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Social network architecture and the maintenance of deleterious cultural traits

Sam Yeaman[1,2,*], Alana Schick[1] and Laurent Lehmann[3]

[1]*Department of Zoology, University of British Columbia, 6270 University Boulevard, Vancouver, British Columbia, Canada V6T 1Z4*
[2]*Department of Biology, University of Neuchâtel, 11 Rue Emile Argand, 2000 Neuchâtel, Switzerland*
[3]*Department of Ecology and Evolution, University of Lausanne, Le Biophore, 1015 Lausanne, Switzerland*

How have changes in communications technology affected the way that misinformation spreads through a population and persists? To what extent do differences in the architecture of social networks affect the spread of misinformation, relative to the rates and rules by which individuals transmit or eliminate different pieces of information (cultural traits)? Here, we use analytical models and individual-based simulations to study how a 'cultural load' of misinformation can be maintained in a population under a balance between social transmission and selective elimination of cultural traits with low intrinsic value. While considerable research has explored how network architecture affects percolation processes, we find that the relative rates at which individuals transmit or eliminate traits can have much more profound impacts on the cultural load than differences in network architecture. In particular, the cultural load is insensitive to correlations between an individual's network degree and rate of elimination when these quantities vary among individuals. Taken together, these results suggest that changes in communications technology may have influenced cultural evolution more strongly through changes in the amount of information flow, rather than the details of who is connected to whom.

Keywords: cultural evolution; social learning; maladaptive culture; susceptible–infected–susceptible; epidemic spread; diffusion

1. INTRODUCTION

While many animals appear to have rudimentary forms of culture [1], humans seem to be the only species to have created innovations that modify the way by which cultural information is transmitted through a population. Successive developments in communications technology, from the hieroglyph to the Internet, have fundamentally altered how humans communicate and transmit different pieces of information. These technologies have probably affected the dynamics of information spread through changes in both the rates of communication and transmission and the architecture of the social networks through which transmission occurs (who is connected to whom). Human cultural networks have likely progressed from approximately isolation-by-distance type architectures in pre-historic times (figure 1*a*) to small-world configurations with the establishment of long-distance trading networks (figure 1*b*,*c*), to more complicated and interconnected architectures with the emergence of modern communication technology [5–7]. For example, the architecture of linkages between documents on the Internet has a highly heterogeneous scale-free degree distribution, with many individual nodes having only a few connections and few nodes having many connections (figure 1*d*; [3]). Similarly, the connections between individuals using various online social networks have degree distributions similar to scale-free networks, but with fewer highly connected nodes [8,9]. The diffusion of information from television or radio broadcasters is unidirectional and even more unevenly distributed, with only a few organizations communicating with audiences numbering in the millions. Along with these changes in network architecture, rates of communication have also probably increased as new technologies reduce the economic costs of communication across long distances.

While such changes have probably facilitated the spread of beneficial new technologies and ideas, they have also likely enabled the increased transmission of information with deleterious consequences (e.g. on biological fitness, welfare, economic utility or accuracy of belief). As the persistence of such misinformation can have negative consequences for various aspects of human society, it is critical to understand how changes in communications technology may have influenced its spread and maintenance. How strong is the effect of a given change in network architecture, relative to

*Author for correspondence (yeaman@zoology.ubc.ca).

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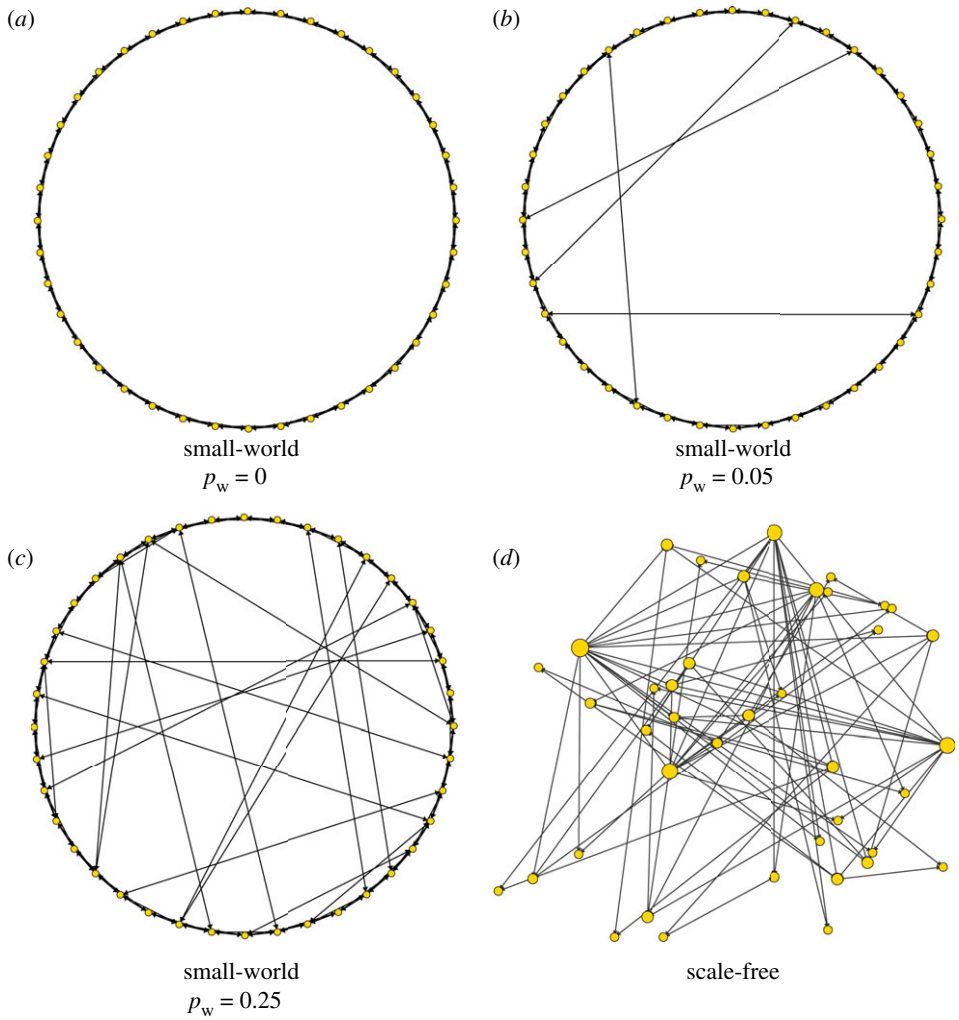

Figure 1. (*a*–*c*) Social networks architectures yielded by the Watts & Strogatz [2] small-world algorithm with varying proportions of long-distance connections (p_w) and by the Barabási & Albert [3] scale-free algorithm (*d*). All four networks have $N = 40$ nodes and an average degree of $k = 4$ links per individual. Networks were drawn using the SocNetV program [4]. (Online version in colour.)

a change in the rate of social communication or the way that individuals process information? Answering these questions is important for understanding human evolutionary history and for predicting the consequences of contemporary shifts in the use of communication technology. For example, a total shutdown of Internet and mobile telephone communications (e.g. similar to January 2011 in Egypt [10]) is likely to result in a drastic change in the way that cultural information could spread through a national population. How might such changes in the availability of communications technology affect the flow and maintenance of misinformation?

While understanding the effects of transmission is critical to answering this question, there are a range of factors that can result in a reduction in the frequency of a deleterious trait in a population. For example, individuals may forget about a trait and therefore no longer carry it or transmit it to others in the population. Deleterious traits may also be effectively 'eliminated' from the population if individuals evaluate these traits, recognize their deleterious value and choose not to use or retransmit them. Regardless of the functional details of any such mechanisms, they can be grouped together in terms of their net effects reducing the frequency of the trait in the population. The maintenance of deleterious traits may therefore be effectively modelled as a product of the tension between the net effects of all factors involved in transmission and elimination of traits, as is common with epidemic models of disease spread [11–13].

Considerable research has explored how the architecture of social networks affects diffusion processes for simple contagions like rumours or diseases [2,11,12,14–17]. When applied to the spread of socially transmitted information, network models have often been modified to scale the probability of transmission by the frequency of social interactions or other complex learning rules [18–22]. In most cases, these studies have focused on deriving 'epidemic thresholds' and other metrics describing the conditions under which such contagions are likely to spread. Generally speaking, these models have found that networks with high variance in connectivity among individuals (e.g. scale-free) reduce the rates of transmission required to realize an epidemic [7,23], whereas networks with high degrees of clustering (e.g. small-world) can have the opposite effect [23]. While these network models are all built around the tension between rates of transmission and rates of elimination, they have not typically represented the *value* of transmitted information as both a factor affecting the dynamics of diffusion and a dynamical endpoint of

interest (i.e. what is the average value of deleterious traits maintained in the population at equilibrium?). Some recent works have examined how network architecture affects the consensus value of transmitted information [24]; but in this case, the concept of misinformation is defined as the deviation from social consensus, rather than as a quality that is specific for value of the traits involved. The maintenance of maladaptive culture has been studied in the context of models of social and individual learning [25,26], but these studies have typically not incorporated the effect of network architecture.

Here, we use a combination of analytical theory and individual-based simulations to compare how the spread and maintenance of deleterious traits are affected by different network architectures (e.g. scale-free, small-world, one-to-many communication) versus variations in the rates that individuals transmit or eliminate these traits. While we use an approach similar to models of epidemic spread [11,23], the processes we represent can be interpreted in a manner similar to models of social versus individual learning [27–31]: traits are innovated (individual learning), transmitted among individuals (social learning) and eliminated from use and retransmission (a form of adaptive individual learning, if the elimination process is interpreted as occurring as a product of an individual evaluating and recognizing a trait's negative value). We assume that the spread of a given cultural trait is affected by two coefficients (each varying from 0 to 1): its transmissibility (c), determining the probability of spread during social contact and its intrinsic value (v), which represents its deleterious effect (e.g. on biological fitness, welfare, economic utility or accuracy of belief; neutral traits have $v = 1$) and also determines the probability that the trait is eliminated. In classical population genetics, 'genetic load' represents the effect of a given deleterious mutation on the mean fitness of a population, calculated as a function of its effect on individual fitness and its frequency in the population [32–35]. Here, we use a similar approach to represent the effect of a given deleterious cultural trait on the 'misinformation' in a population, defining the 'cultural load' that the trait contributes as a function of its intrinsic value and its frequency. We then study how variations in c, v and in the rates of social communication and elimination affect the frequency of the trait in the population and the cultural load that results. In particular, we seek to quantify the effect of variations in network architecture on cultural load relative to changes in the average rates of social communication, as both of these aspects of human interaction have been greatly affected by changes in communications technology.

2. MODEL

We begin by describing the derivation of the analytical models that we use to predict the cultural load maintained at equilibrium in an unstructured population. We then describe the design of the individual-based simulations that we use to study the effect of network architecture on cultural load. Finally, we present and compare the results yielded by these different approaches, referring back to the appropriate equations for reference.

2.1. Analytical model

For the analytical approach, we assume a population of infinite size where individuals interact at random (i.e. a panmictic social network). Individuals can gain a given trait by either innovation (individual learning; at rate μ) or by transmission during social interaction (social learning), which is attempted between two randomly selected individuals at rate β. During a given social interaction, the trait can be passed from the 'sender' to the 'receiver' with a probability equal to its transmissibility (c; hereafter, we use 'rate of communication' to refer to β, and 'rate of transmission' to refer to βc). Individuals can perform the elimination process at rate λ, with the probability that a given trait is actually discarded during an elimination event specified by its discard coefficient (r; hereafter, we use 'rate of elimination' to refer to λ, and the 'realized rate of elimination' to refer to λr). Traits that have been eliminated may be regained through social transmission or innovation, such that this model closely follows the susceptible–infected–susceptible (SIS) model of epidemiology [11,16], and previous models of cultural evolution [36,37].

Following these assumptions, the rate of change in the frequency (ρ) of a focal trait in the population is therefore:

$$\frac{\mathrm{d}\rho}{\mathrm{d}t} = (1-\rho)(\mu + \beta c\rho) - \rho\lambda r. \tag{2.1}$$

The first term in this expression represents the increase in frequency of the trait as individuals that do not have it either innovate (at rate μ) or socially learn it (at rate $\beta c\rho$), whereas the second term represents the decrease in frequency as individuals that have the trait eliminate it (at rate λr). While a composite term could be used to represent the joint effect of the rate of communication (β) and the transmissibility of a trait (c), we keep these terms separate to illustrate how load varies for traits with different transmissibilities under different values of β, as the accumulation of multiple traits is of explicit interest in cultural evolution (likewise for λ and r). As we are most interested in interpreting this model in terms of the effect of individuals making choices to 'retain' or 'discard' a given trait according to its value (as opposed to other factors that might affect the rate of elimination, such as memory), we assume for the remainder of this paper that traits with lower intrinsic value (v) have a higher probability of being discarded during the elimination process (r), such that $r = (1 - v)$. The following derivation could instead incorporate some more complex function relating intrinsic value (which affects the load) to the probability of a trait being discarded (which affects the equilibrium frequency), or incorporate memory into the calculation of r. As we assume that the probability of the trait being discarded is proportional to its value, the effect of the elimination process is somewhat similar to 'guided variation' or 'adaptive filtering' of cultural traits [25,28], but applied to individual traits instead of at the level of the population.

If we assume that the rate of innovation is vanishingly low, setting $\mu = 0$, then this yields two stable

equilibria: one where nobody carries the trait, $\hat{\rho} = 0$, when $\beta c < \lambda(1 - v)$, and one where the trait segregates at intermediate frequency, $\hat{\rho} = 1 - (\lambda(1 - v)/\beta c)$, when $\beta c > \lambda(1 - v)$. When the focal trait is introduced into a small fraction of the population, it will therefore either go extinct or approach the intermediate frequency equilibrium when the transmission rate, βc, exceeds the realized elimination rate, $\lambda(1 - v)$. As all traits considered here are deleterious, the value of not having a trait is assumed to be equal to 1, such that the cultural load (L) can be defined following the usual definition for genetic load based on the mean fitness of the population ($L = 1 - \bar{W}$; [32–35]), but substituting mean value ($\bar{v} = \rho(1 - v) + (1 - \rho)$) for mean fitness ($\bar{w}$). This gives

$$L = 1 - (\hat{\rho}v + (1 - \hat{\rho})) = \hat{\rho}(1 - v), \tag{2.2}$$

such that

$$L = \frac{(1 - v)(\beta c - \lambda(1 - v))}{\beta c}. \tag{2.3}$$

The cultural load varies between 0 and 1 and provides a measure of the deleterious effect of the focal trait at the level of the population, which is proportional to both its frequency and intrinsic value (such that $L = 0$ when the trait is not present in the population). Following the same derivation, the expression for load under recurrent innovation ($\mu > 0$) is given by:

$$L = \frac{(1 - v)(\beta c - \lambda(1 - v) - \mu + \sqrt{(\beta c - \lambda(1 - v) - \mu)^2 + 4\beta c\mu})}{2\beta c}. \tag{2.4}$$

A range of analytical approximations has been developed to illustrate how different networks affect the equilibrium frequency for the SIS model and other related epidemic models [2,6,16], but these often rely upon assumptions that make it difficult to compare results among models. Below, we use individual-based simulations to compare the effect of different network architectures (e.g. scale-free, small-world) on equilibrium trait frequency and cultural load. For continuity, we carry on with our descriptions of two other analytical models of one-to-many transmission, before comparing results among simulations and these various analytical models.

One-to-many transmission occurs whenever one individual (or one coordinated group of individuals) is able to communicate with all other individuals in a population, as might occur with a teacher, leader or mass media source such as a radio or television broadcaster [38]. If individuals in the population communicate normally among themselves at rate $\beta(1 - \tau)$ and receive information unidirectionally from a single central node at rate $\beta\tau$ (with τ scaling from zero to one), then we can modify the approach from equation (2.1) to represent the change in trait frequency in both the members of the general population (ρ_g) and the central node (ρ_l):

$$\left.\begin{aligned} &\frac{d\rho_g}{dt} = (l - \rho_g)\beta c((l - \tau)\rho_g + \tau\rho_l) - \rho_g\lambda_g(l - v) \\ \text{and} \quad &\frac{d\rho_l}{dt} = (l - \rho_l)\beta c\rho_g - \rho_l\lambda_l(l - v). \end{aligned}\right\} \tag{2.5}$$

This modified model assumes that the central node only learns from individuals in the general population, and allows for different rates of the elimination process in the central node (λ_l) and the general population (λ_g).

These equations can be solved for different equilibrium trait frequencies, depending upon the assumptions about the rates of the elimination process. Most simply, when the central node acts as a regular member of the population such that $\lambda_l = \lambda_g = \lambda$, it can be shown by performing a standard linear stability analysis that there is an equilibrium with frequencies $\hat{\rho}_l = \hat{\rho}_g = 0$ which is unstable when $\hat{\rho}c > \lambda(1 - v)$. The cultural dynamics may then converge to an equilibrium given by the frequencies $\hat{\rho}_l = \hat{\rho}_g = 1 - (\lambda(l - v)/(\beta c))$, which does not differ from the expectation in a panmictic population. This shows that there is no effect of this type of network architecture on mean cultural load at equilibrium, without some concomitant change in updating rules (although this architecture may increase the similarity among individuals in the population, relative to panmixia).

If we instead assume that the central node is a persistent source of misinformation that always possesses and never eliminates the focal trait ($\lambda_l = 0$), then $\hat{\rho}_l = 1$, resulting in more load being maintained in the population. In this case, $\rho_g = 0$ is unstable and the dynamics converge to

$$\hat{\rho}_g = \frac{\beta c(1 - 2\tau) - \lambda(1 - v) + \sqrt{(\beta c - \lambda(1 - v))^2 + 4\tau\beta c\lambda(1 - v)}}{2\beta c(1 - \tau)}, \tag{2.6}$$

with the load being determined by the trait in the general population and given by

$$L = \rho_g(1 - v). \tag{2.7}$$

By contrast, if we assume that the central node has essentially limitless capacity for eliminating deleterious traits ($\lambda_l \to \infty$), then $d\rho_l/dt$ can never be positive and the equilibrium frequency at the central node is $\hat{\rho}_l = 0$. The equilibrium $\hat{\rho}_g = 0$ is then unstable when

$\beta c(1-\tau) - \lambda_g(1-v) > 0$. If $\hat{\rho} > 0$ the equilibrium $\hat{\rho}_l = \hat{\rho}_g = 0$ can be unstable even when the realized elimination rate $\lambda_g(1-v)$ does not exceed the transmission rate βc, and the cultural dynamics may then settle in the equilibrium

$$\hat{\rho}_g = \frac{\beta c(1-\tau) - \lambda(1-v)}{\beta c(1-\tau)}, \tag{2.8}$$

where $\lambda = \lambda_g$, resulting in less load being maintained in the population.

We now discuss the design of the individual-based simulations that we use to compare the effects of different network architectures and updating rules on cultural load at equilibrium, before comparing the results yielded by these approaches.

2.2. Individual-based simulations

We implemented individual-based simulations in R the Comprehensive R Archive Network (CRAN) and C with updating rules similar to those used in the above analytical model, but with explicit social interaction networks (source code available upon request from the corresponding author). We assume that the population is of constant size N, with each individual occupying a single node on a static social network describing the potential connections along which traits can be transmitted. Individuals have up to n independently updated traits that are either present or absent, with asynchronous updating following a sequence of transmission, elimination and innovation events at each time step. A social communication event occurs every $1/\beta$ time steps, whereby one link is randomly selected from the social network and one of the nodes is randomly assigned as the sender and the other the receiver (networks are undirected). The sender then attempts to transmit to the receiver each trait that they possess, with $p[\text{transmit}] = c$ for each trait. An elimination event occurs every $1/\lambda$ timesteps, whereby one individual is randomly selected from the population to potentially discard their traits, with $p[\text{discard}] = 1 - v$ for each trait. Finally, every timestep, individuals may gain each trait by innovation with probability μ. We use only very low rates of innovation in the presentation of our results ($\mu = 10^{-3}$), to facilitate comparisons between the analytical predictions and individual-based simulations (this low level of μ has a negligible effect on the results).

Depending on the simulation, the architecture of the network defining social communication and transmission was determined by one of several algorithms. All networks were created at initialization and did not change thereafter, with $N = 1000$ nodes and 2000 bi-directional edges, such that mean network degree, $\bar{k} = 4$ (with the exception of the panmictic case, where all possible connections among nodes are included). Scale-free networks were created following the preferential attachment algorithm of Barabási & Albert [3] with two initial nodes and two edges added for each new node ($m_0 = m = 2$) until reaching 1000 nodes, with two final iterations of the algorithm added at the end to preferentially attach the initial m_0 nodes. Small-world networks were created following the model of Watts & Strogatz [2], such that all individuals were arranged on a circular one-dimensional lattice, with each node connected to its two nearest neighbours (so that again, $\bar{k} = 4$). Small-world connectivity was then added by rewiring a proportion, p_w, of the nodes by randomly choosing one end of a randomly chosen link and reconnecting it to a new node without duplicating an existing link or rewiring more than one link per node (but without controlling for multiple links being rewired to the same node).

There are many potential reasons that rates of elimination could vary among individuals, for example, if some individuals devote more time to determining the value of the cultural traits they carry and choosing whether to continue to use them and retransmit them. In order to examine the effect of variation among individuals in the rate at which they were picked to perform the elimination process, we set the elimination rate per timestep of individual j as:

$$\eta_j = \lambda\left(\frac{k_j^\gamma}{\sum_j^N k_j^\gamma}\right), \tag{2.9}$$

where the term in parentheses is a modified probit choice function [39], k_j is the network degree of individual j (the number of links between individual j and all other individuals) and γ allows us to tune the extent to which an individual is more or less likely to be chosen for updating as a function of network degree disparities. By this function, when $\gamma > 0$, the most highly connected individuals perform the elimination process most often, whereas the opposite occurs when $\gamma < 0$, and when $\gamma = 0$, individuals are picked at random from the network for updating. Varying γ could affect the cultural load at equilibrium through changes in two aspects of the elimination process: the variance in elimination rate (η) among individuals, and the correlation between this rate and the network degree of individuals (k). To test the importance of these two effects of γ, we also ran simulations setting individual rates as above using equation (2.9), but then randomly shuffling the values of η yielded by given values of γ and k_j. This random shuffling maintains the variance in η owing to specific values of γ and k_j, but breaks the correlation between η and k (referred to as 'shuffled η'). We also ran simulations by reverse-ordering the values of η yielded by a given value of γ and k_j, such that when $\gamma > 0$, the most highly connected individual had the lowest value of η (and vice versa). This reverse-ordering also maintains the variance in η owing to γ and k_j, but reverses the correlation between η and k (referred to as 'reverse-ordered η'). We use this generic function and the 'reverse-ordered' versus 'shuffled' variations to illustrate the general importance of variation in η and of correlation between η and k, as we are not attempting to represent any specific cognitive or behavioural processes that define the individual rates of elimination; more complex functions could be used for more specific applications.

In all cases, simulations were run for at least 10^6 time steps, with the mean frequency of each cultural trait recorded at 1000 time-step intervals during the final 10^5 time steps of each simulation (by which point

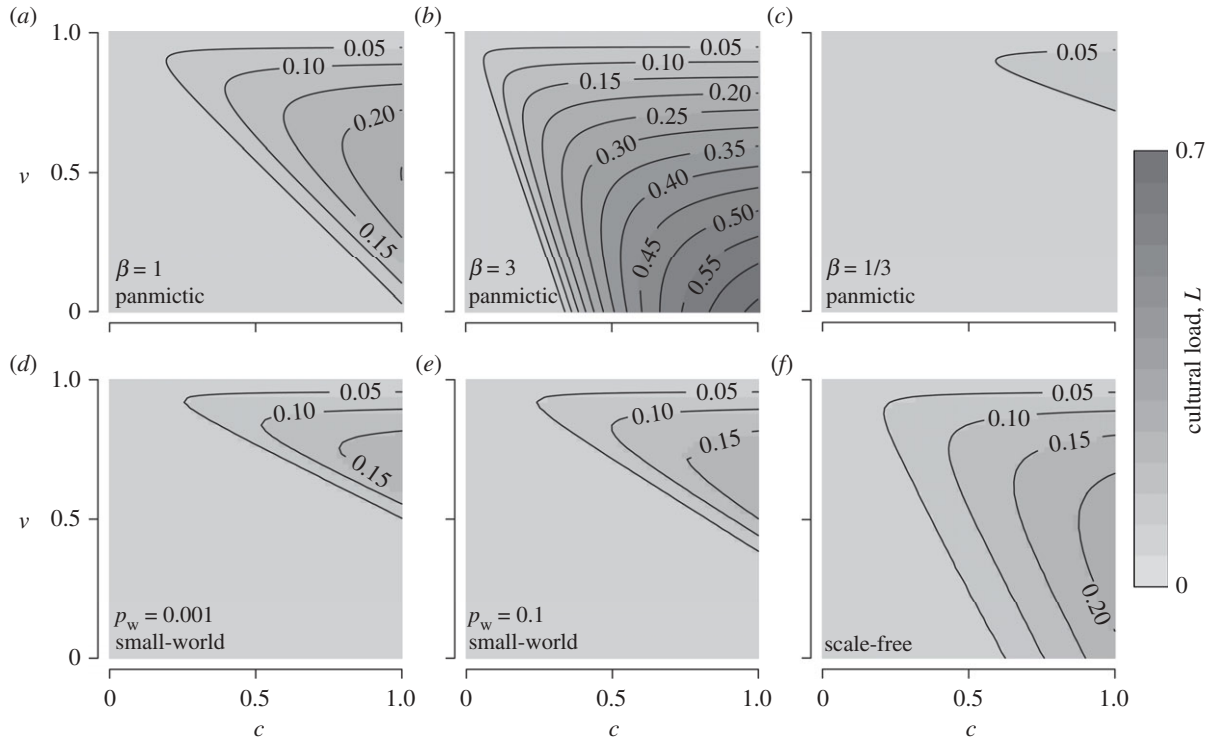

Figure 2. Cultural load at equilibrium contributed by a trait as a function of its intrinsic value, v, and transmissibility, c, under different rates of communication (β) and different network architectures. Lines represent contours of equal load, as indicated by both the numerals plotted over the contour lines and the shading between contours. (a–c) Numerical results for the panmictic network under different rates of transmission using equation (2.4). (d–f) Simulation results for different network architectures with the same rates of communication ($\beta = 1$). (a–f) $\mu = 10^{-3}$, $\lambda = 1$, $\gamma = 0$; for simulations, $N = 1000$. We note that simulations under a panmictic model yielded results that were virtually indistinguishable from the analytical results shown in (a–c).

there was no consistent change in this frequency). As above, the load contributed by each cultural trait was calculated as a function of its frequency and intrinsic value: $L = \rho(1 - v)$. For all simulations, $N = 1000$ and $n = 1000$, with the value (v) and transmissibility (c) coefficients drawn from a uniform distribution with values between 0 and 1. We ran 1000 replicate runs per parameter set, yielding data for the equilibrium trait frequency of 10^6 independently diffusing cultural traits for each parameter set.

3. RESULTS

3.1. Mean transmission rate versus network architecture

Generally speaking, the cultural load contributed by different traits at equilibrium was much more strongly affected by changes in the rates of the communication and elimination processes than by differences in network architecture. The results for the panmictic network under equal rates of communication and elimination ($\lambda = \beta = 1$) provide a baseline for comparison to illustrate these findings (equations (2.3) and (2.4)). In this baseline case (figure 2*a*), there is effectively very little load contributed by any traits with $c < 1 - v$, as they are rapidly eliminated from the population following introduction by innovation (we refer to the shape of the surface represented by these contour plots as the 'load profile'). Even a threefold increase in the rate of communication causes a substantial increase in the proportion of parameter space that maintains cultural load at equilibrium, shifting the load profile so that traits with much lower transmissibilities are maintained in the population (figure 2*b*). By contrast, a threefold decrease in communication results in very little load being maintained across most of the parameter space, with the maximum load occurring for traits with relatively high value (figure 2*c*). While different network architectures also caused shifts in the amount of load maintained at equilibrium for different values of c and v (figure 2*d*–*f*), these shifts were much less pronounced than observed for the relatively small fold-changes in β (or equally, λ). Small-world networks reduced the cultural load (figure 2*d*,*e*), resulting in a profile similar to that found under reduced rates of communication (figure 2*c*). By contrast, scale-free networks increased the load, shifting the load profile so that traits with lower transmissibilities were maintained at non-zero frequencies and contributed to load (figure 2*f*). As the networks modelled in figure 2 represent extremes on a continuum of plausible human social network architectures, but β and λ can plausibly take on values much more extreme than those used here, the load profile has the potential to vary much more strongly with changes in the rate of communication or elimination than with changes in network architecture.

For a population with many traits, the average load will be determined by the distribution of c and v

coefficients of the cultural traits that arise in the population; if newly innovated traits are more commonly of high value or low transmissibility, then the average load will be lower. Here, we have assumed a uniform distribution of both c and v for simplicity; taking an unweighted average over all values of load shown in figure 2, the average load at equilibrium would be (for figure 2*a–f*): $\bar{L}_a = 0.059$; $\bar{L}_b = 0.231$; $\bar{L}_c = 0.008$; $\bar{L}_d = 0.025$; $\bar{L}_e = 0.032$; $\bar{L}_f = 0.079$. Under this assumption of a uniform distribution of both c and v, a panmictic network with a rate of communication of $\beta \sim 1.18$ would generate an average amount of load approximately equal to that found on the scale-free networks in figure 2*f*, whereas $\beta \sim 0.64$ would generate an average load approximately equal to that found on the small-world networks in figure 2*d*.

3.2. Variation among individuals in the rate of elimination

Network models often explore how variation among individuals in rates of communication can affect the likelihood of epidemic spread [23,40,41], but these models rarely examine the effect of variation among individuals in the rate of recovery, perhaps because for biological infections this is relatively constant among individuals. But for cultural traits, it seems likely that there might be considerable variation among individuals in the rate they perform the elimination process and discard deleterious traits. If cultural traits are being eliminated from use and retransmission by individuals being able to determine their value and making choices, then variation among individuals in how much effort in time or energy they spend in making choices would yield considerable differences in η. If we hold the mean rate of elimination constant ($\lambda = \beta = 1$) but vary the rates at which individuals perform the elimination process (η; by setting $\gamma \neq 0$), then we see some surprising effects on the cultural load at equilibrium. When we set $\gamma > 0$, elimination is more often performed by the most highly connected individuals (highest k), whereas for $\gamma < 0$, elimination is more often performed by the least-connected individuals. In either case, the cultural load is substantially increased over the case where all individuals eliminate at equal rates (figure 3 versus figure 2*f*), with $\bar{L}_{\gamma=-3} = 0.177$ and $\bar{L}_{\gamma=-3} = 0.444$, whereas $\bar{L}_{\gamma=-3} = 0.079$. Generally speaking, like the variance in η among individuals (figure 3*c*; thick grey dashed line), the average load always increases with $|\gamma|$, but this increase occurs more rapidly with $\gamma > 0$ (figure 3*c*).

Perhaps more surprisingly, results were almost identical when we either shuffled or reverse-ordered η among individuals, breaking the correlation between an individual's network degree (k) and their rate of elimination (η; 'shuffled η'; figure 3, dashed lines) or reversing the sign of the correlation ('reverse-ordered η'; figure 3, dotted lines). The lack of a substantial effect owing to shuffling or reverse-ordering η shows that the increase in load under $\gamma \neq 0$ is almost entirely owing to the variation in the rate of elimination among individuals induced by γ, rather than from any additional interaction with the architecture of the network. While it might be expected

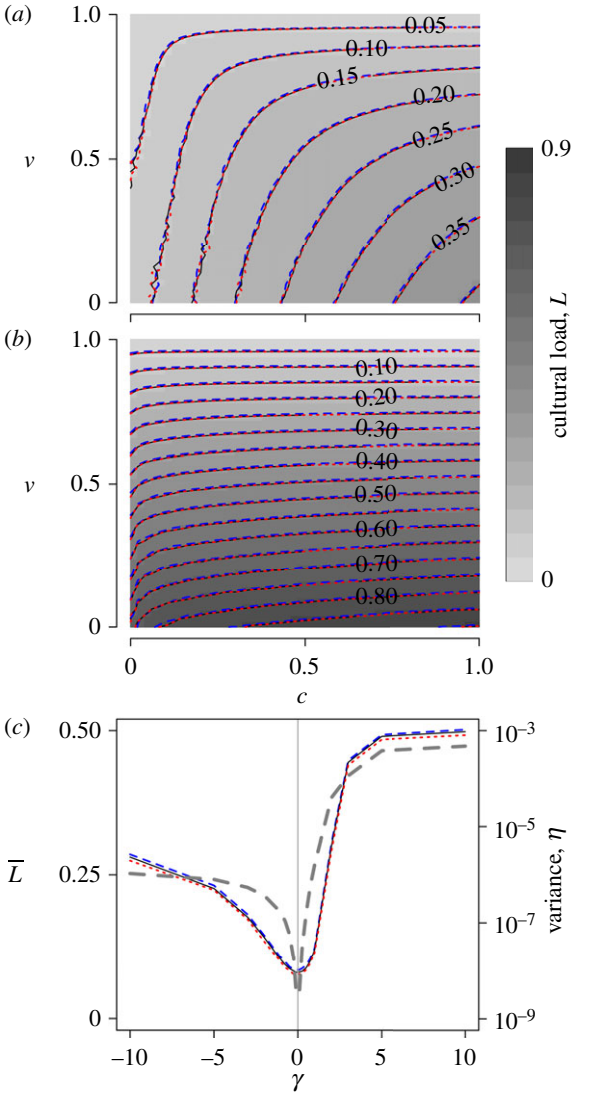

Figure 3. Cultural load at equilibrium under different levels of variation among individuals in the rate they perform the elimination process on a scale-free network. (*a*) Individuals that are the most highly connected perform elimination the least often ($\gamma = -3$; solid lines), whereas (*b*) shows the results for the opposite case ($\gamma = 3$; solid lines), while (*c*) shows the variance in η (thick grey dashed line) and the average load ($\bar{L}$) as a function of γ. In all cases, when the individual rates of elimination (η) set by γ are shuffled (dashed lines; blue online) or reverse-ordered (dotted lines; red online) among individuals, there is vanishingly little change in results (lines are slightly offset for visibility). (*a–c*) $N = 1000$, $\mu = 10^{-3}$, $\beta = 1$, $\lambda = 1$; shading and contours plotted with the same intervals and palette as in figure 2. (Online version in colour.)

that if the most highly connected individuals were also performing the elimination process most often, load might be reduced relative to a case where minimally connected or randomly chosen individuals were performing the elimination process most often (as highly connected individuals would be less likely to carry and therefore transmit the deleterious traits, overall rates of transmission of such traits might be reduced), these results show that this is not the case. While more load is maintained under $\gamma = x$ than under $\gamma = -x$, as shown in figure 3*c*, these results show that this difference is a result of the higher variance in η yielded under

positive values of γ, rather than a result of the most- or least-connected individuals eliminating most often.

We suggest that the reason that load is higher under increased variance in η is that this variation reduces the efficiency of the elimination process. The efficiency of the elimination process can be defined as the net rate that elimination events result in the discarding of a trait, $E = r\sum_j^N p_j\eta_j/N$, where η_j represents the rate of the elimination process for individual j, and p_j represents the probability that individual j carries the trait. In the simplest case, when there is no variance in η and the network is panmictic, each individual will carry the trait with probability $p_j = \rho$ and will be picked to eliminate the trait with probability $\eta = \lambda/N$ (equation (2.9)), yielding $E = \rho\lambda r$. When there is variation in η, individuals with high η often do not carry the trait and therefore tend to have low values of p (and vice versa). Analysis of simulation data shows that individual contributions to total efficiency ($p_j\eta_j$) tend to be very low for individuals with low η (as might be expected), but contributions to efficiency made by individuals with high η do not balance these, because their elimination events are often performed when they do not carry the trait. This reduces the average efficiency of the elimination process at the level of the population. For example, for a trait with $c = 0.75$ and $v = 0.75$ spreading through a scale-free network with $\lambda = \beta = 1$ (as per figure 3), when $\gamma = 0$, the average efficiency of elimination observed in the simulations was $E = 0.148$, whereas when $\gamma = 3$, $E = 0.025$. Basically, because individuals that perform the elimination process most often will typically not carry the trait, their elimination events are often 'wasted' in terms of their effects on the trait frequency at the level of the population, and do not balance the decreases in elimination efficiency owing to individuals that rarely perform the elimination process. Relative to the effect of this decrease in the efficiency of elimination, we found only vanishingly small effects of correlation between individual rates of elimination and network connectivity.

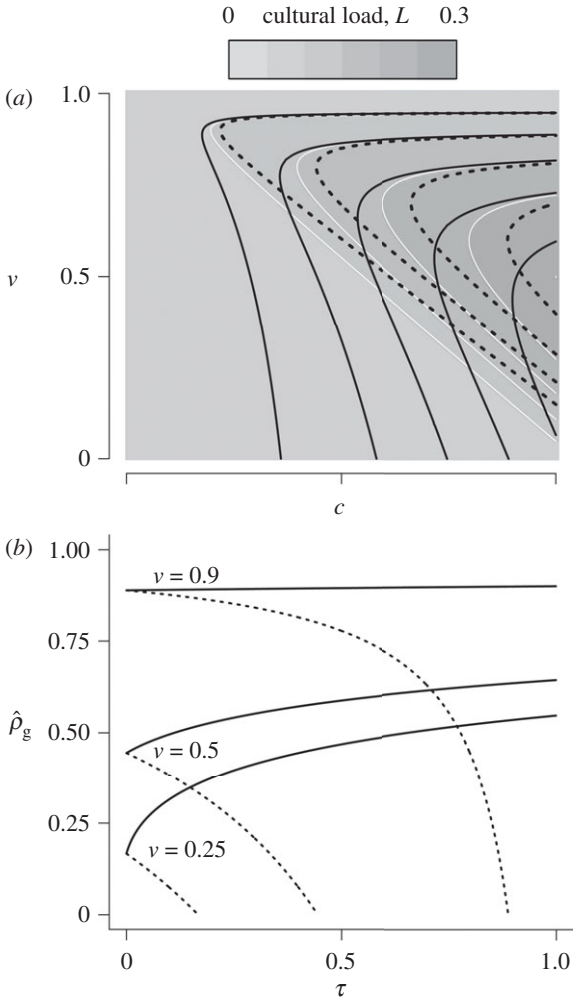

Figure 4. Effect of one-to-many communication on the cultural load at equilibrium (*a*) and the equilibrium trait frequency (*b*) when the central node is either a persistent source of misinformation ($\lambda_l = 0$; $\hat{\rho}_l = 1$; equation (2.6) and (2.7); solid lines) or is never a source of misinformation ($\lambda_l \to \infty$; $\hat{\rho}_l = 0$; equation (2.7) and (2.8); dotted lines). (*a*,*b*) $\mu = 0$, $\beta = 1$, $\lambda_g = 1$; in (*a*) $\tau = 0.1$, with shading and contours showing the load when $\tau = 0$, plotted with the same intervals and palette as in figure 2; (*b*) $c = 0.9$.

3.3. One-to-many communication

To this point, all networks we have discussed have allowed the bidirectional flow of information through links connecting individual nodes. As some modern communications technology allows only the unidirectional flow of information, from one individual (central node) to many (e.g. television, radio), it is important to study the effect of this more extreme type of network on cultural load. As shown analytically in equation (2.5), the trait frequency and the load at equilibrium are independent of the rate of one-to-many communication (τ) if the individual occupying the central node performs the elimination process at the same rate as the rest of the population ($\lambda_l = \lambda_g$). If instead, the individual occupying the central node acts as a persistent source of misinformation ($\rho_l = 1$) and never performs the elimination process ($\lambda_l = 0$), then the equilibrium frequency and load at equilibrium are increased considerably. The equilibrium frequency and load contributed by traits with high v is only slightly affected by τ when the central node is a persistent source of misinformation (figure 4), as most individuals carry the trait. On the other hand, when the central node performs the elimination process at a higher rate than the rest of the population ($\lambda_l \to \infty$) and is therefore never a source of misinformation ($\hat{\rho}_l = 0$), there is a pronounced decrease in equilibrium trait frequency and cultural load with increasing τ (equations (2.7) and (2.8); figure 4, dashed lines). In this case, when individual communication is more strongly oriented to the central node (high τ), the proportion of communication events with others in the general population is decreased (low $1 - \tau$), reducing the equilibrium frequency and the load. These results show that in contrast to the other network architectures examined here (scale-free, small-world), one-to-many communication has no effect on the cultural load at equilibrium if learning rules in the central node are not altered. But if these learning rules are altered,

changing the trait frequency at the central node, then one-to-many communication can have very strong effects on the trait frequency and cultural load at equilibrium. Therefore, this provides another example of how variation among individuals in the rate of elimination can affect the cultural load.

4. DISCUSSION

Cultural traits with deleterious consequences can be maintained in a population when the processes of communication and transmission cause them to spread faster than they are eliminated. Such traits that are maintained in a population at an equilibrium frequency thus constitute a 'cultural load' of misinformation, which represents the average intrinsic value (e.g. effect on fitness, welfare or accuracy of belief) of deleterious cultural traits present in a population in the same way that the 'genetic load' represents the fitness cost of the segregation of deleterious mutations [32–35]. As the load contributed by a given trait depends on both its transmissibility (c) and intrinsic value (v), the average load in a population will depend strongly upon the actual distribution of c and v of all traits arising in the population. Here, we have shown that the cultural load for traits with different c and v (the load profile) is very strongly affected by variations in the mean rates of the communication versus elimination processes (figure 2) and by variation among individuals in the rates they perform the elimination process (figure 3). While we also found that network architecture affected the load profile, with more load under scale-free architectures and less load under small-world architectures (figure 2), the effect of such variations was much more limited than the effect of changes in the processes of communication and elimination. Interestingly, the rate of one-to-many communication had no effect on load unless there were also changes in the rate of elimination at the central node; when the central node never discarded deleterious traits, considerable load could be generated under this architecture (figure 4).

The network models that we have studied may span the extremes of the spectrum of architectures that have likely existed over human history; most human communication networks are thought to fall somewhere between the minimally interconnected small-world networks and the highly heterogeneous scale-free networks [5–9]. We found that an increase in the rate of communication of only approximately 18 per cent could result in a load profile and $\bar{L}$ similar to that of a scale-free network, whereas a decrease of approximately 36 per cent could yield a load profile similar to the small-world network with $p_w = 0.001$. Much larger changes in rates of communication have probably occurred through successive innovations in communications technology, with the economic costs of communication across long distances decreasing considerably as technologies relying on the physical transportation of information (e.g. traditional mail) have been replaced by those that use digital communication (e.g. email). As such, our results suggest that innovations in communications technology have probably affected cultural load more strongly through changes in the rates of communication than through changes in the architecture of the network (who communicates with whom). Thus, while considerable research has focused on how different network architectures affect the dynamics of spreading processes [23,40,41], when applied to problems of cultural evolution, understanding how various aspects of human ecology affect the rates of transmission versus elimination is probably much more important.

Surprisingly, under our modelling assumptions, we found that there was no detectable effect of any correlation between an individual's network connectivity (their individual rate of communication) and their individual rate of elimination (η) on the cultural load at equilibrium (§3.2). While it might be intuitively expected that the cultural load would be reduced when the most highly connected individuals perform the elimination process most often because more communication events would involve individuals that were likely to have purged their deleterious traits, this was not observed (figure 3). However, the load at equilibrium was strongly affected by any increases in the variance in individual rates of elimination (η), suggesting that any genetic or social factors increasing this variance will always increase the load. As increases in social complexity may often be accompanied by individual specialization for certain tasks or professions [42], any such changes in social structure might have been accompanied by some abdication of the 'labour' of evaluating the value of information less relevant to one's specialty. As this practice would increase the variance in η, our results show that it would also increase cultural load in the absence of other changes in learning rules. Therefore, it would be interesting to study the effect of more complex learning rules on the cultural load, such as preferentially learning a trait from individuals with high η for that particular trait (i.e. non-specialists learning preferentially from specialists). In any case, the general effect of varying individual elimination rates in SIS-type models does not seem to have been considered in previous studies of epidemic spreading on networks [23,40,41].

One important assumption implicit in our approach is that there is no evolution and that variation among individuals in their network connectivity and their rates of elimination are fixed, rather than coevolving with culture. To the extent that intrinsic value represents an effect on biological fitness, variation among individuals in the number of deleterious traits they carry will induce natural selection on any factor that affects their own personal cultural load (through changes in the transmission or elimination processes). Such factors could include genetically based traits (according to Gavrilets & Vose [43]), culturally evolved behavioural strategies at the individual level [44] or culturally evolved characteristics of groups that compete with each other for territory or resources (e.g. division of labour or social organization; [28,45,46]). Two other important assumptions of our approach are that all traits have the same intrinsic value for all individuals, and that the rate of discarding of a trait (r) is independent of

the values of r for all other traits carried by an individual and that this rate is directly proportional to the trait's value ($r = 1 - v$). In practice, these assumptions will likely hold only for a small fraction of cultural traits. It would be very interesting to study how the spread of traits that are beneficial for a minority of individuals but deleterious for a majority would be affected by different network architectures. Our model of one-to-many communication without elimination at the central node gives some indication of the possible magnitude of this effect (figure 4*a*, solid lines), and also suggests that different results might be found with directed networks. Further models that relax these assumptions and increase the realism of how traits are transmitted/discarded are necessary to expand our understanding of how misinformation spreads and persists in populations. In particular, as we have shown that considerable cultural load can be maintained at equilibrium, it will be interesting to study how different strategies might evolve that minimize load, both at the individual and at the group level.

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