Modelling transmission and control of the COVID-19 pandemic in Australia

Sheryl L. Chang[1], Nathan Harding[1], Cameron Zachreson[1], Oliver M. Cliff[1], and Mikhail Prokopenko[1,2,*]

[1]   *Centre for Complex Systems, Faculty of Engineering, University of Sydney, Sydney, NSW 2006, Australia*
[2]   *Marie Bashir Institute for Infectious Diseases and Biosecurity, University of Sydney, Westmead, NSW 2145, Australia*
[*]   *Corresponding author: mikhail.prokopenko@sydney.edu.au*

Abstract

In this paper we develop an agent-based model for a fine-grained computational simulation of the ongoing COVID-19 pandemic in Australia. This model is calibrated to reproduce several characteristics of COVID-19 transmission, accounting for its reproductive number, the length of incubation and generation periods, age-dependent attack rates, and the growth rate of cumulative incidence during a sustained and unmitigated local transmission. An important calibration outcome is the age-dependent fraction of symptomatic cases, with this fraction for children found to be one-fifth of such fraction for adults. We then apply the model to compare several intervention strategies, including restrictions on international air travel, case isolation, social distancing with varying levels of compliance, and school closures. School closures are not found to bring decisive benefits. We report an important transition across the levels of social distancing compliance, in the range between 70% and 80% levels. This suggests that a compliance of below 70% is unlikely to succeed for any duration of social distancing, while a compliance at the 90% level is likely to control the disease within 13–14 weeks, when coupled with effective case isolation and international travel restrictions.

Key words: COVID-19, coronavirus, SARS-CoV-2, epidemics, pandemics, interventions, social distancing, mitigation, suppression, computational epidemiology, agent-based modelling

1. Introduction

The 2019-2020 coronavirus pandemic is an ongoing pandemic of coronavirus disease 2019 (COVID-19), caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2). The first outbreak, which originated in December 2019 in Wuhan, the capital of Hubei province, and rapidly spread to the rest of Hubei and all other provinces in China, has been largely eradicated within mainland China by mid- to late March 2020, having generated more than 81,000 cases (cumulative incidence on 20 March 2020 [1]). This was largely due to intense quarantine and social distancing measures, including isolation of detected cases, tracing and management of their close contacts, closures of potential zoonotic sources of SARS-CoV-2, strict traffic restrictions and quarantine on the level of entire provinces (including suspension of public transportation, closures of airports, railway stations, and highways within cities), cancellation of mass gathering activities, and other measures aimed to reduce transmission of the infection [2, 3, 4].

Despite the unprecedented and robust prevention and control measures, the spread of COVID-19 was not contained to China, and the disease spread to other countries. The epidemic has been recognised by the World Health Organization (WHO) as a public health emergency of international concern on 31 January 2020, and on 11 March 2020 the WHO declared the outbreak a pandemic [5]. As of 21 March 2020, over 285,000 cases have been confirmed worldwide, causing more than 11,500 deaths [6, 7]. The disease established a sustained local transmission in many countries around the globe, with the number of confirmed cases exceeding or approaching 10,000 in several nations. More than 180 countries and territories have been affected, including Italy (47,021 cases), Spain (24,926), Iran (20,610), the USA (20,227), Germany (20,099), France (12,612), South Korea (8,799) as the top eight affected nations [8]. The cumulative incidence, incidence, and the growth rate of cumulative incidence are traced for these countries, as well as Australia, in A, see Fig. 8, 9 and 10 respectively.

The scale of the COVID-19 pandemic has grown several orders of magnitude in a matter of weeks, from hundreds to thousands to tens of thousands, with the rate of these transitions varying across countries. Of particular interest to our study is the time periods when the epidemics are sustained locally in these countries, but before the effects of adopted intervention strategies are fully felt. One immediate observation is that during this period, the growth rate of cumulative incidence in many of the traced national epidemics is averaging within the range between 0.2 and 0.3, that is, there are 20% to 30% daily increases in new cases on average. This is particularly evident for Spain, France, and Germany (Fig. 9), as well as China, Iran and Italy (Fig. 8). Average estimates like this may help to reduce uncertainty around key epidemiological parameters which are required to calibrate disease transmission models, before investigating possible effects of various intervention policies.

While worldwide public health emergencies have been declared and mitigated in the past—e.g., the "swine flu" pandemic in 2009 [9, 10, 11, 12]—the scale of socio-economic disruptions caused by the unfolding COVID-19 pandemic is unparalleled in recent history. Effects of the COVID-19 pandemic have quickly spilled over from the healthcare sector into international trade, tourism, travel, energy and finance sectors, causing a panic in the equity markets worldwide [13]. Australia is beginning to feel most of these effects, with the number of confirmed COVID-19 cases crossing 1,000 on 21 March 2020 and a cumulative incidence growth rate consistently above 20%. If the pandemic continues to follow these trends in Australia, the population is likely to experience the same devastating growth as previously seen in other COVID-19 affected nations. In an effort to mitigate this damage, there is an ongoing debate on the utility of specific interventions (e.g., school closures), the low compliance with social distancing measures (e.g., reduction of mass gatherings), and the optimal combination of particular health intervention options balanced against social and economic ramifications, and restrictions on civil liberties. A rigorous and unbiased evaluation of available options is urgently required, and this study aims to provide a timely input to the pandemic response planning in Australia. We quantitatively evaluate and compare several mitigation and suppression measure, using a high-resolution individual-based computational model calibrated to key characteristics of COVID-19 pandemics. In particular, this comparative analysis identifies minimal levels of social distancing compliance required for controlling COVID-19 spread in Australia in the near future, as well as a trade-off between these levels and duration of the interventions. More precisely, our simulations suggest that, without a 80–90% compliance with social distancing strategies, the epidemic will not be effectively controlled.

2. Technical preliminaries and model calibration

In this paper, we present results of the high-resolution (individual-based) pandemic modelling in Australia, using a modified and extended agent-based model, AceMod, previously developed and validated for simulations of pandemic influenza in Australia [14, 15, 16, 17]. AceMod, the *Australian Census-based Epidemic Model*, employs a discrete-time and stochastic agent-based model to investigate complex outbreak scenarios across the nation over time. Stochastic agent-based discrete-time simulation models such as these have been established as useful tools for tracing fine-grained effects of heterogeneous intervention policies in diverse epidemic and pandemic settings [18, 19, 10, 20, 9, 21, 22, 23, 24, 25], including for policy advice currently in place in the USA and the UK [26].

The AceMod simulator comprises over 24 million software agents, each with attributes of an anonymous individual (e.g., age, gender, occupation, susceptibility and immunity to diseases), as well as contact rates within different social contexts (households, household clusters, local neighbourhoods, schools, classrooms, workplaces). The set of generated agents captures average characteristics of the real population, e.g., AceMod is calibrated to the Australian Census data (2016) with respect to key demographic statistics. Potential interactions between spatially distributed agents are represented using data on mobility in terms of commuting patterns (work, study and other activities), adjusted to increase precision and fidelity of commute networks [27]. Each simulation scenario runs in 12-hour cycles ("day" and "night") over the 196 days (28 weeks) of an epidemic, and agents interact across distinct social mixing groups depending on the cycle, for example, in working groups and/or classrooms during a "day" cycle, and their households, household clusters, and local communities during the "night" cycle. The interactions result in transmission of the disease from infectious to susceptible individuals: given the contact and transmission rates, the simulation

2

computes and updates agents' states over time, starting from initial infections, seeded in international airports around Australia [14, 15].

Simulating disease transmission in AceMod requires both (i) specifics of local transmission dynamics, dependent on individual health characteristics of the agents, such as susceptibility and immunity to disease, driven by their transmission and contact rates across different social contexts; and (ii) a natural disease history model for COVID-19, i.e., the infectivity profile from the onset of infection, over an incubation period, to the peak of infectivity, to recovery, for a single symptomatic or asymptomatic individual. It is precisely this part of the model which demands a careful study and calibration to available estimates of key transmission characteristics of COVID-19 spread.

Despite several similarities with influenza, COVID-19 has a number of notable differences, specifically in relation to transmissions across children, its reproductive number R_0, incubation and generation periods, proportion of symptomatic to asymptomatic cases [28, 29, 30], the infectivity of the asymptomatic and presymptomatic individuals, etc. (see Appendix B). While uncertainty around the reproductive number R_0, the incubation and generation periods, as well as the age-dependent attack rates of the disease, have been somewhat reduced [3, 4, 31], there is still an ongoing effort in estimating the extent to which people without symptoms, or exhibiting only mild symptoms, might contribute to the spread of the coronavirus [32]. Furthermore, the question whether the ratio of symptomatic to total cases is constant across age groups, especially children, has not been explored in studies to date, remaining another critical unknown.

Thus, our first technical objective was to calibrate the AceMod model for specifics of COVID-19 pandemic, in order to determine key disease transmission parameters of AceMod, so that the resultant dynamics concur with known estimates. In particular, we aimed to stay within a range of the reproductive number (the number of secondary cases arising from a typical primary case early in the epidemic) $R_0 = [2.0, 2.5]$, which has been reported by the WHO-China Joint Mission on Coronavirus Disease 2019 [3]. Several recent studies estimated that before travel restrictions were introduced in Wuhan on 23 January 2020, the median daily reproduction number R_0 in Wuhan was 2.35, with 95% confidence interval of $(1.15 - 4.77)$ [33]. An agent-based model of the Imperial College COVID-19 Response Team used a baseline assumption that $R_0 = 2.4$, while examining values between 2.0 and 2.6 [26].

In our model, R_0 was investigated in the range between 1.6 and 2.8, by varying a scaling factor κ responsible for setting the contagiousness of the simulated epidemic, as explained in Appendix C [14, 16]. The value of $R_0 = 2.27$ ($\kappa = 2.75$) was found to produce the closest match to our target calibration variables, as shown in Figure 1.

We maintained the incubation period (the interval from the infection to the onset of disease in an individual) around the mean value of 5.0 days, as reported in several studies, e.g., the mean incubation period was reported as 5.2 days, 95% confidence interval (CI), 4.1 to 7.0 [34], while being distributed around a mean of approximately 5 days within the range of 2–14 days with 95% CI [35].

Importantly, we aimed to keep the resultant growth rate of cumulative incidence $\dot{C}$ around 0.2, in order to be consistent with the disease dynamics reported internationally. Another key constraint was a low attack rate in children, reported to be in single digits, for example, only 2.4% of all reported cases in China were children. In doing the calibration, we varied several "free" variables, such as transmission and contact rates, the fraction of symptomatic cases (making it *age-dependent*), the probability of transmission for both symptomatic and asymptomatic agents, and the infectivity profile from the onset of infection.

In calibrating these variables, we minimised changes in order to represent acceptable epidemiological characteristics. For example, we aimed for the generation period (the interval, in days, between successive onsets of symptoms along a transmission chain) to stay in the range $[6.0, 10.0]$ [34, 36, 26]. This is in line with the reported mean serial interval of 7.5 days (with 95% CI of 5.3 to 19) [34] and the 6.5-day mean generation time reported by the Imperial College COVID-19 Response Team [26]. We set the symptoms' duration after the peak of infectivity around the mean value of 12.0 days, on a linearly decreasing profile from the peak. The best match in our calibration, corresponding to $R_0 = 2.27$, was found to produce the generation period of 6.4 days.

The contact and transmission rates across various mixing contexts have been mostly set as in pandemic influenza [14], with the following notable exceptions, as detailed in Appendix C and D. The probability of transmission for asymptomatic/presymptomatic agents was set as 0.3 of that of symptomatic individuals

3

(lower than in the AceMod influenza model). Both symptomatic and asymptomatic profiles were changed to increase exponentially after a latent period of two days, to the infectivity peak, set at the end of the incubation period, see Appendix C.

The fraction of symptomatic cases was set to two-thirds of the total cases ($\sigma = 0.669$), which concurs with the modelling study of Fergusson et al. [26]. However, we found that the best calibration is achieved when this fraction is *age-dependent*, with the fraction of symptomatic cases among children calibrated to one-fifth of the one for adults, that is, $\sigma_c = 0.134$ for children, and $\sigma_a = 0.669$ for adults. This calibration outcome *per se* is in agreement with the reported low attack rates in children worldwide, and the observation that "children are at similar risk of infection as the general population, though less likely to have severe symptoms" [37]. In our study, the attack rates in children have converged to 6%, as shown in Figure 2, that is, only 6% of infections are detected in children, even with a relatively high reproductive number $R_0 = 2.27$ and a relatively long generation period of 6.4 days (in comparison, the generation period for influenza varies in AceMod in the range from 3.35 to 3.39 days).

In summary, this combination of parameters resulted in the dynamics that matched several COVID-19 pandemic characteristics, producing the rate $\dot{C}$ of cumulative incidence during a period of sustained local transmission (i.e., days 40 to 80 since the onset) in the range $[0.15, 0.2]$, as shown in Figure 1. This was achieved while keeping the attack rate in children just over 5%, and agreeing with established range of R_0, and the incubation and generation periods.

3. Intervention strategies

Once the model was calibrated, we focused on our primary objective: evaluating potential effects of several intervention strategies that have been currently deployed in Australia, or have been considered for a deployment.

It is well known that, without efficient and timely interventions, long-distance travel typically carries a virus around the globe within weeks to months of the onset of the outbreak, often causing a worldwide public health emergency [9, 10, 11, 12]. In an attempt to prevent, slow down and eradicate the spread of COVID-19, several pandemic intervention strategies, including various approaches to containment, mitigation and suppression, have been investigated, deployed, and adjusted across the world in the last months. While these strategies inevitably vary across nations, they share fundamental approaches which are adapted by national healthcare systems, aiming at a broad adoption within societies. In the absence of a COVID-19 vaccine, as pointed out by Fergusson et al. [26], *mitigation* policies may include case isolation of patients and home quarantine of their household members, social distancing of the individuals within specific age groups (e.g., the elderly, defined as older than 75 years), as well as people with compromised immune systems or other vulnerable groups. In addition, *suppression* policies may require an extension of case isolation and home quarantine with social distancing of the entire population. Often, such social distancing is supplemented by school and/or university closures.

Some of these intervention strategies and their combinations have shown early promise, while some have been less effective, being delayed by logistical constraints, as well as low adoption level, due to diverse factors often unique to the affected countries. For example, the model developed by the Imperial College COVID-19 Response Team have demonstrated that a combination of mitigation and suppression strategies deployed over three to five months may "reduce peak healthcare demand by 2/3 and deaths by half" [26].

In Australia, an accurate investigation and evaluation of the COVID-19 spread and possible interventions, needs to carefully include demographic specifics, since the population is concentrated mainly along the coast (around urban areas). For instance, it has been previously established that an epidemic of a respiratory disease, such as influenza, typically develops in two waves, initially affecting the more densely populated urban areas, and then spreading to regional and rural areas [15, 16, 17]. In particular, as argued by Cauchemez et al. [38], "back-and-forth waves of transmission between the school, the community, and the household" demand an explicit account of distribution and structuring of schools, grades, and classes in epidemiological models of respiratory diseases. The AceMod simulator has integrated layered school attendance data from the Australian Curriculum, Assessment and Reporting Authority (ACARA), within a
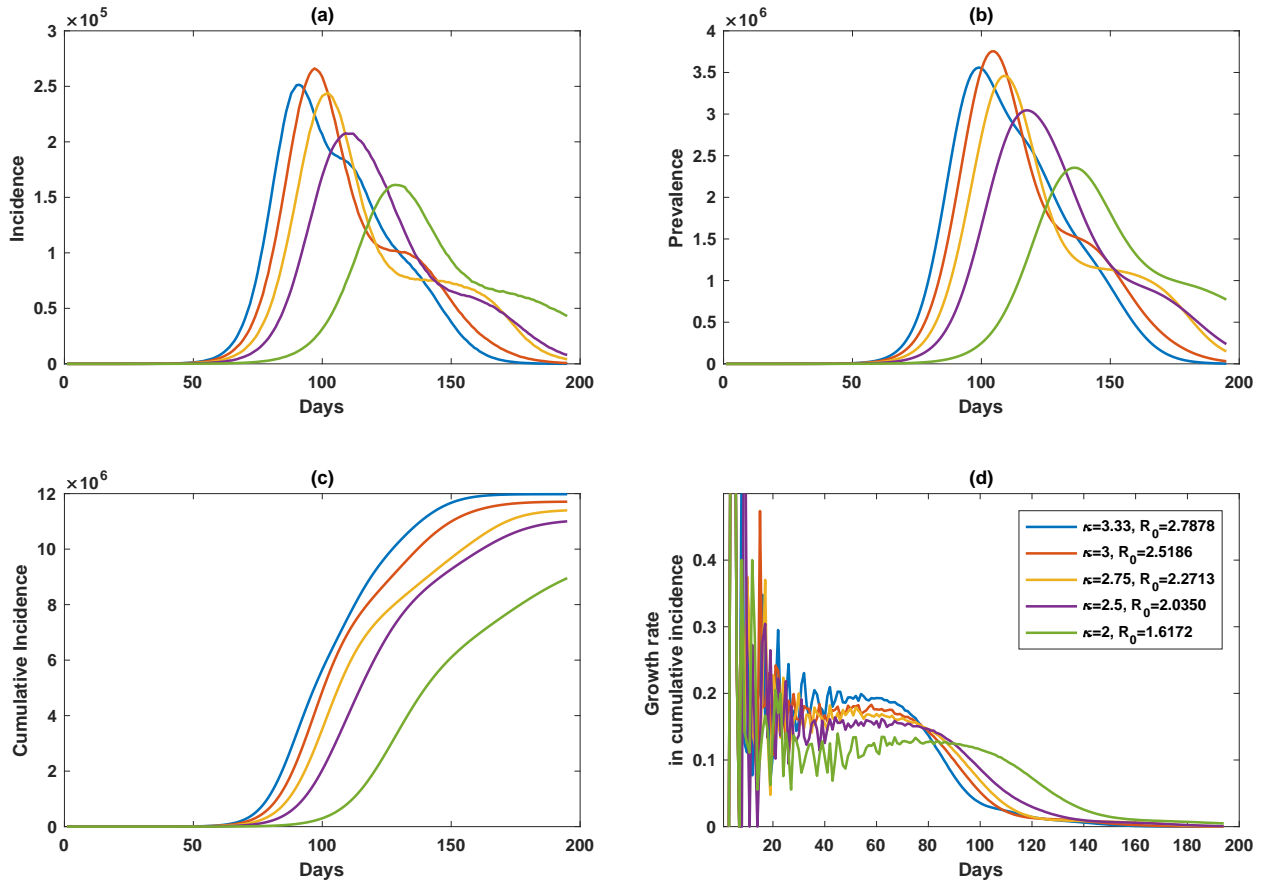
Figure 1: Calibration of AᴄᴇMod parameters to the expected growth rate of cumulative incidence $\dot{C}$ (d), while varying scaling factor κ (i.e, reproductive number R_0), with incidence (a), prevalence (b), and cumulative incidence (c).
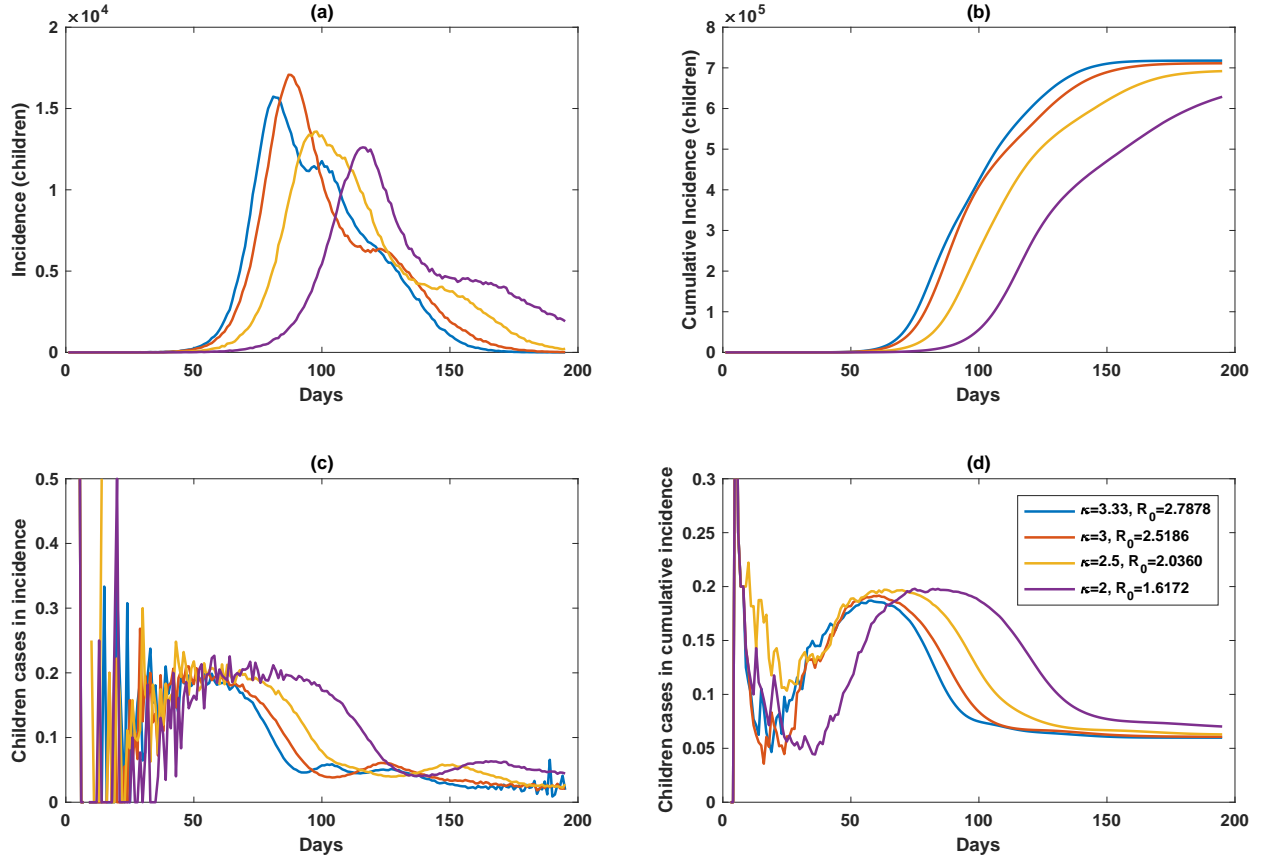
Figure 2: Calibration of AceMod parameters to the attack rate in children (d), while varying scaling factor κ (i.e, reproductive number R_0), with incidence (a), prevalence (b), and cumulative incidence (c) in children.

realistic and dynamic interaction model, comprising both mobility and human contacts. These social mixing layers represent the demographics of Australia as close as possible to the Australian Bureau of Statistics (ABS) 2016 Census and other datasets, as described in Appendix E.

We considered several intervention strategies: case isolation; restriction on international arrivals ("travel ban"); social distancing with the population compliance levels, defined below, varying from 0.0 (no social distancing, i.e., the baseline mode) to 1.0 (full lockdown mode), in increments of 0.1 (10% compliance); and school closures — independently of social distancing. Each of these scenarios were traced over time and compared to the baseline model, in an attempt to quantify their potential for the curtailing the epidemic in Australia, identify minimal levels of the social distancing compliance, and determine the contribution of school closures to the mitigation effort.

The case isolation mitigation strategy was modelled along the lines considered in the study of the Imperial College COVID-19 Response Team [26]: 70% of symptomatic cases stay at home, reduce their working group contacts by 75% (so that their transmission rates decrease to 25% of the standard rate), and maintain their household contacts (i.e., their transmission rates within household remain unchanged). No home quarantine strategy for the members of the affected households is considered in this study, unlike the model of Fergusson *et al.* [26] which considered that 50% of the affected households limit their non-household contacts as well. We opted to exclude this policy at this stage, in order to identify the effects of case isolation more clearly. The case isolation is assumed to be in force from the onset of the epidemic, as has been the case in Australia.

Restriction on international arrivals is set to be enforced from the moment when the number of confirmed infections exceeds the threshold of 1,000 cases. This concurs with the actual epidemic timeline in Australia, which imposed a ban on all arrivals of non-residents, non-Australian citizens, from 9pm of 20 March 2020, with a requirement for strict self-isolation of returning citizens. The number of COVID-19 cases crossed 1,000 cases on 21 March 2020, so the threshold chosen on our model is realistic. The restriction on international arrivals is included in modelling of all other strategies, and is not traced independently, as this mitigation approach is not under debate.

Social distancing (SD) has been implemented in our model by removing all working group contacts, and setting all non-household contacts to 10% of the standard rate, while keeping the contacts within households unaltered. As mentioned above, the SD compliance levels vary from the zero-SD mode to the full lockdown mode, with a fraction of agents (the SD compliance level) following these constraints. Similar to the restriction on international arrivals, SD strategy is triggered by crossing the threshold of 1,000 cases.

Finally, school closures (SC) are considered to remove both students and their teachers from school interactions (their corresponding transmission rates are set to zero), but somewhat increase their interactions within households. Such adjustments may be modelled with a varying degree of the increased household contacts, but in this study we simplified the approach by setting only one level (50% increase), with the intention to consider a more fine-grained approach in the near future. School closures are assumed to be followed with 100% compliance, and may be concurrent with all other strategies described above. For example, they may account for scenarios when, under a partial SD compliance, some household members may choose to leave their households during daytime and interact at work, while their children and/or teacher partners (i.e., other adults) stay at home. SC strategy is also evaluated as triggered by crossing the threshold of 1,000 cases — this is not a current practice in Australia, and so we investigate the SC intervention separately from SD strategy (by setting SD compliance as zero). Hence, the evaluation of school closures is meant to provide an input to policy setting, rather than forecast changes to possible epidemic dynamics at this stage. We note that, at the moment (21 March 2020), Australian Federal Government does not recommend schools closures.

While the case isolation strategy is assumed to last during the full course of the epidemic, the duration of SD and/or SC strategies varies, across a range of time periods, e.g., 28, 49, 70, 91 days, that is, across 4, 7, 10, 13 weeks.

4. Results

The comparative analysis is carried out by AceMod, across all intervention strategies, for the calibrated set of parameters, including $R_0 = 2.27$ (i.e., scaling with $\kappa = 2.75$), and the transmission and contact rates

as detailed in Appendix D. The infectivity of infected agents is set to exponentially rise and peak at 5 days, after an incubation period which includes two days of zero infectivity (latent period). The symptoms are set to last up to 12 days post the infectivity peak, while linearly decreasing — this results in the generation period around 6.4 days. The probability of transmission for asymptomatic/presymptomatic agents is set as 0.3 of that of symptomatic individuals; and the age-dependent fractions of symptomatic cases are set as $\sigma_c = 0.134$ for children, and $\sigma_a = 0.669$ for adults.

All figures below show typical runs, not the averages over multiple runs, in order to illustrate a realistic epidemic development over time, without smoothing. The typical runs are chosen within the range observed over multiple simulations carried out by AceMod, and use identical random number seeds to control for stochastic initial conditions.

4.1. Baseline

A trace of the baseline model—no interventions whatsoever—is shown in Fig. 3, with clear epidemic peaks in both incidence and prevalence evident after 105–110 days from the onset of the disease in Australia, i.e., occurring around mid-May 2020 in the absence of any interventions. The scale of the impact is very high, infecting nearly 50% of the Australian population. This baseline scenario is provided only for comparison, in order to evaluate the impact of interventions, some of which are already in place in Australia. To re-iterate, we aim to consider timely intervention scenarios applicable to current situation in Australia, where the number of confirmed COVID-19 cases crossed 1,000 on 21 March 2020, and the growth rate of cumulative incidence $\dot{C}$ stayed above 20% for longer than a week. We observe that the simulated baseline generates the 20% cumulative incidence increase, in an agreement with actual dynamics.

4.2. Case isolation

All the following interventions include restrictions on international arrivals, triggered by the threshold of 1,000 cases. Two mitigation strategies are of immediate interest: (i) case isolation, and (ii) school closures, combined with case isolation. Both of these strategies are shown in Fig. 3, with the duration of the combined strategy (SC and case isolation) set as 49 days (7 weeks), starting after the threshold of 1,000 cases is reached on day 42 since the onset. The case isolation approach, applied without SC, delays the epidemic peak by just over a week, while reducing the severity at the peak time by around 24% (incidence peak), and 22% (prevalence peak). The overall attack rate, however, is reduced insignificantly, showing that case isolation alone will not be effective for epidemic suppression.

4.3. School closures: SC

Adding school closures to the case isolation approach also does not achieve a significant reduction in the overall attack rate (Fig. 3). While the peaks of both incidence and prevalence are delayed by about two weeks (15 days for both incidence and prevalence), due to a slower growth rate of cumulative incidence, their magnitudes remain practically the same. We also traced the dynamics resulting from SC strategy for two specific age groups: children and individuals over 65 years old, shown by Fig. 4 and 5. The two-week delays in occurrence of the peaks are observed across both age groups, suggesting that there is a strong concurrence in the disease spread across these age groups. We also observe that, under this strategy, there is no difference in the magnitude of the incidence peak for the older age group. Interestingly, for children, the magnitude of the incidence peak increases by about 8% under the SC strategy coupled with case isolation, shown by Fig. 4. This may be explained by increased interactions of children during various household and community social mixing, when schools are closed.

In short, the only tangible benefit of school closures, coupled with case isolation, is in delaying the epidemic peak by two weeks, at the expense of a slight increase in the contribution of children to the incidence peak. Given other societal costs of school closures (e.g., drawing their parents employed in healthcare and other critical infrastructure away from work), this strategy may be less effective than previously suggested (e.g., school closures are considered an important part of pandemic influenza response). There is, nevertheless, one more possible benefit of school closures, discussed in the context of the overall social distancing, as described below.

4.4. Social distancing: SD

Our next step is to compare social distancing strategies, coupled with case isolation, across different compliance levels. Low compliance levels, set at less than 70%, did not show any potential to suppress the disease in the considered time horizon (28 weeks), while the total lockdown, that is, complete social distancing at 100%, managed to reduce the incidence and prevalence to zero, after 49 days of the mitigation. However, since the total lockdown is never perfect, we need to focus on the practically achievable compliance levels: 70%, 80% and 90%. For example, Fig. 3 includes a comparison with the SD level 70% for the entire population, and Figures 4 and 5 trace this strategy for two specific age groups (children and older adults).

Duration of all SD strategies is set to 91 days (13 weeks), and not surprisingly, there is a significant delay of about 12 weeks before the epidemic peak, occurring when the SD strategy (70% compliance) ends. Importantly, during the time period that the SD level is maintained at 70%, the disease is not controlled entirely, with the numbers of new infected cases (incidence) remaining in hundreds, and the number of active cases (prevalence) remaining in thousands. Thus, once social distancing is removed, the epidemic resumes, suggesting that 70% compliance is inadequate. The two higher levels of SD, 80% and 90%, are more effective at suppressing prevalence and incidence during the 13-week social distancing period.

Figure 6 contrasts these three levels of SD compliance, "zooming in" into the key time period, immediately following the introduction of social distancing. Crucially, there is a qualitative difference between the lower levels of SD compliance (70%, or less), and the higher levels (80%, or more). For the SD compliance set at 80% and 90%, we observe a *reduction* in both incidence and prevalence, lasting for the duration of the strategy (91 days). The 80% SD compliance does not completely eradicate the disease, but reduces the new cases to less than 100, with prevalence below 1,000 (during the suppression period, before the resurgence). It is evident that this level of compliance would succeed if the strategy was implemented for longer period, e.g., another 5–6 weeks.

The 90% SD compliance practically controls the disease, bringing both incidence and prevalence to very low numbers of isolated cases. It is possible for the epidemic to spring back to significant levels even under this level of compliance, as the remaining sporadic cases indicate a potential for endemic conditions. We do not quantify these subsequent waves, as they develop beyond the immediately relevant time horizon. Nevertheless, we do share the concerns expressed by the Imperial College COVID-19 Response Team: "The more successful a strategy is at temporary suppression, the larger the later epidemic is predicted to be in the absence of vaccination, due to lesser build-up of herd immunity" [26].

The cumulative incidence for the best achievable scenario (90% SD compliance coupled with case isolation and restrictions on international arrivals) settles around 2,500 cases during the suppression period, before a possible resurgence, at some point after intervention measures are relaxed. This is an outcome several orders of magnitude better than the worst case scenario, developing in the absence of the combined mitigation and suppression strategies. Differences between 70% and 90% SD compliance levels are also visualised in choropleth maps of four largest Australian Capital Cities: Sydney, Melbourne, Brisbane and Perth, shown in F, at day 60, that is, at a time when these two compliance levels result in a tangible divergence.

It is clear that there is a trade-off between the level of SD compliance and the duration of the SD strategy: the higher the compliance, the shorter period it needs to be implemented for. Both 80% and 90% compliance levels are practically achievable within reasonable time periods: 18-19 and 13-14 weeks respectively. We point out, however, that lower levels of compliance (at 70% or less) do not succeed for any duration of the imposed social distancing limits. This qualitative difference is of major policy setting importance, indicating a sharp transition in the performance of these strategies in the region between 70% and 80%. We do not attempt to establish a more precise level of required compliance, e.g., 75%. Such a precision would be of lesser practical relevance than the identification of 80% level as the minimal acceptable level of social distancing, with 90% providing a shorter timeframe.

At this stage we revisit school closures. As shown in Fig. 7, an addition of SC strategy to the SD set at 70% also generates a reduction in incidence, albeit progressing at a higher level than such reductions observed at 80% and 90% SD levels, coupled with school closures. This suggests that another potential benefit of school closures is that it may "compensate" for about 10% lack of SD compliance. This combination, however, would require a much longer duration of the coupled strategy (70% SD and SC), and may be impractical.
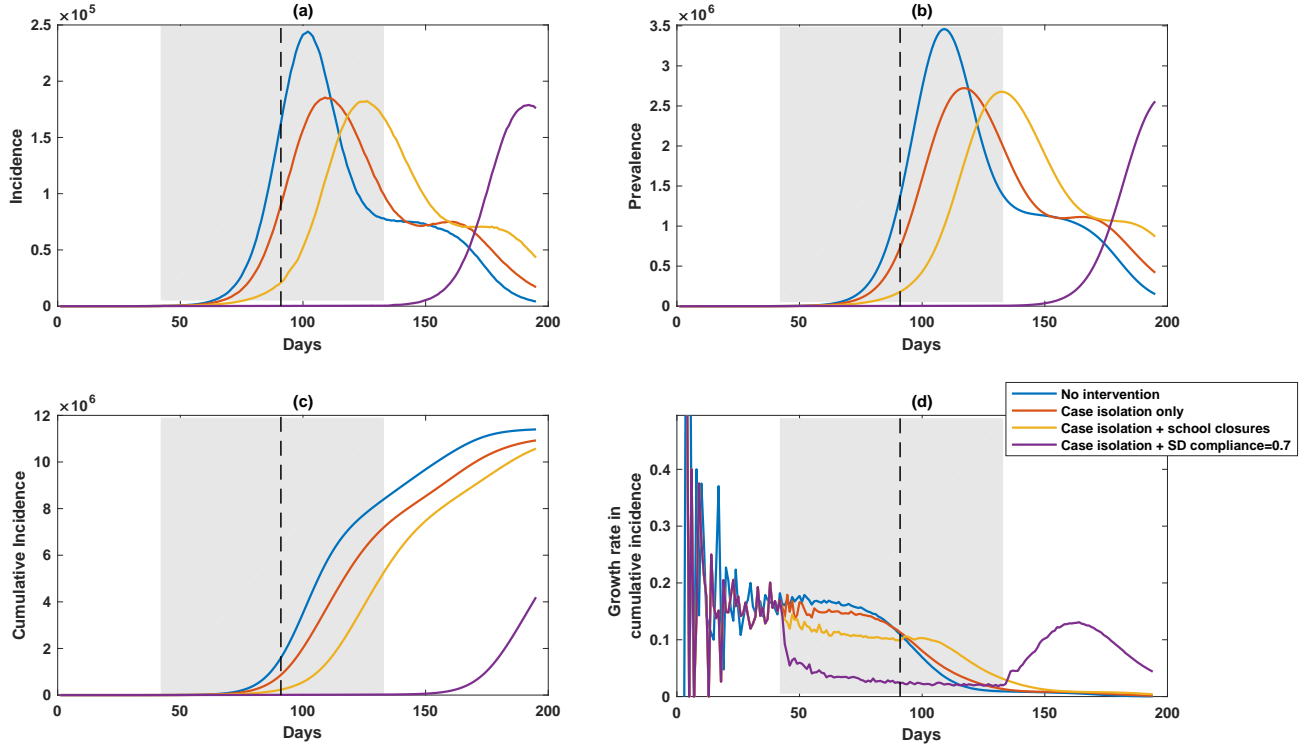
Figure 3: A combination of the case isolation and 70% social distancing measures delay epidemic peaks and reduce their magnitude, whereas school closures have short-term effect. Several baseline and intervention scenarios, traced for incidence (a), prevalence (b), cumulative incidence (c), and the growth rate of cumulative incidence $\dot{C}$ (d). The strategy with SC combined with case isolation lasts 49 days (7 weeks), marked by a vertical dashed line. Duration of the SD strategy is set to 91 days (13 weeks), shown as a shaded area. Restrictions on international arrivals are set to last until the end of each scenario.

4.5. Summary

In short, the best intervention approach identified by AceMod is to combine restrictions on international arrivals (already implemented in Australia), case isolation (also already implemented to a reasonable extent, but demanding increasing testing and monitoring resources), and social distancing with at least 80%–90% compliance and a duration of 91 days (13 weeks). Any compromise on the recommended compliance levels is likely to lengthen the duration of the required suppression measures. We point out that our results are relevant only for the duration of the mitigation and suppression, and a resurgence of the disease is possible once these interventions cease, as shown in Fig. 6. Hence, we do not quantify the precise impact of control measures beyond the selected time horizon (28 weeks), and focus on the time period in the near future, aiming to provide immediately relevant insights. Furthermore, our results should not be seen as policies optimised over all possible parameter combinations, but rather as a clear demonstration of the extent of social distancing required to reduce incidence and prevalence in the next six months.

5. Conclusions

In this study we simulated several possible scenarios of COVID-19 pandemic's spread in Australia. The model, AceMod, was calibrated to known pandemic dynamics, and accounted for age-dependent attack rates, a range of reproductive numbers, age-stratified and social context dependent transmission rates,
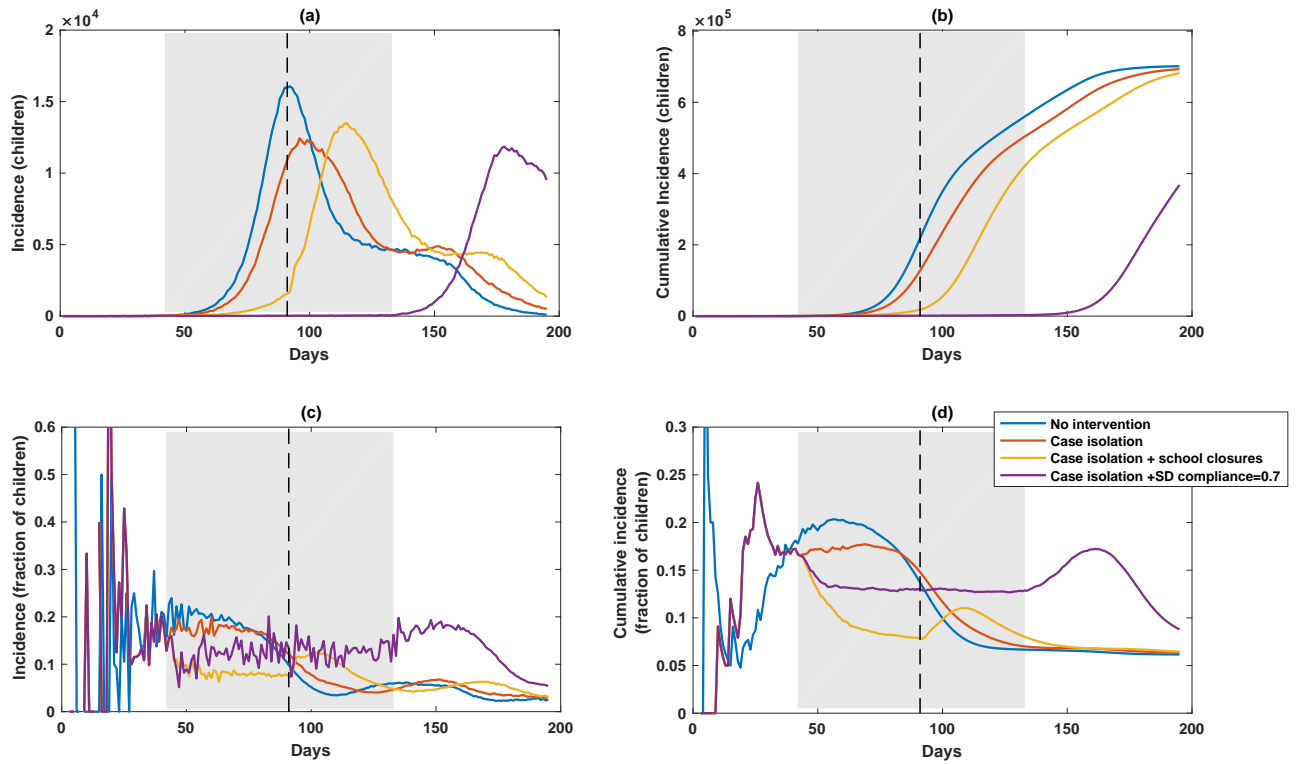
10

Figure 4: School closures delay incidence peak by two weeks, but slightly increase the fraction of new cases in children around the peak time. Epidemic curves for children: incidence (a), cumulative incidence (b), fraction of children in incidence (c), and fraction of children in cumulative incidence (d). The strategy with SC combined with case isolation lasts 49 days (7 weeks), marked by a vertical dashed line. Duration of the SD strategy is set to 91 days (13 weeks), shown as a shaded area. Restrictions on international arrivals are set to last until the end of each scenario.

11
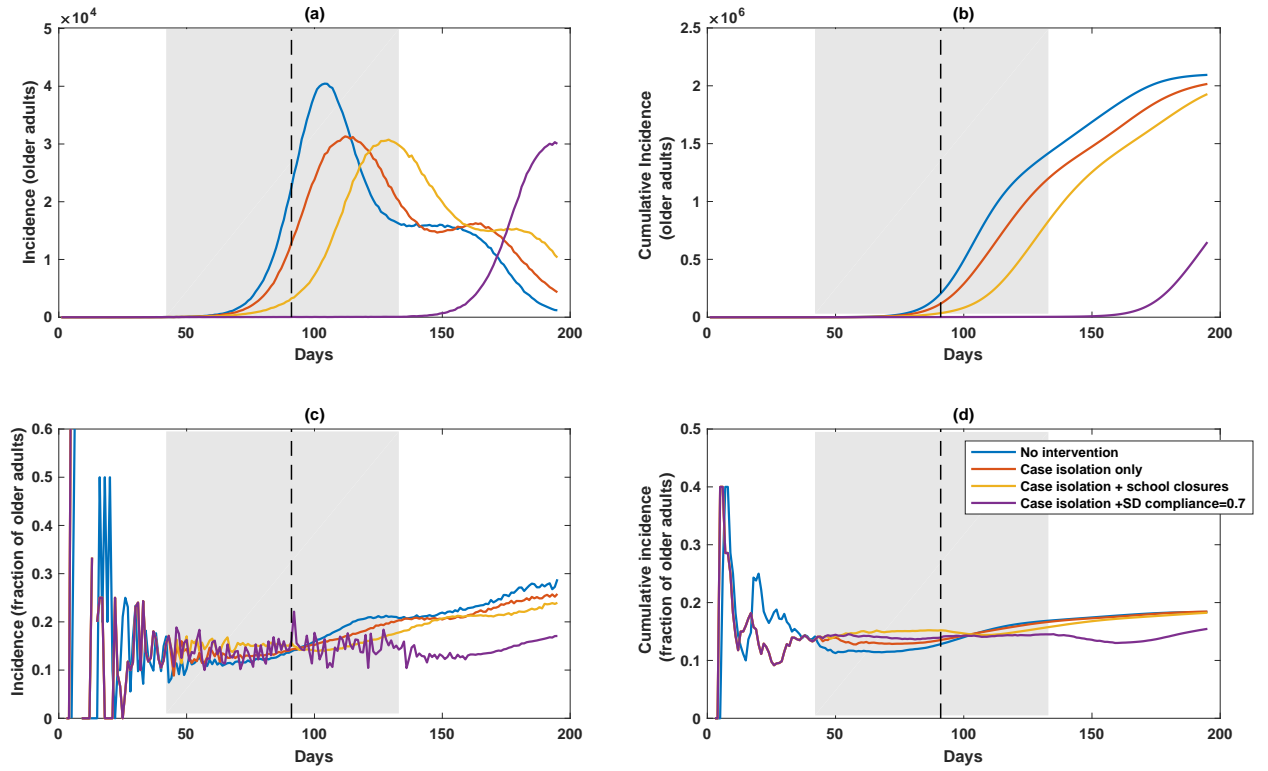
Figure 5: School closures delay incidence peak by two weeks, but do not strongly affect new cases for older adults. Epidemic curves for older adults: incidence (a), cumulative incidence (b), fraction of older adults in incidence (c), and fraction of older adults in cumulative incidence (d). The strategy with SC combined with case isolation lasts 49 days (7 weeks), marked by a vertical dashed line. Duration of the SD strategy is set to 91 days (13 weeks), shown as a shaded area. Restrictions on international arrivals are set to last until the end of each scenario.
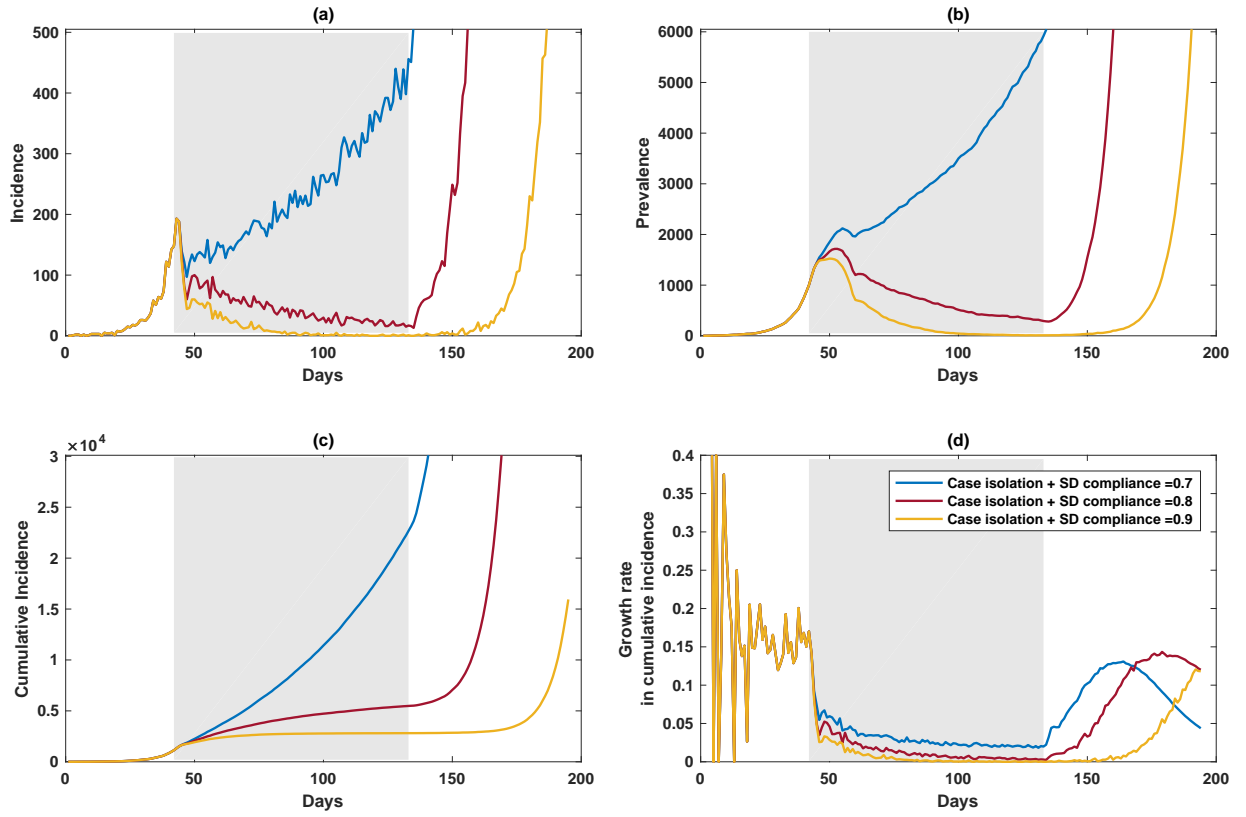
Figure 6: Strong compliance with social distancing (at 80% and above) effectively controls the disease during the suppression period, while lower levels of compliance (at 70% or less) do not succeed for any duration of the suppression. A comparison of social distancing strategies, coupled with case isolation, across different compliance levels (70%, 80% and 90%). Duration of each SD strategy is set to 91 days (13 weeks), shown as a shaded area. Case isolation and restrictions on international arrivals are set to last until the end of each scenario. Traces show incidence (a), prevalence (b), cumulative incidence (c), and the growth rate of cumulative incidence $\dot{C}$ (d).
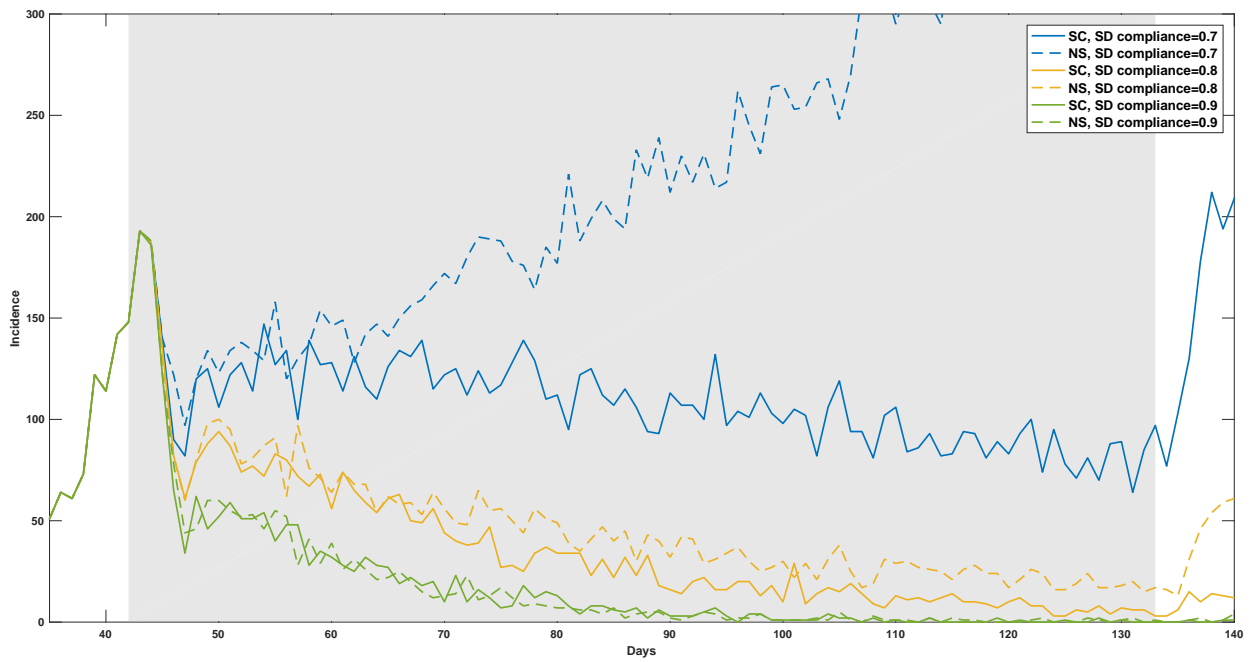
Figure 7: School closures may "compensate" for about 10% lack of SD compliance. A comparison of social distancing strategies, coupled with case isolation and school closures (SC) or no school closures (NS), across different compliance levels (70%, 80% and 90%). Duration of each combined SD and SC strategy is set to 91 days (13 weeks), shown as a shaded area. Case isolation and restrictions on international arrivals are set to last until the end of each scenario.

household clusters and other social mixing contexts, symptomatic-asymptomatic distinction, long and varying incubation periods, and other relevant epidemiological parameters.

An important calibration result was the need for age-dependent fractions of symptomatic agents, with the fraction of symptomatic children found to be one-fifth of that of the adults. While other combinations of parameters may also succeed in calibration, setting the *age-dependent* fractions of symptomatic cases may be important for other modelling studies across the world.

An analysis of spatiotemporal characteristics of COVID-19 pandemic in Australia was carried across a range of intervention strategies, some of which are already in place, while some are under discussion. By running multiple computer simulations, while varying details of the micro-simulation, we estimates epidemic dynamics, in terms of the infection's prevalence and incidence, its peaks and waves, and other indicators, including age-dependent attack rates.

We reported several findings relevant to COVID-19 mitigation and suppression policy setting. The first implication is that the effectiveness of school closures is limited, producing a two-week delay in epidemic peak, without a significant impact on the magnitude of the peak, in terms of incidence or prevalence. The temporal benefit of the two-week delay may be offset not only by logistical complications, but also by some increases in the fractions of both children and older adults during the period around the incidence peak.

The second implication is related to social distancing (SD) strategy, which showed no benefit for lower levels of compliance (at 70% or less) — these levels do not contribute to epidemic control for any duration of the social distancing restrictions. Only when the SD compliance levels exceed 80%, there is a reduction in incidence and prevalence. Our modelling results indicate existence of an actionable transition across these strategies in the range between 70% and 80%. In other words, increasing a compliance level just by 10%, from 70% to 80%, may effectively control the spread of COVID-19 in Australia (during the suppression period). We also reported a trade-off between the compliance levels and the duration of SD mitigation, with 90% compliance significantly reducing incidence and prevalence after a shorter period of 91 days (13 weeks). Although a resurgence of the disease is possible once these interventions cease, we believe that this study could facilitate a timely planning of effective intervention strategies.

Future research will address several limitations of our study, including a more fine-grained implementation of natural history of the disease, incorporation of more recent ABS data from 2020, inclusion of home quarantine strategy for the affected households, as well as a more refined school closures strategy. We also hope to trace specific spatial pathways and patterns of epidemics, in order to enable a detailed understanding of how the infection spreads in diverse circumstances and localities, with the aim to identify the best ways to locate and curtail the pandemic spread in Australia[1]. Another avenue leads to analysis of precursors and critical thresholds for possible emergence of new strains [39, 40, 41], given genomic surveillance data interpreted as complex networks [42, 43, 44, 45, 46], and dynamic models of social behaviour in times of health crises [47, 16, 48, 49].

6. Acknowledgments

---

[1]COVID-19 pandemic is an ongoing and real-time challenge with immediate consequences. We will endeavour to update this manuscript with new information and compare the unfolding events to our simulation in order to improve predictions and policy advice for curtailing the pandemic in Australia.

A. COVID-19 pandemic in top 8 affected countries and Australia

Figures 8 and 9 trace cumulative incidence C, incidence, and growth rate of cumulative incidence $\dot{C} = [C(n+1)-C(n)]/C(n)$, for time step n, for the top eight affected countries: China, Iran, Italy, South Korea 8, Spain, Germany, France, USA 9. The time series begin from the day when the total number of confirmed cases exceeds five. Figure 10 traces these time series for Australia.

B. Natural history of disease

The natural history of disease is a description of the disease from pathological onset to recovery from the perspective of a single individual, profiling their infectiousness over time [50]. In the past, the AceMod simulator has been used to model pandemic influenza within Australia, and here we detail modifications of the natural history aimed to account for COVID-19 specifics. The natural history model considers three distinct phases of infection. The first phase is the LATENT period during which individuals are infected but unable to infect others, set in the COVID-19 model as two days. The second phase is the INCUBATION period, characterised by the onset of symptoms and an increasing infectivity, set in the COVID-19 model to lasts for five days. We model this increase in infectiousness as an exponential increase which varies from 0% to 100% over three days (see figure 11). Following the incubation phase, the infectious reaches its peak, and then decreases linearly over 12 more days, until the recovery, with immunity, occurs after 17 days. Finally, we assume that asymptomatic cases are 30% as infectious as symptomatic cases. Unlike influenza, where we assume that the asymptomatic fraction is the same for adults as for children, for the SARS-COV-2 coronavirus we assume that while 67% of adult cases are symptomatic, a significantly lower fraction (13.4%) is symptomatic in children.

C. Transmission model and reproductive number

The primary dynamics of AceMod are the infection transmissions. At each time-step the simulator determines the probability of infection for an individual, based on the infection levels in each of their mixing contexts. At each time step we consider all daytime or all nighttime contexts. Let $X_i(n)$ be a random variable describing the state of individual i at time step n. At each time step we calculate $p_i(n) = P(X_i(n) = \text{LATENT} | X - i(n-1) = \text{SUSCEPTIBLE})$, the probability that a susceptible individual is infected at n. Each individual belongs to a number of mixing groups with which an agent interacts, denoted $g \in \mathcal{G}_i(n)$, as well as an associated static set of agents $\mathcal{A}_g$. Within each of these contexts, we define a probability $p_{j \to i}^g$ that individual j infects individual i in context g in a single time step. The probability that a susceptible agent i is infected at a given time step n is thus calculated as:

$$p_i(n) = 1 - \prod_{g \in G_i(n)} \left[\prod_{j \in \mathcal{A}_g \setminus i} (1 - p_{j \to i}^g(n)) \right], \tag{1}$$

where $p_{j \to i}^g$ is the context-dependent probability that infected individual j infects susceptible agent i in mixing group g. We also define a scaling factor κ (proportional to the reproductive number R_0), as a free parameter which allows us to vary the contagiousness of simulated epidemic scenarios:

$$p_{j \to i}^g(n) = \kappa \, f(n - n_j \mid j, i) \, q_{j \to i}^g \tag{2}$$

where n_j denotes the time when agent j becomes infected, and $q_{j \to i}^g$ is the probability of transmission from agent j to i at the infectivity peak, derived from the transmission or contact rates. This model assumes that for all contexts, the probabilities of infection over a given time period are known. In cases where this information is unavailable, we instead utilise contact rates reported and calibrated in previous studies. Thus, a majority of the transmission and contact probabilities follow our previous work on pandemic influenza [51, 52, 24, 38, 14, 15], see Tables 1 and 2 in section D. Full details regarding their application can be found in [14].
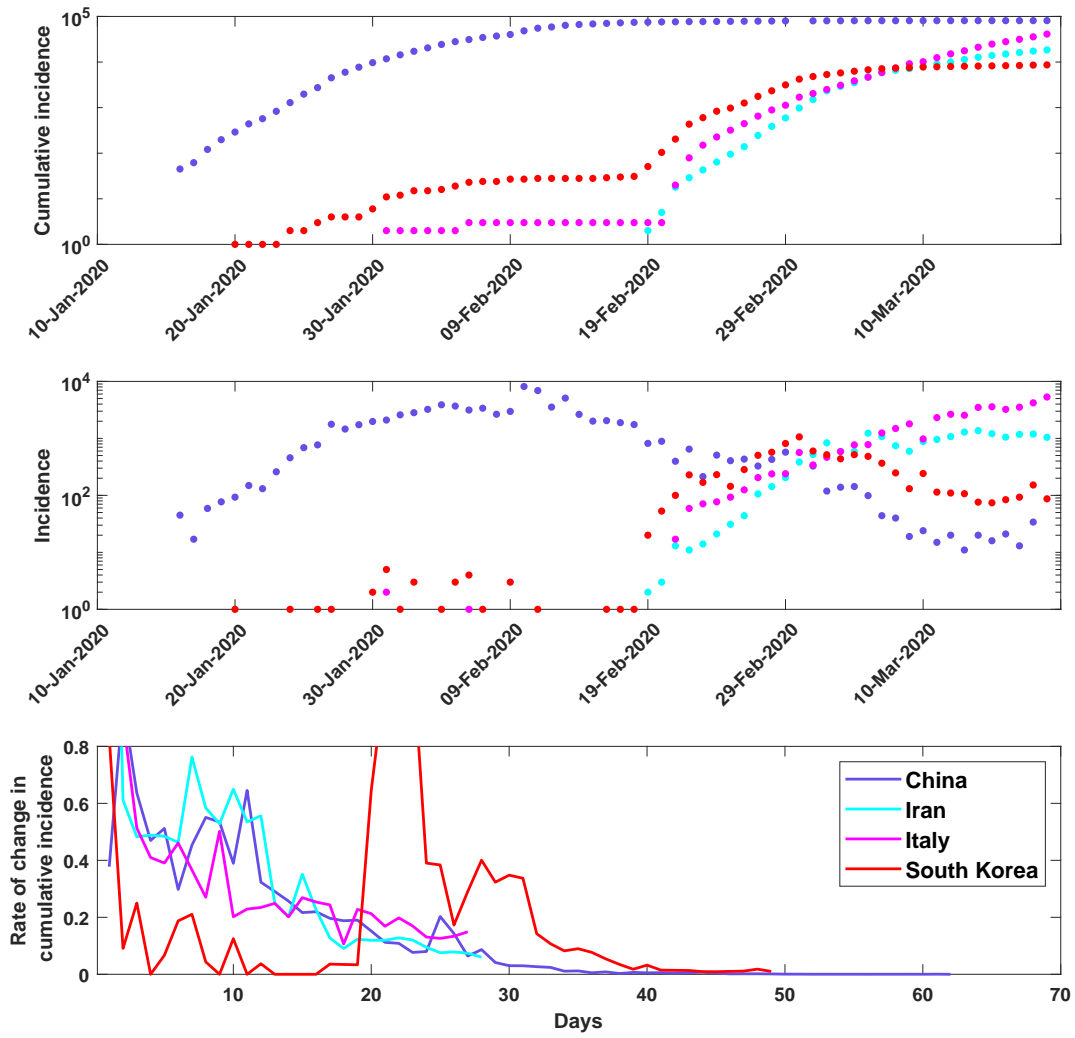
Figure 8: Cumulative incidence (log scale), incidence (log scale), and growth rate of cumulative incidence: China, Iran, Italy, South Korea (up to 19 March 2020).
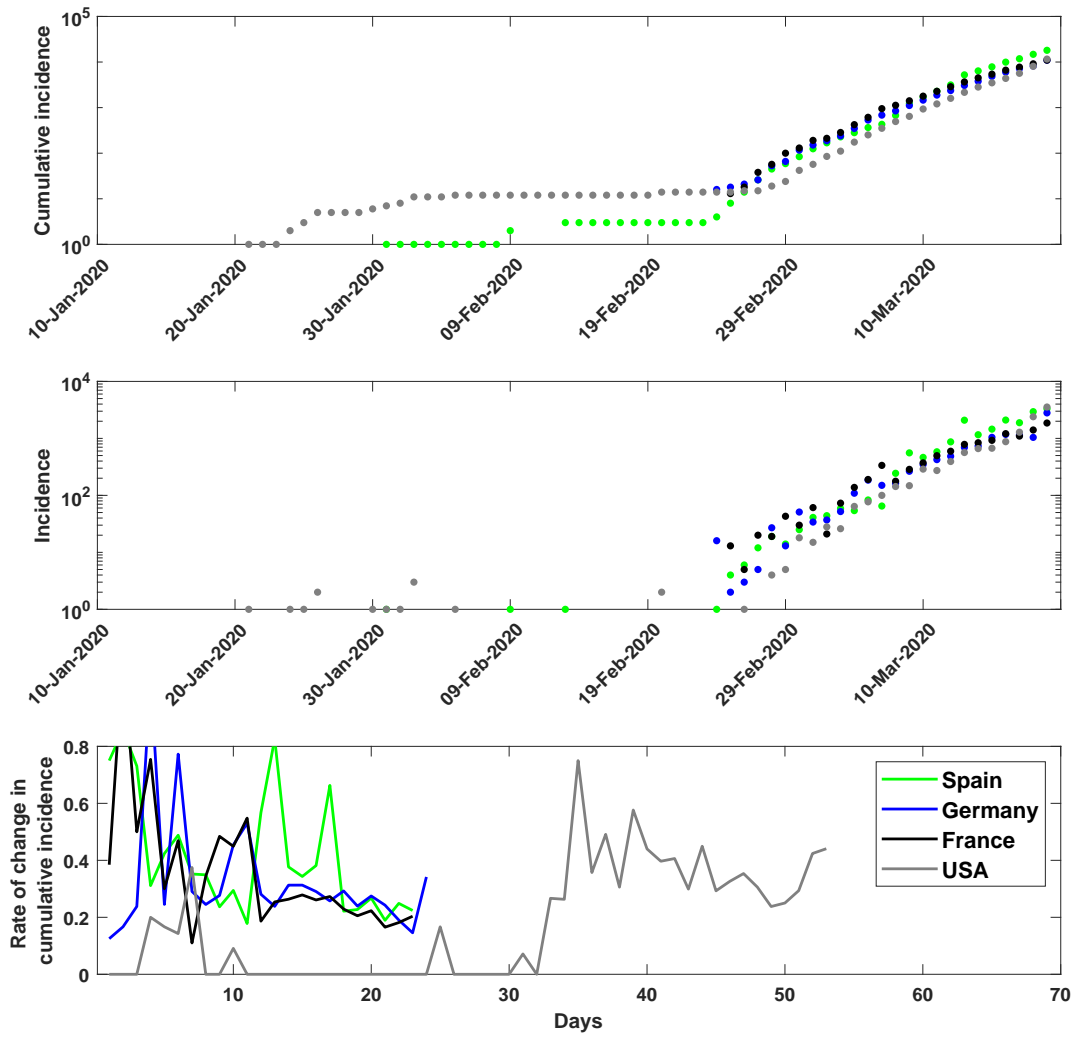
Figure 9: Cumulative incidence (log scale), incidence (log scale), and growth rate of cumulative incidence: Spain, Germany, France, USA (up to 19 March 2020).
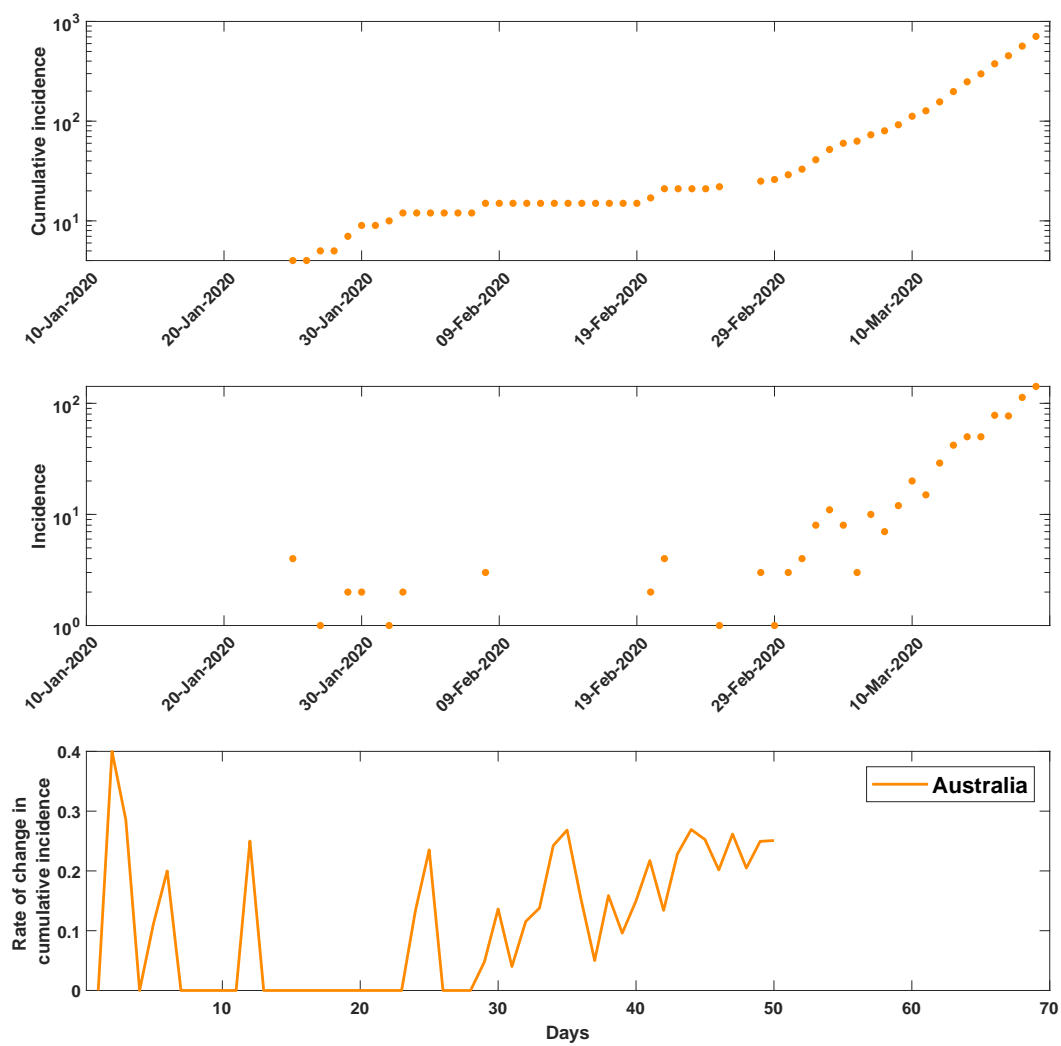
18

Figure 10: Cumulative incidence (log scale), incidence (log scale), and growth rate of cumulative incidence: Australia (up to 19 March 2020)
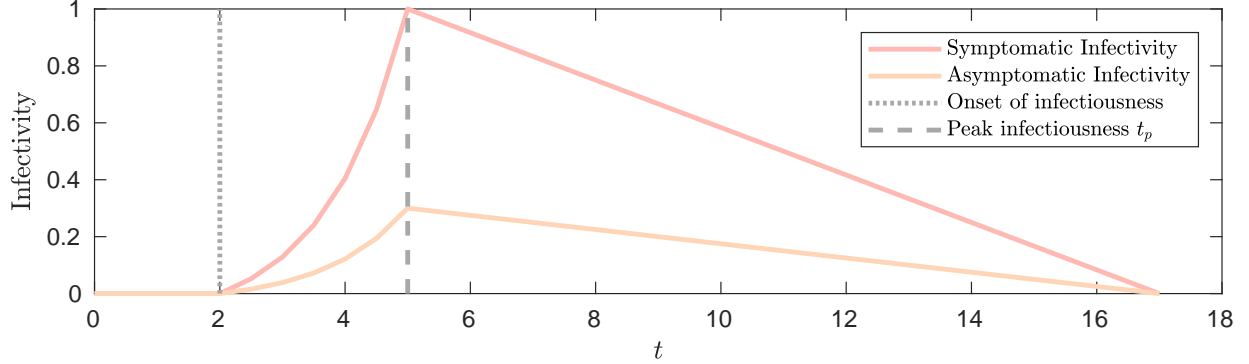
Figure 11: Profile of the infectivity used as the natural history of COVID-19, for both symptomatic and asymptomatic cases. After two days, individuals become infectious, with the infectivity rising exponentially until its peak at five days. After this peak, the infectivity linearly decreases, with full recovery occurring at 17 days. At comparable points within the natural history of disease, asymptomatic individuals are 30% as infectious as symptomatic individuals.

Table 1: Daily contact probabilities $c_{j \to i}^g$ for different contact groups g, reported by [24], reproduced from [14], except for the rates in household clusters.

Mixing group g	Infected individual j	Susceptible individual i	Contact probability $c_{j \to i}^g$
Household cluster	Child (<19)	Child (<19)	0.05
	Child (<19)	Adult (>18)	0.05
	Adult (>18)	Child (<19)	0.05
	Adult (>18)	Adult	0.05
Working Group	Adult (19-64)	Adult (19-64)	0.05
Neighbourhood	Any	Child (0-4)	0.0000435
	Any	Child (5-18)	0.0001305
	Any	Adult (19-64)	0.000348
	Any	Adult (65+)	0.000696
Community	Any	Child (0-4)	0.0000109
	Any	Child (5-18)	0.0000326
	Any	Adult (19-64)	0.000087
	Any	Adult (65+)	0.000174

In this study we used "the attack rate pattern weighted index case" to calculate R_0 [21, 17]. The method is based on age-specific attack rates, computed as averages over many simulation instances, in order to reduce the bias in determining a typical index case, present due to population heterogeneity. As argued in [53, 17], given the correlation between age group and population structure, the age-stratified weights, assigned to secondary cases produced by a sample of index cases, improve the estimation of the reproductive number R_0.

D. Transmission and contact probabilities

Following [14], with some minor adjustments, the transmission and contact probabilities are given in Tab. 1 and Tab. 2, respectively.

E. Population generation, demographics and mobility

At the beginning of AceMod simulation, a surrogate population is generated. This surrogate population is matched to coarse-grained distributions arising from the 2016 Australian census published by the Aus-

Table 2: Daily transmission probabilities $q_{j\to i}^{g}$ for different contact groups g, reported by [38], reproduced from [14].

Contact Group g	Infected Individual j	Susceptible Individual i	Transmission Probability $q_{j\to i}^{g}$
Household size 2	Any	Child (<19)	0.0933
	Any	Adult (>18)	0.0393
Household size 3	Any	Child (<19)	0.0586
	Any	Adult (>18)	0.0244
Household size 4	Any	Child (<19)	0.0417
	Any	Adult (>18)	0.0173
Household size 5	Any	Child (<19)	0.0321
	Any	Adult (>18)	0.0133
Household size 6	Any	Child (<19)	0.0259
	Any	Adult (>18)	0.0107
School	Child (<19)	Child (<19)	0.000292
Grade	Child (<19)	Child (<19)	0.00158
Class	Child (<19)	Child (<19)	0.035

tralian Bureau of Statistics (ABS). In particular we use Statistical Areas (SA1 and SA2) level statistics [54] regarding age, household composition and workplaces in generating this surrogate population. Individuals in the population are separated into 5 different age groups preschool aged children (0-4), children (5-18), young adults (19-29), adults (30-65) and older adults (65+). Along with these assigned characteristics, individuals are assigned a number of mixing contexts based on the census data. The AceMod simulator is a discrete-time simulation, where each simulated day is separated into two distinct portions: 'daytime' and 'nighttime'. In the daytime, workplace and school-based mixing are considered, whereas nighttime mixing considers household spread, and other local spread at the neighborhood (SA1) and community (SA2) levels.

The population generation begins with the contexts needed for nighttime mixing, which can be thought of as "home regions". The simulation iterates through each SA1, creating a cumulative density function (CDF) describing the size and type of households expected based on two dependent probability distributions defined by the ABS. Given this CDF, the procedure begins to randomly generate households, with the generation of agents occurring during this process. Once a household is generated for an SA1, agents are generated to match the size and type of the household (e.g., a single parent family of size four will generate one adult and three children). In order to generate attributes for this surrogate population, the simulation then reads in CDFs describing the population statistics of the given SA, with each of these agents being assigned some attributes based on these population distributions.

Following the population of the home regions, the AceMod simulator assigns work and school regions to individuals within the population. This process is based on the "Travel to work" data published by the ABS, which defines a number of individuals N living in home region i and working in region j. In order to satisfy each of these "worker flows", a number of unassigned working-age individuals (19-64 years old) in region i are selected at random and assigned to work in location j. School allocation, on the other hand, is somewhat more complicated as detailed data about student home locations is not available from the ABS. Instead, we use the available data from the Australian Curriculum, Assessment and Reporting Authority (ACARA), detailing the locations of schools, along with a proximity based model which biases children allocation towards closer schools. More detail about student allocation can be found in previous studies [15].

We trace scenarios of COVID-19 pandemic spread in Australia, initiated by passenger arrivals via air traffic from overseas. This process maintains a stream of new infections at each time step, set in proportion to the average daily number of incoming passengers at that airport [15, 16]. These infections occur probabilistically, generated by binomial distribution $B(P, N)$, where P and N are selected to generate one new infection within a 50 km radius of the airport, per 0.04% of incoming arrivals on average.

F. Comparison of SD compliance levels across several state capitals

Differences between 70% and 90% SD compliance levels are visualised in choropleth maps of four largest Australian Capital Cities: Sydney, Melbourne, Brisbane and Perth (Fig. 12). These snapshots depict the incidence in these cities at day 60, illustrating how these two compliance levels diverge at that time.

References

[1] National Health Commission (NHC) of the People's Republic of China. NHC daily reports, [Online; accessed 21-March-2020] (2020).
http://www.nhc.gov.cn/yjb/pzhgli/new_list.shtml

[2] C. Wang, P. W. Horby, F. G. Hayden, G. F. Gao, A novel coronavirus outbreak of global health concern, The Lancet 395 (10223) (2020) 470–473.

[3] Report of the WHO–China joint mission on coronavirus disease 2019 (COVID-19),
https://www.who.int/docs/default-source/coronaviruse/who-china-joint-mission-on-covid-19-final-report.pdf
(2020).

[4] Vital surveillances: The epidemiological characteristics of an outbreak of 2019 novel coronavirus diseases (COVID-19)–China, 2020. the novel coronavirus pneumonia emergency response epidemiology team, China CDC Weekly 2 (8) (2020) 113–122.

[5] WHO Director-General's opening remarks at the media briefing on COVID-19 – 11 March 2020, [Online; accessed 21-March-2020] (2020).
https://www.who.int/dg/speeches/detail/who-director-general-s-opening-remarks-at-the-media-briefing-on-covid-19---11-mar

[6] E. Dong, H. Du, L. Gardner, An interactive web-based dashboard to track COVID-19 in real time, The Lancet Infectious Diseases.

[7] Coronavirus COVID-19 global cases: Johns Hopkins University,
https://www.arcgis.com/apps/opsdashboard/index.html#/bda7594740fd40299423467b48e9ecf6.

[8] Wikipedia contributors, 201920 coronavirus pandemic — Wikipedia, The Free Encyclopedia, [Online; accessed 21-March-2020] (2020).
https://en.wikipedia.org/w/index.php?title=2019%E2%80%9320_coronavirus_pandemic&oldid=946669971

[9] I. M. Longini, A. Nizam, S. Xu, K. Ungchusak, W. Hanshaoworakul, D. A. Cummings, M. E. Halloran, Containing pandemic influenza at the source, Science (New York, N.Y.) 309 (5737) (2005) 1083–1087.

[10] N. M. Ferguson, D. A. T. Cummings, S. Cauchemez, C. Fraser, S. Riley, A. Meeyai, S. Iamsirithaworn, D. S. Burke, Strategies for containing an emerging influenza pandemic in Southeast Asia, Nature 437 (8) (2005) 209–214.

[11] E. O. Nsoesie, R. J. Beckman, M. V. Marathe, Sensitivity analysis of an individual-based model for simulation of influenza epidemics, PLOS ONE 7 (10) (2012) 0045414.

[12] E. O. Nsoesie, J. S. Brownstein, N. Ramakrishnan, M. V. Marathe, A systematic review of studies on forecasting the dynamics of influenza outbreaks, Influenza and other respiratory viruses 8 (3) (2014) 309–316.

[13] J. Herron, V. Hajric, 'The Market's in Panic Mode.' Stock Markets Plunge 12% Amid Coronavirus Fears, [Online; accessed 21-March-2020] (2020).
https://time.com/5803847/coronavirus-stocks-fall/

[14] O. M. Cliff, M. Harding, M. Piraveen, Y. Erten, M. Gambhir, M. Prokopenko, Investigating spatiotemporal dynamics and synchrony of influenza epidemics in Australia: an agent-based modelling approach, Simulation Modelling Practice and Theory 87 (2018) 412–431.

[15] C. Zachreson, K. Fair, O. M. Cliff, M. Harding, M. Piraveenan, M. Prokopenko, Urbanization affects peak timing, prevalence, and bimodality of influenza pandemics in Australia: results of a census-calibrated model, Science Advances 4 (2018) eaau5294.

[16] N. Harding, R. E. Spinney, M. Prokopenko, Phase transitions in spatial connectivity during influenza pandemics, Entropy 22 (2) (2020) 133.

[17] C. Zachreson, K. Fair, M. Harding, M. Prokopenko, Interfering with influenza: nonlinear coupling of reactive and static mitigation strategies, Journal of Royal Society Interface, accepted.

[18] M. E. Halloran, I. M. Longini, A. Nizam, Y. Yang, Containing bioterrorist smallpox, Science 298 (5597) (2002) 1428–1432.

[19] S. Eubank, H. Guclu, V. A. Kumar, M. V. Marathe, A. Srinivasan, Z. Toroczkai, N. Wang, Modelling disease outbreaks in realistic urban social networks, Nature 429 (6988) (2004) 180.

[20] I. M. Longini, M. E. Halloran, A. Nizam, Y. Yang, Containing pandemic influenza with antiviral agents, American Journal of Epidemiology 159 (7) (2004) 623–633.

[21] T. C. Germann, K. Kadau, I. M. Longini, C. A. Macken, Mitigation strategies for pandemic influenza in the United States, Proceedings of the National Academy of Sciences 103 (15) (2006) 5935–5940.

[22] C. Barrett, K. Bisset, J. Leidig, A. Marathe, M. V. Marathe, An integrated modeling environment to study the co-evolution of networks, individual behavior and epidemics, AI Magazine 31 (1) (2010) 75–87.

[23] D. Balcan, B. Gonçalves, H. Hu, J. J. Ramasco, V. Colizza, A. Vespignani, Modeling the spatial spread of infectious diseases: The global epidemic and mobility computational model, Journal of Computational Science 1 (3) (2010) 132–145.

[24] D. L. Chao, M. E. Halloran, V. J. Obenchain, I. M. Longini Jr, FluTE, a publicly available stochastic influenza epidemic simulation model, PLoS Computational Biology 6 (1) (2010) e1000656.

[25] K. R. Bisset, A. M. Aji, E. Bohm, L. V. Kale, T. Kamal, M. V. Marathe, J.-S. Yeom, Simulating the spread of infectious disease over large realistic social networks using charm++, in: Parallel and Distributed Processing Symposium Workshops & PhD Forum (IPDPSW), 2012 IEEE 26th International, IEEE, 2012, pp. 507–518.

[26] N. M. Ferguson, D. Laydon, G. Nedjati-Gilani, N. Imai, K. Ainslie, M. Baguelin, S. Bhatia, A. Boonyasiri, Z. Cucunubá, G. Cuomo-Dannenburg, et al., Impact of non-pharmaceutical interventions (NPIs) to reduce COVID-19 mortality and healthcare demand, Preprint, Imperial College COVID-19 Response Team.

[27] K. M. Fair, C. Zachreson, M. Prokopenko, Creating a surrogate commuter network from Australian Bureau of Statistics census data, Scientific data 6 (2019) 150.

[28] F. Carrat, E. Vergu, N. M. Ferguson, M. Lemaitre, S. Cauchemez, S. Leach, A.-J. Valleron, Time lines of infection and disease in human influenza: a review of volunteer challenge studies, American Journal of Epidemiology 167 (7) (2008) 775–785.

[29] N. H. Leung, C. Xu, D. K. Ip, B. J. Cowling, The fraction of influenza virus infections that are asymptomatic: a systematic review and meta-analysis, Epidemiology (Cambridge, Mass.) 26 (6) (2015) 862.

[30] L. Feng, P. Yang, T. Zhang, J. Yang, C. Fu, Y. Qin, Y. Zhang, C. Ma, Z. Liu, Q. Wang, et al., Technical guidelines for the application of seasonal influenza vaccine in China (2014–2015), Human Vaccines & Immunotherapeutics 11 (8) (2015) 2077–2101.

[31] W.-j. Guan, Z.-y. Ni, Y. Hu, W.-h. Liang, C.-q. Ou, J.-x. He, L. Liu, H. Shan, C.-l. Lei, D. S. Hui, et al., Clinical characteristics of coronavirus disease 2019 in China, New England Journal of Medicine.

[32] R. Li, S. Pei, B. Chen, Y. Song, T. Zhang, W. Yang, J. Shaman, Substantial undocumented infection facilitates the rapid dissemination of novel coronavirus (SARS-CoV2), Science.

[33] A. J. Kucharski, T. W. Russell, C. Diamond, Y. Liu, J. Edmunds, S. Funk, R. M. Eggo, F. Sun, M. Jit, J. D. Munday, et al., Early dynamics of transmission and control of COVID-19: a mathematical modelling study, The Lancet Infectious Diseases.

[34] Q. Li, X. Guan, P. Wu, X. Wang, L. Zhou, Y. Tong, R. Ren, K. S. Leung, E. H. Lau, J. Y. Wong, et al., Early transmission dynamics in Wuhan, China, of novel coronavirus–infected pneumonia, New England Journal of Medicine.

[35] N. M. Linton, T. Kobayashi, Y. Yang, K. Hayashi, A. R. Akhmetzhanov, S.-m. Jung, B. Yuan, R. Kinoshita, H. Nishiura, Incubation period and other epidemiological characteristics of 2019 novel coronavirus infections with right truncation: a statistical analysis of publicly available case data, Journal of Clinical Medicine 9 (2) (2020) 538.

[36] H. Huang, Y. Wang, Z. Wang, Z. Liang, S. Qu, S. Ma, G. Mao, X. Liu, Epidemic features and control of 2019 novel coronavirus pneumonia in Wenzhou, China, Preprints with The Lancet.

[37] Q. Bi, Y. Wu, S. Mei, C. Ye, X. Zou, Z. Zhang, X. Liu, L. Wei, S. A. Truelove, T. Zhang, et al., Epidemiology and transmission of COVID-19 in Shenzhen China: Analysis of 391 cases and 1,286 of their close contacts, medRxiv.

[38] S. Cauchemez, A. Bhattarai, T. L. Marchbanks, R. P. Fagan, S. Ostroff, N. M. Ferguson, D. Swerdlow, S. V. Sodha, M. E. Moll, F. J. Angulo, et al., Role of social networks in shaping disease transmission during a community outbreak of 2009 H1N1 pandemic influenza, Proceedings of the National Academy of Sciences 108 (7) (2011) 2825–2830.

[39] R. Antia, R. R. Regoes, J. C. Koella, C. T. Bergstrom, The role of evolution in the emergence of infectious diseases, Nature 426 (6967) (2003) 658–661.

[40] E. Erten, J. Lizier, M. Piraveenan, M. Prokopenko, Criticality and information dynamics in epidemiological models, Entropy 19 (2017) 194.

[41] N. Harding, R. Nigmatullin, M. Prokopenko, Thermodynamic efficiency of contagions: a statistical mechanical analysis of the sis epidemic model, Interface Focus 8 (2018) 20180036.

[42] B. Gemeinholzer, Phylogenetic networks, Analysis of biological networks (2008) 255–282.

[43] M. Piraveenan, M. Prokopenko, A. Y. Zomaya, Assortativeness and information in scale-free networks, European Physical Journal B 67 (2009) 291–300.

[44] B. H. Junker, F. Schreiber, Analysis of biological networks, Vol. 2, John Wiley & Sons, 2011.

[45] M. Piraveenan, M. Prokopenko, A. Y. Zomaya, Assortative mixing in directed biological networks, IEEE/ACM Transactions on Computational Biology and Bioinformatics 9 (2012) 66–78.

[46] O. Cliff, V. Sintchenko, T. C. Sorrell, K. Vadlamudi, N. Mclean, M. Prokopenko, Network properties of Salmonella epidemics, Scientific Reports 9 (2019) 6159.

[47] B. J. Marais, S. Williams, A. Li, R. Ofrin, A. Merianos, J. Negin, J. Firman, R. Davies, T. Sorrell, Improving emergency preparedness and response in the Asia-Pacific, BMJ Global Health 4 (1) (2019) e001271.

[48] S. L. Chang, M. Piraveenan, M. Prokopenko, The effects of imitation dynamics on vaccination behaviours in SIR-network model, International Journal of Environmental Research and Public Health 16 (2019) 2477.

[49] S. L. Chang, M. Piraveenan, P. Pattison, M. Prokopenko, Game theoretic modelling of infectious disease dynamics and intervention methods: a review, Journal of Biological Dynamics 14 (1) (2020) 57–89.

[50] M. Porta, A dictionary of epidemiology, Oxford University Press, 2014.

[51] M. E. Halloran, N. M. Ferguson, S. Eubank, I. M. Longini, D. A. Cummings, B. Lewis, S. Xu, C. Fraser, A. Vullikanti, T. C. Germann, et al., Modeling targeted layered containment of an influenza pandemic in the United States, Proceedings of the National Academy of Sciences 105 (12) (2008) 4639–4644.

[52] J. Mossong, N. Hens, M. Jit, P. Beutels, K. Auranen, R. Mikolajczyk, M. Massari, S. Salmaso, G. S. Tomba, J. Wallinga, et al., Social contacts and mixing patterns relevant to the spread of infectious diseases, PLoS medicine 5 (3) (2008) e74.

[53] J. C. Miller, Spread of infectious disease through clustered populations, Journal of the Royal Society Interface 6 (41) (2009) 1121–1134.

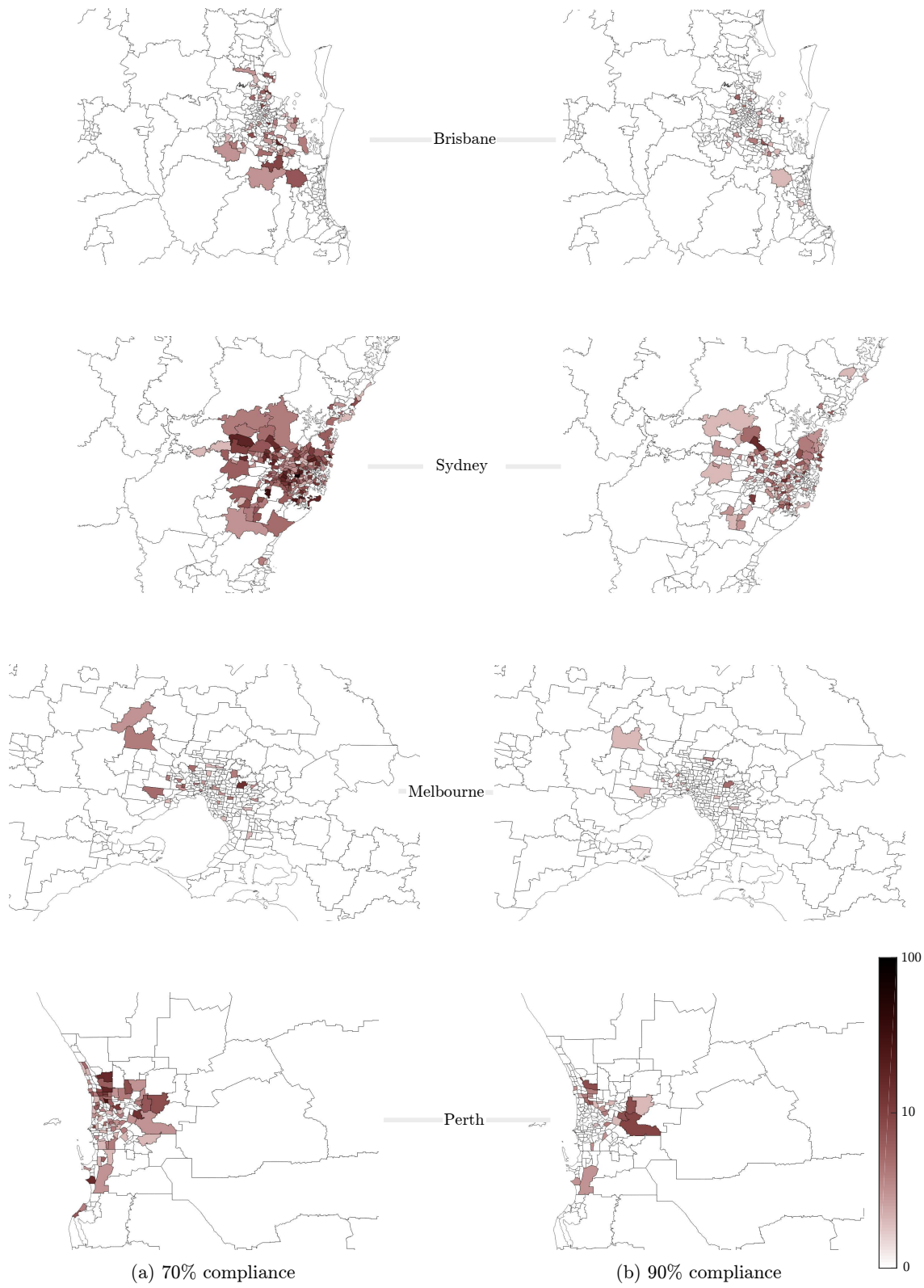[54] Australian Statistical Geography Standard, https://www.abs.gov.au/ausstats/abs@.nsf/Lookup/2901.0Chapter23102011, accessed: 08-10-2019.

Figure 12: Choropleths of four largest Australian Capital Cities: prevalence on a log scale at day 60.

JK

Jared Knofczynski
Undergraduate Research Assistant
University of Oregon

jared@cs.uoregon.edu

https://github.com/j-red

Work Experience

Research Assistant
OCTOBER 2020 — PRESENT

Conducting research in Machine Learning applications to Network Security problems.

Undergraduate Researcher
JUNE 2020 — AUGUST 2020

Conducted research on viral transmission rates of airborne pathogens in university classrooms using computational modeling and agent-based simulation systems.

Computer Science Learning Assistant
SEPTEMBER 2019 — JUNE 2020

Aided professors in the 200-level Computer Science sequence by holding office hours and providing one-on-one student assistance.

Assistant Director at iD Tech Camps
LEWIS AND CLARK CAMPUS, PORTLAND, OREGON
JUNE 2019 — SEPTEMBER 2019

Assisted camp director in overseeing daily operations while also teaching students the fundamentals of graphic design, Python coding, and cybersecurity.

Education

Computer and Information Science
Clark Honors College, University of Oregon

SEPTEMBER 2018 — PRESENT

Studying Computer Science and algorithmic problem solving. Additional minors in Math and Music Technology.

Undergraduate GPA: 4.11   Major GPA: 4.11

Academic Honors

Ripple University Blockchain Research Initiative Scholarship Recipient

2019, 2020

For students interested in conducting research related to cybersecurity as enhanced by secure digital transactions, blockchain and other distributed systems, cryptocurrency, cryptography, and more.

Skills

Coding Languages

Python
JavaScript
C, Java
HTML/CSS
d3.js

Software Programs

Blender
Adobe Creative Suite
Git
Unity
Linux/Unix OS