Reviewer's report

Title: Mitigation of infectious disease at school: targeted class closure vs school closure

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Reviewer: Timo Smieszek

Reviewer's report:

It was a pleasure to review the manuscript "Mitigation of infectious disease at school: targeted class closure vs school closure". The authors presented a well-written and informative manuscript about a very topical problem: how to target infectious disease spread effectively while minimising social / economic costs caused by the interventions. While I believe that the manuscript discussed here is a relevant addition to the scientific literature in this field, I think it needs some revisions before being publication-ready. My subsequent comments and thoughts are in chronological order:

1) Abstract: the authors use the term "airborne infections", a term which I think is misleading in the context of your manuscript. One should distinguish between truly airborne infections and droplet-transmitted infections. While the former term refers to infectious diseases that spread via aerosolised particles that stay suspended in the indoor air for extended periods of time (and, hence, don't require the kind of social action the authors modelled here). The latter refers to infections that spread primarily via droplets of larger size that fall out rather quickly and for which social interaction - as measured and modelled here - might be a good proxy (Cf, e.g., Indoor Air 2007, 17:211–225; see also discussion in BMC Infectious Diseases 2014, 14:136). In short: your model only applies to infections transmitted primarily by larger droplets, not to truly airborne infections. It would be interesting to discuss also what the differences for airborne / aerosol transmitted infections might be.

2) P. 1: "It has been long known [1,2] that children play an important role in the community spread of infectious disease, in particular influenza"; a relevant addition (since it triggered the respective vaccination programme in the UK) might be the following paper: PLoS Med 10: e1001527.

3) P. 2: "...and more recently wearable sensors that detect close-range proximity [30,31,32] and face-to-face contacts [33,34,35,36,37]"; I don not see a fundamental difference between the TelosB motes and the SocioPattern sensors in terms of what they measure. Salathe's contacts are also face-to-face (see, e.g., supplementary figure S4 in Salathe's PNAS paper).

4) P. 2: "This is expected to be a rather general feature of schools, due both to age homophily [38] and schedule constraints"; I agree that there are these tendencies in all schools (especially age assortativity), but I think that the extent
depends vastly on how a school system is organised. There is probably a difference between primary and high school, and there is certainly a difference between, e.g., US and central European schools. The way the US high school system works makes the contacts there more random / less structured (cf. the Salathe group papers) than contacts in a typical European school. I would tone that statement down a bit and discuss potential differences. Maybe a comparison between your data and the Salathé data (with respect to the research question of the manuscript presented here) would be an interesting task for the future?

5) P. 3: "In such a compartmental model each individual at a given time..." from my point of view, your model is not a compartmental model. Since you incorporated the measured contact structure into your model, it must be individual-based. Compartmental models are ODE models that don't represent individuals explicitly, but bin them into "boxes" aka compartments.

6) P. 4: The authors' model assumptions:

6.a) Did you do sensitivity analyses on your most crucial / shaky-ground assumptions, e.g., that asymptomatic individuals are beta/2 or that the probability of being asymptomatic is 1/3?

6.b) Why do you model recovery as a fixed rate (exponential distribution of infection duration)? More realistic would be log-normal / Weibull distributions for the infection duration. Since your model appears to be individual-based (cf. my comment 5), I cannot see a striking mathematical reason to use the (less realistic) exponential distribution. Could you, at least, comment on the implications of this assumptions on model results?

6.c) Where do your beta-values come from?

6.d) A source of references for some of your parameters might be BMC Infectious Diseases 2011, 11:115.

7) P. 7: "inside one school, children do not mix homogeneously" - pls. see my comment 4

8) Tables 1-5: It would be great if you could compare not only "targeted class", "targeted grade", and "whole school", but also a baseline of doing nothing / implementing no intervention.

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 used the senior authors' equipment (sensors & receivers) in a recent study and I
spent a week at Dr Cattuto's institute for the purpose of scientific exchange earlier this year.

I don't feel biased and I think my comments show that I have the necessary critical distance towards Dr Cattuto's and Dr Barrat's work, but I think the editors should be aware of the fact this field (contact measurements) is very small and that, hence, I do know the authors quite well.