



**Supplemental material supporting “Understanding
COVID-19 dynamics and the effects of interventions in
the Philippines: A mathematical modelling study”**

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Contents

1	Base model construction	3
1.1	Platform for infectious disease dynamics simulation	3
1.2	Base COVID-19 model	3
1.3	Age stratification	4
1.4	Clinical stratification	5
1.5	Hospitalisation	5
1.6	Infectiousness	8
1.7	Application of COVID-19-related death	8
2	Case detection	9
2.1	General approach	9
3	Implementation of non-pharmaceutical interventions	10
3.1	Isolation and quarantine	10
3.2	Community quarantine or “lockdown” measures	10
3.3	School closures/re-openings	11
3.4	Workplace closures	11
3.5	Community-wide movement restriction	12
3.6	Household contacts	12
3.7	Microdistancing	13
4	Parameters	13
4.1	Non-age-stratified parameters	13
4.2	Age-specific parameters	15
5	Calculation of outputs	17
5.1	Incidence	17

5.2	Hospital occupancy	17
5.3	ICU occupancy	17
5.4	Seropositive proportion	17
5.5	COVID-19-related mortality	17
5.6	Notifications	18
6	Calibration	18
6.1	General approach	18
6.2	Likelihood function	18
6.3	Variation of infection fatality rate and symptomatic proportions	18
6.4	Calibration parameters	19
6.5	Calibration targets	20
7	Ordinary differential equations	21
8	Supplemental figures and tables to main text	24
8.1	Supplemental Tables	24
8.2	Supplemental Figures	28

1 Base model construction

1.1 Platform for infectious disease dynamics simulation

We developed a deterministic compartmental model of COVID-19 transmission using the AuTuMN platform, publicly available at <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 tuberculosis dynamics, being an infectious disease whose epidemiology differs markedly by setting, such that considerable flexibility is desirable [1]. 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 for the purpose of this project.

1.2 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 (SEEIIR). 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 infectiousness 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 1).

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 [2].

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. applies during the late disease phase only, see Section 2). 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 1.4).

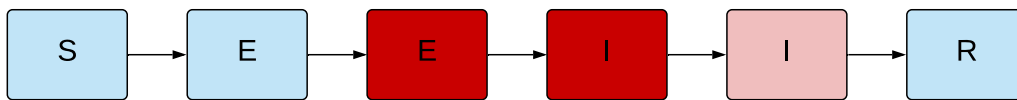


Figure 1 – Unstratified compartmental model structure. S = susceptible, E = exposed, I = active, R = recovered/removed. Depth of pink/red shading indicates the infectiousness of the compartment.

1.3 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 by Prem et al. 2017 [3], who combined survey response data with information on national demographic characteristics to produce age-structured mixing matrices with these age groupings. These are then modified by non-pharmaceutical interventions as described in Section 3. Our modelled age groups were chosen to match these mixing matrices. The automatic demographic features of AuTuMN 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.

1.4 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 2). The proportion of new infectious persons entering stratum 1 (asymptomatic) is age-dependent (as described in Table 4). 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, see Section 2). Of those ever symptomatic (strata 2 to 5), a time-constant but age-specific proportion is considered to be hospitalised (entering strata 4 or 5). Of those hospitalised (entering strata 4 or 5), a fixed proportion was considered to be critically unwell (entering stratum 5, Figure 3).

1.5 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, as indicated in Table 3. 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 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.

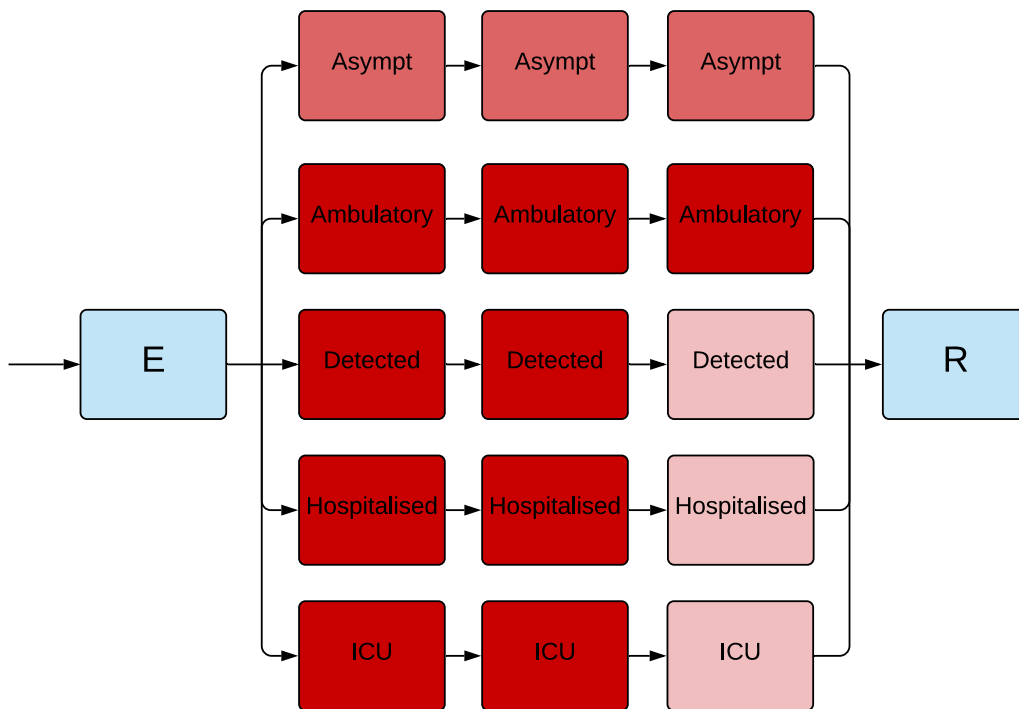


Figure 2 – Illustration of the implementation of the clinical stratification. Depth of pink/red shading indicates the infectiousness of the compartment. Typical parameter values presented, although the infectiousness of asymptomatic persons is varied in calibration.

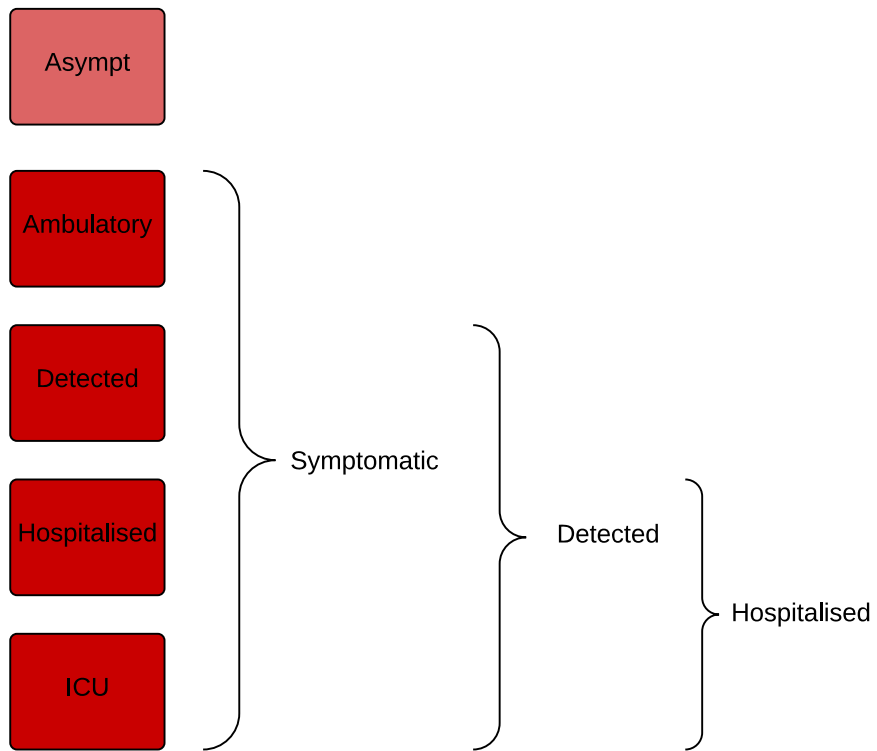


Figure 3 – Illustration of the rationale for the clinical stratification.

1.6 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 both groups). Presymptomatic individuals are presumed to have equivalent infectiousness to those with early active COVID-19.

1.7 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 [4] as indicated in Table 4. We allowed IFRs to vary around the previously published point estimates in order to incorporate uncertainty and to allow the IFRs to differ from the settings in which they were estimated (see Calibration section).

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.

2 Case detection

2.1 General approach

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 31st 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 empirical historical profile of changes

in testing capacity over time.

3 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.

3.1 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 effectively identified through the public health response by any means is determined by the case detection rate as described above.

3.2 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 of Prem et al. [3] are synthetic and do not represent direct observations or reports from surveys (in the case of the 144 countries to which they were extrapolated from observations in the eight “POLYMOD” countries of Western Europe). Although synthetic, the matrices are contextualised to national demographic information, including country-specific data that include household size, workforce participation and school enrolment. Further, the matrices presented are easily machine-readable and appear to be plausible representations of contact structures within these countries.

The matrices also 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.

The corresponding mixing matrix (denoted C_0) is presented using the standard convention that a row

represents the average number of age-specific contacts per day for a contact recipient of a given age-group. In other words, the element $C_{0i,j}$ is the average number of contacts per day that an individual of age-group j makes with individuals of age-group i .

This matrix results from the summation of the four location-specific contact matrices provided by Prem et al.: $C_0 = C_H + C_S + C_W + C_L$, 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.

3.3 School closures/re-openings

Reduced attendance at schools is represented through the function $s(t)$, which represents the proportion of all school 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 and do not fall to zero).

3.4 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 2).

3.5 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 2.

3.6 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 in that they represent the time spent in that location 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.

Prem “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"> • Retail and recreation • Grocery and pharmacy • Parks • Transit stations

Table 2 – Mapping of Google mobility data to contact locations (as defined by Prem et al.)

3.7 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 elements of the mixing matrices by a certain proportion. These time-varying functions multiplicatively scale the location-specific contact rate modifiers $s(t)$, $w(t)$ and $l(t)$.

4 Parameters

4.1 Non-age-stratified parameters

Parameter	Value	Rationale
Incubation period	Calibration parameter, truncated normal distribution, mean 5.5 days	Estimates of the incubation period have included 5.1 days, 5.2 days and 4.8 days [5] [6] [7] [8]. A systematic review [2] found that data are best fitted by a log-normal distribution (mean 5.8 days, CI 5.0 to 6.7, median 5.1 days). Our systematic review [9] found that estimates of the mean incubation period have varied from 3.6 to 7.4 days.
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 [10] and early findings that the incubation period was similar to the serial interval [5]. 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 [11].

Continuation of Table 3

Parameter	Value	Rationale
Active period (regardless of detection/isolation, for clinical strata 1 to 3)	Calibration parameter, truncated normal distribution, mean 8 days	This quantity is difficult to estimate, given that identified cases are typically quarantined. Studies in settings of high case ascertainment and an effective public health response have suggested a duration of greater than 5.5 days [8]. PCR positivity, which may continue for up to two to three weeks from the point of symptom onset [10] [11], is difficult to interpret and does not necessarily indicate infectiousness. Consistent with these findings, the duration infectious for asymptomatic persons has been estimated at 6.5 to 9.5 days [11] (although in our model, this would include the pre-symptomatic infectious period).
Proportion of infectious period before isolation or hospitalisation can occur	0.333	Assumed
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 th October 2020 in Table 6 [12], and similar to the expected mean from earlier reports from ISARIC [13]. 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 [5].
Duration of hospitalisation if not critically unwell (late active sojourn time, stratum 4)	12.8 days	Mean value from the ISARIC cohort, as reported on 4 th October 2020 in Table 6 [12].
ICU duration (late active sojourn time, stratum 5)	10.5 days	Mean duration of stay in ICU/HDU from ISARIC cohort for patients with complete data, as reported on 10 th October 2020 Table 6 [12]. Many other studies reporting on the average duration of ICU stay suffer from right-truncation issues, often estimating 7-10 days length of stay.
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) [12].

Continuation of Table 3

Parameter	Value	Rationale
Relative infectiousness of asymptomatic persons (per unit time with active disease)	0.5	Assumed
Relative infectiousness of persons admitted to hospital or ICU	0.2	Assumed
Relative infectiousness of identified persons in isolation	0.2	Assumed
Proportion of hospitalised patients ever admitted to ICU	0.17	Assumed

Table 3 – 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.

4.2 Age-specific parameters

Age group (years)	Clinical fraction ^a	Relative susceptibility to infection	Infection fatality rate	Proportion of symptomatic patients hospitalised
0 to 4	0.29	0.36	3×10^{-5}	0.0777
5 to 9	0.29	0.36	1×10^{-5}	0.0069
10 to 14	0.21	0.36	1×10^{-5}	0.0034
15 to 19	0.21	1	3×10^{-5}	0.0051
20 to 24	0.27	1	6×10^{-5}	0.0068
25 to 29	0.27	1	1.3×10^{-4}	0.0080
30 to 34	0.33	1	2.4×10^{-4}	0.0124
35 to 39	0.33	1	4.0×10^{-4}	0.0129
40 to 44	0.40	1	7.5×10^{-4}	0.0190
45 to 49	0.40	1	1.21×10^{-3}	0.0331
50 to 54	0.49	1	2.07×10^{-3}	0.0383
55 to 59	0.49	1	3.23×10^{-3}	0.0579
60 to 64	0.63	1	4.56×10^{-3}	0.0617
65 to 69	0.63	1.41	1.075×10^{-2}	0.1030
70 to 74	0.69	1.41	1.674×10^{-2}	1.072
75 and above	0.69	1.41	5.748×10^{-2} , ^b	0.0703
Source/ rationale	Model fitting to age-distribution of early cases in China, Italy, Japan, Singapore, South Korea and Canada taken from upper-left panel of Figure 2b of [14].	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 [15]. ^c	Estimated from pooled analysis of data from 45 countries from Table S3 of O'Driscoll et al [4]. Values consistent with previous estimates using serosurveys performed in Spain [16].	Estimates from the Netherlands as the first wave of infections declined from 4th May to 21st July [17].

Table 4 – Age-stratified parameter values. Age-stratified parameters not varied during calibration, or varied through a common adjuster parameter.

^a Proportion of incident cases developing symptoms.

^b Weighted average of IFR estimates for 70 to 79 and 80 and above age groups.

^c Note the relative magnitude of these values are similar to those estimated by the analysis we use to estimate the age-specific clinical fraction.[14]

5 Calculation of outputs

5.1 Incidence

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

5.2 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.

5.3 ICU occupancy

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

5.4 Seropositive 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.

5.5 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.

5.6 Notifications

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

6 Calibration

6.1 General approach

The model was calibrated using an adaptive Metropolis (AM) algorithm. In particular, we used the algorithm based on adaptive Gaussian proposal functions proposed by Haario *et al.* to sample parameters from their posterior distributions [18]. We ran seven independent AM chains and combined the samples of the seven chains to project epidemic trajectories over time. The definitions of the prior distributions and the likelihood are detailed as follows.

6.2 Likelihood function

Likelihood functions are derived from comparing model outputs to target data at each time point nominated for calibration. The dispersion parameters of these distribution are included as calibration parameters and varied during the calibration approach to improve calibration efficiency.

6.3 Variation of infection fatality rate and symptomatic proportions

Whether age-specific infection fatality rates (IFRs) are significantly different in low- and middle-income settings from those in high-income settings remains highly uncertain. For this application to the Philippines, we adjust the IFRs described above according to a factor that modifies the age-specific IFR for each age group relative to the baseline values described in the previous section, allowing them to vary from those reported up to two-fold those values. The age-specific IFRs used in the model are obtained from:

$$IRF_i^* = \frac{IFR_i \times \omega}{IFR_i(\omega - 1) + 1},$$

where IRF_i^* is the modelled IFR for age group i , IFR_i is the point estimate reported by O'Driscoll *et al.* for the IFR of age group i [4], and ω is the uncertainty adjuster varied during model calibration.

Similarly, we incorporated uncertainty around the age-specific proportions of symptomatic individuals by applying an uncertainty adjuster:

$$s_i^* = \frac{s_i \times \gamma}{s_i(\gamma - 1) + 1},$$

where s_i^* is the modelled symptomatic proportion for age group i , s_i is the point estimate reported by Davies *et al.* for the IFR of age group i [14], and γ is the associated uncertainty adjuster varied during model calibration.

6.4 Calibration parameters

Parameter name	Distribution type	Distribution parameters
Incubation period	Truncated normal	Mean 5.5 days, standard deviation 0.97 days, truncation <1 day
Infectious period (for clinical strata 1 to 3)	Truncated normal	Mean 6.5 days, standard deviation 0.77 days, truncation <4 days
Risk of infection per contact	Uniform	0.03 to 0.05
Infection fatality rate adjuster (ω)	Uniform	Range 1.8 to 2.28
Proportion of symptomatic cases that would be detected with a daily per capita testing rate of one test per ten thousand population	Uniform	Range 0.02 to 0.15

Continuation of Table 5

Parameter name	Distribution type	Distribution parameters
Infectious seed	Uniform	Range 10 to 100
Maximal effect of Minimum Health Standards	Uniform	0.1 to 0.6
Adjuster applied to age-specific proportion of infections leading to symptoms (“Clinical fraction”)	Uniform	0.2 to 2
Adjuster applied to age-specific proportion of symptomatic infections leading to hospitalisations	Uniform	0.2 to 2
Adjuster applied to age-specific infection fatality rate	Uniform	0.2 to 2

Table 5 – Epidemiological calibration parameters.

6.5 Calibration targets

We calibrated each of the Philippines models to the daily notification rate (with seven-day moving average smoothing) observed nationally or for the sub-region simulated over time.

For ICU occupancy, we only considered the most recent estimate of ICU occupancy and did not calibrate to multiple time points for this indicator. Similarly for cumulative infection-related deaths, we only calibrated to the most recent data time point available.

7 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 those presented in the form of ordinary differential equations, we present the following.

$$\begin{aligned}\frac{dS_a}{dt} &= -\lambda_a(t) \times \sigma_a \times S_a \\ \frac{dE_a}{dt} &= \lambda_a(t) \times \sigma_a \times S_a - \alpha E_a \\ \frac{dP_{a,c}}{dt} &= p_{a,c}(t) \times \alpha E_a - \nu P_{a,c} \\ \frac{dI_{a,c}}{dt} &= \nu P_{a,c} - \gamma_c I_{a,c} \\ \frac{dL_{a,c}}{dt} &= \gamma_c I_{a,c} - \delta_{a,c} L_{a,c} - \mu_{a,c} L_{a,c} \\ \frac{dR_a}{dt} &= \sum_c \delta_{a,c} L_{a,c}\end{aligned}$$

where

$$\begin{aligned}\lambda_a &= \beta \left[\sum_{j,c} \frac{\varepsilon \times P_j}{N_j} \times C_{a,j}(t) + \sum_{j,c} \frac{I_{j,c} \times \iota_c + L_{j,c} \times \kappa_c}{N_j} \times C_{a,j}(t) \right] \\ \sum_c p_{a,c}(t) &= 1, \forall t \in \mathbb{R} \\ \mathbf{C}_0 &= \mathbf{C}_H + \mathbf{C}_S + \mathbf{C}_W + \mathbf{C}_L \\ \mathbf{C}(t) &= h(t) \times \mathbf{C}_H + s(t) \times \mathbf{C}_S + w(t) \times \mathbf{C}_W + l(t) \times \mathbf{C}_L \\ l(t) &= \frac{re(t) + gr(t) + pa(t) + tr(t)}{4}\end{aligned}$$

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

Symbol	Explanation
t	Time
a	Compartment of age group a
c	Compartment of clinical stratification c
σ	Relative susceptibility to infection
α	Rate of progression from non-infectious to infectious incubation period
ν	Rate of progression from infectious incubation to early active disease
γ	Rate of progression from early active disease to late active disease
μ	Rate of disease-related death
ε	Relative infectiousness of pre-symptomatic compartment
ι	Clinical stratification infectiousness vector for early active compartment
κ	Clinical stratification infectiousness vector for late active compartments
β	Probability of infection per contact between an infectious and susceptible individual
j	Infectious populations
p	Proportion progressing to each clinical stratification

Symbol	Explanation
C	Mixing matrix
H	Household contribution to mixing matrix
W	Workplace contribution to mixing matrix
O	Other locations contribution to mixing matrix
S	Schools contribution to mixing matrix
<i>l</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

8 Supplemental figures and tables to main text

8.1 Supplemental Tables

Table 6 – Laboratory testing facilities by region.

Facility Name	Region
Batangas Medical Center GeneXpert Laboratory	Calabarzon
Daniel O. Mercado Medical Center	Calabarzon
De La Salle Medical and Health Sciences Institute	Calabarzon
Greencity Medical Center	Calabarzon
Lucena United Doctors Hospital and Medical Center	Calabarzon
Mary Mediatrix Medical Center	Calabarzon
Ospital ng Imus	Calabarzon
Qualimed Hospital Sta. Rosa	Calabarzon
San Pablo College Medical Center	Calabarzon
San Pablo District Hospital	Calabarzon
UPLB Covid-19 Molecular Laboratory	Calabarzon
Allegiant Regional Care Hospital	Central Visayas
Cebu Doctors University Hospital Inc	Central Visayas
CebuTBReferenceLaboratory-MolecularFacilityforCOVID-19Testing	Central Visayas
Chong Hua Hospital	Central Visayas
Governor Celestino Gallares Memorial Medical Center	Central Visayas

Facility Name	Region
Prime Care Alpha Covid-19 Testing Laboratory	Central Visayas
University of Cebu Medical Center	Central Visayas
Vicente Sotto Memorial Medical Center (VSMMC)	Central Visayas
Amang Rodriguez Memorial Center GeneXpert Laboratory	National Capital Region
Asian Hospital and Medical Center	National Capital Region
Chinese General Hospital	National Capital Region
De Los Santos Medical Center	National Capital Region
Dr. Jose N. Rodriguez Memorial Hospital and Sanitarium (TALA) GeneXpert Laboratory	National Capital Region
Dr. Jose N. Rodriguez Memorial Hospital and Sanitarium (TALA) RT PCR	National Capital Region
Fe del Mundo Medical center	National Capital Region
Hi-Precision Diagnostics (QC)	National Capital Region
Lung Center of the Philippines (LCP)	National Capital Region
Lung Center of the Philippines GeneXpert Laboratory	National Capital Region
Makati Medical Center (MMC)	National Capital Region
Marikina Molecular Diagnostics Laboratory (MMDL)	National Capital Region
National Kidney and Transplant Institute	National Capital Region
National Kidney and Transplant Institute GeneXpert Laboratory	National Capital Region
Philippine Children's Medical Center	National Capital Region

Facility Name	Region
Philippine Heart Center GeneXpert Laboratory	National Capital Region
SafeguardDNADiagnosticsInc	National Capital Region
San Miguel Foundation Testing Laboratory	National Capital Region
Singapore Diagnostics	National Capital Region
St. Luke's Medical Center - BGC (HB) GeneXpert Laboratory	National Capital Region
St. Luke's Medical Center - BGC (SLMC-BGC)	National Capital Region
St. Luke's Medical Center - Quezon City (SLMC-QC)	National Capital Region
Sta. Ana Hospital - Closed System Molecular Laboratory (GeneXpert)	National Capital Region
The Medical City (TMC)	National Capital Region
Tondo Medical Center GeneXpert Laboratory	National Capital Region
Tropical Disease Foundation	National Capital Region
University of Perpetual Help DALTA Medical Center Inc	National Capital Region
UP-PGH Molecular Laboratory	National Capital Region
UP National Institutes of Health (UP-NIH)	National Capital Region
UP Philippine Genome Center	National Capital Region
Veteran Memorial Medical Center	National Capital Region
Victoriano Luna - AFRIMS	National Capital Region

8.2 Supplemental Figures

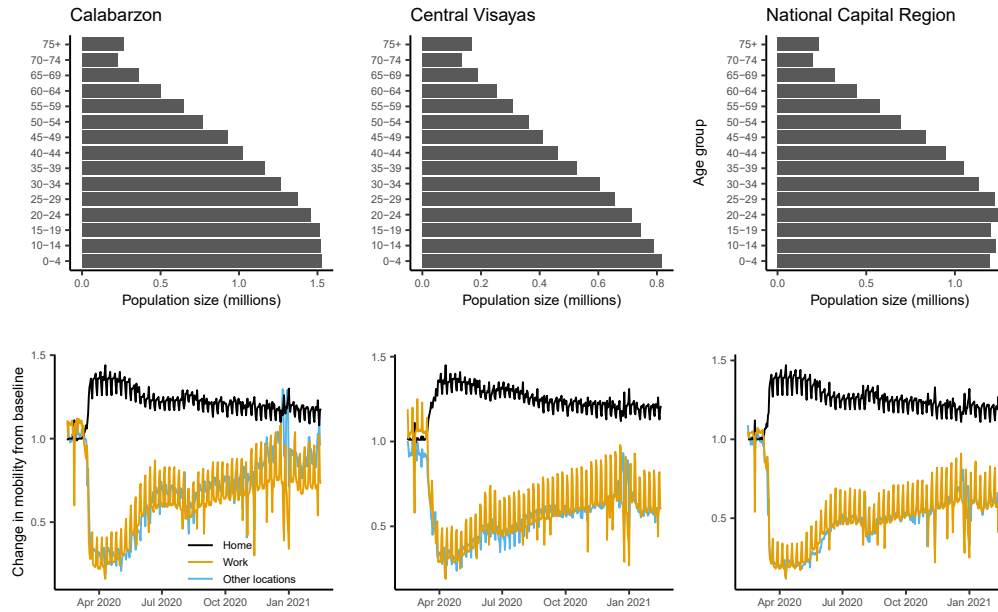


Figure 4 – Population size and mobility included in the age-structured COVID-19 for three regions of the Philippines. Starting population age distribution (top row) and community quarantine driven mobility adjustments applied to the mixing matrices (bottom row) for Calabarzon (left), Central Visayas (middle), and the National Capital Region (right). Other locations in the mobility plots include retail and recreation, supermarket and pharmacy, parks, and public transport.

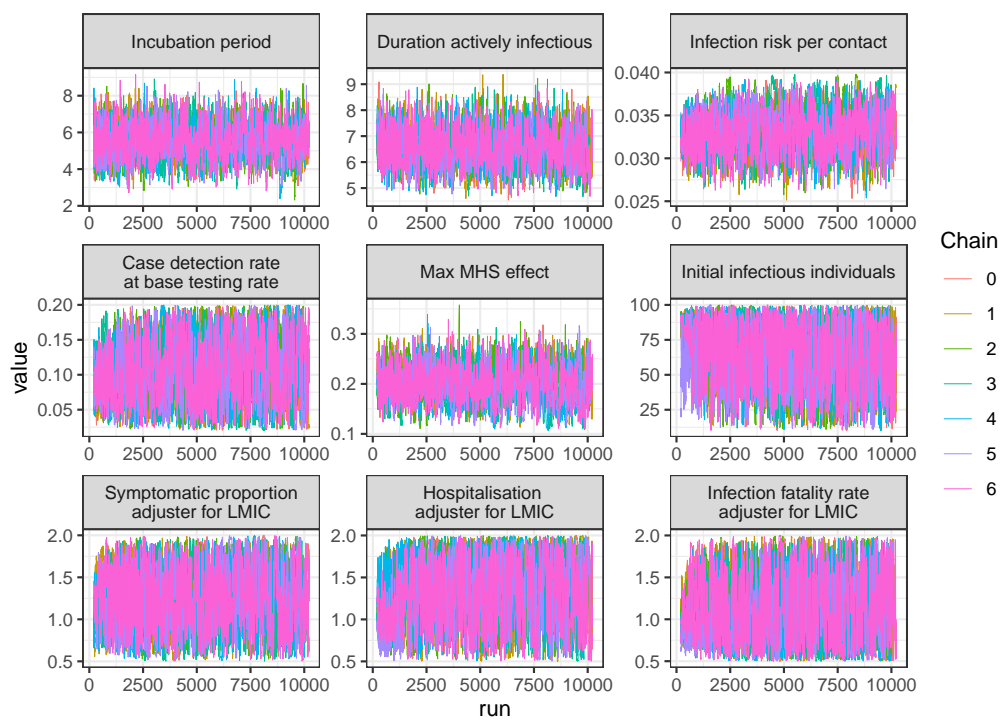


Figure 5 – Trace plots for parameter estimates for the Philippines national model. The seven chains overlap for each parameter, indicating model convergence.

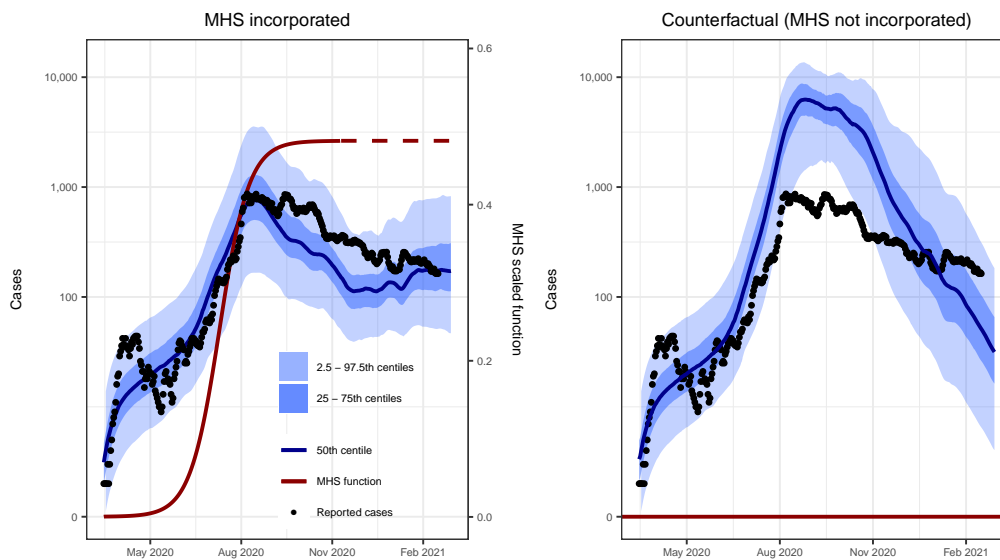


Figure 6 – Comparison of models that included and did not include Minimum Health Standards (MHS) with daily confirmed cases for Calabarzon. We calibrated the Calabarzon model to daily confirmed cases (black dots; same in both plots), which included MHS (left) and ran a counterfactual scenario that did not include MHS (right). The red curve represents the effect of MHS (i.e., reduced transmission risk per contact) in the model through time. The MHS effect value is squared in the model to account for the reduction in the probability of an infected person passing on the infection and the probability of a contact being infected, prior to adjustment of each cell of the mixing matrix.

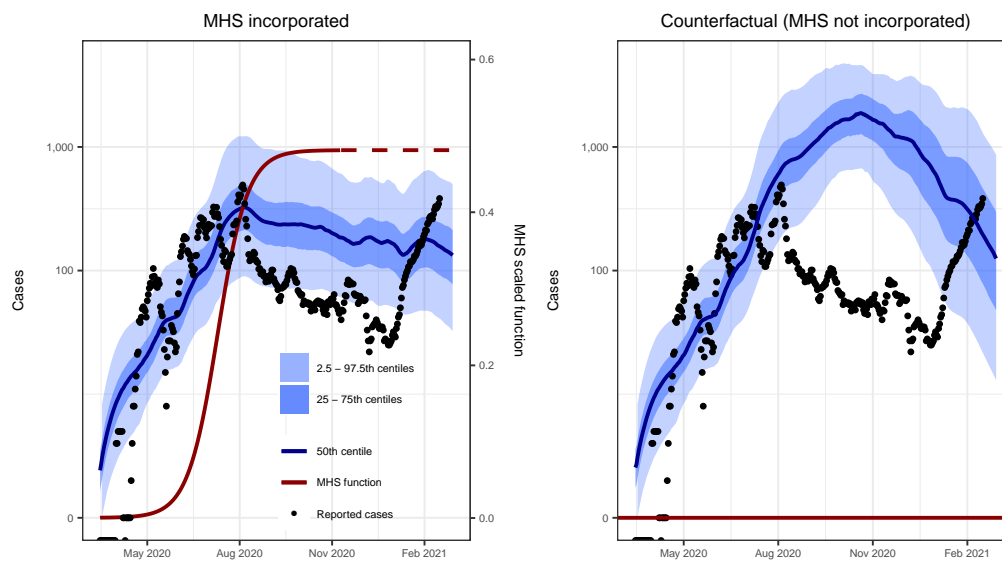


Figure 7 – Comparison of models that included and did not include Minimum Health Standards (MHS) with daily confirmed cases for Central Visayas. We calibrated the Central Visayas model to daily confirmed cases (black dots; same in both plots), which included MHS (left) and ran a counterfactual scenario that did not include MHS (right). The red curve represents the effect of MHS (i.e., reduced transmission risk per contact) in the model through time. The MHS effect value is squared in the model to account for the reduction in the probability of an infected person passing on the infection and the probability of a contact being infected, prior to adjustment of each cell of the mixing matrix. The recent uptick in cases could be due to a new SARS-CoV-2 Variant of Concern.

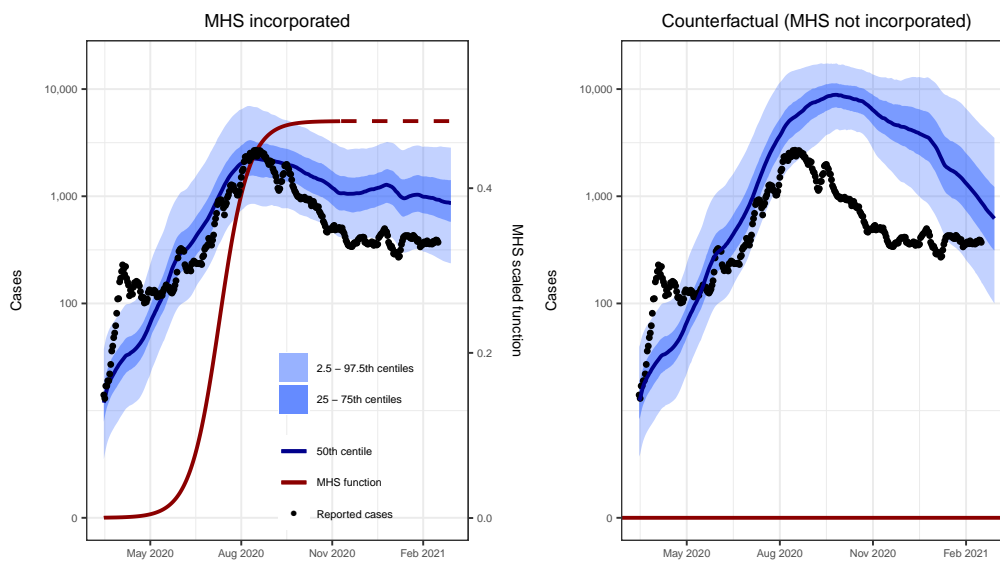


Figure 8 – Comparison of models that included and did not include Minimum Health Standards (MHS) with daily confirmed cases for the National Capital Region. We calibrated the National Capital Region model to daily confirmed cases (black dots; same in both plots), which included MHS (left) and ran a counterfactual scenario that did not include MHS (right). The red curve represents the effect of MHS (i.e., reduced transmission risk per contact) in the model through time. The MHS effect value is squared in the model to account for the reduction in the probability of an infected person passing on the infection and the probability of a contact being infected, prior to adjustment of each cell of the mixing matrix.

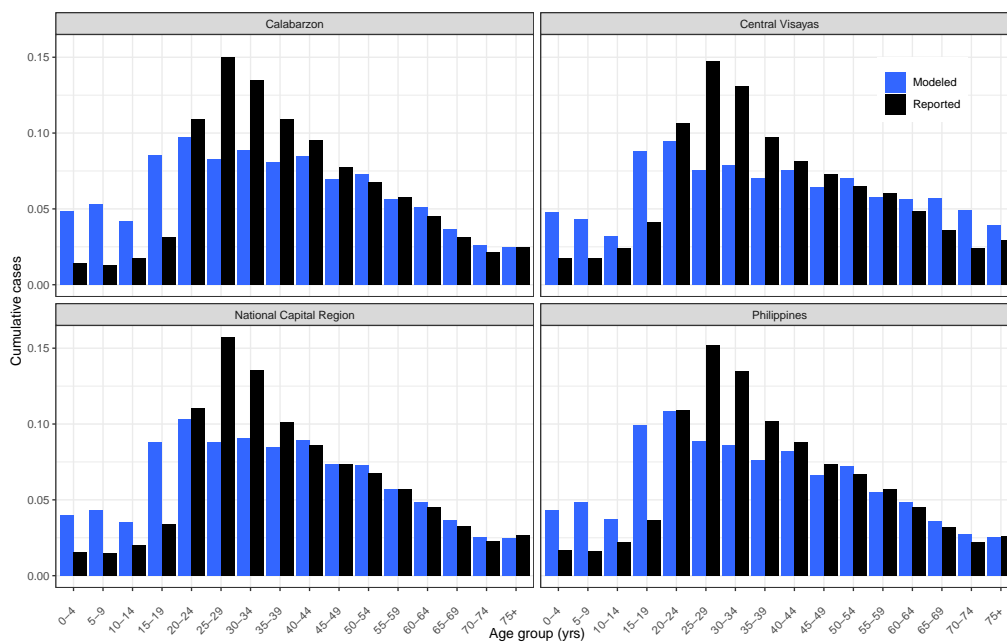


Figure 9 – Proportional modeled (blue bars) versus reported (black bars) cumulative cases by age group. The model over predicted cumulative cases for young age groups and under predicted cumulative cases for middle aged groups, with good fit for age groups over 40 years. There were 2,174 reported cases excluded from these plots because there was no age recorded.

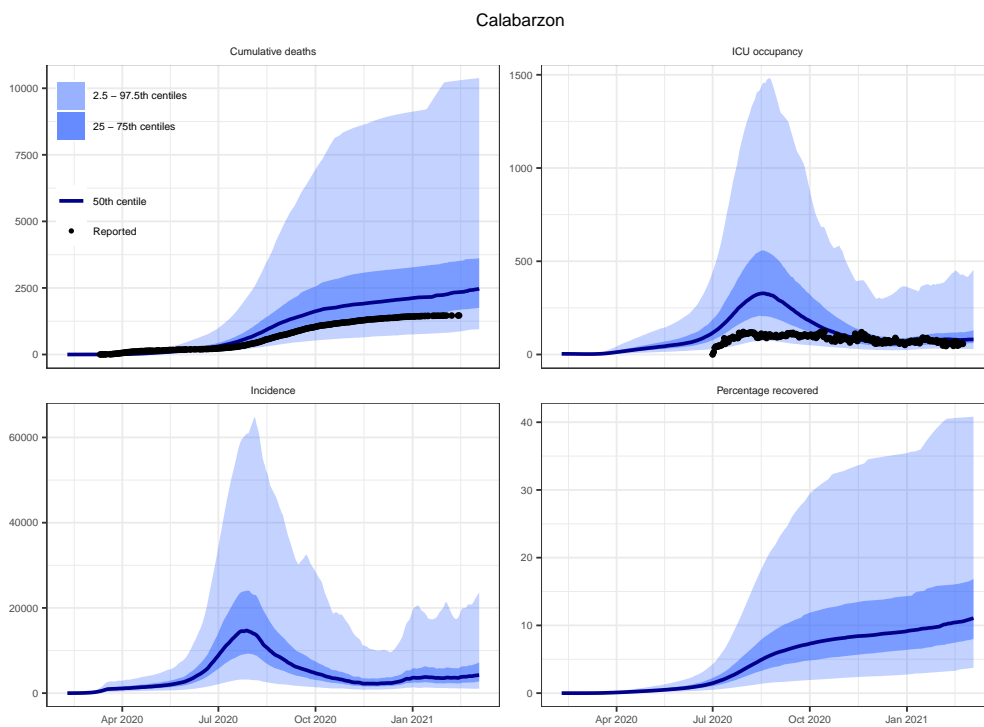


Figure 10 – Model estimated epidemic indices from the calibrated Calabarzon model. Modeled median cumulative deaths, ICU occupancy, incidence, and percentage of the population recovered from COVID-19 (blue line) with shaded areas for 25th to 75th centile (dark blue) and 2.5th to 97.5th centile (light blue) and overlaid with reported cumulative deaths and ICU occupancy (black dots).

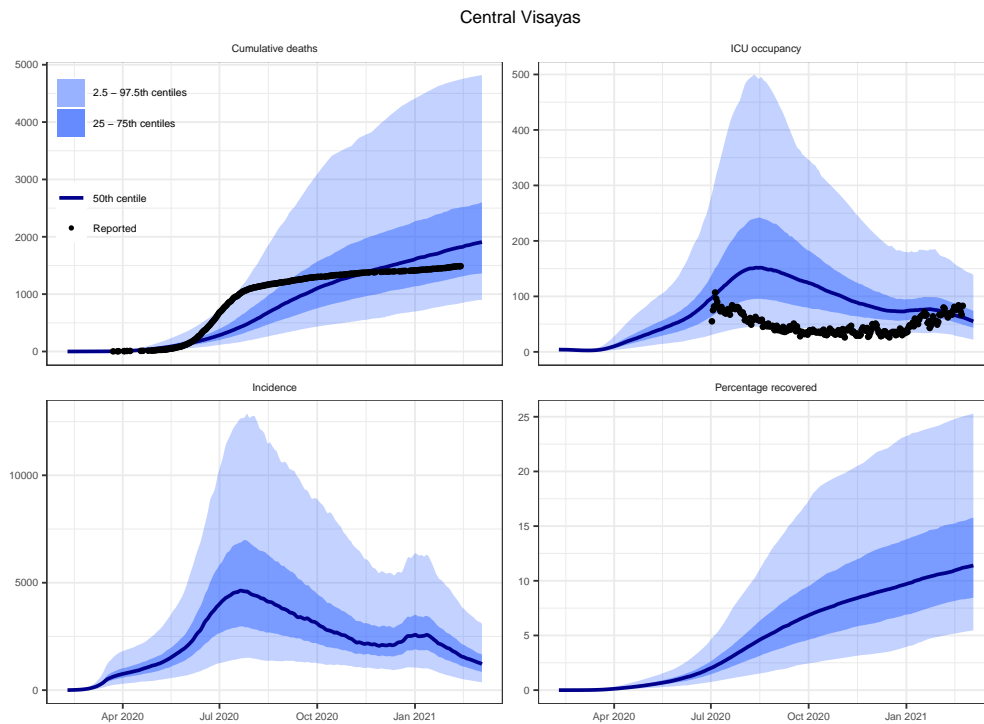


Figure 11 – Model estimated epidemic indices from the calibrated Central Visayas model. Modeled median cumulative deaths, ICU occupancy, incidence, and percentage of the population recovered from COVID-19 (blue line) with shaded areas for 25th to 75th centile (dark blue) and 2.5th to 97.5th centile (light blue) and overlaid with reported cumulative deaths and ICU occupancy (black dots).

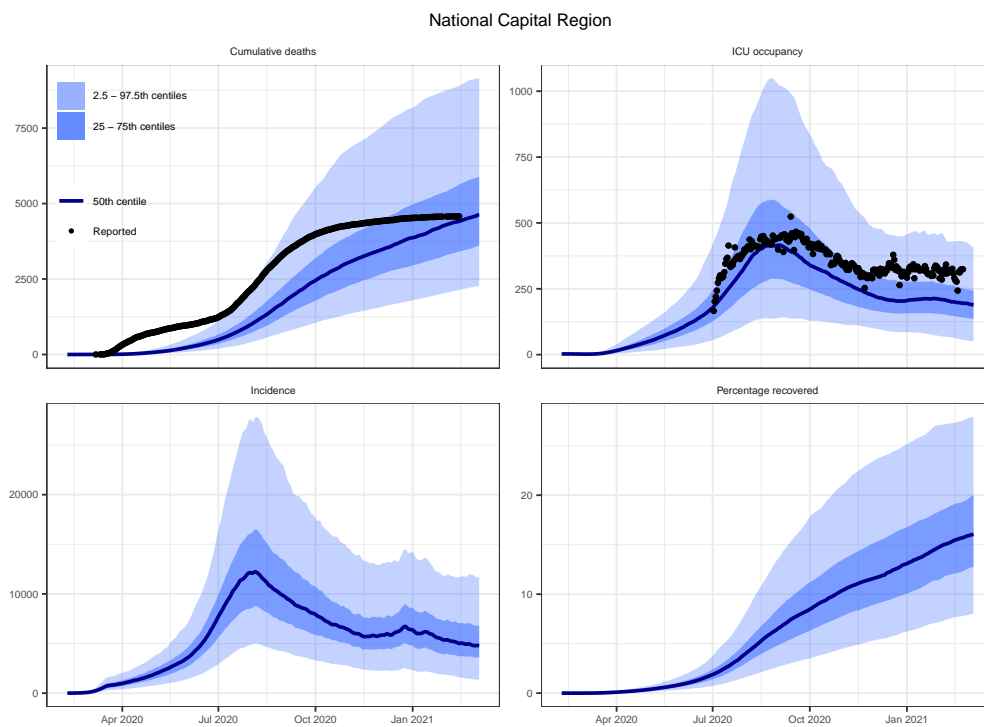


Figure 12 – Model estimated epidemic indices from the calibrated National Capital Region model. Modeled median cumulative deaths, ICU occupancy, incidence, and percentage of the population recovered from COVID-19 (blue line) with shaded areas for 25th to 75th centile (dark blue) and 2.5th to 97.5th centile (light blue) and overlaid with reported cumulative deaths and ICU occupancy (black dots).

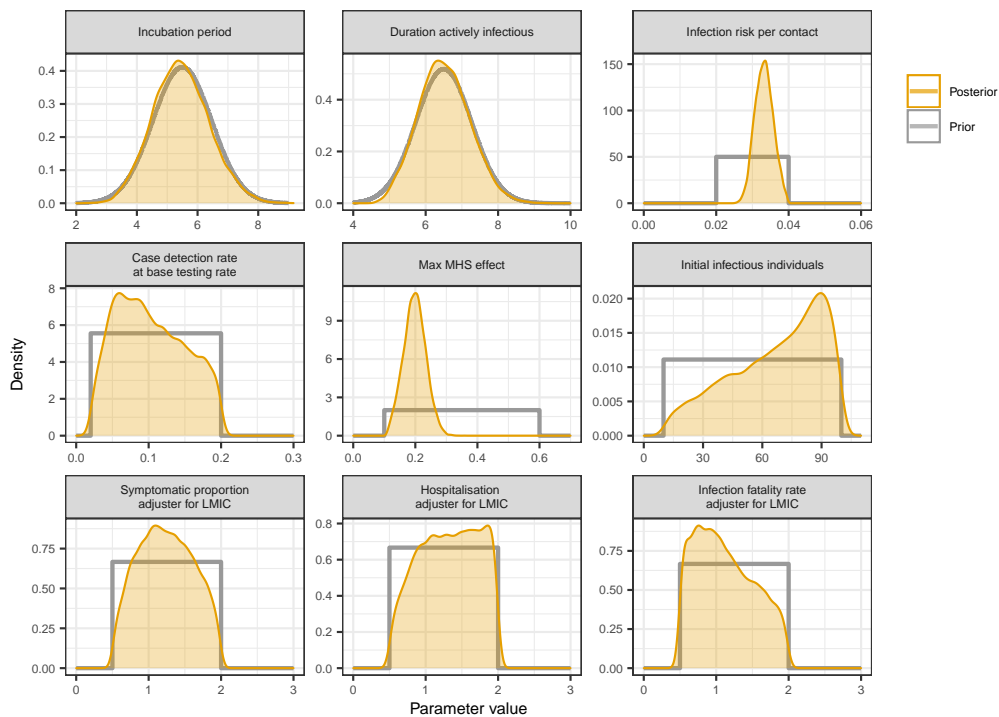


Figure 13 – Prior distributions overlaid with posterior density plots for epidemiological parameters for the Philippines national model. MHS refers to Minimum Health Standards. All parameters with the term “adjuster” allow for modification to the best estimates from the literature (i.e., the priors). The parameter value of the posterior provides the odds ratio used in the model to adjust the odds of an event. Note that the posterior distributions do not exceed the priors even though they appear to do so on some plots because they are plotted as densities.

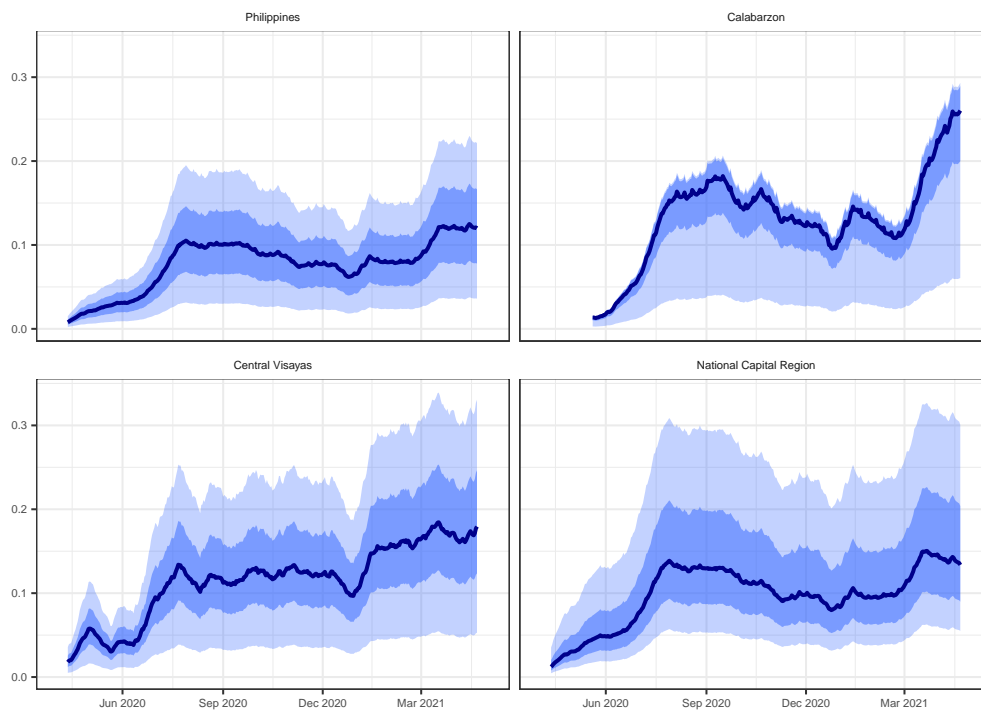


Figure 14 – Model estimated case detection rates varied across regions and through time. We derived values for the symptomatic cases detected through time from the daily number of tests performed, the population size, and the calibrated parameter representing the value of the case detection rate given a testing rate of one test per 10,000 persons per day. We provide a list of laboratory facilities that conducted tests in the three regional models in supplemental Table 6.

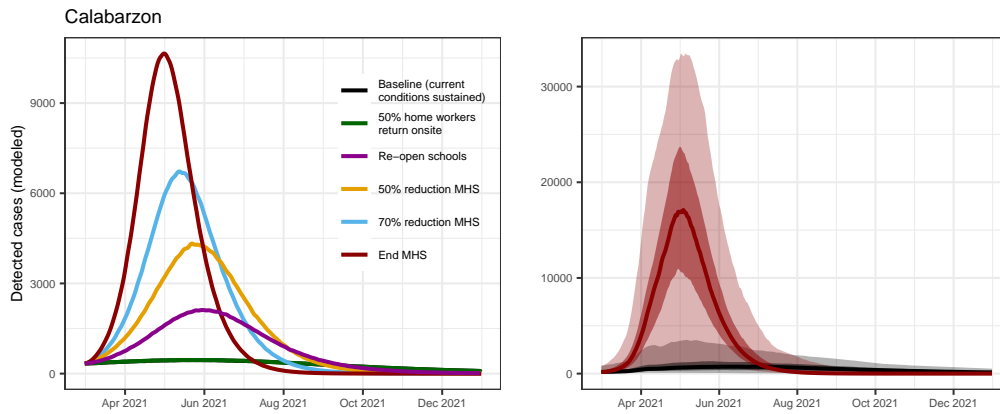


Figure 15 – Epidemic scenario projections for Calabarzon. Median estimates of daily confirmed cases expected under different policy changes (left). Median estimates of daily confirmed cases (lines) with 25th to 75th centiles (dark shading) and 2.5th to 97.5th centiles (light shading) for the baseline scenario (where current conditions are carried forward) and for the scenario where MHS policy ends. Note the different y-axes on each plot.

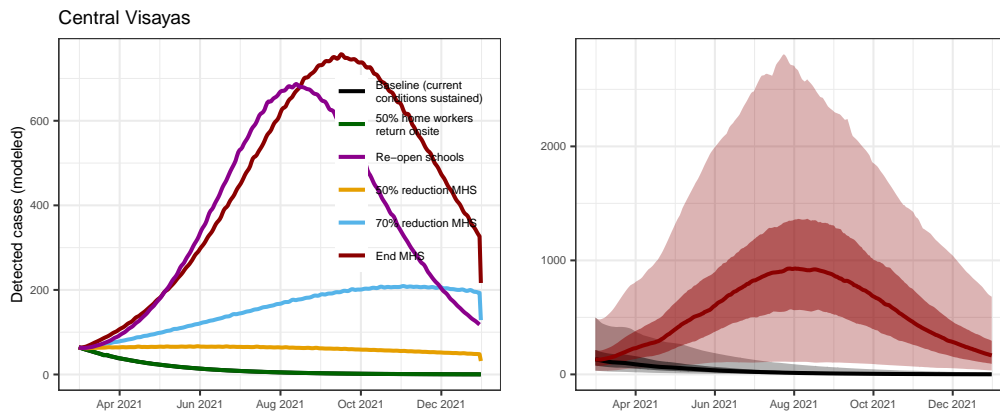


Figure 16 – Epidemic scenario projections for Central Visayas. Median estimates of daily confirmed cases expected under different policy changes (left). Median estimates of daily confirmed cases (lines) with 25th to 75th centiles (dark shading) and 2.5th to 97.5th centiles (light shading) for the baseline scenario (where current conditions are carried forward) and for the scenario where MHS policy ends. Note the different y-axes on each plot.

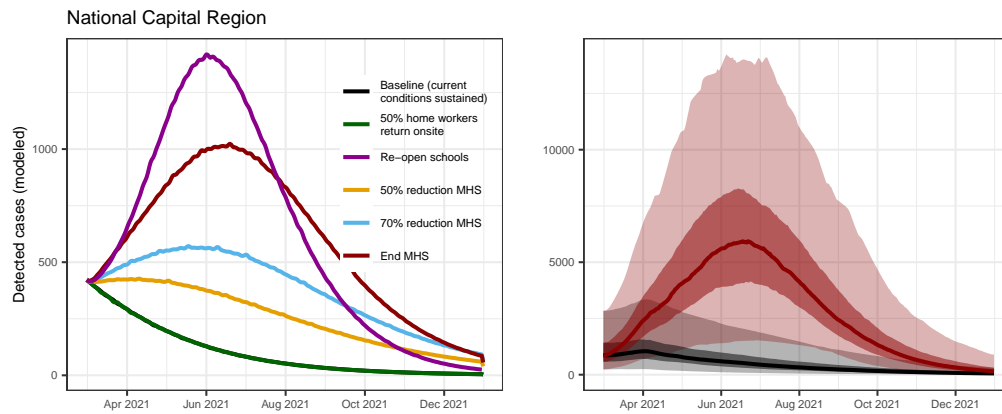


Figure 17 – Epidemic scenario projections for the National Capital Region. Median estimates of daily confirmed cases expected under different policy changes (left). Median estimates of daily confirmed cases (lines) with 25th to 75th centiles (dark shading) and 2.5th to 97.5th centiles (light shading) for the baseline scenario (where current conditions are carried forward) and for the scenario where MHS policy ends. Note the different y-axes on each plot.

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