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Homophily in a multi-group vaccination game and its impact on disease dynamics

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Metapopulation models are among the most frequently used approaches to address spatial heterogeneity in infectious disease modelling. While researchers typically consider the role of heterogeneity in disease transmission by dividing the population into subgroups according to levels of social activity, susceptibility or perceived risk of infection, the role of heterogeneity in vaccination behaviour has received less attention. The tendency to interact with others who share similar beliefs (homophily) can further reinforce such heterogeneity, leading to clustered vaccine sentiments in the population. In this paper, we develop a multi-group vaccination game model coupled with an SIRV epidemic model and examine how interactions among individuals within and between groups affect both vaccination behaviour and disease dynamics. We find that in a three-group system with extremely pro- and anti-vaccine groups, each group can converge to a partially pro-vaccine equilibrium, causing population-level opinion polarization that can persist despite the severity of the disease. Our results highlight the dual role of homophily, offer insights into the design of effective public health interventions and demonstrate the potential of a multi-group framework in real-world disease modelling.

1. Introduction

Vaccination has been one of the most effective ways to control the spread of infectious diseases, as it reduces outbreak size through immunity to individuals. Before the measles vaccine was introduced in the United Kingdom (UK) in 1968, major outbreaks occurred in the UK every 1–2 years in a non-periodic fashion, causing hundreds of deaths annually [1,2]. More complex patterns were observed in some other countries [3–5]. With the help of mass vaccination programmes, measles epidemics became less frequent and smaller in scale. However, in 1998, a series of fraudulent papers sparked widespread fear about a link between the MMR (measles, mumps and rubella) vaccine and autism [6,7]. This led to a decline in vaccine uptake and the resurgence of measles in the UK, highlighting how the success of vaccination programmes greatly depends on people's willingness to get vaccinated.

Vaccination is not just a pharmaceutical intervention but also a strategic decision. People weigh the risk of side effects from vaccines against the often larger risk of infection, which can cause severe illness and long-term health complications. As a result, vaccination is often a more attractive strategy during a pandemic, but when a disease becomes rare, it may seem less appealing. Herd immunity (HI) further complicates this decision. A common misinterpretation of HI is thinking that it eliminates all risk of infection. In fact, HI only implies that the disease cannot persist in a highly immunized population, although localized, sporadic infections can still occur. Since disease elimination does not require everyone to be vaccinated, HI creates opportunities for free riders as the

incentive to get vaccinated decreases. However, if too many people rely on others for protection, population immunity may fall below the threshold needed to prevent outbreaks.

Integrating vaccination behaviour into epidemic models has become a common approach for studying the spread of vaccine-preventable diseases [8–10]. These models usually include two interconnected layers of dynamics: a biological layer that describes how the disease is transmitted in a population and a social layer that captures how individuals imitate others and adjust their vaccination strategies [11]. The biological layer is typically represented using compartmental models, such as the classic SIR (Susceptible–Infected–Recovered) framework or its variations. In these models, individuals are grouped into compartments based on their infection status, and differential equations describe the rates at which individuals move between compartments [12]. The social layer, on the other hand, can be modelled using evolutionary game theory [13–15]. It helps explain vaccination behaviour by treating vaccination as a ‘game’, where individuals make decisions based on both self-interests as well as the choices of others, and captures how these decisions evolve over time.

A substantial body of research explores behaviour–disease dynamics in populations assuming homogeneous mixing [16–22]. This assumption simplifies the model structure, allowing analysis of equilibrium points, stability conditions and threshold parameters like the basic reproduction number R_0 . These models also give insights into the fundamental interplay between behaviour and disease dynamics. However, such an assumption overlooks important features of real-world populations, such as geographic and social structures that could greatly affect the spread of disease and opinions [23–25]. To consider heterogeneity in populations, researchers often apply complex networks, where individuals are represented as nodes, with edges in different layers denoting interactions that enable disease transmission and social contacts [11,24,26–30].

Patch models provide another useful way to capture population heterogeneity by dividing individuals into distinct subgroups [31]. Unlike complex networks that emphasize individual-level contact patterns to trace transmission, patch models focus on group-level interactions to capture dynamics between subpopulations and are easier to analyse, particularly when detailed behavioural data are unavailable. A common practice is to group people by age to reflect differences in social contact patterns, infection susceptibility and vaccine responses, informing targeted vaccination strategies for seasonal influenza and COVID-19 [32–34]. Researchers also assign different levels of risk perception [35–38] and vaccine efficacy [39,40] to subgroups to account for diverse characteristics of the population. However, existing models typically consider heterogeneity only on the biological layer and emphasize its epidemiological consequences, while its effect on the social layer, especially its role in shaping the time evolution of vaccine support, is often ignored. In reality, vaccine sentiments tend to cluster in social and geographic space, creating communities with high or low vaccine acceptance [41,42]. This suggests that a single variable cannot adequately capture population-wide vaccine sentiment. Early studies such as the pioneering work by May & Anderson [43] have recognized the importance of considering heterogeneity in vaccination coverage, showing that assigning different vaccination levels in subpopulations can substantially affect disease transmission. Yet such heterogeneity is imposed as a top-down policy design, rather than emerging from bottom-up social dynamics. Moreover, with the widespread use of social media and online messaging tools, people have more channels to access information and the speed at which they acquire it has greatly increased. People who spend more time online may be more exposed to vaccine-related information, whether accurate or misleading [44–46], while those with limited or no Internet use may rely more on traditional social contacts. As a result, imitation dynamics in vaccination behaviour can vary significantly across groups. These observations highlight the need to account for heterogeneity at the group level in models of vaccination behaviour.

In particular, homophily (or assortativeness, in the language of epidemiological modelling), the tendency for individuals to socially interact with those who share similar views or behaviours [12,47–49], can strongly influence how vaccination strategies or other health behaviours spread in the population [50–53]. A previous study divides the population into social media users and non-users, and explores how homophily and online events affect vaccination behaviour and disease transmission [54]. In this study, we extend the model to more than two groups to examine how small, highly committed pro- and anti-vaccine minority groups interact with the broader majority of the population. Specifically, we ask whether the majority acts as a swing group that shifts under the influence of these extreme groups, or whether the minority groups gradually conform to the prevailing views of the majority over time.

2. Methods

We propose a coupled behaviour–disease model for a paediatric infection such as measles, where vaccination is typically administered at birth [18,21]. On the biological (or disease) layer, we assume homogeneous mixing across the entire population, meaning that all individuals are equally likely to come into physical contact with one another. Disease dynamics follow a standard susceptible–infected–recovered–vaccinated (SIRV) framework with natural births and deaths. However, on the social (or behavioural) layer, we divide the population into n distinct groups of various sizes but assume well-mixing within each subgroup. In modern populations, disease and opinion spread can operate on fundamentally different spatial scales: while infectious disease transmission requires physical contact, opinion change increasingly occurs online through social media and other digital platforms, connecting individuals across wide geographical distances [55]. In the post-COVID-19 era, changes in lifestyle such as reduced travel and increased remote working have altered the dynamics of both disease spread and information flow. As a result, even when considering a small, homogeneous community from a disease transmission perspective, social heterogeneity remains significant. While heterogeneity on the biological layer may be negligible in such cases, differences in information dissemination across social groups must be considered.

On the biological layer, individuals are classified as susceptible (S), infected (I), recovered (R) or vaccinated (V) based on their health status. On the social layer, parents of newborn children act as decision makers in the ‘vaccination game’ [17,18]. Each parent belongs to one of the n social groups and is either pro-vaccine (P) or anti-vaccine (A). We assume that parents strictly adhere to their vaccination strategy and that vaccinated children would gain lifelong immunity. At each time point, the proportion of vaccinated newborns matches the proportion of pro-vaccine parents in the entire population (i.e. the weighted average across all social groups). Effectively vaccinated children enter the system as vaccinated individuals, whereas the rest enter as susceptible.

Let x_i be the proportion of pro-vaccine parents in group i , and thus the proportion of anti-vaccine parents in the same group is $1 - x_i$. The payoffs for adopting a pro-vaccine and an anti-vaccine strategy in group i are $\pi_i(P)$ and $\pi_i(A)$, respectively. $h_i \in [0, 1]$ represents the level of homophily in each group, where higher values indicate stronger homophily, meaning individuals interact less frequently with people from different groups. Said otherwise, the level of homophily is a measure reflecting the preference for socially interacting with individuals of their own group. This has motivated the key concept of preferred mixing in epidemiological modelling [56].

Given that the principal focus of this work is on the ‘pure’ effects of homophily, our multi-group model disregards any other possible sources of heterogeneity. For this reason, we assume that all other parameters related to the diffusion of opinions on vaccination, namely, perceived costs arising from infection or vaccination as well as the strength of social norms, are the same across all social groups. Though easy to include, these additional heterogeneities would make the overall picture less sharp.

To incorporate homophily, we make two additional assumptions: (i) the total amount of learning is fixed; and (ii) increasing the number of groups does not increase the total amount of learning but instead distributes it across the groups. To reflect this, we assign a weight of $[1 + (n - 1)h_i]/n$ for imitation within the group and $(1 - h_i)/n$ for imitation between groups. Given the social learning rate κ , this weighting ensures that the total amount of learning remains constant:

$$\left(\frac{1 + (n - 1)h_i}{n}\right)\kappa + (n - 1)\left(\frac{1 - h_i}{n}\right)\kappa = \kappa.$$

Combining these elements, we obtain the system of ordinary differential equations describing the coupled behaviour–disease dynamics:

$$\frac{dS}{dt} = \mu(1 - \epsilon x) - \beta SI - \mu S, \quad (2.1)$$

$$\frac{dI}{dt} = \beta SI - \gamma I - \mu I, \quad (2.2)$$

$$\frac{dR}{dt} = \gamma I - \mu R, \quad (2.3)$$

$$\frac{dV}{dt} = \mu \epsilon x - \mu V, \quad (2.4)$$

$$\begin{aligned} \frac{dx_i}{dt} = & \left(\frac{1 + (n - 1)h_i}{n}\right) \frac{\kappa N_i}{N} \{x_i(1 - x_i)[\pi_i(P) - \pi_i(A)]\} \\ & + \left(\frac{1 - h_i}{n}\right) \sum_{j \neq i} \frac{\kappa N_j}{N} \{x_j(1 - x_i) \max[\pi_j(P) - \pi_i(A), 0] - (1 - x_j)x_i \max[\pi_j(A) - \pi_i(P), 0]\}. \end{aligned} \quad (2.5)$$

Here, μ denotes the natural birth and death rate, ϵ is the vaccine efficacy, β is the transmission rate of the infection and γ is the recovery rate. Given group sizes N_i and the total population size N , the overall proportion of pro-vaccine individuals (parents) in the population is calculated as $x = \sum_i x_i N_i / N$. κ also accounts for the interaction rate, and the factor N_i / N ensures proportionality to the relative frequency of group i [57]. Since $S + I + R + V = 1$ always holds, the differential equation for R can be eliminated. Details on equation (2.5) and a summary of all parameters used in this system are provided in the electronic supplementary material.

Throughout this study, we assume that the population mainly consists of a large ‘general group’. The remaining individuals who hold strong pro-vaccine or anti-vaccine sentiments are divided into several ‘extreme groups’. To begin, we consider a population with three subgroups ($n = 3$): the general group ($i = 1$), one pro-vaccine group ($i = 2$) and one anti-vaccine group ($i = 3$). In the baseline setting, the general group constitutes 80% of the total population, while each of the two minority groups accounts for 10%. The two minority groups ($i \in \{2, 3\}$) are assumed to exhibit relatively high homophily, reflecting the empirical fact that individuals preferentially interact with others who share similar views, especially in the vaccine debate [58]. For more details on numerical methods and the effects of relative group sizes on model dynamics, see electronic supplementary material.

3. Results

(a) Behavioural dynamics in the absence of diseases

Although global measles eradication has not been achieved and elimination has only been sustained at the national level, in some smaller areas (e.g. cities or counties), measles is extremely rare and can be considered eliminated. Therefore, it is valuable to consider a baseline scenario in which the disease is absent (i.e. $I(0) = 0$), implying that $I(t) = 0$ for all future t . In this case, vaccination dynamics are driven purely by behavioural and social influences.

When a single well-mixed group is assumed on the social layer, the disease-free equilibrium with partial vaccine coverage $x^* = (1 + \omega/\delta)/2$ is always unstable whenever it exists, since the social norm drives the proportion of pro-vaccine individuals either up to 1 or down to 0 [22]. Similarly, if we consider multiple groups with full homophily, each group behaves like an isolated

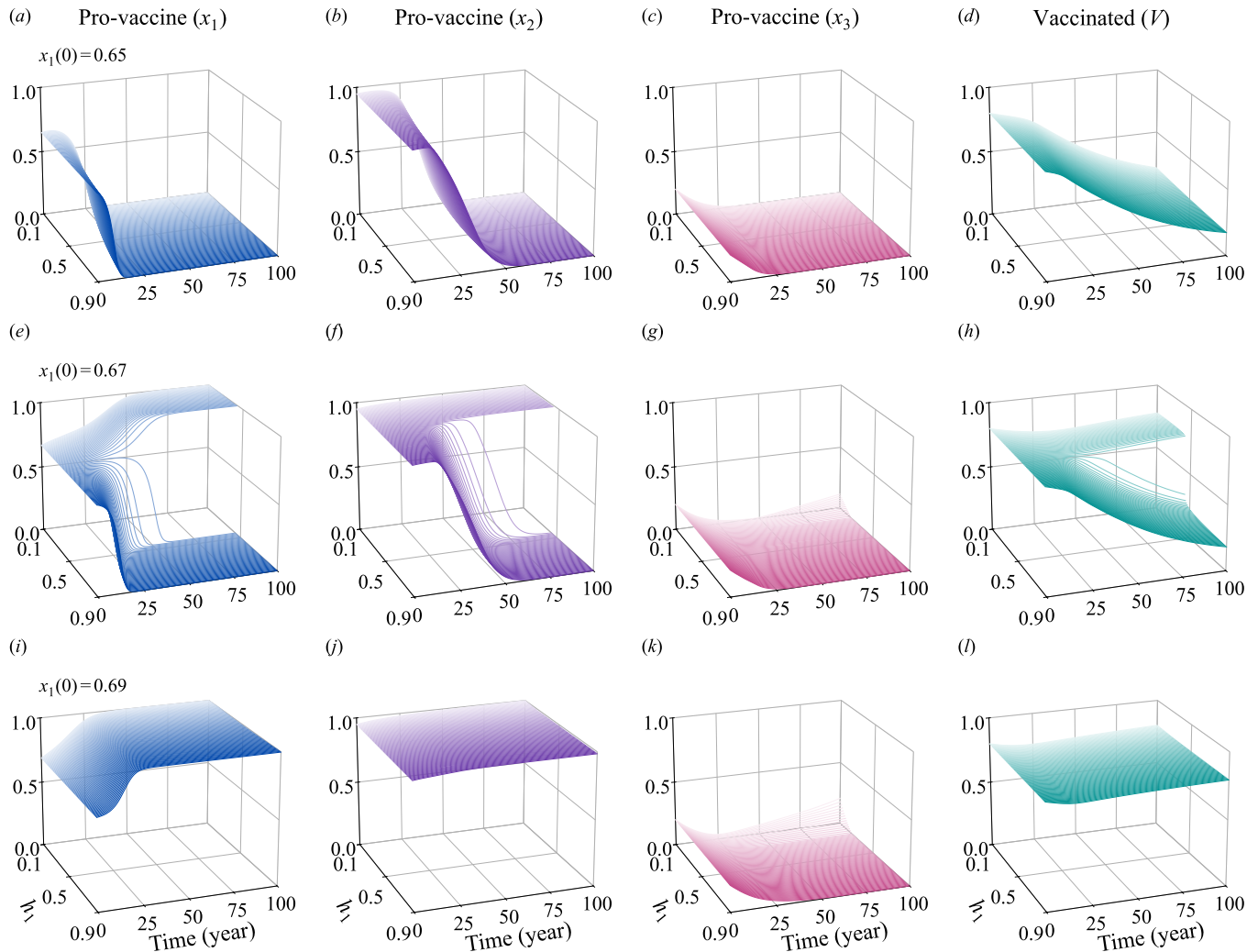


Figure 1. Dynamics of pro-vaccine opinions and vaccine coverage under different levels of homophily in the general group when the disease is absent. Each panel shows the temporal evolution of the proportion of pro-vaccine individuals in (a,e,i) the general group, (b,f,j) the highly pro-vaccine group and (c,g,k) the highly anti-vaccine group, as well as (d,h,l) the overall vaccinated percentage of the entire population. The rows correspond to different initial pro-vaccine percentages in the general group: (a–d) $x_1(0) = 0.65$, (e–h) $x_1(0) = 0.67$ and (i–l) $x_1(0) = 0.69$.

well-mixed population, and the instability of partial vaccine coverage persists within each group. However, when cross-group imitation is allowed, the dynamics become more complex, and the previous threshold condition may no longer hold. If the vaccine support in the out-group is lower than that in the in-group, excessive cross-group interaction may actually hinder vaccine uptake, raising the critical threshold above $(1 + \omega/\delta)/2$. Conversely, if the out-group is highly pro-vaccine, even if the initial support level $x_i < (1 + \omega/\delta)/2$, cross-group imitation may still drive x_i up to 1. On top of this, homophily introduces an additional layer of complexity, as it can either amplify or dampen out-group influence.

Figure 1 illustrates the interplay between initial pro-vaccine percentages and homophily of the majority. We consider two initial pro-vaccine levels in the general group that fall below the critical threshold under full homophily, $(1 + \omega/\delta)/2$. When the opinion flow within the group tends to shift towards anti-vaccine strategies, low homophily, allowing frequent interactions between groups, can temporarily stabilize or even reverse the decline (figure 1a,e). When the initial pro-vaccine level exceeds the critical threshold and the group favours pro-vaccine strategies, low homophily accelerates the approach to full vaccination (figure 1i).

The general group also significantly influences the dynamics of the other two minority groups despite the high homophily levels, since most of the interactions occur with its members (figure 1b,c,f,g,j). We observe that in a high homophily setting, an increase in x_1 fails to raise vaccine support in the highly anti-vaccine group (figure 1k). This is because high homophily reduces the payoff of vaccination, making it a less appealing strategy. The trend in the proportion of effectively vaccinated individuals is also largely driven by the general group, but changes occur gradually rather than abruptly due to relatively low birth and death rates (figure 1d,h,l).

To further investigate how group-specific homophily levels shape long-term vaccination outcomes, we analyse the joint effect of homophily in the two minority groups on the final pro-vaccine proportion (figure 2). We select two initial conditions for x_1 , 0.67 and 0.69, just below and above the threshold $(1 + \omega/\delta)/2$. Lower initial values of x_1 are avoided, as they tend to drive all groups towards a fully anti-vaccine state across almost all combinations of h_2 and h_3 within their targeted range, except when h_2 is very small or becomes unrealistically high. From figure 2a,b, the system consistently converges to a fully pro-vaccine state across all groups when both h_2 and h_3 are small. As h_3 increases, the highly anti-vaccine group becomes increasingly entrenched in vaccine

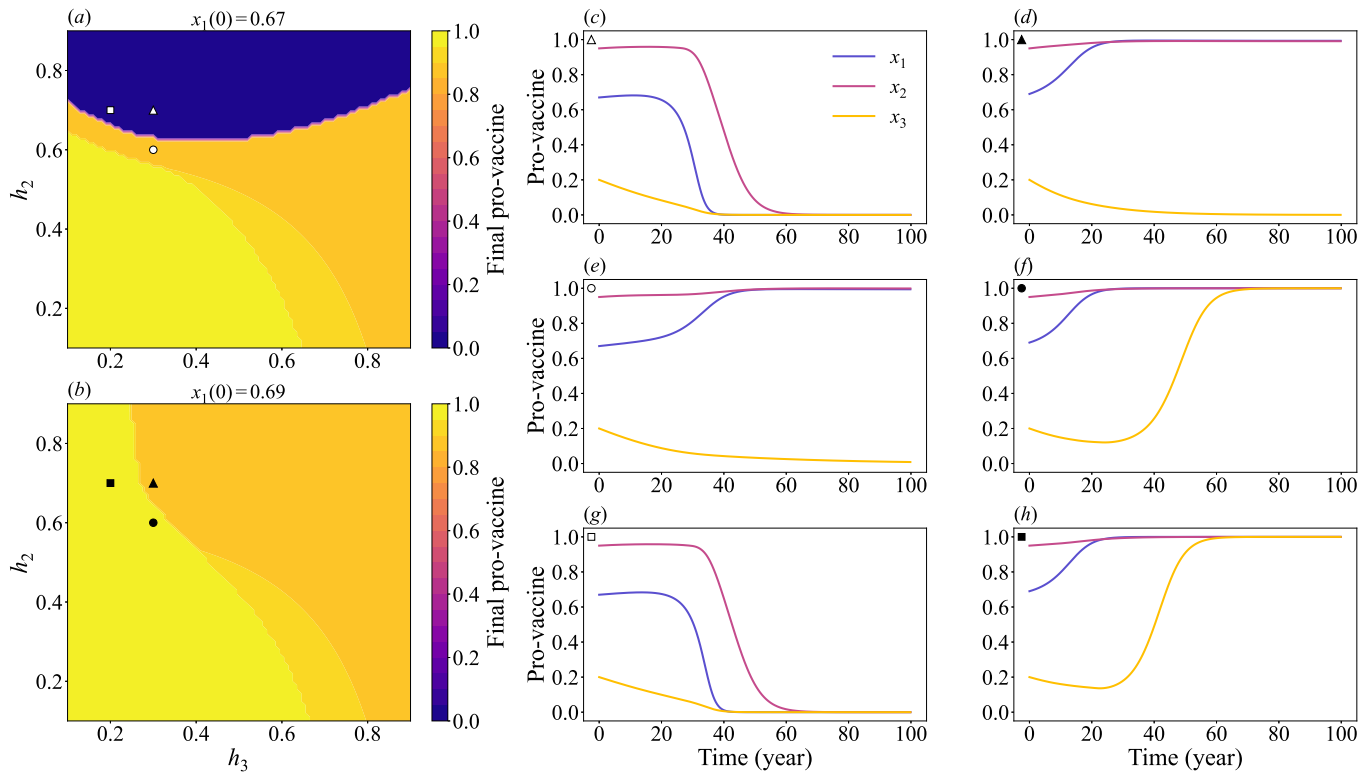


Figure 2. Impact of homophily levels in the minority groups on final pro-vaccine proportion in the entire population when the disease is absent. (a, b) Final pro-vaccine proportion across combinations of homophily levels, with initial pro-vaccine proportion in the general group set to $x_1(0) = 0.67$ and $x_1(0) = 0.69$, respectively. (c–h) Time series plots of pro-vaccine percentages in three groups, corresponding to (c, e, g) the white markers in (a) and (d, f, h) the black markers in (b). The homophily combinations illustrated are: $h = [0.5, 0.7, 0.3]$ (triangle), $h = [0.5, 0.6, 0.3]$ (circle) and $h = [0.5, 0.7, 0.2]$ (square).

refusal, often resulting in a multi-strategy steady state in the population. A rise in h_2 reinforces the pro-vaccine dominance within the group at the beginning, but its long-term trend depends on the general group.

In addition to the influence of group-specific homophily, figure 2a,b also reveal a pronounced sensitivity to initial conditions near the critical threshold: a small increase in $x_1(0)$ from 0.67 to 0.69 significantly expands the region where all groups converge to a fully pro-vaccine equilibrium (yellow), indicating that the requirements to achieve widespread vaccine uptake become less strict. The system can tolerate higher homophily levels in both minority groups while still reaching a favourable outcome. The time series in figure 2c–h further illustrate how such a modest change in initial conditions can qualitatively alter the equilibrium of the system. The system may shift from an anti-vaccine consensus to a multi-strategy state (figure 2c,d) or from a multi-strategy state to a pro-vaccine consensus (figure 2e,f). A more extreme case occurs when the system transitions directly from an anti-vaccine consensus to a pro-vaccine consensus (figure 2g,h). Notably, in figure 2f,h, the non-monotonic trajectories of x_3 suggest that vaccine uptake may temporarily decline before recovering.

(b) Impact of time-varying homophily levels under endemic conditions

In real-world social systems, homophily is hardly a static characteristic. The strength of social ties within and between groups can evolve in response to external factors such as media influence and public health interventions, especially when the disease is present. Social media platforms and search engines often reinforce echo chambers by showing users content similar to their existing views [59], which strengthens group identities and limits exposure to opposing perspectives. Additionally, individuals are more likely to seek social interactions that confirm their beliefs, known as the confirmation bias [60,61]. As a result, homophily often becomes stronger over time. Nevertheless, it can also weaken under certain conditions, such as through educational campaigns by public health agencies and algorithmic adjustments of social media platforms that promote diverse content.

To explore how such temporal changes in social connectivity affect coupled behaviour–disease dynamics, we compare, under endemic conditions, scenarios where the homophily level h_i increases or decreases over the medium/long term against a baseline scenario where homophily remains constant over time. We use the logistic function

$$h_i(t) = \frac{2(h_i^f - h_i^b)}{1 + e^{-\alpha t}} + 2h_i^b - h_i^f,$$

where h_i^b is the baseline value and h_i^f denotes the asymptotic value that h_i approaches in the long term. $\alpha > 0$ controls the rate of change in homophily over time. Whether homophily increases or decreases is determined by the relative values of h_i^b and h_i^f . In the two scenarios with evolving homophily, we consider two distinct values of the time scale of homophily evolution, that is, 20 and 50 years.

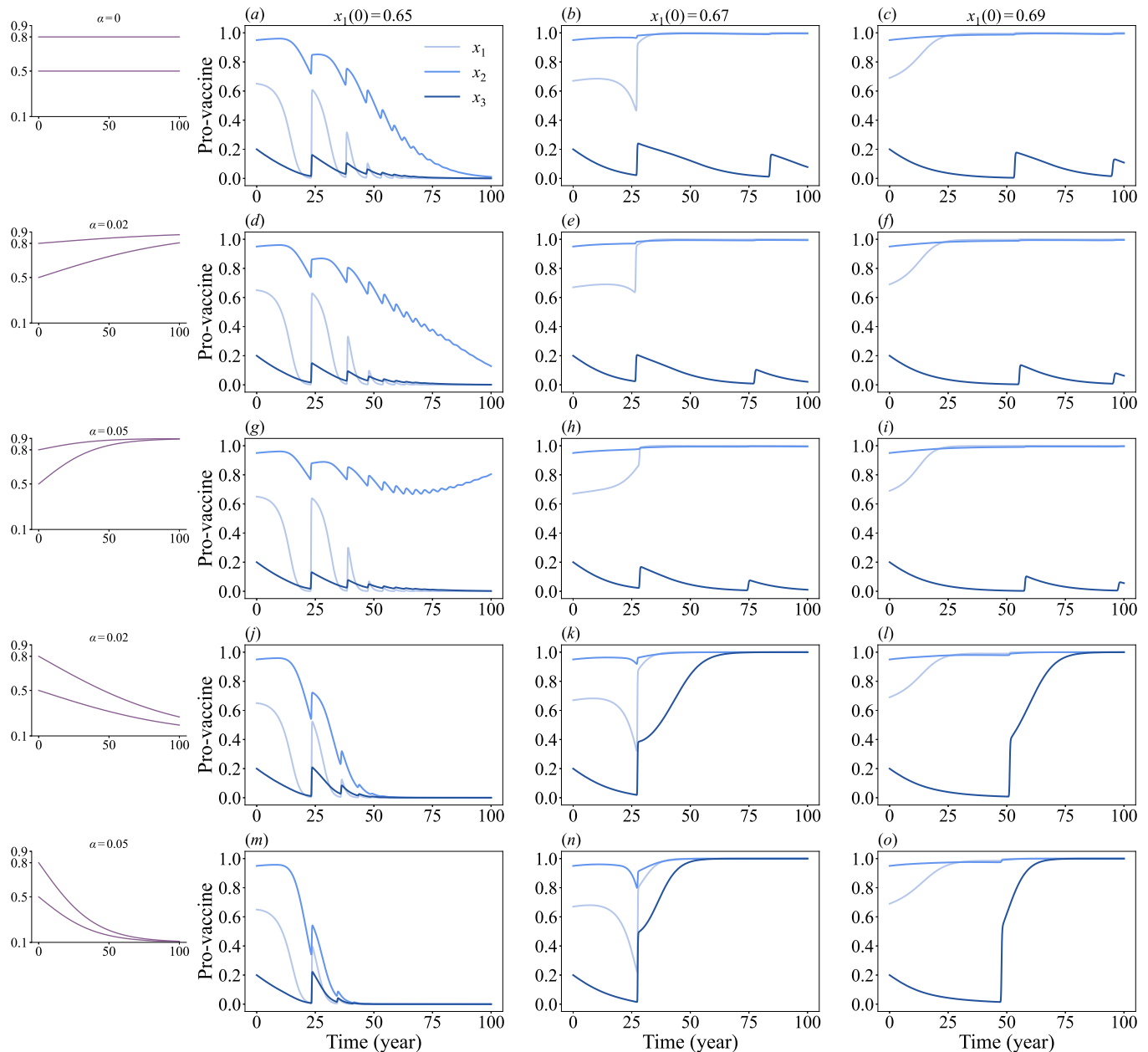


Figure 3. Dynamics of pro-vaccine opinions across three groups under constant and logistically increasing or decreasing homophily levels in the presence of the disease. Each row corresponds to a different rate of change: (a–c) $\alpha = 0$ (constant), (d–f) increasing at $\alpha = 0.02$ /year, (g–i) increasing at $\alpha = 0.05$ /year, (j–l) decreasing at $\alpha = 0.02$ /year and (m–o) decreasing at $\alpha = 0.05$ /year. Each column corresponds to a different initial opinion level in the general group: (a,d,g,j,m) $x_1(0) = 0.65$, (b,e,h,k,n) $x_1(0) = 0.67$ and (c,f,i,l,o) $x_1(0) = 0.69$. The small panels on the left show the time-varying homophily levels for each α . Oscillations in the trajectories represent epidemics (for which graphs are not reported), leading to fast opinion switches over a rather short period of time in each social group.

We begin by examining the baseline case of constant homophily ($\alpha = 0$), shown in figure 3a–c. The dynamics observed in the presence of disease (figure 3) resemble the disease-free scenario (figure 2), where the population can reach an anti-vaccine consensus (figure 3a) or become more polarized (figure 3b,c) in the long run. However, the trajectories in figure 3 exhibit oscillations due to repeated disease outbreaks that cause sudden boosts in pro-vaccine sentiments. Another notable difference is that, when $x_1(0) = 0.67$, the presence of disease fundamentally alters the system's outcome. In the endemic setting, outbreaks drive x_1 and x_2 up, eventually pushing them towards 1 (figure 3b), whereas in the absence of disease, both would decline towards 0.

Our results show that as homophily strengthens, the influence of the general group on minority groups diminishes despite high contact rates. In the anti-vaccine group, support for vaccination remains very low, even when the general group eventually approaches full support. In contrast, maintaining a high level of support within the pro-vaccine group becomes easier. When the vaccine support in the general group drops rapidly and fluctuates at low levels, support in the pro-vaccine group can stabilize or even rebound to full (figure 3d–i).

When homophily declines, the entire population eventually reaches consensus, with all groups becoming either fully pro-vaccine or fully anti-vaccine, and consensus is reached faster as the decline rate increases. Since the difference between within- and cross-group influence becomes smaller, the high contact rate enables the general group to dominate the dynamics of minority groups. Specifically, vaccine support in the anti-vaccine group can no longer remain at a low level independently of other groups;

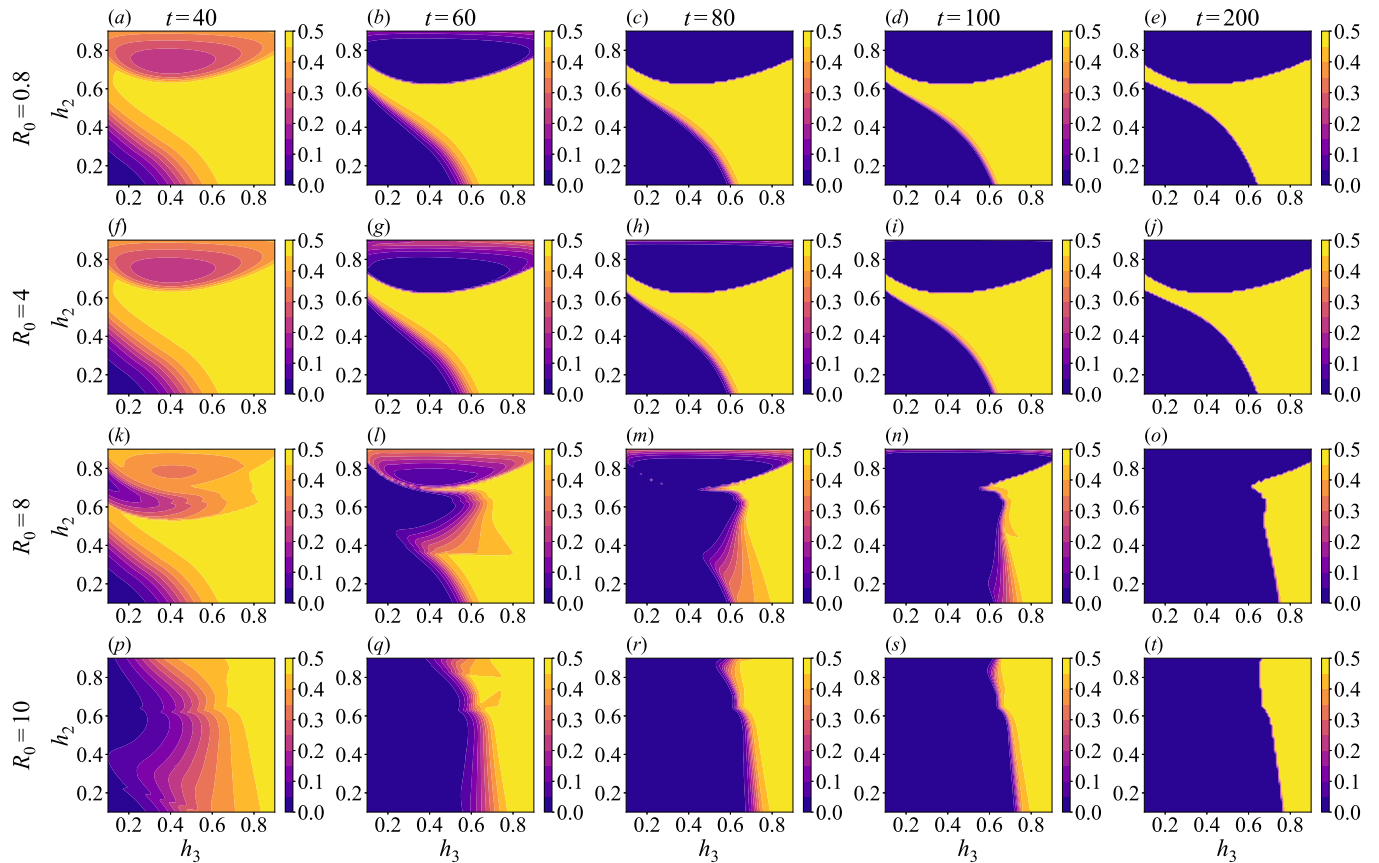


Figure 4. Time evolution of MMD under varying basic reproduction numbers over the h_2 – h_3 parameter plane. Each row corresponds to a different basic reproduction number: (a–e) $R_0 = 0.8$, (f–j) $R_0 = 4$, (k–o) $R_0 = 8$ and (p–t) $R_0 = 10$. Columns represent different time points: (a,f,k,p) $t = 40$, (b,g,l,q) $t = 60$, (c,h,m,r) $t = 80$, (d,i,n,s) $t = 100$ and (e,j,o,t) $t = 200$. The initial pro-vaccine percentage in the general group: $x_1(0) = 0.67$ and the initial minority–majority divergence: $\text{MMD}(0) = 0.375$.

the support in the pro-vaccine group, once substantially reduced, does not bounce back to a high level unless the support in the general group also recovers (figure 3j–o).

Monotonic changes in homophily have a smaller impact on the general group than on the minority groups. Due to its larger size, within-group influence always dominates the dynamics in the general group. When homophily increases over time, this dominance is reinforced, further amplifying the role of internal imitation. When homophily decreases and cross-group influence grows, minority groups tend to follow the general group, so the overall trajectory still reflects its dynamics. That said, the general group can still undergo noticeable changes in the early stages of monotonic homophily. For example, when homophily strengthens faster, vaccine support in the general group declines more slowly, or may not decline at all (figure 3b,e,h).

(c) Time evolution of homophily-driven opinion polarization

The existence of multi-strategy equilibria on the population level revealed in the previous sections highlights that opinion polarization, defined as the divergence in vaccine support across groups, can persist in the long term regardless of the presence of disease. Here, we measure opinion polarization by looking at how much the minority groups deviate from the general group. We define the minority–majority divergence (MMD) at time t as the population-weighted absolute deviation of minority groups' pro-vaccine percentages from the majority group's pro-vaccine percentage,

$$\text{MMD}(t) = \frac{N_2|x_2 - x_1| + N_3|x_3 - x_1|}{N_2 + N_3},$$

and plot the time evolution under different basic reproduction numbers R_0 , shown in figure 4. Small values of MMD indicate that minority groups closely follow the majority, reflecting low opinion polarization, whereas large values indicate strong divergence and high polarization across groups.

When $R_0 < 1$, polarization in vaccine sentiments begins to diminish under two conditions: (i) both h_2 and h_3 are small or (ii) h_2 is large (figure 4a–e). However, these two scenarios lead to different consensus outcomes. When homophily is weak in both minority groups, all three groups converge to a fully pro-vaccine state; when the pro-vaccine group has high homophily, the entire population reaches an anti-vaccine consensus (figure 5a). Since each infectious individual transmits the disease to fewer than one other person on average, the disease eventually dies out, and thus the behavioural dynamics resemble those of a disease-free system, as illustrated in figure 2a, but it is generally more complex.

As the basic reproduction number increases beyond the bifurcation point ($R_0 = 1$) to 4, we do not observe major qualitative differences in the time evolution or the final state of MMD and overall vaccine support across the parameter plane (figures 4f–j and

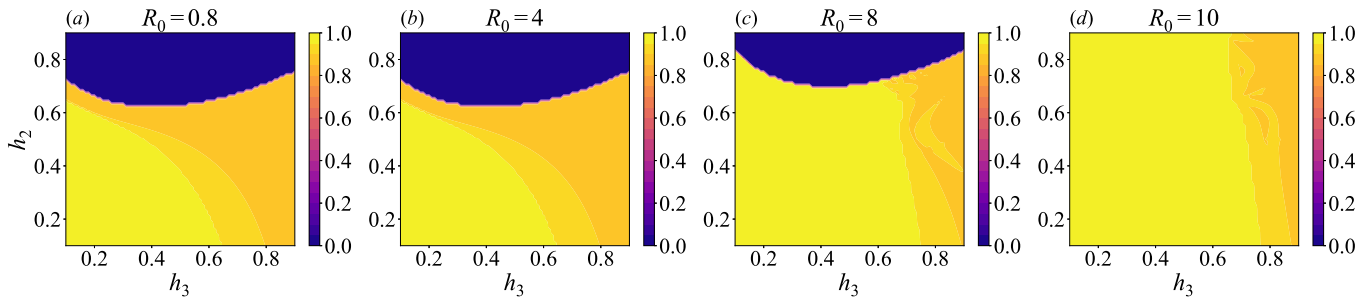


Figure 5. Pro-vaccine percentage in the entire population under varying basic reproduction numbers over the h_2 – h_3 parameter plane. Panels (a), (b), (c) and (d) correspond to panels (e), (j), (o) and (t) of figure 4, respectively.

5b). Due to the occurrence of large-scale outbreaks, the pro-vaccine percentage in each group, after an initial decline that occurs particularly when h_2 is large, experiences varying degrees of increase, leading to periodic and moderate increases in polarization (figure 4g,h).

When R_0 increases further to around 8, a new region of depolarization emerges on the parameter plane, specifically where h_2 ranges from 0.6 to 0.8, and h_3 remains below 0.6 (figure 4k–o). In these scenarios, the higher transmissibility of the disease leads to an earlier initial outbreak, which raises the pro-vaccine percentage across all three groups, eventually driving the system towards near-universal vaccine support. As a result, the pro-vaccine consensus region expands, while the anti-vaccine consensus region shrinks (figure 5c).

Finally, for a very large basic reproduction number $R_0 = 10$, such as that associated with measles, the persistence and disappearance of polarization are almost entirely governed by h_3 (figure 4p–t), and the anti-vaccine consensus will no longer exist (figure 5d).

4. Discussion

Social heterogeneity plays a critical role in infectious disease modelling by shaping individuals' vaccination sentiment and, in turn, influencing disease dynamics. Homophily further amplifies these effects by forming clusters among individuals with similar opinions. In this study, we introduce a multi-group vaccination game model to explore how population heterogeneity and homophily jointly affect disease and behavioural dynamics. We focus on a simple set-up with a majority group that represents the general public, and two minority groups of strongly pro-vaccine and anti-vaccine people.

Our results reveal that partially pro-vaccine equilibria commonly emerge at the group level, and population-level opinion polarization can persist regardless of whether a disease is present, suggesting that social structure alone is enough to maintain divided vaccination behaviour. We also show that time-varying homophily strongly affects the extent of polarization. These findings highlight the importance of evolving social dynamics in shaping long-term public attitudes towards vaccination.

The emergence of a group-level partially pro-vaccine equilibrium, where opposing strategies coexist, can be understood by examining the imitation within and between groups. With incomplete homophily, imitation is shaped by both internal and external payoff comparisons, allowing opposing effects to offset each other. For example, in a pro-vaccine-dominant group, within-group interactions reinforce pro-vaccine strategies, while cross-group interactions may still favour anti-vaccine strategies. As a result, anti-vaccine members adopt the majority stance, but some pro-vaccine individuals may shift due to external influence. The opposite holds in anti-vaccine dominant groups. At the population level, such group-specific dynamics give rise to persistent opinion polarization, as high homophily isolates minority groups from being fully assimilated into the majority's stance.

In a multi-group vaccination game, the dual role of homophily is more prominently exhibited. Although high homophily reduces communication between groups, it can be beneficial when cross-group interaction proves harmful to the interests of a group. Low homophily facilitates broader information exchange and increases the chances of pro-vaccine strategies spreading across groups, but it also allows anti-vaccine strategies to propagate more easily, which amplifies the negative effect of highly anti-vaccine groups. From a population-level perspective, having polarization across groups may be more desirable than convergence to an anti-vaccine consensus. Public health interventions should therefore adopt a targeted strategy reinforcing pro-vaccine norms within supportive groups through trusted messengers, while carefully managing cross-group communication to prevent misinformation spillover, or providing accurate information as a counterpoint to misinformation, when managing cross-group communication is not feasible. Instead of uniformly increasing connectivity between groups, interventions should focus on controlling content flow. In other words, accurate health information needs to be widely disseminated across groups, and (where possible) the spread of anti-vaccine narratives should be limited to reduce their harmful impact. Such interventions can reshape the social layer by helping increase pro-vaccine strategy adoption in anti-vaccine groups, enabling them to pass a tipping point and eventually converge to a fully pro-vaccine equilibrium. This, in turn, will influence the biological layer by enhancing population-level immunity and reducing disease transmission.

When vaccine support is near the critical threshold, the general group with moderate homophily initially behaves like a swing group, influenced by pro- and anti-vaccine minorities. As it approaches a steady state, it begins to shape the behaviour of both minority groups. With high homophily in the minority groups, an asymmetry emerges: the highly anti-vaccine group can sustain low vaccine support even when the broader population moves towards a pro-vaccine consensus, while the highly pro-vaccine

group often fails to maintain its stance as vaccine support in the general group declines towards 0, especially when the disease is not severe. In such cases, the impact of the disease is negligible, and the perceived risk of the vaccine makes vaccination less appealing. Therefore, if cross-group imitation favours anti-vaccine behaviour, strong social norms are needed within the pro-vaccine group to counter the external pressure of vaccine refusal.

At first glance, it may seem surprising that opinion polarization can persist at certain levels of homophily, even as the disease becomes more severe. One might expect that a higher transmission rate would elevate the perceived risk of infection and encourage vaccination across all groups. However, this persistence can be explained by three reasons. First, in the anti-vaccine group with high homophily, behavioural dynamics are largely driven by within-group imitation, which reinforces existing anti-vaccine strategies. Second, although the infection risk increases with higher transmission rates, it may still be insufficient to override the combined effects of the vaccine risk and social norms. Third, higher transmission rates prompt faster vaccine uptake in the general and pro-vaccine groups, leading to widespread immunity in the entire population, which lowers the number of infectious individuals and further reduces the urgency of vaccination within the anti-vaccine group.

It should be noted that the parametric pair (h_2, h_3) induces a first-order phase-transition-like behaviour in the steady state of $x(t)$ (see figure 2; for the legitimacy of using the term ‘phase transition’ in finite-dimensional models, see [62]). When $x_1(0) = 0.67$, the regions corresponding to the low and high phases are separated by a non-monotonic curve in the h_2 – h_3 parameter plane; when $x_1(0) = 0.69$, the transition weakens and the separation curve becomes monotonic. Moreover, considering $\text{MMD}(t)$ as an order parameter, similar first-order transitions are observed at later times (e.g. $t = 200$). At earlier times (e.g. $t = 80$), features of both first- and second-order transitions coexist (figure 4). For small R_0 , the parameter plane is divided into three regions with non-monotonic borders (figure 4e,f). Two of these regions correspond to low phases and the third corresponds to a high phase, based on the degree of polarization. Interestingly, as R_0 increases, the order parameter density plot undergoes a qualitative change, splitting into only two regions: one low phase and one high phase. The high phase shrinks and the boundary between the two regions becomes less smooth (figure 4o,t). Critical transitions in dynamical systems and phase transitions in statistical physics share some similar features, but a rigorous theoretical connection between them is still lacking. Future research could focus on clarifying this link, which may lead to a deeper understanding of early warning signals in coupled behaviour–disease systems [63].

One notable limitation of our model is the lack of heterogeneity at the biological level, which may distort the feedback loop between disease dynamics and vaccination behaviour. Extensive research on age-specific contact patterns in childhood infections has shown that such interactions are highly heterogeneous and assortative, not random [12,49,56,64]. Introducing heterogeneity on the biological layer, such as spatial variation in transmission or contact patterns, could lead to additional complexity in disease dynamics. For example, outbreaks are more likely to occur in high-contact subgroups, where diseases can persist and spread to surrounding low-contact groups. In contrast, social heterogeneity primarily shapes behavioural outcomes and can indirectly affect disease dynamics through feedback on vaccination behaviour [52,53]. Recent work estimated that the basic reproduction number for measles can increase by 70% if heterogeneity in vaccine uptake is taken into account [65]. Consequently, realistic coupled behaviour–disease models should account for a double heterogeneity in both physical contacts and opinion dynamics. However, for childhood diseases, the agents making vaccination decisions are distinct from those involved in disease transmission. Therefore, the biological and social layers should be properly aligned so that each group of individuals on the biological layer is associated with their parents on the social layer, who make vaccination decisions on their behalf. Although age remains the ‘easy to observe’ determinant of the transmission of childhood infections [49,66], future research would benefit from examining the joint heterogeneity of children’s age-structured contacts and parental vaccination opinions, providing stronger empirical support for coupled behaviour–disease models. In principle, social heterogeneity could also affect epidemiological parameters. For example, anti-vaccine individuals may adapt their contact rates based on perceived risk. Clusters that are less willing to accept interventions may face a higher disease burden, which could prompt more individuals within these groups to adopt them, thus potentially dampening the divergences in outcomes observed in our model. Such mechanisms are particularly relevant for diseases in which anti-vaccine individuals themselves participate directly in transmission (e.g. COVID-19), but are less direct for childhood diseases such as measles, where children’s contact networks are largely determined by institutional settings rather than parental choices. Additionally, our model does not include stochastic effects or spatial mobility of agents, both of which may further influence disease spread and behavioural responses in real-world settings. For instance, spatial vaccination games have shown how local versus non-local information about vaccine risks can crucially shape behavioural outcomes [67].

In this study, we develop a multi-group model for an arbitrary number of groups but focus on simulating three-group dynamics to balance computational complexity and interpretability. As additional groups with varying vaccine support levels are considered, they influence both the general public and each other, leading to more complex dynamics. Furthermore, the division into majority and extreme minority groups is primarily a conceptual approach; in practice, the choice of grouping should depend on the available data. For instance, individuals can be grouped by geographic regions, as spatial clustering of vaccination rates and local homophily are often observed [68–70]. In such cases, a large general group may not exist, and group sizes may be more evenly distributed. Importantly, when grouping by region, heterogeneity on the biological layer can become necessary to account for differences in disease transmission across subpopulations. This set-up also allows tracking disease spread in each group in parallel with changes in vaccine strategies.

In our model, we only consider long-term exogenous (i.e. non-epidemiological) changes in homophily to identify stable influences on vaccination behaviour. However, previous research has shown that exogenous conditions can cause rapid homophily shifts [71], which could affect infection trajectories in the short term. In relation to this, an example might be seasonal influenza epidemics over successive seasons. Additionally, homophily might also evolve endogenously; outbreaks and sudden rumours about vaccine adverse events could trigger faster changes in homophily by raising concerns about vaccine effectiveness or safety, even within the majority group.

Our findings highlight that homophily, whether static or dynamic, plays a crucial role in shaping long-term vaccination behaviour and opinion polarization. While lowering homophily can reduce polarization and help the spread of pro-vaccine strategies, it also unintentionally creates conditions for the dissemination of anti-vaccine strategies. As a result, from a public health perspective, simply removing communication barriers is not enough; effective control over the content being spread is equally important. In summary, our multi-group framework offers greater flexibility for simulating the dynamics of vaccination behaviour across diverse population structures. Future work could validate the model with empirical data and extend the framework by incorporating heterogeneous transmission dynamics.

Ethics. This work did not require ethical approval from a human subject or animal welfare committee.

Data accessibility. All code used in this study is available at Zenodo [72]. Data can be generated using the provided MATLAB scripts.

Supplementary material is available online [73].

Declaration of AI use. We have not used AI-assisted technologies in creating this article.

Authors' contributions. Z.H.: conceptualization, formal analysis, investigation, methodology, software, visualization, writing—original draft, writing—review and editing; P.M.: conceptualization, writing—review and editing; A.d.O.: conceptualization, writing—review and editing; C.T.B.: conceptualization, methodology, supervision, writing—review and editing.

All authors gave final approval for publication and agreed to be held accountable for the work performed therein.

Conflict of interest declaration. We declare we have no competing interests.

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