

Methods of the transmission dynamic  
model for projecting COVID-19 burden  
and health service capacity needs in  
Victoria

## Contents

## 1 Preamble

This document describes the methods used for projecting burden of COVID-19 in Victoria for the Victorian Government Department of Health from September 2021 onwards. The model is similar in construction to that used for similar purposes in the latter part of 2020 during Victoria’s second COVID-19 wave. The analysis of the second wave was submitted to a major international journal and has now been revised three times, such that publication is hopefully imminent. This work is also publicly available as a pre-print on the *medRxiv* server here. The pre-print did not include structure for contact tracing, which was added during revision of the re-submitted manuscript and is included in the model described here. The current version of the model also includes structure for vaccination, which has not been used in our analyses for Victoria previously.

## 2 Base model construction

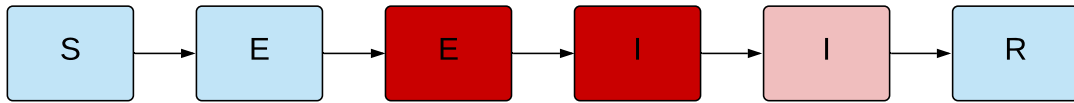
### 2.1 Platform for infectious disease dynamics simulation

We developed a deterministic compartmental model of COVID-19 transmission using the AuTuMN platform, publicly available <https://github.com/monash-emu/AuTuMN/>. Our repository allows for the rapid and robust creation and stratification of models of infectious disease epidemiology and includes pluggable modules to simulate heterogeneous population mixing, demographic processes, multiple circulating pathogen strains, repeated stratification and other dynamics relevant to infectious disease transmission. The platform was created to simulate TB dynamics, being an infectious disease whose epidemiology differs markedly by setting, such that considerable flexibility was desirable [?]. We have progressively developed the structures of our platform over recent years, and further adapted it to be sufficiently flexible to permit simulation of other infectious diseases, such as COVID-19. A similar model has been applied to several countries of the Asia-Pacific, with the application to the Philippines (without structure to represent geospatial stratification or contact tracing) previously described.[?]

### 2.2 Base COVID-19 model

Using the base framework of an SEIR model (susceptible, exposed, infectious, removed), we split the exposed and infectious compartments into two sequential compartments each (SEIIR). The two sequential exposed compartments represent the non-infectious and infectious phases of the incubation period, with the latter representing the “presymptomatic” phase such that infectious-

ness occurs during three of the six sequential phases. For this reason, “active” is a more accurate term for the two sequential “I” compartments and is preferred henceforward. The two infectious compartments represent early and late phases of active disease, during which symptoms occur if the disease episode is symptomatic, and allow explicit representation of notification, case isolation, hospitalisation and admission to ICU. The “active” compartment also includes some persons who remain asymptomatic throughout their disease episode, such that these compartments do not map directly to either persons who are infectious or those who are symptomatic (Figure ??).



**Figure 1: Unstratified compartmental model structure.**  $S$  = susceptible,  $E$  = exposed,  $I$  = active,  $R$  = recovered/removed. Depth of pink/red shading indicates the infectiousness of the compartment.

The latently infected and infectious presymptomatic periods together comprise the incubation period, with the incubation period and the proportion of this period for which patients are infectious defined by input parameters described below. In general, two sequential compartments can be used to form a gamma-distributed profile of transition to infectiousness following exposure if the progression rates for these two compartments are equal, although in implementing this model the relative sojourn times in the two sequential compartments usually differed. Nevertheless, the profiles implemented are broadly consistent with the empirically observed log-normal distribution of individual incubation periods [?].

The transition from early active to late active represents the point at which patients are detected (for those persons for whom detection does eventually occur) and isolation then occurs from this point forward (i.e. applied during the late disease phase only). This transition point is also intended to represent the point of admission to hospital or transition from hospital ward to intensive care for patients for whom this occurs (see Section ??).

### 2.3 Age stratification

All compartments of this base compartmental structure were stratified by age into five-year bands from 0-4 years of age through to 70-74 years of age, with the final age group being those aged

75 years and older. Heterogeneous baseline contact patterns by age were incorporated using age-specific contact rates estimated from survey data from the POLYMOD study, which reported rates of age-specific contacts in various locations between persons of different age groupings (as described below ??). These are then modified by non-pharmaceutical interventions. Our modelled age groups were chosen to match these mixing matrices. The automatic demographic features of the platform that can be used to simulate births, ageing and deaths were not implemented, because the issues considered pertain to the short- to medium-term and the immediate implementation of control strategies, for which population demographics are less relevant.

## 2.4 Contact matrices construction

For each location  $L$  (home, school, work, other locations) the age-specific contact matrix  $\mathbf{C}^L = (c_{i,j}^L) \in \mathbb{R}_+^{16 \times 16}$  is defined such that  $c_{i,j}^L$  is the average number of contacts that a typical individual aged  $i$  has with individuals aged  $j$ . As there is no contact survey available for Australia that is complete across all age groups, the matrices  $\mathbf{C}^L$  were obtained by extrapolating contact matrices from the United Kingdom, being a country included in the POLYMOD study in 2005 [?]. The original matrices from the United Kingdom are denoted  $\mathbf{Q}^L = (q_{i,j}^L) \in \mathbb{R}_+^{16 \times 16}$ , where  $q_{i,j}^L$  is defined using the same convention as for  $c_{i,j}^L$ . The matrices  $\mathbf{Q}^L$  were extracted using the R package “socialmixr” (v 0.1.8) and then adjusted to account for age distribution differences between Victoria and the United Kingdom.

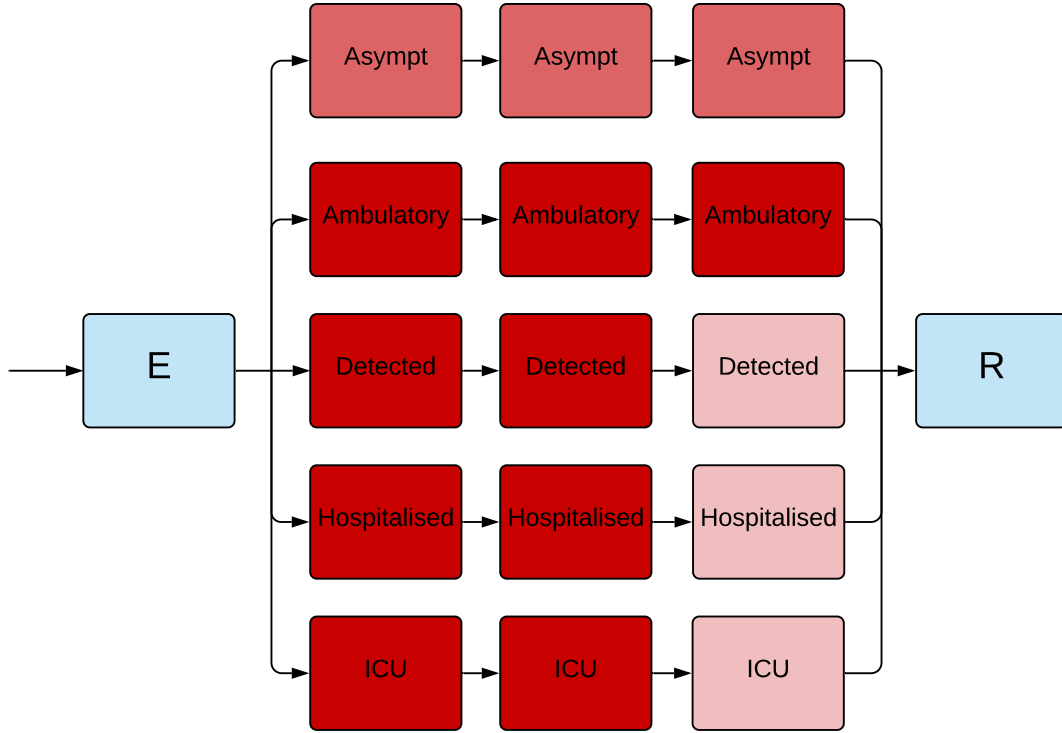
Let  $\pi_j$  denote the proportion of people aged  $j$  in Victoria, and  $\rho_j$  the proportion of people aged  $j$  in the United Kingdom. The contact matrices  $\mathbf{C}^L$  were obtained from:

$$c_{i,j}^L = q_{i,j}^L \times \frac{\pi_j}{\rho_j}.$$

## 2.5 Clinical stratification

The age-stratified late exposed/incubation and both the early and late active disease compartments were further stratified into five “clinical” categories: 1) asymptomatic, 2) symptomatic ambulatory, never detected, 3) symptomatic ambulatory, ever detected, 4) ever hospitalised, never critical, and 5) ever critically unwell (Figure ??). The proportion of new infectious persons entering stratum 1 (asymptomatic) is age-dependent and constant over time. The proportion of symptomatic patients (strata 2 to 5) ever detected (strata 3 to 5) is set through a parameter that represents the time-varying proportion of all symptomatic patients who are ever detected (the case detection rate). Of

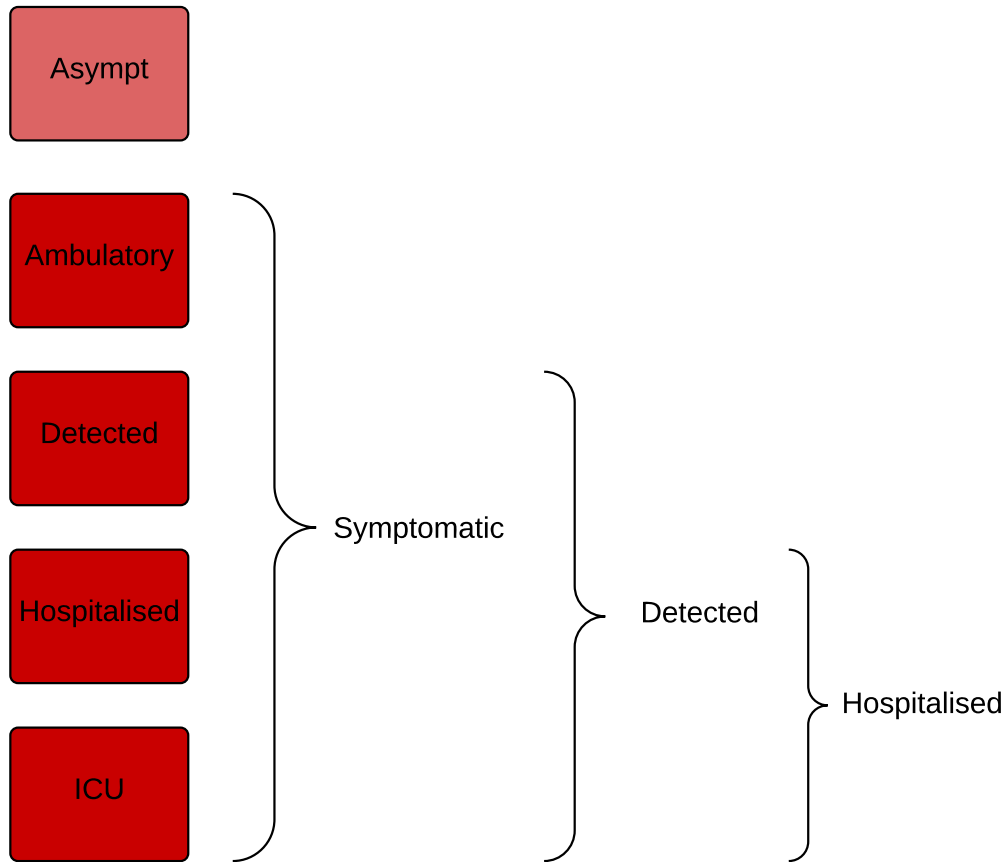
those ever symptomatic (strata 2 to 5), an age-specific proportion is considered to be hospitalised (entering strata 4 or 5, constant over time). Of those hospitalised (entering strata 4 or 5), a fixed proportion was considered to be critically unwell (entering stratum 5, Figure ??, not age-specific, constant over time).



**Figure 2: Illustration of the implementation of the clinical stratification.** Depth of pink/red shading indicates the infectiousness of the compartment. Typical parameter values represented, although the infectiousness of asymptomatic persons is varied in calibration.

## 2.6 Hospitalisation

For COVID-19 patients who are admitted to hospital, the sojourn time in the early and late active compartments is modified, superseding the default values of the sojourn times for these compartments. The point of admission to hospital is considered to be the transition from early to late active disease, such that the sojourn time in the late disease represents the period of



**Figure 3: Illustration of the rationale for the clinical stratification.**

time admitted to hospital. For patients admitted to ICU, admission to ICU occurs at this same transition point. For this group, the period of time hospitalised prior to ICU admission is estimated as a proportion of the early active period, such that the early active period represents both the period ambulatory in the community and the period in hospital prior to ICU admission.

## **2.7 Infectiousness**

Asymptomatic persons are assumed to be less infectious per unit time active than symptomatic persons not undergoing case isolation (typically by around 50%, although this is varied in calibration/uncertainty analysis). Infectiousness is also decreased for persons who have been detected to

reflect case isolation, and for those admitted to hospital or ICU to reflect infection control procedures (by 80% for all groups, parameter value consistent with typically used modelled values but not informed by empiric estimates). Presymptomatic individuals are assumed to have equivalent infectiousness to those with early active COVID-19.

## 2.8 Application of COVID-19-related death

Age-specific infection fatality rates (IFRs) were applied and distributed across strata 4 and 5, with no deaths typically applied to the first three strata. A ceiling of 50% is set on the proportion of those admitted to ICU (entering stratum 5) who die. If the infection fatality rate is greater than this ceiling, the proportion of critically unwell persons dying was set to 50%, with the remainder of the infection fatality rate then applied to the hospitalised proportion. Otherwise, if the infection fatality rate is less than half of the absolute proportion of persons critically unwell, the infection fatality rate is applied entirely through stratum 5 (such that the proportion of critically unwell persons dying in that age group becomes <50% and the proportion of stratum 4 dying is set to zero). In the event that the infection fatality rate for an age group is greater than the total proportion hospitalised (which is unusual, but could occur for the oldest age group under certain parameter configurations), the remaining deaths are assigned to the asymptomatic stratum. This approach was adopted for computational ease and is valid because the duration active for persons entering this stratum is the same as for the other non-hospitalised strata, such that the dynamics are identical to assigning the deaths to any of the first three strata. We used the age-specific IFRs previously estimated from age-specific death data from 45 countries and results from national-level seroprevalence surveys [?].

Clinical stratum	Stratum name	Pre-symptomatic	Early	Late
1	Asymptomatic	0.5	0.5	0.5
2	Symptomatic ambulatory never detected	1	1	1
3	Symptomatic ambulatory ever detected	1	1	0.2
4	Hospitalised never critical	1	1	0.2
5	Ever critically unwell	1	1	0.2

**Table 1: Illustration of the relative infectiousness of disease compartments by clinical stratification and stage of infection. Typical parameter values displayed.**



### 3 Case detection and isolation

#### 3.1 Determining the proportion of cases detected

We calculate a time-varying case detection rate, being the proportion of all symptomatic cases (clinical strata 2 to 5) that are detected (clinical strata 3 to 5). This proportion is informed by the number of tests performed using the following formula:

$$CDR(time) = 1 - e^{-shape \times tests(time)}$$

$time$  is the time in days from the 31<sup>st</sup> December 2019 and  $tests(time)$  is the number of tests per capita done on that date. To determine the value of the shape parameter, we solve this equation based on the assumption that a certain daily testing rate  $tests(t)$  is associated with a certain  $CDR(t)$ . Solving for  $shape$  yields:

$$shape = \frac{-\log(1 - CDR(t))}{tests(t)}$$

That is, if it is assumed that a certain daily per capita testing rate is associated with a certain proportion of symptomatic cases detected, we can determine  $shape$ . As this relationship is not well understood and unlikely to be consistent across all settings, we vary the  $CDR$  that is associated with a certain per capita testing rate during uncertainty/calibration. Given that the  $CDR$  value can be varied widely, the purpose of this is to incorporate changes in the case detection rate that reflect the historical profile of changes in testing capacity over time.

The proportion of persons entering clinical stratum 3 is calculated once the  $CDR$  is known, along with the proportion of all incident cases hospitalised (strata 4 and 5).

#### 3.2 Isolation of detected cases

As described in the Section ?? above, as infected persons progress from the early to the late stage of active COVID-19, infectiousness is reduced for those in the detected strata (3 to 5) to reflect case isolation.

### 4 Contact tracing and quarantine

#### 4.1 Model adaptation

We simulate quarantining of persons identified as first degree contacts of COVID-19 patients explicitly through stratification of the compartments representing active COVID-19. That is, the compartments representing both phases of the incubation period and both phases of active COVID-19 are duplicated into two model strata referred to as “traced” and “untraced”. In model initialisation, all infectious seed is assigned to the untraced stratum. All newly infected persons commence their incubation period in the untraced stratum of the early incubation period. As for isolated and hospitalised patients, those undergoing quarantine have their infectiousness reduced by 80%.

#### 4.2 Contact tracing process

Identification of infected persons through contact tracing is assumed to apply to those in their early incubation period, with flows added to the model that transition persons during their incubation period from the untraced to the traced stratum of this compartment type. The rate of transition from the untraced to the traced stratum of the early incubation period is determined by the proportion of contacts traced. It is assumed that only the contacts of identified cases can be traced, such that the case detection rate (the proportion of symptomatic cases detected) is the ceiling for the proportion of contacts traced. The proportion of contacts of identified cases that is traced is multiplied by the proportion of contacts whose index is detected to determine the proportion of all persons entering the incubation period who are traced. The proportion of all contacts of infectious persons with a detected index case,  $u(t)$ , is calculated as the relative contribution of ever-detected infectious individuals to the total force of infection, and is given as:

$$u(t) = \frac{\sum_{c \in \mathcal{C}} \sum_{s \in \mathcal{D}} prev_{c,s}(t) \times inf_{c,s}}{\sum_{c \in \mathcal{C}} \sum_{s \in \mathcal{S}} prev_{c,s}(t) \times inf_{c,s}},$$

where  $\mathcal{C}$  is the set of infectious compartments,  $\mathcal{S}$  represents all clinical strata and  $\mathcal{D} \subset \mathcal{S}$  is the list of detected clinical strata. The prevalence of infectious compartment  $c$  in clinical stratum  $s$  at time  $t$  is represented by  $prev_{c,s}(t)$ , and  $inf_{c,s}$  is the relative infectiousness of compartment  $c$  in clinical stratum  $s$ .

The proportion of contacts of identified cases that is traced,  $q(t)$ , is considered to decrease as the severity of the COVID-19 epidemic increases, because we expect contact tracing to decline in efficiency as more cases are identified. That is, we assume that contact tracing is universal as COVID-19 prevalence approaches zero and declines exponentially with increasing prevalence. The relationship between the proportion of contacts of identified patients who are quarantined and

prevalence is given as:

$$q(t) = e^{-prev(t) \times \tau}$$

Rather than estimate  $\tau$  directly, we estimate the more intuitive quantity of the proportion of contacts of identified patients who would be quarantined at a particular prevalence. Solving for the previous equation for  $\tau$ , we obtain:

$$\tau = \frac{-\log(q(t))}{prev(t)}$$

or  $\tau = \frac{-\log(q_0)}{prev_0}$  at a specific prevalence that accords with a particular value of  $q$ . Fixing  $prev_0$  at  $10^{-3}$ , we can vary  $q_0$  in calibration as the proportion of contacts of identified cases detected at a prevalence of one active case per thousand population.

Finally,  $q(t) \times u(t)$  gives the proportion of all infected persons who are traced. This proportion of persons entering their early latent period transition to the equivalent compartment in the traced stratum before proceeding to the late latent period.

### 4.3 Testing data

Victorian statewide daily testing data by date of test are obtained from Our World in Data and applied identically to all health service clusters to provide a broad profile of the variation in testing capacity over time. Data sparseness precluded us from implementing separate functions for each individual health service cluster. For this application to Victoria, the case detection proportion corresponding to a per capita rate of testing of one test per thousand population per day was varied as a calibration parameter in creating the time-varying case detection proportion function. Note that testing rates were typically considerably higher than one per thousand per day during the period modelled, such that the actual modelled case detection proportion is considerably higher than the case detection calibration parameter for most of the simulation period.

## 5 Implementation of non-pharmaceutical interventions

A major part of the rationale for the development of this model was to capture the past impact of non-pharmaceutical interventions (NPIs) and produce future scenarios projections with the implementation or release of such interventions.

### 5.1 Isolation and quarantine

For persons who are identified with symptomatic disease and enter clinical stratum 3, self-isolation is assumed to occur and their infectiousness is modified as described above. The proportion of ambulatory symptomatic persons passively identified through the public health response is determined by the case detection rate as described above in Section ??.

### 5.2 Community quarantine or “lockdown” measures

For all NPIs relating to reduction of human mobility or “lockdown” (i.e. all NPIs other than isolation and quarantine), these interventions are implemented through dynamic adjustments to the age-assortative mixing matrix. The baseline mixing matrices have the major advantage of allowing for disaggregation of total contact rates by location, i.e. home, work, school and other locations. This disaggregation allows for the simulation of various NPIs in the local context by dynamically varying the contribution of each location to reflect the historical implementation of the interventions.

For each location  $L$  (home, school, work, other locations) the age-specific contact matrix  $\mathbf{C}^L = (c_{i,j}^L) \in \mathbb{R}_+^{16 \times 16}$  is defined such that  $c_{i,j}^L$  is the average number of contacts that a typical individual aged  $i$  has with individuals aged  $j$ . The original matrices from the United Kingdom are denoted  $\mathbf{Q}^L = (q_{i,j}^L) \in \mathbb{R}_+^{16 \times 16}$ , where  $q_{i,j}^L$  is defined using the same convention as for  $c_{i,j}^L$ . The matrices  $\mathbf{Q}^L$  were extracted using the R package “socialmixr” (v 0.1.8).

Let  $\pi_j$  denote the proportion of people aged  $j$  in the UK, and  $\rho_j$  the proportion of people aged  $j$  in Victoria. The contact matrices  $\mathbf{C}^L$  were obtained from:

$$c_{i,j}^L = q_{i,j}^L \times \frac{\pi_j}{\rho_j}.$$

The overall contact matrix results from the summation of the four location-specific contact matrices:  $C_0 = C_H + C_S + C_W + C_L$ , where  $C_H$ ,  $C_S$ ,  $C_W$  and  $C_L$  are the age-specific contact matrices associated with households, schools, workplaces and other locations, respectively.

In our model, the contributions of the matrices  $C_S$ ,  $C_W$  and  $C_L$  vary with time such that the input contact matrix can be written:

$$C(t) = C_H + s(t)^2 C_S + w(t)^2 C_W + l(t)^2 C_L$$

The modifying functions are each squared to capture the effect of the mobility changes on both the infector and the infectee in any given interaction that could potentially result in transmission.

The modifying functions incorporate both macro-distancing and microdistancing effects, depending on the location.

### **5.3 School closures/re-openings**

Reduced attendance at schools is represented through the function  $s(t)$ , which represents the proportion of all students currently attending on-site teaching. If schools are fully closed,  $s(t) = 0$  and  $C_S$  does not contribute to the overall mixing matrix  $C(t)$ .  $s(t)$  is calculated through a series of estimates of the proportion of students attending schools, to which a smoothed step function is fitted. Note that the dramatic changes in this contribution to the mixing matrix with school closures/re-openings is a more marked change than is seen with the simulation of policy changes in workplaces and other locations (which are determined by empiric data and so do not vary so abruptly or reach a value of zero).

### **5.4 Workplace closures**

Workplace closures are represented by quadratically reducing the contribution of workplace contacts to the total mixing matrix over time. This is achieved through the scaling term  $w(t)^2$  which modifies the contribution of  $C_W$  to the overall mixing matrix  $C(t)$ . The profile of the function  $w(t)$  is set by fitting a polynomial spline function to Google mobility data for workplace attendance (Table ??).

### **5.5 Community-wide movement restriction**

Community-wide movement restriction (or “lockdown”) measures are represented by proportionally reducing the contribution of the other locations contacts to the total mixing matrix over time. This is achieved through the scaling term  $l(t)^2$  which modifies the contribution of  $C_L$  to the overall mixing matrix  $C(t)$ . The profile of the function  $l(t)$  is set by fitting a polynomial spline function to an average of Google mobility data for various locations, as indicated in Table ??.

### **5.6 Household contacts**

The contribution of household contacts to the overall mixing matrix  $C(t)$  is fixed over time. Although Google provides mobility estimates for residential contacts, the nature of these data are different from those for each of the other Google mobility types. They represent the time spent in that location, as opposed to the other categories, which measure a change in total visitors rather

than the duration. The daily frequency with which people attend their residence is likely to be close to one and we considered that household members likely have a daily opportunity for infection with each other household member. Therefore, we did not implement a function to scale the contribution of household contacts to the mixing matrix with time.

location	Approach	Google mobility types
School	Policy response	Not applicable
Household	Constant	Not applicable
Workplace	Google mobility	Workplace
Other locations	Google mobility	Unweighted average of: <ul style="list-style-type: none"> <li>• Retail and recreation</li> <li>• Grocery and pharmacy</li> <li>• Parks</li> <li>• Transit stations</li> </ul>

Table 2: **Mapping of Google mobility data to contact locations.**

### 5.7 Microdistancing

Interventions other than those that prevent people coming into contact with one another are thought to be important to COVID-19 transmission and epidemiology, such as maintaining interpersonal physical distance and the wearing of face coverings. We therefore implemented a “microdistancing” function to represent reductions in the rate of effective contact that is not attributable to persons visiting specific locations and so is not captured through Google mobility data. This microdistancing function reduces the values of all non-household contributions to the mixing matrices by a certain proportion. These time-varying functions multiplicatively scale the location-specific contact rate modifiers  $s(t)$ ,  $w(t)$  and  $l(t)$ .

## 6 Simulation of local NPI implementation during Victoria’s second wave

## 6.1 School closures

The effect of Victorian school closures is captured through the timeline presented in Table ??.

Date of change	Policy change	Modification applied to school contacts contribution to mixing matrix, $s(t)$
From model start	Onsite learning	1
16 <sup>th</sup> July	Schools close for lockdown 5	0.1
27 <sup>th</sup> July	Schools re-open after lockdown 5	1
5 <sup>th</sup> August	Schools close for lockdown 6	0.1
18 <sup>th</sup> September	Four of 13 year levels (foundation to year 2 and year 12) return in regional Vic	0.30769 (regional only)

**Table 3: Timeline used to implement Victorian school closure policies.** The function is applied to both metropolitan and regional services.

## 6.2 Macrod distancing in workplaces and other locations

The functions applied here are determined by the Google mobility data according to Table ??, as described above, but are applied separately for each health service. Because Google mobility data pertain to local government areas (LGAs), whereas health service clusters may receive patients from across the state, it was necessary to map mobility data to services. Health service clusters' overall mobility values in each location were calculated using a weighted average of LGA mobility values according to the historical pattern of the origin of patients presenting to services within each service.

As a hypothetical example, if 50% of patients historically presenting to Barwon South West health services come from the City of Geelong, the mobility data for the City of Geelong will contribute 50% of the Google mobility estimate of Barwon South West.

Historical patterns of patient presentations by health service cluster were provided by the Victorian Department of Health.

### 6.3 Microdistancing approach

In this application to Victoria, the microdistancing function  $m(t)$  is comprised of two components: physical distancing and face coverings. Both physical distancing and face coverings microdistancing are applied to the three non-household locations, such that the microdistancing function for non-household locations is given by:

$$m(t) = d(t)^2 \times f(t)^2$$

The two interventions are assumed to be independent and so are multiplicative. As for the macrodistancing functions, the two functions of time are squared to represent their effects on both the infector and the infectee in any potentially infectious interaction. The face covering function in this analysis is set at the constant value of 0.84, because recent proportions of YouGov survey participants responding “always” to the question “Thinking about the last 7 days, have you worn a face mask outside your home (e.g. when on public transport, going to a supermarket, going to a main road)?” have been consistently around 84%. For the physical distancing function, the proportion of participants reporting zero contacts within two metres in Victoria has increased from around 16% to 24% over recent months. A hyperbolic tan function was used to fit these data, as presented in Figure ??.

## 7 Between service mixing

The preceding section describes the creation of heterogeneous mixing matrices by age for each of the nine health service clusters individually. These mixing matrices are then combined to create a single time-varying heterogeneous mixing matrix by service and age resulting in a 144 by 144 ( $9 \times 16 = 144$ ) square mixing matrix. The force of infection for an index service is calculated from the mixing matrices of the age-assortative matrix for each of the services modelled. The spatial mixing matrix is based on the adjacency of health service clusters as indicated in Table ??.

## 8 Model initialisation

The model is commenced from late March to allow dynamic vaccination to occur prior to seeding of the epidemic. The epidemic is then seeded predominantly through the Metropolitan clusters from early July to initiate transmission.



## 9 Vaccination

### 9.1 Model structure

In this application we introduced separate strata to the model for vaccinated and unvaccinated individuals. All model compartments are replicated into three categories:

- Unvaccinated persons
- Persons who have only received one dose of vaccination
- Persons who have received two doses of vaccination (i.e. the full course)

Note that the model does not contain structure to represent different types of vaccination, despite different schedules likely having somewhat different characteristics. This decision was made in the interest of parsimony, given the extreme level of complexity that the model structure has reached in relation to other features.

### 9.2 Vaccination process

The process of vaccination is only applied to the susceptible and recovered compartments and not to any of the compartments representing current active infection. Transition flows are applied to move persons from the unvaccinated compartments to the one-dose compartments according to the history of vaccination roll-out. The transition flow from the one dose stratum to the two dose stratum is then applied with a rate equal to the reciprocal of the average delay between first and second dose.

## 10 Parameters

### 10.1 Non-age-stratified parameters

Parameter	Value	Rationale
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continuation of parameters table

Incubation period	Calibration parameter, truncated normal distribution, mean 6.1 days, standard deviation 0.8 days	Prior distribution taken from marginal posterior distribution from 2020 analysis
Proportion of incubation period infectious	50%	Infectiousness is considered to be present throughout a considerable proportion of the incubation period, based on analyses of confirmed source-secondary pairs [?] and early findings that the incubation period was similar to the serial interval [?]. The study of source-secondary pairs was also the primary reference cited by a review of the infectious period that identified studies that quantified the pre-symptomatic period, which concluded that the median pre-symptomatic period could range from less than one to four days [?].
Active period (regardless of detection/isolation, for clinical strata 1 to 3)	Calibration parameter, truncated normal distribution, mean 6.4 days, standard deviation 0.7 days	Prior distribution taken from marginal posterior distribution from 2020 analysis
Proportion of infectious period before isolation or hospitalisation can occur	0.333	Assumed

continuation of parameters table

Disease duration prior to admission for hospitalised patients not critically unwell (i.e. early active sojourn time, stratum 4)	7.7 days	Mean value from ISARIC cohort, as reported on 4 <sup>th</sup> October 2020 in Table 6 [?], and similar to the expected mean from earlier reports from ISARIC [?]. This cohort represents high-income countries better than low and middle-income countries, with the United Kingdom contributing data on the greatest number of patients, followed by France. Earlier estimates of this quantity from China included 4.4 days [?].
Duration of hospitalisation if not critically unwell (late active sojourn time, stratum 4)	7.8 days	FluCAN monthly Covid Epi Report to CDNA, 30 <sup>th</sup> August 2020
ICU duration (late active sojourn time, stratum 5)	5.9 days	SPRINT-SARI Australia Project
Duration of time prior to ICU for patients admitted to ICU	10.5 days	Calculated as the sum of the time from symptom onset to hospital admission (7.7 days above) plus the duration from hospital admission to ICU admission reported by October ISARIC report (2.8 days) [?].
Relative infectiousness of persons admitted to hospital or ICU	0.2	Assumed
Relative infectiousness of identified persons in isolation	0.2	Assumed

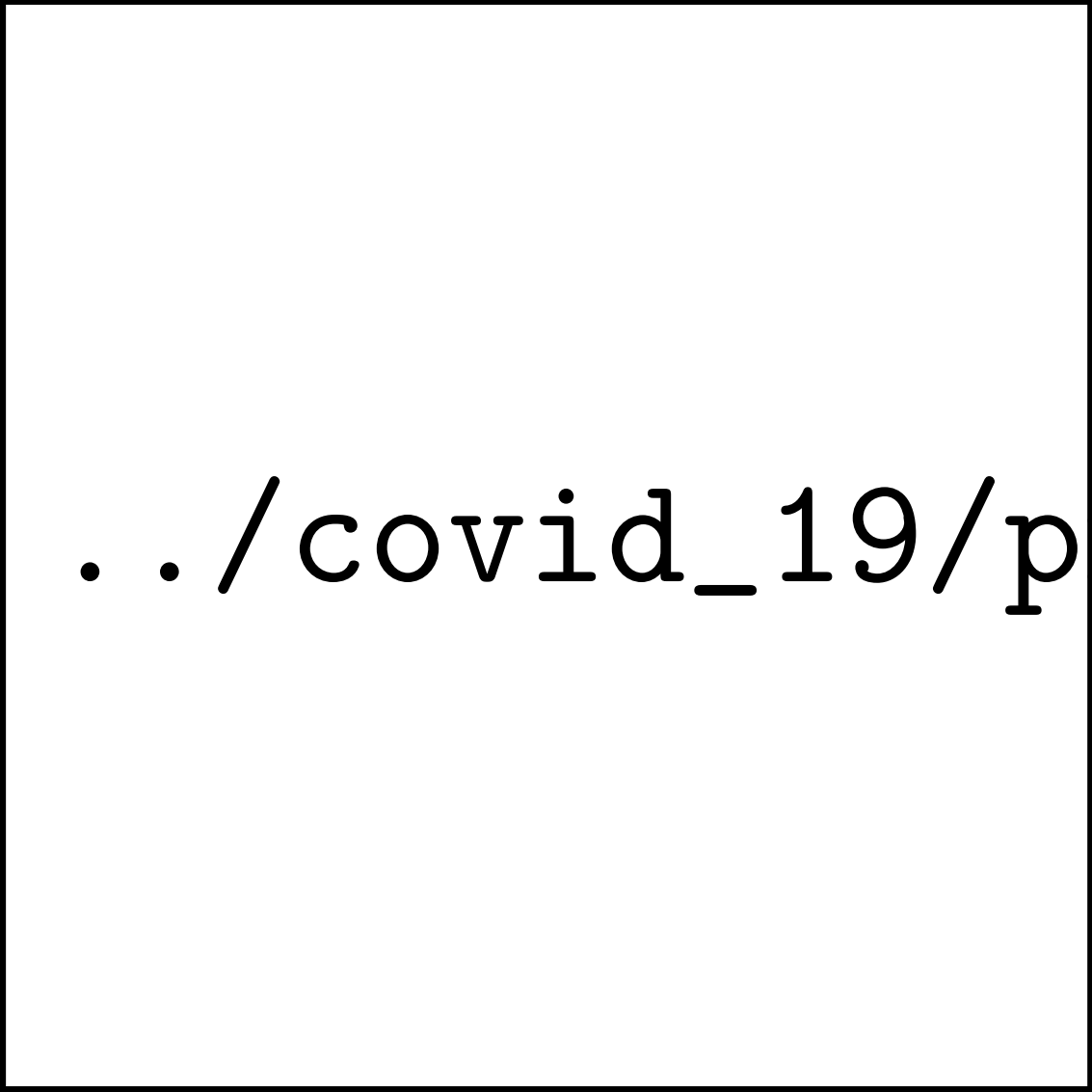
continuation of parameters table

Clinical effectiveness of one vaccine dose	0.49	Mean of one dose effectiveness for BNT162b2 and ChAdOx1 reported in <a href="https://www.medrxiv.org/content/10.1101/2021.08.18.21262237v1">www.medrxiv.org/content/10.1101/2021.08.18.21262237v1</a>
Clinical effectiveness of full course of vaccination	0.775	Mean of full course effectiveness for BNT162b2 and ChAdOx1 reported in <a href="https://www.medrxiv.org/content/10.1101/2021.08.18.21262237v1">www.medrxiv.org/content/10.1101/2021.08.18.21262237v1</a>
Proportion of effect of vaccination mediated through prevention of infection	0.95	Similar estimates for clinical effectiveness and effectiveness in preventing transmission
Reduction in infectiousness of one vaccine dose	0.56	Mean of one dose effectiveness for BNT162b2 and ChAdOx1 reported in <a href="https://www.medrxiv.org/content/10.1101/2021.08.18.21262237v1">www.medrxiv.org/content/10.1101/2021.08.18.21262237v1</a>
Reduction in infectiousness of full course of vaccination	0.77	Mean of full course effectiveness for BNT162b2 and ChAdOx1 reported in <a href="https://www.medrxiv.org/content/10.1101/2021.08.18.21262237v1">www.medrxiv.org/content/10.1101/2021.08.18.21262237v1</a>

**Table 5: *Universal (non-age-stratified) model parameters.*** Point estimates are used as model parameters except where ranges are indicated in calibration parameter table below in calibration table. Note that all vaccination-related parameters pertain specifically to Delta.

## 10.2 Age-specific parameters

Age-structured parameters are presented in Table ??.



../covid\_19/proj

Figure 4: **Physical distancing micro-distancing function with data used for fitting.**

	Barwon South West	Gippsland	Hume	Loddon-Mallee	Grampians	North Metro	South East Metro	South Metro	West Metro
Barwon South West	R	0	0	0	M	M	0	0	M
Gippsland	0	R	M	0	0	0	M	M	0
Hume	0	M	R	M	0	M	M	0	0
Loddon-Mallee	0	0	M	R	M	M	0	0	M
Grampians	M	0	0	M	R	M	0	0	M
North Metro	M	0	M	M	M	R	M	0	M
South East Metro	0	M	M	0	0	M	R	M	0
South Metro	0	M	0	0	0	0	M	R	0
West Metro	M	0	0	M	M	M	0	0	R

**Table 4: Adjacency-based spatial mixing matrix.** 0, no mixing between spatial patches; M, calibrated inter-service mixing parameter for adjacent services; R, the diagonal matrix elements are populated with the complement of the other values for each row/column (and so may take a different value in each cell in which it appears)

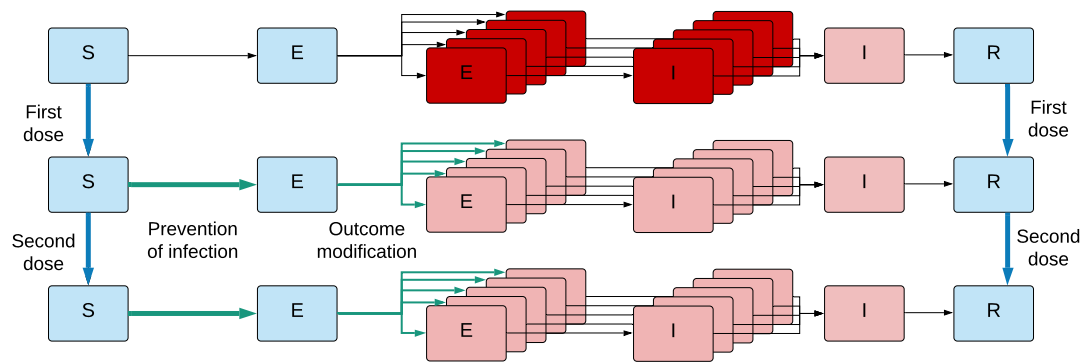


Figure 5: **Approach to model stratification for vaccination.** Vaccination has three modelled effects: prevention of infection, reduction in severe outcomes given infection and reduction in infectiousness.

Age group (years)	Clinical fraction <sup>a</sup>	Relative susceptibility to infection	Infection fatality rate	Proportion of symptomatic patients hospitalised
0 to 4	0.533	0.36	$3 \times 10^{-5}$	0.029
5 to 9	0.533	0.36	$1 \times 10^{-5}$	0.029
10 to 14	0.533	0.36	$1 \times 10^{-5}$	0.014
15 to 19	0.533	1	$3 \times 10^{-5}$	0.014
20 to 24	0.679	1	$6 \times 10^{-5}$	0.030
25 to 29	0.679	1	$1.3 \times 10^{-4}$	0.030
30 to 34	0.679	1	$2.4 \times 10^{-4}$	0.047
35 to 39	0.679	1	$4.0 \times 10^{-4}$	0.047
40 to 44	0.679	1	$7.5 \times 10^{-4}$	0.081
45 to 49	0.679	1	$1.21 \times 10^{-3}$	0.081
50 to 54	0.679	1	$2.07 \times 10^{-3}$	0.128
55 to 59	0.679	1	$3.23 \times 10^{-3}$	0.128
60 to 64	0.803	1	$4.56 \times 10^{-3}$	0.221
65 to 69	0.803	1.41	$1.075 \times 10^{-2}$	0.221
70 to 74	0.803	1.41	$1.674 \times 10^{-2}$	0.389
75 and above	0.803	1.41	$5.748 \times 10^{-2}$ , <sup>b</sup>	0.471
Source/ rationale	Table 1 of systematic review and meta-analysis with appropriate accounting for testing during the pre-symptomatic period [?].	Conversion of odds ratios presented in Table S15 of Zhang et al. 2020 to relative risks using data presented in Table S14 of the same study [?]. <sup>c</sup>	Estimated from pooled analysis of data from 45 countries from Table S3 of O'Driscoll et al [?]. Values consistent with previous estimates using serosurveys performed in Spain [?].	Table 1.1 from NNDSS report on COVID-19 cases in Australia to May 2021

**Table 6: Age-stratified parameters not varied during calibration, or varied through a common multiplier.** <sup>a</sup>Proportion of incident cases developing symptoms. <sup>b</sup>Weighted average of IFR estimates for 70 to 79 and 80 and above age groups. <sup>c</sup>Note the relative magnitude of these values are similar to those estimated by the analysis we use to estimate the age-specific clinical fraction.



## **11 Calculation of outputs**

### **11.1 Incidence**

Incidence is calculated as any transitions into the early active compartment ( “ $I$ ”).

### **11.2 Hospital occupancy**

This is calculated as the sum of three quantities:

1. All persons in the late active compartment in clinical stratum 4, representing those admitted to hospital but never critically unwell.
2. All persons in the late active compartment in clinical stratum 5, representing those currently admitted to ICU.
3. A proportion of the early active compartment in clinical stratum 5, representing those who will be admitted to ICU at a time in the future. This proportion is calculated as the quotient of 1) the difference between the pre-ICU period and the pre-hospital period for clinical stratum 4, divided by 2) the total pre-ICU period. That is, a proportion of the pre-ICU period is considered to represent patients in hospital who have not yet been admitted to ICU.

### **11.3 ICU occupancy**

This is calculated as all persons in the late active compartment in clinical stratum 5.

### **11.4 Recovered proportion**

This is calculated as the proportion of the population in the recovered ( “ $R$ ”) compartment. Although very similar numerically to the attack rate, persons who died of COVID-19 are not included in the denominator.

### **11.5 COVID-19-related mortality**

This is calculated as all transitions representing death, exiting the model. This is implemented as depletion of the late active compartment.

## **11.6 Notifications**

Local case notifications are calculated as transitions from the early to the late active compartment for clinical strata 3 to 5.

## **12 Calibration**

We calibrated the model using the adaptive Metropolis algorithm described by Haario et al. [?]. A standard Metropolis algorithm with fixed proposal distribution parameters was used for the first 500 iterations to initiate the covariance matrix before the adaptive algorithm commenced. Seven chains were then run to ensure 10,000 iterations post-burn-in were achieved.

### **12.1 Rationale for service-specific targets**

For all services (both metropolitan and regional), we included the time series of daily notifications for that service as a calibration target, using a normal distribution for the likelihood function. A normal distribution is preferred because the mapping process for the notifications for each service results in these quantities not being integer-valued.

In addition, we include time series for the following quantities at the state level. Because these quantities are counts, Poisson distributions are used in likelihood calculations:

- Daily new COVID-19 notifications
- Daily new hospital admissions
- Daily new ICU admissions

The daily time series of deaths may be used in future calibrations, but remain too sparse for this purpose at the time of writing.

### **12.2 Assigning targets to services**

Hospital admissions and ICU admissions can be mapped directly to a health service cluster. Health service clusters include all health care (including public hospitals, private, rehab, acute, mental health, etc.) and some metropolitan services have changed service assignment over the years. Mapping was performed as at August 2020. However, for notifications (and deaths where used), mapping was not possible because these events do not necessarily occur within a health service cluster. Therefore, the local government area (LGA) of residence of the person notified or dying is

considered. Each notification and death is split proportionately across the health service clusters to which they would typically present, according to historical data on hospital presentations for each LGA provided by DHHS. (Note that only notifications are considered as calibration targets, although these considerations are relevant to the comparison between data and modelled outputs undertaken for validation purposes.)

### 12.3 Variation of age-specific proportion parameters using “adjuster” parameters

Our parameters included age-specific parameters that were varied up and down together during calibration. These proportion parameters are modified through “adjuster” parameters that are not strictly multipliers, but are rather implemented in such a way as to scale the base parameter value while ensuring that the adjusted parameter remains a proportion (with range zero to one). In each of these cases, the adjuster parameters can be considered as multiplicative factors that are applied to the odds ratio that is equivalent to the baseline proportion to be adjusted. Specifically, the adjusted proportion is equal to:

$$\frac{\text{proportion} \times \text{adjuster}}{\text{proportion} \times (\text{adjuster} - 1) + 1}$$

This approach was applied for the age-specific fraction hospitalised and the age-specific fraction of active cases symptomatic.

### 12.4 Variation of the proportion of patients hospitalised

The modelled proportion of patients hospitalised differs by age group, and is also likely to vary between settings. An adjuster is used to increase or decrease each value for each age group. In the current analysis, the proportion of patients hospitalised is the only quantity adjusted in this way.

## 13 Likelihood function

Likelihood functions are derived from comparing model outputs to target data at each time point nominated for calibration.

The composite likelihood function is given formally as:

$$\prod_t n_t(\theta) h_t(\theta) i_t(\theta) \times \prod_{t,g} n_{t,g}(\theta, \sigma)$$

where  $t$  indexes the date,  $g$  indexes the service,  $n_t$  refers to daily new notifications,  $d_t$  to daily deaths,  $h_t$  to daily new hospitalisations and  $i_t$  to daily new ICU admissions. The contributions

of each state-wide component to the composite likelihood are measured with Poisson distributions (e.g.  $n_t(\theta) = \text{Pois}(\nu_t(\theta))$ , where  $\nu_t(\theta)$  is the number of notifications simulated by the model at date  $t$  under parameter set  $\theta$ ), and normal distributions are used for each  $n_{t,g}$  (because these targets are not integer-valued).  $\sigma$  is the ratio of the peak of each service-specific notification to the corresponding standard deviation of each of the normal distributions used in calculating their contribution to the likelihood. This was included as a calibration parameter to improve calibration efficiency (although results for this parameter are not presented).

## 14 Ordinary differential equations

For the clearest description of the model, we refer the reader to our code repository, because our object-oriented approach to software development is intended to be highly transparent and readable. For those who prefer dynamical systems such as this presented in the form of ordinary differential equations, we present the following.

$$\begin{aligned}
\frac{dS_{a,g,v=1}}{dt} &= -(\lambda_{a,g,v=1}(t)\sigma_a + \zeta)S_{a,g,v=1} \\
\frac{dS_{a,g,v=2}}{dt} &= -(\lambda_{a,g,v=2}(t)\sigma_a + \eta)S_{a,g,v=2} + \zeta S_{a,g,v=1} \\
\frac{dS_{a,g,v=3}}{dt} &= -\lambda_{a,g,v=3}(t)\sigma_a S_{a,g,v=3} + \eta S_{a,g,v=2} \\
\frac{dE_{a,g,q=1,v}}{dt} &= \lambda_{a,g,v}(t)\sigma_a S_{a,g,v} - \alpha E_{a,g,q=1,v} - \chi(t)E_{a,g,q=1,v} \\
\frac{dE_{a,g,q=2,v}}{dt} &= -\alpha E_{a,g,q=2,v} + \chi(t)E_{a,g,q=1,v} \\
\frac{dP_{a,c,g,q,v}}{dt} &= p_{a,c,v}(t)\alpha E_{a,g,q,v} - \nu P_{a,c,g,q,v} \\
\frac{dI_{a,c,g,q,v}}{dt} &= \nu P_{a,c,g,q,v} - \gamma_c I_{a,c,g,q,v} \\
\frac{dL_{a,c,g,q,v}}{dt} &= \gamma_c I_{a,c,g,q,v} - \delta_{a,c} L_{a,c,g,q,v} - \mu_{a,c} L_{a,c,g,q,v} \\
\frac{dR_{a,g,v=1}}{dt} &= \sum_{c,q} \delta_{a,c} L_{a,c,g,q,v} - \zeta R_{a,g,v=1} \\
\frac{dR_{a,g,v=2}}{dt} &= \sum_{c,q} \delta_{a,c} L_{a,c,g,q,v} + \zeta R_{a,g,v=1} - \eta R_{a,g,v=2} \\
\frac{dR_{a,g,v=3}}{dt} &= \sum_{c,q} \delta_{a,c} L_{a,c,g,q,v} + \eta R_{a,g,v=2}
\end{aligned}$$

where

$$\lambda_{a,g,v} = \beta \sum_{g'} \mathbf{G}_{g,g'} \sum_{j,c,v'} \psi_{v'} \frac{\epsilon P_{j,c,g',v'}(t) + \iota_c I_{j,c,g',v'}(t) + \kappa_c L_{j,c,g',v'}(t)}{N_{j,g',v'}(t)} C_{a,j}(t)$$

$$\sum_c p_{a,c,v}(t) = 1, \forall t \in \mathbb{R}$$

$$\chi(t) = \frac{\alpha q(t)u(t)}{1 - q(t)u(t)}$$

$$\mathbf{C}_0 = \mathbf{C}_H + \mathbf{C}_S + \mathbf{C}_W + \mathbf{C}_L$$

$$\mathbf{C}_g(t) = \mathbf{C}_H + s_g(t)^2 \mathbf{C}_S + w_g(t)^2 \mathbf{C}_W + l_g(t)^2 \mathbf{C}_L$$

$$l_g(t) = \frac{re_g(t) + gr_g(t) + pa_g(t) + tr_g(t)}{4}$$

Symbol	Explanation
$S$	Persons susceptible to infection
$E$	Persons in the non-infectious incubation period
$P$	Persons in the incubation period
$I$	Persons in the early active disease period, before isolation or hospitalisation may occur
$L$	Persons in the late active disease period, after isolation or hospitalisation may have occurred
$R$	Persons in the recovered period, from which re-infection cannot occur

## 15 Implementation of the roadmap

The scenario analysis of implementation of the roadmap on schedule is implemented as described in Table ??.

Symbol	Explanation
$t$	Time
$a$	Compartment of age group $a$
$c$	Compartment of clinical stratification $c$
$g$	Compartment of geographical service stratification $g$
$q$	Compartment of tracing stratification $q$
$v$	Compartment of vaccination stratification $v$
$\sigma$	Susceptibility to infection
$\alpha$	Rate of progression from non-infectious to infectious incubation period
$\nu$	Rate of progression from infectious incubation to early active disease
$\gamma$	Rate of progression from early active disease to late active disease
$\mu$	Rate of disease-related death
$\epsilon$	Relative infectiousness of pre-symptomatic compartment
$\iota$	Clinical stratification infectiousness vector for early active compartment
$\kappa$	Clinical stratification infectiousness vector for late active compartments
$\beta$	Probability of infection per contact between an infectious and susceptible individual
$\psi$	Relative infectiousness of vaccination status (unvaccinated, one dose or fully vaccinated)
$\zeta$	Receipt of the first dose of a vaccination schedule
$\eta$	Receipt of the second dose of a vaccination schedule
$j$	Infectious populations
$\rho$	Proportion progressing to each clinical stratification
<b>G</b>	Square matrix of dimensions $9 \times 9$ for nine services, as presented in Table ??

Symbol	Explanation
<b>C</b>	Mixing matrix
<b>H</b>	Household contribution to mixing matrix
<b>W</b>	Workplace contribution to mixing matrix
<b>O</b>	Other locations contribution to mixing matrix
<b>S</b>	Schools contribution to mixing matrix
<i>I</i>	Other locations macrodistancing function of time
<i>w</i>	Function fit to Google mobility data for workplaces
<i>s</i>	Function fit to Google mobility data for schools
<i>re</i>	Function fit to Google mobility data for retail and recreation
<i>gr</i>	Function fit to Google mobility data for grocery and pharmacy
<i>pa</i>	Function fit to Google mobility data for parks
<i>tr</i>	Function fit to Google mobility data for transit stations

Phase	Date	Change	Implementation
A	5 <sup>th</sup> October	VCE Units 3 and 4 on-site (Metropolitan Melbourne)	One thirteenth of 90% remote education on-site
A	18 <sup>th</sup> October	Prep back three days, years 1 and 2 back two days (Metropolitan Melbourne)	2.4 thirteenth of 90% remote education on-site
B	26 <sup>th</sup> October	All students back at least part-time	80% onsite education
B	26 <sup>th</sup> October	Social, recreational, personal care and religious changes	10% return to normal workplace mobility, 20% return to normal other locations mobility
C	5 <sup>th</sup> November	Social, recreational, work, personal care, retail, religious changes	25% return to normal workplace mobility, 40% return to normal other locations mobility
D	19 <sup>th</sup> November	Changes consistent with National Plan	50% return to normal workplace mobility, 60% return to normal other locations mobility

**Table 7: Timeline used to implement Victorian roadmap scenario.** Changes are applied to both Metropolitan and Regional areas except where indicated.