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Vaccination and public trust: A model for the dissemination of vaccination behaviour with external intervention

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HIGHLIGHTS

- The spreading of anti-vaccination opinion is part of a cultural dissemination process.
- The underlying social network is composed by two types of edges: opinion-exchange and personal ones.
- The social adaptive dynamics is given by a process of cultural similarity.
- The mitigating effect of a public health campaign is included as an external field.
- The outbreaks are determined by the degree of clustering of unvaccinated agents.

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ABSTRACT

Vaccination is widely recognized as the most effective way of immunization against many infectious diseases. However, unfounded claims about supposed side effects of some vaccines have contributed to spread concern and fear among people, thus inducing vaccination refusal. MMR (Measles, Mumps and Rubella) vaccine coverage has undergone an important decrease in a large part of Europe and US as a consequence of erroneously alleged side effects, leading to recent measles outbreaks. There is evidence that clusterization of unvaccinated individuals may lead to epidemics way larger that the ones that might appear in the case that unvaccinated agents are distributed at random in the population. In this work we explore the emergence of those clusters as a consequence of the social interaction driven mainly by homophily, where vaccination behaviour is part of a process of cultural dissemination in the spirit of Axelrod's model. The ingredients of this calculation encompass: (i) interacting agents which are to decide if they vaccinate or not their children, (ii) their interaction with a small subset of stubborn agents who believe that the MMR vaccine is not safe and (iii) government sponsored propaganda trying to convince people of the benefits of vaccination. We find that these clusters, which emerge as a dynamical outcome of the model, are the responsible of the increasing probability of the occurrence of measles outbreaks, even in scenarios where the WHO (World Health Organization) recommendation of 95% vaccine coverage is fulfilled. However, we also illustrate that the mitigating effect of a public health campaign, could effectively reduce the impact and size of outbreaks.

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1. Introduction

Vaccination constitutes one of the main ways to prevent the spread of infectious diseases in modern era only behind safe water [1]. It is largely recognized as the most effective method for immunization, with great success in the worldwide eradication of smallpox and in the control of other infectious diseases such as measles, rubella, tetanus, and polio almost all over the world [2,3]. However, recent outbreaks of measles in UK and US have awoken renewed concern about vaccination rejection [4,5]. In the particular case of MMR vaccine against measles, mumps and rubella, quickly refuted claims about a supposed link with autism [6,7] have aroused distrust and fear in people. Thus, people's perception of vaccine safety and efficiency has shown to be an important factor for vaccine uptake that, at the same time, can spread between individuals [8,9].

In those places where vaccination is not mandatory, to vaccinate or not vaccinate a child is a parent's decision which usually involves cultural and belief systems. However, decisions are not made by individuals as isolated entities, but as social agents interacting with other social actors. In this regard, recent works have begun to model the dissemination of vaccination opinion between peers as a pure social imitation process in a mean field approach [10]. As an extension of these previous models, some authors have also introduced vaccine side effects to be assessed by social agents, together with contagion risks, in a game-theoretical framework [11].

Mean field models usually assume that agents' opinions evolve in a homogeneous mixing approach, by which each agent can interact with every other agent. A step forward in the description of opinion dissemination with social contact heterogeneities is provided by agent-based modelling (ABM) where opinions spread over a social network. The clustering of unvaccinated, and then susceptible, agents constitutes one of the most important outcomes from this novel approach [12–15]. The clustering of susceptible agents leads to an effective shift of the safety threshold obtained by homogeneous herd immunity (i.e. the expected herd immunity under the assumption of random mixing), thus increasing the chances of disease outbreaks.

Today we know that opinions not only spread by personal interaction, but also through new ways of remote-socialization provided by current information technologies. Opinions and even sentiments can be spread by virtual friendship networks, as some authors have shown in recent works [16,17]. However, current ABM models to study the dissemination of vaccination behaviour assume that opinions spread over exactly the same personal social ties subsequently involved in disease spreading.

On the other hand, ABM models for dissemination of vaccination behaviour in social networks consider homogeneity at the level of social interactions, by which all social ties are supposed to be equally influentials. However, opinions mostly spread between similar individuals, as has been recently shown for the particular case of health behaviour [18]. The preferential interactions between similar individuals is a well-known concept in sociology, called *homophily* [19,20]. In this context, homophily acts as a weight for the ties between social agents. This last assumption constitutes the cornerstone of Axelrod's model, proposed in order to explain how social consensus arises in human societies [21]. In the particular case of vaccination opinion, we are interested in the opposite transition: how does the initially broad consensus about, for instance, MMR vaccine in developed countries with a wide vaccine availability, such as UK and US [22], begin to decline due to the action of small anti-vaccination movements?

Recent outbreaks of vaccine-preventable diseases have shown that although vaccine availability constitutes a necessary condition to reach global immunization, it is not sufficient. In this regard, massive public health campaigns can contribute to mitigate vaccination refusal by spreading information about the benefits of vaccination. However, as far as we know, this subject has not received much attention in previous models accounting for social network structure, beyond a few exceptions based on mean field approach [23].

In this work, we explore the effects on epidemics outbreaks of opinion spreading by a small group of anti-vaccination stubborn individuals, initially immersed in a totally pro-vaccination population with social network structure. This scenario is compatible, for instance, with the case of measles for the early years after MMR vaccine introduction in developed countries.

We implement and agent based model where two different dynamical process take place over a initial contact network: a social process where anti-vaccination opinion could spread from stubborn agents according to a social adaptive dynamics and an epidemic spreading which takes place in the resulting evolved social network.

The underlying social network is initially composed of two different classes of edges: (a) opinion-exchange edges without direct personal interaction, and (b) direct personal edges which are also involved in the opinion dissemination process and are the only ones able to transmit an infectious disease. These different kinds of edges define two entangled subnetworks over which the process of opinion dissemination takes place.

The social dynamical process is modelled following an extension of Axelrod's model, in order to study the spread of vaccination behaviour as one of the features involved in the cultural exchange between social agents, plus an active adaptation process where each social agent can also replace his dissimilar neighbours by others with which he has higher homophily degree [24]. A public health intervention is included as an external field in this model. We aim to focus on the dynamical basis of clusterization of unvaccinated agents, by placing particular emphasis on the size distribution of these clusters and their relation with the spread of an infectious disease.

We consider measles as case study. We modelled it by means of a simple stochastic compartmental model, in terms of the model describing the possible states that an individual can go through: susceptible (S), exposed (E), infectious (I) and recovered (R).

We compare outbreak sizes of measles over the unvaccinated population yielded by opinion dissemination models, with those obtained assuming that the same unvaccinated population is distributed at random over the initial social network. This comparison is also proposed for the particular case satisfying the recommended 95% MMR vaccine coverage [25]. Finally, we study the effects of massive public campaigns undertaken by health authorities in order to mitigate the action of anti-vaccination activists.

2. Models and methods

The numerical model we implemented here has four main ingredients:

- The opinion dissemination model
- The social adaptive structure
- The public health intervention
- The epidemiological model

The first three ingredients correspond to the social dynamical process which models the dissemination of the anti-vaccination opinion. Once this process is finished, the epidemiological model runs over the resulting social network. We will discuss the main elements of our model to formulate its final algorithmic description at the end of the present section.

2.1. Opinion dissemination model

We adopt a novel variant of the well-known Axelrod's model for cultural dissemination in social networks. In particular, we aim to model the spread of vaccination behaviour. However, this is only one of the features involved in a plentiful cultural exchange between social agents. All the other features evolve in an adaptation process by which the degree of homophily between connected social agents is dynamically developed. At the same time, the degree of homophily acts as a "catalyst" for the spread of vaccination behaviour between social agents.

In the original formulation, Axelrod's model considers a population of interacting social agents with cultural background described through a number of features $F \in N$, representing a particular belief system, involving different subjects such as politics, education, sports, entertainments, etc. Each of such features admits a multiplicity of traits $q \in N$. We adopt the Axelrod's model with $F + 1$ features, and a heterogeneous number of traits q. Vaccination-related opinion is a binary trait ($q = 2$), i.e., only two opposing traits are admitted: vaccinator or non-vaccinator. The remaining F features are assumed to have uniform q, with $q \geq 2$. This modification makes the model be more likely to go to the monocultural state, given that one feature has only two traits. This means that the transition from monocultural to multicultural expected in the Axelrod model takes place for higher values of q.

The cultural background of a given social agent i can be mathematically described by a time-dependent state vector $\mathbf{V}^i(t) = (V_1^i(t), V_2^i(t), \ldots, V_{F+1}^i(t))$ with $V^i(t) \in I^F \times \{0, 1\}$, where $I = \{1, 2, \ldots, q\}$ and $V_{F+1}^i(t) = 0$ corresponds to pro-vaccination opinion while $V_{F+1}^i(t) = 1$ to anti-vaccination opinion at time t. Then, we say that two social agents i and j agree in a particular feature n at time t when $V_n^i(t) = V_n^j(t)$. In addition, we define the homophily degree $h(i, j)$ between an agent i and one of his neighbours j, as the total number of features in which they agree. Thus, we will assume only one opinion vector $\mathbf{V}^i(t)$ representing the decision maker's opinions within each family group.

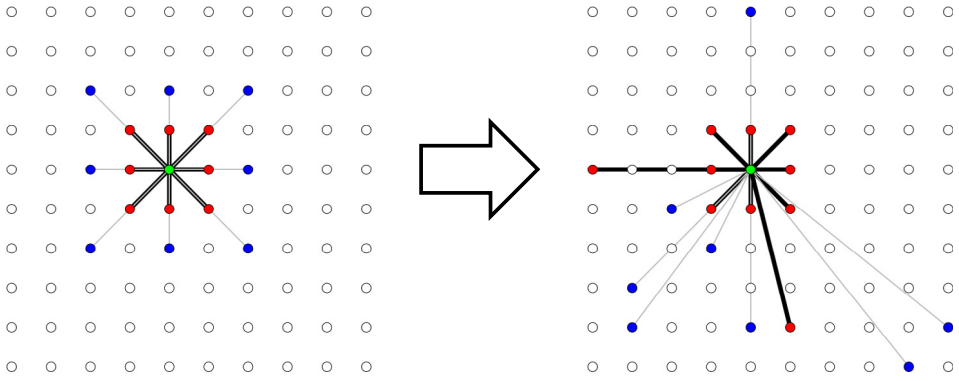
Essentially, the opinion dissemination model consists of social agents performing an adaptation process with asynchronous update. As initial condition, $\mathbf{V}^i(t = 0)$ are assigned uniformly at random in $I^F \times \{0, 1\}$ for all $i \in \{1, \ldots, N\}$. In each time step, a randomly chosen agent i will interact with one of his neighbours j, also chosen uniformly at random, with a probability $P(i \to j)$ proportional to $h(i, j)$. In addition, we define an homophily threshold κ above which the interaction takes place. Then, $P(i \to j)$ is written as:

$$P(i \to j) = \begin{cases} 0 & \text{if} \quad h(i,j) < \kappa \\ \dfrac{h(i,j)}{F+1} & \text{if} \quad h(i,j) \geq \kappa. \end{cases} \tag{1}$$

When the social interaction becomes effective, i adopts the opinion of j for a randomly chosen feature in which they previously disagree, i.e., if $V_n^i(t - \delta t) \neq V_n^j(t - \delta t)$, then $V_n^i(t) = V_n^j(t - \delta t)$. It is worth emphasizing that vaccination opinion stands at equal footing with all the other features. In the context of the model here proposed, we choose $F = 10$ and $\kappa = 2$ to avoid social interactions where the opinion about vaccination was the only coincidence. This last assumption implies that vaccination opinion would be spreading in virtue of cultural affinity between social agents.

In order to consider the action of anti-vaccination movements, we select a fraction of stubborn agents p_{stb} from the total population. Stubborn agents (S_{stub}) preserve their vaccination opinion fixed $V_{F+1}^i(t) = 1$ for all time t, while their opinions about the other features are subject to the adaptation process. Thus, they can influence their neighbours about vaccination behaviour, but cannot be influenced by their neighbours in this respect. At time $t = 0$, they are placed homogeneously at random in the network. Meanwhile, all the other non-stubborn agents are initially vaccinators ($V_{F+1}^i(0) = 0$), because we are interested in studying the impact of anti-vaccination movements in the spread of vaccine rejection behaviour.

We set $p_{stb} = 0.01$ in our simulations assuming that they are very few in the population. Note that higher values of p_{stb} will drive the system to antivaccination consensus for a larger range of traits Q.



(a) Initial. (b) Final.

Fig. 1. Cartoon of initial network and rewiring process Panel (A) shows the initial links for one selected node. The eight nodes corresponding to direct personal contacts are drawn in red. The eight corresponding to non-personal contact are blue. Panel (B) shows the new connections after the homophily – rewiring process as it is explained in the text for this node. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

2.2. Social structure and adaptive network

We model a structured population through a network representation, where nodes and edges are respectively associated to social agents and their social ties. In this context, a social agent may represent a family or an individual agent. Here we refer to a social agent as a family composed by immune adults who must decide about vaccination, or not, of their only son or daughter. Thus, the social ties account for all the social interactions among the members of different households. It is important to distinguish those personal interactions by which an infectious disease could be transmitted, from those which, although having impact on individuals' opinions, do not imply a direct personal interaction. With this in mind, we start by considering two classes of social edges: (a) direct personal and (b) opinion-exchange non-personal edges. Personal edges come from strong, frequent or close contacts implying spatial proximity. They allow not only opinions exchange but also the spread of an infectious disease. Some examples are family ties, or those to close friends, coworkers or neighbours. On the other side, non-personal opinion-exchange edges may represent weak, distant or indirect relations, such as friendship relations in online social networks or "following" relationships in micro-blogging platforms. They represent those social contacts that, in spite of not having spatial proximity, are able to disseminate opinions. As a consequence, the social structure can be represented as two entangled sub-networks, one containing all the direct personal ties, while the other one considering exclusively non-personal opinion-exchange ties.

In order to simplify the representation, we initially assume a square network with $N = 2500$ nodes (50×50 square network) and with homogeneous degree $k = 16$ as is shown in Fig. 1. As can we see, each node has initially $k_p = 8$ neighbours corresponding to direct personal contacts and $k_o = 8$ non-personal contacts.

But social ties are not defined by physical distance neither constrained by it, specially in time of virtual social networks. One of the mechanism by which two individuals can influence each other is because people that share features in common tends to became closer [26]. This mechanism is called *homophily* [27,28] in social sciences and is a powerful tool that shapes social networks. In this work, we implement a mechanism called *linking by homophily* which assumes that each social agent performs preferential connections, in an *active adaptation* process guided by his degree of homophily with the other social agents.

This adaptation mechanism is implemented by an edge rewiring process which comprises two alternative paths depending on whether the chosen edge is personal or non-personal. Both cases are outlined below, in what follows i represents the source agent and j the target agent:

i. Non-personal edge $l(i, j)$: i will attempt to rewire the edge to another agent k chosen uniformly at random (supposing that k was not previously linked to i). If $P(i \to j) < P(i \to k)$, the rewiring will be accepted, otherwise, it will be refused and substituted by an opinion adaptation attempt between i and j as was described in Section 2.1.
ii. Re-wireable personal edge $l(i, j)$: i will attempt a rewiring step to another agent k chosen between their non-personal neighbours. If $P(i \to j) < P(i \to k)$, the edge $l(i, k)$ becomes a personal one, whereas $l(i, j)$ also changes its status becoming a non-personal edge.

These last prescriptions preserve the total number of personal and non-personal edges and, as a direct consequence, do not alter the average degrees $\langle k_o \rangle$ and $\langle k_p \rangle$. Furthermore, the particular prescription for rewiring of personal edges is aimed to reproduce what we expect in real situations. That means that non-personal relations help to develop trust between social agents and, later on, might become personals.

Table 1
Transition probabilities for SEIR measles model.

State transition	Probability
$S \to E$	$1 - \left(1 - \delta t\ c/\langle k_p \rangle\right)^{z(i)}$
$E \to I$	$\nu_E \delta t$
$I \to R$	$\nu_I \delta t$

This adaptive feature of social networks make an important distinction between personal and non-personal edges. By definition, personal edges are robust and, then, hardly prone to active adaptation, so only a small fraction of personal edges (chosen at $t = 0$ with probability p_{pc}) can be rewired. In contrast, non-personal contacts are essentially volatile and therefore we assume that all non-personal contacts can be rewired.

The subgraph G_{pers}, defined only by personal contacts, should be more robust than the corresponding non-personal subgraph, but it is also desirable that satisfies *small-world* conditions [29] as was observed in [30]. Here, a close proximity interaction human network, relevant for infectious disease transmission, was experimentally obtained. In our simulations, small-world properties can be achieved because personal edges are rewired with probability $p_{pc} = 0.1$, given rise to long range connections as in [31]. An example is sketched in panel (B) of Fig. 1.

2.3. Public health intervention: external field

The public health intervention in the dissemination of vaccination behaviour can be represented in the form of an external field ϕ. All the social agents are exposed to ϕ, but in contrast with the vector field proposed in previous works [32–34], here it only acts as a bias for vaccinator-to-non-vaccinator transition. In other words, ϕ represents a public health information campaign in order to prevent vaccinator-to-non-vaccinator transitions. Thus, ϕ is involved in the opinion dissemination process when a vaccinator source agent i has an effective opinion interaction with a non-vaccinator target j, and vaccination behaviour is chosen for interaction. In this case, if $V^i_{F+1}(t) = 0$ and $V^j_{F+1}(t) = 1$, the resulting social imitation process is:

$$V^i_{F+1}(t + \delta t) = \begin{cases} V^j_{F+1}(t) & \text{with probability} \quad (1 - \phi) \\ V^i_{F+1}(t) & \text{with probability} \quad \phi. \end{cases} \tag{2}$$

As it is clear from Eq. (2), the classical social imitation process is recovered by replacing $\phi = 0$. In Section 2.5 could be found a detailed explanation of how the external field intervenes in the dynamical process (Please notice that this implementation of the external field is applied to a system whose initial condition is composed by all pro-vaccination agents, besides the already mentioned stubborn ones).

2.4. Epidemiological model description

Measles is a viral infectious disease of the respiratory system. It is a highly contagious airborne disease mainly transmitted by close person-to-person contact through Flügge droplets. Also it is considered one of the leading causes of death among young children [35], although it can be contracted at any age by individuals who had not been immunized by vaccination or previous contagion.

In this work, we have modelled the dynamics of measles by means of a simple stochastic compartmental model, in terms of the variables describing the possible states that an individual can go through: susceptible (S), exposed (E), infectious (I) and recovered (R). Only those individuals in infectious state are able to transmit the disease, while recovered individuals acquire permanent immunity. We assume a constant population N, i.e. without demographic effects, with a social network structure as described in the main text. Our susceptible population are children because we assume immunized parents. In addition, we conjecture that measles spreads by personal contacts only, and these kind of contacts between children are propitiated mainly by their parents, i.e. young children have limited autonomy.

The possible transition probabilities between states are defined in Table 1. Particular attention deserves the infection probability ($P(S \to E)$) given that we are considering a contact pattern described by the underlying social network, where $z(i)$ corresponds to the number of infectious near-neighbours for the agent i. Thus, $P(S \to E) = 1 - \left(1 - \delta t\ c/\langle k_p \rangle\right)^{z(i)}$ takes a different value for each agent i, being $c/\langle k \rangle$ the mean contagion probability per edge (we consider $c = 2.8$, compatible with known basic reproductive ratio for measles, and $\langle k_p \rangle = 8$ the average degree of personal neighbours for our social network).

In Table 1, ν_E and ν_I are the transition rates from E to I state, and from I to R state, respectively. Furthermore, transition rates are related with the mean time spent in exposed ($T_E = 8$ days, $\nu_E = 1/T_E$) and infectious ($T_I = 8$ days, $\nu_I = 1/T_I$) states [36].

Let S_t, E_t, I_t and R_t being the populations of susceptible, exposed, infectious and recovered agents at time t, respectively. Thus, the population of each state is updated at each time step by the following algorithmic prescriptions:

1. Each susceptible agent $i \in S_t$ gets exposed to measles, or not, with the probability given in Table 1 by only considering those infectious agents j in his neighbourhood with $j \in I_t$. The susceptible population is updated for the next time step: $S_{t+\delta t}$.

2. Each exposed agent $i \in E_t$ gets infectious with probability $\nu_E \delta t$. The exposed population is updated for the next time step: $E_{t+\delta t}$.
3. Each infected agent $i \in I_t$ gets recovered with probability $\nu_I \delta t$. The infected population is updated for the next time step: $I_{t+\delta t}$.
4. Update time $t = t + \delta t$ and go to item 1 until $E_t + I_t = 0$.

We choose $\delta t = 1/2$ day for all our simulations.

2.5. The complete model

Here we gather all previous processes in the final algorithmic structure. We begin with all social agents placed on the initially regular social network. Initially, the vector states $\mathbf{V}^i(t = 0)$ are assigned uniformly at random for all $i \in \{1, \ldots, N\}$, with the exception of vaccination behaviour which is initially set at $V^i_{F+1}(t = 0) = 0$, unless the agent i is a stubborn agent ($i \in S_{stub}$) for which $V^i_{F+1}(t = 0) = 1$ at all time t. As we explained in Section 2.2, we distinguish between rewireable and non-rewireable edges. Non-personal and a small fraction of personal edges (determined at $t = 0$ with probability p_{pc}) are re-wireable. The others are non-rewireables.

The algorithm proceeds as follows:

1. A random source agent i is selected uniformly at random, and a target agent j is selected between the neighbours of i.
   i. If i and j are joined by a re-wireable edge: i follows the prescription of the edge rewiring process described in Social Structure and Adaptive Network Section 2.2.
   ii. If i and j are joined by a personal non-rewireable edge: i follows the opinion adaptation process described in Opinion dissemination model 2.1. If $i \in S_{stub}$ (is a stubborn agent) then it cannot adapt their vaccination opinion.
2. Action of the external field: if $\phi > 0$ and the vaccination opinion of i has changed in previous steps from $V^i_{F+1}(t-1) = 0$ to $V^i_{F+1}(t) = 1$ by the opinion adaptation process, then i becomes vaccinator again ($V^i_{F+1}(t) = 0$) with probability ϕ. In other words, the vaccinator-to-non-vaccinator transition is reverted with probability ϕ.
3. Stop condition: the algorithm stops when the system reaches a metastable configuration (transitions are not longer possible), or a partially frozen configuration without considering small intermittent fluctuations in the vaccination opinion due to the action of stubborn agents (frozen configuration for all features, except vaccination opinion).
4. $t = t + \delta t$. Return to step 1.

Once the social dynamics has finished, we run the epidemiological model as described in Epidemiological model description Section 2.4, in order to analyse and characterize the occurrence of outbreaks.

3. Results

We organize the analysis of the dynamics of the model in three sections:

- The dynamics of the social structure and the effects of the public health campaign,
- the dynamics of the epidemic over the resulting social networks
- the analysis of resulting outbreaks of epidemics in scenarios of 95% of vaccination coverage.

3.1. Analysis of the adaptive social structure and the effects of public health campaign

The first step is to obtain a complete characterization of collective properties of the social model as a function of the main parameters. Our main observable is the amount of unvaccinated individuals, which are the part of the population able to be infected. But also it is important to see how clustered they are, In Fig. 2, we show the dependence of the total unvaccinated population (Panel A), as well as the size of the largest cluster (Panel B) with q for three different values of the external field ϕ. The similarity between these plots suggest that the unvaccinated agents are largely clustered. Also, it can be appreciated that a moderate public campaign promoting vaccination (in the way it is implemented in this model) has a strong impact at the level of individuals' opinion, consequently resulting in a large vaccine coverage.

The original Axelrod's model displays a non-equilibrium phase transition between a mono-cultural (consensus state) and a fully multi-cultural polarization situation [21]. Between these phases, there is a critical region where macroscopic domains with different cultures coexist. In this case, the cultural polarization phase is interpreted as a pro-vaccination consensus forced by the initial condition. Given the action of anti-vaccination stubborn agents, the absorbing state of cultural consensus corresponds to a totally non-vaccinated population, as can be seen in Fig. 2. As the number of traits q increase, the system evolves towards an asymptotic state where non-vaccinator behaviour is no longer dominant and cluster of different cultures coexist. In the absence of external field, this transition takes place in the range of q from 100 to 400 where the richest scenario where different sized clusters are present and non-vaccinator and pro-vaccinator clusters coexist. Hereafter, we set $q = 170$ in order to be close to this critical region

The second step is to understand how many unvaccinated agents do we have as a function of the external field ϕ for $q = 170$ and how they form groups in the underlying social network. In Fig. 3 we plot the histograms of unvaccinated agents

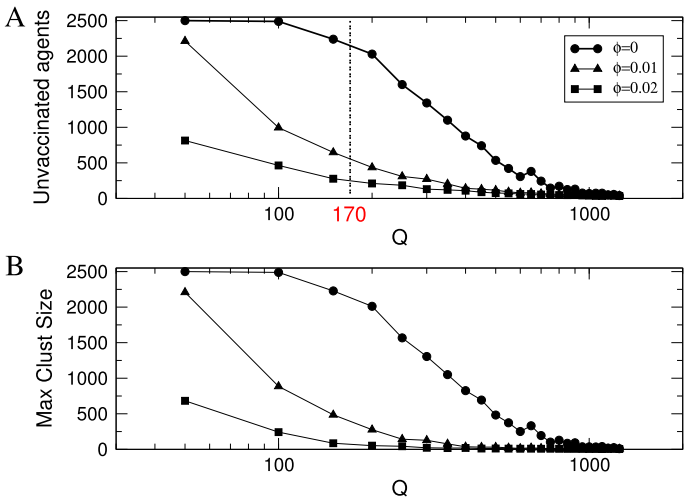


Fig. 2. Unvaccinated agents population as a function of the number of traits q for three different values of external field ϕ. Panel (A) shows the average population of unvaccinated over 500 runs and Panel (B) the size of the largest cluster as a function of q for $\phi = 0, 0.01, 0.02$, under the influence of an anti-vaccination movement which is represented by an 1% of total population. The similarity between these plots suggest that the unvaccinated agents form almost one cluster. Dotted lines corresponds to $q = 170$ which is the value adopted for simulations in next plots.

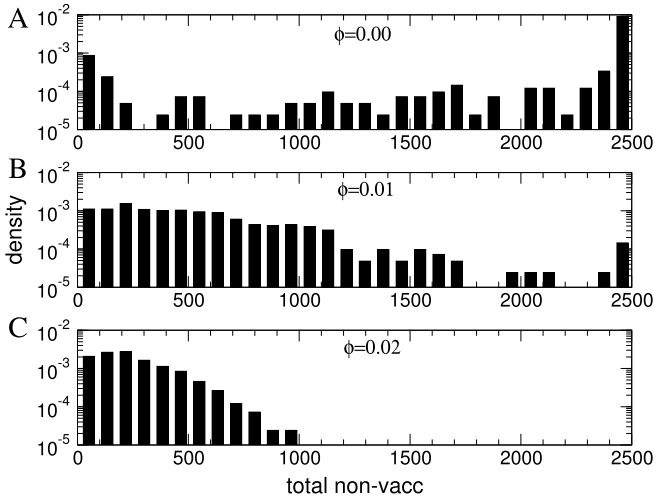


Fig. 3. Histograms of unvaccinated agents under increasing external field strength. They were obtained performing 500 runs with $q = 170$ and 1% of stubborn agents located at the same network sites for each run. The mitigating effect for increasing strength of the external field ϕ, with $\phi = 0.00, 0.01$ and 0.02, is shown in the panels A, B and C.

under increasing strength of public health champaign (ϕ). We observe a transition from U-shaped for $\phi = 0$, to unimodal short-tailed for the case corresponding to an applied external field $\phi = 0.02$ showing the strong mitigating effect induced by ϕ.

Given a population of unvaccinated agents, a particularly relevant aspect is to know how they are distributed over the network. Fig. 4 shows the cluster size distribution corresponding to the unvaccinated distributions of Fig. 3. In this context, a cluster is defined by considering the subgraph G_{pers} involving only direct personal edges, i.e., those through which an infectious disease could be transmitted. The cluster size distributions are compared with those obtained from the equivalent percolation processes over the initial social network, i.e., by distributing uniformly at random the same number of elements constituting the clusters over the initial network. In this initial social network, the structural changes imposed by the active adaptation process has not yet started. As can be seen in Fig. 4, we found differences with respect to their associated percolation results, in particular for $\phi > 0$ and for large clusters. In order to quantify these comparisons we also show in the right panels of Fig. 4 the values of the t-statistic $t_S(i)$ for each bin b, defined as:

$$t_S(b) = \frac{\rho(b) - \hat{\rho}_{perco}(b)}{\hat{\sigma}_{perco}(b)} \tag{3}$$

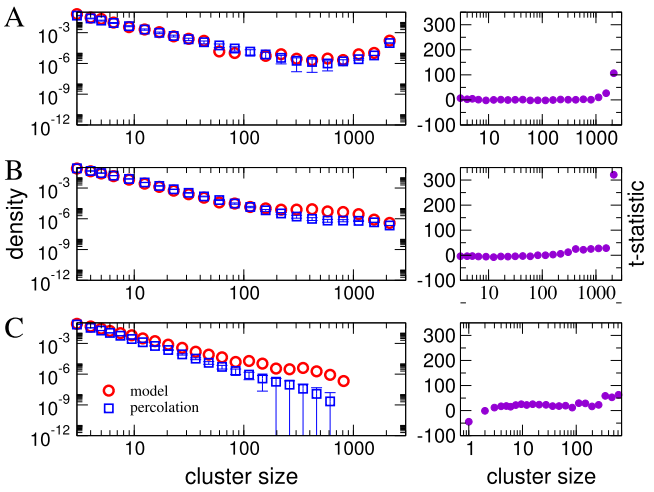


Fig. 4. (Colour online) Cluster size distributions of unvaccinated agents. Cluster size distributions for (A) $\phi = 0.00$, (B) $\phi = 0.01$, and (C) $\phi = 0.02$. Each distribution comprises the accumulated results of 500 runs of the model with $q = 170$ and the indicated value of ϕ. The comparison with percolation is presented and also quantified through the t-statistic (see Eq. (3) in the main text) plotted in the right panels.

being $\rho(b)$ the density corresponding to the bin b given by the model, while $\hat{\rho}_{perco}(b)$ and $\hat{\sigma}_{perco}(b)$ are, respectively, the mean and the standard deviation of the sample given by the percolation realizations, also corresponding to the bin b. Fig. 4 reveals that the bigger values of $t_S(b)$ are mainly obtained for large cluster sizes. This result unveils the tendency towards clusterization of unvaccinated agents in the context of the proposed model for the spread of vaccination behaviour.

3.2. Epidemic dynamics and its relation with underlying social structures

In the previous section we have discussed the dynamics of the underlying social process where the anti-vaccination movement creates clusters of unvaccinated individuals. As we can observe, the risk of contagion of an unvaccinated individual is much higher if he/she is surrounded of unvaccinated rather than vaccinated neighbours.

The goal of this section is to analyse how the dynamics of measles using the SEIR model described in Section 2.4 produces outbreaks of different sizes according to the underlying topology of social personal contacts. The question we would like to answer is: Does the clusterization induced by cultural affinity with stubborns anti-vaccine individuals produce larger-sized outbreaks than those expected if no social process has taken place? Fig. 5 compares the mean outbreak size of measles as a function of the unvaccinated population obtained at the end of the opinion adaptation process, with those which would be obtained if the same unvaccinated population were randomly distributed over the initial personal social network, in what we called *the percolation approach*.

These results are very important because they show the relevance of the social process: at the same amount of unvaccinated individuals, the mean outbreak size is much higher if they are clustered due to the cultural adaptive process described above (red circles in Fig. 5, colour online) than if they were distributed at random in the initial personal social network (blue squares in Fig. 5, colour online).

3.3. Scenarios with 95% of vaccination coverages

Vaccine coverage usually reaches higher levels in developed countries with more vaccine availability. In particular, recommendations of WHO suggest 95% vaccine coverage for the case of MMR vaccine in order to eradicate measles. Is this enough in order to eradicate measles? Or again, the social process induced by homophily also produces clusters of non-vaccinated individuals which promote the occurrence of large outbreaks of measles. Thus, we have repeated our previous analysis but now filtering those realizations leading to a final immunized population between 94.6% to 95.4% (equivalent to an unvaccinated population between 115 to 135 individuals over a total population of 2500) aiming to be close to WHO recommendation.

First, we investigate the cluster size distribution due to the dynamical social process. Fig. 6 shows that the amount of non-vaccinated individuals forms larger clusters due to the underlying social process than if they were located at random in the initial personal social network (percolation approach). In this particular case, the effects of the clustering of unvaccinated are magnified respect the percolation approach when compared with previous results (Fig. 4). This magnifying effect is rooted in the sub-critical percolation regime imposed by the small unvaccinated population.

Then, we analyse the dynamics of measles in this scenario. Fig. 7 clearly shows that the probability of obtaining large outbreaks is higher than in the percolation approach. The outbreak sizes range up to 100 when the social process is driven by the adaptive dynamics meanwhile they are not larger than 10 in the percolation approach. This result confirms the previous statement linking the clustering of unvaccinated with higher outbreak probability and larger mean outbreak size.

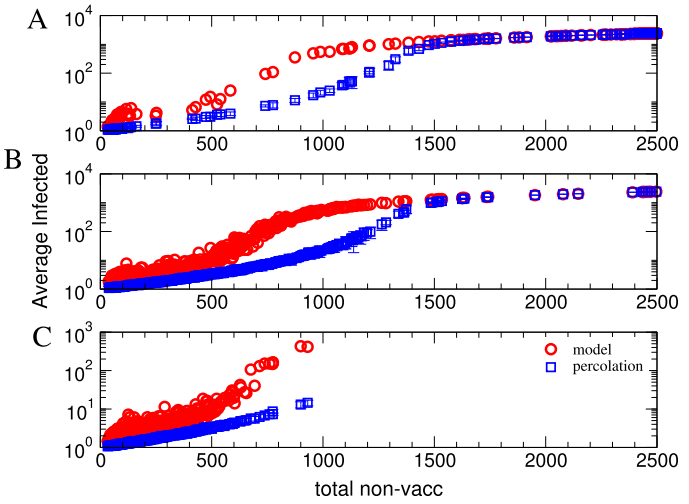


Fig. 5. (Colour online) Mean outbreak size as function of unvaccinated population. Mean outbreak sizes for both opinion dissemination model ($q = 170$) were obtained averaging over 10, 000 realizations of measles spreading model for each unvaccinated population for different values of external field: (A) $\phi = 0.00$, (B) $\phi = 0.01$, (C) $\phi = 0.02$. Results are compared with those obtained through the percolation approach.

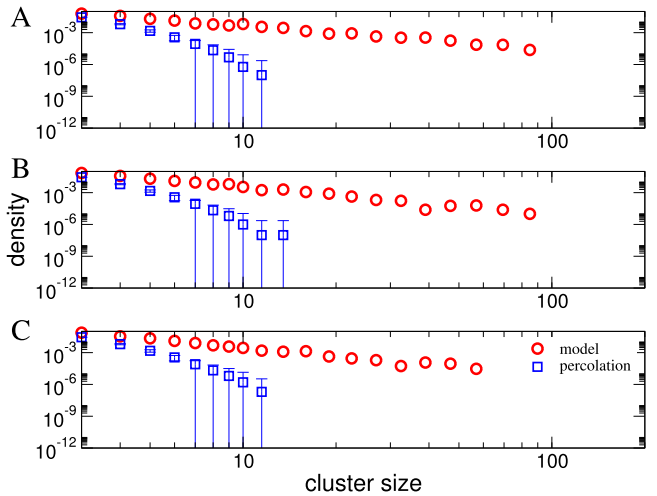


Fig. 6. (Colour online) Cluster size distributions for 95% vaccination coverage. Cluster size distributions restricted to about 5% unvaccinated population, for different values of external field:(A) $\phi = 0.00$, (B) $\phi = 0.01$, and (C) $\phi = 0.02$ compared with the percolation approach results at the same unvaccinated population.

4. Summary and conclusions

In this work, we developed a model where the vaccination opinion is disseminated between social agents as one of their cultural features, suggesting a mechanism where dissemination of culture explains both the clustering and total population of non-vaccinated individuals in a natural way.

We study the impact of a small anti-vaccination movement and show that vaccine refusal spreads more efficiently in the absence of external intervention. On the other hand, the impact of public health campaigns, here represented by the external field ϕ, proved to be very efficient by increasing the average vaccine coverage (i.e. reducing unvaccinated population) and, then, reducing the outbreak probability and its final size.

A strong clustering of unvaccinated agents is apparent when comparing cluster size distributions with those yielded by percolation approach in which unvaccinated agents are randomly placed over the initial networks. As a direct consequence of this, we observe a marked increase in the outbreak size and its probability of occurrence. This clustering effect has deeper consequences on the expected herd immunity, even though the recommended vaccine coverage goal (95% for MMR vaccine) was reached, which suggests that vaccination goals should also consider the chance of clustering of unvaccinated agents. Moreover, the clustering effect is magnified under high vaccination coverage regime, in agreement with previous findings [12].

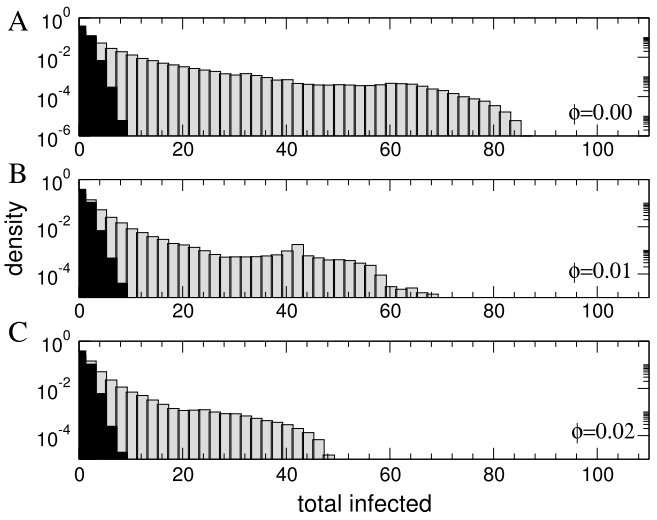


Fig. 7. Outbreak size distributions for 95% vaccination coverage. Simulations of the model results for ((A) $\phi = 0.00$, (B) and $\phi = 0.01$, (C) $\phi = 0.02$, in grey) compared with the percolation approach results at the same unvaccinated population (in black). Each histogram comprises 10, 000 simulations of the measles spreading model.

For future work, it would be interesting to perform runs for different fractions of stubborn agents in order to find the intensity of the external field ϕ that mitigates their effects. Another perspective for the models here introduced would be to study the impact of the initial distribution of stubborn agents (placed at random in this paper) on the final cluster size distribution for all non-vaccinated agents in the network. On the other hand, here we assume a situation involving social agents without perception of the infection risk due to the low initial incidence of measles. However, we could increase the tendency to vaccination in social agents when they are faced to the imminence of an outbreak [37,38]. Relative to the physical aspects of the phase transition in both models, a thorough study of the position and extension of the critical region as a function of the number of features F and total population N remains to be explored.

As final remarks, on one side, our results support the hypothesis stating that vaccine availability is not enough to prevent measles outbreaks if it is not complemented by massive consciousness-raising campaigns, conducted in order to undermine the harmful action of anti-vaccination groups. On the other side, the clustering effect should be extensively studied in future experimental tests, as some authors have already begun to do for the case of influenza [14,39], in order to provide new insight that effectively lead us to the ultimate eradication of measles.

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