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# Homo-psychologicus: Reactionary behavioural aspects of epidemics

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ABSTRACT

We formulate an *in silico* model of pathogen avoidance mechanism and investigate its impact on defensive behavioural measures (e.g., spontaneous social exclusions and distancing, crowd avoidance and voluntary vaccination adaptation). In particular, we use SIR(B)S (e.g., susceptible-infected-recovered with additional behavioural component) model to investigate the impact of *homo-psychologicus* aspects of epidemics. We focus on reactionary behavioural changes, which apply to both social distancing and voluntary vaccination participations. Our analyses reveal complex relationships between spontaneous and uncoordinated behavioural changes, the emergence of its contagion properties, and mitigation of infectious diseases. We find that the presence of effective behavioural changes can impede the persistence of disease. Furthermore, it was found that under perfect effective behavioural change, there are three regions in the response factor (e.g., imitation and/or reactionary) and behavioural scale factor (e.g., global/local) factors $\rho$–$\alpha$ behavioural space. Mainly, (1) disease is always endemic even in the presence of behavioural change, (2) behavioural-prevalence plasticity is observed and disease can sometimes be eradication, and (3) elimination of endemic disease under permanence of permanent behavioural change is achieved. These results suggest that preventive behavioural changes (e.g., non-pharmaceutical prophylactic measures, social distancing and exclusion, crowd avoidance) are influenced by individual differences in perception of risks and are a salient feature of epidemics. Additionally, these findings indicates that care needs to be taken when considering the effect of adaptive behavioural change in predicting the course of epidemics, and as well as the interpretation and development of the public health measures that account for spontaneous behavioural changes.



## 1. Introduction

Infectious disease – ranging from well-known epidemics such as smallpox, AIDS/HIV, SARS, influenza, Ebola to less dramatic unknown pathogens causing significantly high infant morbidity and mortality – pose a constant threat to humanity. These infectious diseases exert a substantial selective pressure on hosts at both biological and cultural/social levels, resulting in different adaptation mechanisms needed to mitigate and/or reduce the costs (e.g., social, economic, political, psychological) posed by these diseases (Anderson, 1992; Cavalli-Sforza and Feldman, 1981; Cox, 2002; Shrewsbury, 2005). In the last few decades, we have witnessed a variety of infectious disease-related behavioural and cultural transformations (e.g., advances in sanitation and hygienic practices; use of alkaline laundry soap, insect repellents and mosquito nest in Africa to combat malaria, condom uses and reduction in sexual partners, see Nesse, 2005; Neuberg et al., 2011; Bishop et al., 1991; Chen, 2004). Many of these behavioural and cultural adaptations have helped minimize or reduce the prevalence of many infectious diseases in communities across the globe. One of the key challenges in public health decision making is understanding the extent to which individuals and communities would modify their behaviours to detect and minimize or eliminate exposures with pathogenic threats. As a result, understanding how behavioural patterns are generated during epidemics as well as how behavioural responses affect the transmission processes is important for predicting and for designing effective public health measures.

To understand the extent to which behavioural changes help reduce the prevalence of an infectious disease, several scholars have recently formulated theoretical models incorporating a variety of behavioural processes into epidemic dynamics. The impetus for this direction owes much to various works in both medical anthropology and health economics, specially the pioneering work of Alland (1975), Philipson (1996) and Fine and Clarkson (1987). Alland

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(1975) employed evolutionary theory to expositorily explore the impact of cultural hygienic practices (e.g., health-promoting and health-demoting practices) in minimizing the risk of disease and maximizing the welfare and health. Philipson (1996) investigated the occurrence of infectious diseases, the effects of public health interventions designed to control them and the incentive structure for vaccination, while the work of Fine and Clarkson provided one of the first empirical studies of the efficacy of vaccine.

Although we have seen an increase in behavioural models, most models – within the extensive literature on epidemiological studies of infectious diseases and dynamics – have focused on the subtleties of host–pathogen interactions and transmission mechanisms, and those addressing preventive behaviours are relatively small. The majority of these models have focused primarily on vaccination demands and strategies (Philipson, 1996; Basu et al., 2008; Bauch et al., 2003; Bauch and Earn, 2004; Bauch, 2005; Breban et al., 2007; Breban, 2011; Perisic and Bauch, 2009; Fu et al., 2010; Galvani et al., 2007; Ndeffo Mbah et al., 2012; Reluga et al., 2006; Reluga, 2010; Plotkin, 2006; d'Onofrio et al., 2008). Recently, other behavioural changes have been studies, including information transmission, social distancing and exclusion such as quarantine and crowd avoidance, participation in antiviral treatment and others (Epstein et al., 2008; Chen, 2004, 2009; Kiss et al., 2010; Funk et al., 2010; Reluga, 2010; van Boven et al., 2008; Perra et al., 2011; Poletti et al., 2011; Del Valle et al., 2005). These models have relied mostly on the use of differential equations formalism and have been limited to deterministic compartmental models, differential game-theoretical approaches, dynamic programming, inductive reasoning games and stochastic modelling of behavioural responses to global information. In particular, models incorporating spontaneous behavioural changes in response to epidemics use imitation in a game-theoretical sense to induce behavioural changes in mean-field or differential equation setting. These approaches rely on the cost-benefit considerations of the perceive risks of infection. For instance, Poletti and colleagues employed such approach in Poletti et al. (2011) to investigate the risk perception on the 2009 H1N1pdm influenza. Also, models incorporating behavioural contagion processes in conjunction with epidemiological dynamics on theoretically and/or empirically derived networks have recently been put forth. In particular, the co-evolution of information diffusion as a proxy for effective preventive behavioural changes and epidemiological contagion has been investigated by Funk and colleagues, (e.g., see Funk et al., 2009, 2010 for a review on different investigations on the impacts behavioural changes on epidemiological dynamics). However, these models, as in other deterministic game-theoretical formulation, rely on a rational construction of agents utilizing payoff maximization (e.g., homo-economicus perspective) to trigger preventive behaviours including vaccinations (Fu et al., 2010; Ndeffo Mbah et al., 2012; Perisic and Bauch, 2009; Epstein et al., 2008; Kelso et al., 2009). In Fu et al. (2010) and Ndeffo Mbah et al. (2012), the authors used game-theoretic approach within an in silico to explore the effect of cost-benefit of imitation of vaccination patterns. In particular, they illustrated the importance of local information on behavioural changes (e.g., imitational behaviours) and its impact on the transmission dynamics.

In this paper, we use an approach that relies on homo-psychologicus perspective to incorporate behavioural component into epidemiological model. In particular, we combine previously separate effects of local and global information as causal determinants of preventive behaviour in an *in silico* model. Instead of relying on the rational utility-maximizing precepts of game-theoretical constructs to determine adoption of preventive behaviour, we utilize a behaviourally enhancement threshold based on *homo-psychologicus* assumptions and mechanisms that respond to the type of information perceived by each individual agent (Curtis et al., 2004, 2011; Duncan et al., 2009; Mortensen et al., 2010; Oaten et al., 2009; Welling et al., 2007). Applying dual-path influence theory (Bandura, 2001; Pryor et al., 2004), which posits that a personal agency and social structure operate as interdependent determinants in an integrated causal structure, we define a causal structure as the sequence of cognitive steps leading to preventive behavioural adaptations. In the proceeding sections, we outline the methods use to generate network, to spread disease on the network, to establish behavioural dynamics and its contagion process, and to simulate the model outcomes.

## 2. Materials and methods

We investigate the impact of reactionary behavioural change and its contagion dynamics on epidemiological processes and quantities. We focus on *homo psychologicus* aspects, rather than *homo economicus*, of behavioural change and provide *in silico* results on epidemic duration. Here both behavioural and epidemiological contagion processes have been abstracted from realistic settings where behavioural dynamics, such as spontaneous social exclusion and distancing, crowd avoidance, voluntary vaccination and facemask wearing (to a lesser extent), in the presence of infectious diseases play significant roles in understanding the course of epidemics. We develop an SIRBS-type model on a quasi-static socio-spatial network, where we associate each node, in the population of size $N$, with three epidemiological and one behavioural state (see Fig. 1a). That is, the population is compartmentalized into four classes, where individuals can be susceptible ($S$), infected ($I$), recovered ($R$) or behaviourally removed ($B$). Susceptible individuals can be infected by infectious neighbours and can subsequently recover from the disease. Entry into the behavioural class is conditioned on the perceived risk or effective behavioural enhancement cue $\mathcal{K}$ and internal psychological threshold $\sigma$ (see the schematics in Fig. 1b and Section 2.3 for details). Although Fig. 1a shows a model structure similar to a SIRS-type model with either vaccination or partially resistant compartment in literature (see for example Hadeler and Van den Driessche, 1997; Alexander et al., 2004; Reluga et al., 2007), the mean-field approximation of the model summarised by schematics shown in Fig. 1a will yield a model with Filippov type dynamical system (or discontinuous piecewise differential equations). In that, the behavioural compartment is conditioned on a state-dependent threshold function. In the next sections, we outline the different processes implemented in the *in silico* model.

### 2.1. Network model

We model the contagion process on a contact network where vertices represented individuals and edges denote contacts between members. An edge represents contact between vertices that can allow direct transmission of either infectious agents or behavioural memes or both. Here we use a half-Gaussian socio-spatial network for its flexibility to generate a wide variety of network structures ranging from highly clustered lattices, small world phenomena to globally connected random network arrangements with a high degree of heterogeneity. To construct the network, we uniformly distribute node/agent or vertices on a square patch/lattice and connect two vertices based on their locations, given rise to a spatial network structure. Agents are connected using half-Gaussian distribution with width $D$ described by:

$$p(k, D) := \frac{\langle k \rangle}{2\pi D^2} \exp\left(-\frac{d_{ij}^2}{2D^2}\right), \tag{1}$$

where $p(k, D)$ is the probability that two nodes will be connected, $d_{ij}$ is the distance between nodes $i$ and $j$, and $\langle k \rangle$ is the average number

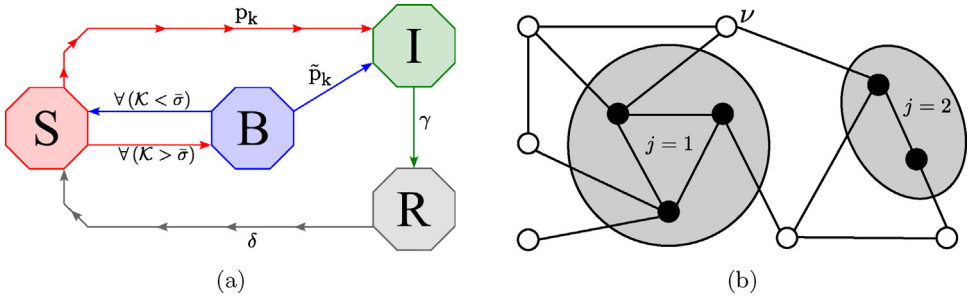


**Fig. 1.** Schematic representation of epidemiological and behavioural contagion models: (a) the sketch shows the flow diagram of disease model in which individuals move between compartmental groups at rates indicated by the arrows. The arrows show the flow directions (see the text). (b) The graph provides a visual representation of behavioural contagion process and illustrates the computation of behavioural enhancement cues. On the graph, the focal vertex is indicated by $\nu$ and grey shadings show subsets of neighbours of the focal vertex with status $\mathcal{X}$ (see the text for details).

of contacts or degree. The parameter $D$ is the spatial length-scale and measures the socio-spatial preferential vicinity. The parameter $D$ tunes how much preference is given to nearest individuals, and control the local and/or long-range interaction between individuals. In addition, $D$ serves as a proxy for clustering coefficient, where low $D$ exhibits local structures with high clustering coefficient (e.g., $D = 2$ generates networks with clustering coefficient $CC = 0.075$, $CC$ denotes the clustering coefficients which measures the degree to which nodes tend to cluster) and high $D$ provides global connections with low clustering coefficient (e.g., $D = 10$ generates networks with clustering coefficient $CC = 0.015$; see Keeling, 1999, 2000 and references therein for further discussion and detailed characterization of socio-spatial network generated by half-Gaussian distribution). Hence the network exhibits more long-range contacts for high $D$ while short-range contact and locally clustered structures are observed for small $D$ (see Fig. 2). We generate a network using this half-Gaussian distribution with $N = 2500$ vertices such that a parametrized average density is achieved while

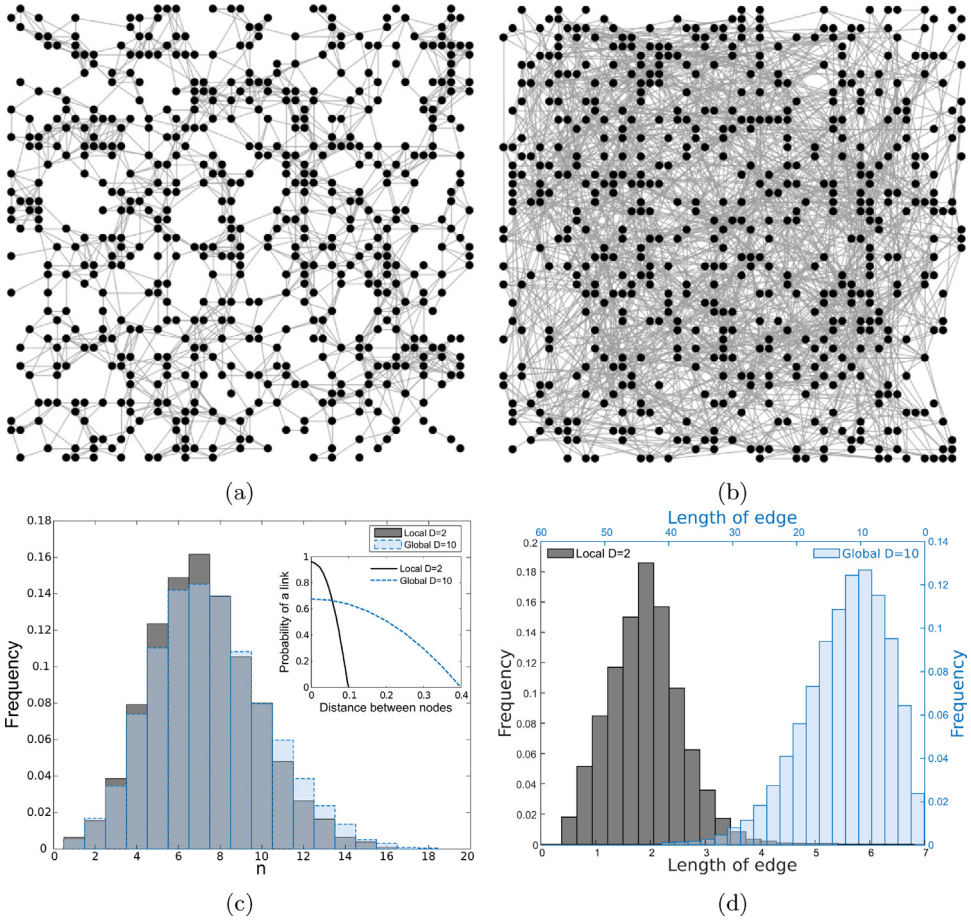


**Fig. 2.** Network generation and properties: graphs with two different $D$ values (a) $D = 2.0$ and (b) $D = 10.0$, where the total number of agents have been set to $N = 600$ for purpose of visual aesthetic. Plots (c, d) are some of the relevant network property statistics and illustrate the use of $D$ as a tuning parameter that affects both the average length of the edge and the clustering coefficients. In all these graphs, we assume an average number of contacts $\langle n \rangle = 7$ and (c) degree distributions are similar, and the probability of links shows that low $D$ is more local while large values of $D$ exhibit long-range contacts (inset). (d) The average length of edges is different for all the networks. Network with $D = 2$ has smaller average length than that of the network with $D = 10$. The results were adopted from Keeling (1999), Read and Keeling (2003), Cohen et al. (2007), Janssen et al. (2010) and Cherif et al. (2009).

**Table 1**
Epidemic and behavioural parameters: the table shows initial conditions and parameter values used in the initialization process.

| Variable | Description | Initial values |
|---|---|---|
| $N$ | Population number | 2500 |
| $S$ | Susceptible group | 2495 |
| $I$ | Infectious group | 5 |
| $R$ | Recovered group | 0 |
| $B$ | Behaviourally removed group | 0 |

| Parameters | Description | Values |
|---|---|---|
| $\langle k \rangle$ | Average degree | 5 |
| $D$ | Length scale | 10 |
| $\beta$ | Transmissibility | 0.1 |
| $\gamma$ | Self-recovery rate | 0.01 |
| $\delta$ | Loss of immunity | 0.001 |
| $\mu$ | Natural mortality | 0.001 |
| $\sigma$ | Internal behavioural threshold | U(0, 1, 0.5)[a] |
| $\mathcal{K}_l$ | Local behavioural enhancement cue | 0 |
| $\mathcal{K}_g$ | Global behavioural enhancement cue | 0 |
| $\mathcal{K}$ | Effective behavioural enhancement cue | 0 |
| $\alpha$ | Behavioural scale factor | $\alpha \in [0, 1]$ |
| $\rho$ | Response factor | $\rho \in [0, 1]$ |
| $\varepsilon_b$ | Behavioural efficacy | 1 |

[a] Uniform distribution with range [0,1] and average 0.5.

maintaining the appropriate distribution. Here we use $D = 10$ and $\langle k \rangle = 5$ (see Table 1).

### 2.2. Spread of infection

The spread of disease follows the standard SIRS model with Reed-Frost stylized infection dynamics to describe the transmission process (Abbey, 1952; O'Neill, 2003), where at each time step a susceptible can become infected with probability $p_k := 1 - (1 - \beta)^k$. Here $k$ is the number of neighbours who are infected and $\beta$ is transmissibility rate, $\beta \in (1, 0)$. Similarly, behaviourally removed individuals can get infected based on the effectiveness of the chosen behavioural practices/changes. They can be infected with probability $\tilde{p}_k := 1 - (1 - (1 - \varepsilon)\beta)^k$, where $\varepsilon$ is the effectiveness of chosen behavioural change. Note that when $\varepsilon = 1$ behavioural change is 100% effective and behaviourally removed individuals are completely protected, and $\varepsilon = 0$ behaviourally removed individuals are completely susceptible to infection and the chosen behavioural change is ineffective. Also, infected individuals recover with probability $\gamma$ and become temporarily immune. After some period, individuals who have recovered lose immunity and become susceptible again with probability $\delta$ (see Fig. 1a). At any time, individuals can leave each class at a rate $\mu$, either due to natural mortality or other means of removal. We have assumed that infected and recovered individuals do not participate in behavioural changes. For the sake of simplicity and consistency, individuals who leave the systems are automatically reintroduced into the system but with different links to maintain constant total population $N$, hence making the network behave quasi-statically. Here dead individuals are not just reincarnated into the system. Rather, a neighbour $i$ of the dead agent moves into its place, and neighbour $j$ of $i$ moves into $i$'s location, and so on. It should be noted that we are not reshuffling the whole network structure, rather the process is equivalent to rewiring the edges of dead individuals when re-introduced into the population on each birth-death event. Then a susceptible individual is introduced into the network, preventing the introduction of spatial correlation between death and birth process while maintaining computational costs. Table 1 summarises the value used in the simulation. Here the values have been chosen such that the dynamics are calibrated to produce an endemic state in the absence of behavioural change. Although mean-field models usually produce endemic equilibrium states that are commensurable with the epidemic parameter values provided the initial conditions be in a given region of attraction, the *in silico* models can sometimes yield a multi-modal steady-state distribution (e.g., bi-stability), where the trajectories can leave a given region of attraction (e.g., boundary crossing of stability regions). Herein, we have fixed the epidemic parameters to explore the impact of behavioural components on the epidemiological quantities such as the average length of outbreaks.

### 2.3. Behavioural change

Behavioural changes involved in the reduction of infection risks usually depend on individual vulnerability to infection, and the necessary cues required to activate behavioural responses are sensitive to any information that suggests increased vulnerability to disease transmission. As illustrated in the recent behavioural reactions to 2009/H1N1pdm (Poletti et al., 2011; Cowling et al., 2010; Liao and You, 2014), this information may come from either internal sources (e.g., phobias, worries and chronic anxieties, greater disgust) or external sources (e.g., news, context-specific perceptual reminders of the epidemiological threats) regardless of the veracity of individual subjective vulnerability or perception. As a result, the activation of disease avoidance behavioural changes is influenced by the subjective perception. For example, if individuals perceive themselves to be more vulnerable to the spread of disease, they are more likely to be perceptually sensitive to cues suggesting the heightened risks and to participate (sometimes exaggeratedly) to aversive responses.

In order to incorporate behavioural dynamics, we condition entry into behaviourally removed class by the level of effective behavioural enhancement cue $\mathcal{K}$ and internal subjective vulnerability factor/threshold $\sigma$. Here we assume $\sigma$ to be constant and uniformly distributed $U(0, 1)$ for the sake of simplicity. At each time step, the subjective vulnerability factor $\sigma$ is compared with the effective behavioural enhancement cue $\mathcal{K}$ which combines both behavioural and epidemiological cues from the environment, and is related to the perceived risk of infection. The effective behavioural enhancement cue $\mathcal{K}$ is given as a convex combination of two dual-influence components weighted by $\alpha$ and $1 - \alpha$, where $\mathcal{K}$ is given as:

$$\mathcal{K} = (\alpha)\mathcal{K}_g + (1 - \alpha)\mathcal{K}_l \tag{2}$$

$$\mathcal{K}_g = (\rho)\left[\frac{\kappa_*(I)}{\kappa_{**}(I)}\right] + (1 - \rho)\left[\frac{\kappa_*(B)}{\kappa_{**}(B)}\right] \tag{3}$$

$$\mathcal{K}_l = (\rho)\left[\frac{\sum_j \eta_*(I)_j}{\sum_j \eta_{**}(I)_j}\right] + (1 - \rho)\left[\frac{\sum_j \eta_*(B)_j}{\sum_j \eta_{**}(B)_j}\right] \tag{4}$$

where $\kappa_*(\mathcal{X})$ denotes the total population size with either epidemiological or behavioural status $\mathcal{X}$, and $\kappa_{**}(\mathcal{X})$ is complement of $\kappa_*(\mathcal{X})$. That is, $\kappa_{**}(\mathcal{X})$ represents the total number of the population that are not in the epidemiological or behavioural class $\mathcal{X}$. Similarly, $\eta_*(\mathcal{X})$ denotes the subset of neighbours with a specific epidemiological or behavioural status $\mathcal{X}$, and $\eta_{**}(\mathcal{X})$ is its complement. Furthermore, $\alpha$ is the behavioural scale factor and measures the extent to which behavioural cues are globally or locally perceived. For instance, when $\alpha = 0$, behavioural change is influenced mostly by local behavioural enhancement cue factor $\mathcal{K}_l$ and when $\alpha = 1$ the global behavioural enhancement cue $\mathcal{K}_g$ affect the course of behavioural adaptation. In addition, both local and global behavioural cues $\mathcal{K}_l$ and $\mathcal{K}_g$ are determined by response factor $\rho$ such that when $\rho = 1$ behavioural change is due primarily to reaction to infectious individuals and imitation of behaviourally removed population when $\rho = 0$. Fig. 1b shows how behavioural enhancement cue factors are determined. For example, if the focal vertex $v$ has $\mathcal{X}$ status neighbours, then $\kappa_*(\mathcal{X}) =$

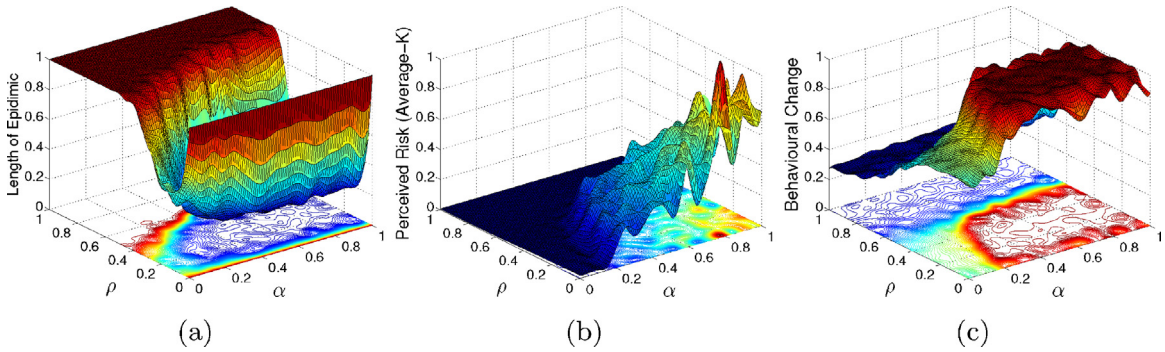


**Fig. 3.** Behavioural and epidemic dynamics: the data show the effects of behavioural scale factor and inclination factors on the average duration of the epidemics, average behavioural enhancement cues and perceived risks, and prevalence of behavioural response. All results are determined from average of 154,350 simulations. (a) The average length of epidemic measures duration of epidemics before its extinction and quantifies the permanence of disease in the population. Here the average length of epidemic has been scaled and is given as $\frac{\mathcal{T}^*}{\mathcal{T}^*_{\max}}$. (b) Perceived risk is measured as average effective behavioural enhancement cues. (c) This figure shows the prevalence of behavioural change in the population under different behavioural parameters. Here $\alpha$ and $\rho$ are behavioural parameters that respectively quantify the behavioural scale factor (e.g., local or global behavioural dynamics) and the inclination to either react to new cases of infectious individuals in the population or to imitate others participating behavioural responses such as social distancing and/or exclusion, crowd avoidance, and voluntary vaccination. All quantities presented on $z$-axis have been scaled by their respective maximum values.

5, $\kappa_{**}(\mathcal{X}) = 6$, $\eta_*(\mathcal{X})_1 = 3$, $\eta_{**}(\mathcal{X})_1 = 5$, $\eta_*(\mathcal{X})_2 = 2$ and $\eta_{**}(\mathcal{X})_2 = 3$. Assuming that $\mathcal{X}$ denotes behaviourally removed status and $\rho = 0$, then $\mathcal{K}_l = \frac{\sum_j \eta_*(B)_j}{\sum_j \eta_{**}(B)_j} = \frac{5}{8}$ and $\mathcal{K}_g = \frac{\kappa_*(B)}{\kappa_{**}(B)} = \frac{5}{6}$. Then $\mathcal{K}$ can easily be obtained. At each time step, Eqs. (2)–(4) are calculated for each agents. However, here we assume that only susceptible individuals can change behaviours. To do this, we compare the behavioural enhancement factor $\mathcal{K}$ and internal threshold $\sigma$. If $\mathcal{K} > \sigma$, individuals change behaviour where they can participate in preventive measures such as social distancing/exclusion, crowd avoidance, vaccination, and remain there while $\mathcal{K} > \sigma$ is satisfied. If $\mathcal{K} \leq \sigma$, they either remained in their current state if that status is susceptible, or return to susceptible group if they were in behaviourally removed class. While individuals are in behaviourally removed class, they are given an efficacy factor $\varepsilon_b \in [0, 1]$ which determines the effectiveness of behavioural response (e.g., high values give more protection to individuals than lower values). Our preliminary results suggest that the epidemic is more sensitive in a very small range of behavioural efficacy $\varepsilon_b$ (i.e., 0.90–1). As a result, studies discussed herein assume a fixed behavioural efficacy and assume that behavioural change provide complete protection with $\varepsilon_b = 1$. It should also be noted that we have assumed endemic infectious disease. However, setting the demographic terms (e.g., birth-death parameters) to zero does not change the results drastically. We found that the equilibrium structure is preserved and is commensurable with the model parameters. These are consistent with results in mathematical models with SIRS-type transmission dynamics.

### 2.4. Agent-based simulations

Putting together both the epidemiological and behavioural dynamics, we implement an *in silico* model using socio-spatial contact structure generated from half-Gaussian distribution. Our simulation scheme consists of network generation, introduction of infected individuals into the susceptible population, and subsequent iterations of both behavioural and transmission mechanisms until the end of simulation time or first extinction time. Table 1 summarises the parameter values used in the simulations, where network of susceptible were generated using Eq. (1) to investigate the effect of the contact structure on the dynamics of behavioural change and epidemiological process. In addition, 0.2% of the total population size are initially infected with the disease. Furthermore, we synchronously update individual status. Here we have fixed all parameters except $\alpha$ and $\rho$ since the effects of epidemics and contact patterns have been investigated elsewhere (Keeling, 1999, 2000; Keeling and Eames, 2005; Perisic and Bauch, 2009; Janssen et al., 2010). While the disease parameters might affect the magnitude of endemicity, the qualitative dynamics do not differ. As such, both the effects of disease parameters and contact structures on epidemics and behavioural dynamics are not studied herein. For the behavioural parameters investigated, we assume that behavioural change provides perfect protection against the disease. The results presented below are for 154,350 simulations, where stochasticity in the model formulation and results is due to the network connections and demographic dynamics that determine it, random initial infections and spread of the disease through the network. The model was implemented in NetLogo (Wilensky, 1999).

## 3. Results and discussion

Here we show that effective behavioural changes can substantially be sufficient to eradicate the disease (see Figs. 3–5). Data in Figs. 3–5 summarise the results of the study investigated herein,

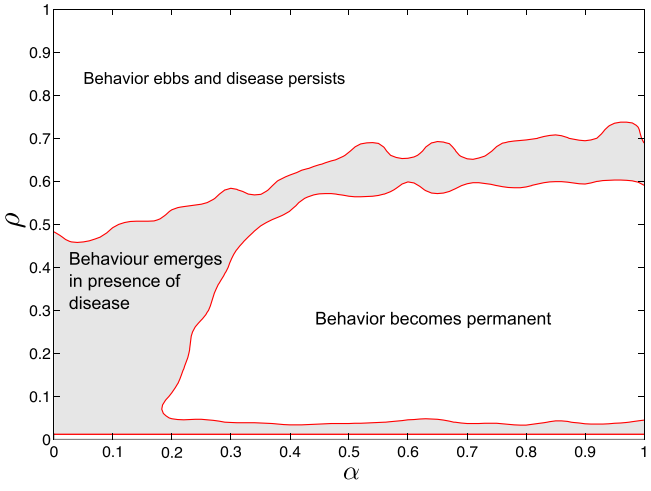


**Fig. 4.** Behavioural parametric outcome space: the diagram illustrates the three regions where different behavioural and epidemiological dynamics are observed in the population. The behavioural space was constructed from the composite of Fig. 3a–c. The two white regions are equivalent to the regions in behavioural change in Fig. 3a and c, where the disease and behavioural change always persist or, equivalent disease persist and goes to extinction, respectively. The grey region is the intermediary phase, where the dynamics exhibit prevalence elasticity.

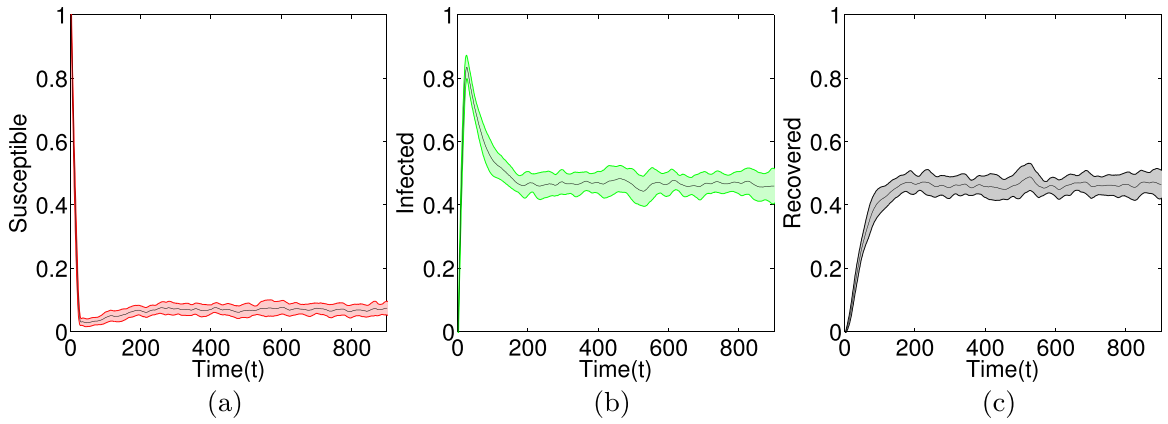


**Fig. 5.** Temporal epidemiological process in the absence of behavioural change: the plots show the temporal dynamics for (a) susceptible, (b) infected, and (c) recovered individuals in the absence of behavioural dynamics. The bands of variation correspond to the 95% confidence interval. It is equivalent to *in silico* representation of SIRS.

and all the results in Fig. 3 have been scaled by their respective maximum values. Under a perfect (effective) behavioural change, we observe that disease endemicity can be affected in 59% of behavioural outcome space. In addition, these regions are affected by co-influence of behavioural factors. Fig. 3a shows the average length of epidemic as functions of the behavioural scale and response factors $\alpha$ and $\rho$, respectively. Here the average length or duration of the epidemic is defined as the first passage time before the disease goes extinct in the population. That is, $\mathcal{T}^* := \inf\{t \geq 0 : I(t) = 0\}$. If the disease is endemic and does not go extinct during the simulation, we set $\mathcal{T}^*$ to be the final time of the simulation. Here we simulate the model for 3500-time steps. The average length of the epidemic in Fig. 3a has been scaled by $\mathcal{T}^*_{\max} = 3500$. Fig. 3a shows that, for high response factors $\rho$, the disease is always endemic even in the presence of some behavioural changes. It implies that when behavioural changes are driven by reactions to infectious individuals, the majority of individuals who initially participated in behavioural response leaves the behaviourally removed class as they replenish the susceptible class. This is illustrated in Fig. 3b–c, where high level of response factors result in very low average effective behavioural enhancement cue $\mathcal{K}$, which determines the perceived risk as shown in Fig. 3b and affects the population size of individual persistently participating in behavioural changes seen in Fig. 3c. As a result of these co-influencing factors, periodic abandonment of behavioural changes (e.g., social distancing and social exclusion, crowd avoidance) contributes to the permanence of disease endemicity in the population.

Furthermore, we find that the effect of behavioural change on the dynamics of epidemic can be organized into three categories in behavioural outcome $\alpha$–$\rho$ space: (i) ebbed behavioural change with endemic disease (disease permanence), (ii) coexistence of behavioural change and disease with prevalence-behaviour plasticity, and (iii) behavioural permanence with disease eradication. These observations are summarised in Fig. 4. In the region where behavioural change is ebbed, we observe that the disease is always endemic despite the existence of behavioural responses. In this region, the behavioural response is unable to eradicate the disease due to its ebb and flow nature in which prevalence-elastic behaviour is seen. This type of behavioural response has been observed in the context of both measles and HIV in some countries (Philipson, 1996; Ahituv et al., 1996).

The remaining regions where we see behavioural characteristic of (ii) and (iii), it was found that permanent behavioural change can eradicate disease, which is consistent with the mathematical results of (Del Valle et al., 2005) with a particular emphasis on behavioural changes in smallpox. In the region (ii) where we observe the coexistence of both behavioural change and endemicity of the disease, the disease can be eradicated provided the epidemic threshold has been crossed. However, in this region, eradication is conditioned on the number of infected individuals in the population and epidemic parameters and has lower averaged epidemic duration $\mathcal{T}^*$. In the third region, it was observed that when the response factor $\rho$ is low resulting in high inclination to imitate neighbours either locally or globally, effective behavioural responses can impede the spread of infectious diseases eventually leading to its eradication. However, strong global influence may typically lead to a rapid adoption of preventive behavioural responses. In Fig. 4, we also observe that, for small $\rho$ and large $\alpha$ (e.g., globally imitational behaviour), behavioral characteristic (ii) where, despite relatively high participation in behavioural changes, disease is persistent. In this region, we observe the formation of clustering of individuals participating in behavioural changes, specially imitational behavioural dynamics. The relative size of the disease does not necessarily decrease as more individuals imitate because the behavioural herd immunity is relatively high within/near these clusters, and further participation in behavioural change within/near these clusters reduces the transmission to a lesser extent than if the behavioural changes were initiated in a region with small proportion of individuals engaged in behavioural changes. Hence, individuals near or surrounded by a large number of clusters of behaviourally removed individuals already benefit from the herd immunity these clusters provide. Therefore, the participation of these individuals within, near or surrounded by these behaviourally removed clusters does not necessarily decrease the size of the disease. A similar dynamic pattern was observed in Ndeffo Mbah et al. (2012), where the authors study the effects of imitation behaviour and contact structure on vaccination coverage and disease dynamics. In Kiss et al. (2010) and Funk et al. (2010), the authors obtained similar results using multigroup (2-core groups) compartmental models and found that disease can be eradicated under certain threshold conditions. Our model introduces heterogeneity, both in the disease transmission process and behavioural dynamics, not considered in Kiss et al. (2010) and Funk et al. (2010); and it does not use multigroup formulation. Fig. 5 shows the temporal dynamics of the model in the absence of behavioural change. It is equivalent to the standard SIRS model. In Fig. 6, the temporal dynamics of the model variables are illustrated in the presence of behavioural changes in the three behavioural parametric outcome space shown Fig. 4.

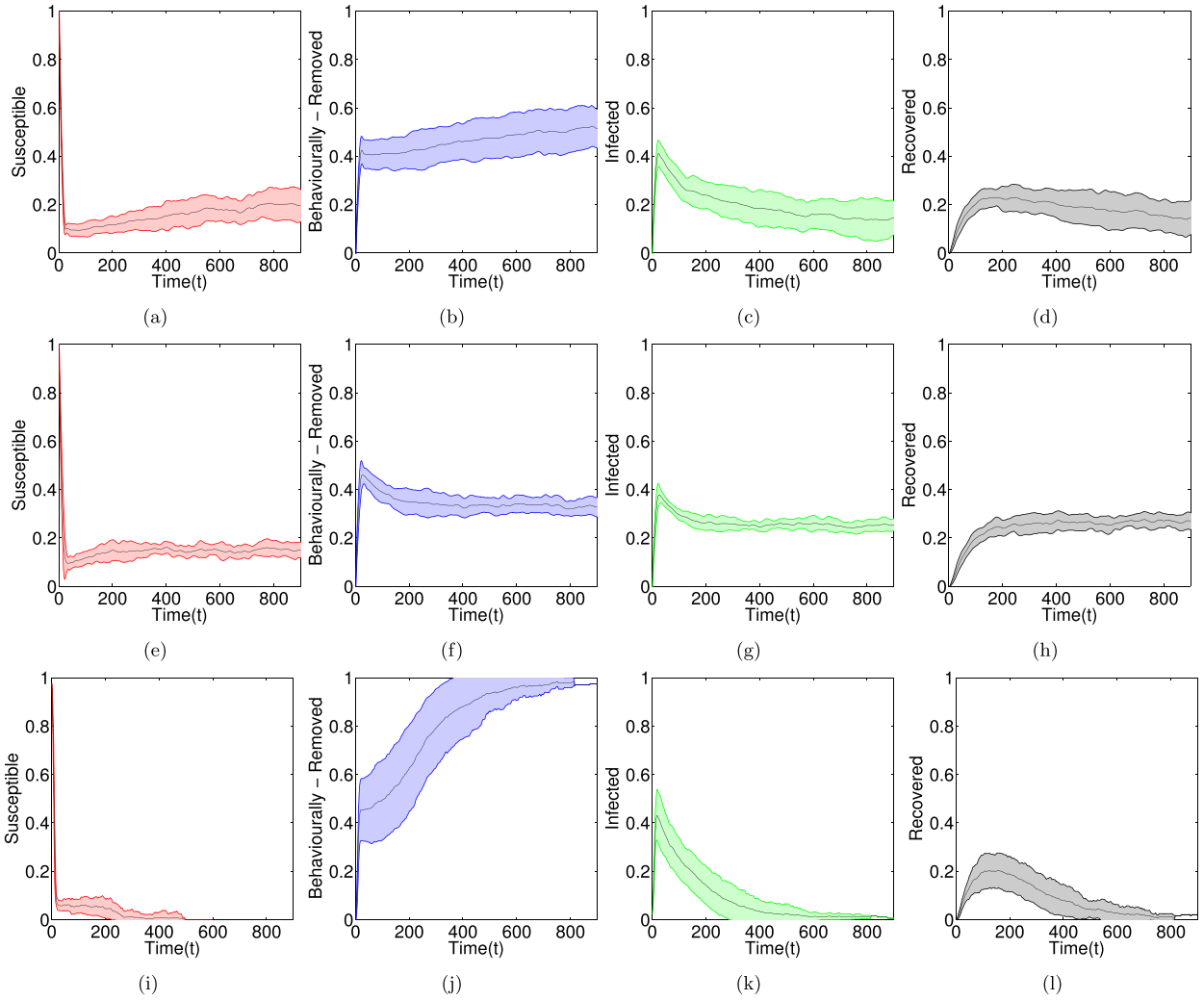


**Fig. 6.** Temporal epidemiological and behavioural contagion dynamics: the plots summarise the temporal dynamics of various quantities in the presence of behavioural change. Temporal dynamics of proportion of both epidemiological and behavioural groups when (a)–(d) behavioural scale factor $\alpha = 0.1$ and response factor $\rho = 0.4$, (e)–(h) behavioural scale factor $\alpha = 0.5$ and response factor $\rho = 0.85$, and (i)–(l) behavioural scale factor $\alpha = 0.5$ and response factor $\rho = 0.25$, respectively. The bands of variation correspond to the 95% confidence interval.

## 4. Conclusion

We have illustrated that behavioural responses to infectious diseases can reduce the prevalence and/or eradicate the disease under certain conditions. However, we find that reactionary behavioural responses are usually not sufficient enough to eradicate the disease due to their ebb-and-flow nature, where individuals who have initially reacted to the disease tend to periodically abandoned their behavioural changes. It consequently leads to behavioural plasticity (e.g., behaviour-prevalence-elasticity), where we found that lower outbreaks are observed, and behavioural response does not affect epidemiological threshold. Under this prevalence-elasticity with high response factor $\rho$ (e.g., a strong inclination to react to the number of infectious individuals), behavioural responses tend to closely follow the prevalence of infected individuals either locally or globally. Because the behavioural change is reactionary, we observe ebb-and-flow dynamics, recurrence and periodicity in the behavioural responses due to the fact that individuals usually change their behaviours when the prevalence is high and abandon them when the prevalence goes down. As a result, the disease persists despite the presence of effective behavioural responses.

In addition, it was shown that imitational behavioural changes that are persistent at high behavioural threshold tend to eradicate diseases when these behavioural responses provide perfect protections against infectious diseases. However, these different epidemiological dynamics are influenced by the interplay between the locality of behavioural scale factor and inclination properties of behaviours. This highlights the drawback of models that rely on the aggregate information to trigger a behavioural response and indicates the possibility of over-estimation of behavioural changes on the epidemiological dynamics.

Although the costs associated with disease and adoption of behavioural responses were not considered in our model, the effects of such additional properties do not greatly impact the outcome of the dynamics observed, and the results will be preserved as long as the associative costs of behavioural responses are less than those of the disease. However, if the associative costs of the disease are less than those of behavioural responses, we could, at the very least, observe smaller or a loss of behavioural outcome space where the permanence of behavioural change affect the persistence of endemic disease.

Insights gained from the model can be improved greatly with a various extension of our model. One such version could incorporate access to prophylactic measures such as vaccination and treatment. Furthermore, other models incorporating rationality could utilise the results outlined herein when constructing agents that

have to manage both local and global information in decision-making processes. Also, our analyses of the model could benefit from the use of empirical data measuring reactions to the threat of infectious disease and perceived personal susceptibility or vulnerability. The use of our model and its extensions in combination with disease-specific empirically derived behavioural responses and exploitation of internet for syndromic surveillance data (e.g., disease-related and health-related information such as Google search trends, Twitter feeds, news stories (see Ginsberg et al., 2009; Jones and Salathé, 2009) could lead to predictive models that substantially improve public health decision-making of current and/or emergent and re-emergent outbreaks of infectious diseases, and could provide a greater insight into behavioural outcomes such as vaccine demands for vaccine-preventable diseases, condom use for sexually transmittable diseases and others. For seasonal epidemic or diseases in a periodic environment, the model presented here could easily be extended. In particular, we can assume behavioural enhancement condition to be of the form $\mathcal{K}\sigma^* \leq \sigma$, where $\sigma^*$ is the constant internal cue and $\sigma$ is a function of state (e.g., $\sigma_{t+1} = \mathcal{H}_{\tilde{I}_t \geq \sigma_t}(\zeta_1\sigma_t + \bar{\sigma}_0) + \mathcal{H}_{\tilde{I}_t < \sigma_t}(\max\{\zeta_1\sigma_t, \zeta_2\tilde{I}\})$, $\tilde{I} = I_t$ or $\tilde{I} = \frac{I}{I+k}$, $\mathcal{H}$ is the Heaviside function). This allows one to incorporate moving behavioural enhancement threshold needed to activate behavioural response in the presence of differential risk and/or perceived vulnerability.

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