Author's response to reviews

Title: How Variations Among States' Plans and Statutes Alter the Impact of School Closure as an Influenza Control Strategy

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Author's response to reviews: see over
May 16, 2012

Jimmar Dizon  
Journal Editorial Office  
BioMed Central  
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Re: Research Article, MS: 1280082065704517

Dear Dr. Dizon:

Submitted herewith to BioMed Central (http://www.biomedcentral.com/my/) is the revised manuscript “How Variations Among States’ Plans and Statutes Alter the Impact of School Closure as an Influenza Control Strategy” (previously submitted as “Closing Schools as an Influenza Control Strategy: Modeling the State-Specific Variations in Plans and Statues”).

Two versions are included in this re-submission: one clean copy and another using “track-changes” to highlight differences from the original submission. The “track-changes” version is attached to this cover letter. Please note that the addition of two references produced a re-numbering of all references not identified by track-changes. The document “Additional File 1” is also resubmitted, due to the change in reference numbering.

Specifically, we address your comments and recommendations with the following changes:

- Title and abstract are modified to emphasize the article’s focus on studying the implications of school-closure specifications in state law and planning documents.
- Earn et al. and Laskowski et al. articles are cited in several sections of the manuscript, noting important areas of distinction and concurrence with our reported work.
- All authors declare no competing interests.

After reviewing the editorial policies of BMC Public Health, we find no further disclosures to be necessary at this time. Recommended reviewers can be provided upon your request.

Thank you for your consideration.

Very truly yours,

Margaret A. Potter, JD, MS  
Associate Dean and Director
How Variations Among States’ Plans and Statutes Alter the Impact of School Closure as an Influenza Control Strategy

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Abstract

Background. States’ pandemic influenza plans and school closure statutes are intended to guide state and local officials, but most faced a great deal of uncertainty during the 2009 influenza H1N1 epidemic. Questions remained about whether, when, and for how long to close schools and about which agencies and officials had legal authority over school closures.

Methods. This study began with analysis of states’ school-closure statutes and pandemic influenza plans to identify the variations among them. An agent-based model of one state was used to represent as constants a population’s demographics, commuting patterns, work and school attendance, and community mixing patterns while repeated simulations explored the effects of variations in school closure authority, duration, closure thresholds, and reopening criteria.

Results. School-level closure criteria appear to be important in disease control because influenza arrives in localities at different times, and commuting and mixing patterns vary locally. The results show no basis on which to justify statewide rather than school-specific or community-specific authority for school closures. Nor do these simulations offer evidence to require school closures promptly at the earliest stage of an epidemic.

Conclusions. This agent-based simulation suggests several ways to improve statutes and influenza plans. First, school closure should remain optional for influenza mitigation. Second, influenza plans need not necessarily specify the threshold for school closures but should clearly define provisions for early and ongoing local monitoring. Finally, school closure authority may be exercised at the statewide or local level, so long as decisions are informed by monitoring incidence in local communities and schools.
Background

School closure has long been considered a useful social-distancing strategy to control the spread of infectious diseases among children [1-4], who are efficient transmitters of influenza virus [5, 6] and whose immunity to circulating virus strains may be lower than that of adults [7]. School closures may delay an epidemic peak, allowing more time for vaccine distribution and readying the healthcare system (e.g., increasing available beds)[8].

Nevertheless, state and local officials faced uncertainty during the 2009 influenza H1N1 epidemic about whether, when, and for how long to close schools. Between April and May, the World Health Organization and the U.S. Centers for Disease Control and Prevention changed their advice from recommending closures unequivocally to deferring to state and local decision makers[9, 10]. But there was inconsistency as to which agencies and officials had legal authority over school closures [11]. Moreover, the evidence base for when and how long to close schools was sparse [12]. Though ordered for some schools in spring, summer, and fall of 2009, school-closures were determined typically by local school districts and individual schools; and closure durations rarely exceeded a few days [13, 14]. It was reported that H1N1 school closure experience had much local variation in decision-making authority, disagreement between school administrators and public health officials, and lack of clarity about the goals for closure [15]. A review of laboratory-confirmed H1N1 cases in Alberta, Canada found that the 2009 summertime closure of schools interrupted virus transmission among school-aged children but failed to prevent a second wave of the pandemic when schools re-opened in the fall[16].
The relatively mild transmissibility and severity of the 2009 H1N1 disease contrasts with the reasonable expectation that another more virulent novel pandemic disease will occur in the future—one accompanied by surging demand for healthcare services, lack of a vaccine, and high morbidity and mortality rates. The need remains to augment the evidence base and to plan meaningfully for school closures.

First, should school closure authority as an influenza mitigation strategy be centralized statewide or decentralized locally? A single, statewide official with recognized authority over all school districts and localities might have greater effect in closing schools uniformly across a state; whereas distributed authority among officials or decentralized authority structure might cause delay or non-uniformity in the timing of school closures decisions and implementation. The effects of these policy alternatives on the spread of infection are unknown.

Second, how early—that is, at what prevalence threshold—should schools close to mitigate a pandemic? School closures are costly [17-19], burdensome on workers and families [20], and a potential cause of absenteeism among public health workers [21]. Caution in imposing such economic and social burdens must be balanced with the health benefits; but neither past experience nor empirical study has yet suggested sound metrics to guide such decision-making.

Third, for how long should schools remain closed? A previous study showed that increasing the duration of school closures reduced the attack rate of infections in a county population and that limiting the duration to two weeks or less—when virus was still circulating—was associated with increased attack rates [8]. Without empirical evidence,
the high costs of school closure will exert pressure on school districts, individual schools, and geographic localities to reopen too soon.

Establishing an evidence base for pandemic planning is challenging, since school closure alternatives cannot be tested in controlled observational studies, experience with one influenza outbreak cannot necessarily be generalized to others, and isolating school closure as a mitigation strategy from other social-distancing or pharmaceutical interventions is infeasible in practice. The present study avoided the limitations of observational studies by using a large-scale computational model to provide insights and guidance for policy makers. Here, we conducted repeated simulations of epidemics with specifications and assumptions held constant while varying school closure policies and observing the outcomes.

**Methods**

This study began with analysis of school-closure statutes and published pandemic influenza plans in all fifty U.S. states and interpreted the variations among them for application to modeling experiments. We chose one state (Pennsylvania) to represent as constants a population’s demographics, commuting patterns, work and school attendance, and community mixing patterns while repeated simulations explored variations in delegation of school closure authority, closure duration, closure thresholds, and reopening criteria. Epidemic simulations using a large-scale agent-based model explored whether and how these variations might affect outcomes as measured by attack rate, peak pandemic day, and peak case incidence.
Throughout this paper, we use the terms “pandemic” and “epidemic” to denote separate meanings. “Pandemic” refers to the H1N1 outbreak of 2009-2010 that was officially declared as such and describes the state plans so labeled. “Epidemic” describes the results of the simulations produced by our agent-based model.

**School Closure Statutes and Pandemic Influenza Plans**

An analysis of states’ school closure statutes and pandemic influenza plans identified variations influencing the uniformity, timing, and duration of closures that lead to a typology of school-closure authorities and criteria.

First, we re-analyzed data from a previously published report [11] of school closure statutes to identify characteristic ways of delegating school closure authority among states. Statutes delegated a single state official (14 states), one state official and one per-locality official (14 states), a single per-locality official (15 states), or two or more state officials and/or two or more per-locality officials (4 states); there was no statutory designation of authority in 3 states. These variations could affect whether or not a school-closure order could be implemented uniformly throughout a state and whether disagreements among designated officials might cause delays in implementation.

Second, we analyzed states’ pandemic influenza plans to identify criteria for initiating and terminating school closures. Every state had developed such a plan under direction of the U.S. Department of Health and Human Services (DHHS) during the avian influenza alert of 2005 and again in 2008. In the spring of 2010, we downloaded and analyzed the plans then posted on the DHHS website and followed links to state websites for further information. When links were broken, we added Google searches using the search phrase
“[state name] pandemic influenza plan,” which led to the specified documents. These methods produced complete plans for 45 states and plan summaries for 3 states (Iowa, North Dakota and Washington) but no plan for 2 states (Massachusetts and Rhode Island); so our analysis used only 48 state plans. These were dated from 2005 to 2010, with the majority written in 2006. All plan documents used in this analysis and downloaded in portable document format (pdf) are maintained on our study’s website [22].

Almost all the plans discussed school closure as an influenza mitigation strategy. Closure criteria were either stated as incidence at the state-wide or community-wide level (35 states), indicating uncertainty as to whether statewide uniformity or local autonomy is likely to be more effective in mitigating the spread of infection. However, plans rarely stated a specific incidence level to trigger closures, probably reflecting the lack of evidence to guide such advice. No closure criteria were mentioned in 14 state plans. State plans stated criteria for re-opening schools either in-depth (13 states), or briefly (8 states), or not at all (28 states). Again, the lack of specificity is attributable to the lack of empirical evidence; however, without such criteria for disease control, official decision makers are left subject to economic and social pressures to re-open schools from parents and employers.

Several examples illustrate how state planners have dealt with the lack of empirical evidence to guide school closure and re-opening thresholds. The Tennessee plan [23] states that schools are to be closed when three conditions are met: 1) pandemic virus causing morbidity and mortality in excess of seasonal influenza; 2) laboratory confirmation of the pandemic virus in a county or surrounding county; and 3) state
surveillance indicating community spread of the pandemic virus in the county or a surrounding county. This plan specifies that schools should re-open based on statewide criteria: when the State Epidemiologist determines that “the pandemic wave has subsided” based on sentinel surveillance.[23] The Kentucky plan instructs decision makers to “Collaborate with the local school board for closing and re-opening of school.”[22] The North Carolina plan asserts that “No data exists [sic] for recommending illness thresholds or rates of change in number of illnesses that should lead to consideration of dismissing or reopening schools.”[24]

**Interpretation of State Variations for Agent-Based Model**

The typical variations among statutory authorities and the uncertainties reflected in state plans are likely to affect school closures in terms of statewide uniformity, timing in relation to disease prevalence, and overall duration. Further, these differences are likely to produce differences in outcome of an epidemic, including attack rate (infected and symptomatic individuals as a proportion of total population), peak epidemic day, and peak case incidence (number of cases by day). These are important outcomes that affect demand for healthcare services overall, hospitalizations, and mortality. Because such these variations and their effects are not readily subject to real-world experimentation and controls, computational modeling offers an alternative. Agent-based modeling can simulate influenza epidemics under variations of school closure implementation patterns, timing, and duration while holding constant population demographics and virus transmission characteristics.

For the purpose of our simulations, the observed variations in states’ statutes and pandemic plans are captured in five “types”:
• Type I represents uniform statewide school closure order based on a standard threshold of statewide prevalence with all schools implementing closure within 1 day and remaining closed for the state duration. We tested 1% prevalence for alternative closure durations of 1, 2, 4, 8, and 16 weeks; we also tested 0.1% and 10% prevalence for the closure duration of 8 weeks.

• Type II represents a local threshold for school closure based on a standard of 5 cases in any school at alternative closure durations of 1, 2, 4, 8, and 16 weeks.

• Type III is similar to Type I but varies the period of implementation for a statewide closure order, testing alternative implementation delays of 3, 5, and 10 days. Such delays could account for time needed to resolve disagreements among state and/or local officials holding concurrent authority for decision making.

• Type IV represents lack of statewide uniformity by introducing randomness in the threshold for closures at the individual school level: 1 to 3 cases, 1 to 5 cases, and 1 to 10 cases. However, once closed, each school remains closed for the full duration.

• Type V represents how local pressures might limit the duration of closures by randomly re-opening schools before the full duration. Prematurity alternatives of 1-3 days, 1-10 days, 1-20 days, and 1-30 days were tested.

Agent-Based Model and Epidemic Simulation

An agent-based simulation model of Pennsylvania was created representing 11,863,395 residents with assigned households, schools, workplaces, commuting patterns, and risk of exposure and illness in a novel influenza epidemic. Detailed specifications and data sources for the agent-based model are presented in Additional File 1.
A simulated epidemic is initiated in the model with 100 infected agents randomly seeded in the state population. We assume an entirely novel virus, thus ruling out the potential effects of variations of immunity to influenza through previous exposures that might affect older age groups in a given population [25]. All agents begin a simulation run in the Susceptible (S) state based on the assumption that the epidemic is caused by an entirely novel virus. Contact with an infectious agent has a probability of transmitting the virus and transitioning a susceptible agent into the Exposed (E) state, which can last up to 3 days (with a mean of 1.2 days) and during which the agent is infected but not yet infectious. After the latent period, the agent progresses to the Infectious (I) state, when it is able to infect others for a period of up to 7 days (with a mean of 4.1 days). In this state, approximately 67% of agents manifest symptoms. Those remaining asymptomatic are 50% less likely to transmit the virus. At the end of the infectious period, agents progress to the Recovered (R) state and remain immune to re-infection so for the remainder of the simulation.

It is assumed that schools are closed on weekends. Community contacts for both students and workers increase by 50% on weekends, and 20% of adults work on weekends. When infected and symptomatic, 50% of students and workers stay home and have no community contacts.

This agent-based model was programmed in C++. Simulations were run at the Pittsburgh Supercomputing Center on its Blacklight architecture. As reported in this paper, each result is the average of 20 successfully initiated epidemics, each of which was run in parallel on a separate compute core (PC computer). Each simulation required 15 minutes.
Results

Simulation results are illustrated by overall attack rate in the population defined the percentage of total population both infected and symptomatic (Table 1) for the five Types of school closure authority. At baseline with no school closures, influenza attack rates were 20% at R0=1.2, over 36% at R0=1.6, and 47% at R0=2.0. These represent the ranges of rates previously reported for seasonal influenza combining all population ages [26], compared to the high R0 of 2.35 reported by Earn et al. for the 2009 H1N1 pandemic for the school-age population alone [16].

With uniform statewide closures in Type I, there was little effect on attack rates unless schools were closed for at least 8 weeks at any R0 tested. At low R0 of 1.2, an 8-week closure resulted in a 14% attack rate when triggered by a 1% statewide prevalence. At higher R0’s of 1.6 and 2.0, school closures of any duration had little effect on attack rates.

The Type I variations of prevalence threshold with low R0 of 1.2 produced counter-intuitive results: the lower statewide prevalence threshold of 0.1% had a higher (16%) attack rate; and the higher statewide prevalence threshold of 10% had a lower (12%) attack rate. These differences have been observed in our previously published school closure studies, [8, 19] and arise from shifting the school-closure period to later, thus having greater impact on the peak and duration of the epidemic curve. This also explains why the benefit of using a higher prevalence threshold disappears from the results at higher R0s, when the epidemic peak occurs earlier.
With Type II using a local threshold (5 cases per school), attack rates resulting from closures of various durations were the same as those with statewide uniform threshold (1% prevalence).

Type III tested another approach to altering statewide uniformity of closure threshold: by delaying implementation, the effective prevalence of infections was allowed to rise before schools were actually closed. At R0=1.2, compared with the attack rate of 14% from 1-day implementation based on 1% statewide prevalence, 3-day delay made no difference (14% attack rate), 5-day delay brought improvement (13% attack rate), and 10-day delay brought even further improvement (10% attack rate). As with the Type I variation of prevalence threshold, these results arise from shifting the school-closure period to later in the epidemic. However, at higher R0s, the delay-related shifting of school closure period produced no benefit because of the epidemics’ earlier peaks.

Type IV shows the effect of deferring to local decision makers and locally determined closure thresholds. Here, with low R0 (1.2) virus transmissibility, allowing randomness among individual schools in the case number to trigger an 8-week closure resulted in attack rates little different than requiring a statewide trigger of 5 cases (14%): 1-3 day triggers had a 16% attack rate; 1-5 day triggers had a 15% attack rate; and 1-10 day triggers had a 15% attack rate. At the higher R0s, local randomness in closure triggers had no effect on attack rates, which remained at 43%—the same as with statewide 5-case triggers.

Type V shows how failure to sustain a school closure for the full 8 weeks for low R0 virus slightly erodes the benefit on attack rates. Compared with attack rate of 14% for full
8-week closure, the rates are 15% with 3-day premature re-opening, 17% with 10-day premature re-opening, and 18% with 20-day or 30-day premature re-opening.

Figures 1, 2, and 3 present epidemic curves to illustrate differences in peak case incidence at various R0s and with various closure and re-opening criteria.

Figure 1 graphically shows Type II, which uses a 5-case per school trigger and 1-day implementation. For R0=1.2, the peak incidence is about 140,000 with 1 week closure; 134,000 with 2 weeks; 120,000 with 4 weeks; 94,500 with 8 weeks; and 12,000 with 16 weeks; the peak day is later for 1, 2, 4, and 8 weeks of closure (day 52, 56, 76, and 112, respectively) but earlier for 16 weeks of closure (day 32). For R0s of 1.6 and 2.0, the peak is lower at each successively longer duration of closure, but nearly all peaks are both earlier and higher than for the same closure durations at R0=1.2. At R0=1.6 the latest peak (day 52) occurs with a 4-week closure, when incidence of 205,000 is higher than with 8- and 16-week closures but well below incidences when closures are 2 weeks or less. At R0=2.0, transmission occurs so rapidly that the peak day occurs between 29 and 32 regardless of closure duration. Also at this high R0, increasing closure durations reduced the peak incidence successively from over 600,000 at 1 week, to 480,694 at 2 weeks; but beyond closure duration of 4 weeks with 419,644 cases, longer closures produced little further reduction of incidence, which remained above 400,000 cases with both 8- and 16-week closures.

Figure 2 shows how with Type IV the epidemic peaks varied as a function of changing the threshold for initiating an 8-week school closure. At all R0’s tested and regardless of whether the per-school case incidence threshold was uniform or randomized, the peak
day and peak incidence were very similar. However, at $R_0=1.2$, the random 1-5 case threshold produced a slightly later peak (day 108) than the alternatives (all at day 104); and notably, at $R_0=1.2$ and 1.6, random thresholds (1-5 cases and 1-10 cases, respectively) produced slightly lower case incidences (91,000 cases and 142,000 cases, respectively) than using the uniform 5-case closure threshold (104,000 and 144,000 cases, respectively). At $R_0=2.0$, using the 1-3 case random threshold resulted in higher incidence than the uniform and random threshold alternatives (425,286 cases compared to alternatives approximating 407,000 cases).

Figure 3 shows with Type V the effects of prematurely re-opening schools. The figure shows epidemic peaks when 8-week closures initiated by a 5-case per-school threshold are prematurely terminated by 3, 10, 20, and 30 days. At $R_0=1.2$, premature re-openings produced successively earlier and higher peaks, except that the 30-day premature reopening had a lower, flatter peak incidence—raising attack rates to 18% (as shown on Table 1) and eroding the benefit of the 8-week closure period. At $R_0=1.6$, the peak occurs at day 40 regardless of premature re-opening schedule, and the incidence varies only slightly among them (between 141,000 and 146,000). At $R_0=2.0$, the peak occurs at day 28 regardless of premature re-opening, and the incidence again varies only slightly among them (between 399,000 and 406,000 cases).

For 8-week closures with $R_0$s of 1.6 and 2.0, there is little or no change in peak day or incidence whether school closure threshold is standardized at 5-cases per school (Type II) or randomized at 1-3, 1-5, and 1-10 cases per school (Type IV) or whether there is premature re-opening (Type V).
Discussion

These simulation experiments permit comparisons among the various state delegations of school closure authority and the variations of specificity for criteria determining the uniformity, timing, and duration of school closure as an influenza mitigation strategy.

These results show no basis on which to justify statewide rather than school-specific or community-specific authority and criteria for school closures. There is little difference in effectiveness as measured by attack rate whether school-closure is statewide or school-by-school.

At low virus transmissibility ($R_0=1.2$), the more important factor is the duration of closure: attack rates remain high unless closures last 8 weeks or more. When transmissibility of infection is high ($R_0=1.6$ and 2.0), differences in closure duration have little effect on attack rates. But transmissibility is difficult to discern in an ongoing epidemic; and lengthy closures impose social and economic burdens on communities and families.

These simulations offer no evidence to support school closures promptly at the earliest stage of an epidemic, given the need for an 8-week closure period and the typically unknown transmissibility of the virus during an epidemic. At low $R_0$, delaying closures shifts the closure period further into the epidemic peak. At higher $R_0$s, this shift is irrelevant because the epidemic peak occurs much sooner; and closing schools at all has minimal effect. Again, since transmissibility is usually unknown during an epidemic, there may be an advantage to delaying school closures while monitoring increasing case numbers over a week or two at the individual community or school level.
An interesting observation concerns the timing of school closures. Earn et al., reported that school closure of 8 to 10 weeks in summer for the 2009 H1N1 pandemic in Alberta, Canada was followed by a spike in the fall infection rate (a so-called “second wave” of the pandemic)[16]. This effect also appears in our simulation results, when uniform, statewide closures initiated early in an epidemic delays the peak incidence but does not quench the epidemic.

School-specific closure criteria, though absent from most states’ existing epidemic influenza plans, appear to be important in disease control because of the dynamics of transmission. Influenza arrives in localities at different times, and commuting and mixing patterns vary locally. Thus, even if the case-per-school closure threshold is non-uniform across school districts and communities, there is a disease-control benefit to local responsiveness. A further advantage of local decision-making is that local authorities may be more knowledgeable about, and more sensitive to, the economic and social concerns of households and workplaces.

The duration of school closures affects not only attack rate but also peak day and peak incidence. Closures of 1-2 weeks or more, regardless of R0 or closure threshold, offer the potential for reducing the likely surge in demand for healthcare services. Closures of less than 8 weeks may also allow time to use other influenza mitigation strategies such as distribution of vaccines and anti-viral medications.

These simulation studies did not consider how the severity of disease caused by an influenza virus might affect the results. When an influenza virus causes only mild disease, long-duration school closure may be economically and socially infeasible. But
when influenza brings high rates of hospitalization and mortality, there may be weaker
economic and social pressures against school closures. Then, longer school closure
periods may have greater sustainability for households and workplaces as well as greater
importance for disease control.

**Conclusions**

Decision makers need evidence on which to base school closure plans and procedures;
but since controlled empirical studies of school closures are infeasible, computational
modeling offers a useful alternative. This agent-based simulation study suggests ways to
improve statutes and influenza plans.

First, school closure should remain an optional strategy for influenza mitigation, given
the uncertainties about virus transmissibility and disease severity that bedevil the early
stages of an epidemic.

Second, the threshold for school closures need not necessarily be specified in states’
influenza plans, but provisions for early and ongoing *local* monitoring of case numbers
should be well defined. It is important to avoid the risk contact-mixing among susceptible
school children when the virus is still circulating widely—a situation more reliably
observed at the community level rather than statewide.

Third, school closure authority may be exercised at the statewide or local level, so long as
decisions are informed by monitoring incidence in local communities and schools. Local
control may be important because of social and economic concerns about prolonged
school closures, which local officials can weigh against the risks of disease perhaps more
sensitively than statewide officials.
Fourth, depending on the transmissibility of an influenza virus, somewhat brief school closures of 1-4 weeks can help to reduce a surge in demand for healthcare services. Thus, school closures might be combined with other social-distancing strategies and with pharmaceutical interventions such as vaccinations.

**Author's contributions**

MAP conceptualized this study, participated in design of modeling experiments, assisted in the interpretation of statutes and pandemic plans, and drafted the manuscript. STB participated in design of modeling experiments, developed the model, performed the modeling experiments, assisted in interpretation of the modeling results, and helped to draft the manuscript. PMS assisted in the conceptualization of the study, assisted with legal and policy data acquisition, created methodology for interpretation of statutes and pandemic plans, assisted in drafting of the manuscript. TBH participated in the study design, analyzed state school closure statutes and state pandemic plans, and helped to draft the manuscript. PCC developed the initial ABM, aided in model validations, and helped to draft the manuscript. SMG assisted in the conceptualization of the study design and helped to draft the manuscript. BYL contributed to the conceptualization and design of the model, interpretation of results, and drafting of the manuscript. CRK assisted in identifying data, participated in analysis, and edited the manuscript. JG contributed to the design of the model, the analysis and interpretation of the data, and helped to draft the manuscript. DSB made substantial contributions to conceptualizing how legal criteria could be used in a computational model and to the overall study design.

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

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10. Update on school (K-12) and child care programs: Interim CDC guidance in response to human infections with the novel influenza A (H1N1) virus [homepage on the internet] [http://www.cdc.gov/h1n1flu/schools/](http://www.cdc.gov/h1n1flu/schools/)


29. Common Core of Data: Build A Table [http://nces.ed.gov/ccd/bat/]


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<td>8 weeks</td>
<td>13%</td>
<td>31%</td>
<td>44%</td>
</tr>
<tr>
<td></td>
<td>1% prevalence, 10-day delay</td>
<td>8 weeks</td>
<td>10%</td>
<td>33%</td>
<td>46%</td>
</tr>
<tr>
<td>TYPE IV</td>
<td>Random, 1-3 cases per school</td>
<td>8 weeks</td>
<td>16%</td>
<td>32%</td>
<td>43%</td>
</tr>
<tr>
<td></td>
<td>Random, 1-5 cases per school</td>
<td>8 weeks</td>
<td>15%</td>
<td>32%</td>
<td>43%</td>
</tr>
<tr>
<td></td>
<td>Random, 1-10 cases per school</td>
<td>8 weeks</td>
<td>16%</td>
<td>32%</td>
<td>43%</td>
</tr>
<tr>
<td>TYPE V</td>
<td>5 cases per school</td>
<td>Random, re-open 1-3 days before 8 weeks</td>
<td>15%</td>
<td>32%</td>
<td>43%</td>
</tr>
<tr>
<td></td>
<td>5 cases per school</td>
<td>Random, re-open 1-10 days before 8 weeks</td>
<td>17%</td>
<td>33%</td>
<td>43%</td>
</tr>
<tr>
<td></td>
<td>5 cases per school</td>
<td>Random, re-open 1-20 days before 8 weeks</td>
<td>18%</td>
<td>33%</td>
<td>43%</td>
</tr>
<tr>
<td></td>
<td>5 cases per school</td>
<td>Random, re-open 1-30 days before 8 weeks</td>
<td>18%</td>
<td>33%</td>
<td>44%</td>
</tr>
</tbody>
</table>

*Infected and symptomatic individuals as a percentage of total population
Additional File 1 (pdf). Data sources for Pennsylvania School Closure Model

Figure 1. Type III - Cases per day with 5-case per-school closure threshold for varied durations at R0 = 1.2, 1.6, and 2.0

Figure 2. Type IV - Cases per day for 8-week closure duration with varied per-school closure thresholds at R0 = 1.2, 1.6, and 2.0

Figure 3. Type V - Cases per day with varied per-school re-openings before 8 weeks at R0 = 1.2, 1.6, and 2.0