Reviewer's report

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

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Reviewer: Gergely Röst

Reviewer's report:

The paper discusses a very important public health question, namely what policy should be followed regarding school closures as influenza mitigation measure. The authors use an agent based model to simulate Pennsylvania. Such a computational modelling study could be a contribution of great importance in providing evidence to the (non)effectivity of various school closure policies.

I find the papers interesting and useful, however, I have several concerns about the paper as it is presented.

Major issues:

(i) The paper discusses only proactive school closures. For 2009 H1N1, reactive closures were typical, after 30% or more of the students fell ill. If the outcome measure is the attack rate, then it is expected that a closure closer to the peak has more effect (as it prevents more contacts between infected and susceptible students). Some of the simulations considered are slipping there, when the trigger is a higher prevalence or when the implementation delay is long (it is an amusing fact that a delayed implementation of a bad policy can improve it a lot).

The authors say that it is counterintuitive that the attack rates decreased in such cases, while I think it is exactly what can be expected. It would have been useful to dig a little into this direction, finding for example what prevalence trigger is the optimal in reducing attack rates.

The 8 and 16 weeks closures seem infeasible (even the 4 weeks), unless this period coincides with regular summer holiday. Is there any example when such a long closure took place?

Has the model been tested on any real data? There are many examples when regular school closure (holiday season) coincided with an ongoing epidemic, thus
providing
information at least for the uniform statewide policies to test.
As one illustratory example, I'd like to point to Figure 8 in our paper
(Knipl D, Röst G, Modelling the strategies for age specific vaccination scheduling
where it can be seen that a regular countrywide autumn school break of one
week
delayed the epidemic for 2009 H1N1 by roughly three weeks, and the
winter break came just around the peak and totally killed the outbreak
(here vaccination campaign was also ongoing so the situation is a little different).
(ii) One would expect more details about how the ABM works, the authors
do not explain the model too much. For example,
the authors say the exposed period can last up to 3 days with a mean of 1.2
days,
but they don't tell us what is the probability distribution for that period.
The same for the infectious period. It is not clear to me how the transmission
parameters
are scaled to the given basic reproduction numbers in the heterogenous agent
population.
What are the contact parameters during school closures: the same as during
weekends
or something else?
(iii) Also, one would expect more detailed results from an agent based model
than the epidemic curves for the total population and the overall attack rates.
How does the epidemic run in various subgroups of the population? What are the
attack rates in the schoolchildren population? In the locally triggered cases, what
was the fraction of schools that needed to be closed and how were these
closures
distributed in time? Due to stochasticity, what are the variations in the outcomes?
20 runs seem too few to me, but it is hard to tell if the variations are not
presented. The advantage of an ABM compared to simple compartmental
models
would be that we could answer such (and many more) questions.

Minor issues:
1) the additional file does not follow the epidemic/pandemic distinction
as the paper does
2) how exactly does the randomness work for Type IV?

3) it would be useful to indicate the time intervals when schools were closed in the figures (when applicable)

4) the additional file mentions vaccine specification while there is nothing about it in the paper

5) about the results in the Table:
what explains the drop of the attack rate to 2% in the Type I / 16 weeks case?
More importantly: for the Type I variation with 10% prevalence trigger, how is the 12% attack rate possible?

**Level of interest:** An article of importance in its field

**Quality of written English:** Acceptable

**Statistical review:** No, the manuscript does not need to be seen by a statistician.

**Declaration of competing interests:**
I declare that I have no competing interests.