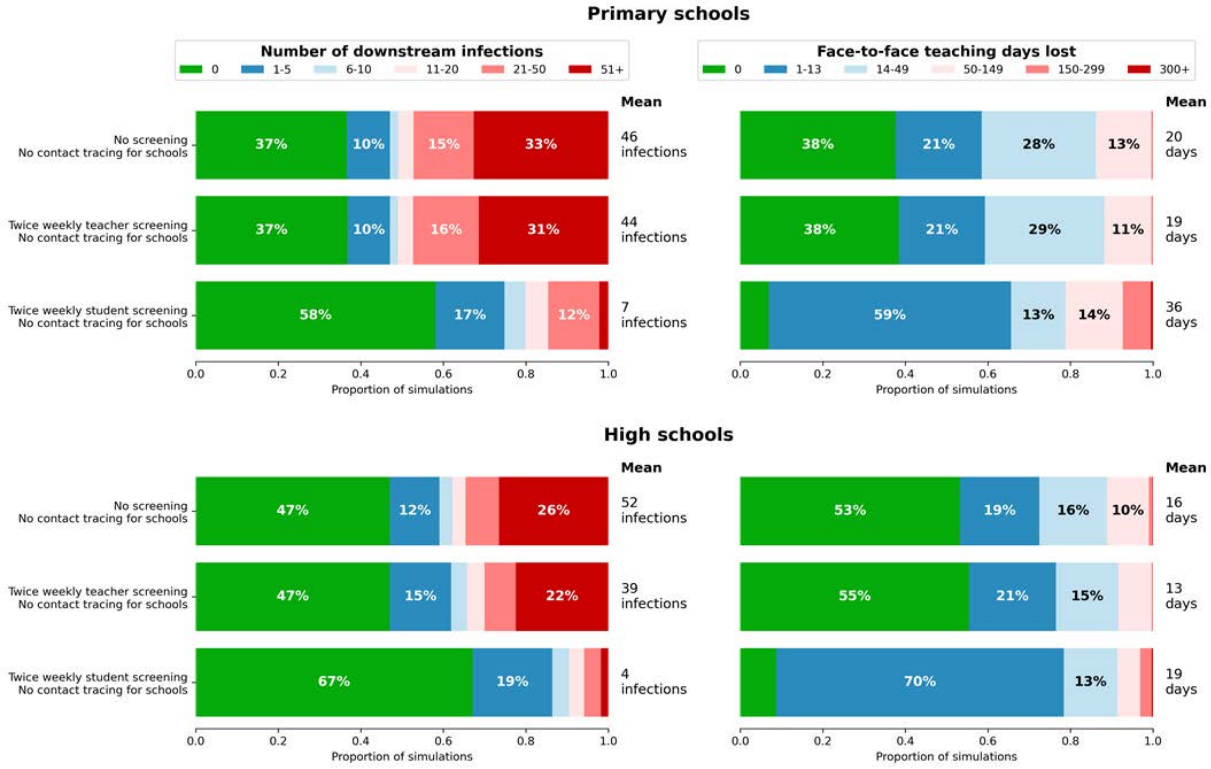
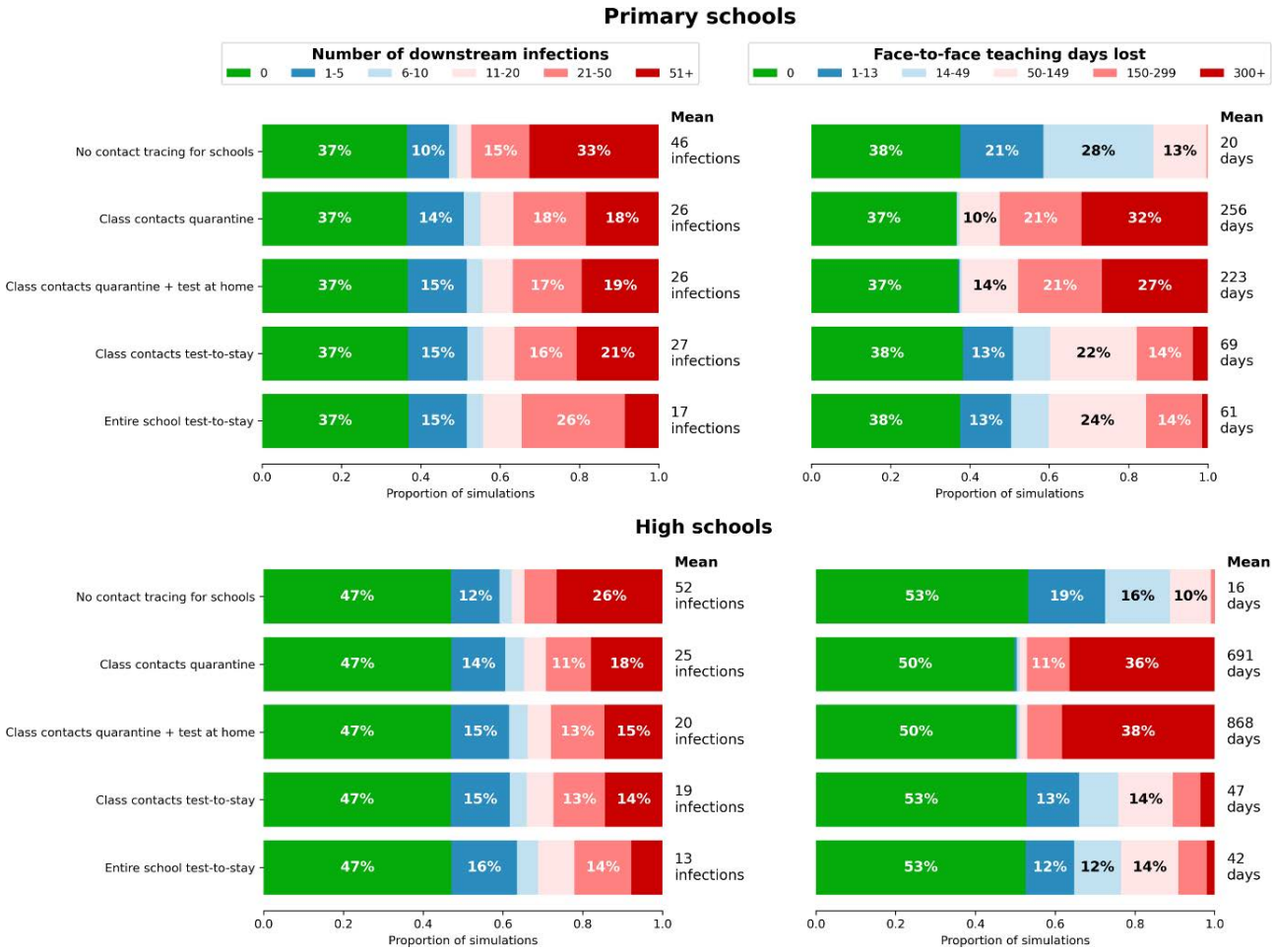


Figure 2.3.2 compares different contact management approaches in the *absence* of surveillance testing. Any form of contact management reduces the chance that outbreaks will grow to 50 or more – whether class contacts are sent home for 7 days or require daily RAT testing 'to stay'. But the number of days of school lost by the quarantine approach is dramatically different. The quarantine option including daily testing at home is to ensure that the likelihood of identifying an infection is equivalent to the 'test to stay' approach. The findings shown are for 70% RAT sensitivity, and 100% compliance. A sensitivity analysis finds that benefits of the test to stay approach are still seen, even if compliance is as low as 50%, because repeated testing increases the likelihood of detection.

Figure 2.3.2 Impact of alternative contact management approaches on size of outbreaks following incursion and days of face to face learning lost. No surveillance is assumed. All outputs are the results of 1,000 simulations.



These model findings reproduce the outcomes observed in a real world study comparing quarantine and test to stay in England. They strongly endorse test to stay as a policy to maintain face to face education and keep schools open. It should be noted that the reduction in outbreaks achieved by this measure is less than surveillance screening.

Additional analyses have demonstrated synergistic benefits of combining twice weekly surveillance screening with test to stay contact management (Attachment D). The greatest number of face to face teaching days gained through this approach occurs when community incidence is highest, resulting in multiple importations.

Evaluation of the ability to implement school based surveillance and testing strategies is recommended as a priority, to support a safe return to face to face learning. Such approaches will have maximum utility in small areas identified as at risk of higher than average community transmission.

Work Package 3: Review border measures & arrivals pathways in context of revised risk tolerance

Key question: How can arrivals caps and pathways be safely modified in the context of the changing risk environment as population vaccine coverage increases?

As Australia opens its borders to international arrivals, it is inevitable that infection importations will occur. We compare the effectiveness of different quarantine and testing requirements to reduce the risk of vaccinated adults and partially vaccinated family groups seeding infections in the community. Vaccination reduces the risk of infected people being released from quarantine into the community, mitigating against shorter duration of quarantine. We then compare scenarios for different numbers of arrivals, quarantine pathways and vaccine coverage for endemic and 'COVID-zero' scenarios, based on pre-COVID-19 traveller volumes. Vaccine uptake in the local population is the dominant determinant of the consequences of importation on local infection numbers in the arrival jurisdiction. Breach importations do not materially impact on established epidemics or lead to large outbreaks at the defined Phase C coverage threshold of 80%, combined with 'low' PHSMs.

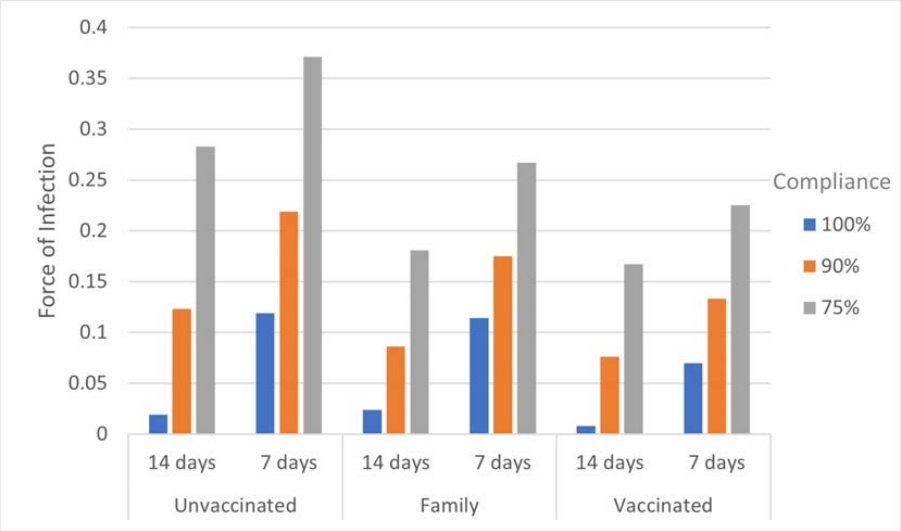
Effectiveness of alternative quarantine pathways

We have modelled a range of home quarantine pathways to compare the exposure days anticipated from an infected arrival passing through that system. Our updated calculations include assessment of the risks posed by family groups composed of adults and children, understanding that children less than 12 years are currently ineligible for vaccination. Vaccination reduces infectiousness and hence the risk posed by quarantine breach events from immunized travelers. In this way it mitigates against the observed increase in community exposure days resulting from shorter duration stays. A full table comparing the force of infection resulting from an infected arrival transiting through various pathways is provided in Attachment E.

Exposure days resulting from infected people being released into the community occur either because the initially infected traveler goes undetected, or they transmit infection to another traveler who goes undetected. We derive a measure called the 'force of infection' that relates those exposure days to their infectiousness, which peaks in the early stages of infection. This measure equates to the expected number of secondary infections produced by one infected arrival in a fully susceptible (ie unvaccinated) population.

Figure 3.1 compares three types of traveler groups– unvaccinated adults, family groups (comprising 2 vaccinated adults and 2 unvaccinated children <12 years) and fully vaccinated adults. Key differences for family groups are: children are not protected by vaccination but if they do contribute a quarantine breach are assumed intrinsically less infectious in the community than adults. If a child is identified as infected in quarantine, they will be isolated with a parent and not alone, so there is an ongoing risk of infection transmission within the isolation facility/medi-hotel that would not apply to adult travelers.

Figure 3.1: Force of infection per infected arrival in home quarantine, for unvaccinated arrivals, family units containing vaccinated parents and unvaccinated children, and unvaccinated arrivals. Results are shown by duration of stay (14 or 7 days) and compliance with quarantine (100%, 90% or 75%)



Different drivers of community exposures are assessed as before. Longer quarantine stays reduce incursion risk. Lower compliance with quarantine requirements is more influential at increasing risk than shortening the length of stay. And vaccination reduces risks across the board. Families contribute an overall risk that is intermediate between fully vaccinated and unvaccinated arrivals.

The estimated force of infection (FOI) for the quarantine pathways in Figure 3.1 has been benchmarked against the previous policy requirement of 14 days hotel quarantine for unvaccinated arrivals (FOI=0.042). Perfect compliance with 14 days home quarantine for a vaccinated adult is associated with an 80% lower FOI than that baseline (FOI=0.008). The scenarios below consider 7 days home quarantine with 90% compliance for vaccinated adults and family groups, for which the relevant FOIs are 3 and 4 fold higher than the previous policy but remain well below one (0.133 and 0.175, respectively). *Note that these values are for a traveler who enters the system infected. Vaccination and pre departure testing reduces the risk that a traveler who is exposed in their country of origin arrives in Australia infected.*

Definition of arrivals scenarios for endemic and 'COVID-zero' settings

We have devised arrivals scenarios in consultation with PM&C, Home Affairs and Treasury that allow us to calculate the aggregate weekly force of infection for different numbers of vaccinated adult and family group arrivals into endemic and 'COVID-zero' jurisdictions 'filtered' through alternative quarantine pathways. We have compared risks associated with 14 or 7 day stays in hotel or home quarantine (the latter assuming 90% compliance), 'no quarantine' (with PCR testing on days 1 and 5) and the previous 14 day hotel quarantine requirement for unvaccinated travelers.

All pathways other than 'no quarantine' are associated with a lower aggregate force of infection than 14 day hotel quarantine for unvaccinated arrivals. This reduction is because of the actions of vaccination prior to departure (preventing infection), as well as within and following release from quarantine (reducing infectiousness). It should be noted that incursion risks are mitigated by testing on days 1 and 5 and are higher if no tests are performed (see Attachment E for full details of all pathways).

The total number of arrivals is calculated as a proportion of 2019 traveler volumes into a large and medium jurisdiction for Australian citizens and permanent residents. We use numbers of travelers up to the age of 12 years from these data to allocate 'family groups' incorporating a corresponding proportion of adults in units of size four (two vaccinated parents, two unvaccinated children). Doubling the number of arrivals from 40% to 80% doubles the force of infection per unit time, *noting that this measure estimates the number of secondary infections anticipated in an unvaccinated population.*

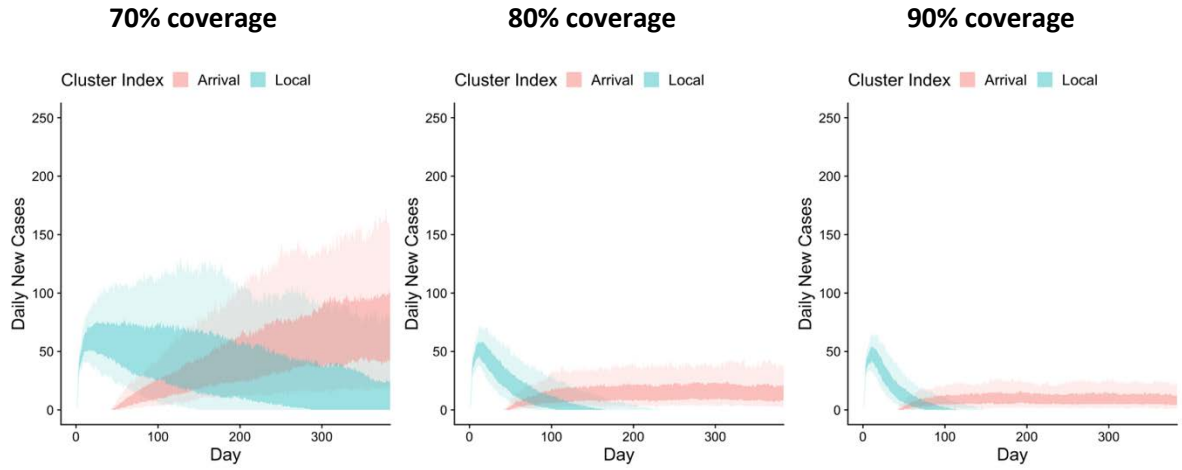
Consequences of importations for local epidemiology

<u>Scenario 1 – Endemic cases</u>

Epidemiological consequences of the arrivals scenarios above are demonstrated in Figures 3.2.1 and 3.2.2. The 'vaccine coverage' in these simulations is fixed at the beginning of the simulations, with no ongoing vaccine rollout assumed. We seed 200 'local' infections on day 0 to establish a local epidemic, with travellers beginning to arrive on simulation day 40. At 70% vaccine coverage, ongoing transmission of local strains occurs and is gradually superseded by new infections resulting from imported strains. For 80 and 90% coverage, locally transmitted strains become extinct at around 100 days.

Ongoing importation of strains is a continuous source of newly seeded infections, but transmission is sufficiently constrained by vaccination that large outbreaks do not occur.

Figure 3.2.1: Impact of incursions on endemic cases given differing vaccine coverage in the arrivals environment. Partial TTIQ and ongoing 'low' PHSMs are additional constraints on transmission. Travellers (vaccinated adults and families) are managed through a 7 day home quarantine pathway, with 90% compliance and PCR testing on days 1 and 5. Traveler volumes are 40% of 2019 citizen/Permanent Residents values from a large jurisdiction*.



Shaded areas denote uncertainty across multiple simulations. Teal shading reflects new cases resulting from local strains present at the beginning of the simulation. Salmon/pink shading denotes cases resulting from transmission chains seeded by importations.

**Estimates of traveller volumes used in the model for 40% are 32,767 per week based on 2019 arrivals into NSW*

Figure 3.2.2: As for Figure 3.2.1, but comparing 40% (left) and 80% (right) of 2019 arrivals, 80% coverage

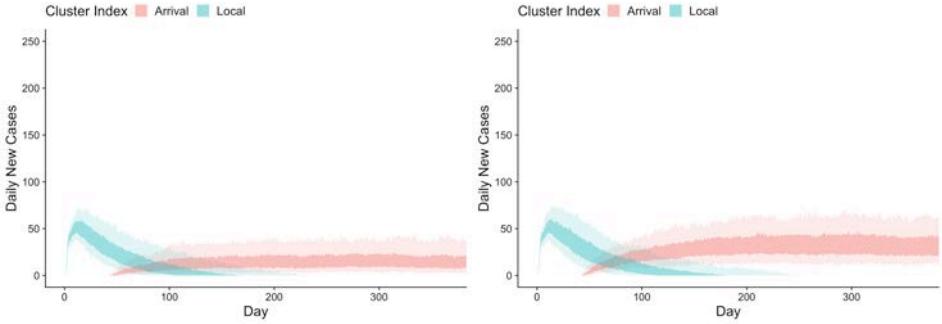
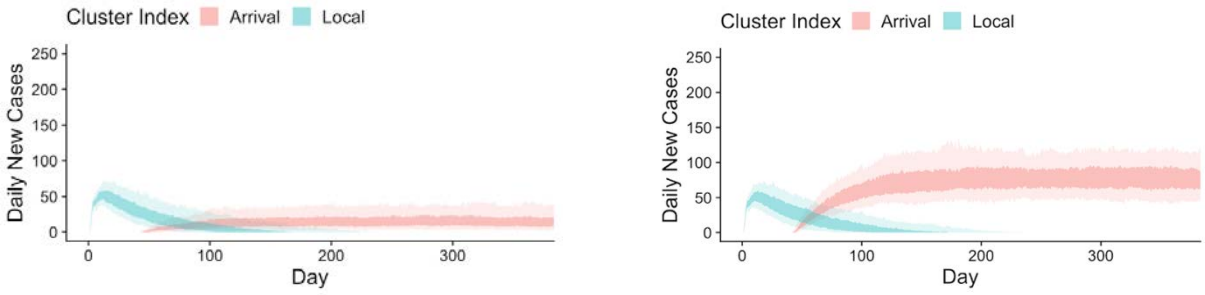


Figure 3.2.2 demonstrates that doubling the number of arrivals results in an approximate two-fold increase in daily incident infections resulting from importations. Further increases in traveller volumes would lead to similar linear impacts on importations, assuming the same mix of arrivals by vaccination status (an increase in the proportion of vaccinated adults compared with families would lead to a slight proportionate reduction in the scaling of this overall risk). The corresponding set point for the 'no quarantine' pathway is approximately three to four-fold higher (Figure 3.2.3) than for 7-day home quarantine.

Figure 3.2.3: As for Figure 3.2.1, but comparing 7 days home quarantine (90% compliance) (left) with the 'no quarantine' pathway with PCR testing on days 1 and 5 (right) and 80% coverage



These differences are explained by the total force of infection calculated for numbers of arrivals, traveller types including vaccine status and the quarantine pathways through which they are processed. Essentially, the level of infection in the arrivals destination relates directly and linearly to this value. However, in terms

of consequences, as in Figure 3.2.1 high vaccine coverage, partial TTIQ and low PHSMs strongly constrain transmission, preventing rapid outbreak growth. Note that these outputs assume homogenous vaccine coverage and transmission potential.

The importance of controls in place in the arrivals environment is demonstrated by an additional scenario for endemic cases considering the impact of partial TTIQ with only *baseline PHSMs* in place, for all the same arrivals considerations as above (Figures 3.3.1, 3.3.2 and 3.3.3). Note the marked difference in axes between these two sets of figures. **At 80% coverage, thousands of incident cases are expected daily with only baseline PHSMs in place, compared with fewer than 100 when ongoing low PHSMs are maintained.**

Such rapidly escalating infections are driven by 'local' cases which far exceed the rate of importation. Incursions do not materially impact on the established local epidemic. This scenario is demonstrative only, as an outbreak of the size shown for the 70 and 80% coverage examples would require imposition of additional measures to reduce disease burden and impacts on the health system and society.

Figure 3.3.1: As for Figure 3.2.1 but assuming Partial TTIQ and 'baseline' PHSMs in place

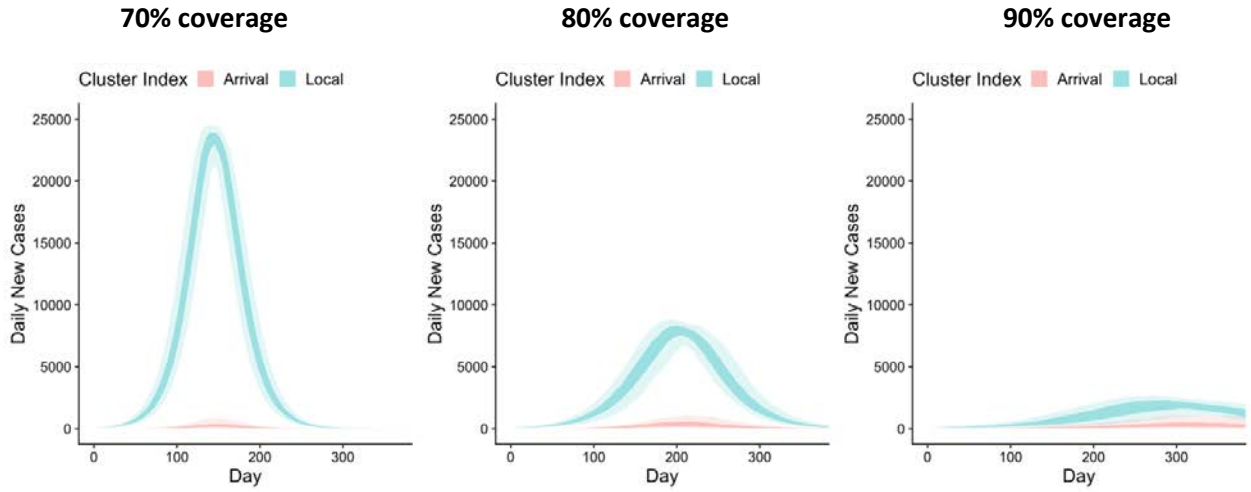


Figure 3.3.2: As for Figure 3.3.1 but comparing 40% (left) and 80% (right) of 2019 arrivals, 80% coverage

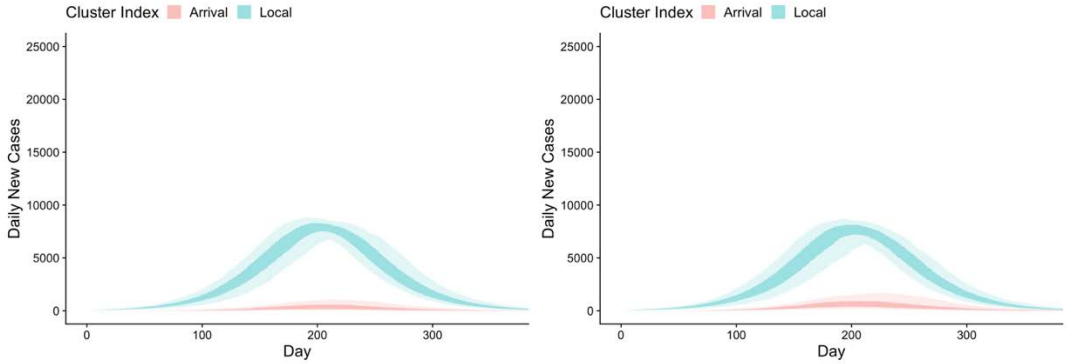
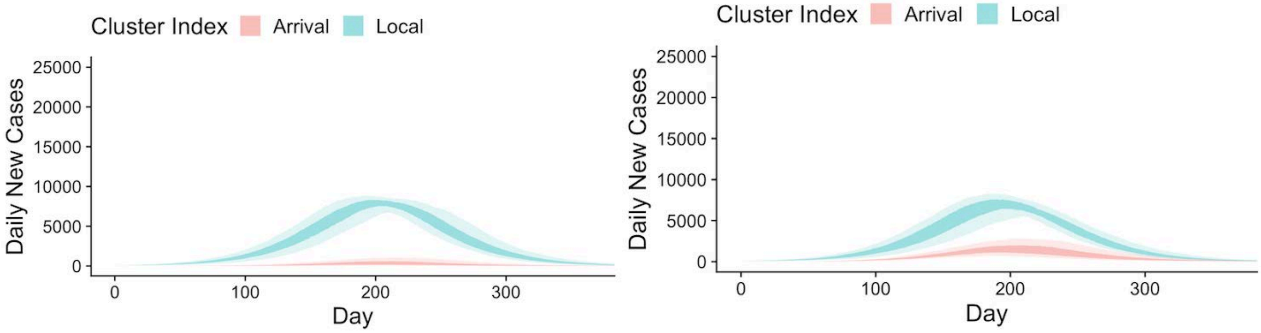


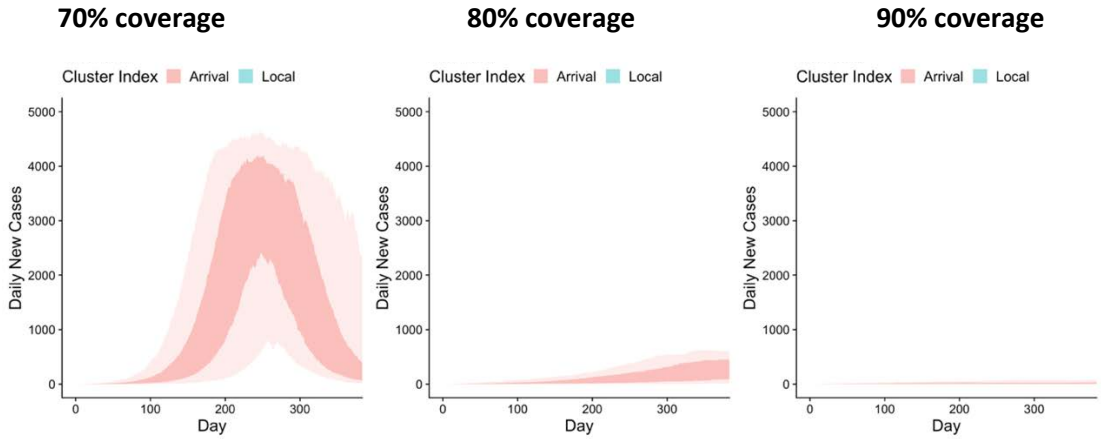
Figure 3.3.3: As for Figure 3.3.1, but comparing 7 days home quarantine (90% compliance) (left) with the 'no quarantine' pathway with PCR testing on days 1 and 5 (right) and 80% coverage



Scenario 2 – 'COVID-zero'

The simulations in Figure 3.3.1 and 3.3.2 share most of the same assumptions as previously but with *optimal TTIQ and baseline PHSMs* in place in a 'COVID-zero' jurisdiction. This difference accounts for the enhanced epidemic growth most apparent in the 70% coverage case, noting that the y axes in these figures are in the 1,000s compared with Scenario 1 (maximum 125). The seeded epidemics grow slowly initially because the transmission potential is just above one but escalate within a few months at 70% coverage. At 80% or higher coverage epidemic growth is slower as further constrained. Because all infections are seeded by 'arrival' strains only one colour is shown on the plots, but in reality it is implausible that only internationally seeded infections would circulate over the one year time frame of the simulations.

Figure 3.4.1: As for Figure 3.2.1 but for 'COVID-zero', and assuming optimal TTIQ and 'baseline' PHSMs. Traveler volumes are 40% of 2019 citizen/Permanent Residents values from a 'medium' jurisdiction*.



**Estimates of traveller volumes used in the model for 40% are 10,363 per week based on 2019 arrivals into WA*

Figure 3.4.2: As for Figure 3.4.1, but comparing 40% (left) and 80% (right) of 2019 arrivals, 80% coverage

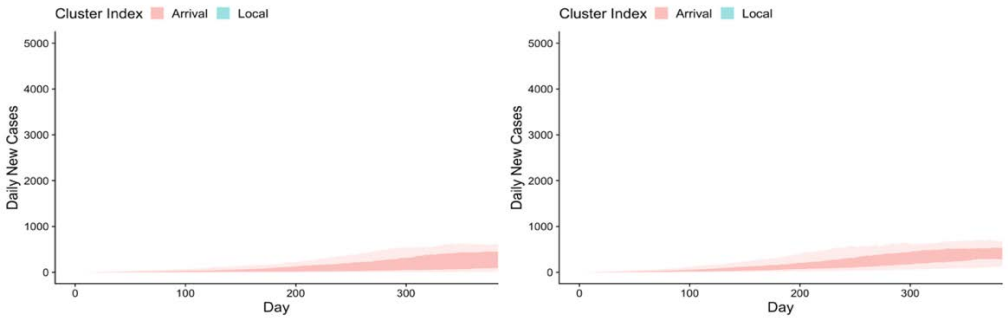
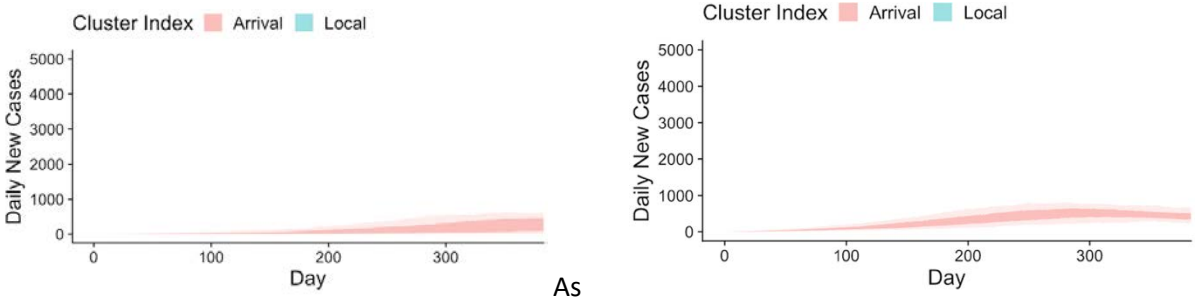


Figure 3.4.3: As for Figure 3.4.1, but comparing 7 days home quarantine (90% compliance) (left) with the 'no quarantine' pathway with PCR testing on days 1 and 5 (right) and 80% coverage



As previously, the set point of daily case numbers scales approximately linearly with the calculated FOI resulting from total arrivals. Other arrivals pathway scenarios are shown in full in Attachment E.

Note that all of these simulations assume consistent vaccine protection over time (ie immunity does not wane) and that the characteristics of imported strains are identical to those initially present in the population (ie they are not more transmissible and are equally preventable by vaccination).

Influential revisions to parameter assumptions

Between the previous and current phases of our modelling work we have extensively reviewed available evidence regarding age-dependent mixing and susceptibility to the Delta variant, vaccine uptake, and vaccine effectiveness assumptions against acquisition, infectiousness and disease outcomes. While values of individual parameters vary between phases of our work, we have assessed the consequences of these changes in aggregate and confirm that our previous recommendations of vaccine coverage thresholds for national plan transition phases remain robust.

Social mixing assumptions

In the first phase of our National Plan modelling, we developed an age-structured transmission matrix characterising infection spread within and between age groups based on population mixing assumptions using widely accepted social contact matrices and age-specific susceptibility and transmissibility estimates published by the London School of Hygiene and Tropical Medicine.

For this phase of work we have updated our mixing assumptions to align more closely with reported observations in the Australian context. We have re-estimated transmission parameters to fit infection age distributions from the UK post-reopening and with full school attendance, which have demonstrated few infections in primary schools in the absence of non-pharmaceutical interventions and vaccination. Our reanalysis finds a reduction in the proportional contribution of children aged 5-11 years to transmission, and some increase for those aged 16-24 years with the following consequences:

(i) A more optimistic expectation of overall vaccine impact on transmission potential (TP) in populations with a high proportion of children than previously anticipated (countered in some populations by large household size);
(ii) A boost in TP reduction associated with vaccination of the 16-24 years group.

Vaccine coverage assumptions

Our initial coverage scenarios considered optimal age-based vaccine distribution strategies to minimise transmission and disease. The Quantium team in Health advise that the actual rollout in the Australian population has most closely approximated the 'all ages' strategy, which resulted in high uptake in the peak transmitting age groups identified above, maximising population wide benefits of the program. Extension of vaccine eligibility to the 12+ years group has further increased whole of population coverage and can be considered as a 'bonus' to the target thresholds.

In addition, the pace of rollout has exceeded expectations, particularly in states with community transmission, enabling threshold targets of 70 and 80% to be reached in a timely manner. Of note, it is anticipated that 'final' vaccine coverage in the order of 90% will be achieved within weeks of the 80% target, which is much faster than in the original simulations provided by Quantium. Should these expectations be realised, we anticipate greater constraint of transmission in the initial weeks following the transition to Phase C than was estimated by our model, in which it took months to achieve this final coverage.

Vaccine effectiveness assumptions

We have updated our assumptions of vaccine effectiveness (VE) against infection and onwards transmission, based on new evidence from the UK specific to the Delta variant. On balance, these changes have resulted in some reduction in overall effectiveness of two doses of the Astra Zeneca vaccine (from 86% to 79%), but none for Pfizer (remains 93%) which has been the predominant vaccine delivered through the Australian program.

Since completion of the first phase of the National Plan modelling, further evidence has emerged regarding vaccine effectiveness (VE) against clinical outcomes for the Delta variant. On balance, these changes have resulted in some reduction in overall effectiveness against symptomatic infection of two doses of the Astra Zeneca vaccine (from 90% to 79%), but minimal change for Pfizer (from 92% to 90%) which has been the predominant vaccine delivered through the Australian program.

Clinical severity assumptions

In our National Plan modelling we reviewed all available evidence on clinical severity of SARS-CoV-2 infections. The bulk of this evidence related to the Wuhan strain, given that it circulated globally over an extended period. From this evidence we derived age-based estimates of the likelihood of hospitalisation and severe disease outcomes following detection of symptomatic infection.

Based on our review of available evidence about Alpha variant infections at that time, we applied an odds ratio (OR) of 1.42 to hospitalisation outcomes across all age groups. At that time, there was uncertainty in the literature about the relative clinical severity of the Delta variant compared with the Alpha variant. Published reports variously described it as milder, about the same, or more severe. On balance we assumed the same severity as for the Alpha strain.

Following completion of that phase of National Plan modelling it has become clear from published studies that the Delta variant is more likely to be associated with severe clinical outcomes than Alpha. The most informative study in the peer reviewed literature reports the odds ratio for hospitalisation given symptoms as 2.08 compared with the Wuhan strain. Given the same 'benchmark' (Wuhan) strain for both viruses, an OR of 2.08 for Delta represents an increase but not a doubling in severity compared to Alpha, for which the assumed OR was 1.42.

An OR is not the same as a percentage increase or decrease. If hospitalisation is rare as is the case for children, then it is approximately true that the OR of 2.08 means hospitalisation is twice as likely. Compared with Alpha, Delta may therefore result in an increase in admissions in this age group by as much as 40-50%. However, for older adults, in whom hospitalisation is a common outcome, the additional increased chance for hospitalisation due to the virus per se will be relatively lower, meaning that absolute numbers of hospitalisations may increase by as little as 10-15%.

More details regarding these parameter choices and tables summarising final assignments are contained in Attachment F.

Work Package 1: TTIQ

Table of contents

Executive summary

Strategies to streamline and focus TTIQ responses as jurisdictions move into Phases B and C of the National Plan were identified through consultation with the Communicable Disease Network of Australia and the Australia Health Protection Principal Committee. This report examines the likely impacts of these modified TTIQ strategies on transmission potential in the context increasing caseloads, the Delta variant, and high vaccine coverage. Further, the report outlines requirements for real-time monitoring of the effectiveness of the entire TTIQ system to support ongoing adaptation of strategies to the epidemiological situation.

- Streamlined and focused test-trace-isolate-quarantine (TTIQ) processes (supported by PHSMs) will be required for future public health responses to remain effective.

  TTIQ is limited by case ascertainment. The fraction of infections displaying symptoms will decrease with increasing vaccination coverage, limiting opportunities for detection.

- Targeting a transmission potential (TP) at or around 1, as per the national strategy, will constrain community transmission. Accordingly, we evaluate TTIQ strategies by estimating the percentage change in TP.

  The impact of even minor changes in TP on the local epidemiology depends critically on how close TP is to the national strategic objective of maintaining a control threshold of 1. If TP at the population level is very close to 1, even a small increase can be sufficient to drive a change from decreasing to increasing epidemic activity, the consequences of which depend on caseloads.

Findings

- Testing on Day 1 of quarantine supports timely identification and isolation of downstream infections and is always the most important test to prioritise

- Case-initiated contact tracing supports timely quarantine in times of system stress

- Reducing quarantine requirements for vaccinated individuals from 14 days to 7 days has no discernible impact on the performance of the TTIQ system. Completely removing isolation and/or quarantine for vaccinated individuals is estimated to increase TP by 3–5%.

- Prioritising the most recently identified cases for contact tracing increases impact of the TTIQ response, as late case finding is associated with diminishing returns on TP reduction

Monitoring of TTIQ impact is required for situation assessment. We describe data requirements and a framework for real-time evaluation of TTIQ impact on transmission.

Random population prevalence surveys would provide gold standard evidence of the number and proportion of infections by vaccination and symptom status over time.

Overview and key question

Overview: Doherty Modelling has clearly identified the critical importance of maintaining optimal test, trace, isolate and quarantine (TTIQ) capability as defined in our previous work into the next phases of our transition to living with COVID-19. This second piece of work has undertaken quantitative risk assessment to inform CDNA and AHPPC consideration of the future requirements of public health responses and inform planning for public health workforce.

Previously, as part of Phase 1 of the National Plan Modelling, we estimated the effectiveness of TTIQ on reducing transmission potential from case data: times from infection to isolation of cases. These delays from infection to isolation are the outcomes of contact tracing which we directly translated into reductions in transmission potential.

To address the key questions in this next phase of work, we now model how different proposed strategies affect transmission potential, via impacts on contact tracing delays, isolation, and quarantine strategies. This requires understanding of component delays and processes within the TTIQ system and the likely impact of vaccination and alternate strategies on system effectiveness.

Key question: What are the most effective and sustainable strategies for TTIQ to manage COVID-19 as vaccination rates increase in Phase A then Phases B and C to achieve the aim of constrained community transmission and avoid lockdown requirement?

National Plan Phases B and C context

The aim of TTIQ is to detect and place cases into isolation as soon as possible. If cases are already in quarantine at the time of onset of infectiousness, the risk to the community is lowered.

In our analyses below, we consider cases detected in the community via testing of symptomatic individuals (passive detection) and via testing of close contacts identified through contact tracing (active detection). Other means of enhanced case finding such as routine asymptomatic screening of individuals in high-risk settings are not included in our analyses. Such strategies, focused on workforce continuity, have the potential to further reduce transmission by increasing the overall proportion of cases ascertained.

The potential impact of TTIQ on transmission is limited by case ascertainment – people with undetected infections are not placed into isolation and so both they and their infected contacts will continue to contribute to transmission. Detection of unlinked symptomatic infections in people presenting for testing (passive symptomatic detection) enables reinitialisation of contact tracing on these undetected chains of transmission. Given that COVID-19 vaccines reduce the probability of developing symptoms, we anticipate lower levels of passive symptomatic detection with increasing vaccination coverage, for similar surveillance effort.

Compared to the pre-vaccination and pre-Delta era, transmission is likely to be concentrated in different population groups and settings (*e.g.*, low coverage groups, schools), which may require different TTIQ strategies and related public health responses.

As we transition to Phases B and C, the objective of TTIQ responses will explicitly change from supporting a goal of 'no community transmission', to one of 'constrained community transmission, that is maintaining transmission potential (TP) around 1'. As a result, TTIQ strategies will need to adapt to higher caseloads. This change in strategy implies a different objective of TTIQ: to reduce transmission in support of overall health and societal goals, rather than the previous aim of minimising the probability of *any* cases being missed and not placed into isolation. Adaptation of measures will therefore likely include pivoting contact tracing procedures to focus on reducing the time infectious in the community for the majority of cases, rather than identifying all downstream and upstream contacts of each case (as previously). With this re-framing, the goal is to identify

strategies where similar reductions in transmission potential can be achieved with a lower per case burden on the contact tracing system.

Key modelling assumptions

The estimated effectiveness of different TTIQ strategies will be sensitive to assumptions about infectiousness and test sensitivity as functions of time since infection, the probability of developing symptoms, and the probability of seeking a test given symptoms, for vaccinated and unvaccinated individuals.

Key biological parameters:

- Infectiousness over time since infection (for vaccinated and unvaccinated)
- Test sensitivity over time since infection (for vaccinated and unvaccinated)
- Probability of developing symptoms for vaccinated and unvaccinated infections

Key behavioural parameters:

- Probability of seeking a test given symptoms (for vaccinated and unvaccinated)

Links to previous work: Estimating TTIQ performance from case data

Figure 1 displays SARS-CoV-2 infection progression over time and how passive and active case detection may each reduce the proportion of infectiousness in the community.

Timely identification and quarantine of contacts (active detection) cuts off a much greater proportion of community infectiousness compared to isolation of symptomatic individuals (passive detection). This is because even if individuals seek a test promptly after symptom onset, they have already contributed several exposure days to the community before they knew they were infected and infectious. However, both modes of detection are critical to the overall impact of TTIQ, since the contact tracing process is initiated by passively detected cases.

Figure 1 also indicates how reductions in the proportion of infectiousness in the community due to passive symptomatic case finding and active contact tracing can be measured from case data. For Phase 1 of the National Plan Modelling, we used times from infection to isolation of cases to estimate the effectiveness of TTIQ on reducing transmission potential. We estimated that periods of 'Optimal' TTIQ reduced transmission potential by 54% and 'Partial' TTIQ reduced transmission potential by 42% (Figure 2).

To address the key questions in this next phase of work, we now model how different proposed strategies affect delays from infection to isolation. This requires understanding of each of the component delays within the TTIQ system and the likely impact of alternate strategies based on vaccination status of cases and/or contacts on system effectiveness.

Component delays include the time from symptom onset to test, test to case notification, case notification to case interview, case interview to contact notification. Figure 3 illustrates the timing of key TTIQ actions, these component delays, and the impact of longer delays on the timeliness of identification and quarantine of contacts.

Figure 1: Schematic of SARS-CoV-2 infection progression (**A**) and two modes of case detection: testing of symptomatic individuals (passive detection, **B**) and testing of close contacts identified through contact tracing (active detection, **C**). We also illustrate two key intervals for estimating TTIQ effectiveness from case data: times from infection to isolation and times from symptom onset to isolation. Isolation times enable more accurate measurement of TTIQ effectiveness than routinely available detection/notification times. Isolation times are not yet available in the National Notifiable Disease Surveillance System (NNDSS). Note: Both the displayed shape of the infectiousness curve (orange) and reductions in transmission are for illustrative purposes only, they do not correspond to specific model parameters and their impacts on transmission.

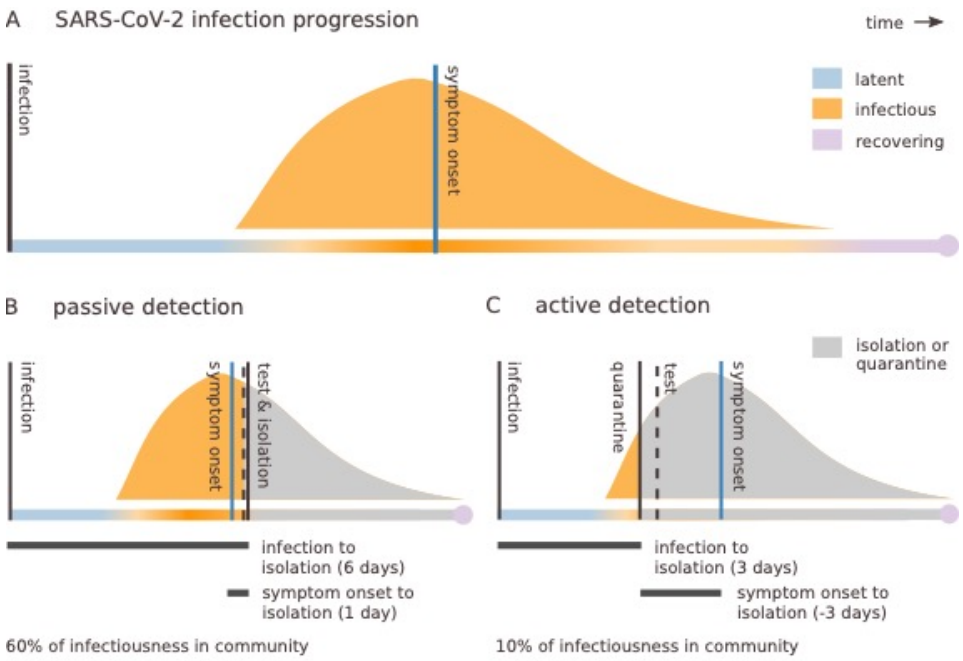


Figure 2: Percentage of cases isolated relative to time of infection for Optimal TTIQ (left) and Partial TTIQ (right) and corresponding reductions in transmission potential. Dashed vertical lines indicate the time of symptom onset.

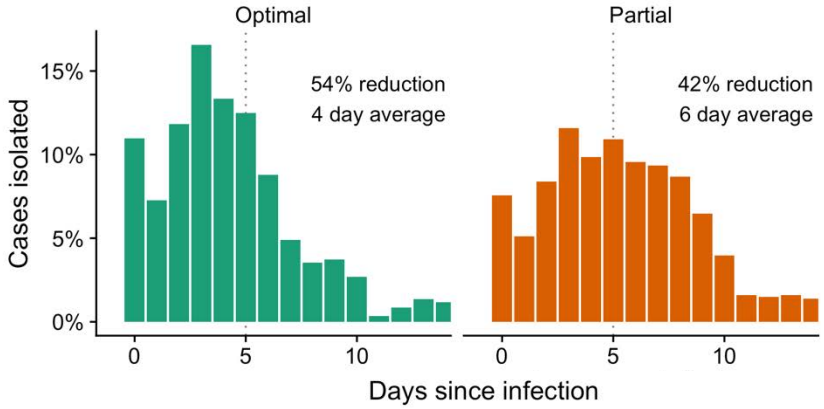
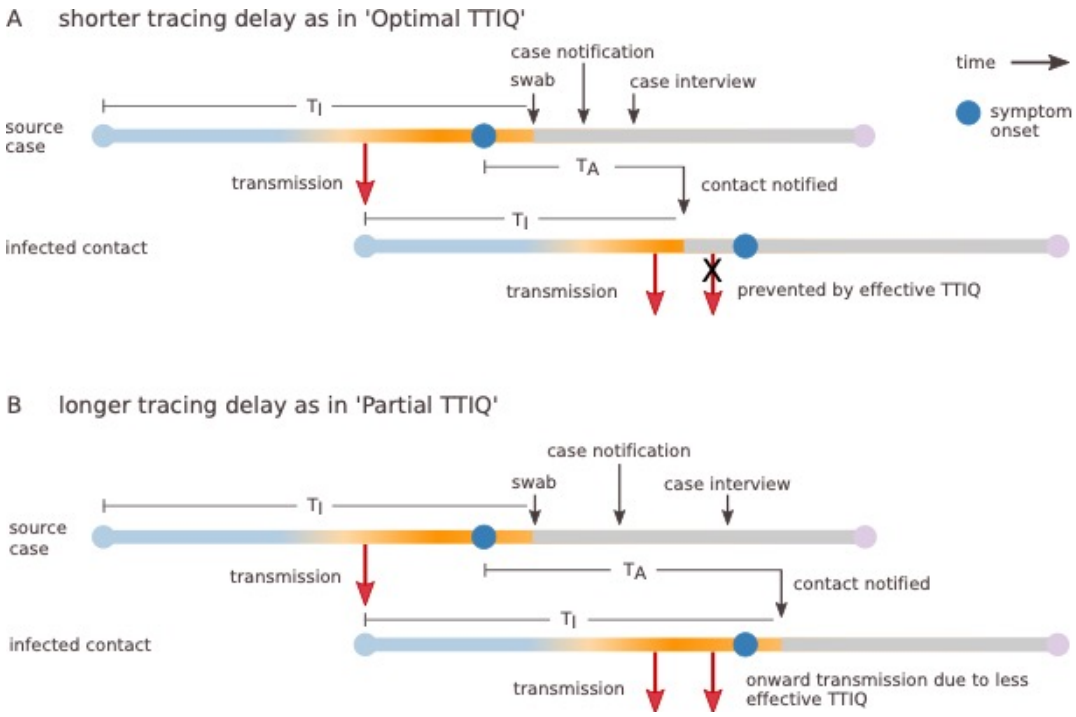


Figure 3: Representation of infection progression for a passively detected case (source case) and an infected contact, with the timings of key TTIQ actions indicated. TTIQ aims to reduce the time from infection to isolation (I_A). Longer tracing delays (T_A) result in longer times to quarantine/isolation and a greater proportion of infectiousness in the community (orange) compared to when delays are short. Further, when delays are long, isolation times of traced contacts are no faster than via passive detection (provided infected contacts develop symptoms).



Case ascertainment in Phases B and C

Detection of infections is central to the impact of TTIQ on transmission. The earlier a case is detected and isolated, the smaller the fraction of infectiousness in the community.

If a case is not detected at all, not only do they spend their entire infectious period in the community, but their infected contacts cannot be detected by downstream contact tracing, and so will be found later (through passive symptomatic detection), if at all.

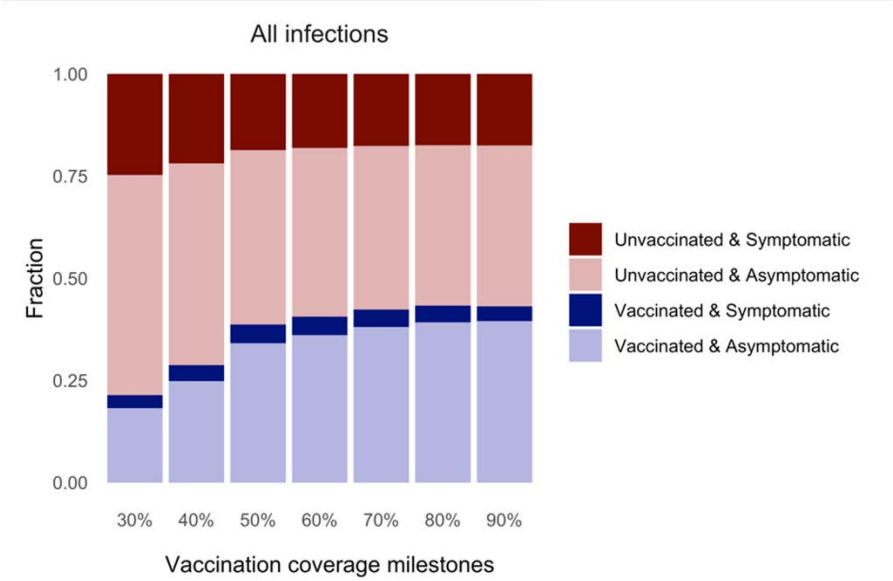
When an otherwise-unlinked infected individual develops symptoms and presents for testing (passive symptomatic detection), they enable public health units to isolate the case and re-start chains of contact tracing, placing more infected people in isolation more quickly. If the fraction of cases ascertained drops, fewer cases are placed into isolation and the TTIQ effect is lessened.

The fraction of cases ascertained by passive symptomatic detection is likely to reduce as Australia moves to higher population vaccination coverage in Phases B and C of the National Plan. COVID-19 vaccines reduce the probability of developing symptoms given infection. Higher rates of vaccination in adults than children will also result in infections being concentrated in children, who have a lower probability of developing symptoms than adults, with or without vaccination. We might therefore expect lower levels of passive symptomatic detection with increasing vaccination coverage, for similar surveillance effort.

Figure 4 displays the expected breakdown of all infections by vaccination and symptom status on the dates Australia crosses milestones of the proportion of the 16+ population fully vaccinated. This is calculated from Australia-wide vaccination coverages by age, vaccine type, and dose, and estimates of age- and location-structured population mixing and age-differences in susceptibility, contagiousness, and the probability of developing symptoms (as per Davies et al 2020 and updated susceptibility estimates). As vaccination coverage increases, the fraction of all infections that are vaccinated increases, and the fraction with symptoms (and therefore detectable by symptomatic screening) decreases.

While Figure 4 displays the split by vaccination and symptom status of all infections, differences in the probability of detection by vaccination and symptom status – driven by properties of the TTIQ system as well as behavioural choices – will result in a different fraction among cases.

Figure 4: Panel A shows the estimated proportion of asymptomatic and symptomatic **infections** stratified by vaccination status for different levels of vaccination coverage across all ages (Australia-wide coverage as per Australian Immunisation Registry (AIR) data and Quantium modelling).



Section 1: TTIQ strategies for evaluation

Table 1 outlines key TTIQ strategies for evaluation as identified through the consultation process undertaken with members of the Office of Health Protection, CDNA and AHPPC.

Table 1: TTIQ strategies for evaluation, expressed as modelling questions, targeting each component of the TTIQ system. The model framework used to address each question is also indicated. PCC = primary close contact. SCC = secondary close contact. TP = transmission potential.

TTIQ component	Modelling question	Model framework
Testing	A. What is the impact of no longer testing vaccinated symptomatic individuals?	Dynamic model
	B. What is the optimal testing schedule for quarantine of PCCs?	Quarantine model
Case interviews (trace)	C. What is the impact of case interview prioritisation based on risk and delays?	TP framework
	D. What is the impact of only contact tracing unvaccinated cases?	Dynamic model
Contact notification (trace)	E. What is the impact of case-initiated contact notification?	TP framework
Isolation	F. What is the impact of shortened isolation for vaccinated cases?	Dynamic model
PCC quarantine	G. What is the impact of no or shortened quarantine for vaccinated PCCs?	Dynamic model

Section 2: Predicted impact of proposed TTIQ strategies on transmission potential

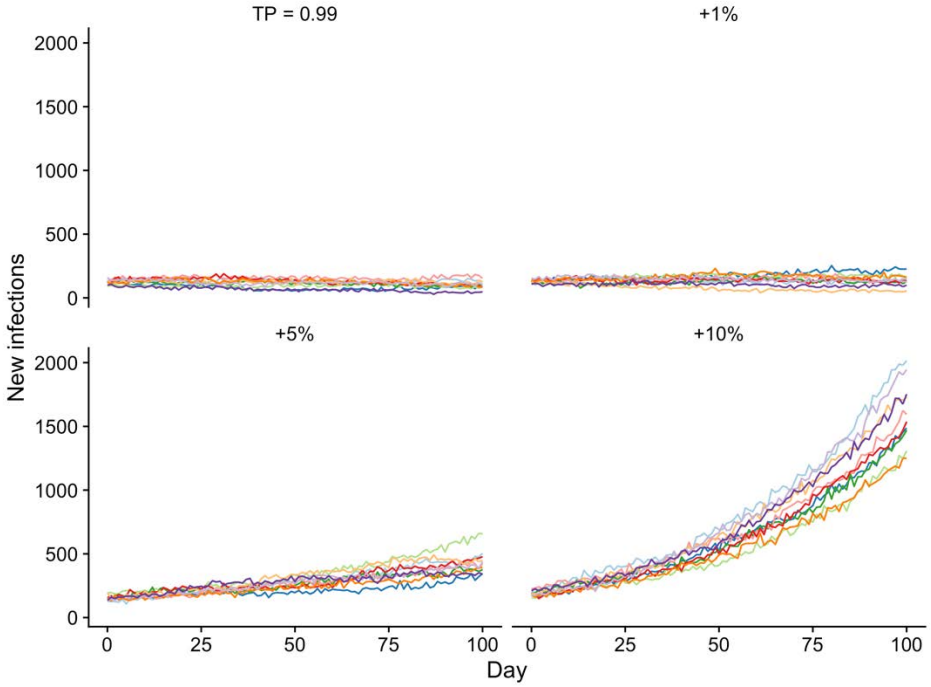
Evaluation and epidemiological context

We evaluate proposed TTIQ strategies by computing a change in transmission potential (TP) under each strategy compared to a reference strategy. The change in TP is reported as either a percentage change or multiplier depending upon the question under consideration.

The epidemiological impact of a change in TP depends strongly on the epidemiological context. The national strategy is to target a transmission potential at or around 1. Near this critical threshold, strategies that marginally increase the TP (by just a few percent) may drive a change from decreasing to increasing epidemic activity. In consequence, strategies need to be considered carefully as they may (ultimately) prompt the need for other measures, including the requirement for increased PHSMs. These considerations emphasise the need for monitoring of TTIQ impact on TP as part of routine epidemic assessment (see Section 3).

Figure 5 illustrates this concept by simulating timeseries of daily infections from our dynamic transmission model under a scenario when small increases in TP can have a strong effect. With a baseline TP of 0.99 epidemic activity is slowly declining. While an increase of 1% has a minor but noticeable effect, increases of 5% and 10% lead to escalating epidemics.

Figure 5: Example simulations of epidemic curves with modest increases in TP, but with an initial TP just below the control threshold of 1.



Modelling approaches and assumptions

To address the questions outlined in Table 1, we use three different modelling approaches. Each model contains features required for addressing a specific question. However, the following biological and behavioural assumptions were employed across all models:

- Probability of seeking a test given symptoms is 0.5.
- 31% of all infections are symptomatic (see Figure 4 above)
- Relative infectiousness of asymptomatics is 0.5.
- Probability that an infected individual is identified as a PCC via downstream manual contact tracing is 0.95.

Vaccine efficacies in the transmission potential framework and dynamic transmission model (against infection, symptomatic disease, and onward transmission) are taken to be the mid-points between the two-dose efficacies for Pfizer and AstraZeneca. The probabilistic quarantine model uses slightly more conservative VEs against onward transmission (0.4) and against infection (0.72).

Transmission potential framework with delays

A stochastic simulation model is used to represent the relationship between contact tracing delays, symptomatic detection, and times from infection to isolation in continuous chains of contact tracing. Sampling from distributions of contact tracing times, this model generates distributions of time from infection to isolation, which can be used to calculate expected reductions in transmission potential, as per figures 1 and 2. See Appendix for details.

Dynamic transmission model

An individual-based infection simulation model is used to simulate TTIQ processes in scenarios where ascertainment may be low (chains of transmission undetected). This model considers the vaccination and symptom status of cases (and therefore the ability to transmit and be detected), but without reduction of transmission due to infection-acquired immunity:

- Cases found by either downstream contact tracing from their source case or testing of symptomatic individuals, following the same process as the transmission potential model (supplementary figure 1).
- All cases are fully isolated when found (*i.e.*, assumes perfect compliance).
- Neither upstream contact tracing nor asymptomatic screening are in effect. With the above parameterization, and baseline TTIQ strategies in place, 38% of all infections are ascertained.
- Baseline isolation and quarantine are assumed to be 14 days from date of swab and date of identification as a case and PCC, respectively. We note that while the COVID-19 Series of National Guidelines (SoNG) recommends a 14-day isolation period for cases, multiple jurisdictions employ a 10-day isolation period.

Unless otherwise stated all analyses assume:

- National vaccination coverage (across all eligible age-groups) as predicted at the date Australia exceeds the threshold of 80% of the 16+ population fully vaccinated. As those aged 12-15 are eligible for the vaccine, the national coverage achieved and used in simulations accounts for current and predicted coverage in that age group.
- Contact tracing delay distributions are as estimated during the 'Optimal' period from NSW case data (directly provided by NSW Health).

In this model, the ratio of TPs (our key reporting metric) is insensitive to the underlying epidemic trajectory (growing or declining). Accordingly, an initial number of infections and the pre-vaccination reproduction number were calibrated for each analysis to ensure the ratio of TPs between strategy scenarios was reliably estimated.

Probabilistic quarantine model

Described in section 2B.

Testing

A. What is the impact of no longer testing vaccinated symptomatic individuals?

To evaluate the impact of no longer testing vaccinated symptomatic individuals via passive detection (*i.e.*, individuals who have not been identified as a PCC), we use the dynamic transmission model.

Note that symptomatic vaccinated individuals who are PCCs of known cases are still tested and their contacts are traced.

We evaluate the impact of this strategy on *transmission potential*. However, testing of symptomatic vaccinated individuals supports other epidemiological and public health priorities including detection of immune evading variants of concern and monitoring of vaccine effectiveness. These other epidemic surveillance objectives are not captured by our analysis.

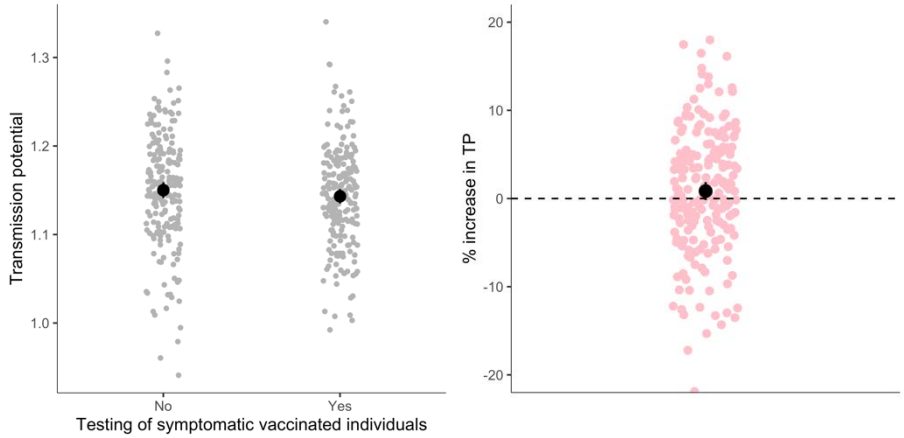
We compute the percentage change in TP between two scenarios:

- 50% of *vaccinated* symptomatic infections are detected via passive screening.
- 0% of *vaccinated* symptomatic infections are detected via passive screening.

In both scenarios, 50% of *unvaccinated* symptomatic infections are detected via passive screening. Asymptomatic infections irrespective of vaccination status can only be identified via contact tracing.

The model is calibrated to a population vaccination coverage corresponding to the 80% national coverage milestone and a pre-vaccination TP of 5.5.

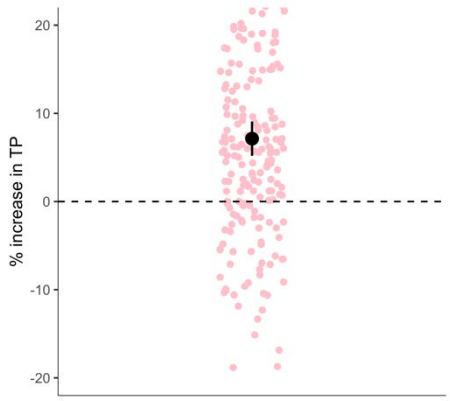
Figure 6: Left: Estimated transmission potential for each of 200 model simulations (grey dots) assuming testing and no testing of symptomatic infected individuals via syndromic surveillance (black dots and lines = mean ± 2SE). **Right:** Percentage increase in TP for randomly paired simulations (pink dots). Black dots and lines show the mean estimated increase in TP of 0.84% (± SE 0.51%). Simulations were initialised with 100 infections, population vaccination coverage corresponding to the 80% national coverage milestone and a pre-vaccination TP of 5.5. Transmission potentials were calculated from a time-average of secondary infections from each infection between days 20 and 50 of each simulation.



Our analysis shows a marginal increase in transmission potential as a consequence of this strategy. Symptomatic infected individuals make up a small proportion of all infections (Figure 6) and are less infectious compared to unvaccinated symptomatic infections.

While this result is unsurprising in terms of anticipated impact of the strategy on whole-of-population transmission dynamics, in certain sub-populations the impact may be different. In Figure 7 we consider a sub-population with high vaccination coverage (90%), a high symptomatic fraction of infections (60%), and a high probability of seeking a test given symptoms (80%), such as a retirement village or township with a particular demographic profile. In this scenario, not testing symptomatic vaccinated individuals results in an increase in TP of approximately 7%. As illustrated in Figure 5, such an increase may drive a change from decreasing to increasing epidemic activity.

Figure 7: Percentage increase in TP for 200 paired simulations (pink dots). Black dots and lines show the mean estimated increase in TP of 7.13% increase (± SE 0.97). All simulations were initialised with 1000 infections and assume 80% of symptomatic individuals present for a testing, 60% of all infections are symptomatic, 90% of the entire population are fully vaccinated, and a pre-vaccination reproduction number of 5.0. Transmission potentials were calculated between days 10 and 300 of each simulation.



B. What is the optimal testing schedule for quarantine of primary close contacts?

Quarantining and testing of primary close contacts (PCCs) aims to reduce transmission in two ways:

- By directly preventing onward transmission from infected PCCs
- By enabling timely tracing of the contacts of infected PCCs

We use a probabilistic model to determine optimal testing schedules for quarantine of primary close contacts that meets both objectives. Accordingly, we measure the impact of each quarantine testing strategy by calculating the average number of infections arising from those infected by the PCCs (herein "IPq").

The model incorporates the following features:

- Infected PCCs can infect others (in the community or household) prior to being identified and placed in quarantine.
- Infected PCCs in quarantine can only infect their household members.
- PCCs not identified through the testing schedule (or if there are no tests) continue to be able to infect household members and are released into the community while potentially still infectious.
- The time taken to identify and isolate the PCC (and thus those they infect) will depend on the current performance of the TTIQ system (e.g., optimal or partial delays).

All analyses make the following assumptions:

- 85% vaccination coverage in 12+ ages which maps to 72% population-level coverage.
- PCCs are assumed to be vaccinated in proportion to the population coverage. This results in approximately 20% of infected PCCs being vaccinated.
- We induce a correlation between the vaccination status of the PCC and their household members. This means that unvaccinated PCCs (who are more likely to transmit the infection) are more likely to have unvaccinated household members (who are more susceptible to infection). We assume household members of a PCC have a 90% chance of having the same vaccination status as the PCC.

- The pre-quarantine, pre-vaccination TP is assumed to be 5. Number of secondary cases are simulated from a Negative Binomial distribution with mean TP, and over-dispersion parameter (k=) 0.2.

We investigate optimal test timing for different quarantine durations (7-day or 14-day) and numbers of tests (1, 2, or 3). Tests may be conducted on any day from day 1 of quarantine through to the final day of quarantine inclusive. The first test must be conducted no later than on day 5 if conducting more than one test and tests must be separated by at least one day.

Figure 8: Estimated IPq for testing strategies where one test is conducted during the 7- or 14-day quarantine period, assuming 'Optimal' delays. The black dot indicates the testing strategy with the lowest IPq, *i.e.*, the optimal strategy in terms of transmission reduction.

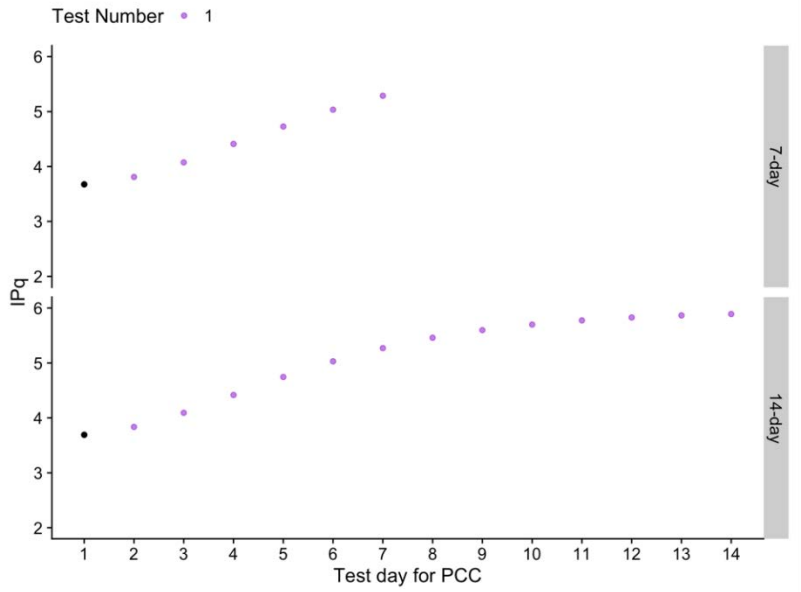


Figure 9: Estimated IPq for testing strategies where two tests are conducted during the 7- or 14-day quarantine period, assuming 'Optimal' delays. Each pair of purple (test 1) and green (test 2) dots, joined by a horizontal line, represent a single testing strategy. The paired black dots indicate the testing strategy with the lowest IPq, *i.e.*, the optimal strategy in terms of transmission reduction.

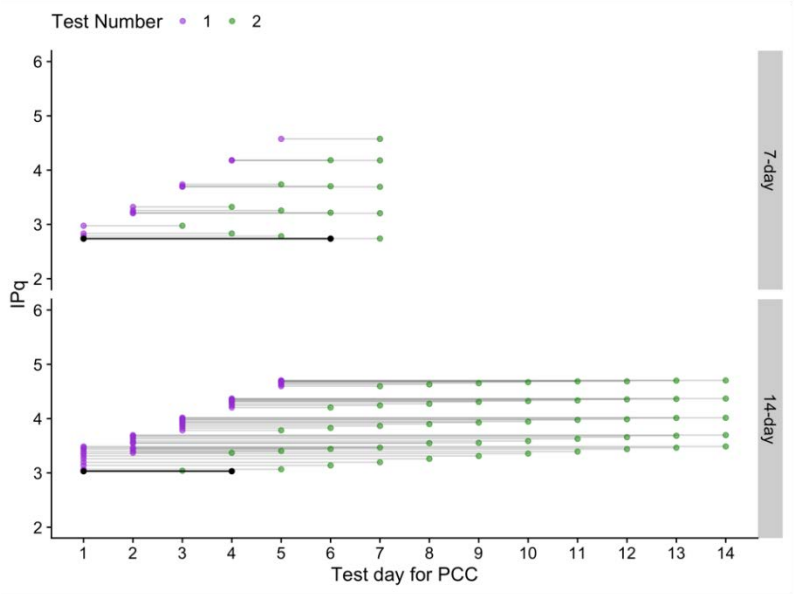


Figure 10: Estimated IPq for testing strategies where three tests are conducted during a 14-day quarantine period, assuming 'Optimal' delays. Each triple of purple (test 1), green (test 2) and orange (test 3) dots, joined by a horizontal line, represent a single testing strategy. The trio of black dots indicate the testing strategy with the lowest IPq, *i.e.*, the optimal strategy in terms of transmission reduction.

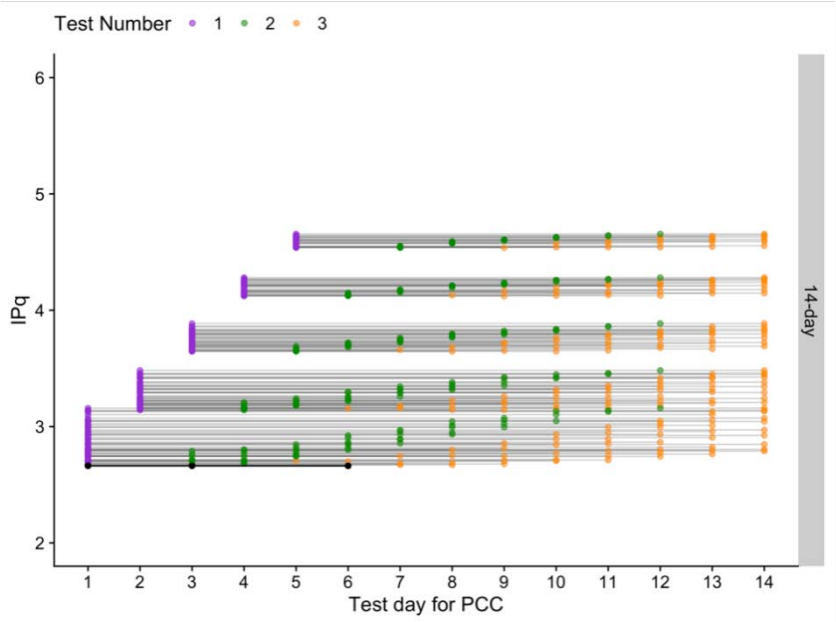


Table 2: Optimal testing strategies for each of 1–3 tests, under 7- and 14-day quarantine, for 'Optimal', NSW current case-initiated, and 'Partial' contact tracing delays.

		Delay distribution		
Quarantine duration (days)	Number of tests	Optimal	Partial	NSW case Initiated
14	3	(1,3,6)	(1,3,10)	(1,3,8)
14	2	(1,4)	(1,4)	(1,4)
14	1	(1)	(1)	(1)
7	2	(1,6)	(1,7)	(1,6)
7	1	(1)	(1)	(1)

Figures 8–10 and Table 2 demonstrate the importance of testing on Day 1 of quarantine. A day 1 test is included in the optimal strategy for all explored quarantine durations and testing schedules. This is because early identification of PCCs that are infected is important for timely identification and isolation of the individuals that they have infected.

Under a 7-day quarantine strategy, a later second test is preferred (e.g., 1, 6), to increase the chance of identifying cases prior to release from quarantine. Under a 14-day quarantine strategy, two early tests are optimal to increase the chance of early identification of infected PCCs and their contacts.

Figures 9 and 10 show that for strategies with 2 or 3 tests, several testing strategies perform similarly well to the optimal testing strategy (i.e., there is some flexibility in testing day). In Table 3 we present a range of testing days for each test that correspond to no more than a 2% loss in strategy performance.

Table 3: Range of testing days for each test that correspond to no more than a 2% loss in strategy performance.

		Delay distribution		
Quarantine duration (days)	Number of tests	Optimal	Partial	NSW Case Initiated
14	3	1, 3–4, 5–11	1, 3–12, 5–14	1, 3–4, 5–12
14	2	1, 3–5	1, 3–14	1, 3–5
14	1	1	1	1
7	2	1, 5–7	1, 3–7	1, 5–7
7	1	1	1	1

When TTIQ system performance is consistent with 'Partial' delays (i.e., PCCs are identified later than under 'Optimal' or current NSW case-initiated delays), in addition to testing on Day 1, testing on nearly any other day is sufficient, resulting in a minimal loss of performance.

Figure 11: Performance of optimal testing strategies (as per Table 1) under each of the 'Optimal', 'Partial', and NSW current case-initiated delays, for 7- and 14-day quarantine.

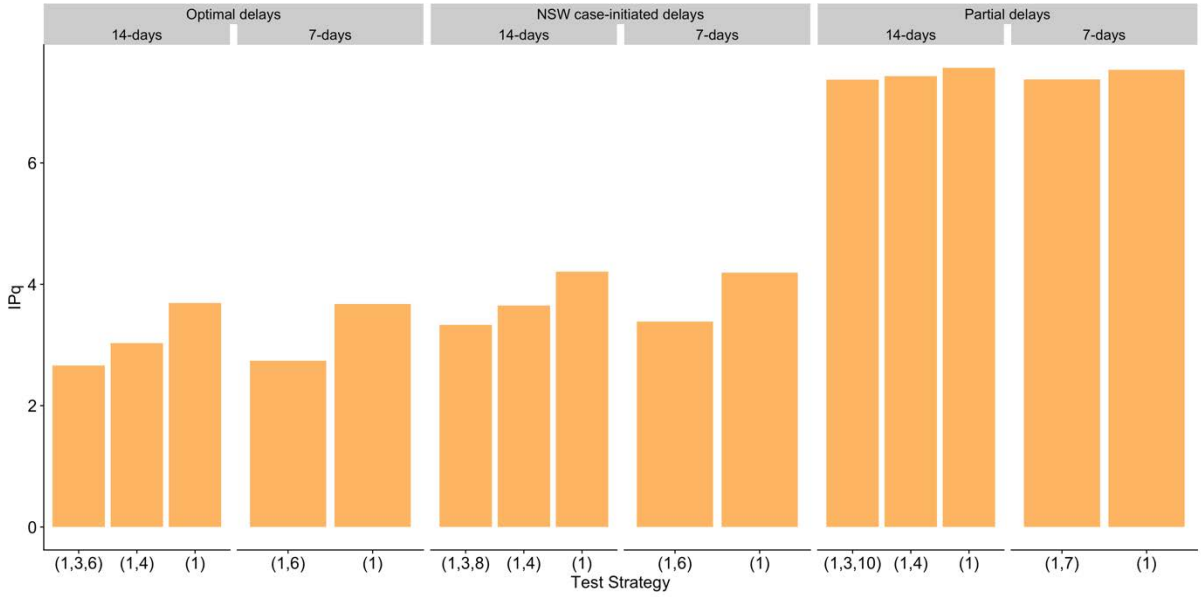


Figure 11 shows that the reduction in transmission achieved by conducting additional optimally timed tests is smaller than the loss of system performance due to increased delays (*e.g.*, from 'Optimal' to 'Partial'). It may therefore be favourable to perform fewer tests for quarantining PCCs if that supports improvements to system performance through a reduction in the time to identify and isolate cases.

In Figure 12, in the context of optimal testing strategies, we explore the impact of differential strategies for quarantine of vaccinated and unvaccinated PCCs on system performance. These results have relevance to Question G.

Figure 12: Evaluation of 7-day quarantine for vaccinated PCCs under 'Optimal' (left) and 'Partial' (right) delays. The reference strategy where both vaccinated and unvaccinated PCCs quarantine for 14 days (left bar in each facet) is compared to two strategies in which vaccinated PCCs quarantine for 7 days. In the first, optimal scheduled testing is present for vaccinated PCCs (middle bar). In the second, there is no scheduled testing (right bar). Under both strategies, unvaccinated PCCs quarantine for 14 days. Where testing is implemented, the optimal strategy for the corresponding delay distribution and quarantine duration is implemented as per Table 1 (*e.g.*, (1,3,6) for 14-day quarantine with 'Optimal' delays, and (1,3,10) with 'Partial' delays).

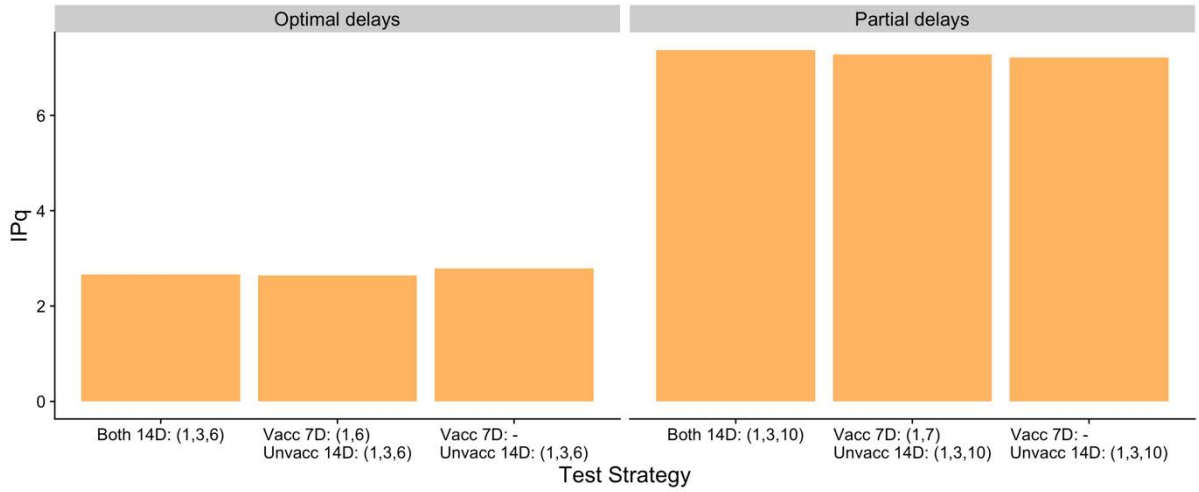


Figure 12 shows that reducing quarantine duration from 14 to 7 days for vaccinated PCCs has no discernible impact on the performance of the TTIQ system. Furthermore, not testing vaccinated PCCs during a 7-day quarantine period has minimal impact on performance. These results follow from the high chance that infections are detectable within the first 7-day period, and that vaccinated PCCs are less likely to acquire infection and, if infected, are less infectious.

Note however that further reductions in quarantine duration for vaccinated PCCs may result in increases in overall transmission, as explored below in Question G.

Tracing

C. What is the impact of case interview prioritisation based on risk and delays?

When the TTIQ system is under stress due to high caseloads, it may no longer be possible for public health units to complete all case interviews on the same day as case notification.

We explore the impact of different strategies for case interview prioritisation by using outputs from a queuing model within the TP framework model.

Queuing model

- Each day a random number of cases (drawn from a time-homogeneous Poisson distribution) are added to the interview queue and a fixed number of cases in the queue are interviewed.
- Cases may be prioritised for interview according to the time since test swab and/or vaccination status.
- Any cases not interviewed within 5 days of notification are removed from the queue (*i.e.*, never interviewed).
- Independent of capacity, we assume that 20% of cases cannot be interviewed on their date of notification due to a range of reasons such as missing contact details or out-of-hours notification.

We examine the impact of four different case interview prioritisation strategies:

1. No prioritisation (*i.e.*, random)
2. Prioritise the most recently swabbed cases
3. Prioritise unvaccinated cases and then the most recently swabbed cases
4. Prioritise the most recently swabbed cases and then unvaccinated cases

We explored these strategies under three different case interview capacities (20%, 50%, 80%). This capacity corresponds to the proportion of average daily incoming cases that the public health unit can interview.

Note that at 100% capacity, the model would assume 80% of cases are interviewed on the date of notification and 20% on the following day. Since some observed times from notification to interview during the 'optimal' TTIQ period in NSW were longer than 1 day (Figure 15, top middle panel), at 100% capacity the model would predict a higher effect than the optimal TTIQ. This is because the model only assesses the impact of prioritisation and does not consider the potential for longer delays for other reasons. We suggest that the TTIQ effect at 80% capacity can be broadly interpreted as representative of the optimal TTIQ scenario, and reductions in TTIQ effect at lower capacities considered relative to that benchmark.

Figure 13: Estimated reduction in transmission potential under four case interview prioritisation strategies: 1) No prioritisation ("Random"). 2) Prioritise the most recently swabbed cases ("New cases"). 3) Prioritise unvaccinated cases and then the most recently swabbed cases ("Unvaccinated then new cases"). 4) Prioritise the most recently swabbed cases then unvaccinated cases ("New cases then unvaccinated"). Results are plotted for three different case interview capacities (20%, 50% and 80%).

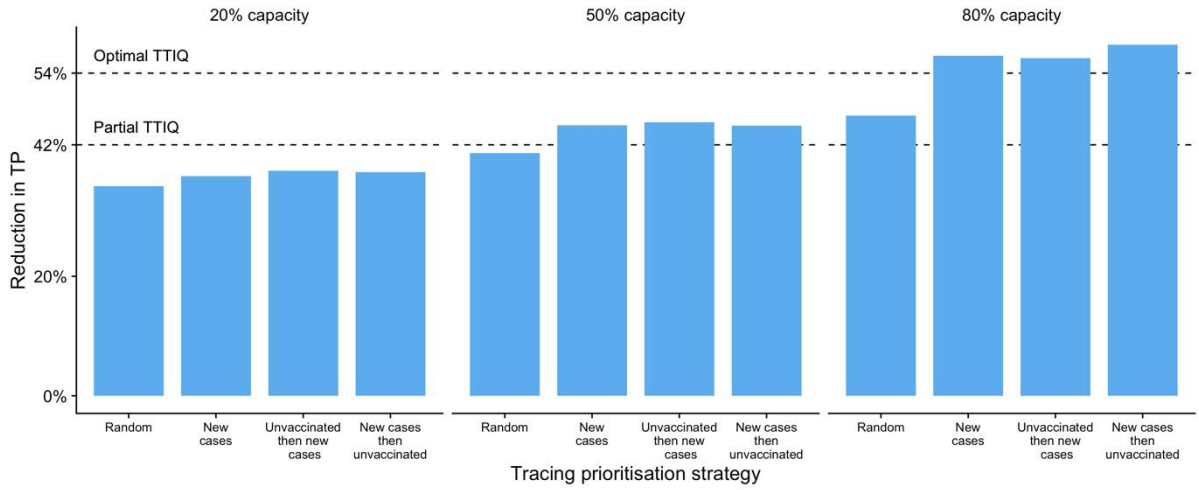


Figure 13 shows that prioritising interviews of the most recently swabbed cases yields the greatest gains in transmission reduction, regardless of contact tracing capacity.

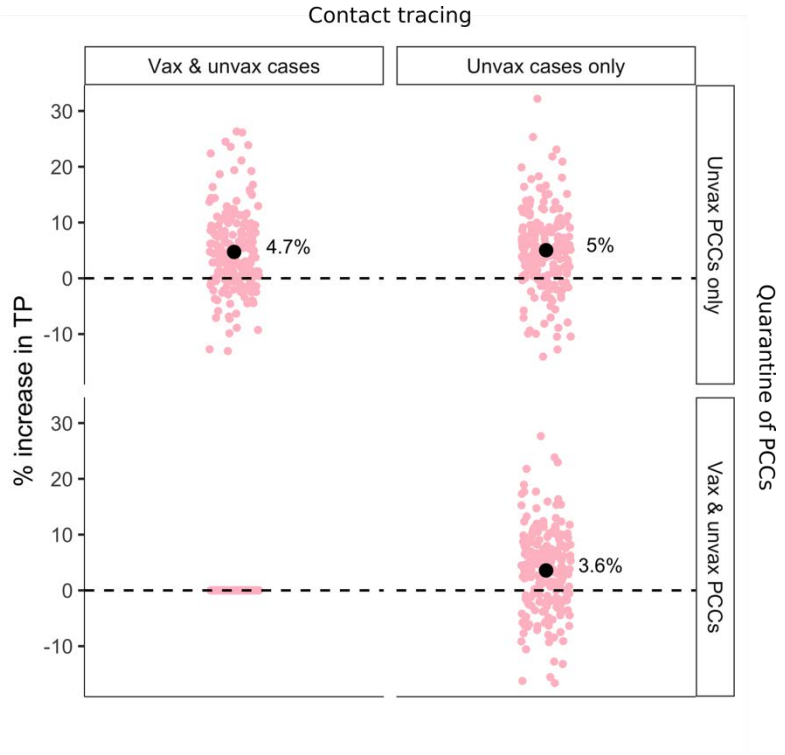
D. What is the impact of only tracing contacts of unvaccinated cases?
G. What is the impact of no or shortened quarantine for vaccinated primary close contacts?

We address questions D and G together, using the dynamic transmission model. We consider a reference strategy where contact tracing of both vaccinated and unvaccinated cases occurs and both vaccinated and unvaccinated PCCs of those cases are placed into quarantine (Figure 14, bottom left).

We then estimate the percentage change in transmission potential between this reference strategy and the following three strategies:

- Contact tracing of both vaccinated and unvaccinated cases is performed, and only unvaccinated PCCs are placed into quarantine (Figure 14, top left)
- Only contact tracing of unvaccinated cases is performed, and both unvaccinated and vaccinated PCCs are placed into quarantine (Figure 14, bottom right)
- Only contact tracing of unvaccinated cases is performed, and only unvaccinated PCCs are placed into quarantine (Figure 14, top right).

Figure 14: Mean percentage change in TP estimated for three TTIQ strategies compared to a reference strategy (black dots) across 200 paired simulations (pink dots). **Bottom left (reference):** Contact tracing of both vaccinated and unvaccinated cases occurs and both vaccinated and unvaccinated PCCs quarantine. **Top left:** Contact tracing of both vaccinated and unvaccinated cases is performed, and only unvaccinated PCCs quarantine. **Bottom right:** Only contact tracing of unvaccinated cases is performed, and both unvaccinated and vaccinated PCCs quarantine. **Top right:** Only contact tracing of unvaccinated cases is performed, and only unvaccinated PCCs quarantine. Simulations were initialised with 100 infections, population vaccination coverage corresponding to the 80% national coverage milestone and a pre-vaccination reproduction number of 5.5. Transmission potentials were calculated between days 20 and 50 of each simulation.



Our analysis shows that ceasing contact tracing for vaccinated cases will increase the TP by approximately 3–4% (bottom right). Similarly, removal of quarantine requirements for vaccinated PCCs will also increase the TP, by an estimated 4–5% (top row).

The increase in TP when vaccinated PPCs are not quarantined contrasts with the finding that a reduced quarantine duration for vaccinated PCCs of 7-days results in minimal additional risk (Question B). This result follows from the fact that a much higher proportion of infected individuals become infectious within the first 7-day period since infection compared to the second 7-day period.

As described at the beginning of this report, with a national plan seeking to constrain community transmission by targeting a transmission potential around 1, a small percentage increase *may* have a significant epidemiological impact. Accordingly, whether or not adjustments to TTIQ processes based on vaccination status (driving a change in the TP) could be considered will depend upon evaluation of the epidemiological context and the expected resultant transmission potential.

E. What is the impact of case-initiated contact notification?

During the NSW outbreak of the Delta variant, seeded in mid-June 2021, NSW Health implemented a policy of case-initiated contact notification to reduce delays associated with high case numbers. This strategy involves instructing confirmed cases to self-identify their primary close contacts and ask them to get tested and quarantine.

We use the TP framework model to consider the likely impact of this approach on transmission potential, given some assumptions about compliance and the proportion of contacts that can be ascertained by this means (see Appendix for details). Empirical and modelled distributions for each scenario and contact tracing delay are shown in Figure 15

We validate our simulations by relating them to available data on timeliness of case isolation from NSW over various time periods. These data were provided by NSW Health.

NSW scenarios

We applied our model to consider three scenarios for NSW:

- *optimal* – the period from July 2020–February 2021 representing 'optimal' TTIQ in the National Plan Modelling report, without case-initiated contact tracing
- *current without case-initiated* – a four-week period commencing August 15 2021, without case-initiated contact tracing,
- *current with case-initiated* – as for current, but with an assumption that 80% of infected contacts are immediately identified by the case.

During the 'current with case-initiated' scenario, we assumed that 80% of close contacts were readily identifiable by the case (e.g., household contacts). This high proportion reflects the fact that stay-at-home restrictions during this period will minimise the number of social contacts and concentrate infected contacts in household and essential workplace settings where contacts are fewer and more easily identifiable. We would expect the fraction of cases found by case-initiated contact tracing to be less under less stringent restrictions.

Results and interpretation

The left most panel of Figure 16 shows that our model can re-produce the TP reduction calculated from observed distributions of times from infection to isolation ('optimal TTIQ' estimated for Phase 1 of the National Plan Modelling, 54% reduction).

The middle panel shows that with a high level of case-initiated contact tracing, current contact tracing delays in NSW can still achieve similar reductions in transmission potential as for the 'partial TTIQ' (42% reduction) estimated for Phase 1 of the National Plan Modelling.

The right most panel shows that the current contact tracing delays in NSW (mid-August to mid-September 2021) would be predicted to result in a much smaller reduction in TP if case-initiated contact tracing (or other strategies to reduce times to isolation) were not in place.

Figure 17 displays estimates of times from infection to isolation from case data. The right most panel (NSW current, mid-August to mid-September 2021) suggests that our model predictions of TP reduction for the case-initiated contact tracing scenario broadly align with estimates from case data in NSW. Both our predictions and the estimates from case data are close to the 'partial TTIQ' benchmark.

Figure 15: Modelled distributions of various delays in the contact tracing process as estimated from NSW data provided by NSW Health (dots = data). These distributions are used as inputs in our model of TTIQ impact on transmission potential (TP). Time from swab to notification and notification to interview are informed by NSW data from July 2020 to February 2021 ('optimal', row 1) and from mid-August to mid-September 2021 ('current without case-initiated', row 2). 'Other delays' is calibrated to match the overall distribution of delays from infection to isolation for the 'optimal' period and has a mean delay of one day. This represents all other delays in the contact tracing process that we are not yet to estimate from data. For example, the time from interview to contact notification and the time from contact notification to isolation. 'current with case-initiated' (row 3) assumes the same delays as for 'current without case-initiated' except that 80% of notification to interview delays are set to zero. This represents a high proportion of contacts being immediately advised by the case to isolate (*e.g.*, household contacts).

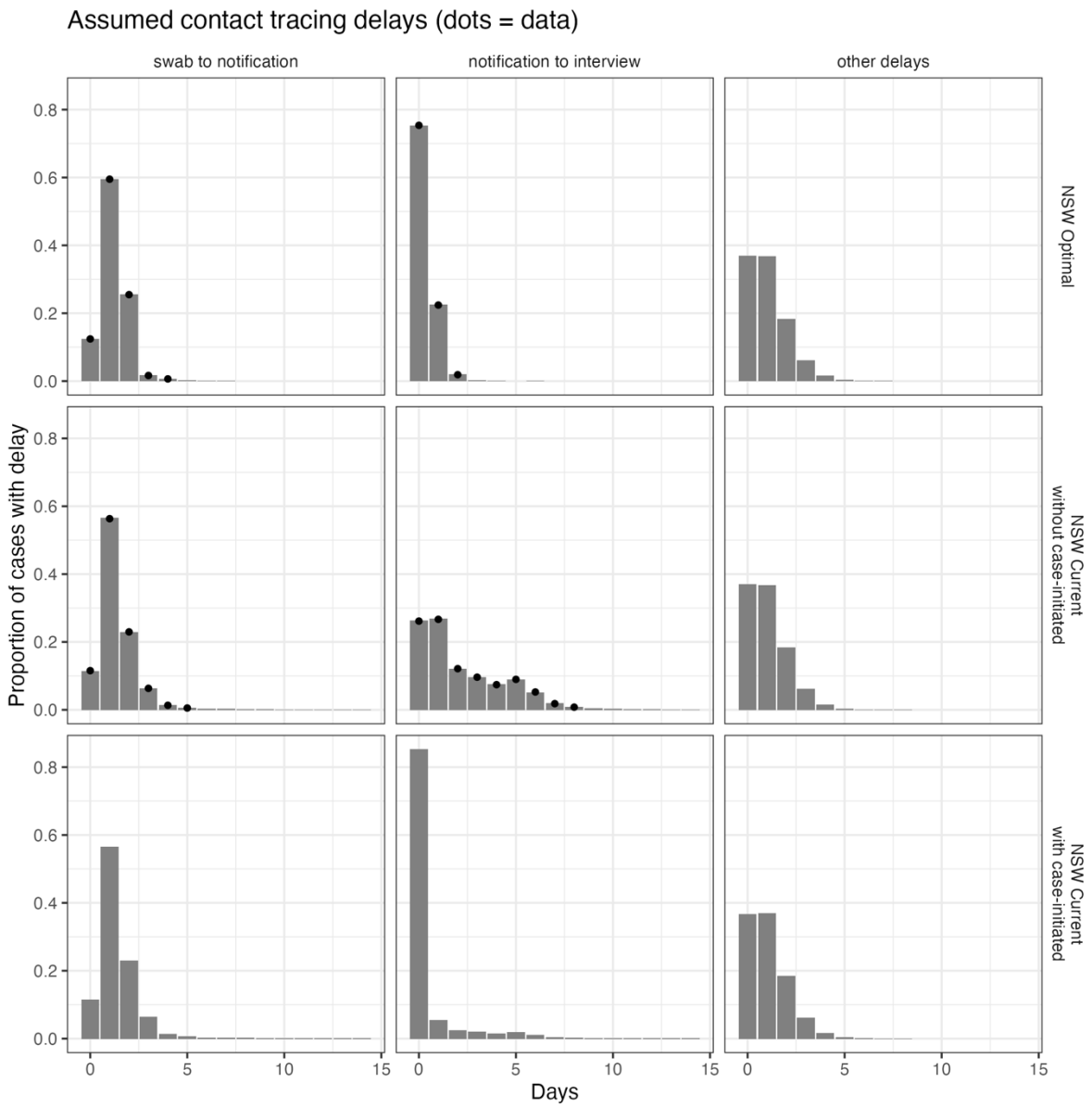

Assumed contact tracing delays (dots = data)
swab to notification
notification to interview
other delays
NSW Optimal
NSW Current without case-initiated
NSW Current with case-initiated
Proportion of cases with delay
0.0
0.2
0.4
0.6
0.8
0
5
10
15
Days

Figure 16: Distribution of delays from infection to isolation, and the resulting % reduction in transmission potential, **predicted by our model** under three delay scenarios (as outlined in Figure 15). Dashed vertical lines indicate the time of symptom onset.

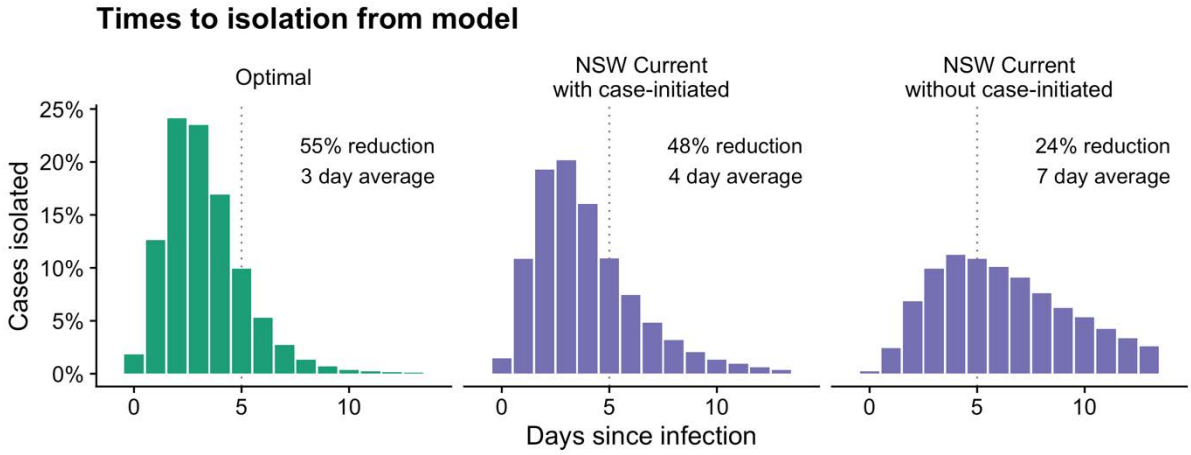
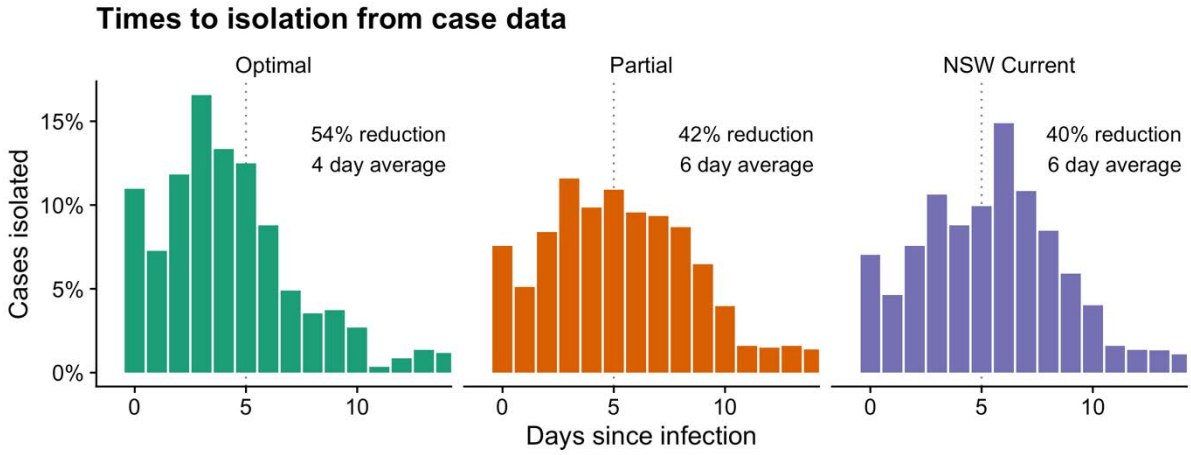


Figure 17: Distribution of delays from infection to isolation, and the resulting % reduction in transmission potential, **estimated from case data** under three delay scenarios (as outlined in Figure 15). 'Optimal' is times from infection to isolation from NSW case data between July 2020 and January 2021, provided by NSW Health. The distribution of times from infection to isolation for 'Partial' and 'NSW Current' are extrapolated from 'Optimal' based on delays from symptom onset to notification measured for VIC on 4 August 2020 (Partial) and for NSW on August 15 2021 (NSW Current). Dashed vertical lines indicate the time of symptom onset.



Isolation and quarantine

F. What is the impact of shortened isolation for vaccinated cases?

To evaluate the impact of shortened isolation from 14 to 7 days for vaccinated cases, we use the dynamic transmission model.

We compute the percentage change in TP between two scenarios:

- Unvaccinated and vaccinated cases isolate for 14 days
- Unvaccinated cases isolate for 14 days and vaccinated cases isolate for 7 days.

Figure 19: Left: Estimated transmission potential (TP) from each of 200 model simulations (grey dots) assuming 14-day and 7-day day isolation for vaccinated cases (black dots and lines = mean ± 2SE). **Right:** Percentage increase in TP for paired simulations (pink dots). Black dots and lines show the mean estimated increase in TP of 1.21% (± SE 0.622)**.** Simulations were initialised with 100 infections, population vaccination coverage corresponding to the 80% national coverage milestone and a pre-vaccination reproduction number of 5.5. Transmission potentials were calculated between days 20 and 50 of each simulation.

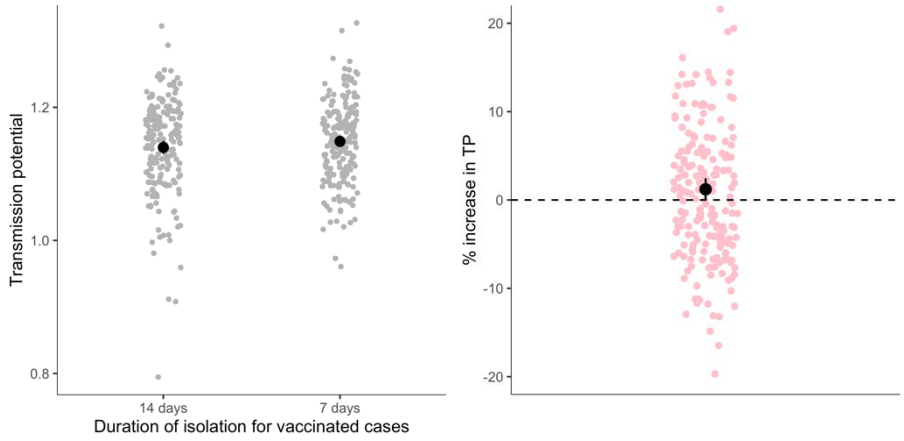


Figure 19 shows a marginal increase in transmission potential as a consequence of shortened isolation from 14 to 7 days for vaccinated cases. This result follows from the high fraction of all infectiousness that occurs in the days around symptom onset.

G. What is the impact of no or shortened quarantine for vaccinated primary close contacts?

Addressed under Question D.

Section 3: Monitoring TTIQ performance

TTIQ is an interdependent system that relies on public health capabilities, community participation, response objectives, and the status of the epidemic. Monitoring the effectiveness of TTIQ at reducing transmission is required to understand the reasons for changes in transmission rates (as measured by the reproduction number) and to anticipate the need for other localised measures, such as PHSM, to maintain a target level of outbreak control.

A monitoring system needs to meet two objectives:

- Measure the overall performance of the TTIQ system
- Measure components of the TTIQ system to enable identification of the source(s) of lowered system performance (if relevant)

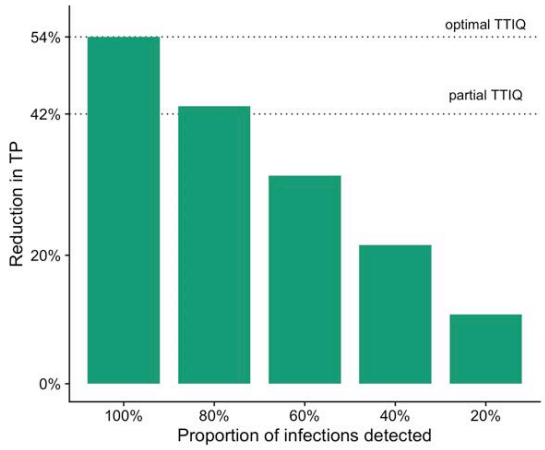
Overall indicator of TTIQ system performance

The principal indicator of TTIQ system performance at controlling outbreaks is the percentage reduction in transmission potential due to TTIQ. This depends on both how quickly cases are found and isolated (Figure 1), and what proportion of infections are detected.

To date in Australia, TTIQ responses have included extensive contact tracing and epidemiological investigation, with the aim of identifying all infections in chains of transmission. This has included strategies such as upstream contact tracing and asymptomatic screening. Consequently, ascertainment of infections has so far been very high. The percentage reduction in transmission potential is approximately proportional to the fraction of infections detected. For example, if only

half of infected people are detected, only half of infections can have any onward transmissions averted due to isolation (Figure 20)

Figure 20: Change in the TTIQ effect (reduction in TP) as ascertainment decreases.



The main overall indicator is the TTIQ effect: the percentage reduction in transmission potential due to TTIQ (Figure 21, panel 1). The TTIQ effect is itself the product of two components (Figure 21, panel 2): the TTIQ effect for *detected* infections (*i.e.*, cases) and the fraction of infections ascertained (case ascertainment). In turn, these indicators can be inferred, using epidemiological knowledge and models, from component indicators and case data (Figure 21, panel 3).

Estimating the TTIQ effect

TTIQ effect for cases

TTIQ reduces transmission through the detection and isolation of cases. The earlier a case is detected and isolated, the smaller the fraction of infectiousness in the community.

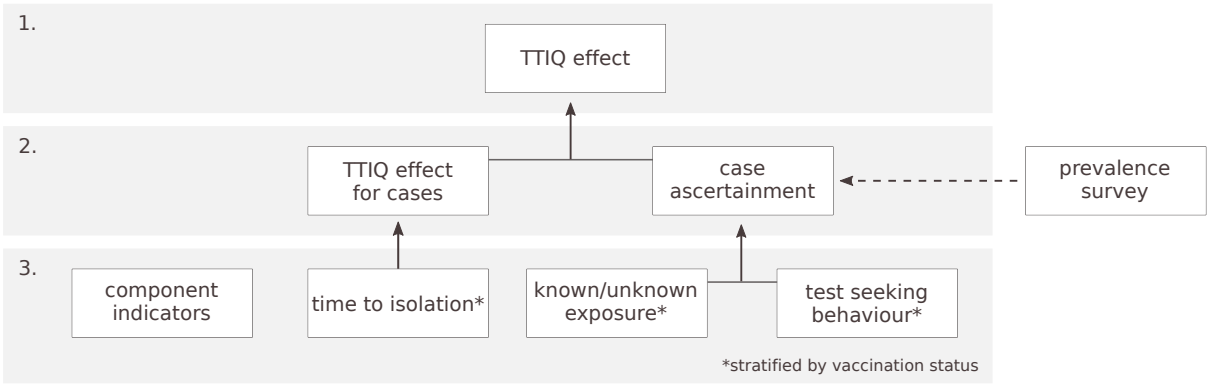
We have a developed a method that uses times from symptom onset to isolation of cases (Figure 1, panel 3) to estimate the percentage reduction in transmission potential for detected infections due to TTIQ.

Case ascertainment

To understand the overall impact of TTIQ on transmission, we need to estimate the fraction of infections which are undetected (and thus unlikely to isolate). To accurately estimate the level of case ascertainment requires temporal information on the prevalence of infection in the community. The UK has been undertaking random population screening of 150,000 people (approximately 0.2% of the population) regardless of symptoms each fortnight throughout the pandemic. This prevalence survey has provided an objective assessment of the total number of both asymptomatic and symptomatic infections over the course of their epidemic by age group and region. These observations are now differentiated by vaccination status, enabling estimation of vaccine effectiveness.

It may be possible to infer the fraction of infections ascertained over time using a model fitted to data on the proportion of cases with known versus unknown exposure at the time of test and other test seeking behaviours, stratified by vaccination status (Figure 21, panel 3). Estimates from such a model would be uncertain and would need to be validated, motivating the need for future consideration of prevalence surveys in Australia.

Figure 21: Elements required to estimate the TTIQ effect (percentage reduction in transmission potential due to TTIQ).



Data requirements for estimating the TTIQ effect

TTIQ effect for cases:

- Date of symptom onset for each case (NNDSS) to compute times from infection to isolation
- Date last in the community for each case (NNDSS) to compute times from infection to isolation
- Vaccination status of each case (NNDSS) because vaccination reduces the probability of onward transmission in infected vaccine recipients.
- Place of acquisition (NNDSS) for each case to determine whether the case has arisen from the local epidemic or was infected overseas/interstate.

Level of case ascertainment:

- Estimates of the prevalence of infection (TBD)
- Probability of seeking a test given symptoms consistent with COVID-19 for vaccinated and unvaccinated individuals in the general population (national behavioural surveys and Flutracking)
- Vaccination status of each case (NNDSS) because:
    - Vaccination reduces the probability of developing symptoms given infection
    - Vaccination likely modifies the probability of seeking a test given symptoms
- Known versus unknown exposure at time of test for all cases (TBD)
- Place of acquisition (NNDSS) for each case to determine whether the case has arisen from the local epidemic or was infected overseas/interstate.

Component performance indicators of the TTIQ system (Figure 21, panel 3)

- Time from when test is taken to public health notification for positive cases
- Time from public health notification to when a case is interviewed (where case interviews are occurring) by public health authorities

There are other delays in the system that matter, but routine collection of data to inform indicators may not be practical. For example, while recording the times from case interview to notification of their contacts (where notification is occurring through public health authorities) would be valuable, it is our understanding that data for cases and their contacts are not easily integrated into existing surveillance reporting systems.

Overall assessment and benchmarking

The TTIQ system performance indicator (the "TTIQ effect") provides an estimate of the percentage reduction in TP due to TTIQ, which can be compared to that estimated for 'optimal' and 'partial' TTIQ periods as per the National Plan Modelling August 2021. Furthermore, when considered in the epidemiological context (alongside other epidemic monitoring components), an assessment of whether this effect is sufficient to achieve a target level of control can be made. Critical thresholds for the TTIQ effect should depend on the status of the epidemic as measured by other indicators (*e.g.*, TP and the effective reproduction number).

Without advance knowledge of the precise combination of TTIQ strategies in place, it is not possible to determine thresholds for component indicators that will flag a substantial reduction in the TTIQ effect. Since TTIQ strategies may be adapted to scale TTIQ system capacity to caseloads, direct monitoring of the TTIQ effect will be a more reliable indicator of whole-of-system performance. In the situation where TTIQ is not performing at a required level, changes in the component performance indicators provide insight into areas of possible lowered system performance, thereby supporting system adjustment.

Appendix

Transmission potential modelling framework with delays

Overview

Our previous estimates of the impact of TTIQ on transmission potential (TP), used in Phase 1 of the National Plan Modelling, were calculated from the observed distribution of times from infection to isolation for all cases (Figure 2). Shorter times from infection to isolation mean that more opportunity for transmission is averted and transmission is reduced (Figure 3).

This piece of work uses a recursive simulation model to link different TTIQ strategies to probability distributions of times from infection to isolation, enabling us to compute the TP reduction expected under proposed TTIQ strategies. This model accounts for two modes to detect each case: active detection by downstream contact tracing from the case's infector, and passive detection by the case developing symptoms and seeking a test (Supplementary Figure 1).

For each detection mode, there is a probability that the case is missed. There is therefore a fraction of cases that will be missed altogether, spending their full duration of infectiousness in the community. Where a case would have been detected and isolated by both active and passive detection modes, the case is isolated via whichever leads to earliest detection. Note that retrospective detection of cases via upstream contact tracing is not explicitly considered in this model.

We translate these distributions and parameter assumptions into a distribution of times from infection to isolation via a numerical simulation. For each simulated case, there is a probability that they would be detected by each mode, and if detected, the time from infection to isolation is sampled at random from a probability distribution of times to isolation.

For passive detection, the overall probability of detection depends on the probability that an infected person will develop symptoms, and the probability that a symptomatic person will seek a test. The distribution of times to detection if passively detected is represented by a lognormal distribution with median of 5 days and 50% density interval 3.6-7 days (parameters log(5) and 0.5).

For active detection, the distribution of times to isolation is given by: the time from infection of the source case to infection of the contact (generation interval); the time from infection to isolation for the source case; and a random sample from the distribution of times from isolation of the source

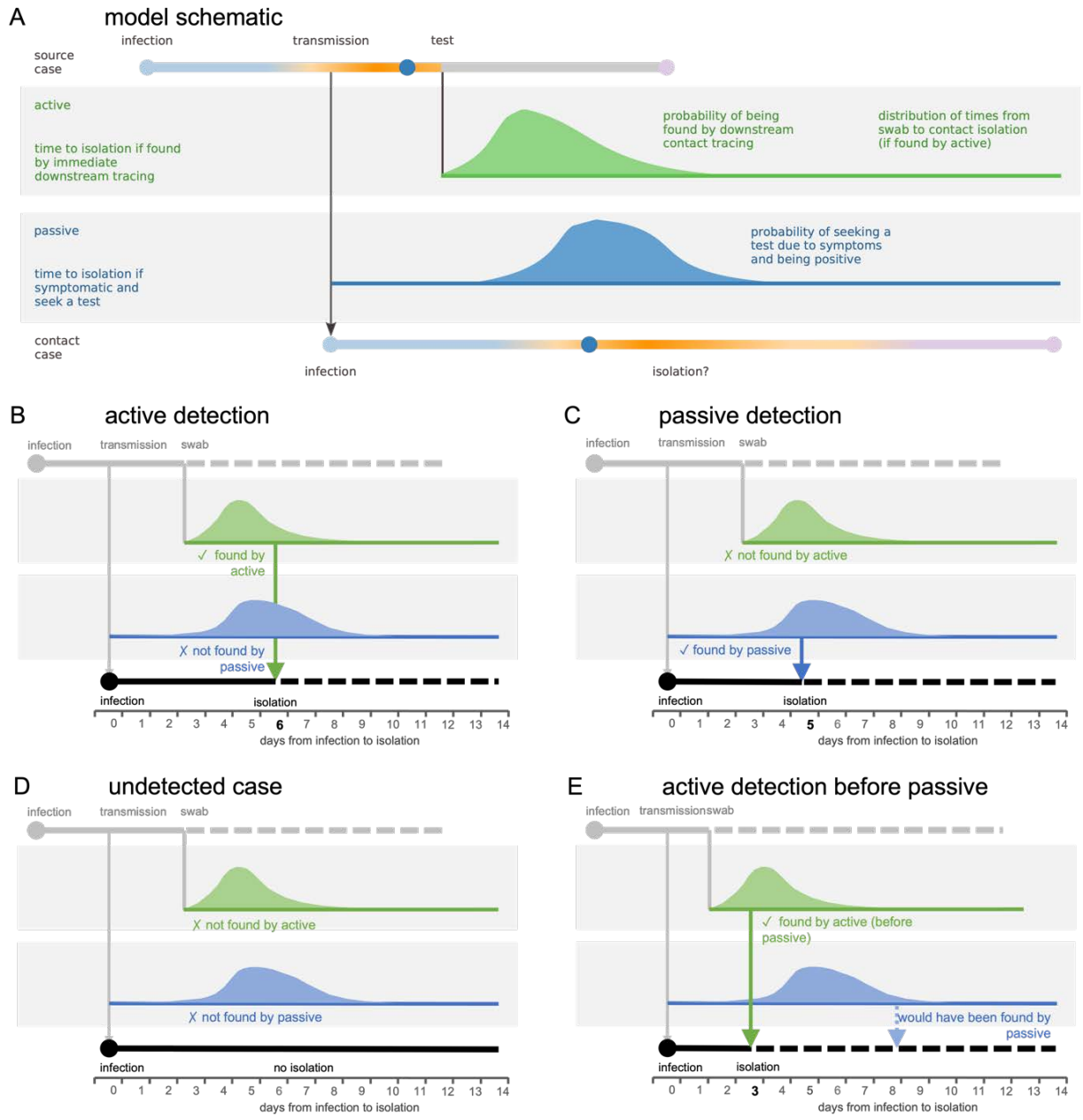
case to isolation of the infected contact (the contact tracing delay). The latter of these is comprised of several different component distributions, including the time from swab to case notification, the time from case notification to case interview, and the times from interview to contact notification and contact swab.

The times from infection to isolation (and therefore the time to onward transmission, which must be before isolation) of the source case also depend on the contact tracing delays and probabilities of detection. We jointly sample the source case times to isolation and times to onward transmission by simulating long chains of transmission via a recursive sampling algorithm (a Gibbs sampler) whereby each infected contact becomes the source for the next infected contact. This yields a distribution of times from infection to isolation for cases from which the reduction in transmission potential can be calculated. The calculation of transmission potential incorporates an assumption about the 'leakiness' of isolation, with a default assumption that when cases are instructed to isolate, they are then completely unable to infect others.

Within this modelling framework, we can investigate the likely impacts of various proposed TTIQ strategies by tweaking parameters and distributions to represent the implementation of those strategies. For example:

- case-initiated contact tracing can be represented by shortening the times from source notification to contact notification;
- prioritisation algorithms can be represented by modifying times from source notification to interview; and
- differences in prioritisation (and therefore contact tracing delays) of vaccinated cases can be modulated by adjusting the lower contribution to transmission of vaccinated infected cases.

Supplementary Figure 1: Illustration of model of active (ascertainment through contact tracing) and passive (symptomatic case finding) detection of each case. **A**) calculation of detection probabilities and times to detection by both modes. **B-E**) Examples of four possible outcomes for a single simulation of detection or non-detection via the two modes. For a given scenario of TTIQ strategies, we generate multiple simulations (each of which may be detected by either process or neither) to build a distribution of times from infection to isolation, from which the reduction in transmission potential can be calculated.



Modelling case-initiated contact tracing

We model scenarios of TTIQ with and without case-initiated contact tracing by modifying the overall contact tracing delay: the distribution of times from source case isolation to infected contact isolation.

We model the contact tracing delay as the sum of three other types of delay (Figure 15): the time from swab to notification, the time from notification to the infected contact being identified (via interview or by the case), and the aggregate of all 'other' delays. These other delays might include the time from source case isolation to swab, and the time from source case interview to the infected contact being instructed to isolate.

For scenarios without case-initiated contact tracing, we estimate the distributions of times from swab to case notification and from case notification to case interview from NSW data. We use a modelled distribution for 'other' delays with mean and variance of one day since we are not able to estimate these directly from the data.

For scenarios with case-initiated contact tracing, we use the same delays for times from swab to notification of the source case, and the 'other' delays, but we modify the times from source case notification to interview so that some fraction of these delays (those infected contacts that are identified by cases) are always set to zero, and the remainder are sampled from the estimated distribution of times (some of which are also zero). This reflects an assumption that instructions to notify contacts are sent to cases immediately, and that the case is immediately able to identify close contacts. It is assumed that the time taken from this point to instruct contacts to isolate is the same as for manual contact tracing, and this is included in the same distribution of 'other' delays.

Note also that this model assumes that in the absence of a formal case-initiated contact tracing policy, infected contacts do not isolate until after being identified by a case interview. However, cases may inform household members and other close contacts of their positive result, and these contacts may choose to self-isolate. The predominance of such self-directed isolation behaviour will likely be difficult to estimate from data, since recorded dates of isolation could represent the date when contacts are instructed to isolate, the date when they began isolating, or the last day in the community, but not all three dates.

TECHNICAL APPENDIX

A.1. Model Details

A.1.1 Response policies

Response policies consist of vaccination, contact tracing, lockdown, and quarantine strategies.

Vaccination

The effect of Pfizer and AstraZeneca vaccines are included in the model. Vaccinated agents aged 60+ are assumed to be vaccinated with AstraZeneca. All other agents are assumed to be vaccinated with Pfizer. Vaccines are assumed to decrease both susceptibility to infection and the probability of developing symptoms, given infection. These reductions depend on vaccine type, the number of doses received, the time since vaccination, and the assumed model of vaccine protection (detailed below).

Vaccine protection. In the main report, we consider a leaky model of vaccine protection. In the leaky model, the effect of vaccination is determined for each contact – there is a per-exposure probability (which depends on age, and the time since receiving their last dose) that vaccinated individuals in the model are protected against infection or the development of symptoms.

Agents reach the maximum dose/vaccine-type vaccine efficacy 3 weeks after their first dose, and 2 weeks after their second dose

In the supplementary results (Section A.3), we also consider an all-or-nothing model of vaccine protection. In this model, the effect of vaccination is to fully protect a proportion (which depends on vaccine efficacy after receiving i doses, $V_{E,i}$) of vaccinated individuals from ever becoming infected, while the complementary proportion will receive zero protection against infection or the development of symptoms. In this model, once an individual receives their first dose of a vaccine, there is a probability, p_1, each timestep for the following *k* timesteps (which equates to three weeks) that they will develop full protection, where,

$$p_1 = 1 - \left(1 - V_{E,1}\right)^{\frac{1}{k}}.$$

This probability per time step p_1 equates to having a probability $V_{E,1}$ that a vaccinated agent will end up being fully protected 3 weeks after receiving their first dose. For individuals who did not develop full protection after their first dose, there is a probability, p_2, each timestep following their second dose (up to *l* timesteps, which equates to two weeks) that they will develop full protection, where,

$$p_2 = 1 - \left(1 - \hat{V}_{E,2}\right)^{\frac{1}{l}} \text{ and } \hat{V}_{E,2} = \left(\frac{V_{E,2} - V_{E,1}}{1 - V_{E,1}}\right).$$

This probability per time step p_2 equates to having a probability $V_{E,2}$ that a vaccinated agent will end up being fully protected 2 weeks after receiving their second dose.

Test, Trace, Isolate, Quarantine (TTIQ) and lockdown.

TTIQ. When an infected individual becomes symptomatic, they get tested without delay and self-isolate. It is assumed that all symptomatic individuals will get tested. There is a delay, d_1, in receiving their test result. If they receive a negative test result, they leave self-isolation. If they receive a positive test result, contact tracing is implemented, and after a delay d_2, they enter isolation for 10 days (Figure A1a).

Contacts of a case are quarantined (there is a delay d_3 between case identification and contacts entering quarantine). Quarantined contacts are immediately tested. There is a delay, d_1, in receiving their test result. If they receive a positive test result, contact tracing is implemented, and after a delay d_2, they enter isolation for 10 days. Quarantined contacts with a negative test result remain in quarantine for 14 days. A clearance test is conducted on day 12 of quarantine. If they have a negative test result, they leave quarantine on day 14. Otherwise, contact tracing is implemented, and after a delay d_2, they enter isolation for 10 days (Figure A1b).

Cases in isolation undergo a clearance test on day 8 of isolation. If they have a negative test result, they leave isolation on day 10, otherwise their isolation period restarts (Figure A1a).

Lockdown. Upon identification of the first case in the community, the whole community goes into lockdown (there is a delay d_4 between case identification and lockdown implementation). Upon entering lockdown, all community members get tested. There is a delay d_5 until the test results are available. Individuals that test positive enter isolation for 10 days and contact tracing is initiated. Individuals that test negative remain in quarantine for 14 days. A clearance test is conducted on day 12. If they have a negative test result, they leave lockdown on day 14. Otherwise, contact tracing is implemented, and after a delay d_2, they enter isolation for 10 days. (Figure A1c).

Contact tracing. When a case is identified, contact tracing is initiated. Contacts are defined to be all household and community contacts of the case, from either

- 3 days before symptom onset (for symptomatic cases)
- 3 days before the day of testing (for asymptomatic cases)

to the day they entered isolation. Contact tracing is assumed to be 100% effective (all contacts are found). Effectiveness can be reduced if it is considered more feasible.

Effect of TTIQ and Lockdown on contact rates and between-household mobility. Self-isolation, isolation, quarantine and lockdown impact the rate at which agents make household and community contacts in the model, and the rate at which they move between households in the community. In the results presented in the main report, all scenarios assume the following effects of TTIQ and lockdown:

Policy	Self-isolated or isolated. Relative reduction in rate of:			Quarantined. Relative reduction in rate of:			Individuals in lockdown. Relative reduction in rate of:		
	Household contact	Comm. contact	Household mobility	Household contact	Comm. contact	Household mobility	Household contact	Comm. contact	Household mobility
CTP1	1	1	1	1	1	1	0	0.9	1
CTP2	1	1	1	0	1	1	0	0.9	1

Delays. In the results presented in the main report, all scenarios have the following delays:

Delay	Values
From onset of symptoms to receiving results of test for a case (includes delay to receiving test, and receiving result) (d_1)	1 day
From time of case identification to case isolation (d_2)	1 day
From time of case identification to quarantining contacts of a case (d_3)	1 day
From time of quarantining contacts to receiving results of test for contact (d_1)	1 day
From time of case identification to enacting lockdown (d_4)	1 day
From time of enacting lockdown to receiving results of testing whole community (includes delay to receiving test, and receiving result) (d_5)	2 days

A.1.2. Severity data for General and Aboriginal and Torres Strait Islander populations, and comparison to general population severity shifted downwards 10 years and 20 years, for ages 20+

Table A1. Percentage of cases hospitalised only (not ICU, not died) in the General and Aboriginal and Torres Strait Islander populations, and comparison to percentages in the general population that have been shifted downwards 10 years and 20 years for ages 20+. General Population data: COVID-19 cases by age group and severity, selected jurisdictions, 1 January 2021 – 12 September 2021. Aboriginal and Torres Strait Islander Population data: as of the 13th of September 2021

Age group	Percentage hospitalised only (total cases)		10-year age shift (for 20+)	20-year age shift (for 20+)
	General Population	Aboriginal and Torres Strait Islander		
0-11	5 (8302)	3 (475)	5	5
12-17	5 (4685)	7 (255)	5	5
18-29	10 (13086)	12 (502)	14	17
30-39	14 (9051)	17 (298)	17	20
40–49	17 (6349)	20 (250)	20	25
50–59	20 (4639)	18 (130)	25	36
60–69	25 (2530)	39 (54)	36	47
70–79	36 (1123)	40 (10)	47	48
80–89	47 (553)	67 (3)	48	48
90+	48 (146)	100 (1)	48	48

Table A2. Percentage of cases ICU only (not hospital only, not died) in the General and Aboriginal and Torres Strait Islander populations, and comparison to percentages in the general population that have been shifted downwards 10 years and 20 years for ages 20+. General Population data: COVID-19 cases by age group and severity, selected jurisdictions, 1 January 2021 – 12 September 2021. Aboriginal and Torres Strait Islander Population data: as of the 13th of September 2021

Age group	Percentage ICU only (total cases)		10-year age shift (for 20+)	20-year age shift (for 20+)
	General Population	Aboriginal and Torres Strait Islander		
0-11	<1 (8302)	<1 (475)	<1	<1
12-17	<1 (4685)	1 (255)	<1	<1
18-29	1 (13086)	<1 (502)	1	3
30-39	1 (9051)	<1 (298)	3	5
40–49	3 (6349)	2 (250)	5	7
50–59	5 (4639)	5 (130)	7	7
60–69	7 (2530)	9 (54)	7	7*
70–79	7 (1123)	40 (10)	7*	7*
80–89	4 (553)	<1 (3)	7*	7*
90+	0 (146)	<1 (1)	7*	7*

** At any point where the probability of a severe outcome decreased by age in the general population, the severity in the Aboriginal and Torres Strait Islander population was assumed to remain constant.*

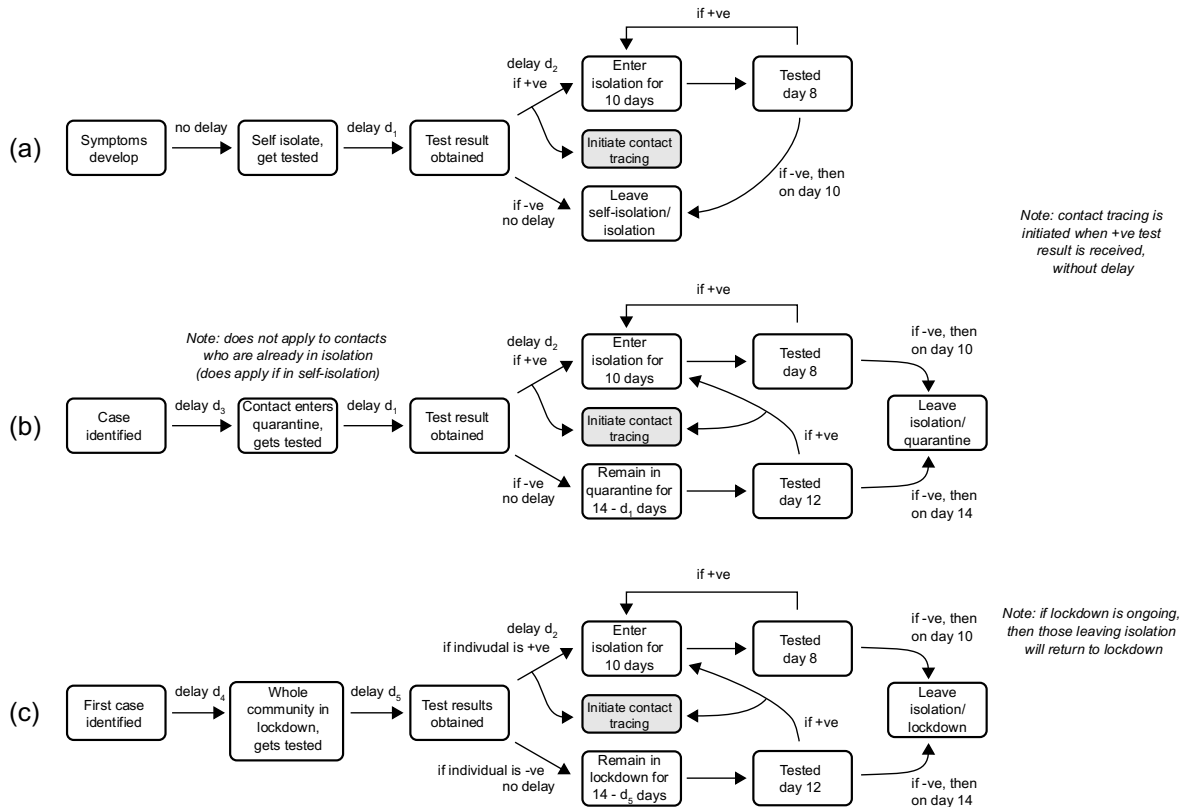


Figure A1. Schematic diagram of the isolation, contact tracing and quarantine in the disease model: (a) represents scenario where case is identified after symptoms develop, (b) represents scenario where case is identified by contact tracing, (c) represents lockdown scenario.

A.2. Model calibration

We applied The Bayesian Optimization for Likelihood-Free Inference (BOLFI) framework to calibrate the model. We used the set of summary statistics (that describe key epidemiological quantities for which we have some prior knowledge) shown in Table A6. These include the basic reproduction number, the mean generation interval, the secondary household attack rate, and the probability of the time of symptom onset being n days earlier than the time of first transmission (TOST), where n is set as -5, -1, 0, 1, 5.

The set of free parameters (estimated by the calibration process) include:

- The mean and standard deviation of the natural logarithm of the distribution describing the duration of symptomatic infection (assuming a lognormal distribution),
- the base probability of transmission per contact,

The values of these free parameters that we use in the model are those which were found to minimise the discrepancy between the model generated summary statistics, and the values of the observed summary statistics. The values of these free parameters that are used in the model are shown in Table A4.

Table A3: Parameters describing community and dwelling characteristics ('AG'= Age Group)

<table>
<tr><th>Parameter/s</th><th>Value</th><th>Source</th></tr>
<tr><td>Community and household size</td><td>
<table>
<tr><th>Scenarios</th><th>Pre-emptive</th><th colspan="3">Reactive</th></tr>
<tr><td>Community size</td><td>1000</td><td>220</td><td>580</td><td>1018</td></tr>
<tr><td>Number of houses</td><td>130</td><td>36</td><td>121</td><td>291</td></tr>
<tr><td>Mean core household size</td><td>7.7</td><td>6.1</td><td>4.8</td><td>3.5</td></tr>
</table>
</td><td>Defined in consultation with the Aboriginal and Torres Strait Islander Advisory Group on COVID-19</td></tr>
<tr><td>Age distribution</td><td>Pre-emptive: Reflective of Aboriginal and Torres Strait Islander Australians in the Northern Territory, Australia.
Reactive: Defined in consultation with the Aboriginal and Torres Strait Islander Advisory Group on COVID-19</td><td>[2]</td></tr>
<tr><td>Within-community mobility</td><td>Individuals stay at main household (core) 66% of the time, second household (regular) 23% of the time, third household (on/off) 9% of the time, and spend their remaining time (i.e. 2%) at a randomly allocated household</td><td>[3,4]</td></tr>
<tr><td>Daily number of contacts with each current household member</td><td>1</td><td>Assumption</td></tr>
<tr><td>Daily number of community contacts (row is age of contact, column is age of agent)
Work package 2 estimate for remote communities (see demographic analysis)</td><td>
<table>
<tr><th>AG</th><th>0,5</th><th>5,10</th><th>10,15</th><th>15,20</th><th>20,25</th><th>25,30</th><th>30,35</th><th>35,40</th><th>40,45</th><th>45,50</th><th>50,55</th><th>55,60</th><th>60,65</th><th>65,70</th><th>70,75</th><th>75,80</th><th>80+</th></tr>
<tr><td>0,5</td><td>2.71</td><td>1.03</td><td>0.23</td><td>0.18</td><td>0.33</td><td>0.49</td><td>0.51</td><td>0.45</td><td>0.39</td><td>0.40</td><td>0.40</td><td>0.29</td><td>0.17</td><td>0.11</td><td>0.11</td><td>0.15</td><td>0.11</td></tr>
<tr><td>5,10</td><td>1.11</td><td>7.54</td><td>1.47</td><td>0.32</td><td>0.37</td><td>0.57</td><td>0.71</td><td>0.73</td><td>0.68</td><td>0.70</td><td>0.73</td><td>0.51</td><td>0.29</td><td>0.19</td><td>0.15</td><td>0.15</td><td>0.13</td></tr>
<tr><td>10,15</td><td>0.36</td><td>1.60</td><td>11.34</td><td>1.84</td><td>0.59</td><td>0.62</td><td>0.77</td><td>0.89</td><td>0.92</td><td>0.89</td><td>0.87</td><td>0.61</td><td>0.35</td><td>0.26</td><td>0.19</td><td>0.13</td><td>0.09</td></tr>
<tr><td>15,20</td><td>0.28</td><td>0.40</td><td>2.02</td><td>10.50</td><td>1.89</td><td>0.82</td><td>0.81</td><td>0.91</td><td>0.98</td><td>0.99</td><td>0.89</td><td>0.63</td><td>0.38</td><td>0.29</td><td>0.25</td><td>0.14</td><td>0.07</td></tr>
<tr><td>20,25</td><td>0.39</td><td>0.34</td><td>0.55</td><td>2.19</td><td>4.94</td><td>1.73</td><td>1.08</td><td>1.04</td><td>1.03</td><td>1.03</td><td>1.00</td><td>0.76</td><td>0.46</td><td>0.33</td><td>0.31</td><td>0.20</td><td>0.07</td></tr>
<tr><td>25,30</td><td>0.62</td><td>0.48</td><td>0.40</td><td>0.72</td><td>2.22</td><td>3.29</td><td>1.76</td><td>1.46</td><td>1.28</td><td>1.16</td><td>1.17</td><td>1.04</td><td>0.65</td><td>0.40</td><td>0.36</td><td>0.28</td><td>0.09</td></tr>
<tr><td>30,35</td><td>0.72</td><td>0.71</td><td>0.51</td><td>0.52</td><td>1.05</td><td>2.04</td><td>2.27</td><td>1.82</td><td>1.56</td><td>1.24</td><td>1.13</td><td>1.09</td><td>0.79</td><td>0.47</td><td>0.37</td><td>0.32</td><td>0.11</td></tr>
<tr><td>35,40</td><td>0.54</td><td>0.70</td><td>0.61</td><td>0.51</td><td>0.70</td><td>1.15</td><td>1.50</td><td>1.69</td><td>1.59</td><td>1.24</td><td>0.98</td><td>0.89</td><td>0.73</td><td>0.48</td><td>0.36</td><td>0.29</td><td>0.11</td></tr>
<tr><td>40,45</td><td>0.34</td><td>0.48</td><td>0.53</td><td>0.52</td><td>0.57</td><td>0.79</td><td>0.93</td><td>1.16</td><td>1.36</td><td>1.22</td><td>0.91</td><td>0.72</td><td>0.57</td><td>0.42</td><td>0.34</td><td>0.27</td><td>0.10</td></tr>
<tr><td>45,50</td><td>0.23</td><td>0.29</td><td>0.35</td><td>0.42</td><td>0.50</td><td>0.64</td><td>0.69</td><td>0.78</td><td>0.96</td><td>1.10</td><td>0.95</td><td>0.70</td><td>0.50</td><td>0.36</td><td>0.30</td><td>0.26</td><td>0.10</td></tr>
<tr><td>50,55</td><td>0.18</td><td>0.19</td><td>0.21</td><td>0.26</td><td>0.37</td><td>0.49</td><td>0.52</td><td>0.53</td><td>0.59</td><td>0.71</td><td>0.84</td><td>0.71</td><td>0.49</td><td>0.32</td><td>0.26</td><td>0.24</td><td>0.11</td></tr>
<tr><td>55,60</td><td>0.15</td><td>0.13</td><td>0.11</td><td>0.12</td><td>0.20</td><td>0.31</td><td>0.35</td><td>0.35</td><td>0.33</td><td>0.36</td><td>0.47</td><td>0.57</td><td>0.46</td><td>0.31</td><td>0.22</td><td>0.19</td><td>0.10</td></tr>
<tr><td>60,65</td><td>0.10</td><td>0.08</td><td>0.06</td><td>0.05</td><td>0.08</td><td>0.14</td><td>0.19</td><td>0.20</td><td>0.17</td><td>0.16</td><td>0.19</td><td>0.28</td><td>0.33</td><td>0.28</td><td>0.19</td><td>0.13</td><td>0.07</td></tr>
<tr><td>65,70</td><td>0.05</td><td>0.04</td><td>0.03</td><td>0.03</td><td>0.03</td><td>0.05</td><td>0.07</td><td>0.10</td><td>0.09</td><td>0.07</td><td>0.07</td><td>0.10</td><td>0.15</td><td>0.19</td><td>0.17</td><td>0.10</td><td>0.04</td></tr>
<tr><td>70,75</td><td>0.02</td><td>0.02</td><td>0.02</td><td>0.01</td><td>0.02</td><td>0.02</td><td>0.03</td><td>0.04</td><td>0.05</td><td>0.04</td><td>0.04</td><td>0.04</td><td>0.06</td><td>0.08</td><td>0.12</td><td>0.09</td><td>0.03</td></tr>
<tr><td>75,80</td><td>0.01</td><td>0.01</td><td>0.01</td><td>0.01</td><td>0.01</td><td>0.01</td><td>0.01</td><td>0.02</td><td>0.02</td><td>0.03</td><td>0.02</td><td>0.02</td><td>0.02</td><td>0.03</td><td>0.05</td><td>0.07</td><td>0.03</td></tr>
<tr><td>80+</td><td>0.00</td><td>0.00</td><td>0.00</td><td>0.01</td><td>0.01</td><td>0.01</td><td>0.01</td><td>0.01</td><td>0.02</td><td>0.02</td><td>0.02</td><td>0.02</td><td>0.02</td><td>0.02</td><td>0.03</td><td>0.04</td><td>0.04</td></tr>
</table>
</td><td></td></tr>
</table>

Table A4: Parameters related to transmission and progression of infection ('AG' = Age Group).

Parameter	Distribution / value	Source
Basic reproduction number	10.7, 5	Highest value: Work package 2 estimate for remote communities (see demographic analysis)
Latent period	Lognormal(mu=1.3, sigma=0.4) which results in mean 4 days, Pr(Latent period < x) = 0.95 of 7.23 days	[6]
Incubation period	Lognormal(mu=1.51, sigma=0.46) which results in mean 5 days, Pr(Incubation period < x) = 0.95 of 9.6 days	[7]
Duration of symptomatic infection	Lognormal(mu=1.40, sigma=0.10) which results in mean 4 days, Pr(Symptomatic period < x) = 0.95 of 4.7 days	Calibrated
Probability of developing symptoms	**AG** **[0,10)** **[10,20)** **[20,30)** **[30,40)** **[40,50)** **[50,60)** **[60,70)** **70+** 0.29 0.21 0.27 0.33 0.4 0.49 0.63 0.69	[8]
Relative susceptibility in community	**AG** **[0,5)** **[5,10)** **[10,15)** **[15,20)** **[20,25)** **[25,30)** **[30,35)** **[35,40)** 0.081 0.098 0.115 0.139 0.197 0.232 0.245 0.243 **AG** **[40,45)** **[45,50)** **[50,55)** **[55,60)** **[60,65)** **[65,70)** **[70,75)** **[75,80)** 0.234 0.230 0.234 0.241 0.248 0.243 0.223 0.210 **AG** **80+** 0.205	Work package 2 estimate for remote communities (see demographic analysis)
Relative susceptibility in household	**AG** **[0,5)** **[5,10)** **[10,15)** **[15,20)** **[20,25)** **[25,30)** **[30,35)** **[35,40)** 0.216 0.264 0.313 0.385 0.571 0.704 0.755 0.747 **AG** **[40,45)** **[45,50)** **[50,55)** **[55,60)** **[60,65)** **[65,70)** **[70,75)** **[75,80)** 0.711 0.696 0.708 0.737 0.770 0.746 0.668 0.620 **AG** **80+** 0.602	Work package 2 estimate for remote communities (see demographic analysis)

Table A5: Parameters related to vaccination and testing

Parameter	Distribution	Source
Test sensitivity	Bernoulli with Pr(positive at t |C, T_{Inc}) = 0, t <= - T_{Inc} , [1 + exp(-(1.5 + 2.2 s))]^(-1) , - T_{Inc} <= t <= - C , [1 + exp(-(1.5 - 0.22 s))]^(-1) , t > - C where s = t + C, C ~ Uniform[0, min(T_{Inc} , 3.5)].	[9]
Vaccine efficacies	Vaccine and dose | Reduction in susceptibility | Reduction in symptomatic infection | Reduction in onwards transmission AstraZeneca Dose 1 | 0.18 | 0.33 | 0.02 AstraZeneca Dose 2 | 0.6 | 0.61 | 0.36 Pfizer BNT Dose 1 | 0.3 | 0.33 | 0.13 Pfizer BNT Dose 2 | 0.79 | 0.83 | 0.65	Doherty modelling consortium, based on literature and expert consultation

Table A6: Summary statistics used in the model calibration process

Summary statistic	Observed values	Source
Basic reproduction number	10.7, 5	Highest value: Work package 2 estimate for remote communities (see demographic analysis)
Generation Interval	4.65 days	Consistent with transmission potential calculation by the Doherty modelling consortium
Secondary household attack rate	0.311	[9]
Pr(TOST < -5)	0.034	[10]
Pr(TOST < -1)	0.325	[10]
Pr(TOST < 0)	0.515	[10]
Pr(TOST < 1)	0.7	[10]
Pr(TOST < 5)	0.9682	[10]

A.3. Supplementary results

A.3.1. Pre-emptive vaccination, leaky vs all-or-nothing vaccine protection

Given uncertainty in the mechanism of vaccine protection, we consider the sensitivity of our transmission model when assuming leaky vaccine protection, versus all-or-nothing vaccine protection. In the scenarios presented in Figure A.2, there is a slight difference in the size of the outbreak peak, with slightly higher peaks observed in the leaky scenarios (top row), compared to the corresponding all-or-nothing scenarios (bottom row). There is little difference in the timing of outbreak peaks and duration of outbreaks.

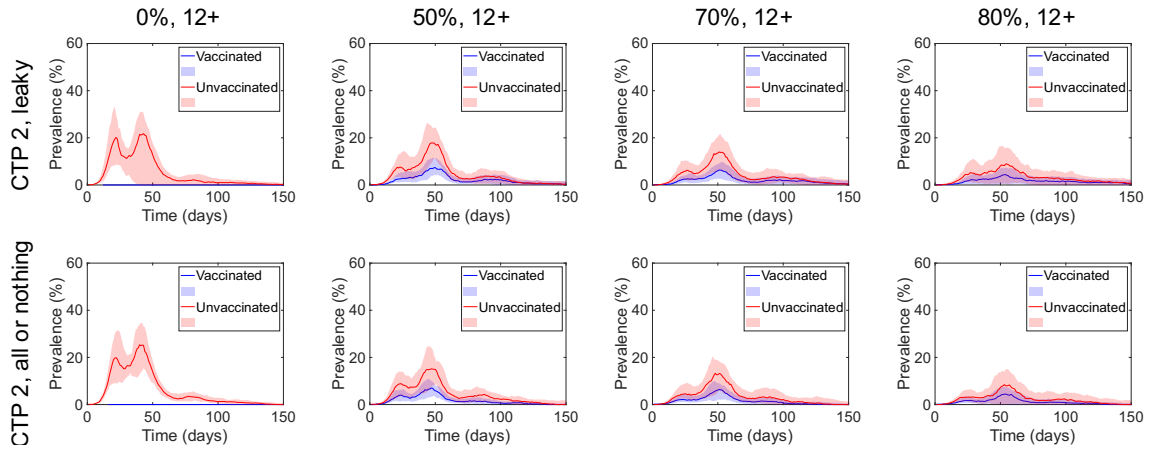


Figure A.2. ***Leaky vs all-or-nothing vaccine protection.*** *Prevalence of infection within the vaccinated (blue) and non-vaccinated (red) subpopulations over time (top row) for response policy CTP2, R0=10.7, leaky vaccine protection; (bottom row) for response policy CTP2, R0=10.7, all-or-nothing vaccine protection, and for each achieved uniform vaccination coverage level (column 1: 0%; column 2: 50%, 12+; column 3: 70%, 12+; column 4: 80%, 12+). Results presented assume 90% level of compliance with lockdown. Solid lines: median prevalence, shaded regions: interquartile range of 100 simulations.*

A.3.2. Pre-emptive vaccination, R_0 = 5 scenarios

Given uncertainty in the basic reproduction number, R_0, for remote communities, the Aboriginal and Torres Strait Islander Advisory Group on COVID-19 was interested in understanding the likely impact of contain and trace response policies with pre-emptive vaccination under the assumption of R_0=5. In the results presented in Figures A3 and Tables A7-A9, it is clear that with a lower starting transmission potential of R_0=5, the impact of contain and trace response policies and pre-emptive vaccination on reducing outbreak size and clinical burdens is far greater, compared to the R_0=10.7 scenarios.

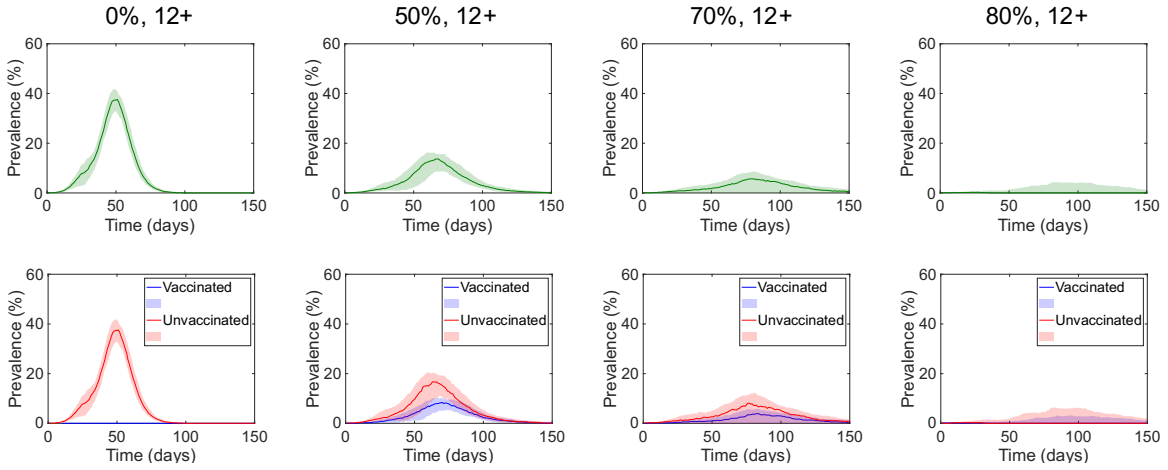


Figure A3. ***Basic reproduction number, R_0=5****. Prevalence of infection (top row) in the whole community; (bottom row) within the vaccinated (blue) and non-vaccinated (red) subpopulations, over time. Here, we assume response policy CTP1 for each achieved uniform vaccination coverage level (column 1: 0%; column 2: 50%, 12+; column 3: 70%, 12+; column 4: 80%, 12+;). Results presented assume 90% level of compliance with lockdown. Solid lines: median prevalence, shaded regions: interquartile range of 100 simulation.*

*Table A7. **Basic reproduction number, R_0=5**. Total cumulative infections for a community of 1000 people, stratified by age and vaccination status. Median and interquartile range of cumulative infections are shown by age group and vaccination status. Results presented assume 90% level of compliance with lockdown and response policy **CTP1**.*

Achieved vaccination coverage scenario	Vaccination status of infected	Age groups				
		<12	12-<15	15-<40	40-<60	60+
No coverage (12+, 0%)	Vaccinated	0 (0, 0)	0 (0, 0)	0 (0, 0)	0 (0, 0)	0 (0, 0)
	Not vaccinated	171 (162, 178)	48 (44, 52)	414 (403, 423)	212 (203, 221)	64 (58, 68)
Uniform coverage 1 (12+, 50%)	Vaccinated	0 (0, 0)	3 (2, 5)	80 (72, 86)	43 (38, 49)	18 (14, 21)
	Not vaccinated	126 (113, 136)	27 (22, 31)	178 (167, 187)	93 (88, 100)	29 (24, 32)
Uniform coverage 2 (12+, 70%)	Vaccinated	0 (0, 0)	3 (0, 5)	82 (1, 94)	48 (0, 56)	18 (0, 22)
	Not vaccinated	97 (1, 114)	17 (0, 22)	94 (1, 100)	49 (0, 56)	14 (0, 17)
Uniform coverage 3 (12+, 80%)	Vaccinated	0 (0, 0)	0 (0, 4)	2 (0, 91)	1 (0, 48)	1 (0, 21)
	Not vaccinated	1 (0, 102)	1 (0, 16)	2 (0, 61)	1 (0, 33)	1 (0, 10)

*Table A8. **Basic reproduction number, R_0=5**. Average cumulative number of symptomatic infections, ward admissions and ICU admissions over the course of outbreaks for achieved two dose vaccine threshold targets of 50, 70, and 80% for ages 12+. Results presented assume 90% level of compliance with lockdown, response policy CTP1, and using the 10-year age shift in severity estimates.*

Average cumulative number	Achieved vaccination coverage scenario		
	50%, 12+	70%, 12+	80%, 12+
Symptomatic infections	138	68	34
Ward admissions	30	14	6
ICU admissions	12	6	2

*Table A9. Average cumulative number of symptomatic infections, ward admissions and ICU admissions over the course of outbreaks for achieved two dose vaccine threshold targets of 50, 70, and 80% for ages 12+, stratified by age and vaccination status. Results presented assume 90% level of compliance with lockdown, response policy CTP1, a basic reproduction number **R_0 = 5**, and using the 10-year age shift in severity estimates.*

Average cumulative number	Achieved coverage scenario	<15 yrs		15-39 yrs		40-59 yrs		60+ yrs	
		Vacc'd	Unvac	Vacc'd	Unvac	Vacc'd	Unvac	Vacc'd	Unvac
Symptomatic infections	50%	0	37	3	43	3	34	3	14
	70%	0	22	3	18	2	15	2	6
	80%	0	13	2	8	1	6	2	3
Ward admissions	50%	0	1	0	3	0	11	2	13
	70%	0	0	0	1	0	5	2	6
	80%	0	0	0	1	0	2	1	2
ICU admissions	50%	0	0	0	1	0	5	1	6
	70%	0	0	0	0	0	2	1	3
	80%	0	0	0	0	0	1	0	1

A.3.3. Pre-emptive vaccination, 20-year age shift in severity

Given uncertainty in the severity of disease in Australian Aboriginal and Torres Strait Islander people, relative to the general population (due to limited data), we also calculated clinical burden in pre-emptive vaccination response scenarios assuming a 20-year age shift in severity estimates (clinical burdens shown in the main report assume a 10-year age shift in severity relative to the general population, which is consistent with the limited data we have to date). In all scenarios, the clinical burden (excluding symptomatic infections) is increased compared to the 10-year age shift scenarios.

*Table A10. **20-year age shift in severity and $R_0 = 10.7$**. Average cumulative number of symptomatic infections, ward admissions and ICU admissions over the course of outbreaks for achieved two dose vaccine threshold targets of 50, 70, and 80% for ages 12+. Results presented assume 90% level of compliance with lockdown, response policy CTP1, a basic reproduction number $R_0 = 10.7$, and using the 20-year age shift in severity estimates.*

Average cumulative number	Achieved vaccination coverage scenario		
	50%, 12+	70%, 12+	80%, 12+
Symptomatic infections	203	147	112
Ward admissions	66	41	29
ICU admissions	27	17	11

*Table A11. **20-year age shift in severity and $R_0 = 5$.** Average cumulative number of symptomatic infections, ward admissions and ICU admissions over the course of outbreaks for achieved two dose vaccine threshold targets of 50, 70, and 80% for ages 12+. Results presented assume 90% level of compliance with lockdown, response policy CTP1, a basic reproduction number **$R_0 = 5$**, and using the **20 year age shift** in severity estimates.*

Average cumulative number	Achieved vaccination coverage scenario		
	50%, 12+	70%, 12+	80%, 12+
Symptomatic infections	138	68	34
Ward admissions	47	22	10
ICU admissions	20	9	4

A.3.4. Reactive vaccination, $R_0 = 5$ scenarios

Given uncertainty in the basic reproduction number, R_0, for remote communities, the Aboriginal and Torres Strait Islander Advisory Group on COVID-19 was interested in understanding the likely impact of contain and trace response policies with reactive vaccination under the assumption of $R_0=5$. In the results presented in Figure A4-A6 and Tables A12-A17, it is clear that with a lower starting transmission potential of $R_0=5$, the impact of contain and trace response policies and reactive vaccination on reducing outbreak size and clinical burdens is far greater, compared to the $R_0=10.7$ scenarios.

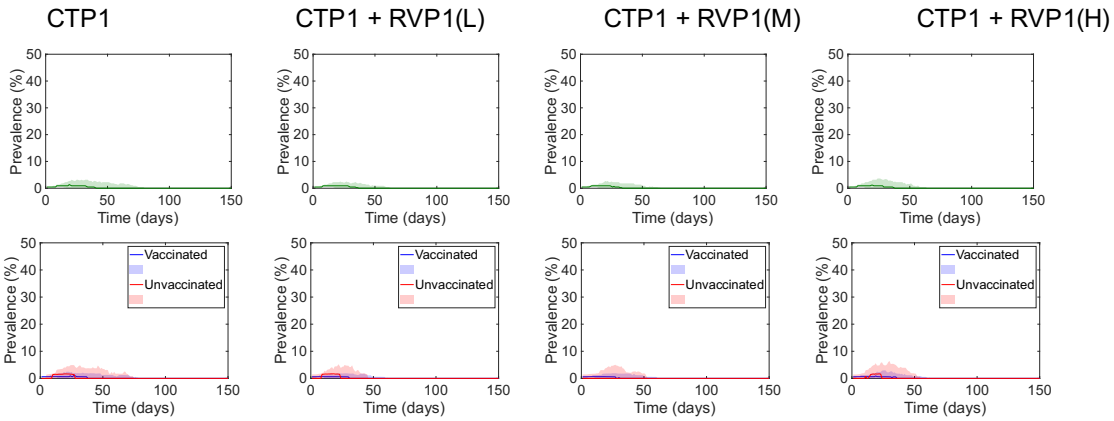


Figure A4. ***Basic reproduction number, R_0=5, exemplar community 1*** *(N = 220, high coverage). Prevalence of infection (top row) in the whole community; (bottom row) within the vaccinated (blue) and non-vaccinated (red) subpopulations, over time, for response policies (column 1) CTP1; (column 2) CTP1+RVP1 (low rate); (column 3) CTP1+RVP1 (medium rate); (column 4) CTP1+RVP1 (high rate). Results presented assume 90% level of compliance with lockdown. Solid lines: median prevalence, shaded regions: interquartile range of 100 simulations.*

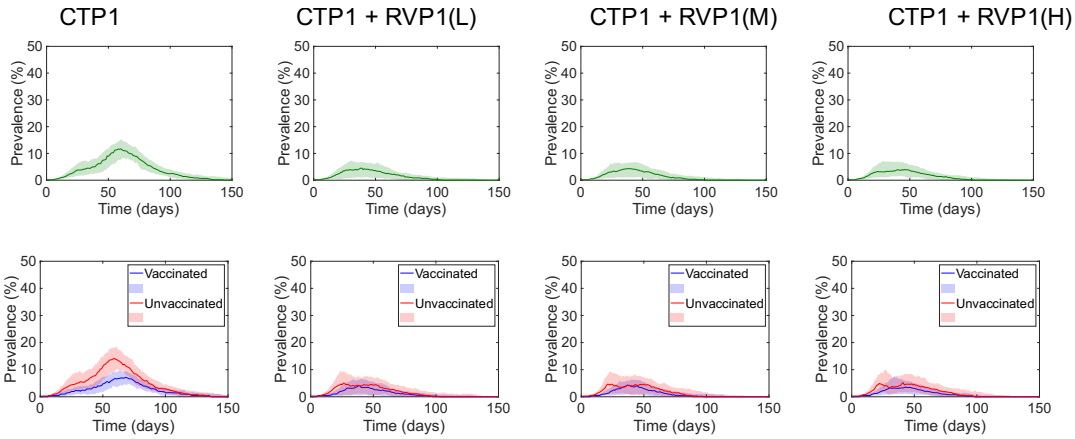


Figure A5. ***Basic reproduction number, R_0=5, exemplar community 2*** *(N = 580, medium coverage). Prevalence of infection (top row) in the whole community; (bottom row) within the vaccinated (blue) and non-vaccinated (red) subpopulations, over time, for response policies (column 1) CTP1; (column 2) CTP1+RVP1 (low rate); (column 3) CTP1+RVP1 (medium rate); (column 4) CTP1+RVP1 (high rate). Results presented assume 90% level of compliance with lockdown. Solid lines: median prevalence, shaded regions: interquartile range of 100 simulations.*

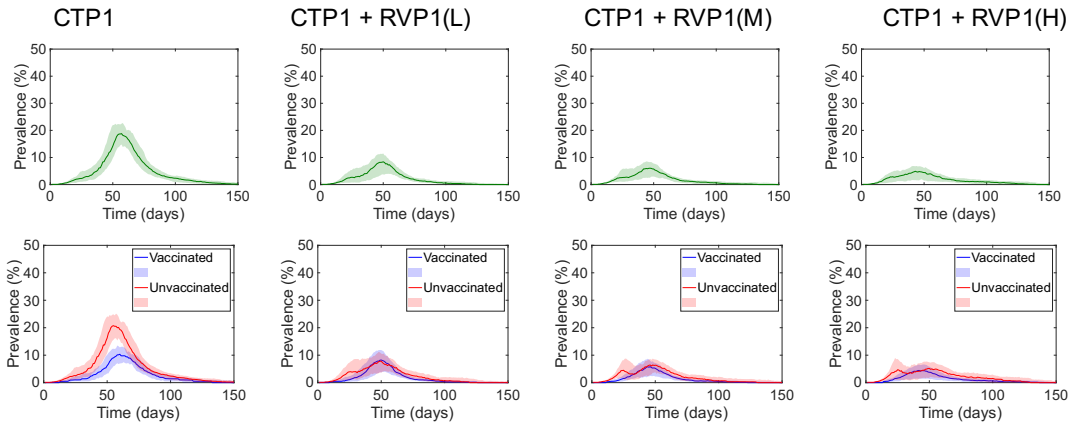


Figure A6. ***Basic reproduction number, R_0=5, exemplar community 3*** *(N = 1018, low coverage). Prevalence of infection (top row) in the whole community; (bottom row) within the vaccinated (blue) and non-vaccinated (red) subpopulations, over time, for response policies (column 1) CTP1; (column 2) CTP1+RVP1 (low rate); (column 3) CTP1+RVP1 (medium rate);. (column 4) CTP1+RVP1 (high rate). Results presented assume 90% level of compliance with lockdown. Solid lines: median prevalence, shaded regions: interquartile range of 100 simulations*

Table A12. ***Basic reproduction number, R_0=5, exemplar community 1*** *(N = 220, high coverage). Total cumulative infections stratified by age and vaccination status. Median and interquartile range of cumulative infections are shown by age group, vaccination status. Results presented assume 90% level of compliance with lockdown, response policy* ***CTP1*** *and reactive vaccination policy* ***RVP1*** *for low, medium and high vaccination rates, R_0=5.*

Reactive vaccination rate	Vaccination status of infected	Age groups				
		<12	12-<15	15-<40	40-<60	60+
0	Vaccinated	0 (0, 0)	0 (0, 0)	3 (0, 10)	1 (0, 6)	1 (0, 4)
	Not vaccinated	2 (0, 9)	0 (0, 2)	1 (0, 4)	0 (0, 1)	0 (0, 0)
Low (30/day)	Vaccinated	0 (0, 0)	0 (0, 0)	3 (0, 8)	1 (0, 4)	1 (0, 2)
	Not vaccinated	1 (0, 5)	0 (0, 1)	0 (0, 2)	0 (0, 0)	0 (0, 0)
Medium (60/day)	Vaccinated	0 (0, 0)	0 (0, 1)	2 (0, 8)	1 (0, 4)	0 (0, 2)
	Not vaccinated	1 (0, 5)	0 (0, 1)	0 (0, 1)	0 (0, 0)	0 (0, 0)
High (100/day)	Vaccinated	0 (0, 0)	0 (0, 1)	2 (0, 8)	1 (0, 4)	1 (0, 2)
	Not vaccinated	2 (0, 7)	0 (0, 2)	0 (0, 2)	0 (0, 0)	0 (0, 0)

Table A13. ***Basic reproduction number, R_0=5, exemplar community 2*** *(580 people, medium coverage). Total cumulative infections stratified by age and vaccination status. Median and interquartile range of cumulative infections are shown by age group, vaccination status. Results presented assume 90% level of compliance with lockdown, response policy* ***CTP1*** *and reactive vaccination policy* ***RVP1*** *for low, medium and high vaccination rates, R_0=5.*

Reactive vaccination rate	Vaccination status of infected	Age groups				
		<12	12-<15	15-<40	40-<60	60+
0	Vaccinated	0 (0, 0)	1 (0, 1)	34 (28, 39)	25 (21, 30)	15 (12, 18)
	Not vaccinated	77 (67, 85)	19 (15, 22)	121 (112, 126)	36 (33, 40)	10 (8, 12)
Low (30/day)	Vaccinated	0 (0, 0)	2 (0, 4)	43 (15, 54)	21 (7, 28)	9 (3, 13)
	Not vaccinated	40 (11, 54)	5 (2, 10)	13 (5, 23)	3 (1, 6)	1 (0, 2)
Medium (60/day)	Vaccinated	0 (0, 0)	2 (1, 3)	39 (20, 56)	19 (10, 26)	9 (3, 12)
	Not vaccinated	39 (17, 54)	6 (2, 8)	12 (6, 24)	4 (1, 7)	1 (0, 1)
High (100/day)	Vaccinated	0 (0, 0)	2 (0, 5)	42 (22, 56)	19 (7, 26)	9 (4, 13)
	Not vaccinated	40 (16, 54)	5 (2, 7)	12 (6, 23)	3 (2, 6)	1 (0, 2)

Table A14. ***Basic reproduction number, R_0=5, exemplar community 3*** *(1018 people, low coverage). Total cumulative infections stratified by age and vaccination status. Median and interquartile range of cumulative infections are shown by age group, vaccination status. Results presented assume 90% level of compliance with lockdown, response policy* ***CTP1*** *and reactive vaccination policy* ***RVP1*** *for low, medium and high vaccination rates, R_0=5.*

Reactive vaccination rate	Vaccination status of infected	Age groups				
		<12	12-<15	15-<40	40-<60	60+
0	Vaccinated	0 (0, 0)	1 (0, 1)	18 (14, 20)	35 (31, 40)	29 (25, 33)
	Not vaccinated	129 (116, 142)	40 (32, 45)	314 (302, 327)	101 (92, 107)	29 (25, 32)
Low (30/day)	Vaccinated	0 (0, 0)	8 (3, 10)	98 (70, 115)	44 (33, 53)	23 (15, 30)
	Not vaccinated	77 (61, 87)	13 (10, 17)	38 (22, 62)	9 (5, 16)	2 (1, 3)
Medium (60/day)	Vaccinated	0 (0, 0)	6 (3, 8)	79 (63, 97)	37 (24, 46)	18 (12, 26)
	Not vaccinated	66 (51, 88)	12 (7, 14)	28 (17, 48)	8 (4, 12)	1 (0, 3)
High (100/day)	Vaccinated	0 (0, 0)	5 (3, 7)	77 (49, 98)	37 (23, 48)	18 (11, 23)
	Not vaccinated	69 (46, 87)	11 (6, 15)	31 (14, 42)	9 (3, 12)	1 (0, 3)

Table A15. ***Basic reproduction number, R_0=5, exemplar community 1*** *(N = 220, high coverage). Average cumulative number of symptomatic infections, ward admissions and ICU admissions over the course of outbreaks with CTP1 and RVP1 (medium rate) switched on or off. Results presented assume 90% level of compliance with lockdown, R0=5, a 2-day delay to initiation of the reactive vaccination program and using the 10-year age shift in severity estimates.*

Average cumulative number	CTP1	CTP1 and RVP1(M)
Symptomatic infections	4	2
Ward admissions	1	0
ICU admissions	0	0

Table A16. ***Basic reproduction number, R_0=5, exemplar community 2*** *(580 people, medium coverage). Average cumulative number of symptomatic infections, ward admissions and ICU admissions over the course of outbreaks with CTP1 and RVP1 (medium rate) switched on or off. Results presented assume 90% level of compliance with lockdown, R0=5, a 2-day delay to initiation of the reactive vaccination program and using the 10-year age shift in severity estimates.*

Average cumulative number	CTP1	CTP1 and RVP1(M)
Symptomatic infections	79	28
Ward admissions	15	4
ICU admissions	6	2

Table A17. ***Basic reproduction number, R_0=5, exemplar community 3*** *(1018 people, low coverage). Average cumulative number of symptomatic infections, ward admissions and ICU admissions over the course of outbreaks with CTP1 and RVP1 (medium rate) switched on or off. Results presented assume 90% level of compliance with lockdown, R0=5, a 2-day delay to initiation of the reactive vaccination program and using the 10-year age shift in severity estimates.*

Average cumulative number	CTP1	CTP1 and RVP1(M)
Symptomatic infections	178	53
Ward admissions	36	8
ICU admissions	14	3

A.3.5. Reactive vaccination, 20-year age shift in severity

Given uncertainty in the severity of disease in Australian Aboriginal and Torres Strait Islander people, relative to the general population (due to limited data), we also calculated clinical burden in reactive vaccination response scenarios assuming a 20-year age shift in severity estimates (clinical burdens shown in the main report assume a 10-year age shift in severity relative to the general population, which is consistent with the limited data we have to date). In all scenarios, the clinical burden (excluding symptomatic infections) is increased compared to the 10-year age shift scenarios.

*Table A18. **20-year age shift in severity and R_0 = 10.7, exemplar community 3** (1018 people, low coverage). Average cumulative number of symptomatic infections, ward admissions and ICU admissions over the course of outbreaks with CTP1 and RVP1 (medium rate) switched on or off. Results presented assume 90% level of compliance with lockdown, R0=10.7, a 2-day delay to initiation of the reactive vaccination program and using the 10-year age shift in severity estimates.*

Average cumulative number	CTP1	CTP1 and RVP1(M)
Symptomatic infections	248	166
Ward admissions	74	40
ICU admissions	31	15

*Table A19. **20-year age shift in severity and R_0=5, exemplar community 3** (1018 people, low coverage). Average cumulative number of symptomatic infections, ward admissions and ICU admissions over the course of outbreaks with CTP1 and RVP1 (medium rate) switched on or off. Results presented assume 90% level of compliance with lockdown, R0=5, a 2-day delay to initiation of the reactive vaccination program and using the 10-year age shift in severity estimates.*

Average cumulative number	CTP1	CTP1 and RVP1(M)
Symptomatic infections	178	53
Ward admissions	55	12
ICU admissions	23	5

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Draft Report 2: Work Package 2, First Nations, Remote Communities
with 1 attachment (Technical Appendix)

Summary

This report uses an agent based infectious disease model to consider protective factors that can reduce the risk of COVID-19 outbreaks in remote Aboriginal and Torres Strait Islander (hereafter respectfully referred to as Indigenous) communities and defines the most effective response strategies in the event of incursions, including reactive immunisation. The model captures key features of age structure, household composition and social connections in remote Indigenous communities of different sizes.

Key questions

What coverage targets are appropriate for populations at higher risk of transmission and disease impacts? What is the role of reactive vaccination in response should outbreaks occur in such localised groups and settings in the context of suboptimal coverage? What additional public health response measures will be most useful to regain control of transmission should outbreaks occur?

Key findings

- High levels of pre-emptive vaccine coverage can substantially reduce COVID-19 transmission and health impacts in remote Indigenous communities.
- Of the strategies recommended in the current remote outbreak response guidelines, a policy that assumes relocation of contacts of cases to a hospital or safe location outside the community for the duration of quarantine is associated with improved outbreak control and lower disease burden.
- Reactive vaccination is a useful adjunct to community engaged and led outbreak response, and can reduce health impacts, particularly in larger communities with low initial vaccine coverage.
- Providing access to effective treatments will further promote health outcomes, particularly where clinical access is limited.

Background

Aboriginal and Torres Strait Islander Australians living in remote communities are anticipated to experience higher than average transmission rates of COVID-19 because of a younger population demographic and household sizes three times larger than the national average. Vaccination of the population aged 12 years and above has less effect on transmission when children under 12 make up a larger proportion of the total population and live in larger households. Both factors increase their contribution to transmission despite lower susceptibility and infectiousness than adults.

Our remote communities model reports outbreak trajectories following silent introduction of infection in the context of different levels of prior vaccine coverage and given different response measures including reactive vaccination. Modelled infections are translated into anticipated clinical outcomes using the clinical pathways model employed in our earlier phase work, with updated assumptions.

Given the high prevalence of underlying health risk determinants in remote Indigenous communities the likelihood of severe health outcomes by age commences from the age of 20 years and in each cohort thereafter maps to the non-Indigenous population 10 years older. This starting assumption has been approved by the Aboriginal and Torres Strait Islander Advisory group and benchmarked as reasonable against available data from NSW which demonstrates a higher prevalence of severe outcomes for Indigenous Australians.

Model

We consider the outcome of silent introduction of an infection into exemplar communities of different sizes, and with different achieved vaccine coverages, under

- **Contain and trace strategies with pre-emptive vaccination** (Table 1), with achieved two dose vaccine threshold targets of 50, 70, and 80% for ages 12 +
- **Contain and trace strategies with reactive vaccination for ages 12+** (Table 2) in three exemplar under vaccinated communities, given differential one and two dose vaccine effectiveness and time to completion of the vaccine course, and under different age-dependent achieved vaccine coverages (Table 3).

The transmission model (Figure 1) assumes transmission and vaccine effectiveness parameters for the Delta variant, consistent with those employed for other projects in this phase of work to support the National Plan. Community contact rates have been estimated for remote communities based on available Australian data.

Clinical outcomes were estimated using the clinical pathways model described in Ref. [2] assuming severity and vaccine efficacy parameters for the Delta variant, inclusion of age-stratified length of stay in clinical states and adapted to estimates of severity for Aboriginal and Torres Strait Islander Australians (Figure 2, section A.1.2, Technical Appendix). Specifically,

- the probabilities associated with severe outcomes are assumed to be the same for people <20 years old compared to the general Australian population.
- for people over 20 years old, the **probabilities associated with severe outcomes are shifted by 10 years** (and 20 years for the sensitivity analysis described in Technical appendix, sections A.3.3 and A.3.5)**;** for example, a symptomatic 30-year-old First Nations Australian will be hospitalised at the rate of a symptomatic 40-year-old person from the general population.
- At any point where the probability of a severe outcome decreased by age in the general population, the severity was assumed to remain constant (the biggest difference here is the probability of being admitted to ICU given hospitalisation in the oldest age groups).
- The age-stratified length of stay in clinical states are also assumed to shift by 10 years (or 20 years for the sensitivity analysis) to coincide with the changes in severe outcomes.
- For the reactive vaccination scenarios, we assume that there is a 14-day delay in gaining protection against severe outcomes after the first dose of a vaccine, and a 5-day delay in gaining the additional protection against severe outcomes from a second vaccine dose.

The clinical pathways model takes inputs of daily symptomatic individuals, stratified by age and vaccination status, from the transmission model, and translates these into a time course of clinical outcomes. There is a delay between the onset of symptoms and presentation to health services. Upon arrival to health services individuals are either admitted to ward immediately, admitted to ICU immediately, or if health services are at capacity, individuals are not admitted and may re-present the next day. We assume that only symptomatic cases requiring hospitalisation present to health services. Individuals who are initially admitted to the ward may have a subsequent ICU stay and vice versa.

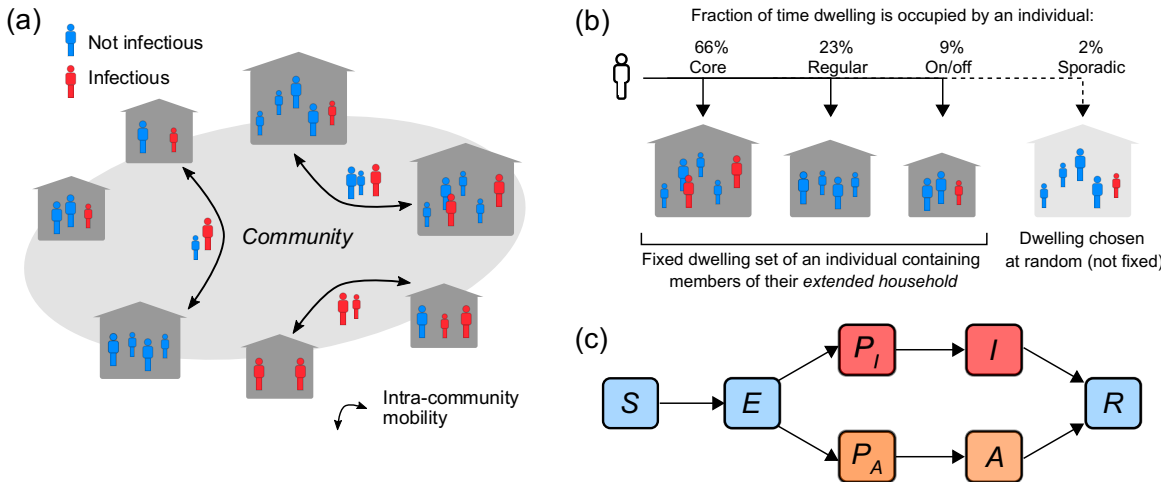

Figure 1. Schematic diagram of the model of (a) community structure, (b) intra-community mobility and (c) disease progression between the Susceptible (S), Exposed (E), Pre-symptomatic Infectious (PI), Symptomatic Infectious (I), Asymptomatic Infectious compartment 2 (A), and Recovered (R) states.

The transmission model is an individual-based model (adapted from Ref. [1], to COVID-19) that explicitly represents each individual in a remote community (Figure 1a), and the impact of pre-emptive vaccination and various public health response strategies on an outbreak. It follows a susceptible, exposed, pre-symptomatic infectious, symptomatic infectious, asymptomatic infectious, recovered paradigm (Figure 1c).

Individuals are assumed to have close family connections across a total of three dwellings in the community, between which their time is distributed as follows: main dwelling (core) 66% of the time, second dwelling (regular) 23% of the time, and third dwelling (on/off) 9% of the time. Their remaining time (i.e., 2%) is spent at a dwelling randomly allocated at the start of each day (Figure 1b). Individuals with the same home dwelling location on a given day are grouped into current households, which we refer to as an individual's *current household*. Individuals who are associated with a dwelling as either a core, regular, or on/off residence are grouped into *extended households*.

Contacts between individuals (that are necessary for transmission of infection) are explicitly modelled and can occur between current household members (household contacts) and among individuals who are not in the same current household (community contacts). Infection is assumed to generally transmit more easily between household contacts (the relative risk of transmission between household contacts compared to community contacts is generally assumed to be greater than one).

An individual's probability of developing symptoms once infected is assumed to depend on age and on vaccination status (number of doses received and vaccine type). The probability of transmission given contact with an infected individual is assumed to depend on the age and vaccination status of both the infector and infectee. Vaccine-induced protection is assumed to reduce infection rates and the chance of developing symptoms on a per-exposure basis and reduce the infectiousness of breakthrough infections. Asymptomatic infections are assumed to be 50% as infectious as symptomatic infections (with the same age and vaccination status). Further details of the model are provided in the Technical Appendix.

Table 1. COVID-19 "Contain and Test" outbreak response policies. Further details of the contain and test policies are provided in the Technical Appendix (section A.1).

Contain and Test policy	Lockdown	Case management	Contact management
CTP 1: contain and test with relocation of cases, home quarantine of contacts	Once return of first positive test, restrict all movement in and out of community for 14 days, and confine all community members to their main house and yard. Multiple rounds of testing whole community while in lockdown (on entry, and on day 12).	Re-locate to hospital or safe location (100% effective isolation) for 10 days. Clearance test on day 8.	Quarantine in main household for 14 days (contact between household members still possible). Test on entry. Clearance test on day 12.
CTP 2: contain and test with relocation of cases and contacts	As above	As above	Re-locate to hospital or safe location (100% effective quarantine) for 14 days. Test on entry. Clearance test on day 12.

Table 2. COVID-19 "Reactive Vaccination" outbreak response policy RVP1. Ages <60 are assumed to be vaccinated with Pfizer, ages 60+ with AstraZeneca. The policy is enacted with either contain and trace policy CTP1 or CTP2. Delays considered: 2, 4 days. Vaccine hesitant = 6.87% of unvaccinated population (NT data). Rate of surge vaccination based on NT estimates with lower bound estimated to be achievable with 3 door-to-door vaccinating teams (team consists of 2 vaccinators and 1 administration/liaison officer), and upper bound estimated to be achievable with 9 vaccinating teams.

Initiation of RVP1 program and scheduling of second dose

Initiated after the first case is identified, and after a delay. Only susceptible individuals are vaccinated. Older individuals are vaccinated first. Second dose scheduled for:
Pfizer: 3 weeks; AstraZeneca: 4 weeks, after first dose.

Daily rate of first dose surge vaccination

Exemplar community (Population size)		1 (220)	2 (580)	3 (1018)
Number to vaccinate	Dose 1	14	220	405
	Dose 2	42	74	204
Vaccination rate (doses per day)	L	30	40	30
	M	60	100	75
	H	100	150	120

Non-surge vaccinations

At the start of the simulation:
- all individuals with one dose are assumed to be scheduled for a second dose and so are assigned a date when they will receive second dose during simulation (time to vaccination assumed to be uniform distribution with bounds, Pfizer: 0-3 weeks, AstraZeneca: 0-4 weeks).
- all individuals who are double dosed are assumed to have reached full vaccine efficacy

Table 3. Starting vaccination coverage in exemplar communities considered in the "Reactive Vaccination" outbreak response scenarios. Characteristics of exemplar communities (size, initial vaccination coverage in age groups) were determined in consultation with the Aboriginal and Torres Strait Islander Advisory Group in COVID-19. Ages <60 are assumed to vaccinated with Pfizer, ages 60+ with AstraZeneca.

Exemplar (Population size)	Initial vaccination coverage, Dose 1					Initial vaccination coverage, Dose 2				
	12-15	16-39	40-59	60-79	80+	12-15	16-39	40-59	60-79	80+
1. (220)	35.3%	30.3%	12.2%	15%	N/A	41.2%	60.7%	83.7%	85.0%	N/A
2. (580)	20.0%	19.2%	14.7%	12.8%	0%	7.5%	21.4%	47.7%	59.0%	100%
3. (1018)	5.5%	9.3%	6.0%	3.8%	0%	5.3%	6.5%	37.7%	57.8%	0%

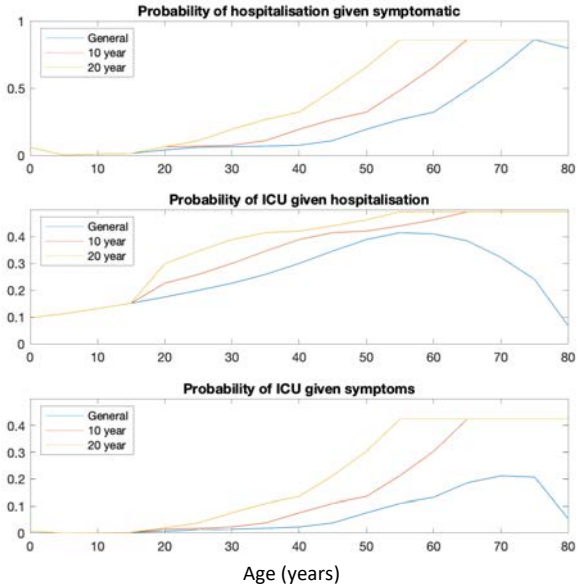

Figure 2. Estimates of severity used in the clinical pathways model for Aboriginal and Torres Strait Islander Australians (10-year age shift in severity, red; 20-year age shift in severity, yellow), compared to whole population estimates (blue). At any point where the probability of a severe outcome decreased by age in the general population, the severity was assumed to remain constant.

In all response scenarios considered, lockdown is assumed to last for 14 days only – it is not reinstated over the remainder of the outbreak, even when escalation of cases occurs so represents a 'worst case' response. Compliance with lockdown is assumed to be 90%. In all results presented in the main report, we assume a starting transmission potential of R_0=10.7 and a downward 10-year age shift in severity relative to the general population, which is consistent with available observations. Given uncertainty in R_0 for remote communities and severity of disease in Australian Aboriginal and Torres Strait Islander people relative to the general population, under the advice of the Aboriginal and Torres Strait Islander Advisory Group on COVID-19, in the Technical Appendix (section A.3) we also considered response scenarios under the assumption of R_0=5 and/or with a downward 20-year age shift in severity relative to the general population.

Results

Pre-emptive vaccination.

Figures 3 and 4 report the prevalence of all infections (with or without symptoms) over time following introduction of infection in a community of size 1000, and Table 4 reports the corresponding cumulative number of infections broken down by age and vaccination status. These results allow comparison of outbreak dynamics under Contain and Test response policies CTP1 and CTP2, and with 0% vaccine coverage and achieved two dose vaccine threshold targets of 50, 70, and 80% for ages 12+. Tables 5 to7 report corresponding clinical burdens. Note that values in these clinical burden tables are central estimates arising from approximately 100 simulations.

These results show that there is sensitivity to both the choice of contain and test response policy, and the level of achieved two-dose vaccine coverage. Higher vaccine coverage levels lead to smaller outbreaks. Contain and Test policy CTP2, where it is assumed that contacts of cases are re-located to hospital or a safe location, outperforms Contain and Test policy CTP1 for all coverage scenarios considered, where it is assumed that contacts of cases

quarantine in main home (outbreak size, size of peak and clinical burden are all smaller in comparison). This is also true in scenarios with a lower starting transmission potential of R_0=5, or when we assume a downward 20-year age shift in severity relative to the general population (Technical Appendix, sections A.3.2-A.3.3). These results provide quantitative support for implementing CTP2 where possible, and for additional wrap around support in contexts where it is only possible to implement CTP1.

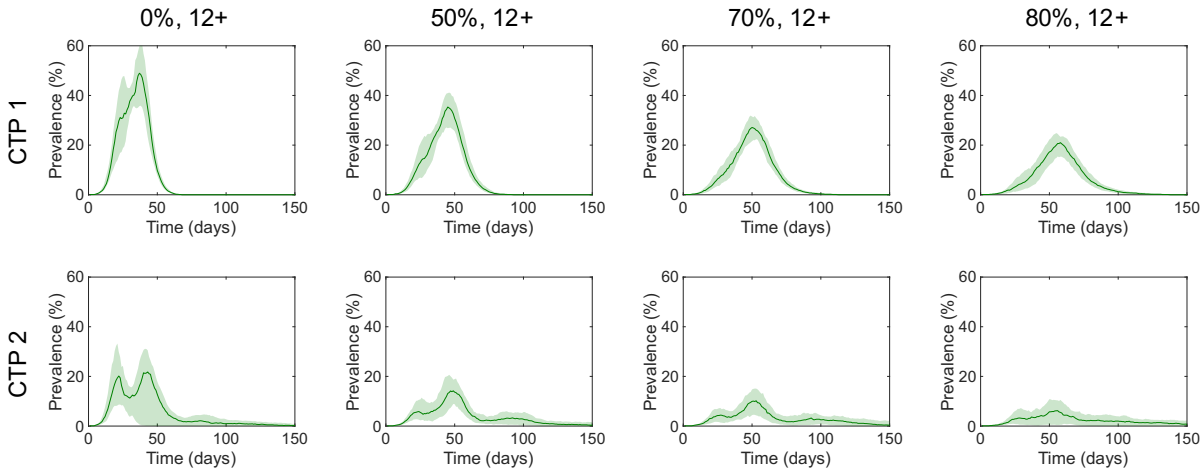

Figure 3. Prevalence of infection in whole community over time, for each response policy (top row: CTP 1; bottom row: CTP 2), and for each achieved uniform vaccination coverage level (column 1: 0%; column 2: 50%, 12+; column 3: 70%, 12+; column 4: 80%, 12+;). Solid lines: median prevalence, shaded regions: interquartile range of 100 simulations.

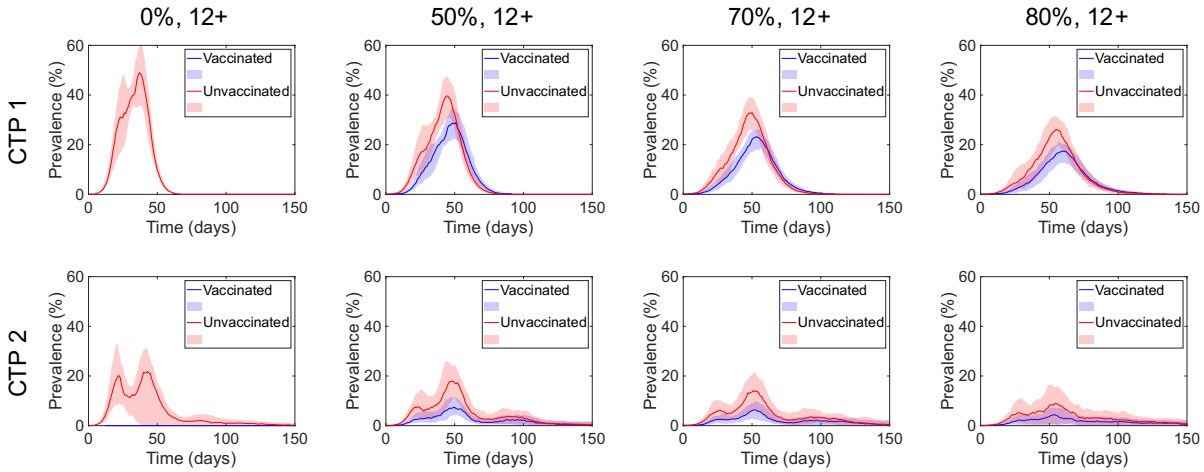

Figure 4. Prevalence of infection within the vaccinated (blue) and non-vaccinated (red) subpopulations over time, for each response policy (top row: CTP 1; bottom row: CTP 2), and for each achieved uniform vaccination coverage level (column 1: 0%; column 2: 50%, 12+; column 3: 70%, 12+; column 4: 80%, 12+;). Solid lines: median prevalence, shaded regions: interquartile range of 100 simulations.

Table 4. Total cumulative infections for a community of 1000 people, stratified by age and vaccination status. Median and interquartile range of cumulative infections are shown by age group, vaccination status, and for each response policy (CTP1, CTP2).

Achieved vaccination coverage scenario	Vaccination status of infected	Outbreak response policy									
		CTP 1					CTP 2				
		<12	12-<15	15-<40	40-<60	60+	<12	12-<15	15-<40	40-<60	60+
No coverage (12+, 0%)	Vaccinated	0 (0, 0)	0 (0, 0)	0 (0, 0)	0 (0, 0)	0 (0, 0)	0 (0, 0)	0 (0, 0)	0 (0, 0)	0 (0, 0)	0 (0, 0)
	Not vaccinated	213 (204, 221)	57 (52, 62)	432 (423, 444)	218 (208, 228)	67 (60, 71)	154 (139, 171)	47 (40, 54)	403 (385, 417)	209 (197, 220)	62 (56, 69)
Uniform coverage 1 (12+, 50%)	Vaccinated	0 (0, 0)	10 (8, 12)	159 (149, 166)	86 (81, 91)	31 (26, 34)	0 (0, 0)	5 (3, 6)	84 (67, 97)	47 (36, 53)	19 (15, 22)
	Not vaccinated	201 (190, 209)	36 (32, 41)	212 (208, 218)	110 (105, 114)	33 (30, 36)	135 (111, 151)	29 (24, 34)	191 (173, 199)	99 (88, 105)	30 (26, 33)
Uniform coverage 2 (12+, 70%)	Vaccinated	0 (0, 0)	12 (9, 14)	199 (190, 208)	109 (101, 117)	39 (34, 42)	0 (0, 0)	5 (3, 7)	111 (80, 127)	60 (48, 71)	24 (18, 30)
	Not vaccinated	184 (176, 195)	29 (25, 34)	124 (120, 128)	66 (62, 69)	20 (17, 21)	131 (104, 147)	24 (18, 28)	112 (93, 119)	59 (48, 62)	17 (14, 19)
Uniform coverage 3 (12+, 80%)	Vaccinated	0 (0, 0)	11 (7, 14)	207 (194, 220)	116 (108, 125)	41 (37, 46)	0 (0, 0)	6 (2, 8)	120 (66, 149)	68 (37, 82)	27 (11, 33)
	Not vaccinated	178 (164, 192)	25 (20, 28)	81 (77, 84)	43 (40, 45)	13 (11, 14)	132 (74, 149)	19 (11, 27)	74 (50, 79)	38 (28, 41)	12 (5, 14)

Table 5. Average cumulative number of symptomatic infections, ward admissions and ICU admissions over the course of outbreaks for achieved two dose vaccine threshold targets of 50, 70, and 80% for ages 12+. Results presented assume response policy CTP1.

Average cumulative number	Achieved vaccination coverage scenario		
	50%, 12+	70%, 12+	80%, 12+
Symptomatic infections	203	147	112
Ward admissions	43	27	19
ICU admissions	17	10	7

The breakdown of infections by severity of clinical outcome by age and vaccine status is reported for contain and trace policies CTP1 and CTP2, for achieved two dose vaccine threshold targets of 50, 70, and 80% for ages 12+ in Tables 6 and 7, respectively. They show that more severe outcomes occur more frequently in the older age groups, and in the unvaccinated subpopulation.

Table 6. CTP1. Average cumulative number of symptomatic infections, ward admissions and ICU admissions over the course of outbreaks stratified by age and vaccination status for achieved two dose vaccine threshold targets of 50, 70, and 80% for ages 12+. Results presented assume response policy CTP1.

Average cumulative number	Achieved coverage scenario	<15 yrs		15-39 yrs		40-59 yrs		60+ yrs	
		Vacc'd	Unvac	Vacc'd	Unvac	Vacc'd	Unvac	Vacc'd	Unvac
Symptomatic infections	50%	0	64	7	56	6	45	5	20
	70%	1	56	8	32	7	26	6	11
	80%	0	49	8	19	7	16	6	7
Ward admissions	50%	0	1	0	4	1	15	4	17
	70%	0	1	0	2	1	8	4	10
	80%	0	1	0	1	1	5	4	6
ICU admissions	50%	0	0	0	1	0	6	1	8
	70%	0	0	0	1	0	3	1	4
	80%	0	0	0	0	0	2	1	3

Table 7. CTP2. Average cumulative number of symptomatic infections, ward admissions and ICU admissions over the course of outbreaks stratified by age and vaccination status for achieved two dose vaccine threshold targets of 50, 70, and 80% for ages 12+. Results presented assume response policy CTP2.

Average cumulative number	Achieved coverage scenario	<15 yrs		15-39 yrs		40-59 yrs		60+ yrs	
		Vacc'd	Unvac	Vacc'd	Unvac	Vacc'd	Unvac	Vacc'd	Unvac
Symptomatic infections	50%	0	39	3	46	3	36	3	16
	70%	0	36	4	25	4	21	4	8
	80%	0	31	5	15	4	12	3	5
Ward admissions	50%	0	1	0	3	0	12	3	14
	70%	0	1	0	2	0	7	3	7
	80%	0	1	0	1	0	4	2	5
ICU admissions	50%	0	0	0	1	0	5	1	6
	70%	0	0	0	1	0	3	1	3
	80%	0	0	0	0	0	1	1	2

Reactive vaccination.

Working closely with the Aboriginal and Torres Strait Islander Advisory Group on COVID-19, we have defined case studies of communities of differing size and current vaccine coverage (Exemplar communities 1-3, Table 2) to consider how reactive vaccine strategies might be used as an adjunct to currently recommended outbreak response measures. We consider how vaccines rolled out at different rates might augment the public health response in these communities. Rates of achievable delivery are based on advice from the Northern Territory, assuming different numbers of teams deployed for implementation. High acceptance is assumed (6.57% hesitancy in the uninfected and unvaccinated).

Vaccination programs continue until all eligible (ages 12+), non-infected, and non-vaccine hesitant people are vaccinated (see Figure 5 for vaccination coverage over time).

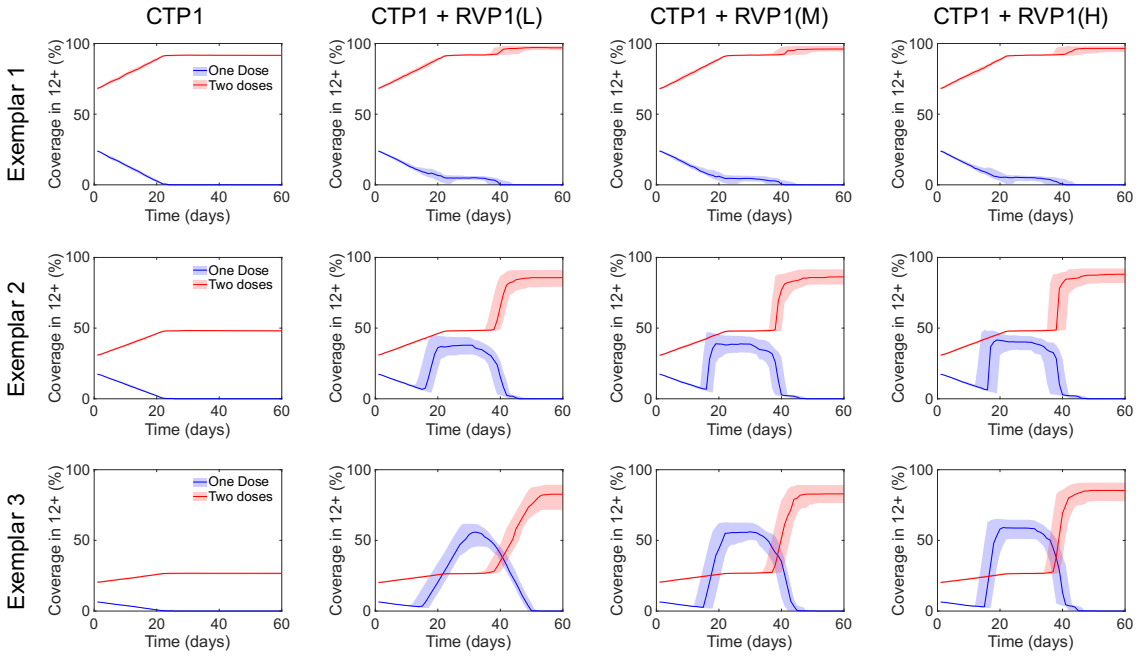

Figure 5. Vaccination coverage in 12+ over time (blue, 1 dose coverage; red, 2 dose coverage) in exemplar communities (top row: community 1, middle row: community 2, bottom row: community 3) for response policies (column 1) CTP1; (column 2) CTP1+RVP1 (low rate); (column 3) CTP1+RVP1 (medium rate); (column 4) CTP1+RVP1 (high rate). Solid lines: median prevalence, shaded regions: interquartile range of 100 simulations.

Figures 6-8 report the prevalence of all infections (with or without symptoms) over time following introduction of infection into the exemplar communities when reactive vaccination policy RVP1 is used in conjunction with Contain and Trace Policy CTP1. Tables 8-10 report the corresponding cumulative number of infections broken down by age and vaccination status. These results allow comparison of outbreak dynamics under increasing rates of reactive vaccination. Tables 11-14 report corresponding clinical burdens.

The greatest benefit of the reactive vaccination program occurs in Exemplar community 3 which has the lowest vaccine coverage before the outbreak (Figure 8). This community has just over 1,000 people and low baseline vaccine coverage. In Exemplar community 3, reactive vaccination reduces ward and ICU admissions by 47% (Table 13) because vaccine protection against severe outcomes kicks in faster than effects against any infection (5 days vs 2 weeks for second dose), even following a single dose (14 days, vs 3 weeks). In this example, lockdown measures are only maintained for 14 days, but if it were possible to extend beyond this duration to slow down spread, greater vaccine benefits might be observed. It is reassuring to note that benefits of immunization are not diminished with the slower pace of rollout in this example (Tables 14, 15), noting reasonably high baseline coverage in the 50+ years at the beginning of the outbreak.

These findings also apply to scenarios with a lower starting transmission potential of R_0=5, or when we assume a downward 20-year age shift in severity relative to the general population (Technical Appendix, sections A.3.4-A.3.5).

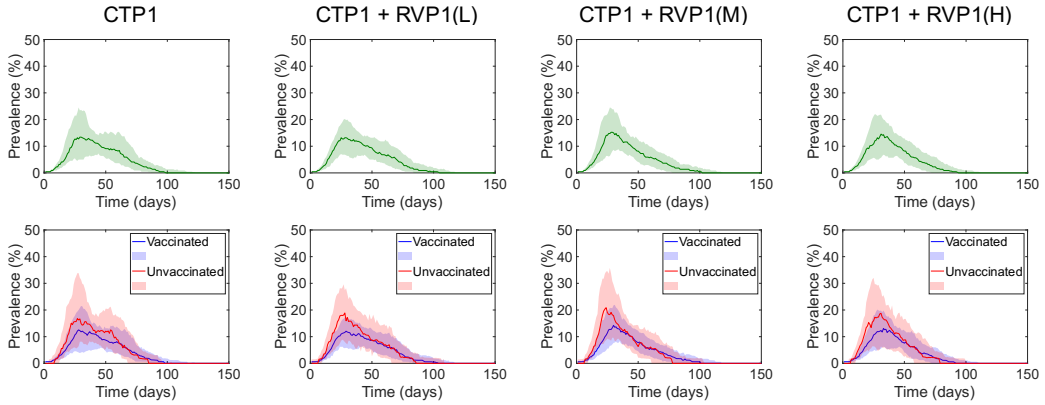

Figure 6. Prevalence of infection in Exemplar community 1 (N = 220, high coverage) within (top row) the whole population; (bottom row) the vaccinated (blue) and non-vaccinated (red) subpopulations, over time, for response policies (column 1) CTP1; (column 2) CTP1+RVP1 (low rate); (column 3) CTP1+RVP1 (medium rate); (column 4) CTP1+RVP1 (high rate). Solid lines: median prevalence, shaded regions: interquartile range of 100 simulations.

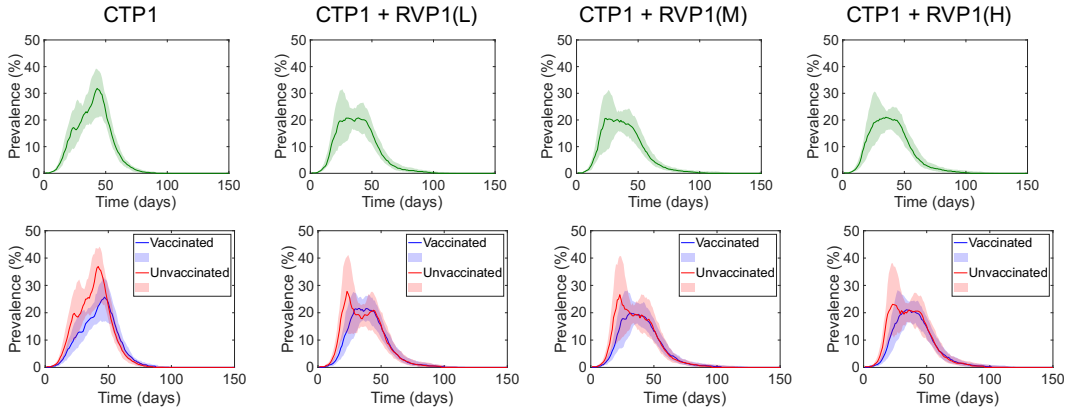

Figure 7. Prevalence of infection in Exemplar community 2 (N = 580, medium coverage) within (top row) the whole community; (bottom row) the vaccinated (blue) and non-vaccinated (red) subpopulations, for response policies (column 1) CTP1; (column 2) CTP1+RVP1 (low rate); (column 3) CTP1+RVP1 (medium rate); (column 4) CTP1+RVP1 (high rate). Solid lines: median prevalence, shaded regions: interquartile range of 100 simulations.

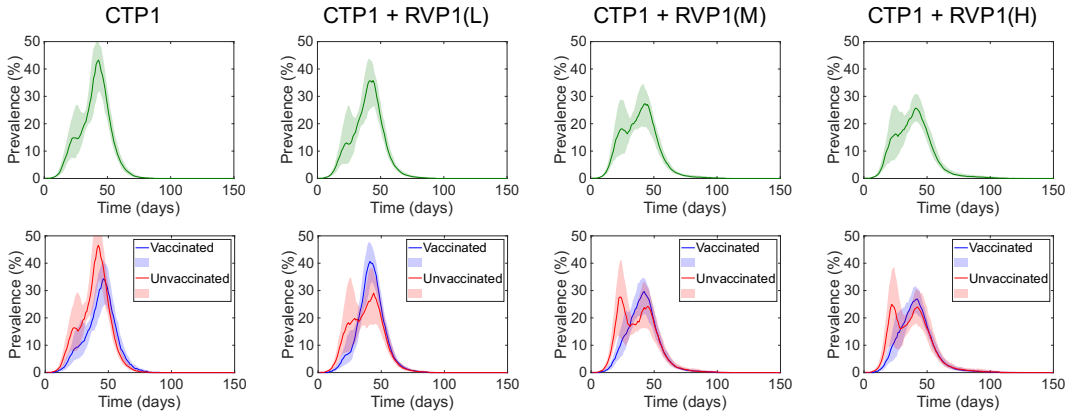

Figure 8. Prevalence of infection in Exemplar community 3 (N = 1018, low coverage) within (top row) the whole community; (bottom row) the vaccinated (blue) and non-vaccinated (red) subpopulations, for response policies (column 1) CTP1; (column 2) CTP1+RVP1 (low rate); (column 3) CTP1+RVP1 (medium rate); (column 4) CTP1+RVP1 (high rate). Solid lines: median prevalence, shaded regions: interquartile range of 100 simulations.

Table 8. Total cumulative infections for Exemplar community 1 (220 people, high vaccination coverage), stratified by age and vaccination status. Median and interquartile range of cumulative infections are shown by age group and vaccination status. Results presented assume response policy CTP1 and reactive vaccination policy RVP1 for low, medium, and high vaccination rates, and a 2-day delay to initiation of the reactive vaccination program.

Reactive vaccination rate	Vaccination status of infected	Age groups				
		<12	12-<15	15-<40	40-<60	60+
0	Vaccinated	0 (0, 0)	2 (1, 4)	48 (44, 53)	30 (25, 33)	15 (12, 18)
	Not vaccinated	31 (27, 36)	5 (4, 7)	9 (8, 10)	2 (1, 3)	0 (0, 0)
Low (30/day)	Vaccinated	0 (0, 0)	3 (2, 4)	49 (41, 56)	27 (23, 33)	15 (12, 19)
	Not vaccinated	32 (27, 36)	4 (2, 5)	3 (2, 5)	1 (0, 1)	0 (0, 0)
Medium (60/day)	Vaccinated	0 (0, 0)	3 (2, 4)	51 (44, 57)	29 (23, 33)	14 (12, 18)
	Not vaccinated	32 (26, 36)	3 (2, 5)	4 (1, 6)	1 (0, 2)	0 (0, 0)
High (100/day)	Vaccinated	0 (0, 0)	3 (1, 4)	49 (40, 56)	27 (21, 33)	15 (11, 17)
	Not vaccinated	30 (22, 34)	3 (2, 5)	3 (1, 5)	0 (0, 1)	0 (0, 0)

Table 9. Total cumulative infections for Exemplar community 2 (580 people, medium vaccination coverage), stratified by age and vaccination status. Median and interquartile range of cumulative infections are shown by age group and vaccination status. Results presented assume response policy CTP1 and reactive vaccination policy RVP1 for low, medium, and high vaccination rates, and a 2-day delay to initiation of the reactive vaccination program.

Reactive vaccination rate	Vaccination status of infected	Age groups				
		<12	12-<15	15-<40	40-<60	60+
0	Vaccinated	0 (0, 0)	3 (2, 4)	68 (63, 74)	50 (44, 54)	28 (25, 31)
	Not vaccinated	121 (113, 128)	26 (23, 29)	145 (140, 150)	43 (40, 47)	13 (12, 15)
Low (30/day)	Vaccinated	0 (0, 0)	9 (7, 11)	128 (116, 143)	68 (63, 77)	33 (29, 37)
	Not vaccinated	109 (103, 119)	15 (13, 18)	45 (23, 66)	10 (7, 18)	3 (1, 5)
Medium (60/day)	Vaccinated	0 (0, 0)	9 (7, 12)	128 (118, 143)	64 (57, 71)	31 (29, 36)
	Not vaccinated	109 (101, 116)	15 (11, 18)	38 (23, 59)	11 (6, 18)	2 (1, 4)
High (100/day)	Vaccinated	0 (0, 0)	9 (7, 12)	132 (120, 142)	66 (58, 73)	32 (28, 36)
	Not vaccinated	112 (104, 123)	14 (11, 18)	37 (22, 56)	10 (7, 18)	2 (1, 4)

Table 10. Total cumulative infections for Exemplar community 3 (1018 people, low vaccination coverage), stratified by age and vaccination status. Median and interquartile range of cumulative infections are shown by age group and vaccination status. Results presented assume response policy CTP1 and reactive vaccination policy RVP1 for low, medium, and high vaccination rates, and a 2-day delay to initiation of the reactive vaccination program.

Reactive vaccination rate	Vaccination status of infected	Age groups				
		<12	12-<15	15-<40	40-<60	60+
0	Vaccinated	0 (0, 0)	2 (1, 2)	35 (32, 38)	67 (63, 70)	48 (45, 52)
	Not vaccinated	199 (190, 209)	54 (49, 59)	368 (360, 379)	114 (109, 119)	34 (32, 38)
Low (30/day)	Vaccinated	0 (0, 0)	20 (15, 24)	256 (199, 295)	130 (117, 143)	65 (60, 72)
	Not vaccinated	187 (179, 197)	30 (25, 34)	98 (53, 164)	24 (15, 37)	4 (3, 8)
Medium (60/day)	Vaccinated	0 (0, 0)	17 (14, 21)	231 (197, 258)	119 (107, 131)	64 (59, 69)
	Not vaccinated	178 (171, 187)	28 (23, 33)	101 (59, 143)	25 (16, 37)	5 (3, 8)
High (100/day)	Vaccinated	0 (0, 0)	18 (15, 21)	224 (201, 247)	117 (109, 126)	61 (57, 68)
	Not vaccinated	178 (166, 189)	27 (22, 31)	84 (49, 126)	21 (13, 34)	5 (3, 8)

Table 11. Average cumulative number of symptomatic infections, ward admissions and ICU admissions over the course of outbreaks in Exemplar community 1 with CTP1 and RVP1 (medium rate) switched on or off. Results presented assume a 2-day delay to initiation of the reactive vaccination program.

Average cumulative number	CTP1	CTP1 and RVP1(M)
Symptomatic infections	21	20
Ward admissions	4	3
ICU admissions	1	1

Table 12. Average cumulative number of symptomatic infections, ward admissions and ICU admissions over the course of outbreaks in Exemplar community 2 with CTP1 and RVP1 (medium rate) switched on or off. Results presented assume a 2-day delay to initiation of the reactive vaccination program.

Average cumulative number	CTP1	CTP1 and RVP1(M)
Symptomatic infections	120	89
Ward admissions	21	14
ICU admissions	8	5

Table 13. Average cumulative number of symptomatic infections, ward admissions and ICU admissions over the course of outbreaks in Exemplar community 3 with CTP1 and RVP1 (medium rate) switched on or off. Results presented assume a 2-day delay to initiation of the reactive vaccination program.

Average cumulative number	CTP1	CTP1 and RVP1(M)
Symptomatic infections	248	166
Ward admissions	49	26
ICU admissions	19	10

Table 14. Varying rate of reactive vaccination in Exemplar community 3. Average cumulative number of symptomatic infections, ward admissions and ICU admissions over the course of outbreaks with CTP1 and RVP1 with a daily rate of surge vaccination of 0, 30, 75 and 150 doses. Results presented assume a 2-day delay to initiation of the reactive vaccination program.

Average cumulative number	Daily rate of vaccination			
	0	30 (L)	75 (M)	150 (H)
Symptomatic infections	248	180	166	160
Ward admissions	49	26	26	26
ICU admissions	19	9	10	9

Table 15. Increase in delay to initiation of the reactive vaccination program in Exemplar community 3. Total cumulative infections stratified by age and vaccination status. Median and interquartile range of cumulative infections are shown by age group and vaccination status. Results presented assume response policy CTP1 and reactive vaccination policy RVP1 for low, medium, and high vaccination rates, and a 4-day delay to initiation of the reactive vaccination program.

Reactive vaccination rate	Vaccination status of infected	Age groups				
		<12	12-<15	15-<40	40-<60	60+
0	Vaccinated	0 (0, 0)	2 (1, 2)	35 (32, 38)	67 (63, 70)	48 (45, 52)
	Not vaccinated	199 (190, 209)	54 (49, 59)	368 (360, 379)	114 (109, 119)	34 (32, 38)
Low (30/day)	Vaccinated	0 (0, 0)	19 (16, 22)	246 (200, 292)	131 (114, 140)	68 (61, 75)
	Not vaccinated	190 (177, 198)	32 (27, 37)	115 (75, 167)	30 (18, 41)	7 (4, 10)
Medium (60/day)	Vaccinated	0 (0, 0)	19 (14, 22)	245 (192, 278)	122 (110, 137)	66 (58, 73)
	Not vaccinated	181 (172, 196)	29 (23, 34)	100 (59, 154)	26 (14, 42)	6 (3, 11)
High (100/day)	Vaccinated	0 (0, 0)	19 (15, 23)	235 (195, 266)	120 (109, 132)	63 (57, 69)
	Not vaccinated	182 (169, 194)	28 (23, 33)	97 (61, 141)	26 (16, 39)	7 (5, 9)

The breakdown of infections by severity of clinical outcome by age and vaccine status is reported for reactive vaccination policy RVP1 in conjunction with contain and trace policies CTP1 and CTP2 in Table 16. These results provide quantitative support for implementing reactive vaccination in conjunction with CTP2 in under vaccinated communities where possible, and for additional wrap around support in contexts where it is only possible to implement reactive vaccination in conjunction with CTP1.

Table 16. Average cumulative number of symptomatic infections, ward admissions and ICU admissions stratified by age and vaccination status over the course of outbreaks in Exemplar Population 3 when reactive vaccination policy RVP1 (medium rate) and either CTP1 or CTP2 are employed. Results presented assume a 2-day delay to initiation of the reactive vaccination program.

Average cumulative number	CTP	<15 yrs		15-39 yrs		40-59 yrs		60+ yrs	
		Vacc'd	Unvac	Vacc'd	Unvac	Vacc'd	Unvac	Vacc'd	Unvac
Symptomatic infections	1	1	58	10	47	8	21	11	9
	2	1	41	7	37	4	16	7	6
Ward admissions	1	0	1	0	3	1	7	6	8
	2	0	1	0	3	0	5	4	6
ICU admissions	1	0	0	0	1	0	3	2	4
	2	0	0	0	1	0	2	1	3

We are continuing to consult with the Advisory Group to develop extended narrative case studies of combined vaccine and other public health measures that may be feasible and implementable in remote settings with different starting vaccination coverage by age to maximise outbreak response impacts. Given the high prevalence of underlying health risk determinants in such settings, our projections of severe clinical outcomes remain uncertain and require ongoing review. Consideration of access to treatments that have been demonstrated to reduce ongoing burden of severe disease is strongly recommended, given limitations of clinical services in regional and remote Australia.

References

[1] Chisholm RH, Crammond B, Wu Y, Bowen AC, Campbell PT, Tong SYC, McVernon J, Geard N. A model of population dynamics with complex household structure and mobility: implications for transmission and control of communicable diseases. PeerJ. 2020; 8: e10203.
[2] Doherty Institute Modelling Report for National Cabinet. https://www.doherty.edu.au/uploads/content_doc/DohertyModelling_NationalPlan_and_Addendum_20210810.pdf

Work Package 2 – LGA Substream

Table of Contents

Key Messages

- Baseline transmission potential (TP) differs by small area, as do vaccine and PHSM impacts (ability to work from home).
  - Drivers include larger mean household size (leading to more household contacts), larger working-age populations (leading to more workplace contacts) and social determinants such as housing quality and crowding.
  - These factors tend to be geographically clustered and are often reported at the LGA-level.
  - Such variation also influences likely vaccine impacts at subpopulation level as LGAs with a higher proportion of children will be more likely to observe ongoing transmission in those aged less than 12 years, who are currently ineligible for vaccination.
  - In addition, inability to work from home reduces the impact of public health stay at home orders, and often correlates with higher baseline and post-vaccination transmission potential.
  - A model based on employment industry type correlates well with empirical survey data reporting 'working from home' under lockdown, by local government area (LGA).
- Focussed TTIQ and wrap around supports will be needed to constrain TP in high-risk areas, not lockdowns, and may include additional measures in schools and workplaces.

Aims

The national vaccination targets will have different effects across different sub-populations. Areas of socio-economic disadvantage are likely to require higher vaccination thresholds compared to the average to achieve a target level of control because of differences in baseline transmission potential and related impacts of vaccine coverage.

Here, we adopt the transmission potential framework used in previous pieces of modelling advice to account for these location specific differences.

The transmission potential (TP) represents average or expected transmissibility of SARS-CoV-2 in a population. In previous work and weekly situational assessment, we estimate and report on TP at a state-level. By adjusting the TP for various spatial factors, we can measure this expected transmissibility at the level of Local Government Areas (LGAs) rather than states.

Key questions

1. What is the spatial variability in the underlying ability for SARS-CoV-2 to spread in the population?
2. How much can this be modified with vaccination?
3. How much can this be modified with strong public health and social measures (PHSMs)?

Transmission potential at LGA level

Baseline TP and the effect of vaccination will vary by LGA, based on age profile. The vaccine rollout does not currently target children under 12 years of age, and so in LGAs with a high proportion of children, the relative impact on transmission of a vaccine will be reduced.

Experience has also shown that the ability of lockdowns to modify mixing and so reduce transmission differ across geographical areas. While a number of behavioural changes result in PHSM impacts, the ability to work from home can be anticipated with reasonable certainty based on occupation and validated on the basis of survey data. In some LGAs, there is a high proportion of people whose work cannot be done remotely and are considered ‘essential’, who will continue to have workplace contacts even under the most restrictive of PHSMs.

The combination of an increased baseline TP, lower vaccination coverage and lower PHSM effect combine to make understanding of these spatial differences complex, as highlighted in Figure 1. In many locations, these factors – population structure, vaccine impact, and ability to adhere to lockdowns – co-occur with socioeconomic disadvantage.

Figure 1 shows how population characteristics influence baseline transmission potential (upper limit of salmon bars) and vaccine impacts (blue shading) between 50 and 80% coverage. Compared with the ‘all Australian’ population, small area TP and vaccine impacts will be heterogeneous, as demonstrated by five exemplar LGAs each for greater Melbourne and Sydney.

Kingston (left panel) and Sutherland Shire (right panel) are most ‘typical’ of the national average. Affluent areas comprised of small households and a high proportion of working age adults (Port Phillip, Stonnington, North Sydney, Mosman) have an average baseline TP but larger than average vaccine change impacts. Areas like Greater Dandenong and Fairfield have a higher than average proportion of working age adults, which accounts for a higher starting TP but also marked reductions achieved following vaccination. Murrindindi and Oberon both have lower baseline transmission potential and vaccine impacts arising from higher proportions of children and older adults than the national average, respectively.

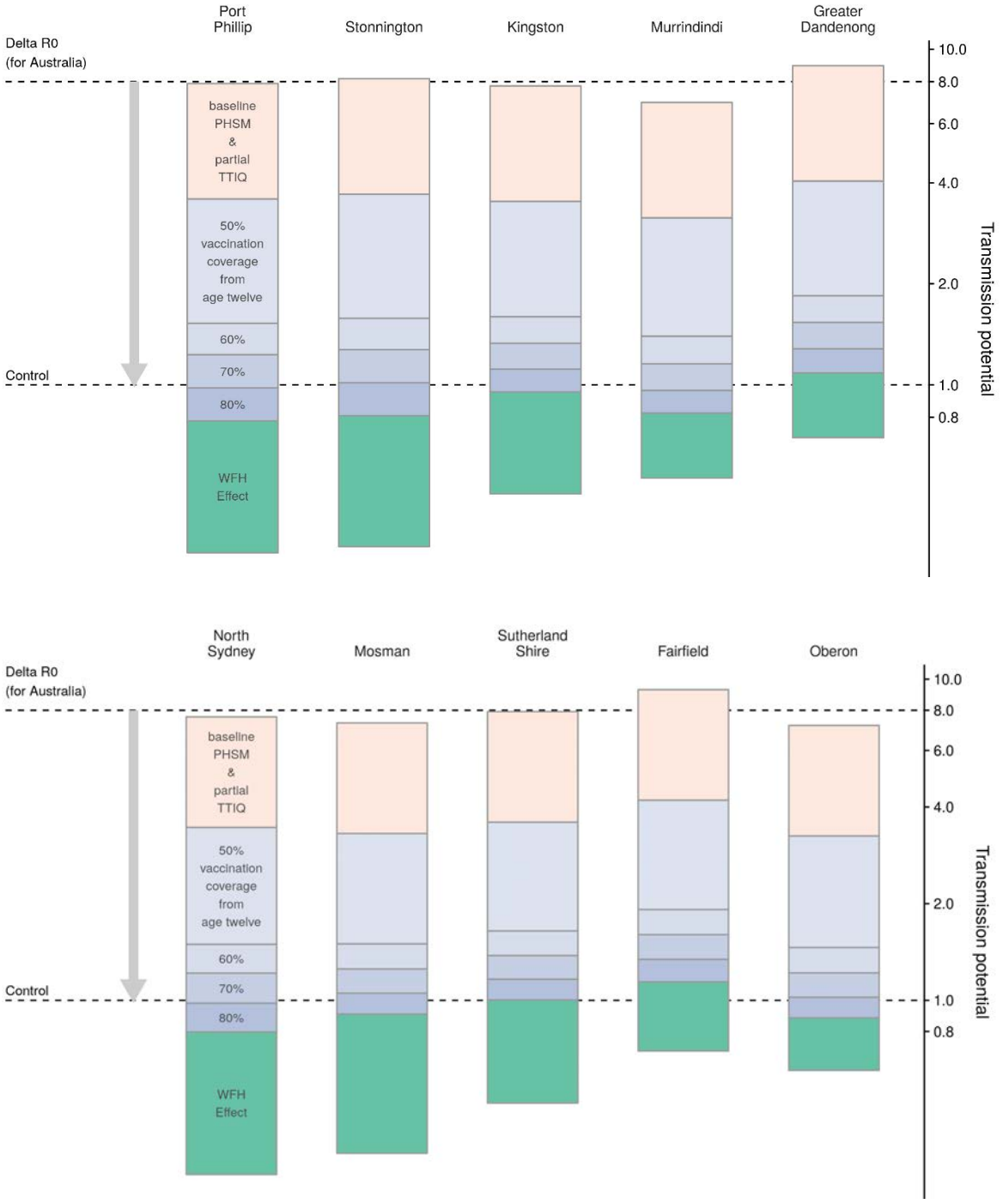

Figure 1: Visualisation of baseline TP, vaccination effect and ability to work from home on the overall TP achievable for LGAs in Greater Melbourne (upper panel) and Greater Sydney (lower panel)

Varying ability to work from home is reflected in the differences between the green components of Figure 1. Port Phillip, Stonnington, North Sydney and Mosman have large population proportions in professional occupations that are amenable to stay at home working. Greater Dandenong and Oberon each have higher than the national average proportion of machinery operators and labourers, who cannot work from home. Murrindindi and Fairfield have a larger than average proportion of children who are not in employment, lessening the impact of work from home requirements on overall levels of mixing in these areas under public health orders.

Spatial variability in transmissibility

Figure 2 maps the estimated transmission potential for LGAs in the Melbourne and Sydney Metropolitan areas. These baseline transmission potentials include LGA-specific R0 estimates (as per Figure 1) with the assumed effects of baseline PHSMs and partial TTIQ (i.e. these are comparable to a national TP of 3.6). Note that the resident populations of major city-centre LGAs (e.g. Melbourne and Sydney) have a very high transmission potential when considered by this metric, due to the small numbers of school-age children living there. However, in reality, CBD residents will have many contacts outside of the LGA, making these unreliable estimates of transmission in these settings.

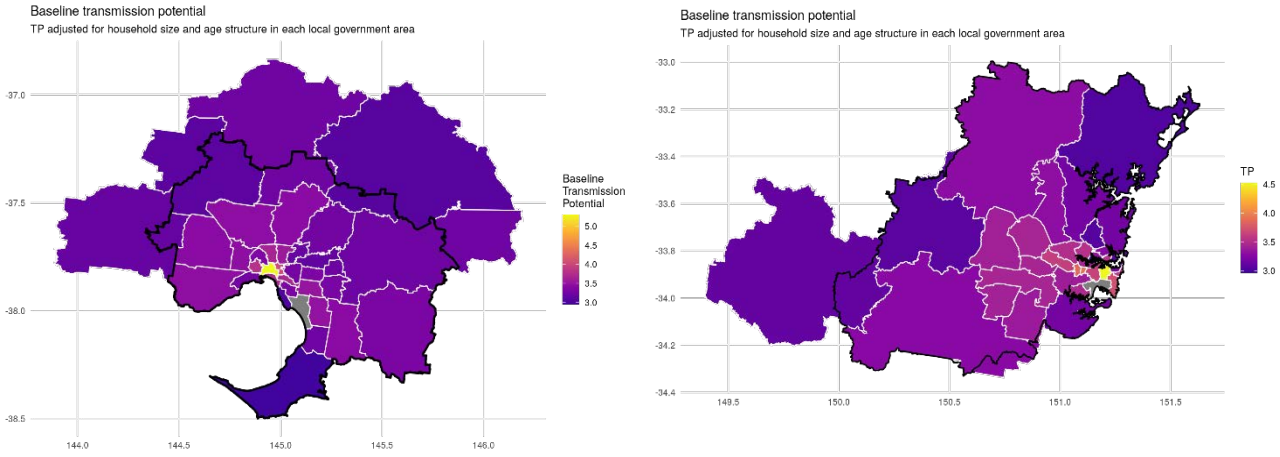

Figure 2: Baseline transmission potential, including differences in age structure and mean household size, by LGA in metropolitan Melbourne (left) and Greater Sydney (right).

Overall, many LGAs are below the national average transmission potential (calibrated to be 3.6, in line with previous national modelling work), although generally the risk increases the closer an LGA is to a metropolitan centre.

Note that this work focusses only on structural changes in the population (age structure and household size) and translates these into changes in contact patterns. It does not consider the measured changes in contact patterns in population subgroups. Substantially more observational data would be required to capture these patterns at a smaller resolution

Spatial variability in impact of vaccination

Figure 3 maps the percentage reduction in TP in each LGA from the baseline TP in Figure 2 to the TP expected after 80% coverage of the 12+ population. This includes a proportion of the population having received only a single dose as in the phase 1 report. There appears to be significantly more variation in this quantity than baseline TP. Further, the baseline transmission potential is not correlated with the percentage reduction, since changes in the numbers of contacts due to age structure and household size are not exactly offset by the effect of vaccination.

There appears to be significantly more variation in this quantity than baseline TP. Further, the baseline transmission potential is not correlated with the percentage reduction, since changes in the numbers of contacts due to age structure and household size are not exactly offset by the effect of vaccination.

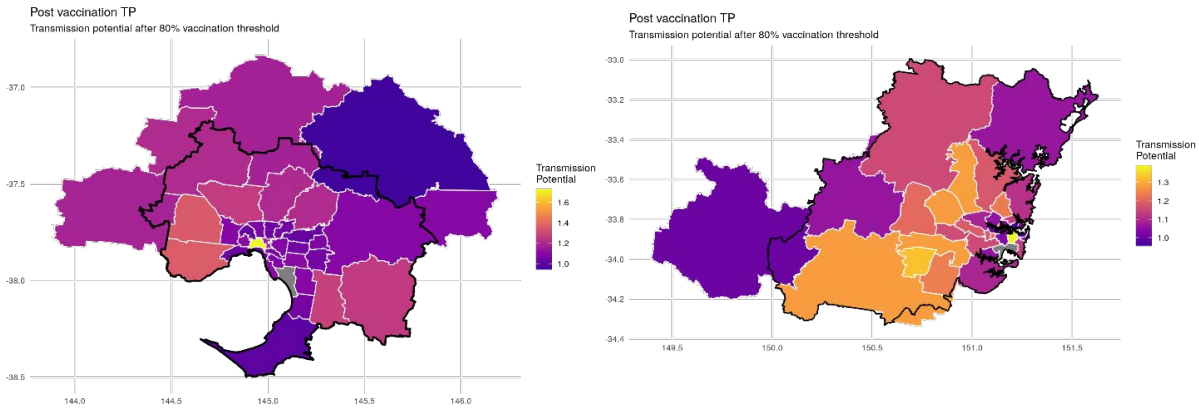

Figure 3: Transmission potential including the reduction due to vaccination at 80% coverage, by LGA in metropolitan Melbourne (left) and Greater Sydney (right).

Spatial variation in the ability for lockdowns to reduce TP

While an LGA (or region of an LGA) may have a high baseline TP, an important factor if infection is established is the ability for response measures to modify (reduce) that TP. It should not be assumed that regions with a higher baseline TP as necessarily those for which TP under lockdowns is also highest. Similarly, LGAs with an intermediate baseline TP may vary in how much change can be induced.

Within an LGA the proportion of working-age adults who are able to work from home is a possible metric for the population's ability to reduce their out-of-home contacts (and thus TP). Analysis of weekly-collected survey data shows variation in the proportion of individuals reporting to be working from home across LGAs under different levels of restrictions (Figure 4).

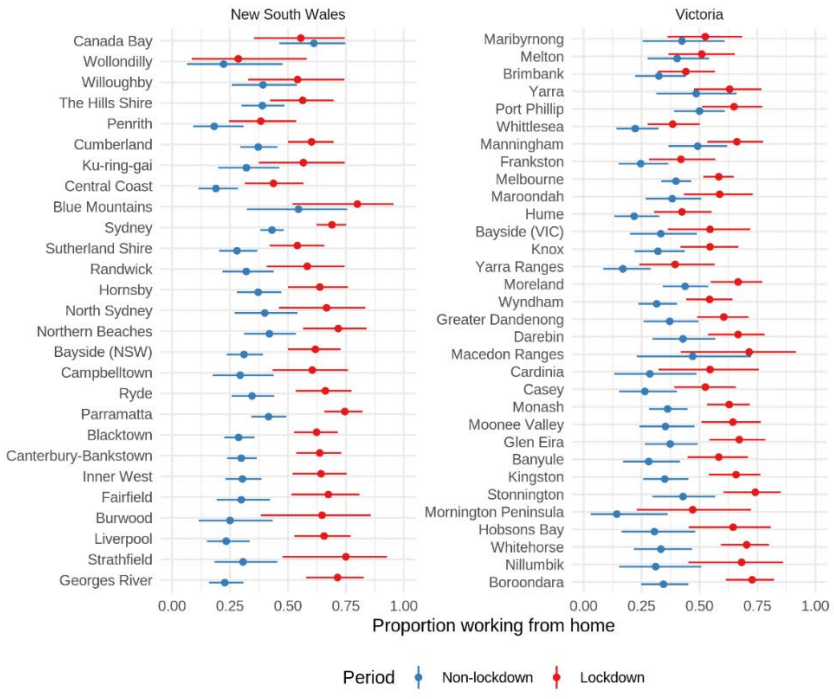

Figure 4: Survey responses on whether individuals are working from home or not, during lockdown and non-lockdown periods.

The survey data is relatively sparse, particularly in regional areas, and is difficult to extrapolate to all LGAs. To address this, we assess the correlation between the survey responses and the ability to work from home derived from occupation data from the 2016 census (1,2), termed the "modelled WFH ability". For metropolitan LGAs, the modelled WFH ability correlates well with survey-based responses. For non-metropolitan LGAs, there is insufficient resolution in available data to assess the validity of the approach (Appendix A: Validation of modelled WFH ability).

Figure 5 shows the modelled WFH ability for metropolitan Melbourne and Greater Sydney. As with the other measures, there is clear visible heterogeneity across the region, although the likelihood of being able to WFH appears correlated with proximity to a city centre.

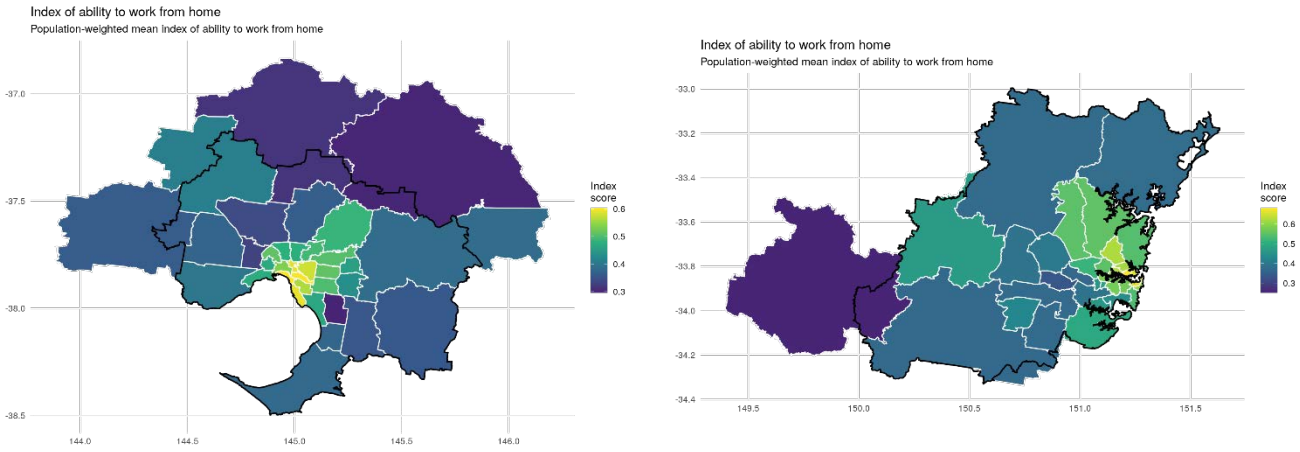

Figure 5: Modelled WFH ability for metropolitan Melbourne and Greater Sydney.

Sub-LGA heterogeneity

We anticipate heterogeneity in lockdown impacts at the sub-LGA level. If managing the epidemic at an LGA level, the required response will likely by driven by sub-sections of the LGA with the lowest ability to reduce out-of-household contacts and thus reduce transmission.

As the modelled WFH-ability is based on census data, it can be evaluated at different spatial scales to assess the variability. Figure 6 shows the modelled WFH-ability calculated for each SA2 that makes up an LGA, ordered by the lower confidence interval (left-hand endpoint of the black line). This figure highlights how the heterogeneity varies across LGAs, with areas such as Liverpool and Canterbury-Bankstown in NSW, as well as Brimbank and Mornington Peninsula in VIC having very high heterogeneity.

Where there is high variability within an LGA, the ability of the virus to spread in certain sub-populations may be higher than the population average (the LGA-wide TP). Sub-LGA analyses may reveal populations at-risk and therefore guide anticipated needs. Insight into sub-LGA level epidemic dynamics could be gained in real-time by comparing observed epidemic growth (R_{eff}) to LGA-level TPs.

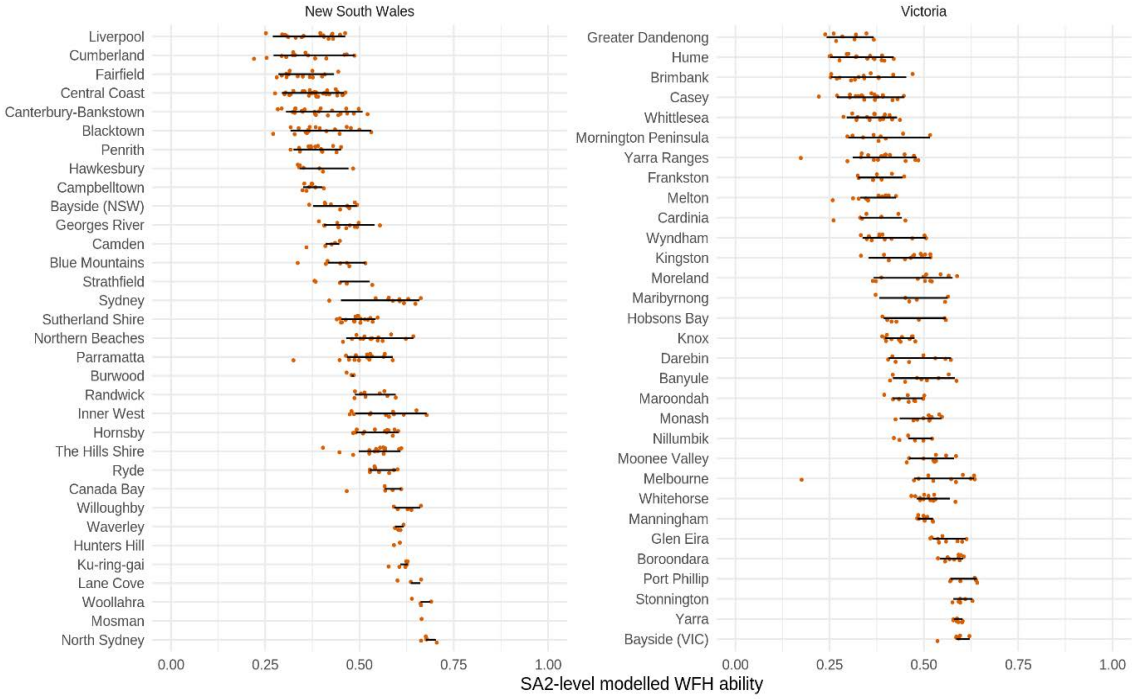

Figure 6: Proportion and 95% CI of modelled WFH ability in Greater Sydney and metropolitan Melbourne, calculated on each SA2 that is part of an LGA.

WFH Effect on TP

The WFH Effect, represented by the green bars in Figure 1, is calculated for each LGA and shown in Figure 7 assuming a static 80% vaccination coverage in each LGA. Here, the ability to WFH is much greater in city centres compared to the more regional areas. When all factors are included – vaccination, WFH effects and TTIQ measures – the transmission potential generally increases further away from city centres, indicating the reduction in transmission risk gained from WFH measures is critical in keeping case numbers in control, and that is far less effective in more regional centres.

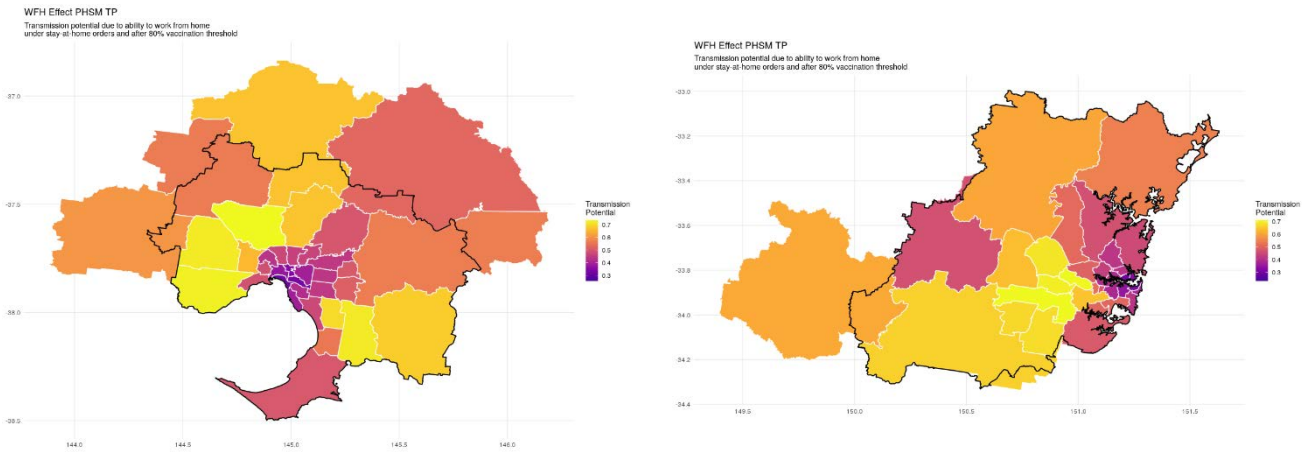

Figure 7: Transmission potential including 80% vaccination coverage, population demographics, and WFH effect, for metropolitan Melbourne and Greater Sydney.

Summary and next steps

This work has thus far shown that the heterogeneity in population factors is complex and multi-faceted. For example, an increase in baseline TP does not necessarily correlate to an increased risk at high vaccination thresholds.

It must also be stressed that LGAs are large geographical structures, and sub-structures within these areas may drive the behaviour of the entire LGA. Targeted measures, including increased messaging and community engagement, could have a greater impact than in more homogenous areas.

WFH measures have a highly varied effect across space. Stay at home orders will not necessarily mitigate importation and outbreak risks in many LGAs that would be anticipated to have higher than average ongoing risks of transmission, even with high 12+ vaccine coverage. Focused TTIQ responses, wrap around supports and school and workplace measures are more likely to effectively reduce transmission and disease impacts in these settings.

References

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Appendix A: Validation of modelled WFH ability

The modelled WFH ability was compared to the survey-based responses to working from home. These measures differ slightly in that the survey responses measure whether an individual *is* working from home, whereas the modelled WFH ability measures whether an individual *can* work from home. Figure 8 shows the modelled WFH ability score against the empirical survey responses, by non-lockdown and lockdown periods. There is a visible correlation between the two measures, although the trend appears somewhat confounded by the LGAs of concern in NSW, and the differences in what public health measures were applied across the state.

Figure 9 is a Bland-Altmann plot, showing the difference in modelled WFH ability and the survey responses, against the modelled WFH ability, stratified by whether an LGA is metropolitan or regional. Good model performance is represented by the points being contained in a single horizontal band. For metropolitan LGAs, this visually appears true.

There is a constant bias between the two measures, indicating that more people are going to work who could have worked from home according to the model. This bias will be captured in the quantification of how WFH affects the number of workplace contacts in the TP model.

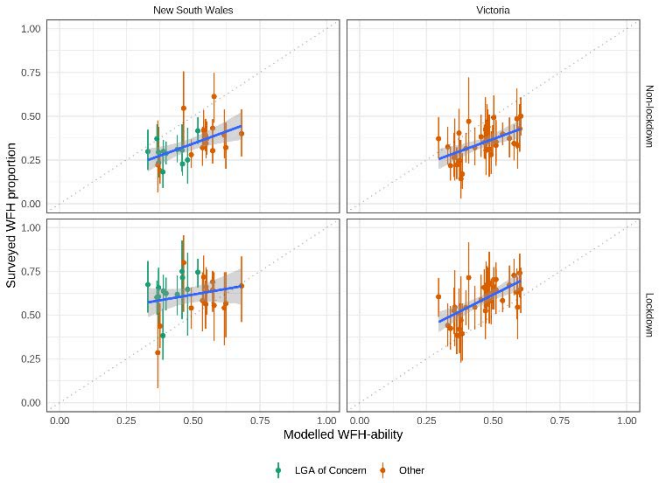

Figure 8: Modelled WFH ability against survey-based responses for NSW and VIC, separated by non-lockdown and lockdown time periods.

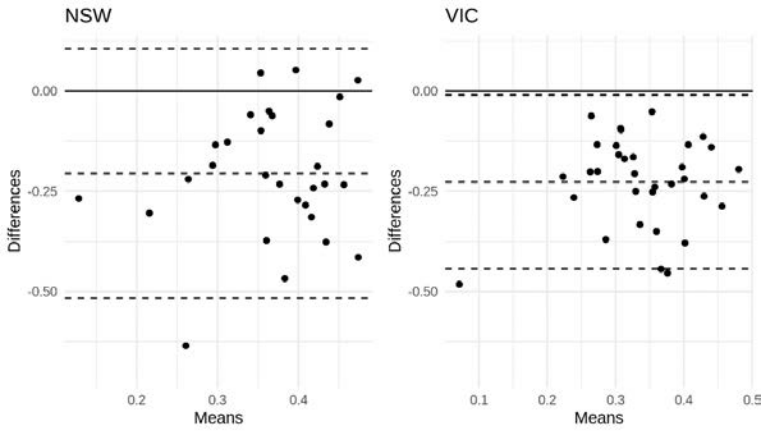

Figure 9: Bland-Altmann plot considering modelled WFH ability and survey based responses.

Work package 2.3: Schools

Executive summary

Returning students to in-person learning and keeping schools open has been identified as a national priority. This work assesses the effectiveness of a variety of school-based surveillance, contact tracing and quarantine strategies to prevent outbreaks, reduce transmission in schools, and maximize face-to-face teaching.

Key findings

1. **Early infection detection and high vaccine coverage markedly reduce outbreak risk.**
2. **Allowing ongoing school attendance for class contacts of a case through a 'test to stay' strategy achieves equivalent outbreak containment to home quarantine and enables face to face learning.**
   - This was true for primary and secondary schools.
   - The effectiveness of test-to-stay requires at least partial compliance with testing.
   - The high frequency of testing compensates for the reduced sensitivity of rapid antigen tests.
3. **Regular screening of students in areas at risk of outbreaks can result in even fewer infections and in-person teaching days lost.**
   - By detecting cases faster, there are fewer infections present when the first diagnosis is made and a lower risk of larger outbreaks occurring.
   - Identifying and isolating cases earlier leads to fewer downstream cases requiring isolation.
   - This was true for primary and secondary schools.
4. **School based surveillance testing will have maximum utility in areas with higher-than-average transmission.**
   - The benefits of student surveillance testing for reducing infections and days of face-to-face teaching lost increase as incursion rates increase.
   - More frequent screening provides greater benefits.
5. **Surveillance of teachers had minimal benefit for reducing outbreaks in schools.**
   - Teachers only comprise a small proportion of the school community.
   - However, this analysis only considered outcomes following an incursion in a school, and does not capture potential benefits that screening teachers may have on preventing of incursions.
6. **Findings are sensitive to assumptions for the number of non-classroom contacts students have.**
   - Quarantine or test-to-stay strategies focus on classroom contacts rather than close contacts as they are more practical to identify.
   - Strategies are less effective if a greater proportion of risk comes from non-class contacts.

This analysis focuses on transmissions taking place within schools, and does not consider the benefits of community public health responses on reducing incursions into schools, nor the benefits of school closure on reducing overall community transmission.

Background and aims

Background

Returning students to in-person learning and keeping schools open has been identified as a national priority.

The current public health response to COVID-19 cases in schools involves school closure following a positive case for cleaning (often three days), as well as 7 or 14-day quarantine for all close contacts and their households. If schools reopen with high levels of COVID-19 transmission in the community, rates of incursions into schools will also be higher, and the current approach to managing cases in schools may be unsustainable and inconsistent with the national priority of maximizing face-to-face teaching. Equally, allowing infections to spread within schools and the school community can lead to adverse health outcomes for students, their households and family members (e.g., parents and grandparents). Hence, different approaches to managing cases in schools and keeping schools open may be required.

Aim and scope of work

This work assesses the effectiveness of a variety of school-based surveillance, quarantine and testing strategies to determine which are likely to be the most appropriate for preventing outbreaks, reducing transmission in schools, and maximizing in-person learning. Due to the different epidemic situations across the country, the analysis is conducted for differing levels of community transmission and school incursion rates.

The analysis does not consider the benefits of community public health responses on reducing incursions into schools, nor the benefits of school closure on reducing overall community transmission. Reduced community transmission would lead to reduced school incursions, and the impact of higher or lower incursion rates are tested in sensitivity analyses.

The modelling considers primary and secondary schools and does not consider early learning or specialized settings (e.g., specialist schools and boarding schools).

Methods

Model overview

We used an established agent-based microsimulation model, *Covasim* [1], developed by the Institute for Disease Modelling (USA) and previously adapted by the Burnet Institute to model epidemics in Australia [2-5]. The model is open-source and available online [6]. Additional model details are provided in the appendix.

For this analysis, primary and secondary schools are modelled to have different social and mixing networks within and so are reported on separately.

Primary schools

Primary schools are modelled as a collection of classrooms, aggregated into schools. Each student is assigned to a classroom with others of the same age, and each classroom has an assigned teacher (Figure 1). Primary school mixing includes student-student contacts within classrooms, student-student contacts between students in different classrooms, teacher-teacher contacts and teacher-student contacts within the classrooms that they are assigned to.

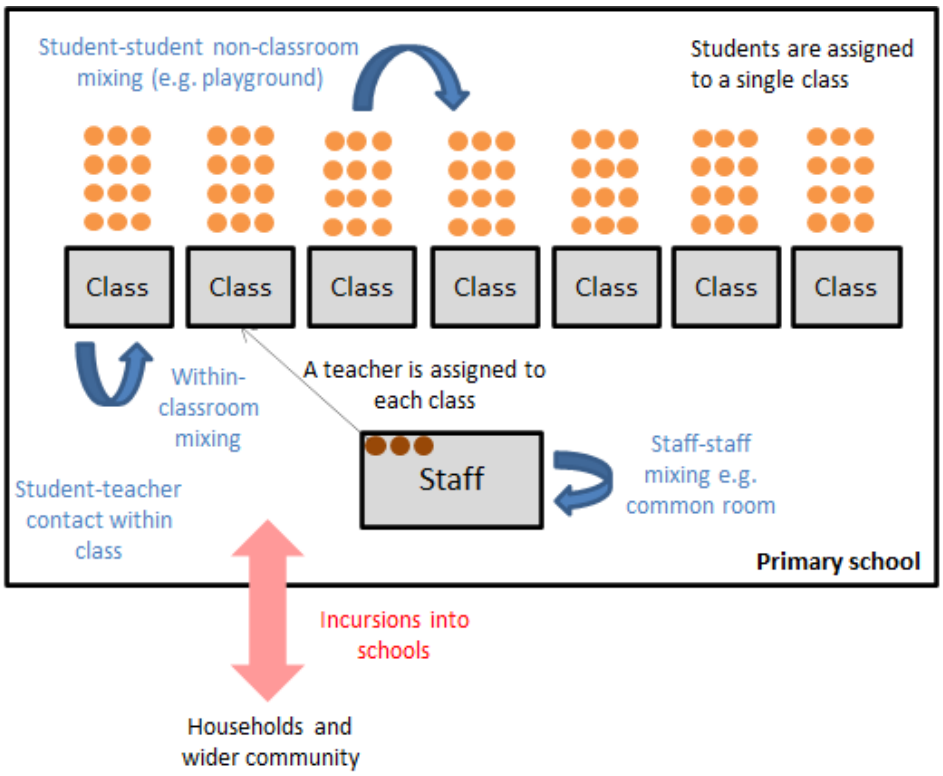

Figure 1: Contact networks within primary schools in the model. Primary schools are modelled as a collection of classrooms, where students of the same age are assigned a teacher. Primary schools include student-student classroom contacts, student-student non-classroom contacts, teacher-teacher contacts and teacher-student contacts.

Secondary schools

Secondary schools are modelled with a lower emphasis on assigned classrooms reflecting attendance at classes for multiple core and elective subjects. Hence secondary school students have a greater number of classroom contacts than primary school students. Secondary schools in the model include student-student classroom contacts, student-student non-classroom contacts, teacher-teacher contacts and teacher-student contacts (Figure 2).

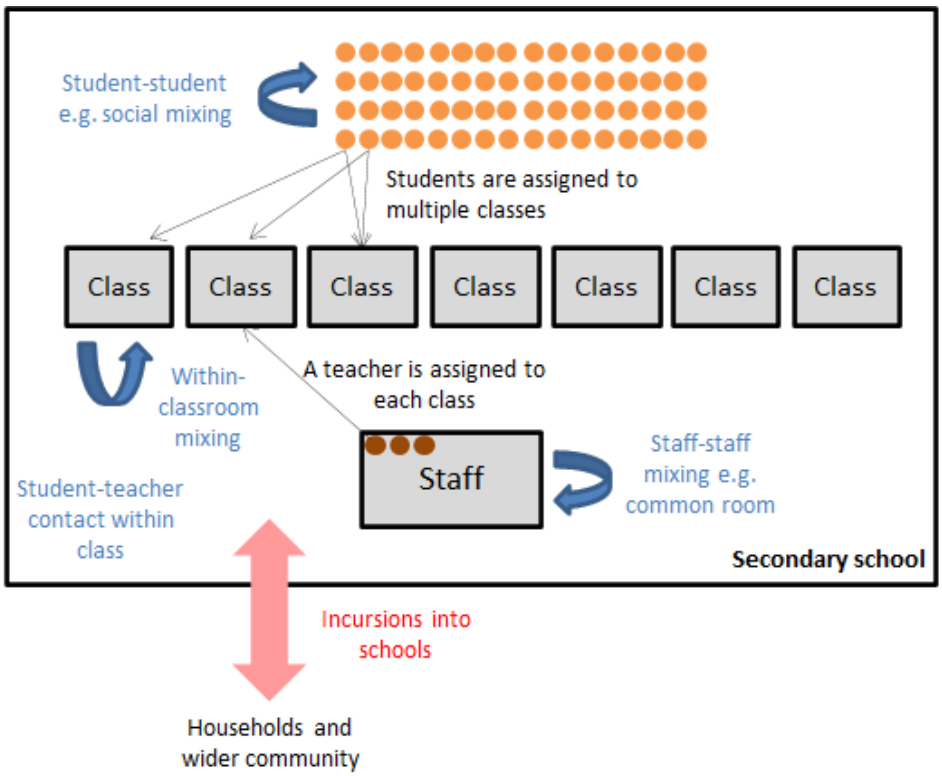

Figure 2: Contact networks within secondary schools in the model. Secondary school mixing includes student-student classroom contacts, student-student non-classroom contacts, student-teacher contacts, and teacher-teacher contacts. Secondary school students have more contacts than primary school students because they attend multiple classes.

Transmission in schools

Transmission is modelled to occur when a susceptible individual is in contact with an infectious individual through one of their contact networks. The overall transmission probability per contact per day has been calibrated based on the delta variant epidemic wave in Melbourne over the July-September 2021 period [5]. For individual contacts this transmission risk is further weighted according to the setting of the contact (e.g., classroom, home), the time-varying viral load of the person infected, whether or not they have symptoms (based on an age-specific probability of being symptomatic), and an age-specific disease susceptibility (Table 1).

Symptomatic testing probability (COVID-19 cases)

All people with severe disease are assumed to be tested. For people with mild symptoms, the model includes a per-day probability of seeking a test, which is necessary for the first case to be diagnosed when surveillance is not in place (noting that the first case to be detected may be a household member of a student at the school, which would trigger contact tracing for the student). Symptomatic testing assumes that people who have mild symptoms and are not identified through contact tracing or exposure site notification will seek testing during their symptomatic period with a per-day testing probability of 11% (varied in a sensitivity analysis).

The rest of the community

The non-school community is included in the model, to capture dynamics such as infected students transmitting to household members. This is relevant because adult household members who become infected may be more likely to seek symptomatic testing leading to detection of the outbreak, or siblings who become infected at home can reintroduce the infection to the school (noting that the model replicates the age and household structure of Australia). For all simulations, we assume that symptomatic testing and contact tracing in the general community continues, but that no public health restrictions are in place or introduced outside of schools.

School surveillance strategies

School surveillance strategies considered were no surveillance, twice weekly teacher screening with rapid antigen tests (RAT), and twice weekly student screening with RAT. These scenarios were considered with and without contact tracing in place.

Contact tracing and quarantine strategies in schools

In all scenarios, students or teachers diagnosed with COVID-19 were assumed to be removed from the school and required to isolate until no longer infectious.

Contact tracing scenarios were based around classroom contacts, as opposed to close contacts, as classroom contacts were deemed more practical to identify and apply policies to. Options considered were no contact tracing; 7-day quarantine of classroom contacts with/without daily RAT; daily RAT of classroom contacts who remain at school ("test-to-stay"); entire school test-to-stay with daily RAT after initial case detection. The inclusion of a 7-day quarantine with RAT was to create a fairer comparison to test-to-stay by allowing equivalent likelihood of case ascertainment.

Model simulations and outcomes

The model was initialized with a single infection allocated randomly within a school. The model was run for 45 days, recording the number of cumulative infections in students or teachers attending the school. Infections were used as the primary outcome measure as opposed to diagnoses to avoid biasing strategies with lower testing rates.

For each scenario, the simulation was repeated 1000 times and reported outcomes are based on the distributions of (1) secondary infections occurring in the same school; and (2) days of face-to-face teaching lost. Days of face-to-face teaching lost are calculated for the school as the total student-days spent in isolation or quarantine as a result of a school quarantine policy over the 45 day period.

Sensitivity analyses

Sensitivity analyses were conducted to consider how outcomes varied with different assumptions or inputs for:

- School incursion rates: model initialization with 1, 2 or 3 simultaneous incursions
- Vaccination coverage:
    - 0%, 60%, 80% coverage among students 12+ years

    - 0%, 60%, 80% coverage among students 5-11 years
    - 60%, 80%, 100% coverage among teachers
- Non-pharmaceutical interventions (e.g. ventilation, physical distancing): efficacy at reducing transmission probability per contact of 0%, 25% or 50%
- Surveillance testing frequency (weekly or daily compared with twice weekly)
- Compliance with test-to-stay (also an equivalent sensitivity analysis for lower test sensitivity): 0-100%
- Average number of non-classroom contacts per student
- Symptomatic testing rate

Except for incursion rate and compliance with test-to-stay, these are provided in the appendix.

Table 1: Model parameters related to schools

Parameter area	Estimate	Source
Primary school		
Average school size	298	Number of primary students (2,267,564 in 2020; ABS [7] Table 42b) divided by number of Primary + Primary/secondary schools (6249+1363 in 2021; ABS [7] Table 35b).
Average class size	22	Average class size of primary schools. Victorian government [8]
Average number of student-student non-classroom contacts per day, per student	2	Assumption; tested in sensitivity analysis. This impacts the efficacy of test-to-stay of class contacts verses close contacts or entire school.
Average number of teacher-teacher contacts per day, per teacher	20	Number of FTE primary teachers (152,281 in 2020; ABS [7]) divided by number of primary schools (6249+1363)
Secondary school		
Average students per school	622	Number of secondary students (1,738,083 in 2020; ABS [7] Table 42b) divided by number of Secondary + Primary/secondary schools (1433+1363; [7] Table 35b)
Average teacher/student ratio	12	ABS data. [7] suggesting secondary schools have on average 12.1 students to one teacher.
Average number of student-student classroom contacts per day	44	Average class size in secondary school of 22 ([9]; page 354), assuming two unique classrooms of contacts per student per day.
Average number of student-student non-classroom contacts per day	5	Assumption; tested in sensitivity analysis. This impacts the efficacy of test-to-stay of class contacts verses close contacts or entire school.
Average number of teacher-teacher contacts per day	5	Assumption.
Average number of teacher-student contacts per day, per student	6	Assumes students have six classes per day
Probability of transmission per contact per day (without vaccines or NPIs)		
Student-student (primary classroom)	0.25	Delphi process; Scott et al. [2] Measured as relative to household transmission per contact - e.g. a typical day's worth of contact in school is 75% less likely to result in transmission than a typical day's worth of contact at home. Non-classroom primary school contacts equivalent to outdoor contacts; secondary school classroom contacts halved to account for shorter interactions. All transmission probabilities are scaled in sensitivity analyses when NPI efficacy is tested.
Student-student (primary non-classroom)	0.03	
Student-student (secondary class contact)	0.12	
Student-student (secondary close/social contact)	0.12	
Teacher-teacher	0.25	Assumption that transmission risks in schools are equivalent for all types of contacts. Note that the model has independent parameters to account for differences in susceptibility by age
Teacher-student (primary)	0.25	
Teacher-student (secondary)	0.12	
Age-susceptibility (relative to 20-49 year old)		
Age 0-4	0.349	Derived from Davies et al. [10]
Age 5-9	0.423	
Age 10-14	0.495	

Age 15-19	0.599	
Age 20-24	0.846	
Age 24-29	1	
Probability of being symptomatic		
Age 0-9	0.28	Davies et al. [10]
Age 10-19	0.20	
Age 20-29	0.26	
Rapid antigen testing (RAT)		
Sensitivity	0.773	Muhi et al. [11] Lower bound selected to account for inconsistent self-use. Note that PCR is modelled as having 87% sensitivity in real world use (systematic review Arevalo-Rodriguez et al. [12])

Results

Surveillance strategies, without contact tracing / quarantine

Even though secondary school students have a greater number of contacts, the chances of an incursion leading to zero secondary cases (after 45 days) was greater in secondary schools than in primary schools (

Figure 3, left green bars) – largely a result of secondary school students being vaccinated.

Twice weekly screening of teachers had minimal impact on reducing infections in primary schools, and only a marginal impact in secondary schools, since teachers make up a small percentage of the school community. However, this analysis focuses on transmission within schools, and considered outcomes given a random incursion into the school. It therefore does not capture differences between students and teachers in their probability of acquiring COVID-19 in the community. The analysis presented here likely underestimates the overall benefits of screening (and vaccinating) teachers through preventing incursions from taking place.

Twice weekly screening of students leads to earlier detection of an incursion and reduces the number of exposure days in the school. This increases the chances of an incursion leading to no secondary infections in both primary and secondary schools, because the index cases are often detected and removed from the school before transmission occurs. Screening of students increased the average days of face-to-face teaching lost compared with no screening and no contact tracing due to the detection of asymptomatic cases; however the days of face-to-face teaching lost were entirely due to positive cases isolating.

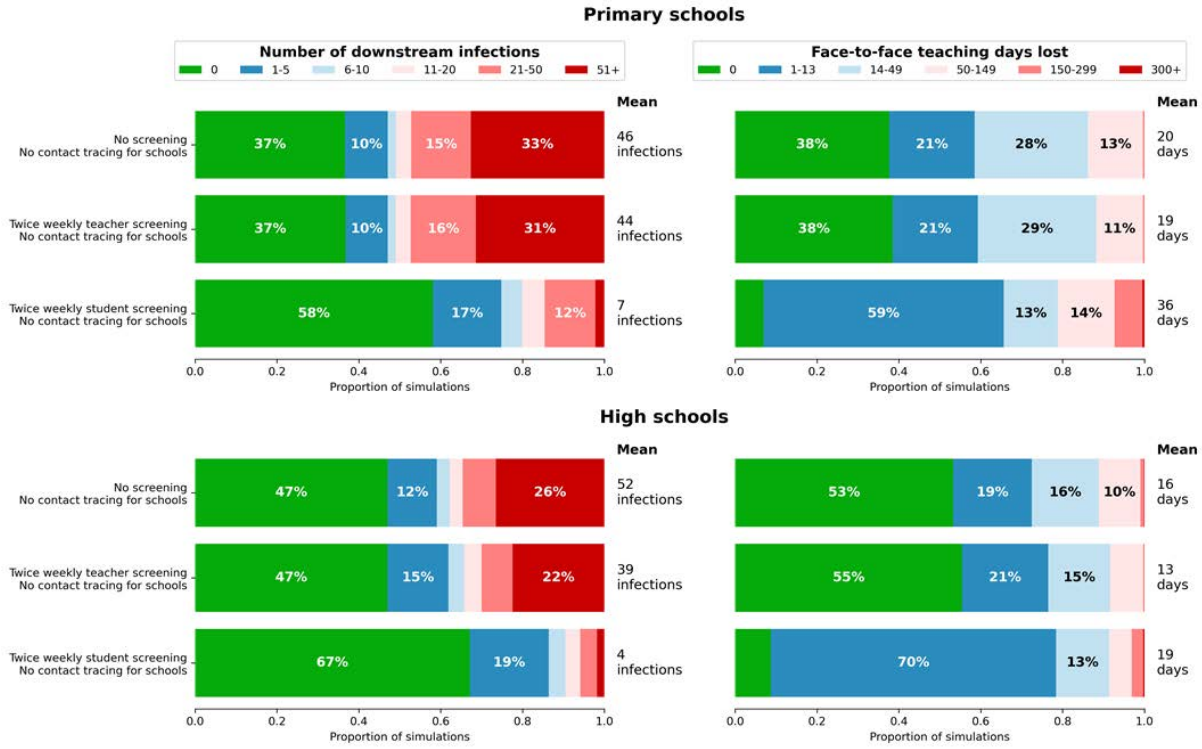

Figure 3: Impact of surveillance strategies on the distribution of outcomes for cumulative infections (left) and days of face-to-face teaching lost (right) in a single school following a single incursion. Outcomes are from 1000 model simulations run for 45 days following first diagnosis. Scenarios assume no contact tracing or quarantine (only isolation for positive cases that are detected) and from top to bottom are based on: no screening; twice weekly testing of teachers with rapid antigen tests; twice weekly testing of students with rapid antigen tests.

Contact tracing and quarantine strategies

Following detection of a case, different responses made some difference to the distribution of outcomes. Test-to-stay of classroom contacts was approximately equivalent to 7-day quarantine of classroom contacts in both primary and secondary schools, but with a significantly lower number of face-to-face teaching days lost (Figure 4). The incremental benefit of test-to-stay for the entire school, in place of just the classroom contacts, was small; however it was sensitive to assumptions about the number of non-classroom contacts that students have.

The effectiveness of test-to-stay was dependent on compliance with the daily rapid antigen testing (Figure 5), but even at partial (e.g. 50%) compliance was effective relative to no test-to-stay or quarantine.

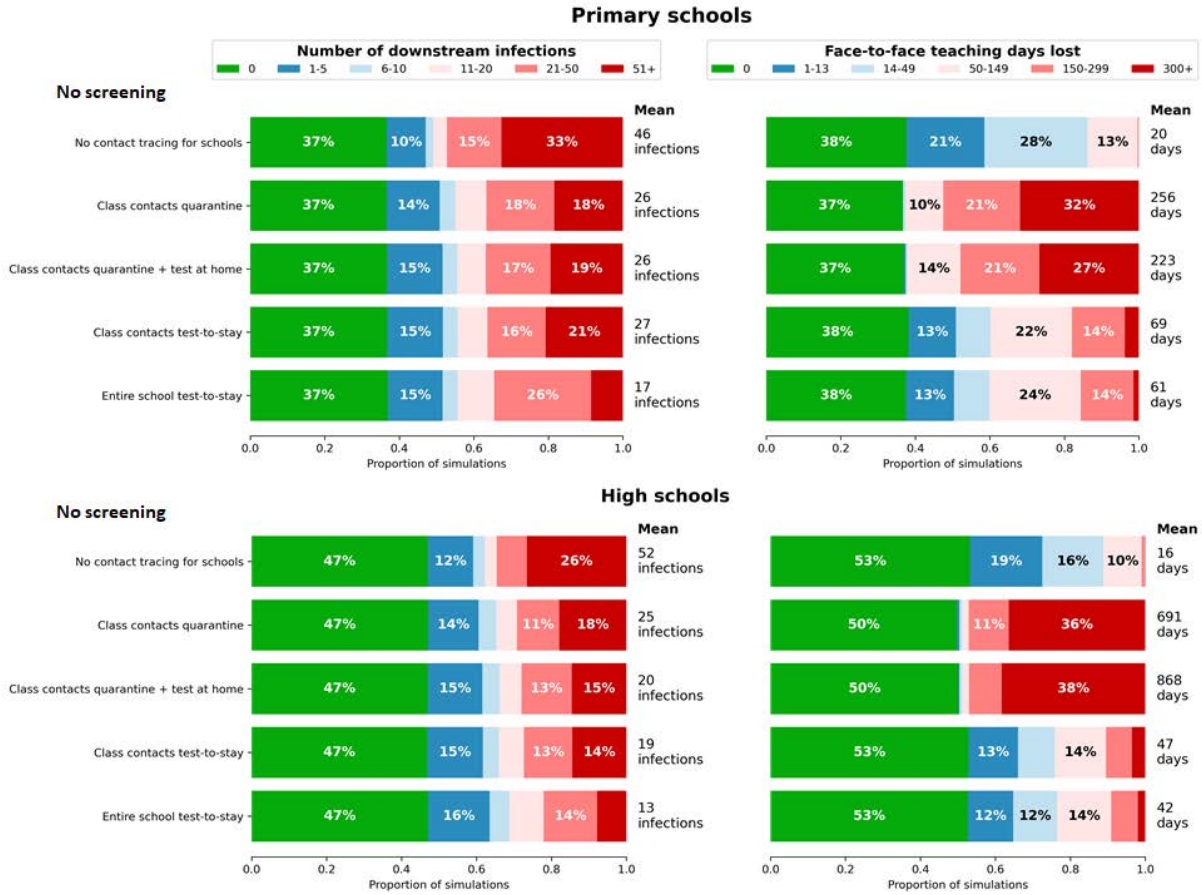

Figure 4: Impact of contact tracing and quarantine strategies on the distribution of outcomes for cumulative infections (left) and days of face-to-face teaching lost (right) in a single school following a single incursion. Outcomes are from 1000 model simulations run for 45 days following first diagnosis. Scenarios top to bottom: no contact tracing; class contacts have 7-day quarantine without / with testing; class contacts test-to-stay with rapid antigen tests; entire schools test-to-stay with rapid antigen testing. Top: Primary schools; bottom: secondary schools.

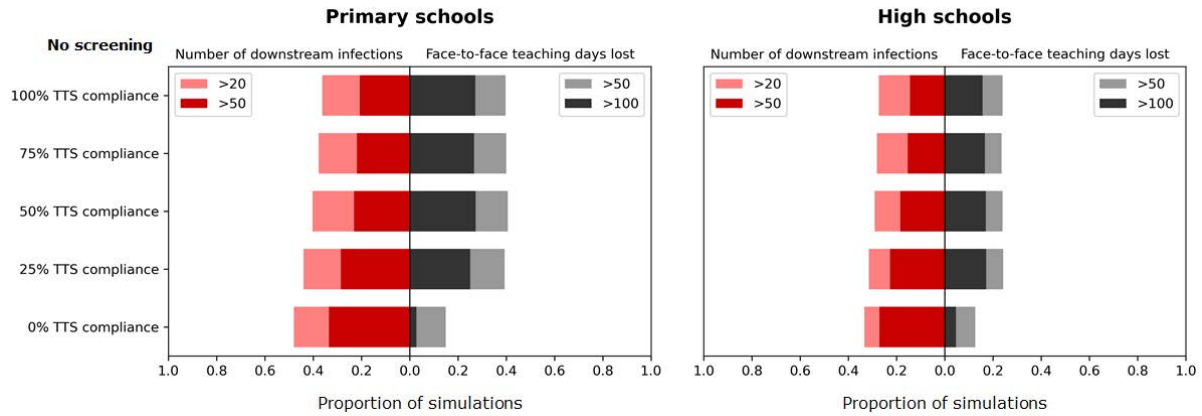

Figure 5: Impact of compliance on the effectiveness of a test-to-stay (TTS) strategy. Left bars: the percentage of simulations with more than 20 or 50 cumulative infections after 45 days of first diagnosis, for different surveillance strategies and number of initial incursions. Right bars: the percentage of simulations with more than

50 or 100 days of face-to-face teaching lost in a single school following the incursions. Outcomes are from 1000 model simulations run for 45 days following first diagnosis.

Surveillance strategies combined with contact tracing / quarantine

An additional analysis was undertaken to assess the incremental impact of surveillance strategies when contact tracing was in place. Test-to-stay strategy was used as a baseline for this analysis due to its superiority to other contact tracing and quarantine strategies in terms of minimizing infections and maximizing face-to-face teaching.

With contact tracing (test-to-stay) in place, twice weekly screening of students still had benefits in terms of reducing infections and had additional benefits in terms of reduced face-to-face teaching days lost (Figure 6). Since contact tracing is effective at detecting and isolating positive cases once an outbreak is identified, larger outbreaks in schools generate more days of face-to-face teaching lost. Therefore, by detecting and removing cases earlier, student screening combined with test-to-stay for class contacts could reduce the number of downstream infections following an incursion, reduce the likely outbreak size, and reduce the average days of face-to-face teaching lost per incursion. Despite student screening leading to fewer instances of zero days of face-to-face teaching lost – due to most incursions being detected and at least one infected student being isolated – there was also a significant reduction in the proportion of simulations where more than 150 days were lost.

With contact tracing (test-to-stay) in place, the relative benefits of twice weekly screening of students on reducing secondary infections in schools and days of face-to-face teaching lost increased as the number of incursions increased (Figure 7).

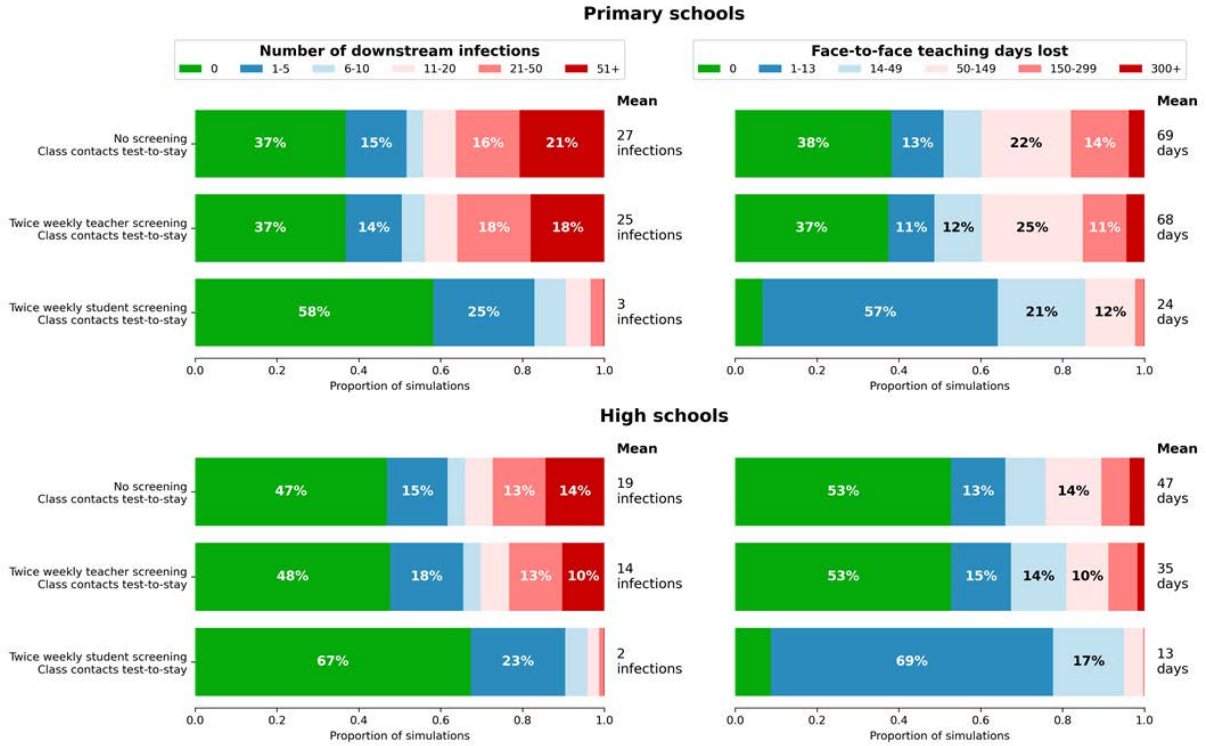

Figure 6: Impact of surveillance strategies on the distribution of outcomes for cumulative infections (left) and days of face-to-face teaching lost (right) in a single school following a single incursion. Outcomes are from

1000 model simulations run for 45 days following first diagnosis. Scenarios assume classroom contacts test-to-stay and from top to bottom are based on: no screening; twice weekly testing of teachers with rapid antigen tests; twice weekly testing of students with rapid antigen tests.

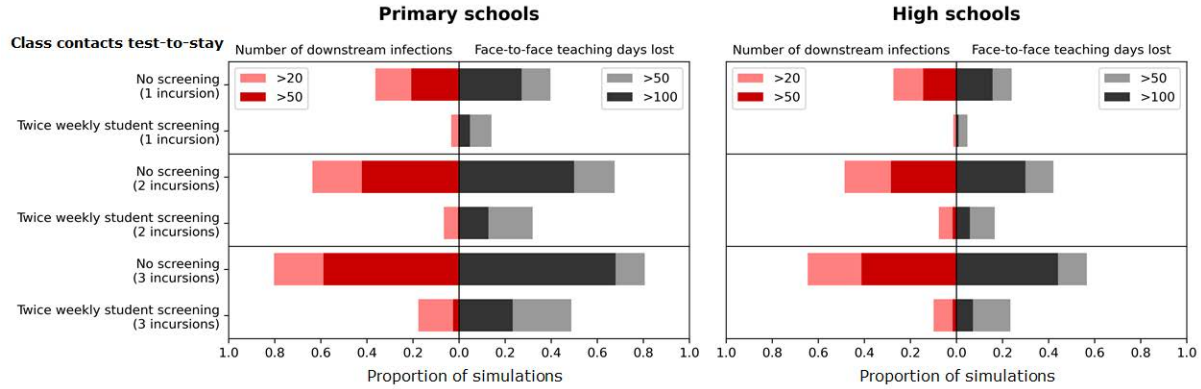

Figure 7: Impact of multiple incursions on the benefits of surveillance testing. Left bars: the percentage of simulations with more than 20 or 50 cumulative infections after 45 days of first diagnosis, for different surveillance strategies and number of initial incursions. Right bars: the percentage of simulations with more than 50 or 100 days of face-to-face teaching lost in a single school following the incursions. Outcomes are from 1000 model simulations run for 45 days following first diagnosis. Scenarios assume classroom contacts test-to-stay and from top to bottom have: no screening; twice weekly testing of teachers with rapid antigen tests; twice weekly testing of students with rapid antigen tests.

Total days of face-to-face teaching gained

The above outputs relate to the number of face-to-face teaching days lost following a single incursion; however, schools will experience ongoing incursions, with an incursion rate influenced by transmission in the surrounding community. By early 2022, empirical data will be available to measure the actual incursion rate. In the absence of these data, we estimate the incursion rate here to outline how a cost-effectiveness analysis for screening may be performed.

Between June and October 2021 in NSW and Victoria approximately 30% of new diagnoses occurred in people aged 18 and under, and this was consistent across high and low transmission settings (regional NSW: 28.4%, Sydney: 29.9%, Victoria: 30.2%). However, 12-15 year olds only became eligible for vaccines from 13 September so this may partly explain this outcome, which may change over time.

The current relative stability in the proportion of new cases that occur in school-age children makes it possible to infer crude estimates of the total number of face-to-face teaching days gained through student screening. For a particular community, this could be simplistically estimated by multiplying:

a) New daily cases in local community (diagnoses/day not in quarantine)
b) Proportion of new cases that occur in school-age children
c) School attendance in the community (a mixture of enrollment rates and any other community restrictions modifying attendance)
d) Screening period (days) of testing in schools (e.g. to estimate the potential impact of a term of screening)
e) Average days of in-person learning gained from a single incursion in a single school due to screening (i.e. difference in average model outputs from (Figure 6).

However, caveats to this approach must be noted. Most notably, stability in the proportion of cases occurring in school-age children between June-October is an artefact of the restrictions that were in place in NSW and Victoria at that time, particularly those enforcing school closure. There is also uncertainty in the percentage of all infections that are diagnosed, which depends on community testing rates – this is likely to underestimate incursion rates. Conversely, for communities with high transmission and frequent incursions, the outcomes of each incursion may not be independent and so this may overestimate the face-to-face teaching days gained.

Example: test-to-stay with/without twice weekly screening

When comparing test-to-stay with or without twice weekly screening of students, the average number of face-to-face teaching days gained per school per incursion is estimated to be (Figure 6):

- 45 for twice-weekly screening of students in primary schools;
- 34 for twice-weekly screening of students in secondary schools.

Using these results, the number of days of face-to-face teaching days gained due to screening have been estimated for a population of 100,000 over a 45-day period (Figure 8).

The greatest number of face-to-face teaching days gained through screening occur when incidence is highest.

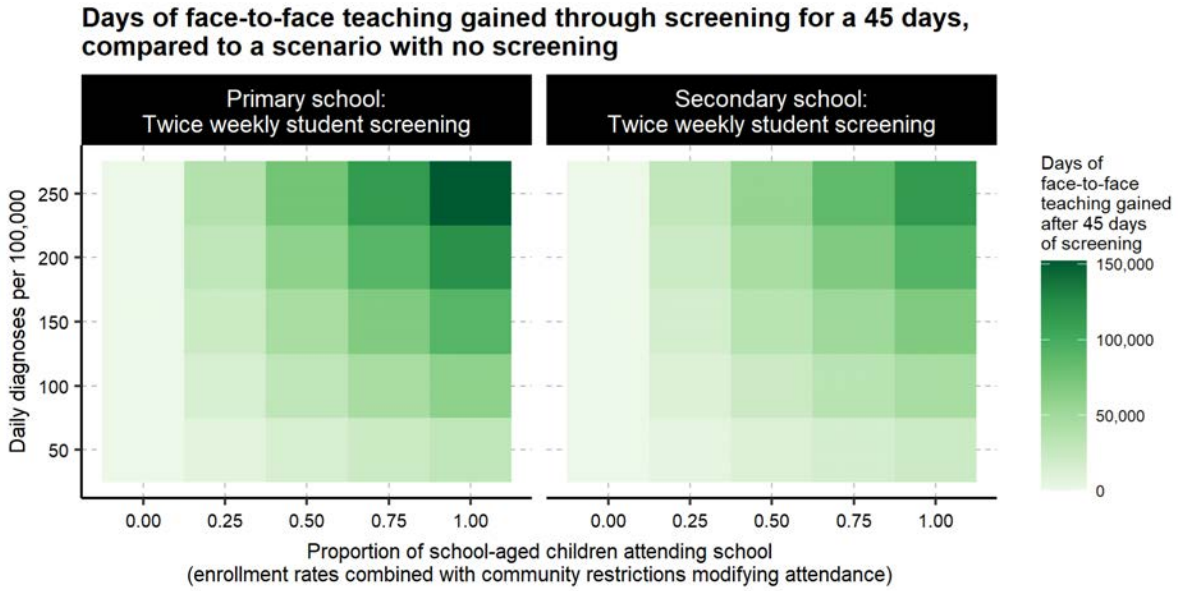

Figure 8: Estimated total days of face-to-face teaching gained through twice weekly student screening for 45 days of screening in a community with 100,000 population. Example assumes test-to-stay is in place alongside the screening. Left: Primary school. Right: secondary school. Assumes 30% of community infections are in school-aged children, an average of 45 and 34 days of face-to-face teaching are gained per incursion in primary and secondary schools respectively. Outcomes are shown for a range of community infection rates and school attendance rates (percentage of school-aged children attending school).

Limitations

The findings presented are derived from an individual-based model, which is an imperfect representation of the real world.

- Mixing within schools in the model is approximated as classroom and non-classroom contacts, where students are allocated at random to classrooms and randomly mix with other students outside of classrooms. In reality, within-school mixing is likely to include clustering due to subject selection and social mixing.
- Incursion risk was not modelled explicitly and model simulations started from a single assumed incursion. Actual incursion rates will depend on community prevalence, vaccination coverage and public health restrictions and interventions in place.
- The initial incursion that was modelled was randomly allocated to a member of the school (student or teacher); however, there may be social or other factors that make teachers or older/younger students more likely to be exposed in the community, and hence more likely to be the index case within the school.
- These results do not consider early learning or specialized settings (e.g. specialist schools and boarding schools) or small rural schools.
- Model parameters are based on best-available data at the time of writing. Results from new studies could change estimates of social mixing, contact networks, adherence to policies, quarantine advice, and disease characteristics (e.g. asymptomatic cases), and these could change these results.

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Appendix: Sensitivity analyses

Non-pharmaceutical Interventions in schools

The impact of non-pharmaceutical interventions (NPIs; e.g. masks, ventilation) were tested by running scenarios where the risk of transmission per contact was reduced by either 25% or 50%. NPIs can reduce outbreak risks in schools and reduce the number of days of face-to-face teaching lost.

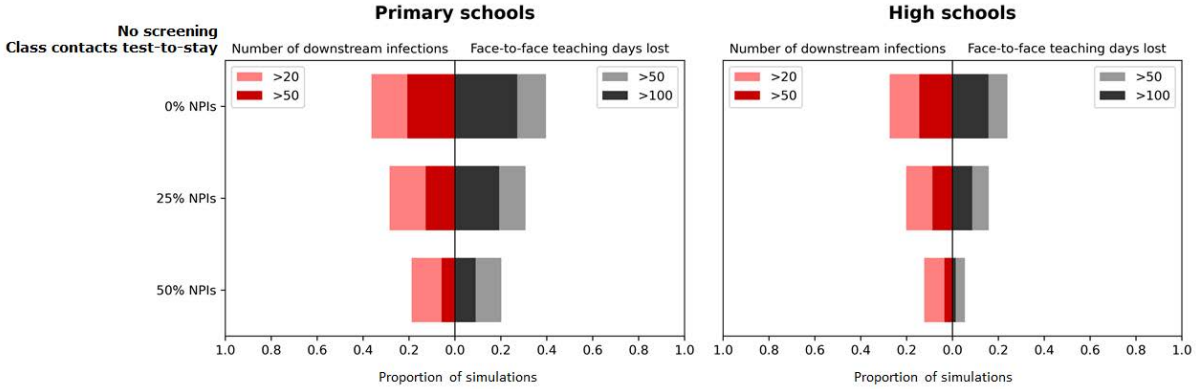


Figure 9: Impact of non-pharmaceutical interventions (NPIs) on outbreaks in schools. Red bars: the percentage of simulations with more than 20 or 50 cumulative infections after 45 days of first diagnosis. Grey bars: the percentage of simulations with more than 50 or 100 days of face-to-face teaching lost in a single school following an incursion. Scenarios assume test-to-stay is in place for class contacts and no surveillance testing.

Vaccine coverage in students

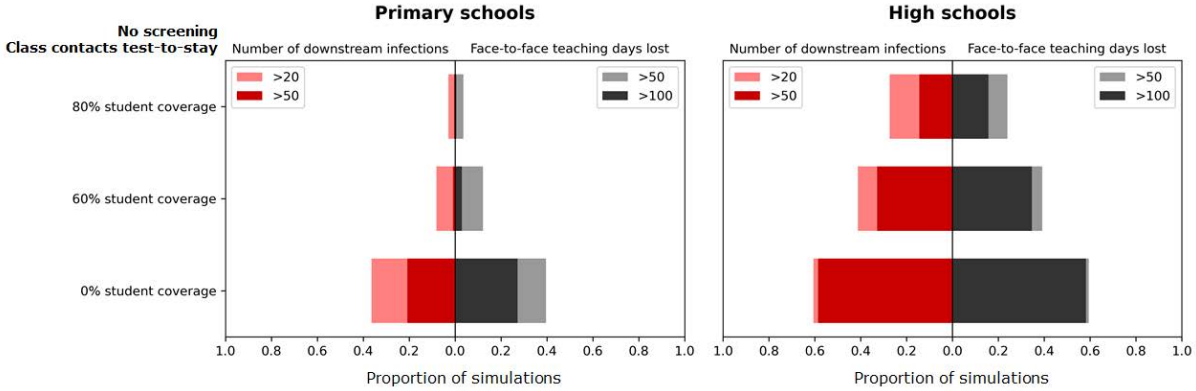


Figure 10: Impact of vaccines for students on outbreaks in schools. Red bars: the percentage of simulations with more than 20 or 50 cumulative infections after 45 days of first diagnosis. Grey bars: the percentage of simulations with more than 50 or 100 days of face-to-face teaching lost in a single school following an incursion. Scenarios assume test-to-stay is in place for class contacts and no surveillance testing.

Vaccine coverage in teachers

Note that the benefits of vaccinating teachers are not fully captured in this analysis, since the model does not account for potential reduced incursions as a result of teacher vaccination – only reduced transmission within the school once an incursion has already occurred.

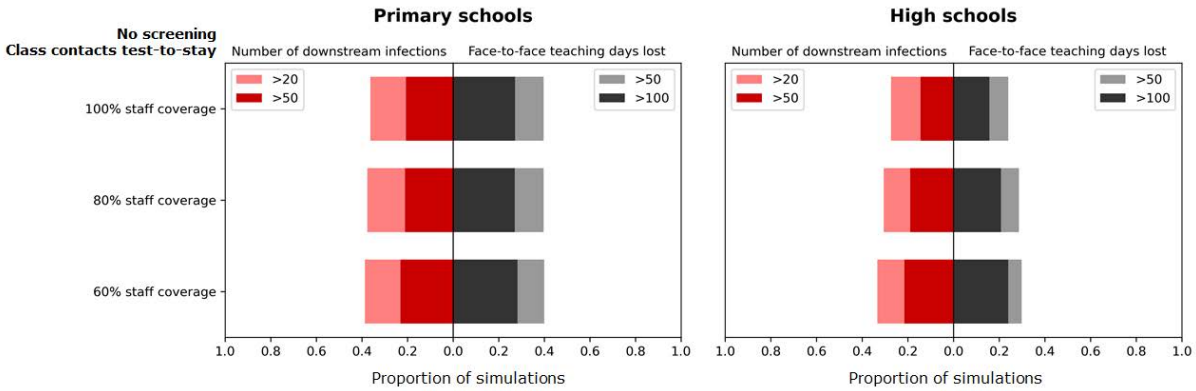


Figure 11: Impact of vaccines for teachers on outbreaks in schools. Red bars: the percentage of simulations with more than 20 or 50 cumulative infections after 45 days of first diagnosis. Grey bars: the percentage of simulations with more than 50 or 100 days of face-to-face teaching lost in a single school following an incursion. Scenarios assume test-to-stay is in place for class contacts and no surveillance testing.

Frequency of surveillance screening

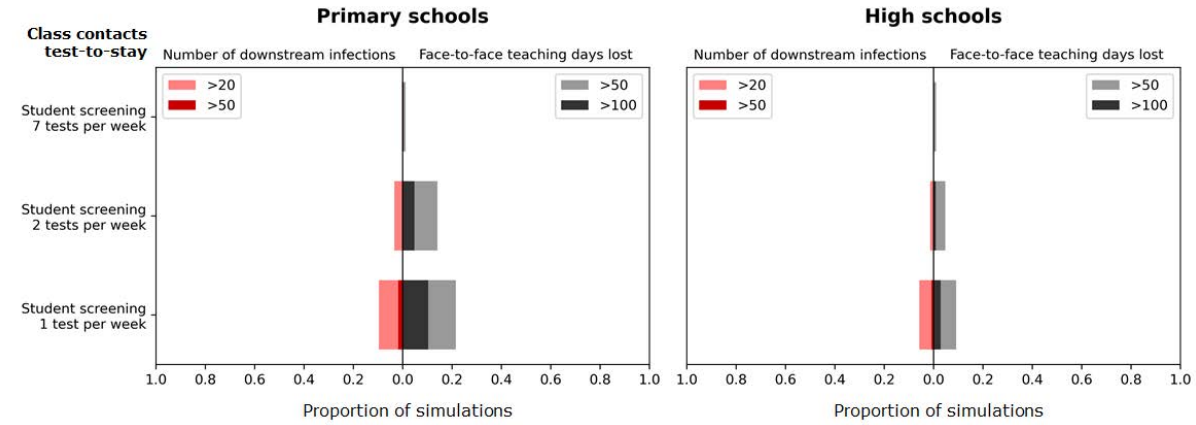


Figure 12: Impact of different frequencies of surveillance testing on outbreaks in schools. Red bars: the percentage of simulations with more than 20 or 50 cumulative infections after 45 days of first diagnosis. Grey bars: the percentage of simulations with more than 50 or 100 days of face-to-face teaching lost in a single school following an incursion. Scenarios assume test-to-stay is in place for class contacts.

Symptomatic testing rate

The model has an underlying parameter for the per-day probability that an individual with mild COVID-19 symptoms will have a test. This parameter plays an important role in determining how long it takes to detect an outbreak in scenarios where regular testing of students or teachers are not in place. Hence a sensitivity analysis was run to understand what influence this parameter had on key outcomes. Figure 13 shows that maintaining symptomatic testing is important for earlier detection of outbreaks and reduced outbreak size.

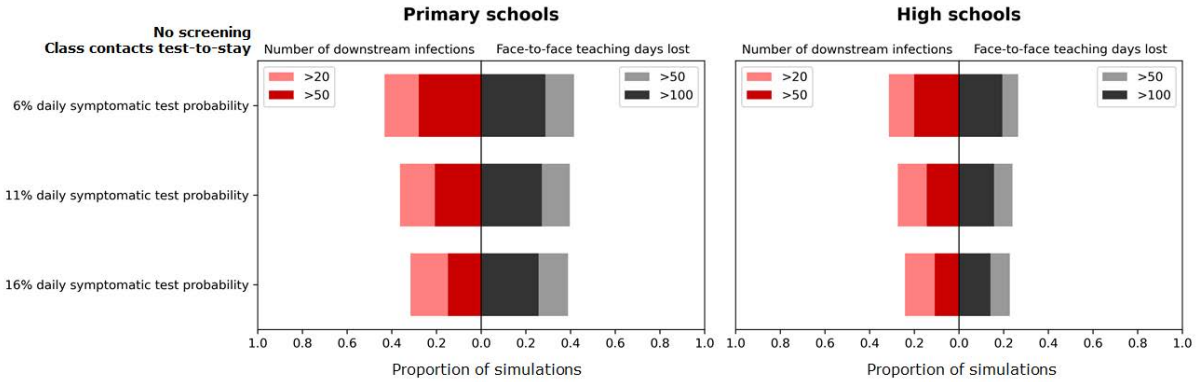


Figure 13: Impact of symptomatic testing probability on outbreaks in schools. Red bars: the percentage of simulations with more than 20 or 50 cumulative infections after 45 days of first diagnosis. Grey bars: the percentage of simulations with more than 50 or 100 days of face-to-face teaching lost in a single school following an incursion. Scenarios assume test-to-stay is in place for class contacts and no surveillance testing.

Sensitivity to number of non-classroom contacts

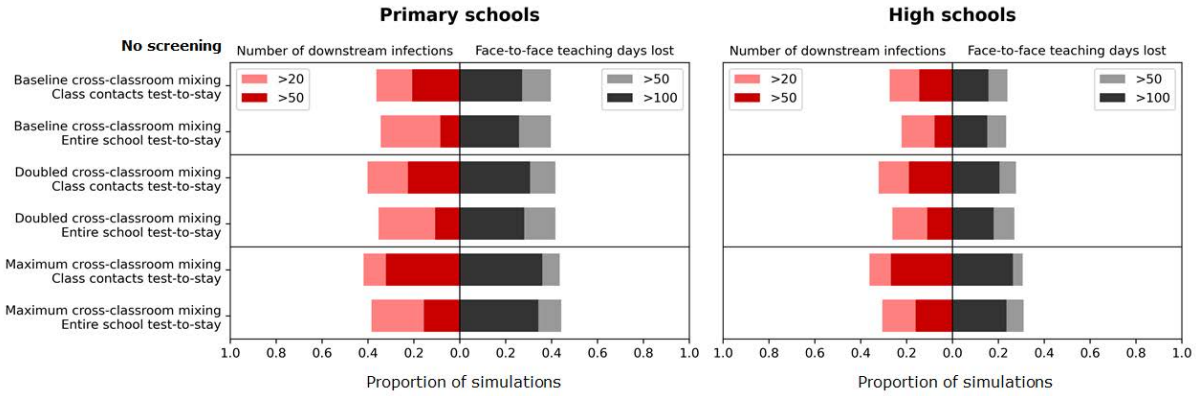


Figure 14: Impact of assumptions around number of non-classroom contacts per student. Doubled cross-classroom mixing assumes 4 and 10 non-classroom contacts for primary and secondary school students respectively. Maximum cross-classroom mixing assumes 11 and 22 non-classroom contacts for primary and secondary school students respectively. Red bars: the percentage of simulations with more than 20 or 50 cumulative infections after 45 days of first diagnosis. Grey bars: the percentage of simulations with more than 50 or 100 days of face-to-face teaching lost in a single school following an incursion. Scenarios assume no surveillance testing.

Appendix: Additional methodological details

The agent-based model Covasim models the spread of COVID-19 by simulating a collection of agents representing people. Each agent is characterised by a set of demographic and disease properties:

- Demographics:
    - Age (one-year brackets)
    - Household size, and uniquely identified household members
    - Uniquely identified school contacts (for people aged 5-18)
    - Uniquely identified work contacts (for people aged 18-65)
    - Average number of daily community contacts (multiple settings / contact networks modelled, described below)
- Disease properties:
    - Infection status (susceptible, exposed, recovered or dead)
    - Whether they are infectious (no, yes)
    - Whether they are symptomatic (no, mild, severe, critical; with probability of being symptomatic increasing with age, and the probability of symptoms being more severe increasing with age)
    - Diagnostic status (untested vs tested)

Transmission is modelled to occur when a susceptible individual is in contact with an infectious individual through one of their contact networks. The probability of transmission per contact is calibrated to match the epidemic dynamics observed and is weighted according to whether the infectious individual has symptoms, and the type of contact (e.g. household contacts are more likely to result in transmission than community contacts). Transmission dynamics depend on the structure of these contact networks, which are randomly generated but statistically resemble the specific setting being modelled. The layers included are described below, and the model parameters values are provided for each layer that was included.

Model population

For this analysis a synthetic model population was initialized comprising of 100,000 people. The age and household size structure of the model population was based on the Australian population.

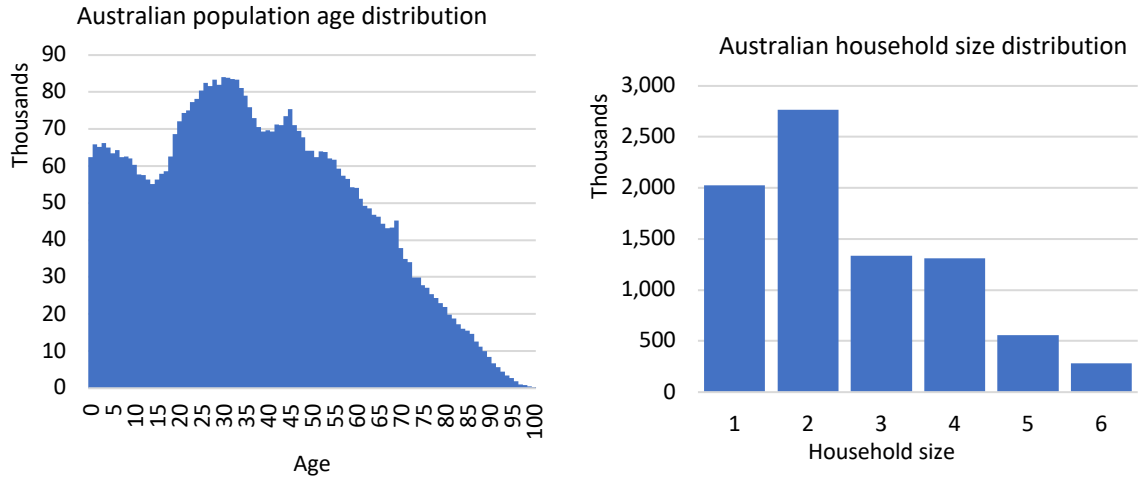


Figure 15: Population age structure and household size distribution [13].

Household contact network: household size and age structure

The household contact network was set up by explicitly modelling households. The households size distribution for Australia [13] was scaled to the number required for the number of agents in the simulation. Each person in the model was uniquely allocated to a household. To assign ages, a single person was selected from each household as an index, whose age was randomly sampled from the distribution of ages of the Household Reference Person Indicator in the 2016 Census [13]. The age of additional household members were then assigned according to Australian age-specific household contact estimates from Prem et al. [14], by drawing the age of the remaining members from a probability distribution based on the row corresponding to the age of the index member.

School contact networks

Schools and school contact networks were set up as described in the main report.

Work contact networks

Two different workplace types are included: public facing (e.g. retail, hospitality) and non-public facing. Contact networks for non-public facing workplaces were created as a collection of disjoint, completely connected clusters for the percentage of people aged 18-65 who worked in those settings. The mean size of each cluster was equal to the estimated average number of daily work contacts (Table S1). For the percentage of people aged 18-65 who worked in public facing workplaces, their workplace networks consisted of a completely connected cluster with other work colleagues, as well as each day having a number of random contacts with the community.

Additional contact networks

An arbitrary number of additional networks can be added. Each network layer requires inputs for: the proportion of the population who undertake these activities; the average number of contacts per day associated with these activities; the risk of transmission relative to a household contact (scaled to account for (in)frequency of some activities such as pubs/bars once per week); relevant age range; type of network structure (random, clustered, or specialized [as per schools/workplaces]); and effectiveness of quarantine and contact tracing interventions. Parameters for the networks currently in the model are in Tables S1 and S2.

Parameter values for each contact network

Tables S1 and S2 show the parameters that define each contact network in the model. Unless otherwise noted, parameters are derived in [2] from a mix of published and grey literature and a Delphi parameter estimation process. The columns refer to:

- **Network structure type:** Clustered refers to a network structure comprised of disjoint, completely connected groups of contacts. Random refers to individuals being allocated connections to anyone else in the network. Random networks are also dynamic and regenerated each day. Public facing networks are a combination of completely connected clusters for staff, who are then connected to random community members

- **Mean contacts:** The average number of contacts per person in each network. Each person in the model has their individual number of contacts draw at random from a Poisson distribution with these values as the mean. For the social network layer, a negative binomial distribution was used with dispersion parameter 2 to account for a longer tail to the distribution.
- **Mean public-public contacts:** For the percentage of people who participate in an activity, the average number of contacts they have with other members of the public (draw at random from a Poisson distribution with these values as the mean)
- **Mean public-staff contacts:** For the percentage of people who participate in an activity, the average number of contacts they have with staff (draw at random from a Poisson distribution with these values as the mean)
- **Relative transmission risk:** The transmission probability per contact is expressed relative to household contacts, and reflects the risk of transmission depending on behaviour. For example, a casual contact in a public park is less likely to result in a transmission event compared to a contact on public transport. Similarly, the relative transmission risks between staff-staff, public-public and staff-public are characterised for public-facing workplaces.
- **Quarantine effect:** If a person is quarantined, the transmission probability is reduced by this factor. For example, an individual on quarantine at home would likely not work or use public transport, but they may still maintain their household contacts.
- **Population proportion:** Each network will only include a subset of the population e.g. every person has a household, but not every person regularly uses public transport.
- **Age bound:** Each network will only include agents whose age is within this range.
- **Contact tracing probability:** Probability that each contact can be notified in order to quarantine
- **Effectiveness of quarantine and isolation:** When a close contact is asked to quarantine for 14 days, or a confirmed case asked to isolate while they are infected, these parameters represent he effectiveness of at reducing transmission through the specific networks. For example quarantine is assumed to have no impact on household transmission and greater impact on other contacts, reflecting compliance.

Table S1: Contact parameters for each of the networks in the model.

Contact network	Network structure type*	Mean contacts	Mean public-public contacts	Mean public-staff contacts	% of workforce	Relative transmission risk	Relative transmission risk (staff-staff)	Relative transmission risk (public-public)	Relative transmission risk (staff-public)	% of population	Age bound
House	Specialized	4				1.00					
School	Specialized										5-17
Non-retail work	Specialized	5			0.80	0.28					
Retail work	Public facing	5	8	2	0.11		0.28	0.04	0.04	0.70	12+
Community (general)	Random	1				0.10				1.00	
Places of worship	Clustered	20				0.04				0.11	
Community sport	Clustered	30				0.07				0.34	4-30
Entertainment	Public facing	25	8	2	0.02		0.28	0.01	0.01	0.30	15+
Cafe/restaurant	Public facing	5	8	2	0.02		0.28	0.04	0.04	0.60	12+
Pub/bar	Public facing	5	8	2	0.03		0.28	0.06	0.06	0.40	18+
Public transport	Random	25				0.16				0.11	15+
Public parks	Random	10				0.03				0.60	
Child care	Clustered	20				0.25#				0.55	1-6
Social	Random	6 (disp=2)				0.12				1.00	15+
Aged care	Clustered	12				0.58				0.07	65+

Table S2: Contact tracing parameters for each of the networks in the model.

Contact network	Assumed contact tracing probability	Assumed effectiveness of quarantine on network	Assumed effectiveness of isolation on network
House	1	0.00	0.80
School	0.95	0.99	0.99
Non-retail work	0.95	0.90	0.90
Retail work	0.95	0.90	0.90
Community (general)	0.1	0.80	0.80
Places of worship	0.5	0.99	0.99
Community sport	0.5	1.00	1.00
Entertainment	0.5	1.00	1.00
Cafe/restaurant	0.5	1.00	1.00
Pub/bar	0.5	1.00	1.00
Public transport	0.1	0.99	0.99
Public parks	0.1	1.00	1.00
Child care	0.95	0.99	0.99
Social	0.75	0.50	0.80
Aged care	0.95	0.80	0.80

Contact tracing: non-school contacts

Following detection of a positive case, the model initiates a contact tracing algorithm. *For cases detected in schools, this is described in the main report*. For cases in the community, the testing/contact tracing system was approximated as follows:

1. Day 0: Test is taken by index case
2. Day 1 (24 hours following test): Positive test results are returned, index case is notified and enters isolation.
3. Day 2 (48 hours following test being taken^): Contact tracing completed, with contacts having a setting-specific probability of being detected (Table S2), reflecting differences in the level of difficult in identifying contacts in that network (e.g. households vs public transport contacts). Identified contacts are tested and quarantined for 14 days regardless of test results, along with their entire households. Contacts are additionally tested on day 11 of quarantine, regardless of symptoms.
4. Day 3 (72 hours following test): Test results for contacts become available, and any contacts who returned a positive initial test would then have their contacts traced within the next 24 hours, in the same manner as the index case.

It was assumed that contact tracing deteriorated as case numbers increased. Caps on contact tracing assumed: at 0, 25, 75, 150 and 500+ cases per day, 100%, 80%, 50%, 30% or 20% of detected cases are subject to the above algorithm. The cap does not apply to household, school or childcare contacts who are assumed able to conduct their own tracing.

Virus strain

The model was based on transmission of the delta variant, with infectiousness calibrated to outcomes of the 2021 Victorian epidemic wave. The incubation period was shortened to a mean time from exposure to becoming infectious of 3.71 days, compared to 4.50 days for the wild type virus [15].

Vaccine properties

In the model, vaccination acts to reduce the probability of acquiring an infection when a contact occurs with an infectious case, as well as the probability of developing symptoms (both mild and severe) for people who are vaccinated and become infected. The assumed efficacy values used in this modelling are as per the main report.

The vaccine's prevention of infection is approximated as "leaky", meaning that each person vaccinated has reduced but non-zero risk of becoming infected based on the vaccine efficacy (as opposed to an "all or nothing" vaccine, where 80% efficacy means that 80% of people have perfect protection and 20% have no protection).

Model calibration

Model parameters for transmission and testing were calibrated to data on daily new detected cases, hospitalisations and ICU from the delta COVID-19 epidemic wave in Melbourne over the July-September 2021 period [5]. The model was initialised with a population of 100,000 agents, and the overall transmission risk per contact (which multiplies the transmission probabilities in Table S1 for each layer), the per-day probability of a symptomatic individual seeking testing were varied such that the distribution of model outcomes for diagnoses, hospitalizations and number of tests was centred near the actual epidemic trajectory. For additional details see [5].

For this analysis, the model was initialized with only a single case in a school, as described in the main report, however the transmission and testing parameters were based on this previous calibration.

Work Package 3: Review border measures & arrivals pathways in context of revised risk tolerance

Key question: How can arrivals caps and pathways be safely modified in the context of the changing risk environment as population vaccine coverage increases?

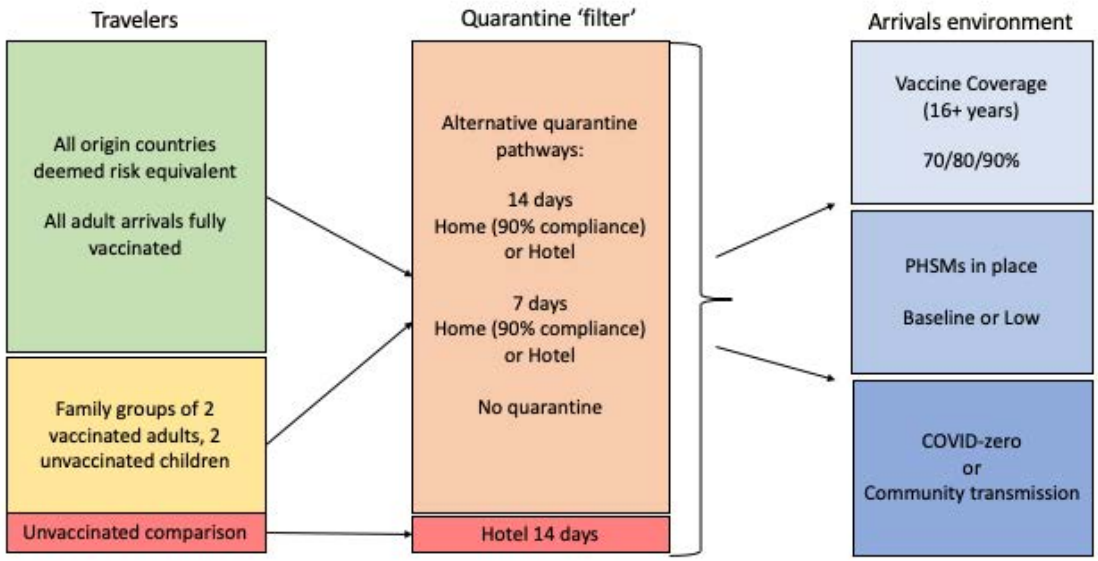
Overview: This work package extends on earlier models of quarantine and importation risk assessment to consider alternative arrivals arrangements for vaccinated individuals including family quarantine, and the impact of increased arrivals caps on outbreak risk during Phases B and C of the National Plan.

The purpose of this report is to:

1. Review the quarantine pathways risk assessment using our updated parameters for vaccine effectiveness against the Delta strain, including assessment of family pathways, reduced quarantine durations and alternative testing regimens;
2. Demonstrate the relative infection risk associated with hypothesised arrivals scenarios developed in consultation with PM&C, Home Affairs and Treasury, based on pre-COVID-19 travel volumes for Australian citizens and permanent residents into a large and medium jurisdiction;
3. Demonstrate the local epidemiological impact associated with these arrivals scenarios for jurisdictions where COVID-19 transmission is established (endemic cases) or absent (COVID-zero) for different vaccine coverage levels and application of public health and social measures (PHSMs).

Figure 1 shows an overview of the risk assessment pathway by which the influence of arrivals on local epidemiology will be characterised in this reporting phase. In the first instance, we will assume uniform risk of exposure in the country of origin for travellers from the majority of destinations, deemed 'green'. We will consider the relative effectiveness against importations of alternative quarantine approaches, applied to fully vaccinated travellers and partially vaccinated families. Consequences of importations are determined by three characteristics of the arrivals environment: vaccine coverage, the level of intensity of public health and social measures (PHSMs) and the level of pre-existing transmission of COVID. 'Partial' or 'optimal' test-trace-isolate-quarantine (TTIQ) responses are assumed to be ongoing.

Figure 1: Overview of the risk assessment pathway



1. Updated quarantine pathways risk assessment

In previous work we defined the *force of infection* associated with quarantine breach events as the number of days an infectious individual is in the community adjusted for their relative infectiousness. It can be interpreted as the expected number of secondary cases produced per infected arrival through a given quarantine pathway in a *fully susceptible* community. It exceeds one for an unvaccinated infected individual without quarantine or testing. This metric also allows comparison of the relative risks posed by vaccinated and unvaccinated travellers, given that we assume vaccinated breakthrough infections are less infectious per unit time.

Hotel quarantine is modelled as previously, with a 14-day duration as the benchmark, for comparison with a 7-day stay. Compliance in this system is assumed to be 100% due to oversight. Testing is an important risk mitigation measure for both travellers and workers, the former according to a fixed schedule (days 1, 5 and 13 for 14-day duration; days 1 and 5 for 7-day duration), the latter corresponding to their days of work. Confirmed cases are removed to an isolation facility for 10 days, and the quarantine duration of their travelling party contacts is extended by 14 days in situ. All measures are implemented in accordance with the national minimum standard, which may be exceeded by some jurisdictions to further reduce risks.

Home quarantine is modelled as previously, with a 14-day duration as the benchmark, for comparison with a 7-day stay. The household unit is assumed to be bound by the restrictions imposed for the same duration as the arriving traveller, whether they have returned together from overseas or represent the home-based contacts of a returning single traveller. Testing is conducted according to the same schedule as hotel quarantine. We consider a range of compliance levels:

- 100% compliance with social restrictions, allowing quantification of the additional risks of transmission within the hotel quarantine system to other travellers (and workers);
- 90% compliance with social restrictions, deemed reasonably achievable by jurisdictions who have offered this arrivals pathway to exempt travellers;
- 75% compliance with social restrictions.

In addition, this report includes evaluation of 2 or 3-day home quarantine durations, with rapid antigen testing (RAT) on days 0,1 or 0,1,2 respectively.

No quarantine is modelled as previously, with no constraints placed on arrivals. Polymerase Chain Reaction (PCR) testing is the only substantive risk mitigation measure, with tests required on days 1 and 5. Testing is an important risk mitigation for arrivals through the 'no quarantine' pathway, reducing the force of infection by about four times compared with the 'no testing' option, which is now also shown in the pathway for reference.

Our updated calculations include assessment of the risks posed by family groups composed of adults and children, understanding that children less than 12 years are currently ineligible for vaccination. Vaccination reduces infectiousness and hence the risk posed by quarantine breach events from immunized travellers. In this way it mitigates against the observed increase in community exposure days resulting from shorter duration stays. Given the anticipated intensity of transmission pressure within the confined family group, we assume all members are equally infectious and susceptible in the quarantine environment.

Updated estimates for the force of infection per infected arrival are shown in Tables 1.1 and 1.2 for adult arrivals and families, respectively, by vaccination status, quarantine pathways and testing regimens.

Table 1.1: Force of infection contributed by an infected adult arriving in a group of four travellers, by vaccination status, quarantine pathway and testing regimen

Pathway	Duration (days)*	Vaccinated	Compliance (%)	Force of infection per infected arrival	Force of infection relative to baseline
Hotel	**14**	**No**	**100**	**0.042**	**1**
		Yes		0.013	0.31
	7	No		0.17	4.05
		Yes		0.081	1.93
Home	14	No	100	0.019	0.45
			90	0.123	2.93
			75	0.283	6.74
		Yes	100	0.008	0.19
			90	0.076	1.81
			75	0.167	3.98
	7	No	100	0.119	2.83
			90	0.219	5.21
			75	0.371	8.83
		Yes	100	0.07	1.67
			90	0.133	3.17
			75	0.225	5.36
Home (daily RAT)[#]	3	Yes	100	0.597	14.21
			90	0.641	15.26
			75	0.695	16.55
	2	Yes	100	1.207	28.74
			90	1.236	29.43
			75	1.282	30.52
No quarantine	0	No	NA	1.121	26.69
		Yes		0.69	16.43
No quarantine or testing	0	No	NA	4.85	115.48
		Yes		2.807	66.83

**For quarantine durations of 14 days, arrivals are PCR tested on days 1, 5 and 13; for quarantine durations of 7 or 0 days, arrivals are PCR tested on days 1,5.*

[#]For 2-3 day home quarantine options, individuals are rapid antigen tested (RAT) on days 0,1 (2 day stay) OR days 0,1,2 (3 day stay)

Table 1.2: As for Table 1.1 but for family group arrivals comprising 2 vaccinated adults and 2 unvaccinated children aged <12 years

Pathway	Duration (days)*	Vaccinated	Compliance (%)	Force of infection per infected arrival	Force of infection relative to baseline
Hotel	14	No	100	0.048	1.14
		Yes		0.035	0.83
	7	Yes		0.125	2.98
Home	14	Yes	100	0.024	0.57
			90	0.086	2.05
			75	0.181	4.31
	7	Yes	100	0.114	2.71
			90	0.175	4.17
			75	0.267	6.36
No quarantine	0	Yes	NA	0.73	17.38
No quarantine or testing	0	Yes	NA	2.89	68.81

**For quarantine durations of 14 days, arrivals are PCR tested on days 1, 5 and 13; for quarantine durations of 7 or 0 days, arrivals are PCR tested on days 1, 5*

Key differences for family groups are that children are not protected by vaccination but if they do contribute a quarantine breach are assumed to be intrinsically less infectious in the community than adults. In addition, if a child is identified as infected in quarantine they will be isolated with a parent and not alone, so there is an ongoing risk of infection transmission within the isolation facility/medi-hotel that would not apply to adult travellers.

Figure 2: Force of infection per infected arrival in home quarantine, for unvaccinated arrivals, family units containing vaccinated parents and unvaccinated children, and unvaccinated arrivals. Results are shown by duration of stay (14 or 7 days) and compliance with quarantine (100%, 90% or 75%)

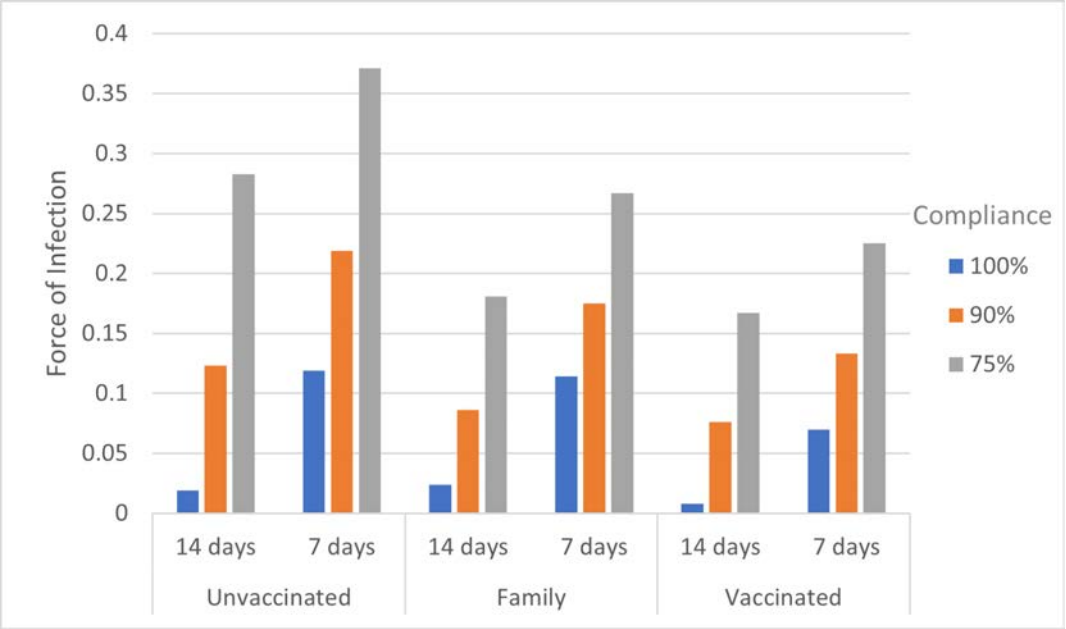


Figure 2 demonstrates the drivers of risk associated with quarantine breaches. Longer quarantine stays reduce breach risk. Lower compliance with quarantine requirements is more influential at increasing risk for a given length of stay. And vaccination reduces risks across the board. Families contribute an overall risk that is between fully vaccinated and unvaccinated arrivals.

2. Aggregate force of infection associated with hypothesised arrivals scenarios

Importation risks associated with alternative arrival scenarios can be readily calculated by grouping different volumes of arrivals into their allocated quarantine pathways. Given uncertainty about the true risks of infection across all potential countries of origin, we have assumed 1% of travellers are exposed to infection but remain undetected on embarkation. Vaccination reduces the likelihood that 'exposed' travellers will arrive infected by 80% (ie to 0.2%). This percentage is used to calculate the absolute number of infected arrivals entering the quarantine system for given traveller volumes. The quarantine pathways through which they are processed will determine the aggregate weekly force of infection imposed on the arrival jurisdiction.

We have devised arrivals scenarios in consultation with PM&C, Home Affairs and Treasury that allow us to calculate the aggregate weekly force of infection for different numbers of vaccinated adult and family group arrivals 'filtered' through alternative quarantine pathways. The total number of arrivals is calculated as a proportion of 2019 Australian citizen and permanent resident traveller volumes to inform scenarios representative of large and medium sized jurisdictions. Based on the threshold age for vaccine eligibility, we use numbers of travellers up to the age of 12 years from these data to allocate 'family groups' incorporating a corresponding proportion of adults in units of size four (two vaccinated parents, two unvaccinated children).

Scenario 1 – Endemic cases

Table 2.1: Calculated force of infection resulting from breaches in the quarantine system, assuming weekly arrivals of 65,534 or 32,767*, with 17.9% of travellers arriving in family units. The first five scenarios assume that all adult arrivals are fully vaccinated and compare different lengths of stay in home or hotel quarantine. 90% compliance is assumed for all home quarantine pathways. Regular PCR testing increases infection ascertainment on days 1, 5 and 13 for 14 day stays, and days 1 and 5 for 7 day stays and 'no quarantine'. Forces of infection through these pathways are compared with the previous policy of 14 days' hotel quarantine for unvaccinated travellers.

		14d home	14d hotel	7d home	7d hotel	None	Unvaccinated
80% **65,534 weekly arrivals**	**Adult FOI**	8.17	1.40	14.31	8.71	70.24	22.59
	Family FOI	6.06	2.47	12.34	8.81	45.55	5.64
	Total FOI	**14.24**	**3.87**	**26.65**	**17.53**	**115.79**	**28.23**
40% **32,767 weekly arrivals**	**Adult FOI**	4.09	0.70	7.15	4.36	35.12	11.29
	Family FOI	3.03	1.23	6.17	4.41	22.78	2.82
	Total FOI	**7.12**	**1.93**	**13.32**	**8.76**	**57.90**	**14.11**

**Arrivals figures represent 80% and 40%, respectively, of 2019 Australian citizen/Permanent resident arrivals into NSW in 2019*

All pathways other than 'no quarantine' (None) are associated with a lower aggregate force of infection than 14 day hotel quarantine for unvaccinated arrivals, which was the requirement prior to the era of vaccination. It should be noted that incursion risks in the absence of a quarantine stay are mitigated by testing on days 1 and 5 and are four times higher if no tests are performed. Doubling the number of arrivals from 40% to 80% doubles the force of infection per unit time, *noting that this aggregate force of infection estimate applies to an unvaccinated population.*

Scenario 2 – 'COVID-zero'

Table 2.2: As above, but assuming weekly arrivals of 20,726 or 10,363*, with 18.2% of travellers arriving in family units.

		14d home	14d hotel	7d home	7d hotel	None	Unvaccinated
80% **20,726 weekly arrivals**	**Adult FOI**	2.58	0.44	4.51	2.75	22.14	7.12
	Family FOI	1.95	0.79	3.96	2.83	14.61	1.81
	Total FOI	**4.52**	**1.23**	**8.47**	**5.57**	**36.76**	**8.93**
40% **10,363 weekly arrivals**	**Adult FOI**	1.29	0.22	2.26	1.37	11.07	3.56
	Family FOI	0.97	0.40	1.98	1.41	7.31	0.90
	Total FOI	**2.26**	**0.62**	**4.23**	**2.79**	**18.38**	**4.47**

**Arrivals figures represent 80% and 40%, respectively, of 2019 Australian citizen/Permanent resident arrivals into WA in 2019*

Consistent with findings above, reduced traveller volumes for this example jurisdiction are associated with a lower overall risk of infection importation.

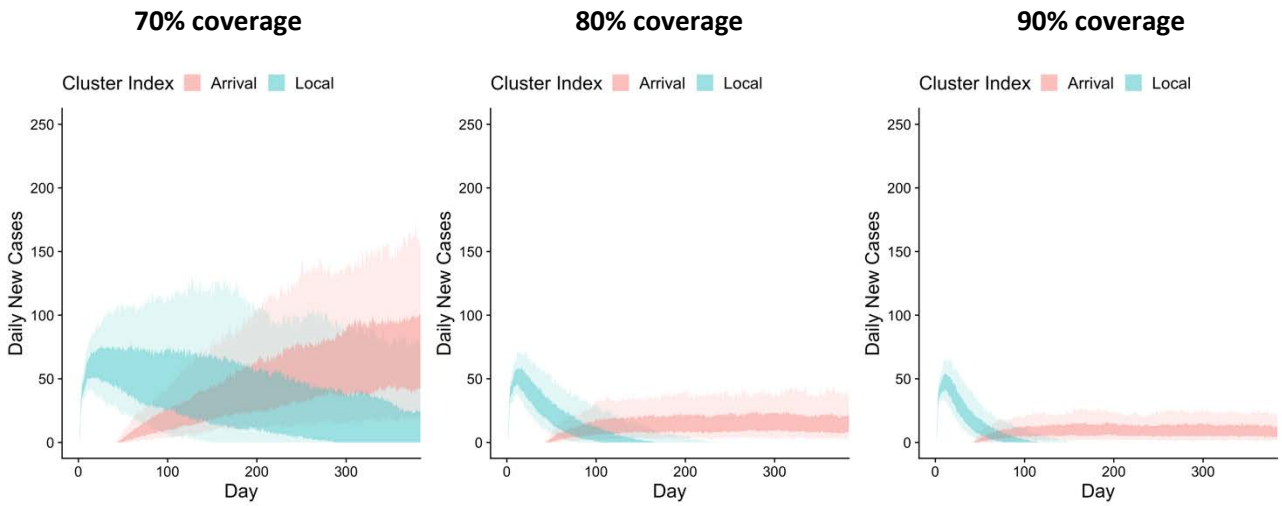
3. Consequences of importations for local epidemiology

Scenario 1 – Endemic cases

Epidemiological consequences of the arrivals scenarios above are demonstrated in Figures 3.1.1, 3.1.2 and 3.1.3. The 'vaccine coverage' (16+ years) in these simulations is fixed at the beginning of the simulations, with no ongoing vaccine rollout assumed. We include additional coverage of the 12-15 years age group at the time of achieving the threshold, based on estimates provided by the Quantium team in Health. 200 'local' infections are seeded on day 0 to establish a local epidemic, with travellers beginning to arrive on simulation day 40.

At 70% vaccine coverage, ongoing transmission of local strains occurs and is gradually superseded by new infections resulting from imported strains. For 80% and 90% coverage, locally transmitted strains become extinct at around 100 days. Ongoing importation of strains is a continuous source of newly seeded infections, but transmission is sufficiently constrained by vaccination that large outbreaks do not occur.

Figure 3.1.1: Impact of incursions on endemic cases given differing vaccine coverage in the arrivals environment. Partial TTIQ and ongoing 'low' PHSMs are additional constraints on transmission. Travellers (vaccinated adults and families) are managed through a 7 day home quarantine pathway, with 90% compliance and PCR testing on days 1 and 5. Traveller volumes are 40% of 2019 citizen/PR values.



Shaded areas denote uncertainty across multiple simulations. Teal shading reflects new cases resulting from local strains present at the beginning of the simulation. Salmon/pink shading denotes cases resulting from transmission chains seeded by importations.

Figure 3.1.2: As for Figure 3.1.1 but comparing 40% (left) and 80% (right) of 2019 arrivals, 80% coverage

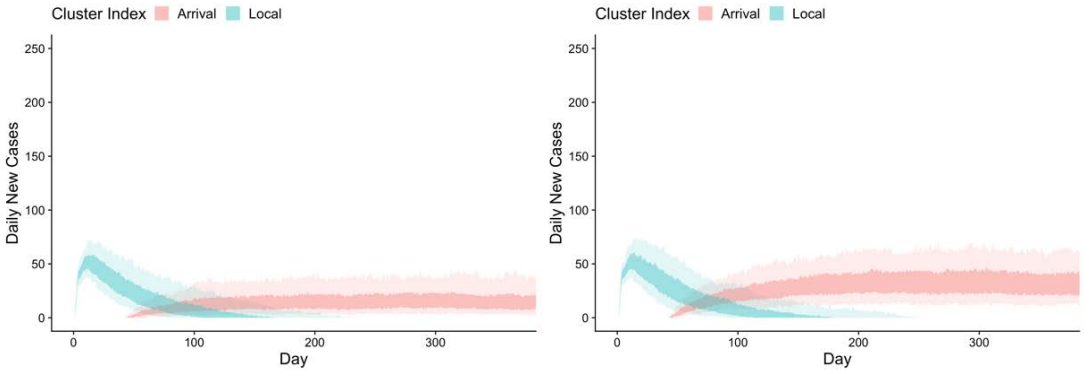


Figure 3.1.3: As for Figure 3.1.1, with 80% vaccine coverage but for different quarantine requirements

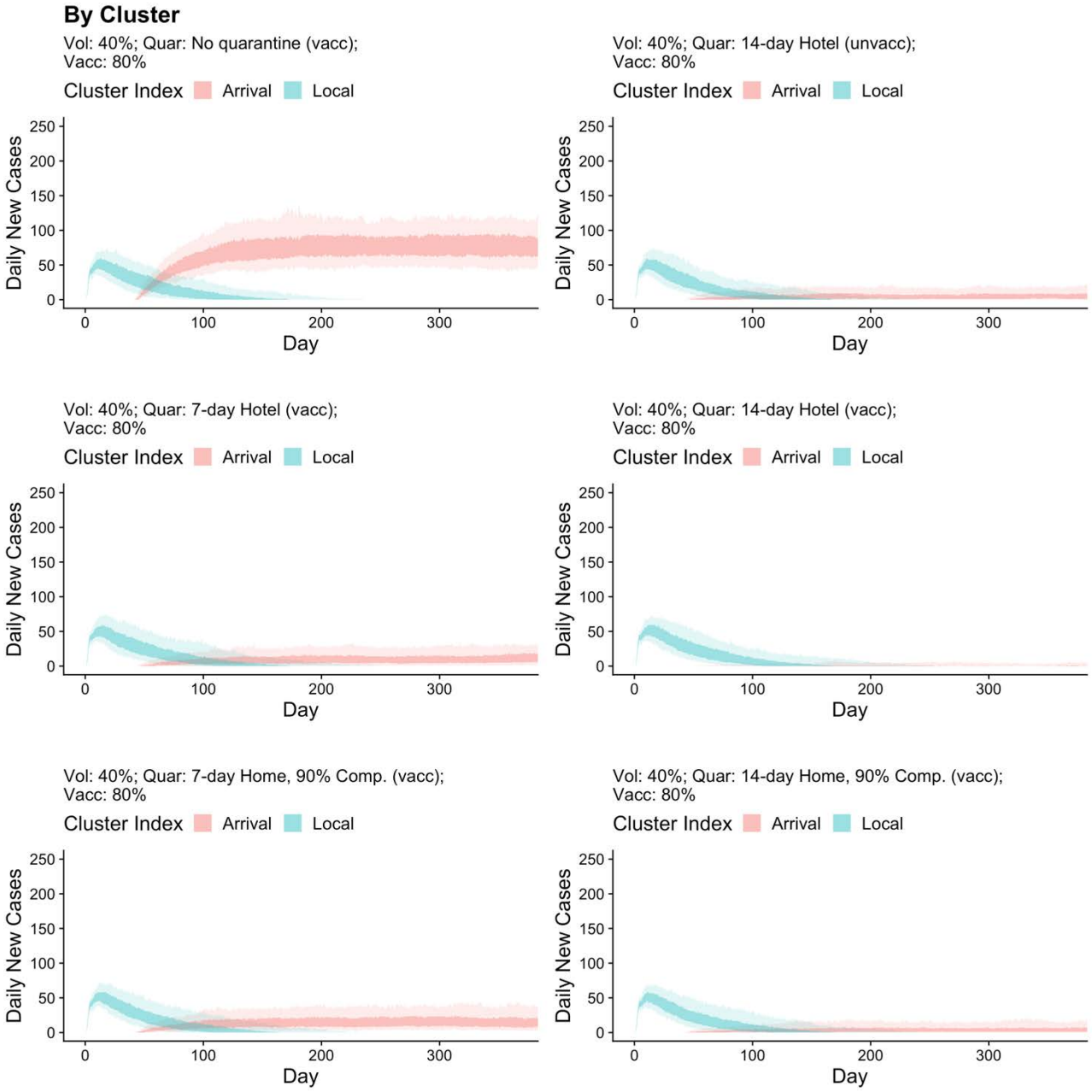


Figure 3.1.2 shows an approximate doubling of set point infection prevalence as traveller volumes increase. Figure 3.1.3 demonstrates that the 'no quarantine' pathway is associated with approximately a three to four fold increase in daily incident infections resulting from importations. This difference is explained by the higher aggregate force of infection associated with this pathway in Table 2.1. However, as in all simulations with vaccine coverage of 80% or more transmission is strongly constrained, preventing explosive outbreaks.

The importance of controls in place in the arrivals environment is demonstrated (Figure 3.2.1) by an additional scenario for endemic cases considering the impact of partial TTIQ with only *baseline PHSMs* in place, for all the same arrivals considerations as above (Figures 3.2.1, 3.2.2 and 3.2.3). Note the marked difference in axes between these two sets of figures. **At 80% coverage, thousands of incident cases are expected daily with only baseline PHSMs in place, compared with fewer than 100 when ongoing low PHSMs are maintained.**

Such rapidly escalating infections are driven by 'local' cases which far exceed the rate of importation. Incursions do not materially impact on the established local epidemic. This scenario is demonstrative only, as an outbreak of the size shown for the 70% and 80% coverage examples would require imposition of additional measures to reduce disease burden and impacts on the health system and society.

Figure 3.2.1: As for Figure 3.1.1 but assuming Partial TTIQ and 'baseline' PHSMs in place

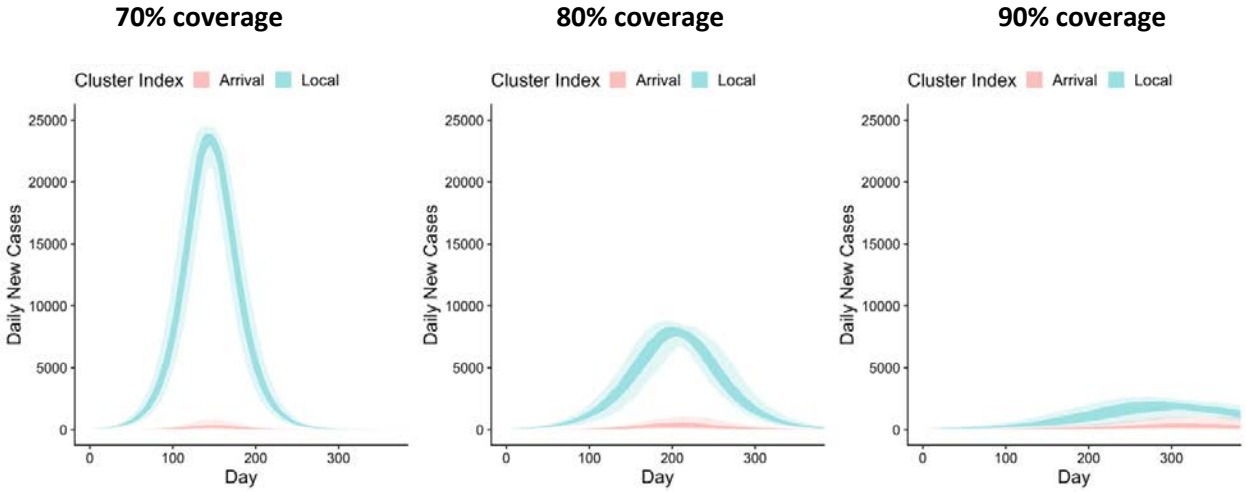


Figure 3.2.2: As for Figure 3.2.1 but comparing 40% (left) and 80% (right) of 2019 arrivals, 80% coverage

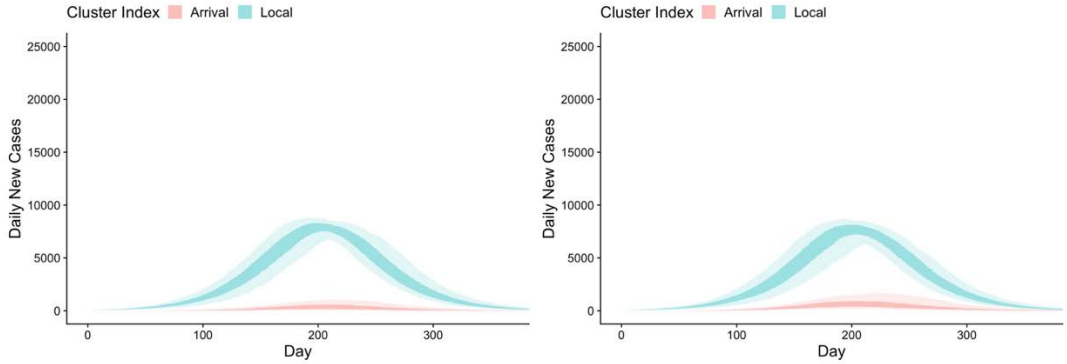
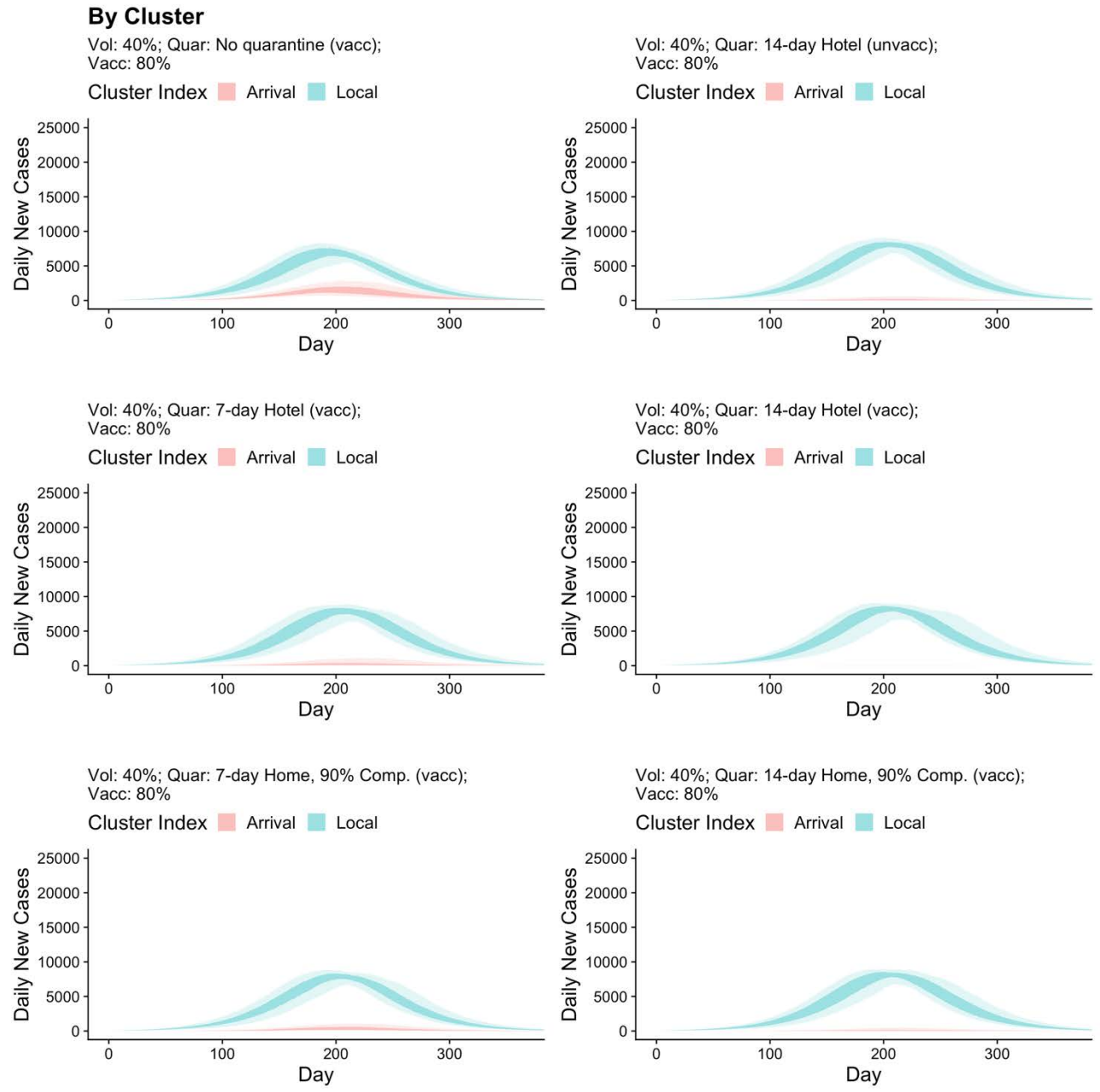


Figure 3.2.3: As for Figure 3.2.1, with 80% vaccine coverage but for different quarantine requirements



Scenario 2 – 'COVID-zero'

The simulations in Figures 3.3.1, 3.3.2 and 3.3.2 share most of the same assumptions as previously but with *optimal TTIQ and baseline PHSMs* in place in a 'COVID-zero' jurisdiction. These differences account for the enhanced epidemic growth most apparent in the 70% coverage case, noting that the y axes in these figures are in the 1,000s compared with the first Scenario 1 example (maximum 125).

The seeded epidemics grow slowly initially because the transmission potential is just above one but escalate within a few months at 70% coverage. At 80% or higher coverage epidemic growth is slower as further constrained. Because all infections are seeded by 'arrival' strains only one colour is shown on the plots, but in reality it is implausible that only internationally seeded infections would circulate over the one year time frame of the simulations.

Figure 3.3.1: As for Figure 3.1.1 but for 'COVID-zero', and assuming optimal TTIQ and 'baseline' PHSMs.

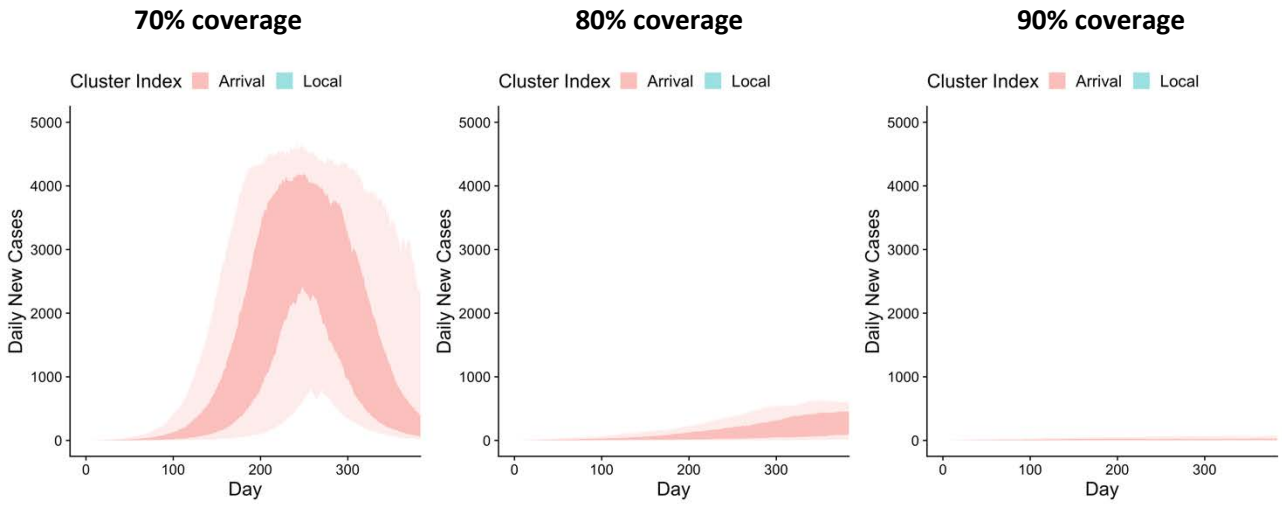
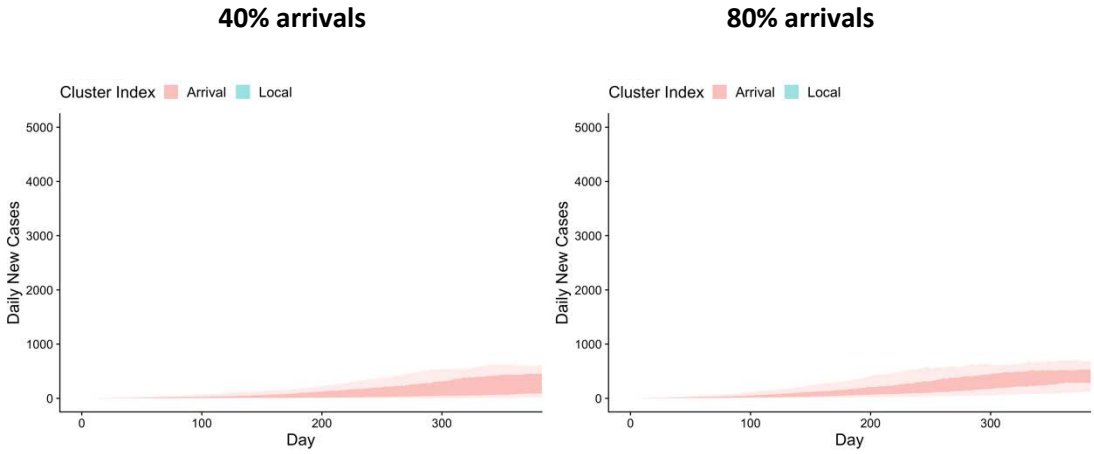
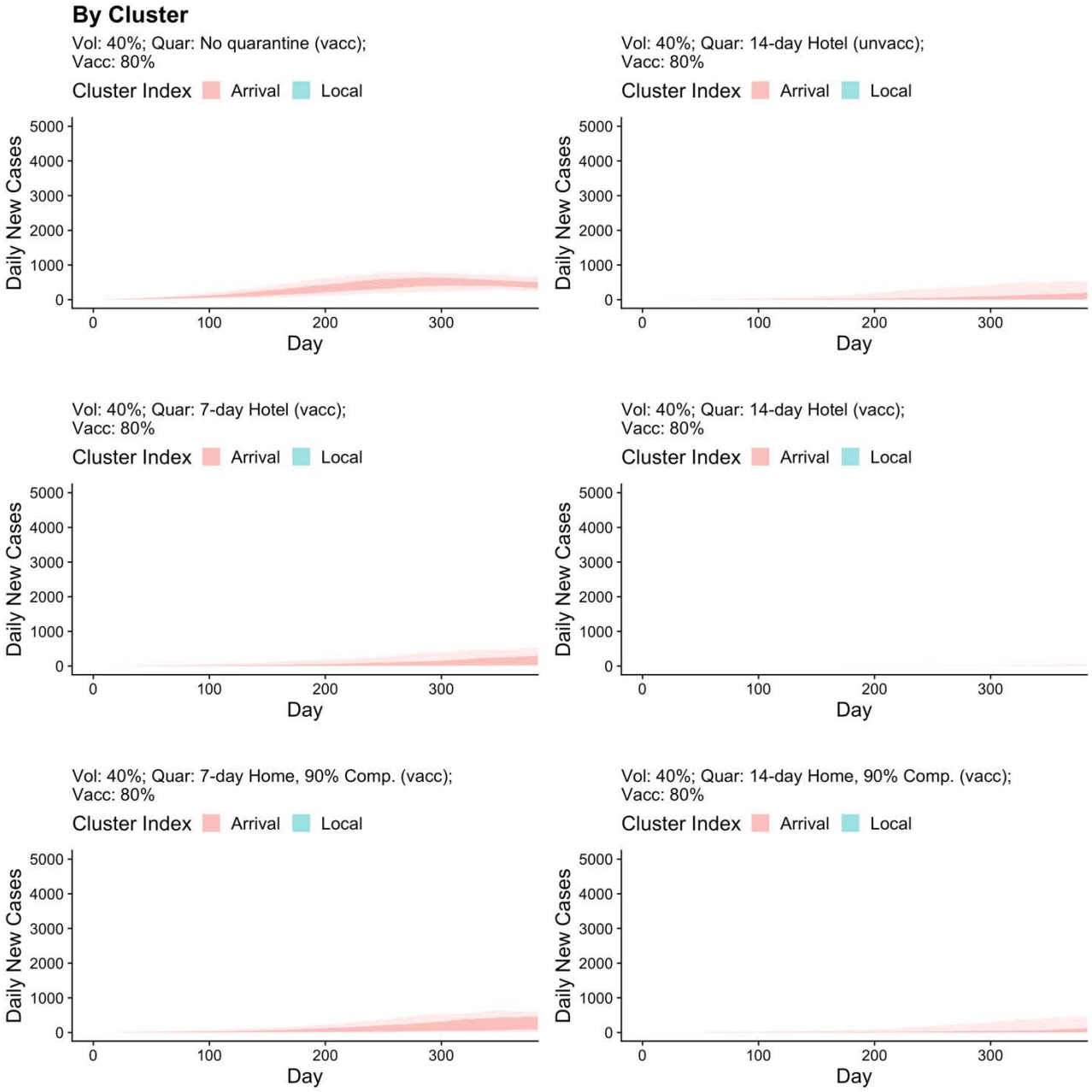


Figure 3.3.2: As for Figure 3.3.1 with 80% vaccine coverage, comparing 40% and 80% of 2019 arrivals



Doubling the number of arrivals at 80% coverage results in a modest increase in the number of infections anticipated on a given day in this scenario (Figure 3.2.2) but less than the difference with 'no quarantine' for 40% of arrivals (Figure 3.2.3). In all cases, the timing of epidemic growth is not materially different and case numbers escalate over several months, allowing time for situational assessment.

Figure 3.3.3: As for Figure 3.3.1 with 80% vaccine coverage, but for different quarantine requirements



Note that all of these simulations assume consistent vaccine protection over time (ie immunity does not wane) and that the characteristics of imported strains are identical to those initially present in the population (ie they are not more transmissible and are equally preventable by vaccination).

Attachment F: Influential revisions to parameter assumptions used in Doherty Modelling

Summary

Between the previous and current phases of our modelling work we have extensively reviewed available evidence regarding age-dependent mixing and susceptibility to the Delta variant, vaccine uptake, and vaccine effectiveness assumptions against acquisition, infectiousness and disease outcomes.

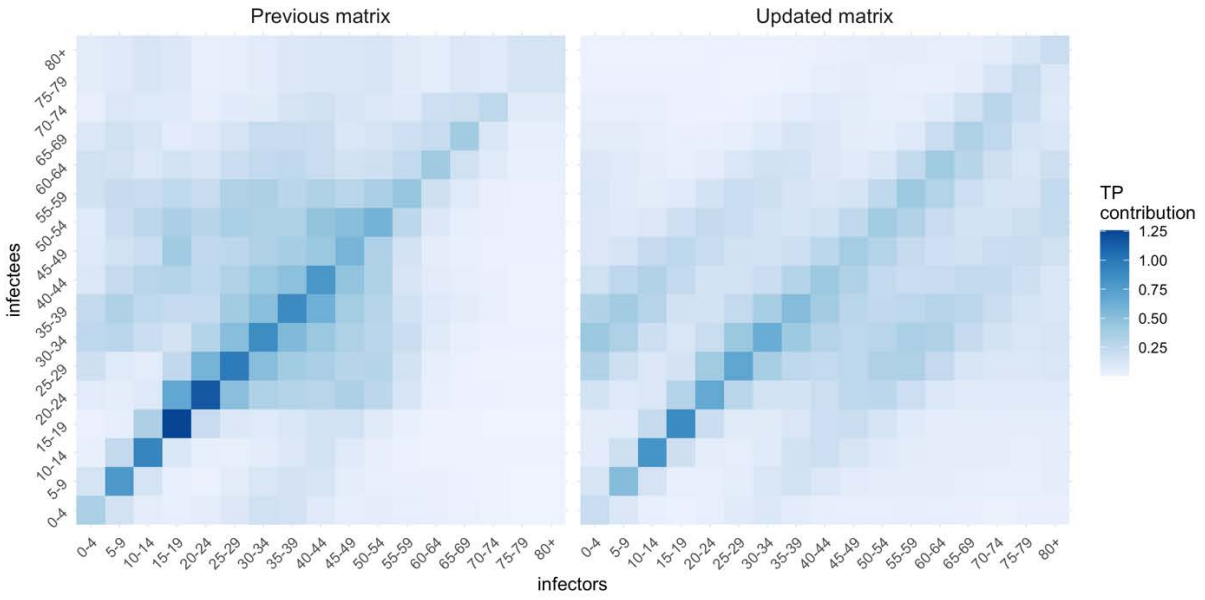
While values of individual parameters vary between phases of our work, we have assessed the consequences of these changes in aggregate and confirm that our previous recommendations of vaccine coverage thresholds of 70% and 80% for national plan transition phases remain robust.

Social Mixing Assumptions

In the first phase of our National Plan modelling, we developed an age-structured transmission matrix characterising infection spread within and between age groups based on population mixing assumptions using widely accepted social contact matrices published by Prem et al [1]. The matrix (left panel, Figure 1) was extended to include an 80+years cohort and weighted using age-specific susceptibility and transmissibility estimates from Davies et al [2].

For this phase of work we have updated the social mixing assumptions from the Prem paper to align more closely with reported observations in the Australian context. In this process we have identified errors in the original work by Prem, including an apparent overestimation of workplace contacts in Australia. The relative probability of transmission between household and non-household contact settings was also re-estimated and included in the transmission matrix resulting in an upweighting of household contacts.

Figure 1: Age based transmission matrices used in previous work (left) based on assumptions of the Prem [1] and Davies [2] papers, and updated (right) to incorporate emerging evidence on age-based mixing (Australia) and the relative susceptibility of individuals aged <16 years (England)



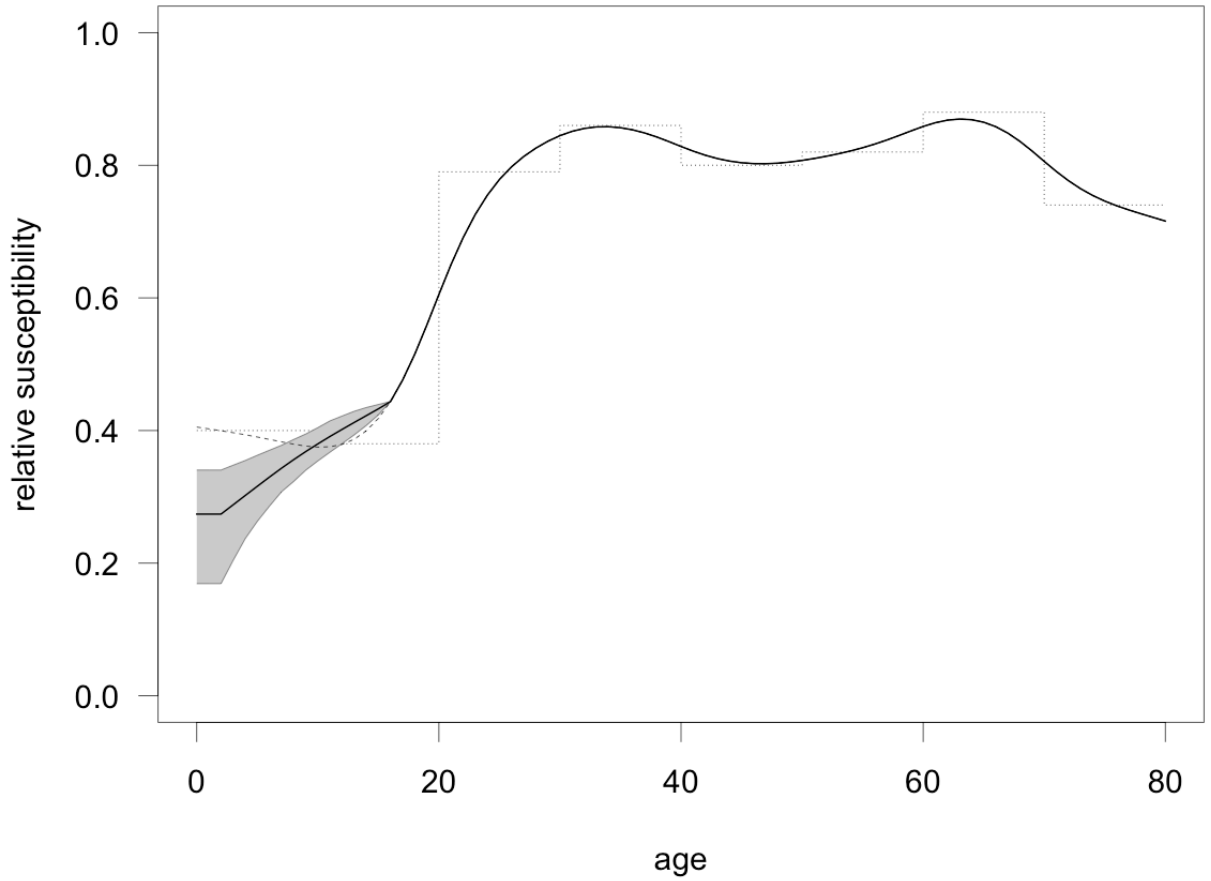
We have re-estimated transmission parameters to fit infection age distributions from the UK post-reopening and with full school attendance since the beginning of September. There has been very limited vaccination of the 12-15 years cohort in the UK, with current first dose coverage approximately 15%

compared with ~60% in Australia. The UK also has a nationwide infection survey that randomly screens 150,000 people each fortnight (approximately -0.2% of the population).

Given biases in acquisition of childhood infections due to the low symptomatic fraction, data on the age distribution of infections among under-16s in the UK is probably the best source of information on the relative susceptibility/infectiousness of the 5-11 cohort versus the 12-15 cohort, and therefore of the likely effectiveness of our 12+ (and hopefully imminent 5+) vaccination program on transmission with minimal restrictions. After a delay since schools reopened, prevalence in the 12-15 cohort in England has increased markedly to more than 8%, while in the 2-11 years group it has only increased from 2% to 3%[1].

Figure 2 compares previous and revised estimates of relative susceptibility by age, based on these most recent observations.

Figure 2: Relative susceptibility by age. New mean estimates are shown by the black line (grey region reflects 95% CIs), with previous estimates represented by dotted/dashed lines for comparison.



As shown in the right panel of Figure 1, the net consequence of this reanalysis has been an overall reduction in the proportional contribution of children aged 5-11 years to transmission, and some increase in attribution to individuals aged 16-24 years.

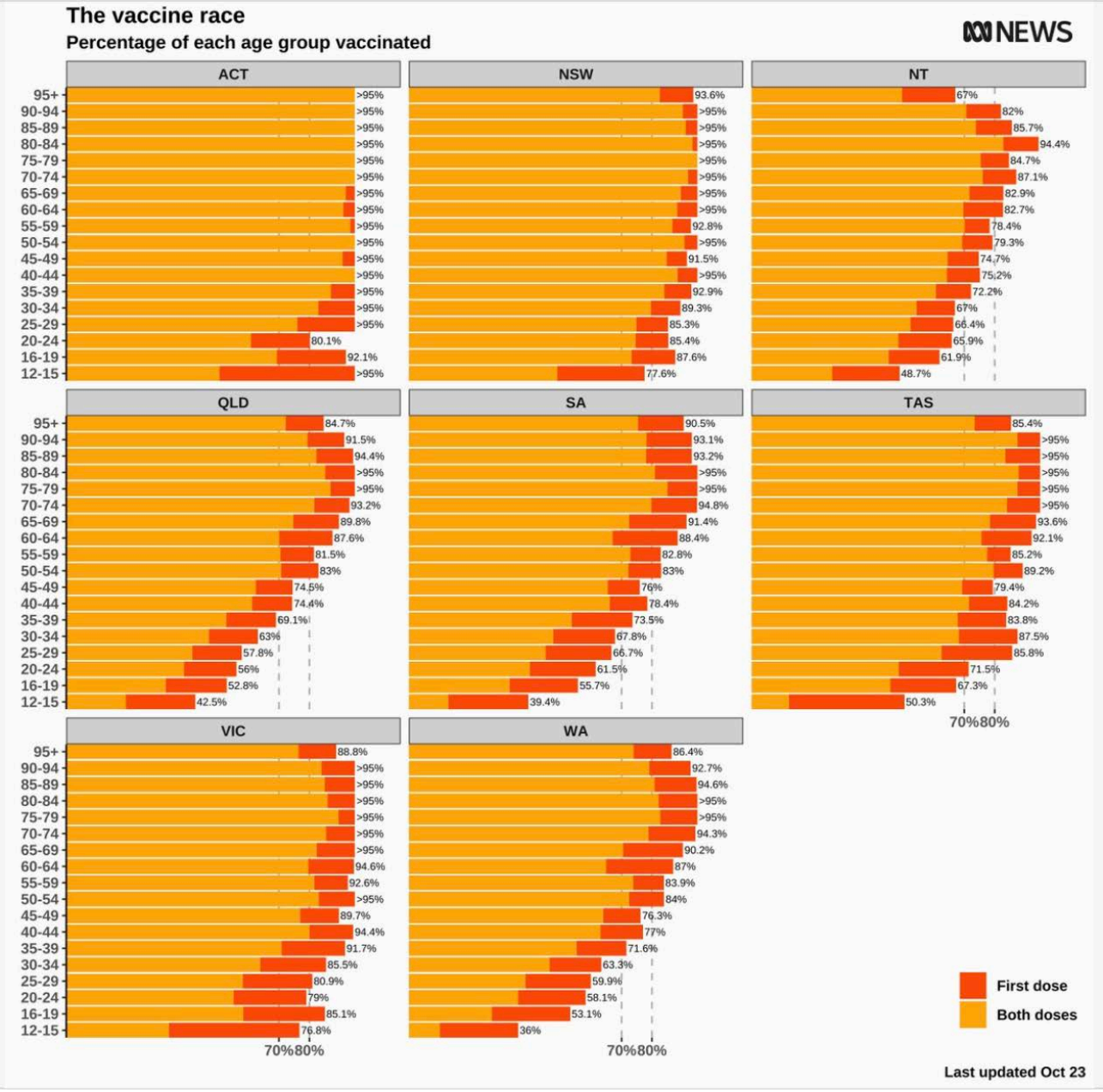
The new model enables extrapolation to any population within Australia. The main consequence of this change has been a more optimistic expectation of overall vaccine impact on transmission potential (TP) in populations with a high proportion of children than previously anticipated (countered in some populations by large household size), and a boost in TP reduction associated with vaccination of the 16-24 years group.

[1] https://www.ons.gov.uk/peoplepopulationandcommunity/healthandsocialcare/conditionsanddiseases/bulletins/coronaviruscovid19infectionsurveypilot/15october2021#age-analysis-of-the-number-of-people-who-had-covid-19.

Vaccine coverage assumptions

Our initial coverage scenarios considered optimal age-based vaccine distribution strategies to minimise transmission and disease. The Quantium team in Health advise that the actual rollout in the Australian population has most closely approximated the 'all ages' strategy, which resulted in high uptake in the peak transmitting age groups identified above, maximising population wide benefits of the program. Extension of vaccine eligibility to the 12+ years group has further increased whole of population coverage (Figure 3). In addition, the pace of rollout has exceeded expectations, particularly in states with community transmission, enabling threshold targets of 70 and 80% to be reached earlier in some states than the dates anticipated in our earlier work, which were 1st and 22nd November respectively.

Figure 3: Visualisation of one and two dose vaccine coverage by age and state, as of 23 October 2021 *(source: https://twitter.com/CaseyBriggs/status/1451771648412045315)*



Of note, it is anticipated that 'final' vaccine coverage in the order of 90% will be achieved within weeks of the 80% target, which is much faster than in the original simulations provided by Quantium. Should these expectations be realised, we anticipate greater constraint of transmission in the initial weeks following the transition to Phase C than was estimated by our model.

Vaccine effectiveness assumptions

We have updated our assumptions of vaccine effectiveness (VE) against infection and onwards transmission, based on new evidence from the UK specific to the Delta variant. On balance, these changes have resulted in some reduction in overall effectiveness of the Astra Zeneca vaccine, but none for Pfizer which has been the predominant vaccine delivered through the program.

Table 1A: Vaccine effectiveness estimates (%) against overall (asymptomatic and symptomatic) infection of SARS-CoV-2 Delta variant based on **Shiek et al 2021** [3] (as per ATAGI July 2021 advice document).

Vaccine	**Dose 1***			**Dose 2†**		
	Lower limit	**Point estimate**	Upper limit	Lower limit	**Point estimate**	Upper limit
AstraZeneca	9	**18**	25	53	**60**	66
Pfizer BNT	17	**30**	41	75	**79**	82

**estimates in study for ≥28days post dose 1 and pre dose 2*

†estimates in study for ≥14days post dose 2

Table 1B: Vaccine effectiveness estimates (%) against overall (asymptomatic and symptomatic) infection of SARS-CoV-2 Delta variant based on **Pouwels et al 2021** [4].

Vaccine	**Dose 1***			**Dose 2†**		
	Lower limit	**Point estimate**	Upper limit	Lower limit	**Point estimate**	Upper limit
AstraZeneca	35	**46**	55	62	**67**	71
Pfizer BNT	50	**57**	63	77	**80**	83

**estimates in study for ≥21days post dose 1 and pre dose 2*

†estimates in study for ≥14days post dose 2

Pouwels et al's second dose estimates for the Delta variant broadly agree with Shiek et al's estimates. However, Pouwels et al's estimates are less likely to be biased by differential test-seeking behaviour according to vaccination status. They used data from the Office for National Statistics COVID-19 Infection Survey, a large community-based survey of individuals living in randomly selected households across the UK, where testing was performed according to a pre-determined schedule, irrespective of symptoms, vaccination status or prior infection.

Note that Eyre et al [6] also provide delta-specific estimates of VE against acquisition but caution against using these as overall estimates of VE since the study mostly captured symptomatic infections. Thus, the reduction in infection of vaccinated contacts in the study cannot account for the increased chance of asymptomatic infection in the vaccinated contacts (who are less likely to be detected based on the study design).

ACTION TAKEN: for acquisition VE parameters use values in Table 1B rather than Table 1A.

Table 2A: Vaccine effectiveness estimates (%) reasonable to use as against onward transmission to **household members (i.e., 100% household contacts)** in case of breakthrough infections in vaccine recipients for the **Alpha variant** based on **Harris et al 2021** [5] (as per ATAGI 2021 advice document).

Vaccine	**Dose 1**			**Dose 2**
	Lower limit	**Point estimate**	Upper limit	**Point estimate**
AstraZeneca	38	**48**	57	**65***
Pfizer BNT	38	**46**	53	**65***

**These estimates are an ATAGI expert view 3 May and 7 July 2021.*

Table 2B: Vaccine effectiveness estimates (%) against onward transmission to **contacts (70% household contacts)** in case of breakthrough infections in vaccine recipients for the **Alpha variant** based on **Eyre et al 2021** [6].

Vaccine	**Dose 1***			**Dose 2†**		
	Lower limit	**Point estimate**	Upper limit	Lower limit	**Point estimate**	Upper limit
AstraZeneca	12	**18**	24	37	**63**	78
Pfizer BNT	20	**26**	30	71	**82**	88

†estimates in study for ≥14 days post dose 2

Table 2C: Vaccine effectiveness estimates (%) against onward transmission to **contacts (70% household contacts)** in case of breakthrough infections in vaccine recipients for the **Delta variant** based on **Eyre et al 2021** [6].

Vaccine	**Dose 1***			**Dose 2†**		
	Lower limit	**Point estimate**	Upper limit	Lower limit	**Point estimate**	Upper limit
AstraZeneca	0	**2**	10	28	**36**	43
Pfizer BNT	6	**13**	19	52	**65**	74

†estimates in study for ≥14 days post dose 2

Both Harris et al and Eyre et al primarily capture symptomatic infections. For the values in Table 2C to be considered a VE against onward transmission, we need to assume that the fraction of infections in contacts that are symptomatic is independent of the vaccination status of the source case. This would seem reasonable from a virological and immunological perspective.

ACTION TAKEN: for breakthrough transmission VE parameters use values in Table 2C rather than Table 2A.

Table 3A: Combined vaccine effectiveness assumptions on transmission for the Delta variant based on **Sheik [3] and Harris [5]** (as per ATAGI July 2021 advice document).

Vaccine	Reduction in infection *(E_i)*	Reduction in onward transmission *(E_t)*	Calculated overall reduction in transmission*
AstraZeneca Dose 1	18%	48%	57%
AstraZeneca Dose 2	60%	65%	86%
Pfizer BNT Dose 1	30%	46%	62%
Pfizer BNT Dose 2	79%	65%	93%

**Calculated overall reduction in transmission = $1-(1-E_i)*(1-E_t)$*

Table 3B: Combined vaccine effectiveness assumptions on transmission for the Delta variant based on **Pouwels [4] and Eyre [6].**

Vaccine	Reduction in infection *(E_i)*	Reduction in onward transmission *(E_t)*	Calculated overall reduction in transmission*
AstraZeneca Dose 1	46%	2%	46%
AstraZeneca Dose 2	67%	36%	79%
Pfizer BNT Dose 1	57%	13%	63%
Pfizer BNT Dose 2	80%	65%	93%

**Calculated overall reduction in transmission = $1-(1-E_i)*(1-E_t)$*

ACTION TAKEN: for combined VE parameters on transmission for the Delta variant use values in Table 3B rather than Table 3A.

Since completion of the first phase of the National Plan modelling, further evidence has emerged regarding vaccine effectiveness (VE) against clinical outcomes for the Delta variant.

Table 4A: Vaccine effectiveness estimates (% reduction) against symptomatic disease, hospitalisation, ICU admission and death for the Delta variant used in National Plan Modelling.

Vaccine	Symptomatic infection[a]	Hospitalisation[b]	ICU admission[c]	Mortality[b]
AstraZeneca Dose 1	33%	69%	69%	69%
AstraZeneca Dose 2	61%	86%	86%	90%
Pfizer BNT Dose 1	33%	71%	71%	71%
Pfizer BNT Dose 2	83%	87%	87%	92%

[a] Sheik et al [3]. Study reports VE against asymptomatic and symptomatic infection. We use their estimates of VE against *symptomatic* infection.

[b] London School of Hygiene and Tropical Medicine central estimates used for UK roadmap modelling on 9 June 2021 for Delta, see Table 3 [7]. These Delta VE assumptions are scaled from VE estimates for pre-existing and Alpha variants. The starting Alpha assumptions for *hospitalisation* and *second dose mortality* are based on a range of studies and are in line with Public Health England's (PHE) COVID-19 vaccine surveillance report for pre-Alpha and Alpha (week 22) [8]. The starting Alpha assumptions for *first dose mortality* are informed by findings from Dagan et al [9] and Lopez Bernal et al [10]. Note that these assumptions are consistent with PHE's week 31 report (5 August 2021). To obtain estimates for Delta, the Alpha VE assumptions for both hospitalisation and mortality were reduced by half of the relative reductions by dose and product estimated by Lopez Bernal et al for symptomatic infection [11] (see Table 2). See LSHTM roadmap report from 9 June for further details [7].

[c] Few studies report VE against ICU admission for either ancestral or Delta variants. One study conducted in India (Victor et al [12]) reports 95% and 94% reductions in ICU admission after dose 1 and dose 2 of AstraZeneca, respectively. The findings from this study are unlikely to be directly transferable to the Australian setting due to health system differences. In the absence of relevant data for our setting, we assume the same reductions in ICU admission given vaccination as for hospitalisation.

Table 4B: Vaccine effectiveness estimates (% reduction) against symptomatic disease, hospitalisation, ICU admission and death for the Delta variant updated according to studies published since National Plan Modelling work.

Vaccine	Time post dose	Symptomatic infection[a]	Hospitalisation[b]	ICU admission[c]	Mortality[b]
AstraZeneca Dose 1	≥28 days	40%	81%	81%	88%
AstraZeneca Dose 2	≥14 days	71%	93%	93%	93%
AstraZeneca Dose 2	≥20 weeks	-	77%	77%	79%
Pfizer BNT Dose 1	≥28 days	58%	92%	92%	89%
Pfizer BNT Dose 2	≥14 days	84%	97%	97%	95%
Pfizer BNT Dose 2	≥20 weeks	-	93%	93%	90%

[a]Pouwels et al [4]. Study reports VE against asymptomatic and symptomatic infection. We use their estimates of VE against *symptomatic* infection.

[b] Andrews et al [13]. Estimates in study are for ≥28 days post dose 1 and ≥14 days post dose 2 with ≥20 weeks post dose 2 in parentheses following the primary immunisation course.

[c] Few studies report VE against ICU admission for either ancestral or Delta variants. One study conducted in India (Victor et al [12]) reports 95% and 94% reductions in ICU admission after dose 1 and dose 2 of AstraZeneca, respectively. The findings from this study are unlikely to be directly transferable to the Australian setting due to health system differences. In the absence of relevant data for our setting, we assume the same reductions in ICU admission given vaccination as for hospitalisation.

ACTION TAKEN: for clinical outcomes VE parameters use values in Table 4B rather than Table 4A and ≥ 20 weeks post dose 2 estimates for VEs against hospitalisation, ICU admission and mortality.

Clinical severity assumptions

Table 5. Disease severity assumptions for *unvaccinated individuals*

Parameter	Description	Source		Value(s)
Wildtype severity parameters				
Pr(symptoms|wt)	Probability of symptomatic disease given wildtype infection	Davies et al. Nature Medicine (2020) [2] Clinical fractions estimated for 10-year age groups.	Age group	Symptomatic fraction
			0-9	0.28
			10-19	0.20
			20-29	0.26
			30-39	0.33
			40-49	0.40
			50-59	0.49
			60-69	0.63
			70+	0.69
Pr(hosp|symptoms)	Probability of hospital admission given symptomatic wildtype infection	Knock et al. Pre-print [14]. Prepared for UK roadmap modelling by Imperial group. UK data first wave.		Age-specific. See Tables S6 and S8 of Knock et al.
Pr(ICU|hosp)	Probability of ICU admission given hospital admission	Same as above.		Same as above.
Pr(death|ward)	Probability of death for ward patients (no ICU stay)	Same as above.		Same as above.
Pr(death|ICU)	Probability of death for ICU patients	Same as above.		Same as above.
Pr(death|post-ICU ward)	Probability of death for post-ICU patients	Same as above.		Same as above.

Alpha severity parameters (versus wildtype)			
Pr(symptoms|alpha)	Probability of symptomatic disease given Alpha infection	A number of studies using UK data suggest that the probability of reporting symptoms is consistent for wildtype and Alpha Walker et al. Pre-print [15]. Graham et al. Lancet Public Health (2021) [16].	RR=1
Pr(hosp|alpha)	Probability of hospitalisation given Alpha infection	Bager et al. Lancet Infect Dis (2021) [17]. Denmark data.	OR=1.42
Pr(ICU|alpha)	Probability of ICU admission given Alpha infection	Patone et al. Lancet ID [18]. UK data.	HR=2.15
Pr(death|alpha)	Probability of death given Alpha infection	Davies et al. Nature (2021) [19]. UK data.	HR=1.61
Delta severity parameters (versus Alpha)			
Pr(hosp|delta)	Probability of hospitalisation given Delta infection	Bager et al. Lancet ID (2021) [20]. Denmark data.	RR = 3.01
Delta severity parameters (versus wildtype)			
Pr(hosp|delta)		Fisman & Tuite. Pre-print [21]. Canada data.	*OR = 2.08
Pr(ICU|delta)	Probability of ICU admission given Delta infection	Fisman & Tuite. Pre-print [21]. Canada data.	*OR = 3.35
Pr(death|delta)	Probability of death given Delta infection	Fisman & Tuite. Pre-print [21]. Canada data.	*OR = 2.33

*Note that for Pr(hosp|delta), Pr(ICU|delta) and Pr(death|delta) is direct estimate of Delta versus wildtype (rather than Alpha).

ACTION TAKEN: Incorporate delta severity parameters into overall estimates of disease severity.

References

1. Prem KP et al. Projecting social contact matrices in 152 countries using contact surveys and demographic data. *PLoS Computational Biology 2017;* 13(9):e1005697.
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