Simulation suggests that rapid activation of social distancing can arrest epidemic development due to a novel strain of influenza

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Abstract

Background: Social distancing interventions such as school closure and prohibition of public gatherings are present in pandemic influenza preparedness plans. Predicting the effectiveness of intervention strategies in a pandemic is difficult. In the absence of other evidence, computer simulation can be used to help policy makers plan for a potential future influenza pandemic. We conducted simulations of a small community to determine the magnitude and timing of activation that would be necessary for social distancing interventions to arrest a future pandemic.

Methods: We used a detailed, individual-based model of a real community with a population of approximately 30,000. We simulated the effect of four social distancing interventions: school closure, increased isolation of symptomatic individuals in their household, workplace nonattendance, and reduction of contact in the wider community. We simulated each of the intervention measures in isolation and in several combinations; and examined the effect of delays in the activation of interventions on the final and daily attack rates.

Results: For an epidemic with an R₀ value of 1.5, a combination of all four social distancing measures could reduce the final attack rate from 33% to below 10% if introduced within 6 weeks from the introduction of the first case. In contrast, for an R₀ of 2.5 these measures must be introduced within 2 weeks of the first case to achieve a similar reduction; delays of 2, 3 and 4 weeks resulted in final attack rates of 7%, 21% and 45% respectively. For an R₀ of 3.5 the combination of all four measures could reduce the final attack rate from 73% to 16%, but only if introduced without delay; delays of 1, 2 or 3 weeks resulted in final attack rates of 19%, 35% or 63% respectively. For the higher R₀ values no single measure has a significant impact on attack rates.

Conclusion: Our results suggest a critical role of social distancing in the potential control of a future pandemic and indicate that such interventions are capable of arresting influenza epidemic development, but only if they are used in combination, activated without delay and maintained for a relatively long period.
Background

Concern exists that the avian H5N1 influenza virus may become readily transmissible between humans, leading to a pandemic with significant mortality [1].

Social distancing interventions, such as school closure, reducing workplace numbers, reducing social and community contacts, and increasing home isolation are embedded within the pandemic influenza preparedness plans of most countries [2-4] and appear in current WHO recommendations. Social distancing interventions are important as they represent the only type of intervention measure guaranteed to be available against a novel strain of influenza in the early phases of a pandemic. The goal of these interventions is to reduce the overall illness attack rates and the consequential excess mortality attributed to the pandemic, and to delay and reduce the peak attack rate, reducing pressure on health services and allowing time to distribute and administer antiviral drugs and, possibly, suitable vaccines.

Modelling [5] has suggested that early interventions which increase social distancing may postpone the time to reach peak attack rates and limit the total number of cases and deaths attributed to pandemic influenza. This theoretical work has recently been supported by archival studies of excess deaths attributed to the 1918–19 pandemic in the largest US cities [6] and by the work of [7] for an Australian city. While these studies show that the historically implemented measures were not effective in preventing any local epidemics, they do show a strong correlation between the delay in introduction of intervention measures and excess mortality (both total and peak).

However, the potential impact and possible limitations of social distancing measures are not fully understood. An evaluation of the evidence base for non-pharmaceutical interventions concluded that there is a general lack of scientific evidence or expert consensus for school closure, workplace closure or banning of public gatherings during a pandemic [8].

Epidemiological simulation models have been used to analyse the effects of alternative containment measures. Various studies have simulated influenza pandemics at the scale of the whole world [9], whole countries [10-13], and individual communities [14,15]. A picture that emerges from a comparison of such simulation studies is that the predicted efficacy of social distancing intervention measures can depend strongly on the particular assumptions made about the operation of each intervention [15]. For example, little can be predicted about the outcome of school closure without specifying the contact behaviour of students when schools are closed, the timing of introduction of closure, or the other intervention measures that are concurrently in effect.

The purpose of this study was to extend the scope of simulations of social distancing interventions in an influenza pandemic by examining several important assumptions which have been not previously studied in a systematic way. We present results from an examination of the timing-of-activation and combination of social distancing interventions to determine how these factors impact their effectiveness, and thus to inform policy decisions regarding reactive strategies for mitigating the effects of an influenza pandemic.

Methods

In previous work, we constructed a detailed, individual-based model of a real community in the south west of Western Australia (Albany) with a population of approximately 30,000, and applied the model to conduct simulations of the spread of pandemic influenza – full details of the model can be found in [15].

We used census data and state and local government data to construct a population of virtual individuals and households that matched the age and household structure of the real town. Individuals were also grouped into a number of “contact hubs” such as schools, child care facilities, adult educational facilities, workplaces and the regional hospital. Additional random contact in the community was modelled, with contacts biased towards meetings between individuals with nearby household locations.

Each simulation proceeded in a sequence of 12-hour cycles. During each cycle, a nominal location of each individual was calculated; taking into account the type of cycle (day or night, weekend or weekday), the contact hub the individual was a member of (if any), and the infection status of the individual and so forth. Individuals occupying the same household or contact hub during the same cycle were deemed to come into potentially infectious contact.

When a susceptible and infectious individual came into contact, a probability of infection transmission was calculated, based on the underlying infectivity of the viral strain, the age of the susceptible individual, and the progress and severity of the infection of the infectious individual. Influenza infection was assumed to proceed for 6 days, with 1 day latent, 1 day asymptomatic and infectious, followed by 4 days infectious (either symptomatic or asymptomatic). Although there is little evidence for spread from asymptomatic subjects for pandemic influenza, we adopted the conservative assumption that the proportion of individuals experiencing asymptomatic infection matched that of seasonal influenza.
Full details of the methodology and data sources used to select the various model parameter values can be found in [15], and the resulting parameter value settings are recorded in Supporting Information Text S1 of that publication. Since the infectivity of an epidemic arising from a new strain of influenza is uncertain, we simulated epidemics with basic reproduction numbers ($R_0$) of 1.5, 2.5 and 3.5, which, assuming no intervention, gave final symptomatic attack rates of 33%, 65% and 73% respectively. Characteristics of these baseline epidemics are given in Table 1.

For the study described in this paper we extended that model to allow delayed introduction of social distancing intervention measures, and conducted a new simulation experiment series. We simulated four different social distancing intervention measures. For each measure (and for several combinations of simultaneously applied measures), we simulated the effect of introducing each intervention measure (and several combinations of simultaneously applied measures) at different points in time, ranging from 0 to 8 weeks after the introduction of the first infectious case into the community. Once introduced, it was assumed that interventions continued until the end of the simulation.

The four intervention measures simulated were as follows:

**School closure**
We assumed that when schools were closed, students and teachers spent weekday daytime cycles at home rather than at school. This meant that no contact took place at that school, but that these individuals would contact any other individuals present in their household during the day. We also assumed that if school closure would result in a child being present in a household alone, one adult from the household stayed home (and did not make hub contacts).

**Increased case isolation**
When the increased case isolation intervention was in effect, there was a 90% (100% for children) chance that, upon becoming symptomatic, an adult (or child) would withdraw to their household for the duration of their infection (in the no-intervention case these probabilities were 50% for adults and 90% for children). We assumed that withdrawn individuals made only household contacts thereafter.

**Workplace non-attendance**
When this measure was in effect, each person attending a workplace had a 50% chance each day of staying home instead of attending the workplace. Individuals staying at home made contacted all other individuals at home during the day.

**Community contact reduction**
When this measure was in effect, individuals made half as many community contacts with other individuals per day.

### Results

**Impact of intervention activation delay on epidemic attack rates**
We use a final symptomatic attack rate of <10% as a criterion for determining that an epidemic due to a novel pandemic strain of influenza has been prevented. For an epidemic with an $R_0$ value of 1.5, we found that the only single intervention measure capable of preventing an epidemic was the 90% case isolation measure, and only if applied within 3 weeks. We found that a combination of all four social distancing measures could reduce the final attack rate from 33% to 9% if introduced within 6 weeks from the introduction of the index case. If applied preemptively, anticipating the arrival of the index case into the otherwise uninfected community, this combination of measures reduces the total attack rate to 1.6%, with a correspondingly significant reduction in attendant mortality rates. Figure 1 shows the relationship between final attack rate and the delay in intervention activation.

| Characteristic                        | $R_0 = 1.5$ | $R_0 = 2.5$ | $R_0 = 3.5$ |
|---------------------------------------|-------------|-------------|-------------|
| Final infection rate (%)              | 39.7        | 79.7        | 91.2        |
| Final attack rate (%)                 | 33.3        | 64.8        | 73.2        |
| Peak symptomatic population (%)       | 4.8         | 25.7        | 43.0        |
| Peak daily attack rate (per 10,000)   | 87          | 481         | 856         |
| Peak attack day                       | 57          | 29          | 20          |
| Serial interval                       | 2.97        | 2.74        | 2.45        |

This table gives characteristics of simulated baseline epidemics (that is, epidemics where no intervention measures were applied) with $R_0$ values of 1.5, 2.5 and 3.5. The mean and standard deviation (S.D.) are for 40 independent randomly seeded simulation runs.
rates and intervention delays for each intervention measure on its own and the combination of all measures.

In contrast, for an epidemic with an $R_0$ value of 2.5 the combination of all intervention measures must be introduced within 2 weeks of the index case to prevent an epidemic developing; delays of 2, 3 and 4 weeks resulted in final attack rates of 7%, 21% and 45% respectively (see Figure 1). No single intervention measure could reduce the final attack rate to less than 48%, even if activated preemptively. While not controlling the epidemic, the combination of school closure plus 90% case isolation approximately halved the final attack rate (from 65% to 31%), illustrating the value of layering multiple intervention measures, especially for high values of $R_0$.

For an epidemic with an $R_0$ of 3.5, perhaps considered to be a worst-case scenario, our results indicate that the combination of all interventions was unable to reduce the final illness attack rate to less than 10% and unable to prevent an epidemic occurring. However, the rapid activation of measures may significantly arrest epidemic development, giving final attack rates of 16%, 19% and 35% if activated pre-emptively or with a 1 or 2 week delay, respectively.

We found a similar effect of intervention delay on peak daily attack rates (see Figure 2). For an epidemic with an $R_0$ of 1.5, any of the intervention measures except 50% workplace non-attendance reduced the peak daily attack rate from 90 cases per 10,000 to below 35 if introduced within 4 weeks. For an $R_0$ of 2.5, only the combination of all measures applied within 2 weeks could reduce the peak daily attack rate from 474 to below 35 cases per 10,000. Delays of 2, 3 or 4 weeks resulted in peak daily attack rates of 28, 151 or 422 cases per 10,000 respectively. To put this
into context, if it is assumed that 1% of cases require hospitalisation [16], delays of 2, 3 or 4 weeks would require the 120-bed regional hospital in the 30,000 person community to handle 0.84, 4.5 or 12.7 influenza admissions per day at the peak of the epidemic, respectively. For an epidemic with an $R_0$ of 3.5, the situation is even more stark: the combination of all measures applied within one week reduced the peak daily attack rate from 856 to 33; delays of 2 or 3 weeks resulted in peak daily attack rates of 187 or 801 cases per 10,000 respectively. Figure 2 shows the relationship between peak daily attack rates and intervention measure activation delay.

In order to illustrate the effect of delays in activation of intervention measures on the time course of an epidemic, Figure 3 shows cumulative and daily attack rate epidemic curves for school closure, for delays of between 2 and 8 weeks. The figure clearly shows the contrast between epidemics with $R_0$ values of 1.5 and 2.5. In the former case the rate of infection peaks on day 58; pre-emptive school closure is capable of making large reductions in final and peak daily attack rates, and each additional delay of 2 weeks steadily reduces the effectiveness of the intervention. In the latter case the epidemic peaks on day 28; school closure does not make a large reduction in final attack rate even if applied pre-emptively, and reduction in peak daily attack rate declines suddenly between delays of 2 and 4 weeks.

**Relationship between intervention activation delay and observed case trigger thresholds**

We have examined the effect of delaying intervention in terms of the time between the first infected individual appearing in the population and the activation of intervention measures. In reality, it is unlikely that the first case of pandemic influenza in a community would be identi-
Figure 3
Epidemic curves for school closure for a range of activation delays. Cumulative (top) and peak daily (bottom) attack rates are shown for epidemics with unmitigated $R_0$ values of 1.5 (left) and 2.5 (right).
half of the 18–64 age group, or 29%; compared to all of
approximately the same sized sub-population each day:

The interventions would need to continue for approximately 5
months to prevent an epidemic with an \( R_0 \) of 2.5, which is
clearly unrealistic. While long-term imposition of socially
and economically disruptive measures is not possible,
social distancing interventions may be used to buy time
for the establishment of an antiviral containment pro-
gramme and/or the distribution of a vaccine [4,3,2].

The historical record indicates that social distancing
measures may be implemented, relaxed and sometimes re-

Impact of interventions on age-specific attack rates

Our results showed that the simulated intervention mea-
sures affected age groups differently. The interventions
that caused the largest deviation from the baseline age-
specific attack rate profile were school closure, which
caus a larger proportional reduction in the attack rate of
the school age range (6–17 years) compared to other age
groups; and workplace nonattendance, which resulted in
a proportionally larger reducing the attack rate of the 18–
64 age group. The age-specific attack rates for the baseline
epidemic (which was calibrated to resemble that of sea-
onal influenza), and epidemics mitigated by each of the
interventions would need to continue for approximately 5
months to prevent an epidemic with an \( R_0 \) of 2.5, which is
clearly unrealistic. While long-term imposition of socially
and economically disruptive measures is not possible,
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Discussion

We consider social distancing interventions for a number of
reasons: to better understand the effect which individ-
ual social distancing measures have on the attack rate and
consequential mortality rate; to determine their sensitivity
to the time of activation; to address scenarios where sup-
plies of antiviral drugs and suitable vaccines are in limited
supply, due to an outbreak occurring in a country
with little access to these resources, due to logistical
delays in their distribution and administration or due to
the unavailability of an appropriately “typed” vaccine,
particularly at the early states of a pandemic.

Antiviral drugs and social distancing interventions share
several common characteristics: neither confers long-last-
ing immunity, and both deplete limited resources once in
operation (a drug stockpile on one hand, and public
patience on the other). The use of antiviral drugs is a core
feature of the pandemic preparedness plans of many
countries, such as the United States, the United Kingdom,
and Australia [4,3,2]. Their use in an influenza pandemic
is however an untested strategy: more experience is
needed to determine their likely effectiveness and optimal
use, especially given the possibility of the development of
antiviral drug resistance during or prior to a pandemic
[17–19]. It therefore seems prudent to consider social dis-
 tancing interventions as an alternative or complement to
antiviral-based strategies.

Social distancing interventions are important as they rep-

implemented [6,7,20]. Other models have investigated the optimal timing for rescinding and re-implementing social distancing interventions [21]. However our results show what may be expected of social distancing interventions when implemented early and maintained indefinitely (or in practical terms, until an effective vaccination programme has been completed); and we establish maximum activation delays allowable if such interventions are to fulfil their potential.

The results are applicable to industrialised populations and are possibly not applicable to developing countries having lower population mobility and/or higher population densities. In such countries we may find higher daily contact rates and hence reduced opportunities for limiting contact and in achieving isolation in the household by non-pharmaceutical means.

When comparing our results to those of other models, differences arise which may be due to alternative assumptions being made regarding the demographics and contact behaviour of the population, and to different assumptions regarding interventions and the methods for deploying them. However, we are able to comment at a general level on how our results relate to that of methodologically similar studies.

The work of Glass et al [5] most closely resembles that of ourselves, whereby they utilise a population of 10,000 individuals and, like us, examine only non-pharmaceutical interventions. Their model represents the estimated structure and contact patterns of a synthetic town in the USA; it is unclear to us how the differences in the detail modelled, between an actual population (Albany, Australia in our case) and this synthetic town affect the quality of the results obtained. The results coincide well for an $R_0$ of 1.5 to 1.6, when considering school closure as the only intervention. With an $R_0$ of 2.5 and all non-pharmaceutical interventions activated together, the results in [5] suggest a reduction in the illness attack rate in the range

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**Figure 4**

**Age-specific attack rates for social distancing interventions.** Final attack rates are shown for each of 7 age groups for a baseline (unmitigated) epidemic, and for epidemics mitigated by 4 intervention measures. An $R_0$ value of 1.5 is assumed; interventions are assumed to be applied pre-emptively.
10% to 20%, depending on variation of the contact patterns assumed. By contrast, the results presented here suggest that an attack rate of as low as 3% may result if all interventions are activated optimally, that is within the first week of the arrival of an outbreak-producing index case and held indefinitely. One difference which may explain this variance is that Glass et al assume a threshold of 10 diagnosed cases in a school before closure is effected compared with the optimal strategy adopted by ourselves. This again highlights the significance of rapid intervention if we are to prevent an epidemic, or to substantially reduce its rate of growth.

In comparing whole-country models such as that produced for the USA by Germann et al [13], the only control interventions which can be directly compared is that of school closure in isolation. The simulated attack rate for unmitigated epidemics with $R_0 = 1.5/1.6$ are reduced to 1% and 13%, comparing the Germann et al result with that presented here. What is perhaps more interesting is that for $R_0$’s in the range 1.9 to 2.4 significant reductions in attack rates to a level where an epidemic may be prevented are only achieved by Germann et al by combining non-pharmaceutical interventions with either targeted antiviral prophylaxis or vaccination, with the exception of child-first vaccination for an $R_0$ of 1.9. Our results suggest that for $R_0$’s up to and including 2.5 epidemics may be prevented by combined non-pharmaceutical interventions alone, provided they are activated immediately and are sustained indefinitely. Given the logistics of vaccination and antiviral drug deployment it is highly likely that non-pharmaceutical interventions may be activated more rapidly and our results suggest a similar ability to prevent epidemic development as that achieved by a combined pharmaceutical/non-pharmaceutical strategy.

The need to react rapidly when activating interventions is also highlighted by Ferguson et al [12] where antiviral treatment is very sensitive to initial delays of 24 hours, due to the use of an early, peaked infectiousness function. This contrasts with our more abstract flat infectivity profile. Single non-pharmaceutical interventions, such as school closure or home quarantine, are shown to have little impact for $R_0$ in the range 1.7 – 2.0, whilst more significant reductions are suggested in the results that we derive. Similarly, school closure and 50% workplace reduction has less effect (an approximately 4 percentage point reduction in attack rate) in the Ferguson et al model compared to our results, where the suggested reduction in attack rate is of the order of 20 percentage points. While there are clear differences in assumptions between the two models and direct comparison is difficult, key factors may be their requirement to diagnose one case in a school before closure is effected, and their assumption that additional community contact occurs when schools are closed.

The range of modelling estimates for the potential effectiveness of social distancing interventions such as school closure is considerable, and may stem from the range of modelling assumptions about the operation of school closure and associated behavioural changes of individuals [15]. Observations of actual school closures do not seem to provide conclusive evidence on the effectiveness of school closure. Based on observations made during a teacher’s strike in Israel, it was estimated by Heymann et al [22] that diagnoses of respiratory infections decreased by 42%. In contrast Cowling et al [23] observed that a school closure episode in Hong Kong in 2008 had little impact on influenza attack rates – although in that case school closure appears to have occurred after the epidemic peak. The largest scale study known to the authors that provides an estimate of the effectiveness of school closure on influenza epidemics is the work of Cauchemez et al [24]. Based on surveys of seasonal influenza during and between school terms in France, this work estimated that school closure could achieve at most a 17% reduction in attack rates, indicating that school closure may not be as effective as predicted our model.

**Conclusion**

Our results suggest a critical role of combined social distancing measures in the potential control of a future pandemic. They indicate that non-pharmaceutical social distancing interventions are capable of preventing influenza epidemics with $R_0$ values of up to 2.5, and of significantly reducing the rate of development and overall burden of epidemics with $R_0$ values of up to 3.5, but only if used in combination, activated without delay, and
maintained for a relatively long period. Our results also confirm the importance of rapid, decisive and robust action if social distancing interventions are to be useful in pandemic control. While such draconian measures seem unlikely to be mandated given their impact on personal freedom, they appear to have a key role to play in delaying the development of a ‘worst case’ influenza epidemic (i.e. with a reproductive value of 3.5). They may be critical in holding back an epidemic until vaccines are deployed on a sufficient scale that subsequent relaxation of these rigorous measures will not result in a consequential acceleration in the scale of the outbreak.

Competing interests
The authors declare that they have no competing interests.

Authors’ contributions
GM and JK were responsible for the conception and design of the simulation experiments. HK contributed epidemiological and public health expertise that informed model parameters. JK was responsible for software development and conducted simulation experiments. All authors were involved in analysis of simulation results and writing the manuscript.

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