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

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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 models [15]. 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 (r0) 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 r0 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 occured 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) r0 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 advice [21-23]. 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 to [26]). 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 elsewhere [2]. 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 stories [27]. 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 r0 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 r0 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 r0 or number of generations was achieved. We also tested (stage 3.4) if

"immunising" 90% of agents could reduce r0 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 r0s in stage 1 models, when mean TP = 50%, and the change in TP (ΔTP) required upon each information exposure to raise the r0 by 40% (stage 2 models). Table 3 shows model run results (r0, 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 r0 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), r0 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 r0 (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 rates [41-50]). 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 process [57-59], 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 r0 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

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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 > 25th 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	75thperc #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	75thperc #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	75thperc #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	75thperc #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 $0.01 < p < 0.05$.