

Misinformation making a disease outbreak worse: Outcomes compared for influenza, monkeypox and norovirus

Julii Brainard* and Paul R. Hunter
Norwich Medical School

*Dr. Julii Brainard, j.brainard@uea.ac.uk, tel. +44-1603-591151
ORCID 0000-0002-5272-7995

Other ORCID
Paul R. Hunter 0000-0002-5608-6144

SUGGESTED RUNNING TITLE Fake news in disease outbreaks

CONFLICT OF INTEREST: We declare that we have no conflicts of interest.

ACKNOWLEDGEMENTS

Adrian Pratt, Tom Finnie, Steve Leach and other staff at Public Health England (PHE) gave many helpful comments. Thanks to Soroush Vosoughi, Shannon Fast and Anil Doshi for answering questions about their research. Anonymous referees gave useful comments on previous versions of this article. James Rubin of Kings College London suggested monkeypox. Our study was funded by the National Institute for Health Research (NIHR) Health Protection Research Units in Emergency Preparedness and Response and Gastrointestinal infections in partnership with PHE. The views expressed are those of the authors and not necessarily those of the National Health Service, the NIHR, the Department of Health or PHE. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript and associated documents.

Word Count: 8000 including abstract, tables and references
4000 words without abstract (150 words), tables (n=3) or references (n=78)
3 supplemental files

Misinformation making a disease outbreak worse: Examples for norovirus, influenza and monkeypox

ABSTRACT

Health misinformation can exacerbate infectious disease outbreaks. Especially pernicious advice could be classified as ‘fake news’: manufactured with no respect for accuracy and often integrated with emotive or conspiracy-framed narratives. We built an agent-based model that simulated separate but linked circulating contagious disease and sharing of health advice (classified as useful or harmful). Such advice had potential to influence human risk-taking behaviour and therefore risk of acquiring infection, especially as people are more likely in observed social networks to share bad advice. We test strategies **proposed in recent literature** for countering misinformation. Reducing harmful advice from 50% to 40% of circulating information, or making at least 20% of the population unable to share or believe harmful advice mitigated the influence of bad advice in the disease outbreak outcomes. How feasible it is to try to make people “immune” to misinformation or control spread of harmful advice should be explored.

Keywords: agent-based models, norovirus, influenza, monkeypox, fake news, social networks

1. INTRODUCTION

Previously we constructed an agent based model (ABM) ^{1,2} about what could happen during a norovirus outbreak exacerbated by circulating misinformation (which we call '*bad advice*'). Bad advice is relevant when it changes human behaviour to be more risky (= higher risk of getting disease). Examples of possible risky behaviour can include not washing hands, sharing food with ill people, not disinfecting potentially contaminated surfaces or fomites, sneezing, coughing or vomiting in places where there is a high risk of spread onto food or surfaces, lack of disinfection and other unprotected physical contact with infectious persons or their bodily fluids.

Some people in disease outbreak situations take few precautions to avoid getting disease, even when awareness of the outbreak is widespread and many official sources are widely disseminating information about how to avoid illness. Compliance with quarantine protocols during a 2003 outbreak of SARS-coronavirus was found to be uneven. Compliance related to risk perception which in turn related to how trustworthy and credible sources of health advice were perceived to be, as well as difficulties respondents had in accepting parts of the situation they could not be in control of. ³ During a large and well publicised norovirus outbreak at a Canadian university, 25% of symptomatic students were observed to not avoid contacts, while 17% of the observed cohort did not comply with recommended handwashing practices ⁴. In a 2019 survey in the UK ⁵, 14% of 2000 surveyed parents reported sending a child to school with symptoms of contagious chickenpox, violating school policies and official advice to quarantine such children.

That misinformation can be linked to taking fewer measures to effectively prevent disease transmission is especially well documented with respect to Ebola viral disease (EVD). For instance, persons affected by the West Africa outbreak (2013-2016) who believed other types of misinformation about EVD (eg., that it can be airborne or transmitted via mosquito bites) were more likely to report practicing unsafe burial practices.⁶ Low trust in institutions and more belief in EVD misinformation were associated with fewer preventive behaviours in the Congolese outbreak that started in 2018. ⁷ Following ineffective advice or disbelieving official recommendations often led to pursuing other self-care actions first and thus delayed actions that could reduce transmission (such as formally getting tested for EVD).⁸ Most sources that have studied disease-precaution behaviour, especially vaccine refusal (all diseases), note that cultural identity outside perceived mainstream society is strongly positively linked to tendency to reject expectations of 'good citizen behaviour' ⁹⁻¹², including disease avoidance actions recommended by public health authorities.

Our objective was to model possible interactions between misinformation spread and disease outcomes. The modelling was done within an agent-based modelling ¹³ environment, using Netlogo software ¹⁴. Most model parameters were available from sources reporting actual human behaviour, such as about speed and

frequency of social media and real life information sharing. An ABM environment was attractive because it afforded many opportunities to include complex aspects of behaviour and response that might vary individually. We use the term *complex* to distinctively mean processes that are thought to be inherently unpredictable and have uncertain outcomes (as opposed to the often misused near synonym *complicated*, which can be better interpreted as describing events that can be modelled using consistent albeit possibly very multifaceted decision trees).

Complexity in our models meant that, for instance, clusters of individuals with frequent contact with each other (physical or information sharing contact) might have very different traits from the population averages. But these differences could not be consistently predicted; nor could location or encounters with other agents. Location and chances of getting disease or encountering misinformation were best estimated for our purposes using reiterative modelling and probabilistic distribution of relevant attributes. These skewed traits could lead to localised hotspots or low activity zones of disease and/or information transmission. Multiple types of feedback loops could be in operation that affected behaviour choices. These feedback processes could be modelled at individual level using an ABM. At the same time, many aspects of behaviour and response seemed likely to be probabilistic but perhaps on uncertain distributions or with unclear central tendency points. The iterative nature of ABMs meant that we could try to use real world data to describe some behaviour aspects in the models, but also use iterations to estimate what some central values for other behaviour tendencies might be.

In the ABM that we constructed, disease spread was via direct or indirect physical contact, while misinformation spread was via social contact, particularly within social groups ('bubbles'). These social groups were constructed to have relatively similar susceptibility for believing the misinformation. Members of one's bubble were often the same persons that were physically encountered, so these could be the same people with whom it was likely that the disease could be exchanged. Our initial work focused on norovirus because although very common, gastrointestinal illness is rarely modelled in individual infectious disease models¹⁵. Norovirus also had the advantage of being unlikely to cause flight or death (so those things could be defensibly not included in the model). The incubation period is relatively short so travel outside the residence area was excluded.

The purpose of this study is to adapt our misinformation (agent based) model to other communicable diseases and outbreak conditions. We compare results for norovirus, influenza and monkeypox (*Orthopoxvirus*). The latter diseases can be substantially different from norovirus in many respects (such as mean incubation period) and are often not previously vaccinated for. Influenza is an important communicable respiratory illness globally, while monkeypox is an emerging disease of high concern that has triggered biosecurity concerns^{16, 17}.

These pathogens gave good opportunities to show that our modelling approach could be adapted to multiple types of disease and transmission risks.

2. METHODS

2.1 Overview

Table 1 shows model assumptions and targets, separated by disease. Model assumptions and design features such as rate of information injections are described in greater detail elsewhere ². The underlying Netlogo model code is available from the authors upon request. At least 100 iterations were run for the most likely candidate thresholds or parameters to verify their reliability. Extra simulations (above 100 minimum) were run until we found that additional model runs did not produce mean or median estimates that were any closer to the target conditions (i.e., a monotonic state in target outputs had been achieved).

2.2 Stage 1

A baseline stage 1 model was constructed for each circulating disease. Target basic reproduction numbers (r_0) were determined from consulting relevant literature for norovirus and influenza, and a likely maximum number of generations during a monkeypox outbreak (see Table 1). The stage 1 models were designed to achieve the target r_0 or number of generations. Real life transmission of disease depends on multi-faceted factors: biological, social, structural and behavioural ¹⁸. We conceptually break the risk of transmission down into three components: the probability of (1) infectious persons or (2) susceptible persons taking adequate precautions to avoid catching disease, as well as (3) viral shedding (which can be linked to pathogen and illness characteristics, not truly under individual control). The involuntary shedding is separated from behavioural risks for many reasons. It makes sense that a small part of the risk is purely biological not behavioural. Separation lets individual behaviour be fully separated from involuntary shedding, while involuntary shedding may be adjusted to vary over the course of illness in future more sophisticated models (the amount of virus shed does vary with stage of illness in real infections ¹⁹).

In a model run, during each time step and in a random direction, well agents move one step. Well agents move five times further than ill agents (who move 0.2 steps). Each time step, susceptible agents near infectious agents were tested for possible disease transmission. Disease was transmitted if neither side took sufficient precautions and viral shedding was sufficient for transmission. Individuals had baseline take-precautions (TP) % values assigned at the start of model runs. TP is the percentage of the time that agents took effective precautions against catching disease. TP did not vary in the stage 1 mode, but was important in stage 2-3 models when it could change in response to circulating (mis)information. To establish the

initial (baseline) stage 1 models, an exercise (described in the next paragraph) was undertaken to estimate the proportion of risk that could be attributed to and reserved for just viral shedding.

TP was assigned to each individual agent, generated stochastically and assumed to have a normal distribution of values around the population mean, which was preset to 50% for several reasons. Constraining TP to range only between 0 and 1.0 (but with a fixed central population mean) gave TP the maximum room for change. Also, prespecifying TP allowed us to estimate and specify separately the small proportion of risk of transmission that could be attributed to viral shedding alone (meant to be pathogen-specific and due to presence of infection, not the risk of transmission due to agents' good or poor behaviour choices). Designating a small separate viral shed risk from behaviour seemed desirable because only with fairly extreme precautions (such as wearing personal protection equipment) could the risk of disease transmission be truly reduced to effectively zero. The mechanism of disease transfer was: transmission could happen when susceptible and infectious agents were in close proximity and neither took high enough precautions to avoid transmission. A small proportion of the risk was also linked to viral shedding for each disease. The variations in how disease was transmitted (with and without a separate viral shedding risk) is helpful in showing that our modelling approach can be flexible and adapted for different disease or outbreak conditions.

Table 1 lists other model parameters and assumptions. 1-2% of agents were infected at simulation start time. Thereafter, agents moved around and tests were made about potentially transmitted disease each time step (one hour). The model starts at 7am, and agents return 'home' to the same location each evening, which means the highest disease transmission risk is with others near their home location. Agent density, grid size and movement rules were designed such that, in the absence of any disease, the daily contact rate with other agents averaged very close to 11.74 unique others/day (a target drawn from published UK contact rates in non-epidemic situations ²⁰). Empirically, we found that 1600 agents achieved the target contact rate, on a torus world shape (e.g.. going off the bottom meant re-entry at the top), with visible area measuring 88x90 patches that agents could move around on. Incubation periods, assumptions about shedding before or after illness, duration of active illness and infectious period and case fatality rates were drawn from relevant literature. In Table 1, chances of hospitalisation or fatality were plausible, not meant to be definitive. Because health care in the UK is free at the point of use to and urgent care facilities are widely available, we assume that all very ill individuals will seek medical advice. We assume that only very ill individuals will die from disease. Therefore, the models assume that only hospitalised cases ever died and that no transmission occurred after someone was hospitalised due to effective infection control measures. Vaccine efficacy was assumed to be 100%, but vaccine availability varied by disease. Uptake was assumed to be high but not universal (75% of those who could benefit). Hospitalisation is the only form of quarantine considered.

TABLE 1 AROUND HERE

2.3 Stage 2

The stage 2 model used the same disease and information circulating assumptions as the stage 1 model, but with exacerbation due to spread of misinformation that often reduced taking effective precautions (TP). Recall that the mean TP value was assigned randomly on a normal distribution with preset mean = 50% at start. In stage 2, each exposure to misinformation changed individual TP, with limits at 0 and 100%. The possible change in TP (ΔTP) was determined using repeated model runs, to achieve a 40% worse (higher) r_0 for norovirus or influenza, or more generations of transmission (from target 4 to target 7 in 75% of simulations) for monkeypox. The magnitude of change in response to advice was equal whether good or bad advice²¹⁻²³. ΔTP is the key response that individuals have to circulating information (or misinformation). Exposure to ‘good’ advice increases taking precautions; exposure to bad advice decreases taking precautions.

2.4 Social contacts and information sharing

Each agent had a list of other agents that they might share information with (their own unique ‘information bubble’). There were typically 80-230 members of this ‘bubble’ (mean number = 150, to conform with estimates of significant friendship circle sizes Dunbar numbers^{24, 25}). The list of social contacts was created such that (on average) two-thirds of the social contacts had similar propensity (randomly assigned around population mean = 38.9%) to believe misinformation (from experimental and observation data, that typically members of the British public believe in an average 38.9% of conspiracy theories that they are exposed to²⁶). About 20% of social contacts were located near the agent’s home location; physical proximity made it more likely that disease would be shared with these specific same social contacts with whom (mis)information was shared. In every model run, agents moved around, potentially transmitted disease, and made decisions about whether to share information they had been exposed to. Susceptible agents with lower TP values were at highest risk of acquiring infection.

138 times per hour a single agent chosen at random was exposed to a piece of information and made a decision (stochastically) whether to share the information onwards to a small percentage (2.5%) of their social contacts. The cascades of resulting information (number of times the information passed through unique sharers) were monitored and the model performance in this regard is documented elsewhere². The frequency of relevant information sharing and the likelihood of sharing advice were both determined empirically so that the resulting information cascades would conform with information sharing patterns reported recently on Twitter for true and false stories²⁷. Distinguishing true from false (good and bad

advice in our model) was important because false stories were observed to be four times more likely to be shared in the Twitter study. In stage 2, the ratio of good:bad advice exposed to agents was 50:50.

2.5 Stage 3: Intervention Strategies

Proposed strategies to fight fake news from previous literature include:

- 1) Provide counter-information that is equally or better evidenced, or more persuasive ²⁸⁻³⁴
- 2) Tax the advertising or tax the profits of products sold via misinformation ³⁵
- 3) Drown bad info with good information ³⁵
- 4) Regulate information ³³, possibly impose civil or criminal liabilities ²⁹ which could lead to explicit censorship ^{29, 33}
- 5) Revise financial models available to fake news disseminators (incentives) to stop encouraging production and sharing of false (or even just very salaciously written) stories over truth and accuracy ^{27, 34, 36-39}
- 6) Labelling (reliability rating or counter-arguments provided) by news provider ^{27, 29, 33, 34}
- 7) Encourage individuals to actively strive to make their own social filter bubbles more diverse ³³
- 8) “Immunise” recipients to disregard fake news (education-based strategy) ^{36, 40}

We do not model effects of intervention strategy 1 because the results are predictable; any changes will be linear responses if good advice increases without a reduction in bad advice or if good and bad advice are equally contagious. Therefore, in stage 3, two strategies for reducing misinformation impacts were tested because their impacts could not be easily foreseen: 1) increasing the proportion of ‘good advice’ that encourages more protective behaviours, and 2) “immunising” individuals so that they do not respond or share bad advice. Possible thresholds (to reduce the r_0 from stage 2 to stage 1 values, or even lower) were explored both for the proportion misinformation adjustment and “immunisation” strategies. Stage 3 models were run under stage 2 conditions but with the below modifications and objectives:

- **Stage 3.1:** Reduce bad advice injections from 50% to a value that achieved conditions similar to our stage 1 models (stage 1 target r_0 or number of generations), as well as **stage 3.2** test what happened if only 10% of circulating advice is bad advice.
- “Immunise” against bad information (but not immunised against the virus, and still able to react positively to good advice): a percentage of randomly selected agents were selected to be fully resistant (“immunised”) to bad advice. The exact percentage was found empirically, such that **(stage 3.3)** the stage 1 r_0 or number of generations was achieved. We also tested **(stage 3.4)** if

“immunising” 90% of agents could reduce r_0 below 1.0. “Immunisation” also meant no sharing of bad advice. This strategy simulated approaches that were based on education or diversifying social contacts (social filter bubbles).

Statistical differences in the outcomes under each set of modelling assumptions were calculated using Wilcoxon rank sum tests using Stata v. 16.0 (stage 1 was the reference condition).

3. RESULTS

Table 2 shows estimated viral shed risk values found to consistently get closest to target r_0 s in stage 1 models, when mean TP = 50%, and the change in TP (Δ TP) required upon each information exposure to raise the r_0 by 40% (stage 2 models). Table 3 shows model run results (r_0 , duration of outbreak, final attack rate and prevalence of disease at peak) for each separate disease. Supplemental files S1-S3 show additional results for alternative model parameters. For all diseases, reducing bad advice from 50% to 40% of all relevant circulating advice changed stage 2 outbreak conditions to stage 1 levels. Similarly, making 20-25% of individuals “immune” to believing or sharing bad advice changed stage 2 outbreak outcomes back to stage 1 levels (with respect to r_0 or number of generations of transmission, duration of outbreak, peak attack rate, case fatality rate or final total attack rate).

We find it interesting to note that even if the strategies are applied relatively drastically and effectively (i.e., quite large reductions in proportion of bad advice circulating or high percentage “immunised” against bad advice), disease spread was not stopped. The results in Table 3 suggest that even if bad advice were only 10% of circulating advice (stage 3.2), r_0 for norovirus and influenza may reach 1.0, while number of generations of transmission for monkeypox will be ≥ 3 in at least 25% of simulations. Similarly, even if 90% of agents are “immune” to bad advice (stage 3.4), outbreak r_0 (norovirus and influenza) will tend to be ≥ 1.0 and at least 25% of outbreaks will have ≥ 3 generations of transmission. The Wilcoxon rank sum tests (Table 3) show that broadly, outcomes were similar between stage 1 models and stage 3.1 and 3.3 models (which was the objective). Stage 2, 3.2 and 3.4 model outcomes were quite different from stage 1 models (most p values well below 0.01).

TABLE 2 AROUND HERE

TABLE 3 AROUND HERE

4. DISCUSSION

No previous studies have integrated information spread with disease spread to the level of sophistication that we have done. Prior models often considered information spread in disease outbreak development, but information awareness was typically equally available to all agents, and benign at worst. Thus, information spread in the models nearly always led to greater protective measures (such as increasing vaccine uptake or decreasing contact rates⁴¹⁻⁵⁰). Most previous similar disease and awareness spread models had awareness increases that could only happen following physical contact or as a result of global conditions^{42, 45, 48, 50-55}. Our modelling is unusual because information spread was individual and separated from the physical interactions that could transmit disease. Our model is unique and original in attempting to consider the potentially deleterious role of information sharing with stochastic and individually assigned elements. The need for research such as ours has been recognised before^{17, 56}.

More sophisticated information sharing networks than we tried to create could make these models more credible. There exist more sophisticated models on rumour spread that we could possibly replicate for the information spreading process⁵⁷⁻⁵⁹, and simultaneously merge with existing sophisticated disease spread models. More ambitious models than ours would describe more agents and more complicated movement patterns, such as including flight as a behaviour option. Many rumour spreading models have borrowed ideas and methods from epidemiological models^{60, 61}; but not many (if any) previous models have integrated both rumour and disease spread as separate but interacting processes into one unified probabilistic model.

This study describes spread of three viral diseases; misinformation affecting spread of bacterial diseases could be modelled equally well. The ideas could be applied to non-communicable diseases and health outcomes, but would need to change the time scale to be much longer to model chronic and lifestyle diseases and how their incidence might change in response to circulating misinformation. A much longer time scale would mean incorporating many other lifestyle factors into the models.

Model construction relied heavily on a small number of existing studies about such factors as number of contact rates, social contacts (i.e., Dunbar numbers), how much bad or good advice can change behaviour, and the propensity to believe in misinformation (the finding that on average, British people believe in 38.9% of conspiracy theories that they are exposed to). More reliably estimating any of these and many of the other factors would also increase the credibility of our results. Our threshold for a 'worse' outbreak situation was r_0 being 40% worse or number of generations of disease transmission increased from 4 to 7; these thresholds were decided for convenience in this set of demonstration models.

Given our definition of stage 2 as an outbreak 'made worse by circulating misinformation', stage 3.1 modelling concluded for all three diseases that a ratio of about 60:40 good:bad advice circulating would

reduce the stage 2 conditions to those of stage 1. The models also suggested that “immunising” about 20% of the population against misinformation was likely to revert stage 2 to stage 1 conditions (for all diseases, stage 3.3). Since these apparent consistencies could be artefacts of shared model design, tests to explore the true consistency of these findings for multiple diseases would be worthwhile. It is possible that more sophisticated, detailed or larger models or more flexible modelling software ⁶² would facilitate better insights into risk distributions and behaviour choices.

There is uncertainty in the reliability of these findings because the models are experimental and have not been tested in real world situations. There is a general lack of reliable quantification for how much misinformation spread impacts real life risk-taking behaviour with regard to communicable diseases.

5. CONCLUSIONS

We applied three stages of modelling (1 = no misinformation spread, 2 = misinformation making outbreaks worse and 3 = strategies to reduce the influence of misinformation). Our modelling approach and design is adaptable to many different types of diseases. Controlling spread of misinformation or susceptibility to it could reduce communicable disease burdens. Our stage 3.1 modelling found that a ratio of about 60:40 good:bad circulating advice reduced stage 2 conditions to those of stage 1 in three types of disease. “Immunising” about 20% of the population against misinformation (stage 3.3) was likely to revert stage 2 to stage 1 conditions (for all diseases). The feasibility of implementing these types of strategies (“immunisation” or changing the proportions of types of advice in circulation) should be explored. The efficacy of implementing such strategies to fight ‘fake news’ needs to be tested in real world settings, with costs and benefits ideally compared with real world disease reduction.

Supplemental Material

S1: Additional results for Influenza
S2: Additional results for Monkeypox
S3: Additional results for Norovirus

References:

1. Brainard J, Hunter PR and Hall IR. An agent-based model about the effects of fake news on a norovirus outbreak. *East of England Public Health Conference 2018*. Stansted Airport, UK. 2018.
2. Brainard J, Hunter PR and Hall I. An agent-based model about the effects of fake news on a norovirus outbreak (under review; also at https://osf.io/cyq6t/?view_only=e422e51b6ab441b0badadd0963821221): 18 pp.
3. Cava MA, Fay KE, Beanlands HJ, et al. Risk perception and compliance with quarantine during the SARS outbreak. *J Nurs Scholarsh* 2005; 37: 343-347.

4. Surgeoner BV, Chapman BJ and Powell DA. University students' hand hygiene practice during a gastrointestinal outbreak in residence: what they say they do and what they actually do. *J Environ Health* 2009; 72: 24-29.
5. Francis G. Millions of children are sent to school or nursery when ill, poll claims. *The Independent*, 17 Sept 2019.
6. Tenkorang EY. Effect of knowledge and perceptions of risks on Ebola-preventive behaviours in Ghana. *International Health* 2018; 10: 202-210.
7. Vinck P, Pham PN, Bindu KK, et al. Institutional trust and misinformation in the response to the 2018–19 Ebola outbreak in North Kivu, DR Congo: a population-based survey. *The Lancet Infectious Diseases* 2019; 19: 529-536.
8. Yamanis T, Nolan E and Shepler S. Fears and misperceptions of the Ebola response system during the 2014-2015 outbreak in Sierra Leone. *PLoS Negl Trop Dis* 2016; 10: e0005077.
9. Blume S. Anti-vaccination movements and their interpretations. *Soc Sci Med* 2006; 62: 628-642.
10. Hobson-West P. 'Trusting blindly can be the biggest risk of all': organised resistance to childhood vaccination in the UK. *Social Health Illn* 2007; 29: 198-215.
11. Bass SB, Ruzek SB, Ward L, et al. If you ask them, will they come? Predictors of quarantine compliance during a hypothetical avian influenza pandemic: Results from a statewide survey. *Disaster Med Public Health Prep* 2010; 4: 135-144.
12. Blendon RJ, DesRoches CM, Cetron MS, et al. Attitudes Toward The Use Of Quarantine In A Public Health Emergency In Four Countries: The experiences of Hong Kong, Singapore, Taiwan, and the United States are instructive in assessing national responses to disease threats. *Health Aff (Millwood)* 2006; 25: W15-W25.
13. Wellman MP. Putting the agent in agent-based modeling. *Autonomous Agents and Multi-Agent Systems* 2016; 30: 1175-1189.
14. Yang C and Wilensky U. Netlogo epidem basic model. *Center for Connected Learning and Computer Based Modeling Northwestern University, Evanston IL* 2011.
15. Willem L, Verelst F, Bilcke J, et al. Lessons from a decade of individual-based models for infectious disease transmission: A systematic review (2006-2015). *BMC Infect Dis* 2017; 17: 612.
16. Federal Select Agent Program. Select Agents Regulations. Centers for Disease Control and Prevention, Animal and Plant Health Inspection Service, 2017.
17. Tian D and Zheng T. Comparison and analysis of biological agent category lists based on biosafety and biodefense. *PLoS One* 2014; 9: e101163.
18. Swift L, Hunter PR, Lees AC, et al. Wildlife trade and the emergence of infectious diseases. *EcoHealth* 2007; 4: 25.
19. Lau LL, Cowling BJ, Fang VJ, et al. Viral shedding and clinical illness in naturally acquired influenza virus infections. *The Journal of Infectious Diseases* 2010; 201: 1509-1516.
20. Mossong J, Hens N, Jit M, et al. Social contacts and mixing patterns relevant to the spread of infectious diseases. *PLoS Med* 2008; 5: e74.
21. Gallagher KM and Updegraff JA. Health message framing effects on attitudes, intentions, and behavior: A meta-analytic review. *Ann Behav Med* 2011; 43: 101-116.
22. Lithopoulos A, Bassett-Gunter RL, Martin Ginis KA, et al. The effects of gain-versus loss-framed messages following health risk information on physical activity in individuals with multiple sclerosis. *Journal of Health Communication* 2017; 22: 523-531.
23. O'Keefe DJ and Jensen JD. The advantages of compliance or the disadvantages of noncompliance? A meta-analytic review of the relative persuasive effectiveness of gain-framed and loss-framed messages. *Annals of the International Communication Association* 2006; 30: 1-43.
24. Dunbar RI, Arnaboldi V, Conti M, et al. The structure of online social networks mirrors those in the offline world. *Social Networks* 2015; 43: 39-47.
25. Mac Carron P, Kaski K and Dunbar R. Calling Dunbar's numbers. *Social Networks* 2016; 47: 151-155.
26. Brotherton R, French CC and Pickering AD. Measuring belief in conspiracy theories: The generic conspiracist beliefs scale. *Front Psychol* 2013; 4: 279.
27. Vosoughi S, Roy D and Aral S. The spread of true and false news online. *Science* 2018; 359: 1146-1151.
28. Jervelund SS. How social media is transforming the spreading of knowledge: Implications for our perceptions concerning vaccinations and migrant health. *Scand J Public Health* 2018; 46: 167-169. editorial.
29. Lazer DM, Baum MA, Benkler Y, et al. The science of fake news. *Science* 2018; 359: 1094-1096.

30. Oyeyemi SO, Gabarron E and Wynn R. Ebola, Twitter, and misinformation: a dangerous combination? *Br Med J* 2014; 349: g6178.
31. Haer T, Botzen WW and Aerts JC. The effectiveness of flood risk communication strategies and the influence of social networks—Insights from an agent-based model. *Environ Sci Policy* 2016; 60: 44-52.
32. Berger J and Milkman KL. What makes online content viral? *J Marketing Res* 2012; 49: 192-205.
33. Garrett RK. The “echo chamber” distraction: Disinformation campaigns are the problem, not audience fragmentation. *Journal of Applied Research in Memory and Cognition* 2017; 6: 370-376.
34. Lyons T. Replacing Disputed Flags with Related Articles, <https://newsroom.fb.com/news/2017/12/news-feed-fyi-updates-in-our-fight-against-misinformation/> (2017, accessed 24 July 2018).
35. Glaeser EL and Ujhelyi G. Regulating misinformation. *J Public Econ* 2010; 94: 247-257.
36. MSNBC. "Finding Foothold in the Age of Trump" Television broadcast on April 10. 2018.
37. Allcott H and Gentzkow M. Social media and fake news in the 2016 election. *J Econ Perspect* 2017; 31: 211-236.
38. Ball J. *Post-truth: How bullshit conquered the world*. Biteback Publishing, 2017.
39. Shane S. From Headline to Photograph, a Fake News Masterpiece. *The New York Times*, Jan 18 2017.
40. Rochlin N. Fake news: Belief in post-truth. *Library Hi Tech* 2017; 35: 386-392.
41. Andrews MA and Bauch CT. Disease interventions can interfere with one another through disease-behaviour interactions. *PLoS Comput Biol* 2015; 11: e1004291.
42. Bisset KR, Feng X, Marathe M, et al. Modeling interaction between individuals, social networks and public policy to support public health epidemiology. In: *Winter Simulation Conference* 2009, pp.2020-2031. Winter Simulation Conference.
43. Chen F, Jiang M, Rabidoux S, et al. Public avoidance and epidemics: Insights from an economic model. *J Theor Biol* 2011; 278: 107-119.
44. d’Onofrio A and Manfredi P. Information-related changes in contact patterns may trigger oscillations in the endemic prevalence of infectious diseases. *J Theor Biol* 2009; 256: 473-478.
45. Durham DP and Casman EA. Incorporating individual health-protective decisions into disease transmission models: A mathematical framework. *J Royal Soc Interf* 2011: rsif20110325.
46. Guo D, Li KC, Peters TR, et al. Multi-scale modeling for the transmission of influenza and the evaluation of interventions toward it. *Sci Rep* 2015; 5: 8980.
47. Hatzopoulos V, Taylor M, Simon PL, et al. Multiple sources and routes of information transmission: Implications for epidemic dynamics. *Math Biosci* 2011; 231: 197-209.
48. Mao L. Predicting self-initiated preventive behavior against epidemics with an agent-based relative agreement model. *Journal of Artificial Societies and Social Simulation* 2015; 18: 6.
49. Smith MC and Broniatowski DA. Modeling influenza by modulating flu awareness. In: Cham, 2016, pp.262-271. Springer International Publishing.
50. Zhang H-F, Xie J-R, Chen H-S, et al. Impact of asymptomatic infection on coupled disease-behavior dynamics in complex networks. *EPL (Europhysics Letters)* 2016; 114: 38004.
51. Epstein JM, Parker J, Cummings D, et al. Coupled contagion dynamics of fear and disease: Mathematical and computational explorations. *PLoS One* 2008; 3: e3955.
52. Fenichel EP, Castillo-Chavez C, Ceddia MG, et al. Adaptive human behavior in epidemiological models. *Proceedings of the National Academy of Sciences* 2011; 108: 6306-6311.
53. Fu F, Rosenbloom DI, Wang L, et al. Imitation dynamics of vaccination behaviour on social networks. *Proceedings of the Royal Society of London B: Biological Sciences* 2011; 278: 42-49.
54. Nunez F, Ravello C, Urbina H, et al. A rule-based model of a hypothetical zombie outbreak: Insights on the role of emotional factors during behavioral adaptation of an artificial population. *arXiv preprint arXiv:12104469* 2012.
55. Tully S, Cojocar M and Bauch CT. Coevolution of risk perception, sexual behaviour, and HIV transmission in an agent-based model. *J Theor Biol* 2013; 337: 125-132.
56. Fajebe A. *Computational modeling of spontaneous behavior changes and infectious disease spread*. PhD, Georgia Institute of Technology, 2016.
57. Lukasik M, Cohn T and Bontcheva K. Point process modelling of rumour dynamics in social media. In: *Proceedings of the 53rd Annual Meeting of the Association for Computational Linguistics and the 7th International Joint Conference on Natural Language Processing (Volume 2: Short Papers)* 2015, pp.518-523.
58. Wang A, Wu W and Chen J. A novel rumour propagation model on social networks. *International Journal of Sensor Networks* 2017; 25: 126-133.

59. Nekovee M, Moreno Y, Bianconi G, et al. Theory of rumour spreading in complex social networks. *Physica A: Statistical Mechanics and its Applications* 2007; 374: 457-470.
60. Pearce CE. The exact solution of the general stochastic rumour. *Mathematical and Computer Modelling* 2000; 31: 289-298.
61. Huo La, Huang P and Guo C-x. Analyzing the dynamics of a rumor transmission model with incubation. *Discrete Dynamics in Nature and Society* 2012; 2012.
62. Jenness SM, Goodreau SM and Morris M. EpiModel: An R package for mathematical modeling of infectious disease over networks. *Journal of Statistical Software* 2018; 84.
63. Gaythorpe K, Trotter CL, Lopman B, et al. Norovirus transmission dynamics: A modelling review. *Epidemiol Infect* 2018; 146: 147-158.
64. Biggerstaff M, Cauchemez S, Reed C, et al. Estimates of the reproduction number for seasonal, pandemic, and zoonotic influenza: A systematic review of the literature. *BMC Infect Dis* 2014; 14: 480.
65. Blumberg S and Lloyd-Smith JO. Inference of R_0 and transmission heterogeneity from the size distribution of stuttering chains. *PLoS Comput Biol* 2013; 9: e1002993.
66. Sklenovská N and Van Ranst M. Emergence of monkeypox as the most important orthopoxvirus Infection in humans. *Frontiers in Public Health* 2018; 6.
67. Centers for Disease Control and Prevention. Norovirus in Healthcare Facilities Fact Sheet, <https://www.cdc.gov/hai/pdfs/norovirus/229110-ANoroCaseFactSheet508.pdf> (2011, accessed Oct 24 2018).
68. Lee RM, Lessler J, Lee RA, et al. Incubation periods of viral gastroenteritis: A systematic review. *BMC Infect Dis* 2013; 13: 446.
69. Centers for Disease Control and Prevention. Clinical Signs and Symptoms of Influenza, <https://www.cdc.gov/flu/professionals/acip/clinical.htm> (2008, accessed Oct 24 2018).
70. McCollum AM and Damon IK. Human monkeypox. *Clin Infect Dis* 2013; 58: 260-267.
71. Devasia T, Lopman B, Leon J, et al. Association of host, agent and environment characteristics and the duration of incubation and symptomatic periods of norovirus gastroenteritis. *Epidemiol Infect* 2015; 143: 2308-2314.
72. Mesquita J and Nascimento MS. A foodborne outbreak of norovirus gastroenteritis associated with a Christmas dinner in Porto, Portugal, December 2008. *Eurosurveillance* 2009; 14: 19355.
73. Centers for Disease Control and Prevention. The Symptoms of Norovirus, <https://www.cdc.gov/norovirus/about/symptoms.html> (2015, accessed Oct 24 2018).
74. Hadler JL, Konty K, McVeigh KH, et al. Case fatality rates based on population estimates of influenza-like illness due to novel H1N1 influenza: New York City, May–June 2009. *PLoS One* 2010; 5: e11677.
75. World Health Organization. Monkeypox, <http://www.who.int/news-room/fact-sheets/detail/monkeypox> (2018, accessed Oct 24 2018).
76. Gerdil C. The annual production cycle for influenza vaccine. *Vaccine* 2003; 21: 1776-1779.
77. Centers for Disease Control and Prevention. Smallpox Vaccine Guidance, <https://www.cdc.gov/poxvirus/monkeypox/clinicians/smallpox-vaccine.html> (2015, accessed Oct 24 2018).
78. Centers for Disease Control and Prevention. Key Facts About Seasonal Flu Vaccine, <https://www.cdc.gov/flu/protect/keyfacts.htm> (2018, accessed Oct 24 2018).

Table 1. Model assumptions and targets (stage 1)

	Norovirus	Influenza	Monkeypox
Model baseline target r0 or generations	r0=1.9 ⁶³	r0=1.47 ⁶⁴	Insufficient data to estimate r0 ⁶⁵ but assume 75% outbreaks have ≤ 4 generations of p2p transmission ⁶⁶
Targets in stage 2 (worse r0 or gen's)	r0=2.66	r0=2.06	Increase to 75% outbreaks have ≤ 7 generations
% agents who start model ill (infectious)	2%	2%	1%
Incubation period?	36 hours (mean) ^{67, 68}	48 hrs (mean), range 1-4 days SD = 0.5 d ⁶⁹	12d (mean) ⁷⁰ (range 7-17d)
Viral shedding pre- illness?	Assumed none	Yes: 1 day before illness starts	Assumed none
Viral shedding post- illness?	Mean 48 hours	Only if illness < 6 days	Assumed none
Infectious period while ill	Entire duration of illness ⁷¹⁻⁷³	First 6 days (mean) after symptoms onset, or until no longer ill (if < 6 days) ⁶⁹	Entire duration of illness
Duration of illness	46 hours ⁷¹⁻⁷³	7 days, with SD = 1.75 day ⁶⁹	21d (mean) ⁷⁰ (range 14-28d)
Chances of hospitalisation	0%	5%/d (when ill)	70%/d (only after 4d)
Case fatality rate	0% ⁶⁷	0.65% ⁷⁴	Low because assumed high income country setting, 1-2% ⁷⁵
Vaccination available	No	After 5 months ⁷⁶	After 18 wks ⁷⁷
Vaccination efficacy	Not applicable	100% if not incubating ⁷⁸	100% if < 4d after exposure, else 0 ⁷⁷
Vaccination uptake	All those with TP > 25 th percentile of TP% & susceptible (or incubating < 4 days if monkeypox)		
Model tests relevant to disease transmission	If neither side takes enough precautions AND viral shed risk is high enough		

Notes: **CFR = Case fatality rate; p2p = person to person.** Targets are from literature (influenza pandemics after 2009). The CFR for monkeypox is plausible ⁶⁵ but there is lack of data from high income country

settings. Model assumes that no shortage of vaccine (once available, there is always enough to meet demand). Delay in influenza is for production timeline; delay in monkeypox vaccine is for time to procure supply and to recognise need.

Table 2. Performance metrics and results to generate stage 1-2 models.

	Norovirus	Influenza	Monkeypox
Stage 1. Proportion of transmission risk due to viral shedding (5-95th percentiles for linked r0)	8.3% (1.75-2.06)	3.6% (1.27-1.58)	0.8% (0-0.53)
Stage 1. Mean case fatality rate % (CFR, 5-95th percentiles)	n/a	0.70% (0.42-1.03%)	1.04% (0-2.93%)
Stage 2. ΔTP value required to consistently increase r0 or gens (5-95th percentiles for linked r0)	1.9% (2.44-2.89)	1.05% (1.78-2.32)	1.1% (0-0.89)
Stage 2. Mean case fatality rate % (CFR, 5-95th percentiles)	n/a	0.62% (0.42-1.14%)	1.39% (0-1.68%)

Note: CFR = case fatality rate; gens = generations. These values most closely enabled meeting the r0 or #generations targets (in Table 1). Values in () are range of r0 or CFR, 5th to 9th percentiles, over ≥ 100 simulations. More detailed results are in supplemental information.

Table 3. Stage 1 (no sharing), stage 2 (outbreak exacerbated by bad advice), and stage 3 (results with intervention strategies). Mean values for given outbreak characteristics, with 5-95th percentiles to indicate range without the most extreme values.

Stage 1 No circulating advice					
	r0 or #gens	Duration (weeks)	Final Attack Rate	Peak attack rate	Case fatality rate
Norovirus	1.90 (1.75-2.06)	9.4	78.6%	8.6%	n/a
Influenza	1.46 (1.27-1.58)	13.6	59.2%	14.0%	0.70%
Monkeypox	75 th perc #gens = 4	7.3	1.3%	0.98%	1.04%
Stage 2. Circulating advice makes outbreak worse, r0 increased by 40% or #gens from 4 to 7. Good:Bad advice ratio is still 50:50					
	r0 or #gens	Duration (weeks)	Final Attack Rate	Peak attack rate	Case fatality rate
Norovirus	2.66 (2.44-2.89)**	8.7**	91.8%**	10.7%**	n/a
Influenza	2.08 (1.78-2.32)**	14.9**	82.7%**	18.2%**	0.62%**
Monkeypox	75 th perc #gens = 7	9.9	2.2%	1.2%	1.39%**
Stage 3.1 Strategies to reduce impacts of circulating bad advice in Stage 2 conditions: Ratio advice needed to revert to stage 1 r0 or #gens					
	Good:Bad advice ratio	Duration (weeks)	Final Attack Rate	Peak attack rate	Case fatality rate
Norovirus	59:41*	9.2	79.2%*	8.9%	n/a
Influenza	60:40	14.4	59.0%	13.4%	0.73%
Monkeypox	61:39	7.1	7.3%	1.0%	1.33%
Stage 3.2 Stage 2 conditions, but if Good:Bad advice ratio is 90:10					
	r0 or #gens	Duration (weeks)	Final Attack Rate	Peak attack rate	Case fatality rate
Norovirus	0.99 (0.95-1.03)**	3.9**	21.1%**	3.9%**	n/a
Influenza	0.88 (0.76-0.99)**	5.1**	12.8%**	5.0%**	0.74%**
Monkeypox	75 th perc #gens = 3**	5.6**	1.2%*	1.0%	0.96%
Stage 3.3: Stage 2 conditions, what % of agents need to be “immunised” against bad advice, needed to revert to stage 1 r0 or #gens					
	% “immunised”	Duration (weeks)	Final Attack Rate	Peak attack rate	Case fatality rate
Norovirus	20%	9.3	78.5%	8.9%	n/a

Influenza	22.5%	15.2**	59.6%	13.1%*	0.42%
Monkeypox	20-40%	6.9-8.4	1.3-1.5%	0.98-1.04%	0.86-1.34%

Stage 3.4: Stage 2 conditions, but if 90% of agents are “immunised”

	r0 or #gens	Duration (weeks)	Final Attack Rate	Peak attack rate	Case fatality rate
Norovirus	1.11 (1.03-1.22)**	5.0**	31.6%**	4.8%**	n/a
Influenza	1.02 (0.88-1.13)**	7.1**	22.4%**	7.5%**	0.15%**
Monkeypox	75 th perc #gens = 3*	5.6*	1.2%	1.0%	0.96%

Note: “immunised” means acquired perfect resistance against believing or sharing bad advice, rather than inability to catch norovirus, influenza or monkeypox. 75th perc #gens = the 75th percentile value (among all eligible simulations) for number of disease transmission generations; 75% of simulations had this number or fewer transmissions. Statistical significance: Wilcoxon rank sum tests with stage 1 outcomes as reference: ** means $p < 0.01$, while * means $0.01 < p < 0.05$.