Theoretical Population Biology 170 (2026) 11–25

Contents lists available at ScienceDirect

Theoretical Population Biology

journal homepage: www.elsevier.com/locate/tpb

Correlation between multiple protective health behaviors can determine the outcome of disease spread

Rohan S. Mehta

Department of Biology, Elmhurst University, 190 Prospect Ave, Elmhurst, IL, 60126, USA

ARTICLE INFO

Dataset link: https://github.com/rohansmehta/mask_vaccine_interaction_code

Keywords:
Epidemic modeling
Coupled contagion
Social contagion
Vaccination
Mask-wearing
Disease-behavior dynamics
Dynamical systems

ABSTRACT

The impact of human health-related behavior on the spread of the disease continues to be prominent due to the proliferation of sentiment against public health-promoting behaviors. The recent SARS-COV-2 pandemic brought these effects into sharp focus. In the process, we have learned that our current understanding of how human behavior affects disease spread is insufficient in scope and intricacy. In particular, how different health-related behaviors themselves interact with each other, and how those interactions affect disease spread, is largely unknown. In this paper, we use a coupled-contagion model to study the interaction of two different health behaviors — vaccination and mask-wearing. We study how a positive or negative association between these behaviors — e.g. a pro-vaccine person may be more or less likely to be pro-mask-wearing — affects disease dynamics. We find that the strength of such an association alone can determine the outcome of disease spread in the short term and the long term. In addition to studying human health-related behaviors separately, it is vitally important to understand how these behaviors interact with each other and how these associations affect the outcomes of epidemics.

1. Introduction

The effects of human behavior on disease spread are as old as modern medical interventions themselves; the first successful vaccine for smallpox was met with anti-vaccine sentiment from many different groups of people (Nelson and Rogers, 1992). Despite the recent dramatic increase in efforts to model the effects of human behavior on disease spread (Funk et al., 2010; Bauch and Galvani, 2013; Manfredi and D'Onofrio, 2013; Funk et al., 2015; Verelst et al., 2016; Weston et al., 2018; Perra, 2021; Proverbio et al., 2025), the SARS-COV-2 pandemic revealed that our understanding of human health-related behavior and how it affects disease spread is currently inadequate for large-scale infectious disease management. Existing compartmental models leave room for improvement in capturing the complexity of human behavior (Iranzo and Pérez-González, 2021), although there has been progress in game-theoretical models that incorporate a wide variety of individual decision-making and economic factors (Brüne and Wilson, 2020; Kabir and Tanimoto, 2020; Nagurney, 2021). On the other end of the modeling spectrum, the successful application of an agent-based model that incorporated several aspects of human behavior (Elbanna et al., 2020) required a large amount of coordination and real-time changes to intervention strategies (Ranoa et al., 2022). In addition, health behavior is culture-dependent (Rosal and Bodenlos, 2009), and so models must be flexible enough to incorporate the potential diversity of interactions between health-related behaviors.

Despite these difficulties, it has recently been shown that even relatively simple compartmental models that incorporate behavioral feedback mechanisms can perform as well as and even occasionally outperform models that are driven by real-time data; incorporating behavior can improve the success of an epidemiological model (Gozzi et al., 2025).

Much of the effort towards incorporating human health behavior into epidemiological models has focused on one behavior at a time. If multiple behaviors have been studied, then they have been studied without potential correlation within the individual. However, it is possible that a person's opinion towards one health-related behavior may be correlated to, or even anticorrelated to, their opinion towards a different health-related behavior. An online experiment (Patel, 2026) demonstrated that there can be complex correlations between vaccination sentiment and an NPI (in this case, social distancing). In particular, Patel (2026) found that unvaccinated individuals socially distance further if they have a stronger pro-vaccine sentiment, whereas fully vaccinated individuals socially distance less if they have a stronger pro-vaccine sentiment; sentiments can be correlated and anticorrelated. The recent work of Chen et al. (2025) studied the effect of a correlation between vaccination and adherence to a generic NPI and found that this correlation level can exert some control over disease prevalence over time. In this study, we use coupled contagion dynamics to present

E-mail address: rohan.mehta@elmhurst.edu.

https://doi.org/10.1016/j.tpb.2026.04.003
Received 27 October 2025
Available online 11 June 2026

a framework that can be used to study specific interactions between multiple health-related behavior sentiments and their effect on disease dynamics. We focus on the specific health-related behavior sentiments of vaccination and mask-wearing.

One modeling strategy that can be exploited to specifically study the interaction between multiple different human health-related behaviors and disease dynamics is the coupled contagion approach. In this approach, the disease and a human opinion on a health-related behavior are treated as contagions, where the spread of the opinion affects health-related behavior which then affects the spread of the disease (Funk et al., 2010; Wang et al., 2015). Early models using the coupled contagion framework focused on either generic traits (Tanaka et al., 2002), disease awareness (Funk et al., 2009), or disease fear (Epstein et al., 2008; Perra et al., 2011). Mehta and Rosenberg (2020) defined the term "cultural pathogen" to refer to a contagious opinion for health-related behavior that decreases the health of the carrier of the opinion. Note that this concept is distinct from the "parasitic contagion" from Hébert-Dufresne et al. (2020), which describes a contagion that simultaneously harms and benefits from the presence of another contagion. The model from Mehta and Rosenberg (2020) and other recent models (e.g. Salathé and Bonhoeffer, 2008; Wang et al., 2016; Fu et al., 2017) used a coupled contagion approach to study the effects of vaccine opinion on disease dynamics, and found that these opinions can determine both short- and long-term outcomes of disease spread. In the last two decades, models have also studied the effect of mask-wearing behavior on disease spread (Tracht et al., 2010; Brienen et al., 2010; Eikenberry et al., 2020), also see citations in Perra (2021), but few have studied this behavior under a coupled contagion framework (e.g. Qiu et al., 2022).

In one of the first studies to use a coupled contagion model with more than one behavior and a disease, Epstein et al. (2021) notably studied a model that combined a disease fear contagion with a vaccine sentiment contagion, with the result that multiple waves of infection, and changes in infection form and intensity, are significantly affected by the characteristics of both behaviors. Teslya et al. (2022) studied the effects of two mutually-exclusive, competing health opinions (one health-positive and one health-neutral) and found complex results, including the fact that both opinions can coexist and that the initial state of population opinion can affect the disease dynamics. Other models have studied the effects of multiple non-pharmaceutical interventions (NPIs) simultaneously (see citations in Perra, 2021), but relatively few have studied their joint dynamics under a coupled contagion framework and incorporated correlations between behaviors (e.g. Chen et al., 2025).

In this study, we consider a model of disease spread coupled with two pairs of competing behavioral contagions: pro-vaccine sentiment, anti-vaccine sentiment, pro-mask sentiment, and anti-mask sentiment. In addition to studying these two different health-related behaviors as cultural pathogens, we model a potential tendency of humans to adopt one type of behavior if they have already adopted another type of behavior. For example, people may be either more or less likely to adopt anti-masking sentiment if they have already adopted anti-vaccine sentiment. We study the long-term behavior of this model and establish conditions under which we expect endemic disease, endemic anti-mask sentiment, and endemic anti-vaccine sentiment. We find situations where different sentiment and disease states are potentially stable at the same time, and characterize the set of initial conditions of population sentiment in which each such state is reached. We also study the effect of the spread of masking sentiments and vaccination sentiments in the short-term on a single epidemic of a new disease. We find that the strength of the association between the two cultural pathogens can can strongly influence, and in some cases determine the outcome of disease spread regardless of the specific parameters of the social or biological contagions themselves, provided that the biological contagion does not self-extinguish in isolation. In addition, we show how a simple model of mask-wearing sentiment acting as a cultural pathogen affects the dynamics of a disease, and show that the spread of masking behavior is overall less important than the spread of vaccination behavior unless mask effectiveness is very high.

2. Model

This model is a compartmental model based off of the standard Kermack–McKendrick SIR model (Kermack and McKendrick, 1927). Each individual is placed into compartments from three categories: disease state, vaccine sentiment state and mask sentiment state. In the framework of Proverbio et al. (2025), this is an explicit mean-field model, with disease compartments split up by behavior in a manner similar to e.g. Proverbio et al. (2024); the dynamics of a general form of SIR model with split compartments is studied by Van den Driessche and Watmough (2002).

The key assumptions of this model are as follows: the disease follows standard SIR dynamics and resembles measles. There are four sentiments: pro-vaccine, anti-vaccine, pro-mask, and anti-mask. Each of these acts as a simple contagion, and an individual exhibits one of each opinion at any given time (similar to the approach of Teslya et al., 2022). These behaviors have previously been observed and modeled as social contagions (e.g. Fu et al., 2017; Mehta and Rosenberg, 2020; Konstantinou et al., 2021; Qiu et al., 2022; Karashiali et al., 2023), but generally not at the same time.

The disease state is the same as in a simple SIR model with vital dynamics and vaccination: individuals can be susceptible to the disease (S), infected with the disease (I), or recovered from the disease (R). The R compartment also includes vaccinated individuals. Individuals enter the population as S, can transition from S to I by interacting with an I individual, and can transition from S to R by vaccination. Infected individuals can also transition from S to R by recovering from the disease.

For the vaccine sentiment state, individuals can either be pro-vaccine (P) or anti-vaccine (A). Individuals transition between the two states by interacting with individuals from the other state. Only pro-vaccine individuals can be vaccinated (i.e. can make the $S \to R$ transition.)

For the mask sentiment state, individuals can either be pro-mask (M), or anti-mask (N). Individuals transition between the two states by interacting with individuals from the other state. Pro-mask individuals exhibit a decrease in the transmission probability of the disease (i.e. the rate of the $S \to I$ transition is decreased).

We chose a contagion model for sentiment as opposed to a game-theoretic opinion dynamics model (e.g. Huang and Zhu, 2022) because the behavioral phenomenon that is of primary study here is the tendency to be receptive to one kind of opinion if you already possess another kind of opinion, which is naturally modeled as a contagion. In addition, there is empirical evidence that health-related opinions act as social contagions (Konstantinou et al., 2021; Karashiali et al., 2023). In particular, transitions between these sentiment states have been observed in both directions using Twitter sentiment analysis (Sediqin, 2021).

Each of the 12 compartments in this model is labeled by the disease state, vaccine sentiment state, and mask sentiment state, in that order. Table 1 describes the notation for each compartment. We denote marginal variables by either one or two letters (e.g., $PM = SPM+IPM+RPM$ and $N = SPN+SAN+IPN+IAN+RPN+RAN$). Table 2 describes the parameters used in the model.

We can think of this model as consisting of three "subsystems" that interact: the disease subsystem, the vaccine sentiment subsystem, and the mask sentiment subsystem. At any given point, any individual inhabits one compartment from each subsystem. In addition, the vaccine and mask sentiment subsystems can be combined into a larger subsystem — the sentiment subsystem — that does not depend on the disease subsystem.

Fig. 1A depicts the interactions between the disease, vaccine sentiment, and mask sentiment subsystems. Each of the four sentiment states acts as a simple contagion; for example, an individual transitions from pro- to anti-vaccine at a rate a upon interaction with an anti-vaccine person. Only individuals who are in the pro-vaccine state (P) can

Table 1
Description of model variables (compartments). The marginal variables (labeled with fewer than three letters) are sums of the 12 main variables of the model.

Compartment	Disease state	Vaccine sentiment State	Mask sentiment state
SPM	Susceptible	Pro-vaccine	Pro-mask
SPN	Susceptible	Pro-vaccine	Anti-mask
SAM	Susceptible	Anti-vaccine	Pro-mask
SAN	Susceptible	Anti-vaccine	Anti-mask
IPM	Infected	Pro-vaccine	Pro-mask
IPN	Infected	Pro-vaccine	Anti-mask
IAM	Infected	Anti-vaccine	Pro-mask
IAN	Infected	Anti-vaccine	Anti-mask
RPM	Recovered	Pro-vaccine	Pro-mask
RPN	Recovered	Pro-vaccine	Anti-mask
RAM	Recovered	Anti-vaccine	Pro-mask
RAN	Recovered	Anti-vaccine	Anti-mask
SM	Susceptible		Pro-mask
SN	Susceptible		Anti-mask
PM		Pro-vaccine	Pro-mask
PN		Pro-vaccine	Anti-mask
AM		Anti-vaccine	Pro-mask
AN		Anti-vaccine	Anti-mask
S	Susceptible		
I	Infected		
R	Recovered		
P		Pro-vaccine	
A		Anti-vaccine	
M			Pro-mask
N			Anti-mask

Table 2
Description of model parameters. The ranges supplied here are the ranges used in the simulations in this study; in principle, most of these parameters are unbounded.

Parameter	Description	Range
b	Birth rate	$[0, 0.03]$
r	Disease transmission rate	$[0, 3]$
z_S	Reduction in disease susceptibility due to masking	$[0, 1]$
z_I	Reduction in disease transmission due to masking	$[0, 1]$
g	Recovery rate	$[0, 1]$
v	Vaccination rate	$[0, 1]$
p	Pro-vaccine transmission rate	$[0, 1]$
a	Anti-vaccine transmission rate	$[0, 1]$
m	Pro-mask transmission rate	$[0, 1]$
n	Anti-mask transmission rate	$[0, 1]$
x	Sentiment association	$[-1, 1]$

transition from the susceptible state (S) directly to the recovered state (R) by vaccination. Following Tracht et al. (2010), Eikenberry et al. (2020), and Qiu et al. (2022), mask-wearing decreases transmission rate of the disease by a fraction z_S if the susceptible individual is wearing the mask and z_I if the infected individual is wearing the mask. These effects are multiplicative, so that if both the susceptible and the infected individual are wearing a mask, then the transmission rate is multiplied by $z_S z_I$.

Fig. 1B depicts how the vaccine and mask sentiment systems interact with each other. For a particular sentiment, the pro- and anti-state contagions compete with each other; the one with a higher transmission rate wins. This interaction is affected by the association parameter x, which ranges from -1 to 1 and describes how an individual's tendency to exhibit a position on vaccines is similar to that individual's tendency to exhibit a position on masks. For example, $x > 0$ means that if an individual is pro-vaccine, that individual is more likely to turn pro-mask when interacting with a pro-mask individual than if the individual were anti-vaccine. An individual is also more likely to turn pro-vaccine if the individual is pro-mask. The opposite occurs for $x < 0$; an individual is more likely to turn pro-vaccine if that individual is anti-mask and more likely to turn anti-vaccine if pro-mask. When $x = 0$, there is no association between behaviors, so the two contagions are independent. The association parameter x should be interpreted as modulating transmission probabilities during pairwise interactions; it summarizes the extent to which exhibiting one sentiment affects the probability of adopting another sentiment. This formulation is consistent with many different theoretical frameworks, including cognitive dissonance/consistency, social identity alignment, and network homophily; we do not attempt to distinguish between these options as the model is intended to capture their aggregate effect.

Fig. 1C depicts all the possible transitions between all the compartments of the model. The four states that correspond to a particular disease state and all possible sentiment state combinations can each transition among each other via the four different sentiment contagions. The four susceptible states can become infected, but the infection rates for SPM and SAM (the pro-mask states) are decreased compared to those for SPN and SAN (the anti-mask states) by a factor of z_S and the infection rates are also decreased by a factor of z_I if the infected individual is pro-mask (IPM or IAM). We use the standard assumption that only one state transition can happen at once (so that e.g. the transition $SPM \to IAN$ is not allowed.)

Eq. (1) shows the full system of equations corresponding to the model from Fig. 1C. Note that all variables must sum to 1, so we can omit one variable (RAN) from most further analysis.

$$
\begin{aligned}
\frac{dSPM}{dt} &= b(PM) + (1+x)pP(SAM) + (1+x)mM(SPN) \\
&\quad - SPM\left[rz_S\left[IN + z_I(IM)\right] + (1-x)(aA+nN) + b + v\right] \\
\frac{dSAM}{dt} &= b(AM) + (1-x)aA(SPM) + (1-x)mM(SAN) \\
&\quad - SAM\left[rz_S\left[IN + z_I(IM)\right] + (1+x)(pP+nN) + b\right] \\
\frac{dSPN}{dt} &= b(PN) + (1-x)pP(SAN) + (1-x)nN(SPM) \\
&\quad - SPN\left[r\left[IN + z_I(IM)\right] + (1+x)(aA+mM) + b + v\right] \\
\frac{dSAN}{dt} &= b(AN) + (1+x)aA(SPN) + (1+x)nN(SAM) \\
&\quad - SAN\left[r\left[IN + z_I(IM)\right] + (1-x)(pP+mM) + b\right] \\
\frac{dIPM}{dt} &= rz_S(SPM)\left[IN + z_I(IM)\right] + (1+x)pP(IAM) \\
&\quad + (1+x)mM(IPN) - IPM\left[g + (1-x)(aA+nN) + b\right] \\
\frac{dIAM}{dt} &= rz_S(SAM)\left[IN + z_I(IM)\right] + (1-x)aA(IPM) \\
&\quad + (1-x)mM(IAN) - IAM\left[g + (1+x)(pP+nN) + b\right] \\
\frac{dIPN}{dt} &= r(SPN)\left[IN + z_I(IM)\right] + (1-x)pP(IAN) \\
&\quad + (1-x)nN(IPM) - IPN\left[g + (1+x)(aA+mM) + b\right] \\
\frac{dIAN}{dt} &= r(SAN)\left[IN + z_I(IM)\right] + (1+x)aA(IPN) \\
&\quad + (1+x)nN(IAM) - IAN\left[g + (1-x)(pP+mM) + b\right] \\
\frac{dRPM}{dt} &= v(SPM) + g(IPM) + (1+x)pP(RAM) + (1+x)mM(RPN) \\
&\quad - RPM\left[(1-x)(aA+nN) + b\right] \\
\frac{dRAM}{dt} &= g(IAM) + (1-x)aA(RPM) + (1-x)mM(RAN) \\
&\quad - RAM\left[(1+x)(pP+nN) + b\right] \\
\frac{dRPN}{dt} &= v(SPN) + g(IPN) + (1-x)pP(RAN) + (1-x)nN(RPM) \\
&\quad - RPN\left[(1+x)(aA+mM) + b\right] \\
\frac{dRAN}{dt} &= g(IAN) + (1+x)aA(RPN) + (1+x)nN(RAM) \\
&\quad - RAN\left[(1-x)(pP+mM) + b\right].
\end{aligned}
\tag{1}
$$

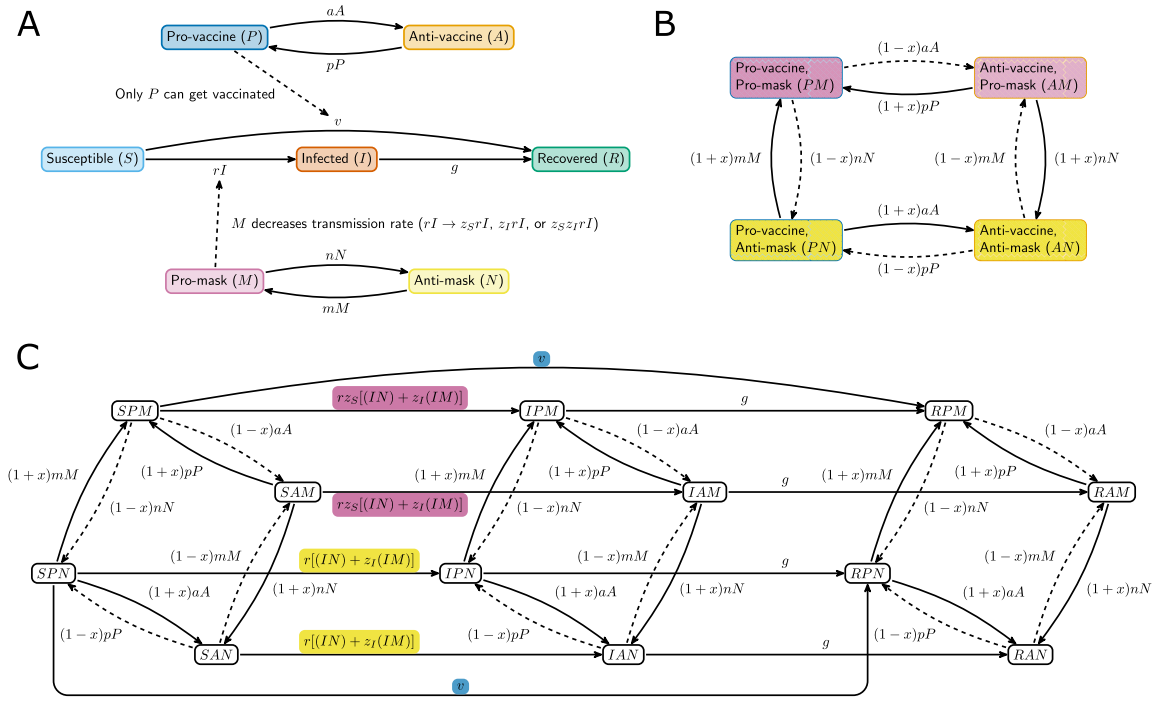

Fig. 1. Diagrams of the model interactions. (A) Interaction between the sentiment subsystems the and disease subsystem. Pro- and anti-vaccine and pro- and anti-mask sentiments act as simple contagions. Only pro-vaccine individuals can be vaccinated. Pro-mask individuals have a modified, decreased, probability of disease transmission. (B) Interaction between sentiment subsystems. The contagion behavior of the two sentiments is mediated by the association parameter x. For large positive x, transitions labeled by solid arrows are more frequent, and for large negative x, transitions labeled by dashed lines are more frequent. (C) Full model with all transitions between states. The two possible vaccination transitions are colored blue. The infection rates are colored by the mask sentiment: green for pro-mask (and so the infection rate is decreased), yellow for anti-mask (and so the infection rate is increased).

3. Results

3.1. Long-term dynamics

3.1.1. Sentiment equilibria

In the long run, this system reaches one of several possible stable equilibria. The dynamics of the sentiment subsystem are independent of the dynamics of the disease subsystem (but not vice versa). The sentiment subsystem is

$$\begin{aligned}
\frac{dPM}{dt} &= (1+x)pP(AM) + (1+x)mM(PN) - (1-x)(PM)(aA+nN)\\
\frac{dPN}{dt} &= (1-x)pP(AN) + (1-x)nN(PM) - (1+x)(PN)(aA+mM)\\
\frac{dAM}{dt} &= (1-x)aA(PM) + (1-x)mM(AN) - (1+x)(AM)(pP+nN)\\
\frac{dAN}{dt} &= (1+x)aA(PN) + (1+x)nN(AM) - (1-x)(AN)(pP+mM)
\end{aligned} \tag{2}$$

There are four sentiment subsystem equilibria that are nontrivial and do not rely on specific equalities in parameter values, each corresponding to one particular sentiment type (PM, PN, AM, or AN), taking over the population. See Appendix for details.

To express the stability conditions for these equilibria, we use the following two quantities:

$$\Delta_V = \frac{p-a}{p+a} \tag{3}$$

$$\Delta_M = \frac{m-n}{m+n}. \tag{4}$$

Δ_V (Eq. (3)) is the relative transmissability or persuasiveness of pro-vaccine sentiment compared to anti-vaccine sentiment, and Δ_M (Eq. (4)) is the equivalent value for masking sentiment. Table 3 summarizes the stability conditions for the four possible equilibria. From Table 3 and the knowledge that $-1 \le x, \Delta_V, \Delta_M \le 1$, we can deduce that ranging x from -1 to 1 will yield all possible sentiment states as stable at some point, at least in a situation where bistability is possible (i.e., the bottom two columns of Table 3).

Table 3
Equilibria and stability conditions for the sentiment subsystem. Note that the last two rows are for bistability; the population is not ever simultaneously in a state where $\widehat{PM} = \widehat{AN} = 1$ or $\widehat{PN} = \widehat{AM} = 1$.

$\widehat{PM}$	$\widehat{PN}$	$\widehat{AM}$	$\widehat{AN}$	$\lambda_1 < 0$	$\lambda_2 < 0$	$\lambda_3 < 0$
1	0	0	0	$x > -\Delta_V$	$x > -\Delta_M$	$x < 1$
0	1	0	0	$x < \Delta_V$	$x < -\Delta_M$	$x > -1$
0	0	1	0	$x < -\Delta_V$	$x < \Delta_M$	$x > -1$
0	0	0	1	$x > \Delta_V$	$x > \Delta_M$	$x < 1$
1	0	0	1	$x > \lvert\Delta_V\rvert$	$x > \lvert\Delta_M\rvert$	$x < 1$
0	1	1	0	$x < -\lvert\Delta_V\rvert$	$x < -\lvert\Delta_M\rvert$	$x > -1$

The effect of x can be summarized by stating that a strong enough association can override existing relationships between sentiments to "drag" a weaker position into dominance if the corresponding position in the other sentiment is strong enough. For example, PM can be stable even if anti-vaccine or anti-mask sentiment is dominant ($\Delta_V, \Delta_M < 0$) so long as x is large enough. If one of those "anti" states is dominant, "large enough" only depends on the strength of that state.

The conditions in Table 3 leave the possibility open for multiple equilibria to be stable at once. The first possible situation involves a very large positive x, in which case the association can overpower sentiment transmission and "drag" the double-pro (PM) or double-anti (AN) state into dominance. The second possible situation involves a very large negative x, which has the opposite effect and can drag one of the "mixed" states (PN or AM) into dominance.

Because there is complete symmetry between the two sentiments in the sentiment subsystem, we can choose exactly one of each option for several choices without loss of generality as an illustrative example of

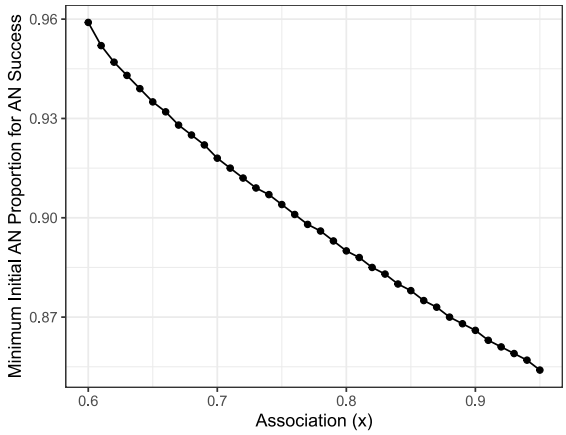

Fig. 2. Association affects which equilibrium is attained in a bistable regime. The minimum required initial proportion for AN to dominate over PM when both are potentially stable decreases with increasing association x. Thus, a stronger association between behaviors can change a situation from PM to AN, potentially dramatically shifting the outcome of the disease spread. Parameters used in these simulations are $p = 0.8$, $a = 0.2$, $m = 0.6$, $n = 0.4$, $x \in [0.6 - 0.95]$, and $PM_0 \in [0, 1]$.

the potential for bistability: we focus on the case where the double-pro sentiments PM and AN are possible, we let $\Delta_V > \Delta_M > 0$, and range over a variety of initial conditions to see when which cases AN dominates vs PM. In this case, we require x to be large but less than 1, and we expect that we are in the basin of attraction of the AN equilibrium more when we start close to the equilibrium (i.e., $AN_0 \approx 1$) and when x is larger. Because $\Delta_V, \Delta_M > 0$, the two "positive" sentiment contagions are stronger than the "negative" ones, so the pair PM should dominate under more initial conditions.

This reasoning is shown in Fig. 2, where we fixed $p = 0.8$, $a = 0.2$, $m = 0.6$, and $n = 0.4$. Thus, $\Delta_V = 0.6$ and $\Delta_M = 0.2$. We ranged x from 0.6 to 0.95. These parameters satisfy the condition in Table 3 for bistability of PM and AN. For simplicity, we fixed $PN_0 = AM_0 = 0.01$ and ranged PM_0 from 0 to 1 in intervals of 0.01. In this case, $\widehat{AN} = 1$ when AN_0 is large enough (and PM_0 is small enough). Fig. 2 shows the minimum value of AN_0 such that $\widehat{AN} = 1$ for a range of values of x such that bistability is possible. We see that as x increases, it becomes easier for the unfavored pair AN to dominate over the favored pair PM, suggesting that association alone between behaviors is sufficient to switch the outcome of the dynamics.

3.1.2. Disease equilibria

We can use the sentiment subsystem results to find the different disease subsystem equilibria. The disease subsystem is

$$\begin{aligned}\frac{dS}{dt} &= b[1-S] - r\left[SN + z_S(SM)\right]\left[IN + z_I(IM)\right] - v(SP)\\ \frac{dI}{dt} &= r\left[SN + z_S(SM)\right]\left[IN + z_I(IM)\right] - (g+b)I\\ \frac{dR}{dt} &= v(SP) + gI - bR\end{aligned} \tag{5}$$

First, we define the standard epidemiological quantity

$$R_0 = \frac{r}{g+b}. \tag{6}$$

There are two possible options for the equilibrium value of infected individuals $\widehat{I}$: either $\widehat{I} = 0$ and the disease is extinct in the long term or $\widehat{I} > 0$ and the disease is endemic. Each of these options can combine with all four of the sentiment subsystem equilibria to create 8 possible situations. Table 4 lists these situations with the corresponding equilibrium values of the variables. As seen in Table 4, the presence of both vaccination and masking decreases endemic disease.

Fig. 3 displays how the critical R_0 (aka the value at which R_0 must exceed for the disease to be endemic) varies with the association parameter x and the sentiment subsystem equilibrium in the case where $\Delta_V < \Delta_M < 0$ (so each anti-contagion is stronger than each pro-contagion, and anti-vaccine sentiment is stronger than anti-mask sentiment). For illustrative purposes, we chose $b = 0.02$, $v = 0.14$, and $z_S z_I = 0.3$. The critical R_0 is lowest when both anti-sentiments dominate ($\widehat{AN} = 1$) and highest when both pro-sentiments dominate ($\widehat{PM} = 1$). The multiplicative nature of the joint effect of masking and vaccination is visible in how much higher the critical R_0 is for $\widehat{PM} = 1$ than for both $\widehat{PN} = 1$ and $\widehat{AM} = 1$, where only one pro-sentiment is dominant. From Fig. 3, we see that the fate of a non-self-extinguishing disease (aka $R_0 > 1$) can be entirely determined by the value of x, so long as its R_0 is less than the critical value for PM.

3.2. Example

Fig. 4 displays the equilibrium value of disease ($\widehat{I}$) for each sentiment equilibrium, a range of values of z and v and disease parameters $b = 0.02$, $r = 2.14$, $g = 0.014$, $R_0 \approx 63$ (chosen for visibility of the boundary curves). In a situation with a highly contagious disease, in order to eliminate the disease in the long-term, it is critical for pro-vaccine sentiment to dominate. Mask-wearing sentiment has a comparatively smaller effect on endemic disease. The effect of the two sentiments combined is multiplicative.

3.3. Sensitivity analysis

Following the technique outlined by Marino et al. (2008), we performed a sensitivity analysis on all the parameters and initial conditions on all variables, as well as the marginal variables S, I, P, and M. The full results of this are provided in the Appendix and Table 5. Parameters that significantly correlated positively with the infected rate I are the anti-vaccine sentiment transmission rate a, the anti-mask transmission rate n, the disease transmission rate r, and, notably, the interaction term x. Parameters that significantly correlated negatively with I are pro-vaccine transmission rate p, pro-mask transmission rate m, and recovery rate g.

The primary effect of increasing the interaction term x was to increase the proportion of compartments where the sentiments "match" (both pro- or both anti-) and decrease the proportion of compartments where the sentiments are opposed.

3.4. Short-term dynamics

Here we study the effect of associated masking and vaccination behavior on a single epidemic of a new disease. We assume that a population is generally pro-vaccine and pro-mask with respect to this disease. The only non-susceptible, non-pro-vaccine, and non-pro-mask individuals in the population are one infected individual, one anti-mask individual, and one anti-vaccine individual, with every other state of each of these individuals pro-sentiment and/or disease susceptible. Our initial condition is

$$\begin{aligned}SPM &= 1 - \frac{3}{1000}\\ SAM &= \frac{1}{1000}\\ SPN &= \frac{1}{1000}\\ IPM &= \frac{1}{1000},\end{aligned}$$

and everything else is zero. This initial condition is chosen to model a situation that is highly unfavorable for disease spread, allowing for the sentiment dynamics to be potentially important. These results are not intended to be exhaustive.

For our parameters, we use disease parameters representative of measles ($r = 2.14$ and $g = v = 0.14$ as used in Mehta and Rosenberg,

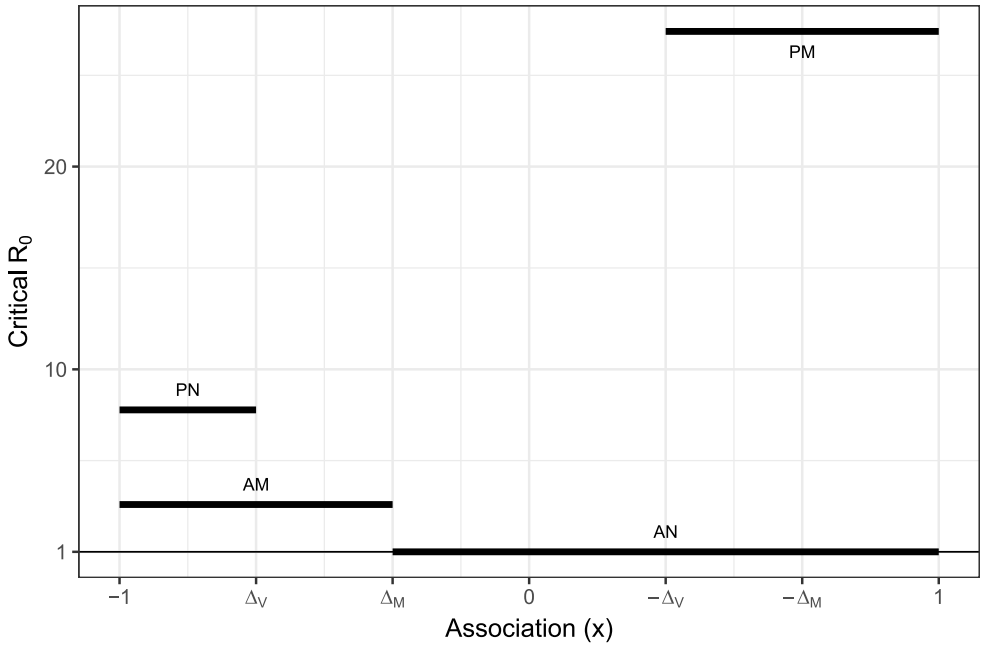

Fig. 3. Association can determine disease endemism. The critical value of R_0 that a disease must exceed to be endemic is highest when both pro-sentiments are dominant and lowest when both anti-sentiments are dominant. The association parameter x determines which sentiment state is dominant, and any dominant sentiment state can be obtained with a particular value of x. Critical R_0 values are obtained from Table 4. The horizontal lines indicate critical values of R_0, labeled by which sentiment state is dominant. Δ_V and Δ_M are defined in Eqs. (3) and (4), respectively. For this illustration, $b = 0.02$, $v = 0.14$, and $z_S z_I = 0.3$.

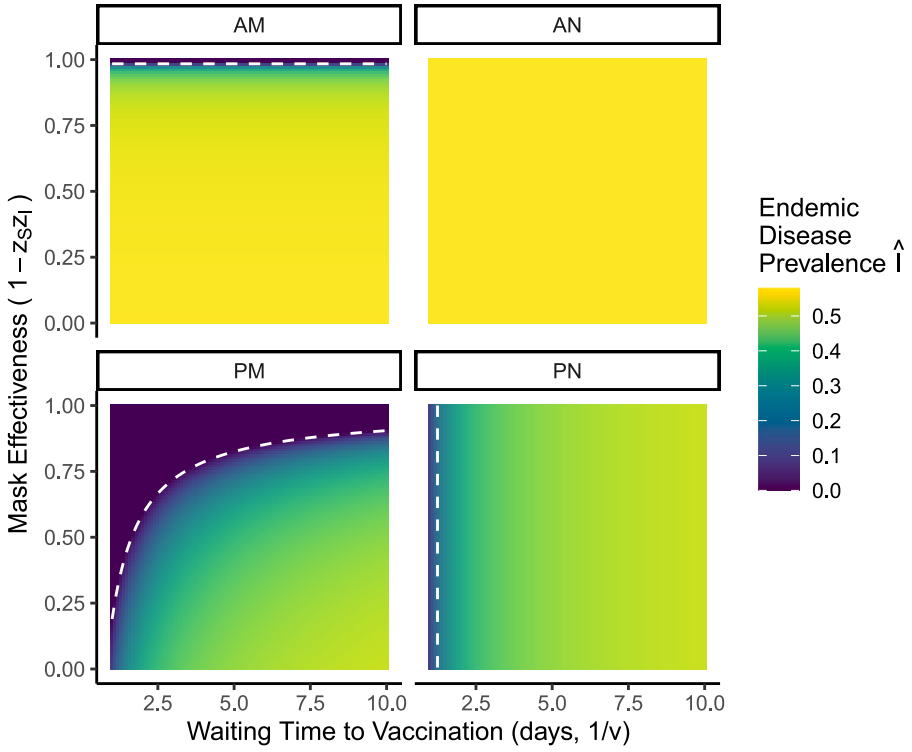

Fig. 4. Endemic disease prevalence (the equilibrium value $\hat{I}$ from Table 4) is affected by which sentiment state is dominant. For AM, the population is anti-vaccine and pro-mask. For AN, the population is anti-vaccine and anti-mask. For PM, the population is pro-vaccine and pro-mask. Finally, for PN, the population is pro-vaccine and anti-mask. For the two anti-vaccine sentiment states AM and AN, mask effectiveness makes little difference on endemic disease. For the two pro-vaccine states PM and PN, vaccination rate and mask effectiveness have strong effects, leading to situations where sentiment presence can affect disease endemism. The white dashed lines represent the boundary between disease endemism at equilibrium and disease extinction at equilibrium. For this illustration, $b = 0.02$, $r = 2.14$, and $g = 0.014$. For AM, this line is $1 - z_S z_I = 1 - \frac{1}{R_0} \approx 0.98$ which is slightly higher than the value of 0.97 obtained by using the masking effects that are used by Qiu et al. (2022); for PM, this curve is $1 - z_S z_I = 1 - \left(1 + \frac{v}{b}\right) \frac{1}{R_0}$; finally, for PN, this line is $\frac{1}{v} = b(R_0 - 1) \approx 1.24$, which is within the reasonable range of timescales with regards to practical vaccination (at least one day).

Table 4
Disease equilibrium values for each sentiment equilibrium scenario. Note that for each scenario, only one pair of sentiment states exists, so there are only three potential nonzero states. The recovered state can be obtained by subtracting the susceptible and infected states from 1.

Sentiment equilibrium	$\hat{S}$	$\hat{I}$	Stability condition
$\widehat{PM}=1$	$\frac{b}{v+b}$	0	$R_0 < \frac{1}{z_S z_I}\left(1+\frac{v}{b}\right)$
$\widehat{PM}=1$	$\frac{1}{z_S z_I R_0}$	$\frac{b}{g+b}\left[1-\left(1+\frac{v}{b}\right)\frac{1}{z_S z_I R_0}\right]$	$R_0 > \frac{1}{z_S z_I}\left(1+\frac{v}{b}\right)$
$\widehat{PN}=1$	$\frac{b}{v+b}$	0	$R_0 < 1+\frac{v}{b}$
$\widehat{PN}=1$	$\frac{1}{R_0}$	$\frac{b}{g+b}\left[1-\left(1+\frac{v}{b}\right)\frac{1}{R_0}\right]$	$R_0 > 1+\frac{v}{b}$
$\widehat{AM}=1$	1	0	$R_0 < \frac{1}{z_S z_I}$
$\widehat{AM}=1$	$\frac{1}{z_S z_I R_0}$	$\frac{b}{g+b}\left[1-\frac{1}{z_S z_I R_0}\right]$	$R_0 > \frac{1}{z_S z_I}$
$\widehat{AN}=1$	1	0	$R_0 < 1$
$\widehat{AN}=1$	$\frac{1}{R_0}$	$\frac{b}{g+b}\left[1-\frac{1}{R_0}\right]$	$R_0 > 1$

2020), with $b = 0$ so that we look at a single instance of potential disease spread over a short time scale. As we are primarily concerned with the effect of x, we vary $x \in [-1, 1]$. Because we do not know the sentiment parameters but also want to maintain some simplicity, we set $p + a = m + n = 1$ and vary $p, m \in [0, 1]$.

We describe the transient dynamics of this system using three measurements: the number of epidemic peaks, the maximum disease proportion, and the total infection load (defined as the integral of the disease proportion).

Fig. 5 displays the number of observed epidemic peaks versus the model parameters. There are a small number of cases where there are multiple epidemic peaks; these cases occur in general for lower or negative association x, longer waiting time to vaccination v^{-1}, sufficiently ineffective masking (large $z_S z_I$), strong anti-vaccine sentiment transmission (negative Δ_V), and strong anti-mask transmission (negative Δ_M). For $z_S z_I$, the critical value that separates the two phases is $\approx R_0^{-1}$, corresponding to the disease endemism stability condition in Table 4 as well as a necessary condition for having no epidemic peaks (see the next section). All of these situations can be summarized as those that make it easier for the disease to spread. The initial condition for this section has almost everyone at pro-mask and pro-vaccine, so a negative x would be able to "pull apart" these pairs and make it easier for anti-sentiments to spread.

We divide the remainder of this analysis among the cases where there are no epidemic peaks and where there is one epidemic peak.

3.5. No epidemic peaks

When there is no epidemic peak, the infected proportion starts at the initial proportion of 0.001 and then monotonically decreases. Thus, the maximum infection proportion is 0.001. The total infection load thus depends on how quickly the infected proportion decreases.

Fig. 6 shows that the total infection load (the area under the infection curve) increases in any situation where you would expect the disease to have an easier time transmitting: stronger anti-vaccine sentiment, stronger anti-mask sentiment, less effective masking, and longer waiting times to vaccination. In addition, the effect of the association parameter is complex, with significant infection loads occurring for intermediate positive and all negative associations. The same reasoning that allows negative x to lead to more epidemic peaks applies here to allow for a greater infection load; a negative association uncouples the two pro-sentiments that initially dominate the population.

A necessary (but not sufficient) condition for there to be no peaks is for $\frac{dI}{dt} < 0$ at $t = 0$ (the condition would be sufficient if it is true for all t). In general, if we assume that there is one anti-mask and one infected individual in a population of size n, this condition is equivalent to

$$\frac{z_I}{n} + z_S z_I \left(1 - \frac{2}{n}\right) < \frac{1}{R_0}, \tag{7}$$

which, using our $R_0 = 15.29$, for large n yields $z_S z_I < 0.065$ (which is the critical value in Fig. 5C). If z_S and z_I were both less than about 0.25, this condition would be satisfied. In general, for a new disease that is approximately as infectious as measles, where sentiment is almost entirely pro-mask, mask effectiveness has to be at least moderate in both directions to prevent an epidemic.

3.6. Two epidemic peaks

Here we observe an example situation where the presence of a positive association x results in two epidemic peaks. Using the same initial conditions as previously, and the following parameters: $b = 0$, $p = 0.1$, $m = 0.12$, $v = 0.25$, $a = 0.9$, $n = 0.88$, $z_S = 0.6$, and $z_I = 0.5$, we obtain a situation where $x = 0$ leads to a single epidemic peak, but $x = 0.2$ leads to two epidemic peaks

The infection dynamics are broken down in Fig. 7. In both Fig. 7A ($x = 0$) and Fig. 7B ($x = 0.2$), the infected proportion is initially driven by the spread of IPM individuals (as those were the only infected individuals at the start). Then, at some point, anti-vaccine and anti-mask sentiment takes over the population (Fig. 7C), and then after a brief transition through the IAM and IPN states, the IAN state takes over and leads to the epidemic in Fig. 7A and the second epidemic in Fig. 7B. The effect of the positive association $x = 0.2$ is in this case to delay the spread of the anti-sentiments slightly (Fig. 7C). Because we start with almost everyone pro-vaccine and pro-mask, and no individuals anti-vaccine and anti-mask, positive association between the traits actually takes longer for the two anti-traits to "find" each other than if they were independent. This delay is enough to allow the initial pro-sentiment epidemic to settle down before the anti-sentiment epidemic starts up. Note that the positive association epidemics are much smaller than the independent epidemic in this case; the delay of the sentiment spread also mitigates the overall impact of the epidemic.

3.7. One epidemic peak

Fig. 8 displays the relationship between the maximum infection load and the model parameters. Strong, expected effects include those of negative association (Fig. 8A); because the initial condition has the two anti-sentiments separate, negative association can act like genetic recombination and bring them together, allowing faster transmission of disease in those individuals, strong anti-vaccine transmission (Fig. 8B), strong anti-mask transmission (Fig. 8C), and longer vaccination waiting times (Fig. 8F). Mask effectiveness (Fig. 8DE) leads to the highest peaks when it is intermediate. The nonmonotonic effect of mask effectiveness is further explored in Fig. 9.

In Fig. 9, four scenarios are considered. With a parameter set of $b = 0$, $r = 2.13$, $g = v = 0.14$, $m = 0.4$, $n = 0.8$, $p = 0.2$, and $a = 0.8$

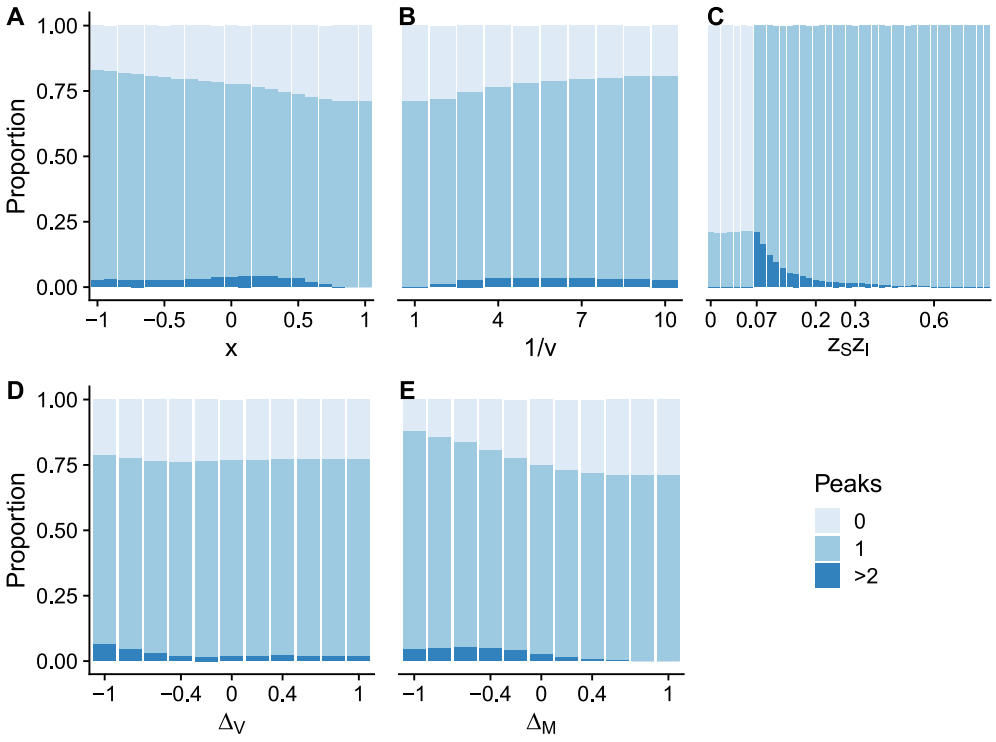

Fig. 5. The number of observed epidemic peaks depends on the values of the model parameters. (A) As the association x increases, fewer peaks occur. (B) As the waiting time to vaccination v^{-1} decreases, fewer peaks occur. (C) If $z_S z_I < R_0^{-1}$, there is either one peak or no peaks. Multiple peaks only occur when $z_S z_I > R_0^{-1}$, which is the condition for endemic disease when anti-vaccine, pro-mask sentiment dominates. (D) Multiple peaks are more likely to occur when pro-vaccine transmission is weaker than anti-vaccine transmission. (E) Multiple peaks are more likely to occur when pro-mask transmission is weaker than anti-mask transmission.

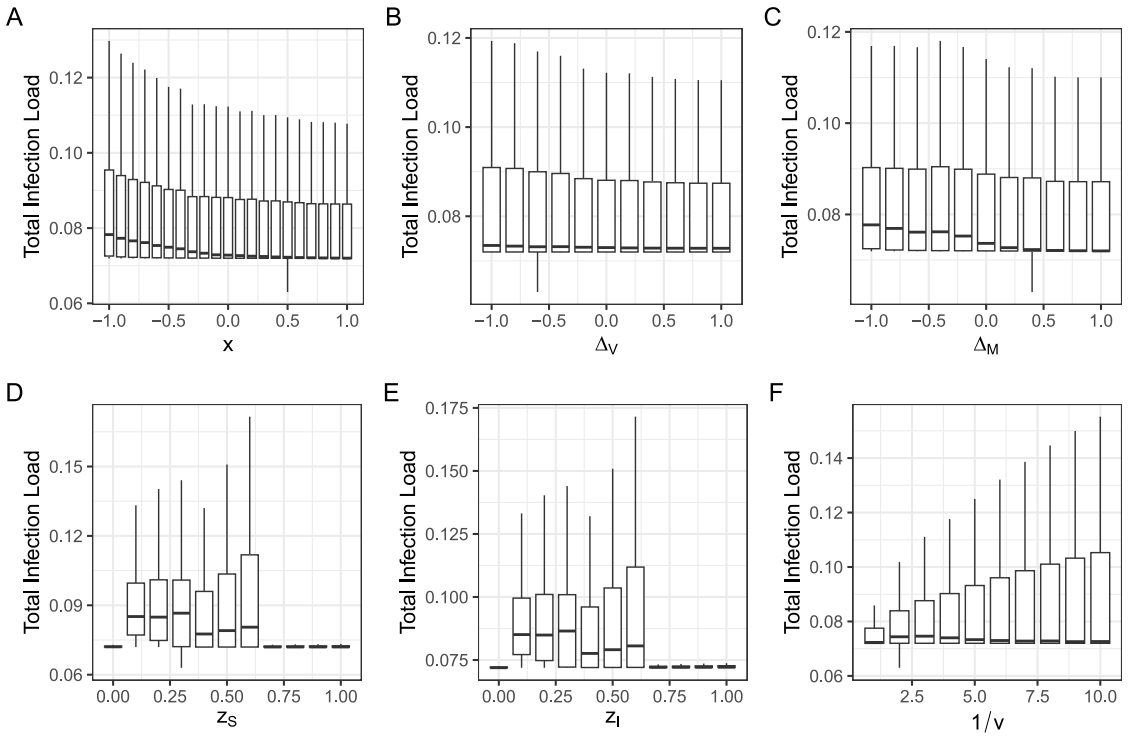

Fig. 6. Total infection load (the area under the infection curve) increases with (A) decreasing/negative association, (B) stronger anti-vaccine transmission, (C) stronger anti-mask transmission, (D) poor/intermediate susceptible mask effectiveness, (E) poor/intermediate infected mask effectiveness, and (F) intermediate/longer waiting time to vaccination. The main effect of the masking transmission rate modifiers (D and E) is that if the masks are too ineffective, we leave this regime and get epidemic peaks. Outliers have been removed from the boxplots.

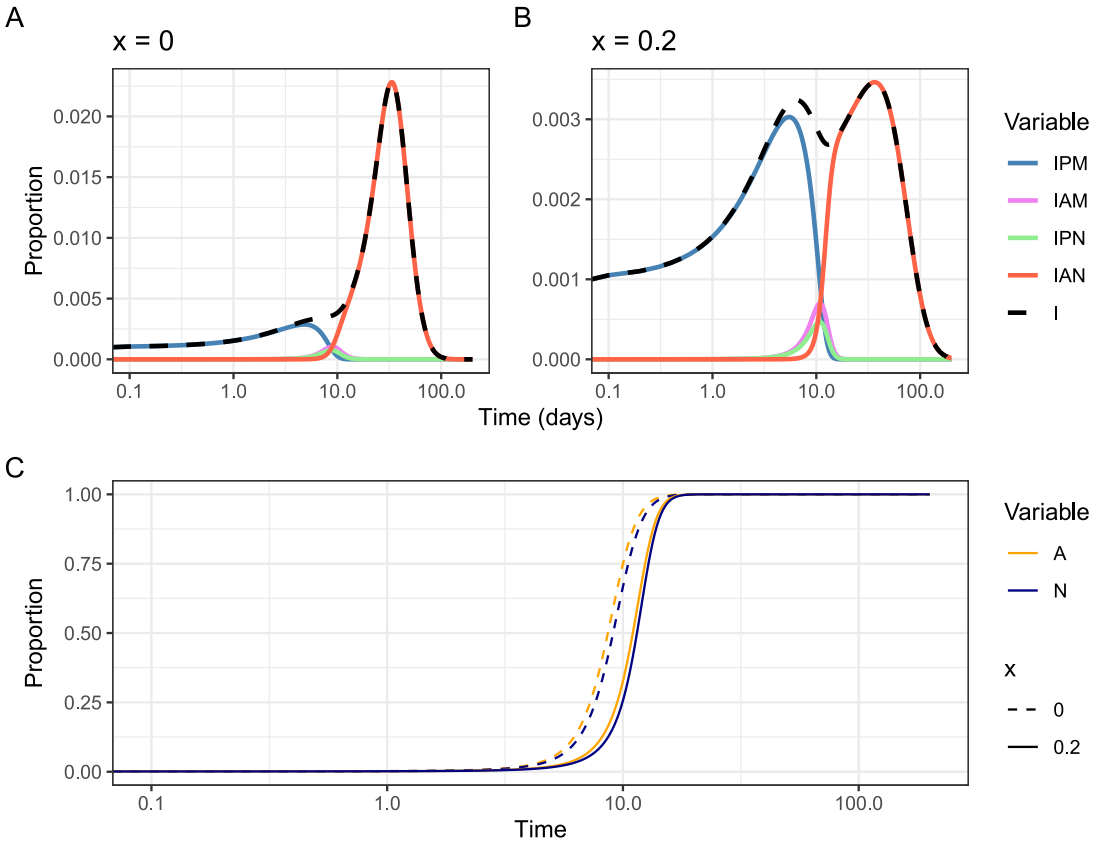

Fig. 7. Association between sentiments can lead to multiple epidemic peaks. (A) Infection dynamics when there is no association ($x = 0$). (B) Infection dynamics when there is an association ($x = 0.2$). (C) Sentiment dynamics in both cases. The positive association $x = 0.2$ delays the spread of anti-sentiments (because the initial condition had the sentiments separate), which then delays the spread of AN individuals and delays the IAN-based epidemic until after the initial IPM-based epidemic subsides in (B).

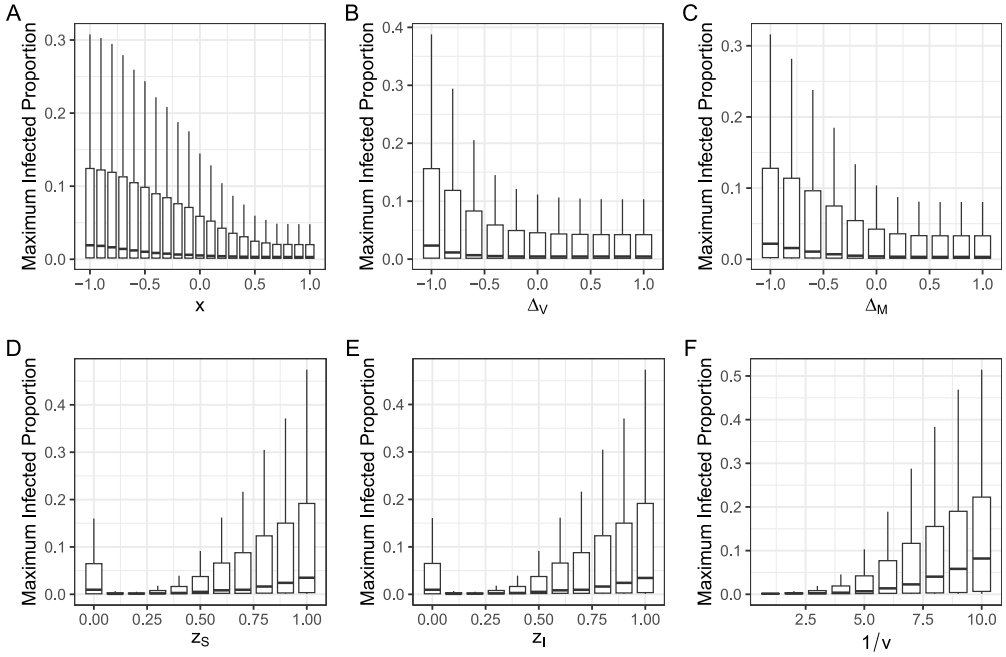

Fig. 8. Model parameters can affect the height of the epidemic peak. The height is larger when (A) there is a stronger negative association, (B) anti-vaccine sentiment transmission is stronger, (C) anti-mask sentiment transmission is stronger, (D) susceptible mask effectiveness is either strong or weak, (E) infected mask effectiveness is either strong or weak, and (F) the waiting time to vaccination is longer. The effects of the two mask effectiveness parameters are nearly identical in this case.

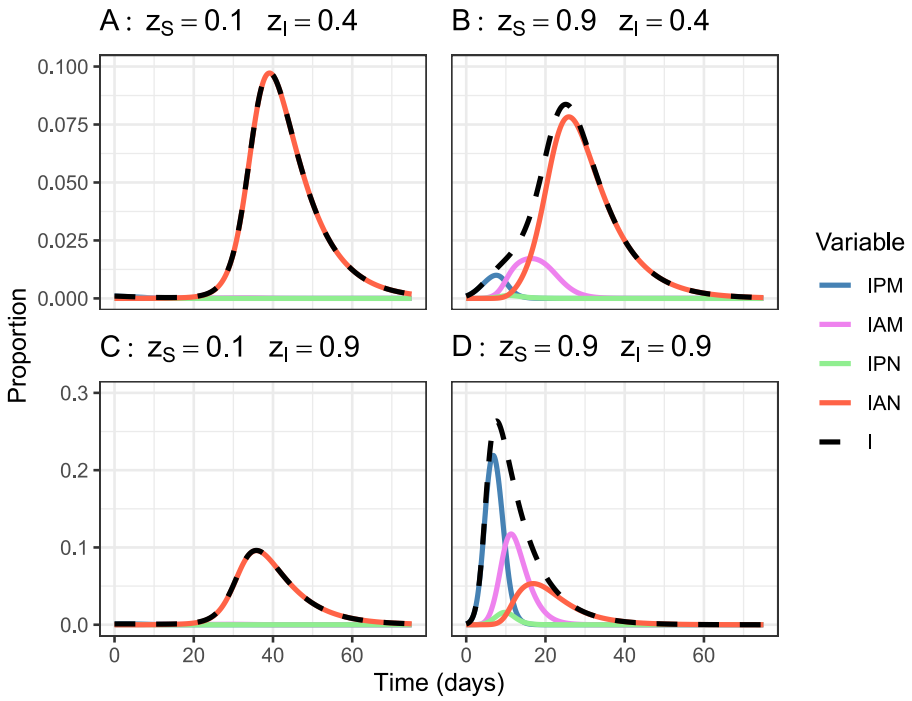

Fig. 9. Decreasing mask effectiveness can either increase or decrease the intensity of an epidemic. (A and B) In the context of moderate infected mask effectiveness, decreasing susceptible mask effectiveness actually decreases the epidemic peak. With a highly effective susceptible mask (A), the epidemic consists entirely of IAN individuals (aka unmasked). With a highly ineffective susceptible mask (B), the infection spreads through pro-mask individuals, but because infected mask effectiveness is sufficient, this slows down the epidemic. (C and D) In the context of poor transmission effectiveness, decreasing susceptible mask effectiveness increases the epidemic peak. With a highly effective susceptible mask (C), the epidemic again consists entirely of IAN individuals. With a highly ineffective susceptible mask (D), the infection spreads primarily through pro-mask individuals, and the masks are both poor enough that the epidemic is more severe. Other parameters used here include $b = 0$, $x = -0.1$, $p = 0.2$, $a = 0.8$, $m = 0.4$, $n = 0.8$, $r = 2.13$, and $g = v = 0.14$. Both anti-sentiments transmit stronger than the pro-sentiments in this example.

(so both anti-sentiments transmit better than their corresponding pro-sentiments, and the disease behaves like measles), we use four different types of masks: a mask that is good for the susceptible individual and moderate for the infected individual ($z_S = 0.1$, $z_I = 0.4$), a mask that is bad for the susceptible individual and moderate for the infected individual ($z_S = 0.9$, $z_I = 0.4$), a mask that is good for the susceptible individual and bad for the infected individual ($z_S = 0.1$, $z_I = 0.9$), and a mask that is bad for both the susceptible individual and the infected individual ($z_S = 0.9$, $z_I = 0.9$).

It might be expected that decreasing mask effectiveness will increase the intensity of an epidemic, but this is not always the case. In Figs. 9AB, we see that decreasing mask effectiveness for the susceptible individual actually decreases the epidemic peak. In this case, because masks are effective at preventing transmission, infecting masked individuals slows the disease down. Masked individuals can get infected because their masks do not prevent themselves from getting infected, but the masks still prevent them from passing the disease on, so instead of spreading through a predominantly AN population, the disease encounters transmission-limiting pathways in the AM and PM population.

By contrast, if masks are poor at preventing transmission (Figs. 9CD), then spreading through the masked population does not hinder the disease very much, and a poor mask for the susceptible individual will increase the spread of the disease as compared to a good one.

4. Discussion

We have developed a model of two cultural pathogens: one for mask-wearing sentiment, and one for vaccine sentiment, that affect the transmission of a disease. Our focus was to see how a tendency to associate one cultural pathogen with another affects the disease dynamics. We modeled this tendency using a parameter that affected the rate of transmission of a social contagion based on the current sentiment state of the receiver. A positive value of this parameter indicated that there was an association between "like" sentiments (e.g. pro-vaccine people are more receptive to pro-mask sentiment, and anti-vaccine people are more receptive to anti-mask sentiment), and a negative value of this parameter indicated that there was an association between "unlike" sentiments (e.g. pro-vaccine people are more receptive to anti-mask sentiment, and anti-vaccine people are more receptive to pro-mask sentiment).

Negative values of this parameter may seem unlikely to exist in reality, but empirical and published anecdotal evidence suggests that negative associations may arise, at least locally or over a short period of time. For instance, people may be anti-vaccine because they are worried about the vaccine, but are still concerned about the disease itself, and therefore would be more likely to adopt other behaviors that would prevent them from getting the disease, such as masking. Likewise, people who are vaccinated may be less likely to want to wear a mask as well, as masks are uncomfortable and the protection from the vaccine may be judged to be sufficient. For example, Floyd et al. (2022) found that mask-wearing decreased once the COVID-19 vaccine was broadly available in the USA, even among individuals who were not vaccine-hesitant. In addition, in their study of social media sentiment, Martin and Vanderslott (2022) found examples of pro-mask, anti-vaccine Tweets (e.g. "I trust the mask but not the vaccine.") and evidence of pro-vaccine, anti-mask behavior (e.g. "Young, healthy Senator, who spoke at rallies packed w/thousands w/o masks, who supports Trump -who's down-played COVID & mocked those who wear masks, is 1st to get vaccine while most medical workers, elderly & infirm Americans, wait. Congratulations on ur privilege, @user"). Furthermore, not all anti-science skepticism comes from the same source or has the same basis (Rutjens et al., 2018), and so allowing the potential

for decoupling or even negative coupling of multiple sentiments better matches reality than assuming a positive coupling.

We found that such a tendency can have a dramatic effect on the outcome of disease spread, to the extent that it can play a determining role in the outcome of the spread of a disease. In the long term, for any particular social contagions, there exist critical values of the association such that any outcome of social contagion transmission can be reached. Some of these values can be quite extreme and require a very strong association between the two traits, but in principle they exist. Thus, not only can the dynamics of the social contagions themselves determine the outcome of disease spread, but the association between the social contagions can as well.

We also found a wide variety of short-term effects of the joint spread of associated vaccine and mask sentiments. The spread of these sentiments can affect how quickly a disease dies out without an epidemic, the overall strength of a single-peaked epidemic, and the possibility of multiple epidemic peaks. In addition, the association between the two sentiments can have complex effects on the spread of the sentiments and the spread of disease. If the association is strong enough, sentiments that normally would not spread in isolation can still spread in the presence of a more favorably-spreading associated sentiment, leading to changes in the existence, strength, and timing of epidemics.

Previous modeling studies have also observed multiple epidemic waves as a consequence of intrinsic human behavior (Jo et al., 2026). Examples include models of behavior change with decaying memory (Harris et al., 2023; Ochab et al., 2023) or a time delay (d'Onofrio and Manfredi, 2009; Bhattacharyya and Konar, 2021; Mahmud et al., 2025), as well as "injection"-style effects where individuals self-isolate and then return to the population based on disease prevalence (Epstein et al., 2008; Valle et al., 2012; Epstein et al., 2021) and other forms of behavioral reversion (Martcheva et al., 2021; Nguyen et al., 2025). The general pattern for these cases is that multiple waves are triggered by some aspect of the system changing over time or a time delay in some effect. In our case, this time-delayed effect is the "background" spread of sentiment dynamics; multiple epidemics can be triggered by anti-sentiments spreading in the population and making the situation worse after an initial epidemic has already burned out. This particular mechanism is similar to that observed in the related model of Mehta and Rosenberg (2020) as well as a fear-contagion model in Perra et al. (2011).

The association parameter is used here as a way to capture how individuals adopt ideas when they already hold other ideas. These individuals can be thought of as integrating a new idea into their existing framework. This idea has a long history in the social sciences, starting with Blau and Schwartz (1984)'s concept of "consolidation". The matching nature of existing and new ideas is related to Heider's Balance Theory (Heider, 1946) and cognitive dissonance theory (Harmon-Jones and Mills, 2019). The underlying assumption in this model is that regardless of whether the association is positive or negative, when adopting the new trait, the individual is trying to reconcile their opinions with some sort of internal model (which may naturally lead to either a positive or negative association). Connecting this type of dynamic with existing psychological and sociological models could lead to a deeper understanding of how humans develop health-related beliefs and how those beliefs affect human health.

The association parameter can be considered in the context of a "parasitic contagion" defined by Hébert-Dufresne et al. (2020), in which a contagion can increase its own spread by using another contagion. Depending on the sign of the association, different pairs of contagions in this model can be considered as having a parasitic relationship. This framework is useful for classifying social contagions that may behave in similar ways despite affecting different behaviors.

Humans vary in their individual behavior. In particular, humans may vary in their tendency to accept certain sentiments when they possess others. Thus, the association parameter x could be an individual quantity as opposed to a population average. Future work could allow x to vary randomly among the population; this variation when combined with population structure may lead to different quantitative results. However, we hypothesize that similar qualitative effects of the association parameter may persist in the context of heterogeneous behavior due to the mean-field nature of SIR models. Another potential extension would include time-varying association (perhaps tied to disease prevalence); in those cases, it is important to know what association does when fixed so that time-varying situations can be properly interpreted.

Individuals can change their behavior based on factors other than social contagion. For instance, the Health Belief Model used by Ryan et al. (2024) is an example of a behavioral-sciences-driven model formulation of individual decision-making that takes into account multiple sources of information and risk-reward assessment. Using this or similar frameworks could help pinpoint exactly in which way associations between sentiments can occur and be most impactful.

The simplest SIR models, with and without vital dynamics, are unobservable and unidentifiable in general (unless certain information is known) (Cunniffe et al., 2024). This suggests the existence of similar identifiability problems in this model, which is a more complicated SIR model with more parameters that are difficult to measure. The practical effort of estimating model parameters from data is therefore challenging. The approach we would recommend is to measure individual parameters from specific observations of the particular dynamic (e.g. measure sentiment transmission parameters from experiments) and then use this model as a forward-projection model as opposed to an inference model. The association parameter x can be inferred from a contingency table of joint sentiment observations, where deviation from independence implies association. Potential data sources for these sentiment transmission parameters would include sentiment-analyzed social media posts or message-passing experiments.

This model is relatively high-dimensional. There are 12 compartments, and computing the eigenvalues of the full Jacobian matrix is analytically intractable. Future research that models multiple behaviors at once in conjunction with a disease may be better served by agent-based models, where less bookkeeping is required. Another potential source for future complexity with regard to this model is the simple treatment of the effect of mask-wearing on disease transmission. It is well-known that mask-wearing has nuanced effects on disease transmission (Brienen et al., 2010). Our simplified choice was made purely for modeling tractability and interpretability; there is certainly room for future work that uses more realistic modeling of disease transmission with masking.

Incorporating human behavior into epidemiological modeling is both difficult and necessary. This study shows that when multiple health behaviors interact, the nature and strength of the interaction itself can have a strong effect on the outcome of disease spread. As we continue to learn how human behavior in all of its myriad facets affects disease dynamics, we must pay attention to the interactions between those behaviors themselves.

Funding

This work was supported by a 2024 Summer Faculty Research Grant from Elmhurst University.

Acknowledgments

We thank Noah Rosenberg, Daniel Weissman, and two anonymous reviewers for helpful comments on the manuscript.

Appendix

To simplify the analysis of this system, we can study the equilibria and stability conditions of the disease and sentiment subsystems obtained by tracking marginal variables.

A.1. Disease subsystem equilibria

If we track the disease states S and I (noting that $R = 1 - S - I$), we obtain

$$\frac{dS}{dt} = b[1-S] - r\left[SN + z_S(SM)\right]\left[IN + z_I(IM)\right] - vSP$$
$$\frac{dI}{dt} = r\left[SN + z_S(SM)\right]\left[IN + z_I(IM)\right] - (g+b)I$$

Let the dummy variables $k = \frac{SP}{S}$, $p = \frac{SM}{S}$, and $q = \frac{IM}{I}$:

$$\frac{dS}{dt} = b - rSI\left[(1-p) + z_S p\right]\left[(1-q) + z_I q\right] - (vk+b)S$$
$$\frac{dI}{dt} = rSI\left[(1-p) + z_S p\right]\left[(1-q) + z_I q\right] - (g+b)I.$$

As with the standard SIR model, there are two choices for $\widehat{I}$. First, $\widehat{I} = 0$:

$$\frac{dS}{dt} = b - (vk+b)S$$
$$\widehat{S} = \frac{b}{vk+b}.$$

Second, $\widehat{I} > 0$. First, we solve for $\widehat{S}$:

$$0 = r\widehat{S}\left[(1-p) + z_S p\right]\left[(1-q) + z_I q\right] - (g+b)$$
$$\widehat{S} = \frac{g+b}{r}\frac{1}{\left[(1-p) + z_S p\right]\left[(1-q) + z_I q\right]}$$
$$= \frac{1}{R_0}\frac{1}{\left[1-(1-z_S)p\right]\left[1-(1-z_I)q\right]}.$$

Then, we solve for $\widehat{I}$:

$$0 = b - r\widehat{S}\widehat{I}\left[(1-p) + z_S p\right]\left[(1-q) + z_I q\right] - (vk+b)\widehat{S}$$
$$r\widehat{S}\widehat{I}\left[(1-p) + z_S p\right]\left[(1-q) + z_I q\right] = b - (vk+b)\widehat{S}$$
$$\widehat{I} = \frac{b}{r\widehat{S}\left[(1-p) + z_S p\right]\left[(1-q) + z_I q\right]} - \frac{vk+b}{r\left[(1-p) + z_S p\right]\left[(1-q) + z_I q\right]}$$
$$= \frac{b}{g+b} - \frac{vk+b}{r\left[1-(1-z_S)p\right]\left[1-(1-z_I)q\right]}.$$

We still need to know the values of k, p, and q at equilibrium, which require knowing the sentiment equilibria.

A.2. Sentiment subsystem equilibria

If we track the sentiment marginal variables PN, PM, and AN (noting that $AM = 1 - PN - PM - AN$), we obtain

$$\frac{dPN}{dt} = (1-x)pP(AN) + (1-x)nN(PM) - PN\left[(1+x)\left[a(1-P) + m(1-N)\right]\right]$$
$$\frac{dPM}{dt} = (1+x)pP(1-P-AN) + (1+x)m(1-N)PN - PM\left[(1-x)\left[a(1-P) + nN\right]\right]$$
$$\frac{dAN}{dt} = (1+x)a(1-P)PN + (1+x)nN(1-P-AN) - AN\left[(1-x)\left[pP + m(1-N)\right]\right].$$

From this system of equations, it is not obvious what the equilibrium values of these variables are. Verifying that all the boundaries where either one or none of the three variables is 1, and the rest are zero, are indeed equilibria, requires only inspection. We label these equilibria by $(\widehat{PN}, \widehat{PM}, \widehat{AN})$.

Because the Jacobian of this system is very wide, we display it by element:

$$J_{11} = (1-x)\left[n(PM) + p(AN)\right] - (1+x)\left[a(1-PM) + m(1-AN) - 2(PN)(a+m)\right]$$
$$J_{12} = (1-x)\left[nN + p(AN)\right] + (1+x)a(PN)$$
$$J_{13} = (1-x)\left[n(PM) + pP\right] + (1+x)m(PN)$$
$$J_{21} = (1+x)\left[(p+m)(1-AN-2(PN)) - 2p(PM)\right] - (1-x)(n-a)(PM)$$
$$J_{22} = (1+x)p\left[1-2P-AN\right] - (1-x)\left[a(1-PN-2(PM)) + nN\right]$$
$$J_{23} = -(1+x)\left[(p+m)P + p(PM)\right] - (1-x)n(PM)$$
$$J_{31} = (1+x)\left[(a+n)(1-PM-2(PN)) - n(AN)\right] - (1-x)(p-m)(AN)$$
$$J_{32} = -(1+x)\left[(a+n)(PN) + n(AN)\right] - (1-x)p(AN)$$
$$J_{33} = (1+x)n(1-PM-2(PN)-2(AN)) - (1-x)\left[pP + m(1-PN) - 2m(AN)\right] \tag{8}$$

The eigenvalues of the equilibrium $(1,0,0)$ (which corresponds to $\widehat{p} = 1$ and $\widehat{k} = 0$) are:

$$\lambda_1 = (m+n)x + m - n$$
$$\lambda_2 = -(1+x)(p+n)$$
$$\lambda_3 = (p+a)x + a - p.$$

This equilibrium is stable when:

$$x < \frac{n-m}{m+n} \tag{9}$$
$$x > -1 \tag{10}$$
$$x < \frac{p-a}{p+a}. \tag{11}$$

The eigenvalues of the equilibrium $(0,1,0)$ (which corresponds to $\widehat{p} = 1$ and $\widehat{k} = 1$) are:

$$\lambda_1 = -(m+n)x + n - m$$
$$\lambda_2 = -(1-x)(p+n)$$
$$\lambda_3 = -(a+p)x + a - p.$$

This equilibrium is stable when:

$$x > \frac{n-m}{m+n} \tag{12}$$
$$x < 1 \tag{13}$$
$$x < \frac{p-a}{p+a}. \tag{14}$$

The eigenvalues of the equilibrium $(0,0,1)$ (which corresponds to $\widehat{p} = 0$ and $\widehat{k} = 0$) are:

$$\lambda_1 = -(m+n)x + n - m$$
$$\lambda_2 = -(1-x)(c+n)$$
$$\lambda_3 = -(p+a)x - a + p.$$

This equilibrium is stable when:

$$x > \frac{m-n}{m+n} \tag{15}$$
$$x < 1 \tag{16}$$
$$x < \frac{a-p}{p+a}. \tag{17}$$

Finally, the eigenvalues of the equilibrium $(0,0,0)$ (which corresponds to $\widehat{p} = 0$ and $\widehat{k} = 1$) are:

$$\lambda_1 = (m+n)x - m + n$$
$$\lambda_2 = -(1+x)(c+m)$$
$$\lambda_3 = (a+p)x - a + p.$$

This equilibrium is stable when:

$$x < \frac{m-n}{m+n} \tag{18}$$

$$x > -1 \tag{19}$$

$$x < \frac{a-p}{p+a}. \tag{20}$$

There are non-corner equilibria, but they only exist when certain parameters are equal and are therefore non-generic. First, suppose $M = 1$. Then we have

$$\frac{dPM}{dt} = PM(1-PM)\left[p(1+x) - a(1-x)\right] = 0.$$

The only solution here where $PM \neq 0, 1$ requires $p(1+x) = a(1-x)$. Now suppose $N = 1$. Then we have

$$\frac{dPN}{dt} = PN(1-PN) - \left[p(1-x) - a(1+x)\right] = 0.$$

The only solution here where $PN \neq 0, 1$ requires $p(1-x) = a(1+x)$. Analogously, when $P = 0$ a non-corner equilibrium requires $(1+x)n = (1-x)m$, and when $A = 0$ it requires $n(1-x) = m(1+x)$.

If only $PM = 0$, then we have

$$\frac{dPM}{dt} = (PN)(1-N)\left[p(1+x) + m(1+x)\right] = 0,$$

which is impossible. Analogous arguments can be used to show that there cannot be exactly one variable equal to 0 and all others nonzero at equilibrium for the other three choices.

No nongeneric interior equilibrium exists (confirmed by numerical simulation sweeping over parameter combinations). Demonstrating this with algebra is at least very involved and may be intractable.

These stability conditions are essentially outcomes of duels between competing contagions for the same sentiment. They are mostly determined by which rate of spread (p vs a or m vs n is bigger, modulated by x).

A.3. Disease subsystem stability

Because we are only concerned with situations where $\hat{p} = \hat{q}$ and $\hat{k}$ are either 0 or 1, we can now finish our analysis of the disease subsystem.

The Jacobian of the disease subsystem is

$$\begin{bmatrix} rI\left[1-(1-z_S)p\right]\left[1-(1-z_I)q\right] - (\upsilon k + b) & -rS\left[1-(1-z_S)p\right]\left[1-(1-z_I)q\right] \\ rI\left[1-(1-z_S)p\right]\left[1-(1-z_I)q\right] & rS\left[1-(1-z_S)p\right]\left[1-(1-z_I)q\right] - (g+b). \end{bmatrix}$$

When $\hat{I} = 0$, the eigenvalues are

$$\lambda_1 = -(\upsilon k + b) < 0$$

$$\lambda_2 = -(g+b) + r\hat{S}\left[1-(1-z_S)p\right]\left[1-(1-z_I)q\right].$$

When $\hat{k} = \hat{p} = \hat{q} = 0$, $\hat{S} = 1$, and

$$\lambda_2 = -(g+b) + r,$$

so this equilibrium is stable when $R_0 < 1$.

When $\hat{k} = 0$ and $\hat{p} = \hat{q} = 1$, $\hat{S} = 1$, and

$$\lambda_2 = -(g+b) + rz_S z_I,$$

So this equilibrium is stable when $R_0 < \frac{1}{z_S z_I}$.

When $\hat{k} = 1$ and $\hat{p} = \hat{q} = 0$, $\hat{S} = \frac{b}{\upsilon+b}$, and

$$\lambda_2 = -(g+b) + r\frac{b}{\upsilon+b},$$

So this equilibrium is stable when $R_0 < \frac{\upsilon+b}{b}$.

When $\hat{k} = 1$ and $\hat{p} = \hat{q} = 1$, $\hat{S} = \frac{b}{\upsilon+b}$ and

$$\lambda_2 = -(g+b) + rz_S z_I \frac{b}{\upsilon+b},$$

So this equilibrium is stable when $R_0 < \frac{\upsilon+b}{bz_S z_I}$.

As demonstrated by Van den Driessche and Watmough (2002) for SIR systems with heterogeneous compartments (of with this system is an example, where S, I, and R compartments are subdivided into compartments with transitions between them), these R_0 conditions define the boundaries between stability of a DFE and an endemic equilibrium, so the reverse conditions are where the DFE is unstable and the corresponding endemic equilibrium is stable.

These stability conditions can be interpreted in the standard SIR manner, by thinking of the transmissability of the disease being affected by the disease parameters as well as masking and vaccination, all summarized in a threshold for R_0.

The disease equilibria here are structurally identical to a standard SIR model with vaccination, with the additional complication of mask-wearing behavior. Where this model adds to existing models is the ability to study how the spread of masking and vaccination behavior affects the dynamics of a disease that would otherwise spread by standard SIR dynamics.

A.4. Condition for no epidemic peaks

The condition for no epidemic peaks means that I has to monotonically decrease, so $\frac{dI}{dt} < 0$ for all t. In particular, with our initial conditions, we have

$$\frac{dI}{dt}|_{t=0} < 0$$

$$r\left[SN + z_S(SM)\right]\left[IN + z_I(IM)\right] < (g+b)I$$

$$r\left[\frac{1}{1000} + z_S\frac{98}{1000}\right]\left[z_I\frac{1}{1000}\right] < (g+b)\frac{1}{1000}$$

$$\left[1 + 98z_S\right]z_I < 1000\frac{1}{R_0}.$$

This condition is approximately $z_S z_I < 0.065$ for large population size.

A.5. Sensitivity analysis

We performed a sensitivity analysis following Marino et al. (2008), using Latin hypercube sampling to sample the parameter space (including initial conditions, which are normalized to sum to 1) and evaluating parameter effects using Partial Rank Correlation Coefficients (PRCC). We used 10,000 sample parameter sets. Table 5 displays the PRCCs for each parameter (with a dash if the result was not significant at an $\alpha = 0.05$ level, Bonferroni-corrected). In general, the directionality of effect is roughly as expected, with pro-vaccine p and pro-mask m transmission rates strongly positively correlated with P and M, respectively, and their corresponding anti-sentiments a and n strongly negatively correlated with P and M. Vaccination rate υ is negatively correlated with S, recovery rate g is strongly negatively correlated with I, and disease transmission rate r is positively correlated with I and negatively correlated with S (these are relevant in only a subset of cases where bistability is possible). The only initial conditions that are correlated with anything are the susceptible, antivaccine conditions which are mildly positively correlated with S. The parameters b, z_S, and z_I were not significantly correlated with any output; this makes sense for b because its effect is minimized due to the restriction of a constant population size, and for z_S and z_I because they only modify transmission rate, which is already only weakly correlated with output variables.

The interaction parameter x has strong positive correlations with any state that has the sentiment in the "same direction" (SAN, IPM, IAN, and RPM) except for SPM and RAN (which was not tested as it is not independent of the others). Regarding marginal variables, x is positively correlated with I and negatively correlated with the two pro-sentiments P and M.

Data availability

No empirical data were generated or analyzed for this study. Simulation code is available at https://github.com/rohansmehta/mask_vaccine_interaction_code.

Table 5
Partial Rank Correlation Coefficients (PRCCs) for each model parameter against each model variable. Dashes indicate values that were not significant at a Bonferroni-corrected $\alpha = 0.05$ level. No correlations were significant for b, z_S, z_I or any initial value other than SAM_0 and SAN_0.

Parameter	SPM	SPN	SAM	SAN	IPM	IPN	IAM	IAN	RPM	RPN	RAM	S	I	P	M
x	−0.07	–	−0.33	0.27	0.20	−0.04	−0.08	0.12	0.34	−0.29	−0.28	–	0.08	−0.07	−0.07
p	−0.04	−0.04	−0.44	−0.52	0.17	0.27	−0.18	−0.16	0.27	0.47	−0.26	−0.59	−0.11	0.56	−0.05
m	0.14	−0.18	0.29	−0.21	0.19	−0.16	0.29	−0.13	0.29	−0.28	0.45	0.04	−0.04	−0.04	0.59
v	−0.19	−0.23	−0.11	−0.13	–	0.05	–	–	–	0.04	0.05	−0.30	–	–	–
a	−0.27	−0.28	0.25	0.42	−0.34	−0.36	0.09	0.19	−0.43	−0.52	0.15	0.50	0.15	−0.57	–
n	−0.34		−0.40	0.16	−0.34	0.07	−0.39	0.14	−0.42	0.17	−0.52	–	0.05	–	−0.58
g	–	–	–	0.07	−0.72	−0.72	−0.73	−0.85	–	0.04	–	0.13	−0.97	–	–
r	–	–	–	−0.05	–	–	0.05	0.05	–	–	–	−0.09	0.09	–	–
SAM_0	–	–	–	–	–	–	–	–	–	–	–	0.06	–	–	–
SAN_0	–	–	–	–	–	–	–	–	–	–	–	0.06	–	–	–

References

Bauch, Chris T., Galvani, Alison P., 2013. Social factors in epidemiology. Science 342 (6154), 47–49.

Bhattacharyya, R., Konar, Partha, 2021. Modelling the influence of progressive social awareness, lockdown and anthropogenic migration on the dynamics of an epidemic. Int. J. Dyn. Control. 9 (2), 797–806.

Blau, Peter, Schwartz, Joseph, 1984. Crosscutting Social Circles: Testing a Macrostructural Theory of Intergroup Relations. Routledge.

Brienen, Nicole C.J., Timen, Aura, Wallinga, Jacco, Van Steenbergen, Jim E., Teunis, Peter F.M., 2010. The effect of mask use on the spread of influenza during a pandemic. Risk Anal. 30 (8), 1210–1218.

Brüne, Martin, Wilson, Daniel R., 2020. Evolutionary perspectives on human behavior during the coronavirus pandemic: Insights from game theory. Evol. Med. Pub. Health 2020 (1), 181–186.

Chen, Jiangzhuo, Espinoza, Baltazar, Chou, Jingyuan, Gumel, Abba B., Levin, Simon A., Marathe, Madhav, 2025. A simple model of coupled individual behavior and its impact on epidemic dynamics. Math. Biosci. 380, 109345.

Cunniffe, Nik, Hamelin, Frédéric, Iggidr, Abderrahman, Rapaport, Alain, Sallet, Gauthier, 2024. Identifiability and Observability in Epidemiological Models. Springer.

d'Onofrio, Alberto, Manfredi, Piero, 2009. Information-related changes in contact patterns may trigger oscillations in the endemic prevalence of infectious diseases. J. Theoret. Biol. 256 (3), 473–478.

Eikenberry, Steffen E., Mancuso, Marina, Iboi, Enahoro, Phan, Tin, Eikenberry, Keenan, Kuang, Yang, Kostelich, Eric, Gumel, Abba B., 2020. To mask or not to mask: Modeling the potential for face mask use by the general public to curtail the COVID-19 pandemic. Infect. Dis. Model. 5, 293–308.

Elbanna, Ahmed, Wong, George N., Weiner, Zach J., Wang, Tong, Zhang, Hantao, Liu, Zhiru, Tkachenko, Alexei, Maslov, Sergei, Goldenfeld, Nigel, 2020. Entry Screening and Multi-Layer Mitigation of COVID-19 Cases for a Safe University Reopening. Cold Spring Harbor Laboratory Press, pp. 1–13, MedRxiv.

Epstein, Joshua M., Hatna, Erez, Crodelle, Jennifer, 2021. Triple contagion: A two-fears epidemic model. J. R. Soc. Interface 18 (181), 20210186.

Epstein, Joshua M., Parker, Jon, Cummings, Derek, Hammond, Ross A., 2008. Coupled contagion dynamics of fear and disease: Mathematical and computational explorations. PLoS One 3 (12), e3955.

Floyd, Christopher J., Joachim, Grace E., Boulton, Matthew L., Zelner, Jon, Wagner, Abram L., 2022. COVID-19 vaccination and mask wearing behaviors in the United States, August 2020-June 2021. Expert. Rev. Vaccines 21 (10), 1487–1493.

Fu, Feng, Christakis, Nicholas A., Fowler, James H., 2017. Dueling biological and social contagions. Sci Rep. 7 (1), 43634.

Funk, Sebastian, Bansal, Shweta, Bauch, Chris T., Eames, Ken T.D., Edmunds, W. John, Galvani, Alison P., Klepac, Petra, 2015. Nine challenges in incorporating the dynamics of behaviour in infectious diseases models. Epidemics 10, 21–25.

Funk, Sebastian, Gilad, Erez, Watkins, Chris, Jansen, Vincent A.A., 2009. The spread of awareness and its impact on epidemic outbreaks. Proc. Natl. Acad. Sci. USA 106 (16), 6872–6877.

Funk, Sebastian, Salathé, Marcel, Jansen, Vincent A.A., 2010. Modelling the influence of human behaviour on the spread of infectious diseases: A review. J. R. Soc. Interface 7 (50), 1247–1256.

Gozzi, Nicolò, Perra, Nicola, Vespignani, Alessandro, 2025. Comparative evaluation of behavioral epidemic models using COVID-19 data. Proc. Natl. Acad. Sci. USA 122 (24), e2421993122.

Harmon-Jones, Eddie, Mills, Judson, 2019. An Introduction to Cognitive Dissonance Theory and an Overview of Current Perspectives on the Theory. American Psychological Association.

Harris, Mallory J., Cardenas, Kimberly J., Mordecai, Erin A., 2023. Social divisions and risk perception drive divergent epidemics and large later waves. Evol. Hum. Sci. 5, e8.

Hébert-Dufresne, Laurent, Mistry, Dina, Althouse, Benjamin M., 2020. Spread of infectious disease and social awareness as parasitic contagions on clustered networks. Phys. Rev. Res. 2 (3), 033306.

Heider, Fritz, 1946. Attitudes and cognitive organization. J. Psychol. 21 (1), 107–112.

Huang, Yunhan, Zhu, Quanyan, 2022. Game-theoretic frameworks for epidemic spreading and human decision-making: A review. Dyn. Games Appl. 12 (1), 7–48.

Iranzo, Valeriano, Pérez-González, Saúl, 2021. Epidemiological models and COVID-19: A comparative view. Hist. Philos. Life Sci. 43 (3), 104.

Jo, Youngji, Sharbayta, Sileshi Sintayehu, Buonomo, Bruno, 2026. Behavioral change models for infectious disease transmission: A systematic review (2020–2025). arXiv preprint arXiv:2602.16633.

Kabir, K.M., Tanimoto, Jun, 2020. Evolutionary game theory modelling to represent the behavioural dynamics of economic shutdowns and shield immunity in the COVID-19 pandemic. R. Soc. Open Sci. 7 (9).

Karashiali, Christiana, Konstantinou, Pinelopi, Christodoulou, Andria, Kyprianidou, Maria, Nicolaou, Christiana, Karekla, Maria, Middleton, Nicos, Kassianos, Angelos P., 2023. A qualitative study exploring the social contagion of attitudes and uptake of COVID-19 vaccinations. Hum. Vaccines Immunother. 19 (2), 2260038.

Kermack, William Ogilvy, McKendrick, Anderson G., 1927. A contribution to the mathematical theory of epidemics. Proc. R. Soc. Lond. A 115 (772), 700–721.

Konstantinou, Pinelopi, Georgiou, Katerina, Kumar, Navin, Kyprianidou, Maria, Nicolaides, Christos, Karekla, Maria, Kassianos, Angelos P., 2021. Transmission of vaccination attitudes and uptake based on social contagion theory: A scoping review. Vaccines 9 (6), 607.

Mahmud, Md Shahriar, Eshun, Solomon, Espinoza, Baltazar, Kadelka, Claus, 2025. Adaptive human behavior and delays in information availability autonomously modulate epidemic waves. PNAS Nexus 4 (5), pgaf145.

Manfredi, Piero, D'Onofrio, Alberto, 2013. Modeling the Interplay Between Human Behavior and the Spread of Infectious Diseases. Springer Science & Business Media.

Marino, Simeone, Hogue, Ian B., Ray, Christian J., Kirschner, Denise E., 2008. A methodology for performing global uncertainty and sensitivity analysis in systems biology. J. Theoret. Biol. 254 (1), 178–196.

Martcheva, Maia, Tuncer, Necibe, Ngonghala, Calistus N., 2021. Effects of social-distancing on infectious disease dynamics: An evolutionary game theory and economic perspective. J. Biol. Dyn. 15 (1), 342–366.

Martin, Sam, Vanderslott, Samantha, 2022. "Any idea how fast 'It's just a mask!' can turn into 'It's just a vaccine!'": From mask mandates to vaccine mandates during the COVID-19 pandemic. Vaccine 40 (51), 7488–7499.

Mehta, Rohan S., Rosenberg, Noah A., 2020. Modelling anti-vaccine sentiment as a cultural pathogen. Evol. Hum. Sci. 2, e21.

Nagurney, Anna, 2021. Game theory and the COVID-19 pandemic. In: Tutorials in Operations Research: Emerging Optimization Methods and Modeling Techniques with Applications. INFORMS, pp. 83–130.

Nelson, Marie Clark, Rogers, John, 1992. The right to die? Anti-vaccination activity and the 1874 smallpox epidemic in Stockholm. Soc. Hist. Med. 5 (3), 369–388.

Nguyen, Maximilian M., Freedman, Ari S., Cheung, Matthew A., Saad-Roy, Chadi M., Espinoza, Baltazar, Grenfell, Bryan T., Levin, Simon A., 2025. The complex interplay between risk tolerance and the spread of infectious diseases. J. R. Soc. Interface 22 (225).

Ochab, Magdalena, Manfredi, Piero, Puszynski, Krzysztof, d'Onofrio, Alberto, 2023. Multiple epidemic waves as the outcome of stochastic SIR epidemics with behavioral responses: A hybrid modeling approach. Nonlinear Dynam. 111 (1), 887–926.

Patel, Krishane, 2026. Risk compensation: How vaccination impacts social distancing in an online natural experiment. Risk Anal. 46 (3), e70201.

Perra, Nicola, 2021. Non-pharmaceutical interventions during the COVID-19 pandemic: A review. Phys. Rep. 913, 1–52.

Perra, Nicola, Balcan, Duygu, Gonçalves, Bruno, Vespignani, Alessandro, 2011. Towards a characterization of behavior-disease models. PLoS One 6 (8), e23084.

Proverbio, Daniele, Tessarin, Riccardo, Giordano, Giulia, 2024. Data informed epidemiological-behavioural modelling. In: GIMC-SIMAI Workshop for Young Scientists. Springer, pp. 279–289.

Proverbio, Daniele, Tessarin, Riccardo, Giordano, Giulia, 2025. Recent trends in socio-epidemic modelling: Behaviours and their determinants. Boll. Unione Mat. Ital. 1–25.

Qiu, Zirou, Espinoza, Baltazar, Vasconcelos, Vitor V., Chen, Chen, Constantino, Sara M., Crabtree, Stefani A., Yang, Luojun, Vullikanti, Anil, Chen, Jiangzhuo, Weibull, Jörgen, et al., 2022. Understanding the coevolution of mask wearing and epidemics: A network perspective. Proc. Natl. Acad. Sci. USA 119 (26), e2123355119.

Ranoa, Diana Rose E., Holland, Robin L., Alnaji, Fadi G., Green, Kelsie J., Wang, Leyi, Fredrickson, Richard L., Wang, Tong, Wong, George N., Uelmen, Johnny, Maslov, Sergei, et al., 2022. Mitigation of SARS-CoV-2 transmission at a large public university. Nat. Commun. 13 (1), 3207.

Rosal, Milagros C., Bodenlos, Jamie S., 2009. Culture and health-related behavior. In: Shumaker, Sally A., Ockene, Judith K., Riekert, Kristin A. (Eds.), The Handbook of Health Behavior Change. Springer New York, NY.

Rutjens, Bastiaan T., Sutton, Robbie M., van der Lee, Romy, 2018. Not all skepticism is equal: Exploring the ideological antecedents of science acceptance and rejection. Pers. Soc. Psychol. Bull. 44 (3), 384–405.

Ryan, Matthew, Brindal, Emily, Roberts, Mick, Hickson, Roslyn I., 2024. A behaviour and disease transmission model: Incorporating the Health Belief Model for human behaviour into a simple transmission model. J. R. Soc. Interface 21 (215).

Salathé, Marcel, Bonhoeffer, Sebastian, 2008. The effect of opinion clustering on disease outbreaks. J. R. Soc. Interface 5 (29), 1505–1508.

Sediqin, Mohammadreza, 2021. Semantic Analysis of Vaccine and Mask Sentiments in COVID-19 Twitter Data. The University of Wisconsin-Milwaukee.

Tanaka, Mark M., Kumm, Jochen, Feldman, Marcus W., 2002. Coevolution of pathogens and cultural practices: A new look at behavioral heterogeneity in epidemics. Theor. Popul. Biol. 62 (2), 111–119.

Teslya, Alexandra, Nunner, Hendrik, Buskens, Vincent, Kretzschmar, Mirjam E., 2022. The effect of competition between health opinions on epidemic dynamics. PNAS Nexus 1 (5), pgac260.

Tracht, Samantha M., Del Valle, Sara Y., Hyman, James M., 2010. Mathematical modeling of the effectiveness of facemasks in reducing the spread of novel influenza A (H1N1). PLoS One 5 (2), e9018.

Valle, Sara Y. Del, Mniszewski, Susan M., Hyman, James M., 2012. Modeling the impact of behavior changes on the spread of pandemic influenza. In: Modeling the Interplay Between Human Behavior and the Spread of Infectious Diseases. Springer, pp. 59–77.

Van den Driessche, Pauline, Watmough, James, 2002. Reproduction numbers and subthreshold endemic equilibria for compartmental models of disease transmission. Math. Biosci. 180 (1–2), 29–48.

Verelst, Frederik, Willem, Lander, Beutels, Philippe, 2016. Behavioural change models for infectious disease transmission: a systematic review (2010–2015). J. R. Soc. Interface 13 (125), 20160820.

Wang, Zhen, Andrews, Michael A., Wu, Zhi-Xi, Wang, Lin, Bauch, Chris T., 2015. Coupled disease–behavior dynamics on complex networks: A review. Phys. Life Rev. 15, 1–29.

Wang, Zhen, Bauch, Chris T., Bhattacharyya, Samit, d'Onofrio, Alberto, Manfredi, Piero, Perc, Matjaž, Perra, Nicola, Salathé, Marcel, Zhao, Dawei, 2016. Statistical physics of vaccination. Phys. Rep. 664, 1–113.

Weston, Dale, Hauck, Katharina, Amlôt, Richard, 2018. Infection prevention behaviour and infectious disease modelling: a review of the literature and recommendations for the future. BMC Pub Health 18, 1–16.