

Emotions as infectious diseases in a large social network: the SISa model

Alison L. Hill^{1,2,6,*}, David G. Rand^{1,3,†}, Martin A. Nowak^{1,4}
and Nicholas A. Christakis^{5,7}

¹Program for Evolutionary Dynamics, ²Biophysics Program, ³Berkman Center for Internet and Society, ⁴Departments of Mathematics and Organismic and Evolutionary Biology, and ⁵Department of Sociology, Harvard University, Cambridge, MA 02138, USA

⁶Harvard-MIT Division of Health Sciences and Technology, Massachusetts Institute of Technology, Cambridge, MA 02139, USA

⁷Departments of Medicine and Health Care Policy, Harvard Medical School, Boston, MA 02115, USA

Human populations are arranged in social networks that determine interactions and influence the spread of diseases, behaviours and ideas. We evaluate the spread of long-term emotional states across a social network. We introduce a novel form of the classical susceptible–infected–susceptible disease model which includes the possibility for ‘spontaneous’ (or ‘automatic’) infection, in addition to disease transmission (the SISa model). Using this framework and data from the Framingham Heart Study, we provide formal evidence that positive and negative emotional states behave like infectious diseases spreading across social networks over long periods of time. The probability of becoming content is increased by 0.02 per year for each content contact, and the probability of becoming discontent is increased by 0.04 per year per discontent contact. Our mathematical formalism allows us to derive various quantities from the data, such as the average lifetime of a contentment ‘infection’ (10 years) or discontentment ‘infection’ (5 years). Our results give insight into the transmissive nature of positive and negative emotional states. Determining to what extent particular emotions or behaviours are infectious is a promising direction for further research with important implications for social science, epidemiology and health policy. Our model provides a theoretical framework for studying the interpersonal spread of any state that may also arise spontaneously, such as emotions, behaviours, health states, ideas or diseases with reservoirs.

Keywords: social networks; happiness; emotional contagion; SIS model; depression; social contagion

1. INTRODUCTION

Social network effects are of great importance for understanding human behaviour. People interact with different numbers of individuals and with some individuals more than others, and this affects behaviour in fundamental ways. Investigations from diverse fields, including sociology, economics, physics, mathematics and public health, have studied social networks, both to understand their structure and their affect on social dynamics. Economic behaviour, such as purchasing decisions (Lazarsfeld & Katz 1955), employment prospects (Marmaros & Sacerdote 2002; Calvo-Armengol & Jackson 2004), bankruptcy (Uzzi 1996), investment (Cohen *et al.* 2008) and productivity (Oswald *et al.* 2009) have been shown to be influenced by network contacts (reviewed in Jackson 2009). Other behaviours such as college GPA (Sacerdote 2001) and crime (Glaeser *et al.* 1996; Haynie 2001) have been related to peer effects. Recently, social networks have been studied as determinants of health (reviewed by Smith & Christakis 2008), ranging from determining the patterns of infectious disease spread (Keeling & Eames 2005) to the

propagation of behaviours such as smoking cessation (Christakis & Fowler 2008), obesity (Christakis & Fowler 2007) and suicidal ideation (Bearman & Moody 2004). Networks also play an important role in evolutionary biology, where population structure determining interactions can facilitate the evolution of cooperative behaviour (Ohtsuki *et al.* 2006; Tarnita *et al.* 2009a,b; Nowak & May 1992; Fu *et al.* 2007).

Human emotions are strongly influenced by social contacts. ‘Emotional contagion’ (Hatfield *et al.* 1994) is known to occur over short timescales between people in frequent close contact, such as families (Larson & Almeida 1999), roommates (Howes *et al.* 1985) and team-mates (Barsade 2002), and even occurs in experimental situations (Fowler & Christakis 2010). Positive and negative moods also spread during workplace interactions such as cooperation (Barsade 2002), negotiation (Van Kleef *et al.* 2004) and successful leadership (Bono & Iles 2006). The spread of positive mood is a common technique in interpersonal relations, and the ‘service with a smile’ technique is a well-known example (Pugh 2001). Depressive moods and symptoms have been found to be contagious both by media and by personal contact with strangers or acquaintances (Joiner & Katz 1999). Over longer timescales, clinically relevant depressive states can also spread between social contacts (Ueno 2005). Unlike most of these previous studies, in the present work we demonstrate the interpersonal

* Author for correspondence (alhill@fas.harvard.edu).

† These authors contributed equally to this work.

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spread of long-lasting emotional states over protracted timescales.

Recently, Fowler & Christakis (2008) have studied the properties of happiness in relation to a network of social contacts, extracted from the Framingham Heart Study (Dawber 1980). They observe correlations in happiness between individuals who are connected in a social network. If your friends are happy, then you are more likely to be happy in the future. This correlation, they suggest, is caused in part by an inductive effect of happiness spreading from person to person. Thus, happiness could be thought of as a form of social infection. Here, we evaluate this conceptual approach to emotional transmission in a mathematically rigorous way, by formally modelling the spread of positive and negative emotions using theoretical tools from epidemiology.

Mathematical models for the spread of microbial infections help us to understand and predict the spread of disease (Anderson & May 1991). Such models have also been used to explore the spread of non-microbial infections, such as rumours (Daley & Kendall 1964) or computer viruses (Pastor-Satorras & Vespignani 2001). Studying the dynamics of epidemiological models on social networks is currently an active area of research. Many models have been developed to study how the structure of networks affects disease spread (Keeling & Eames 2005). These models have been used to understand contagious diseases where the network describes contacts in geographical proximity (for example severe acute respiratory syndrome (SARS), Meyers *et al.* 2005; foot-and-mouth disease, Ferguson *et al.* 2001) or for studying sexually transmitted diseases where the network consists of sexual partnerships (Eames & Keeling 2002).

Here, we introduce a novel approach for studying the spread of emotions in a social network. We can think of emotions at three levels: ‘moods’, ‘states’ or ‘traits’. These levels have increasing duration and permanence, and differ in terms of the extent to which they are deeply characteristic of the individual, and therefore the extent to which they are mutable or immutable. We will use the word ‘long-term emotional states’ to describe the phenomena of interest to us here, lying in between fleeting moods and permanent traits (like personality traits). We use the terms *content* and *discontent* for these phenomena that have been characterized, in prior work, as akin to happiness and depression, with validated scales. We modify a classical infectious disease model to represent the spread of emotions, to account for the fact that emotions can be contracted both spontaneously and through transmission. We then use this model to ask whether emotional states in the Framingham Heart Study dataset (Dawber 1980) do indeed act like infectious diseases. For an emotional state such as content to fit the classical definition of an infectious disease, (i) the probability of becoming content must depend on the number of content contacts (and not on the number of non-content contacts), and (ii) the probability of switching from content back to neutral (‘recovering’ from content) should be independent of any properties of social contacts. The particular structure of the Framingham Heart Study dataset, with a social network, reliable emotional evaluation and longitudinal follow-up, allows application of our model to the spread of content and discontent over long timescales. We find that both content

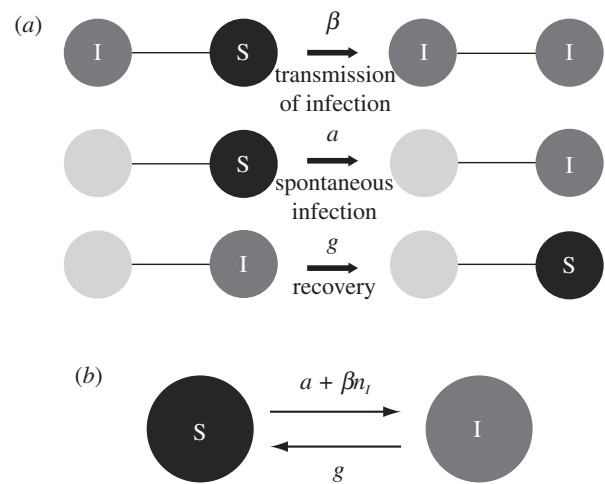


Figure 1. The SISa model of infection. (a) There are three processes by which an individual’s state can change. (i) An infected individual transmits infection to a susceptible contact with rate β . (ii) A susceptible individual spontaneously (‘automatically’) becomes infected at rate a , regardless of the state of her contacts. (iii) An infected individual returns to being susceptible at rate g , independent of the state of her contacts. (b) The rates of movement for an individual between the susceptible and infected states. n_I is the number of infected contacts.

and discontent behave like infectious diseases. The data allow us to then estimate values for model parameters, and to calculate derived quantities which give insight into the dynamics of emotional contagion.

2. MATERIAL AND METHODS

(a) Basic infectious disease model

In the simplest infectious disease models (Anderson & May 1991), individuals are classified as occupying one of two states: ‘susceptible’, meaning they do not have the disease, and ‘infected’, meaning they do have the disease. The disease can be transmitted to a susceptible person when they come into contact with an infected person. The rate of this disease transmission from infected to susceptible is defined as β , the *transmission rate*. Once an individual is infected, he/she recovers from the disease at a constant rate g , regardless of his/her contact with susceptible or infected others. In one class of disease models (susceptible–infected–recovered (SIR)), recovered individuals become immune to further infection and enter a ‘recovered’ state. However, an emotion can occur many times over an individual’s life, and therefore we use assume infected individuals return to the susceptible state after recovering. This form of susceptible–infected–susceptible (SIS) model is used to model infectious diseases that do not confer immunity, such as many sexually transmitted diseases.

In the standard SIS model, infection can only be transmitted by having a contact between an infected and a susceptible individual. Emotional ‘infections’, however, can also arise owing to spontaneous factors other than transmission. Therefore, we extend the SIS model by adding a term whereby uninfected individuals spontaneously (or ‘automatically’) become infected at a constant rate a , independent of infected contacts. A diagrammatic representation of our modified SIS model, which we will call SISa, is shown in figure 1.

The corresponding differential equations for a well-mixed population are described in equation (2.1). When the population is not well-mixed but instead constrained on a social network, the transmission rate for each individual depends on the number of infected contacts (n_I) instead of the total number of infected individuals (I).

$$\left. \begin{aligned} \frac{dS}{dt} &= -\beta SI + gI - aS, \\ \frac{dI}{dt} &= \beta SI - gI + aS \end{aligned} \right\} \quad (2.1)$$

and $I + S = N$,

where S is the number of susceptible individuals, I the number of infected individuals β the transmission rate, g the recovery rate, and a the rate of spontaneous infection. This model assumes a constant population size, neglecting birth and death.

(b) Parameter estimation

The classical definition of an infectious disease in the SIS/SIR context is that (i) the probability of an individual transitioning from susceptible to infected is an increasing function of the number of infected contacts, while (ii) the probability of a transition from infected to susceptible (i.e. recovery) is independent of the number or state of contacts. Exposure to infected individuals makes you more likely to become sick, but once you are infected you recover at a constant rate regardless of contacts. In addition to infectious contagion, the SISa model has an additional spontaneous mode of acquiring disease. To both test that a behaviour displays infectious-disease-like dynamics, and to estimate values for the model parameters a , β and g , we can use dynamic information about transitions between states based on multiple time points in longitudinal data. For data points separated by time intervals (Δt) smaller than the average time between transitions, the transition probabilities can be linearized. The probability of a transition from susceptible to infected after a time Δt can be given by $P(S \rightarrow I, \Delta t) \sim (a + \beta n_I)\Delta t$, and the probability of transition from infected to susceptible after time Δt , by $P(I \rightarrow S, \Delta t) \sim g\Delta t$. It is necessary for the time between measurements to not be comparable to or greater than the average lifetime of a state to keep the probability of double transitions within a time interval low.

(c) Dataset

In this study, we evaluate the assumption that emotions behave like infectious agents. We use data from the Framingham Heart Study (Dawber 1980) and the framework discussed above to test whether content and discontent fit the dynamics of infectious diseases. The Framingham study was initiated in 1948 in Framingham, Massachusetts and has continued enrolling subjects till the present. Subjects were examined at regular intervals of approximately three years. They were administered a psycho-social exam called the CES-D, which allowed us to classify each individual as *content*, *discontent* or *neutral*. We used the well-defined cut-off of a score of 16 on the full CES-D scale to classify subjects as *discontent*. This measure has previously been called ‘depression’, though it is not specific to clinical depression and represents

general distress or depressive symptoms (Rush 2007). *Content* was defined as a score of 12 on the positively worded questions of the CES-D, and is the same subscale used by others and called ‘positive affect’, ‘well-being’ or ‘happiness’ (Moskowitz 2003; Ostir et al. 2000; Pressman & Cohen 2005; Fowler & Christakis 2008). The terms ‘content’, ‘discontent’ and ‘emotional state’ as used in this paper represent long-term states reflecting general life satisfaction, as opposed to short-lived moods evinced in response to stimuli. Additionally, social networks were constructed for each subject based on recorded information on friends, family, co-workers and residential neighbours. For details, see the electronic supplementary material.

We hypothesize that a neutral emotional state may act like a susceptible state, and that content and discontent act like ‘infected’ states (figure 1). We validate this classification using the data analysis presented below. Note that we exclude the possibility of *coinfection* by both content and discontent simultaneously, as this doubly infected state very rarely appears in the data (see the electronic supplementary material, figure S1). However, *superinfection* may be possible: individuals could potentially move directly between being content and discontent without passing through neutral. The data are used to validate the model assumptions and estimate values for the parameters β , a and g . These parameters are then used to calculate derived quantities of interest, such as the average lifetime of an emotional infection, the probability an infected person passes the infection to a given contact sometime during the course of her infection, and the average frequency with which infections arise spontaneously. Additionally, agent-based simulations are used to compare model dynamics and clustering with the observational data.

(d) Measuring transmission of emotions

To study the transmission of emotions, we examine changes in emotional state between the two sequential exams of the Framingham Heart Study in which the CES-D was administered (exams 6 and 7). The average fraction of the network that was classified as content or discontent was very similar between the two exams, suggesting the transmission process is at steady state (exam 6: 65% content, 9% discontent; exam 7: 62% content, 9% discontent). Additionally, these exams were closely and consistently spaced ($\Delta t = 3 \pm 1$ year). We include only subjects who answered all questions on the CES-D in both exams.

Subjects were initially classified as first as either ‘content’ or ‘not content’, and then separately as ‘discontent’ or ‘not discontent’. Individuals who are neither ‘content’ nor ‘discontent’ are classified as ‘neutral’. As expected, very few people who are classified as ‘content’ are also classified as ‘discontent’ (less than 1/1000). Thus we have three possible emotional states. A diagram of the distribution of subjects between emotional states is included in the electronic supplementary material, figure S1.

A given state x is considered infectious if having more contacts in state x makes you more likely to switch to state x . That is, a positive relationship between the number of contacts in state x and the probability to

transition from state y to state x indicates that state x is infectious with respect to state y . Therefore, to test whether a given state x is infectious with respect to another state y , we perform an ordinary least squares (OLS) linear regression as follows. Each subject in state y in exam 6 is coded as either having transitioned to state x (transition = 1) or not (transition = 0) in exam 7. We then regress this binary transition variable for each subject against that subject's number of contacts in state x during exam 6. A significant positive correlation indicates that having more friends in state x at the earlier exam makes you more likely to switch to state x in the later exam. If state x is infectious (a significant positive correlation exists), then the value of β can be calculated from the slope of the regression line, and the value of a can be calculated from the intercept. If state x is not infectious (no significant correlation exists), then the value of g can be calculated from the intercept. Δt was taken as the average time between examinations, which was 2.5 years. Using logistic regression as opposed to OLS regression gives very similar results, as the datapoint line is within the linear range of the logistic model.

It is important for our analysis that subjects do not change emotional states rapidly (days or weeks), but instead on the timescale of years or longer. While the CES-D asks subjects to evaluate feelings in the last week, it is known to be a reliable measure of long-term emotions, as opposed to short-lived moods evinced in response to stimuli. Several studies have shown that CES-D scale scores to be stable for up to 12 months, with high intra-individual test-retest correlations (Radloff 1977; Roberts 1980). Furthermore, we find a strong positive correlation between each subject's emotional state in the two measurement periods ($r = 0.47$). This suggests that emotional states are not fluctuating on the timescale of weeks or months, but rather on the timescale at which we are measuring: if they were changing faster than 3–5 years, then a subject's state in exam 6 would not be strongly predictive of her state in exam 7. Note that this correlation is also confirmed by a more detailed analysis (Fowler & Christakis 2008). Thus, there is strong evidence that the emotions we are measuring change only on the timescale we are measuring, as opposed to fluctuating much more rapidly.

Our epidemiological approach to social contagion has important differences from other models which look at correlations in present and past states of connected individuals (Christakis & Fowler 2007, 2008; Fowler & Christakis 2008). Here, we look at how contacts influence the transitions between states, which better captures the nature of contagion. Since we use pre-existing social ties, we do not see effects from selection bias in choosing friends with similar emotional states. Additionally, confounding events that lead to concurrent changes in connected individuals will not show up as contagion effects in this model.

3. RESULTS

In the Framingham Heart Study network, individuals may be connected to neighbours, co-workers, first-degree relatives and self-nominated friends. An individual's degree is the number of contacts that individual has to other people in the network. Restricting to only individuals who had been administered the CES-D at

both exams 6 and 7, and their connections that exist at exam 6, gives a population of size $n = 1880$, with an average degree of 3.1. The network is approximately Poisson distributed, with a long tail, although with some subjects having no connections. It has been previously found that family and friends living more than 2 miles away are not significantly associated with emotional influence (Fowler & Christakis 2008), suggesting that face-to-face contact is important for transmission. Thus, we restrict our analysis to connections involving only friends and relatives living closer than 2 miles away, next-door or same address neighbours and co-workers. With these further constraints, the degree of the network was reduced to 1.25 ± 1.35 .

The results of the infectiousness analysis for the spread of content and discontent between exams 6 and 7 are shown in figure 2. Consistent with the SISa model formulation, we find a significant positive correlation between the probability of transitioning from neutral to content and the number of content contacts (figure 2*a*, coeff. = 0.052, $p = 0.024$), and no significant relationship between the transition from content to neutral and the number of neutral contacts (figure 2*b*, coeff. = -0.011 , $p = 0.59$). Additionally, we find no significant relationship between the probability of transitioning from neutral to content and the number of neutral contacts (coeff. = 0.48, $p = 0.10$), or the probability of transitioning from content to neutral and the number of content contacts (coeff. = 0.22, $p = 0.98$). This suggests that content can indeed be modelled as an infectious process in the SISa framework, with neutral susceptibles more likely to become content infecteds with each content contact they have. The parameters for the SISa model can be calculated from the transition probabilities using equations mentioned previously, by dividing slope and intercept values by Δt , the average time between exams. The transmission rate, β , is found to be 0.02 yr^{-1} . The spontaneous infection parameter a is found to be 0.18 yr^{-1} . The recovery parameter g is found to be 0.088 yr^{-1} . These results are dependent on restricting analysis to contacts who had exams separated by less than three-and-a-half years (75%). For longer times it becomes increasingly probable that more than one transition has been made between exams.

We now repeat this analysis for discontent. We find that discontent can be also described in the SISa framework as a contagious emotion with neutral as the susceptible state. The probability of transitioning from neutral to discontent is positively correlated with the number of discontent contacts (figure 2*c*, coeff. = 0.097, $p = 0.027$), and the probability of transitioning from discontent back to neutral is independent of the number of neutral contacts (figure 2*d*, coeff. = 0.032, $p = 0.56$). Additionally, we find no significant relationship between the probability of transitioning from neutral to discontent and the number of neutral contacts (coeff. = 0.11, $p = 0.75$), or the probability of transitioning from discontent to neutral and the number of discontent contacts (coeff. = 0.002, $p = 0.99$). The model parameters are $\beta = 0.04 \text{ yr}^{-1}$, $a = 0.040 \text{ yr}^{-1}$, $g = 0.13 \text{ yr}^{-1}$. Interestingly, the rate of transmission through a contact is similar for both content and discontent, while the rate of spontaneous infection is much larger for content and the rate of recovery is larger for discontent.

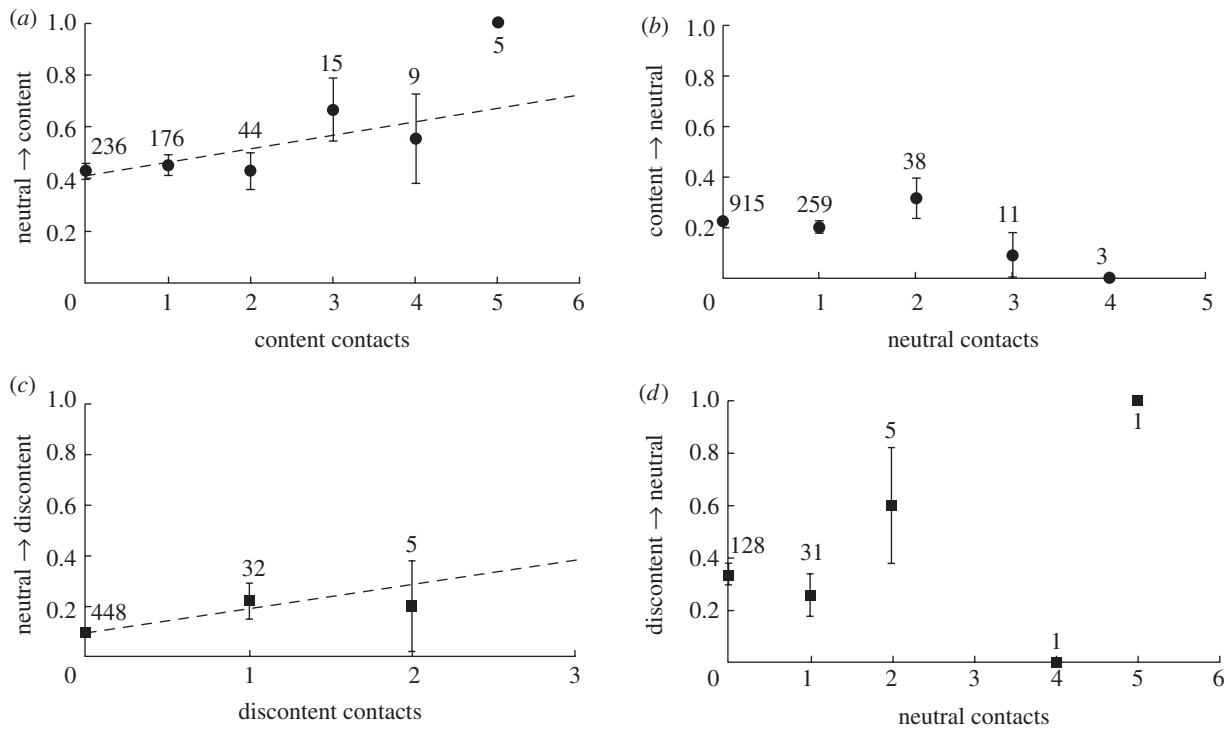


Figure 2. Both content and discontent behave like disease agents, infecting those in a susceptible neutral emotional state. (a) Becoming content: the probability of transitioning from neutral to content increases in the number of content contacts (slope = 0.052, $p = 0.024$, intercept = 0.42). (b) Recovering from content: the probability of recovering from content to the neutral state does not depend on the number of neutral contacts (slope = -0.011 , $p = 0.59$, intercept = 0.22). (c) Becoming discontent: the probability of transitioning from neutral to discontent increases in the number of discontent contacts (slope = 0.097, $p = 0.027$, intercept = 0.095). (d) Recovering from discontent: the probability of recovering from discontent to the neutral state does not depend on the number of neutral contacts (slope = 0.032, $p = 0.56$, intercept = 0.32). Error bars represent the standard error of the mean, and labels are the number of individuals averaged for each data point. We also find no significant dependence of these transitions on age, sex or education ((a): age, coeff = 0.0014, $p = 0.52$; education, coeff = 0.0024, $p = 0.81$; sex, coeff = -0.0074 , $p = 0.87$. (b): age, coeff = 0.0016, $p = 0.21$; education, coeff = -0.0095 , $p = 0.08$; sex, coeff = 0.0109, $p = 0.64$. (c): age, coeff = 0.0001, $p = 0.93$; education, coeff = 0.0008, $p = 0.90$; sex, coeff = 0.0485, $p = 0.08$. (d): age, coeff = -0.0006 , $p = 0.88$; education, coeff = -0.0165 , $p = 0.35$; sex, coeff = -0.024 , $p = 0.77$).

We also include for the possibility of direct transitions between content and discontent ('superinfection'), which are sometimes observed. We calculate the rate of direct transitions from content to discontent to be $s_{cd} = (0.009 \pm 0.003)$ per year and the rate from discontent to content to be $s_{dc} = (0.07 \pm 0.02)$ per year. See the electronic supplementary material for details. The estimated rates of all transitions are visualized in figure 3.

From the SISa model parameter estimates, various quantities of interest can be calculated. The average lifetime of an emotional state is the average length of time an individual spends in this state before recovering. For both content and discontent, 'recovering' usually means going to the neutral state (g_c or g_d), but also includes the possibility of transitioning directly to the other 'infected state' (s_{cd} or s_{dc}). The total recovery rate (g_T) is the sum of both these rates. The average lifetime of a contentment infection is 10.1 years, and for discontentment it is 5.0 years. Thus, the average contentment infection appears to last twice as long as the average discontentment episode. The 'influence' of an emotion is the cumulative probability that the infection will be passed from an infected to a susceptible connection before the infected individual recovers. This is calculated as $1 - e^{-\beta/g_T} \approx \beta/g_T$. For both content and discontent, this value is 0.18. The 'cycle length' is the average length of time between spontaneous infections, and is

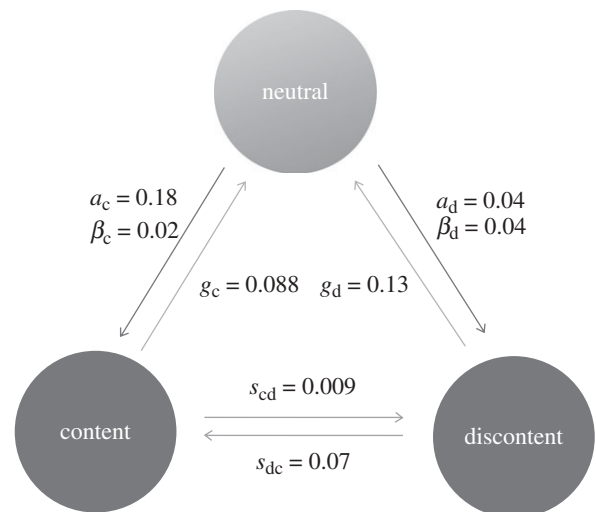


Figure 3. Parameter estimates for content and discontent using the SISa model framework. From these results, derived from figure 2, we show that content and discontent are infectious emotions that can be 'caught' by neutral (susceptible) individuals from content or discontent (infected) contacts. There is also some 'superinfection' present: direct transitions between content and discontent. a_c (a_d) = rate of spontaneously becoming content (discontent), g_c (g_d) = rate of recovery from content (discontent), β_c (β_d) = transmission rate of content (discontent), s_{cd} (s_{dc}) = rate of spontaneous superinfection from content to discontent (discontent to content).

5.6 years for content and 25 years for discontent. This suggests that an individual may spontaneously become content many times throughout life.

In the regular SIS or SIR models, the average number of secondary infections caused before recovery is called the *basic reproductive ratio*, R_0 . An epidemic only occurs for $R_0 > 1$. In the SISa model, there is no thresholding behaviour as seen in traditional models, because of the spontaneous infection term. Even if there are no individuals initially infected and no contagion ($\beta = 0$), spontaneous infection will nonetheless lead to a non-zero steady-state level of infected. In the absence of spontaneous infection, the effective R_0 can be estimated as $\beta n/g$, where n is the average degree. For content, R_0 is 0.28 and for discontent it is 0.39. Since for both R_0 is less than one, self-sustained epidemics of either emotion could not be sustained if people did not become spontaneously infected.

We also perform further analysis to examine differences in local network structure for individuals in different emotional states. Content individuals have on average 1.26 connections to other people in the data, neutral individuals 1.37 connections and discontent individuals 0.97 connections. The smaller number of connections among discontent individuals suggests isolation, which could be contributing to or secondary to depressive symptoms. We can also ask whether people in a given state tend to cluster. Clustering is a measure of the spatial correlation (in the network sense) between individuals in the same emotional state. Clustering is defined as the ratio of the observed number of connections between two types of individuals to the number of connections expected if the positioning of individuals in the network was random. Clustering of like individuals can be caused by an infective process spreading within a network (Keeling 1999), but may also be caused by confounding environmental factors influencing behaviour, or selection of contacts based on similar behaviour. In a purely infectious process on a network, the average correlation between two infected individuals will be greater than one, and the average correlation between an infected and a susceptible individual will be less than one. To compare observed clustering to that expected from a pure infection, we perform agent-based simulations of SISa epidemics on networks with the FHS network topology and calculate the predicted clustering (table 1). For content, there is good agreement between observed and predicted clustering, suggesting that the inductive spread of content can explain the observed clustering of content individuals. For discontent, however, there is less clustering in the data than predicted by the infectious disease simulations. Thus discontent people are more isolated in the network than predicted by the infectious model, which is consistent with the reduced average degree observed above. This could be explained by a tendency for depression to cause people to interact less with others, or for social isolation to lead to depression (Beck & Alford 2009).

4. DISCUSSION

Here, we have shown that long-term emotional states can spread between socially connected individuals. The transmission rate for content is found to be $\beta = 0.02 \text{ yr}^{-1}$,

and the spontaneous infection parameter is found to be $a = 0.18 \text{ yr}^{-1}$, thus each content contact increases the probability per year of becoming content by 11%. For discontent, the rate of spontaneous infection is $a = 0.040 \text{ yr}^{-1}$ and the rate of transmission is $\beta = 0.04 \text{ yr}^{-1}$, so each discontent contact makes an individual 100% more likely to become discontent each year. The average lifetime of a contentment ‘infection’ is measured to be 10 years, and a discontentment ‘infection’, 5 years. We have introduced a novel framework for formalizing social contagion derived from the study of infectious diseases, which can be used to study the spread of emotions or other social phenomena. This infectious disease approach has several advantages over methods which examine correlations in behaviours of connected individuals. Our method controls for selection bias in choosing contacts with similar behaviour, as well as confounding environmental factors synchronously affecting contacts. Moreover, we find no dependence of the probability of changing emotional state on age, sex or education, which have been found in previous studies of content and discontent to affect the propensity for emotions and confound the correlations between contacts (Fowler & Christakis 2008; Angst 1992). This lack of dependence when we focus on transitions suggests that our approach is more robust to variation across individuals than previous methods.

	content	discontent	neutral
average degree	1.26	0.97	1.37
observed clustering	$C_{cc} = 1.04$ $C_{cn} = 0.91$	$C_{dd} = 1.07$ $C_{dn} = 0.77$	—
simulated clustering	$C_{cc} = 1.09$ $C_{cn} = 0.95$	$C_{dd} = 1.92$ $C_{dn} = 1.0$	—

Much of the appeal of our approach is the significance of our results despite the great simplicity of the model used, and the assumptions made about the dataset. Determining the various transition rates for the model requires the implicit assumption that all individuals are identical in their susceptibility to various emotional states. These rates therefore represent ensemble averages over the population. In reality, individuals are likely to differ in their predispositions to various emotional states. The ability to observe transmission of emotional states over long time frames requires some particular properties of the Framingham Heart Study. These include large sample sizes, longitudinal follow-up, extensive information on social contacts and various relationships, closely and consistently spaced exams, and reliable measures of emotions and behavioural states. This study was not designed to study social networks or mental health, and so there are some drawbacks that could be improved upon in more specifically designed projects.

For example, we only examine changes between two time points, which give us stationary values for the rates of transmission, although it is plausible that these rates themselves change over time. Future work exploring the dynamic spread of emotions with higher resolution data collection is called for. This may also allow for more complex and perhaps realistic models, such as modeling emotions as continuous variables rather than discrete states, with transmission depending on the severity of infection.

One possible limitation of this study is the social network dataset used. We have also assumed that the social network collected through the Framingham Heart Study is the same as the contact network for the spread of emotions, which is almost certainly not true given the sparseness of the Framingham network. Because the Framingham Heart Study was not designed as a study of social networks, no attempt was made to capture all of a person's important contacts. Many close friends of a person could be missing (usually only one was recorded) and family and co-workers who are not part of one's actual social network may have been counted. Even if under-sampling of real-world contacts did occur, it does not change our results qualitatively: our data clearly show that rates of becoming 'content' or 'discontent' increase with the number of 'infected' contacts (i.e. are contagious) while the rates of 'recovery' to a 'neutral' state do not depend on contacts. However, it could change the measured rate constants. If we missed a constant number of contacts for each person, the intercept of the transition graphs would be shifted to the right, then the actual a would be smaller than the a we measured. If we missed a constant fraction of contacts for each person, then the x -axis would be compressed and the slope would be increased, so then the actual value of β would be smaller than the β we measured. However, while it is probable that the Framingham network underestimates the total number of contacts, the relationship to the number of 'influential' contacts is unclear. In this sense, the observed value of the transmission rates, β , are network dependent. Additionally, network connections may be weighted differently according to their ability to transmit emotions. It would be ideal to design longitudinal study with the intent of measuring social networks and health and carefully define contacts, such as by amount of time spent together per day, influence, etc.

A CES-D score of greater than 16 (which we defined as 'discontent') can occur with major depression (94% specific) as well as alcohol or drug dependence, anxiety, dysthymia or bipolar disorder. Thus, it is a measure of depressive symptoms or general distress and not necessarily clinical depression (Rush 2007; Radloff 1977). The 10 year prevalence of all affective disorders is close to 30 per cent (Angst 1992) which suggests that a large portion of the population is susceptible to depressive symptoms. This fact is consistent with our ensemble treatment, and with the observed cycle length for discontent of 25 years. Treating discontent as a spontaneously acquired or transmitted infection has one major drawback: it assumes that the probability of becoming discontent is independent of an individual's history of depressive episodes. In reality, depression is known to be a recurrent condition, with relative risk increasing with history of illness (Angst *et al.* 2003). It has been found that 72 per cent of people with major or minor

depression experience recurrent episodes (Kessler *et al.* 1997). No comparable data exist for the CES-D measurement of content, which is less routinely used. Relevant data for comparison will probably become available as the study of 'positive psychology', and especially its relation to health (Pressman & Cohen 2005), becomes more widespread. There have been no studies to our knowledge which examine the lifetime or cycle of discontent or content in the general population, as defined by the CES-D.

Our study provides evidence for a transmissive component to the dynamics of the emotions of content and discontent. However, this dataset does not give insight into the mechanism of infection. It is possible that displayed emotions are directly contagious (via mimicry, pheromones, etc.), or that emotions spread indirectly, for example by sharing some type of good fortune. While short-term emotional states have been suggested as a mechanism for systemically coordinating body functions to react in the most favourable way to an environmental stimulus, and as a means of 'quick-and-dirty' communication of this stimulus to others (Lewis & Haviland-Jones 2004), it is not obvious how this argument extends to the contagion of long-term emotional states. The observed fact that 'catching' content and discontent depends on social contacts, while 'recovering' to a neutral state does not, suggests that infection involves a process similar to teaching a life skill that can then persist without a constant stimulus. The mechanisms behind the transmission of long-term emotional states merit further study.

We have formally demonstrated that emotions can be thought of as infectious diseases spreading across social networks. We have introduced a novel form of mathematical infectious disease model for describing the spread of emotions which has advantages over previous methods. We have validated this model by studying content and discontent propagating across a large social network. Our results give insight into the transmissive nature of positive and negative emotions, and our model provides a theoretical framework for studying the spread of other emotions, as well as a wide range of other social phenomena. Determining to what extent particular emotions or behaviours are infectious is a promising direction for further research with important implications for social science, epidemiology and health policy.

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REFERENCES

- Anderson, R. M. & May, R. M. 1991 *Infectious diseases of humans: dynamics and control*. Oxford, UK: Oxford University Press.

- Angst, J. 1992 Epidemiology of depression. *Psychopharmacology* **106**, 71–74. (doi:10.1007/BF02246240)
- Angst, J., Gamma, A., Sellaro, R., Lavori, P. W. & Zhang, H. 2003 Recurrence of bipolar disorders and major depression. *Eur. Arch. Psychiatr. Clin. Neurosci.* **253**, 236–240. (doi:10.1007/s00406-003-0437-2)
- Barsade, S. G. 2002 The ripple effect: emotional contagion and its influence on group behavior. *Admin. Sci. Q.* **47**, 644–675. (doi:10.2307/3094912)
- Bearman, P. S. & Moody, J. 2004 Suicide and friendships among American adolescents. *Am. Public Health Assoc.* **94**, 89–95.
- Beck, A. T. & Alford, B. A. 2009 *Depression: causes and treatment*. Pennsylvania, PA: University of Pennsylvania Press.
- Bono, J. E. & Iliès, R. 2006 Charisma, positive emotions and mood contagion. *Leadership Q.* **17**, 317–334. (doi:10.1016/j.leaqua.2006.04.008)
- Calvo-Armengol, A. & Jackson, M. O. 2004 The effects of social networks on employment and inequality. *Am. Econ. Rev.* **94**, 426–454. (doi:10.1257/0002828041464542)
- Christakis, N. A. & Fowler, J. H. 2007 The spread of obesity in a large social network over 32 years. *N. Engl. J. Med.* **357**, 370–379. (doi:10.1056/NEJMsa066082)
- Christakis, N. A. & Fowler, J. H. 2008 The collective dynamics of smoking in a large social network. *N. Engl. J. Med.* **358**, 2249. (doi:10.1056/NEJMsa0706154)
- Cohen, L., Frazzini, A. & Malloy, C. 2008 The small world of investing: board connections and mutual fund returns. *J. Polit. Econ.* **116**, 951–979. (doi:10.1086/592415)
- Daley, D. J. & Kendall, D. G. 1964 Epidemics and rumours. *Nature* **204**, 1118. (doi:10.1038/2041118a0)
- Dawber, T. R. 1980 *The Framingham study: the epidemiology of atherosclerotic disease*. Cambridge, MA: Harvard University Press.
- Eames, K. T. D. & Keeling, M. J. 2002 Modeling dynamic and network heterogeneities in the spread of sexually transmitted diseases. *Proc. Natl Acad. Sci. USA* **99**, 13 330–13 335. (doi:10.1073/pnas.202244299)
- Ferguson, N. M., Donnelly, C. A. & Anderson, R. M. 2001 The foot-and-mouth epidemic in Great Britain: pattern of spread and impact of interventions. *Science* **292**, 1155–1160. (doi:10.1126/science.1061020)
- Fowler, J. H. & Christakis, N. A. 2008 Dynamic spread of happiness in a large social network: longitudinal analysis over 20 years in the framingham heart study. *Br. Med. J.* **337**, a2338. (doi:10.1136/bmj.a2338)
- Fowler, J. H. & Christakis, N. A. 2010 Cooperative behaviour cascades in human social networks. *Proc. Natl Acad. Sci. USA* **107**, 5334–5338. (doi:10.1073/pnas.0913149107)
- Fu, F., Chen, X. J., Liu, L. H. & Wang, L. 2007 Social dilemmas in an online social network: the structure and evolution of cooperation. *Phys. Lett. A* **371**, 58–64. (doi:10.1016/j.physleta.2007.05.116)
- Glaeser, E. L., Sacerdote, B. & Scheinkman, J. A. 1996 Crime and social interactions. *Q. J. Econ.* **111**, 507–548. (doi:10.2307/2946686)
- Hatfield, E., Cacioppo, J. T. & Rapson, R. L. 1994 *Emotional contagion*. Cambridge, UK: Cambridge University Press.
- Haynie, D. L. 2001 Delinquent peers revisited: does network structure matter? *Am. J. Sociol.* **106**, 1013–1057. (doi:10.1086/320298)
- Howes, M. J., Hokanson, J. E. & Loewenstein, D. A. 1985 Induction of depressive affect after prolonged exposure to a mildly depressed individual. *J. Personal. Soc. Psychol.* **49**, 1110–1113. (doi:10.1037/0022-3514.49.4.1110)
- Jackson, M. O. 2009 Networks and economic behavior. *Annu. Rev. Econ.* **1**, 489–513. (doi:10.1146/annurev.economics.050708.143238)
- Joiner, T. E. & Katz, J. 1999 Contagion of depressive symptoms and mood: meta-analytic review and explanations from cognitive, behavioral, and interpersonal viewpoints. *Clin. Psychol.: Sci. Practice* **6**, 149–164. (doi:10.1093/clipsy/6.2.149)
- Keeling, M. J. 1999 The effects of local spatial structure on epidemiological invasions. *Proc. R. Soc. Lond. B* **266**, 859–867. (doi:10.1098/rspb.1999.0716)
- Keeling, M. J. & Eames, K. T. D. 2005 Networks and epidemic models. *J. R. Soc. Interface* **2**, 295–307. (doi:10.1098/rsif.2005.0051)
- Kessler, R. C., Zhao, S., Blazer, D. G. & Swartz, M. 1997 Prevalence, correlates, and course of minor depression and major depression in the national comorbidity survey. *J. Affective Disorders* **45**, 19–30. (doi:10.1016/S0165-0327(97)00056-6)
- Larson, R. W. & Almeida, D. M. 1999 Emotional transmission in the daily lives of families: a new paradigm for studying family process. *J. Marriage Fam.* **61**, 5–20. (doi:10.2307/353879)
- Lazarsfeld, P. F. & Katz, E. 1955 *Personal influence: the part played by people in the flow of mass communications*. New York, NY: Free Press.
- Lewis, M. & Haviland-Jones, J. M. 2004 *Handbook of emotions*, 2nd edn. New York, NY: The Guilford Press.
- Marmaros, D. & Sacerdote, B. 2002 Peer and social networks in job search. *Eur. Econ. Rev.* **46**, 870–879. (doi:10.1016/S0014-2921(01)00221-5)
- Meyers, L. A., Pourbohloul, B., Newman, M. E. J., Skowronski, D. M. & Brunham, R. C. 2005 Network theory and SARS: predicting outbreak diversity. *J. Theor. Biol.* **232**, 71–81. (doi:10.1016/j.jtbi.2004.07.026)
- Moskowitz, J. T. 2003 Positive affect predicts lower risk of AIDS mortality. *Psychosom. Med.* **65**, 620–626. (doi:10.1097/01.PSY.0000073873.74829.23)
- Nowak, M. A. & May, R. M. 1992 Evolutionary games and spatial chaos. *Nature* **359**, 826–829. (doi:10.1038/359826a0)
- Ohtsuki, H., Hauert, C., Lieberman, E. & Nowak, M. A. 2006 A simple rule for the evolution of cooperation on graphs and social networks. *Nature* **441**, 502–505. (doi:10.1038/nature04605)
- Ostir, G. V., Markides, K. S., Black, S. A. & Goodwin, J. S. 2000 Emotional well-being predicts subsequent functional independence and survival. *J. Am. Geriatr. Soc.* **48**, 473–478.
- Oswald, A. J. & Wu, S. 2010 Objective confirmation of subjective measures of human well-being: evidence from the USA. *Science* **327**, 576–579. (doi:10.1126/science.1180606)
- Oswald, A. J., Proto, E. & Sgroi, D. 2009 Happiness and productivity. IZA Discussion Paper no. 4645. Institute for the Study of Laor (IZA), Bonn.
- Pastor-Satorras, R. & Vespignani, A. 2001 Epidemic spreading in scale-free networks. *Phys. Rev. Lett.* **86**, 3200–3203. (doi:10.1103/PhysRevLett.86.3200)
- Pressman, S. D. & Cohen, S. 2005 Does positive affect influence health? *Psychol. Bull.* **131**, 925. (doi:10.1037/0033-2909.131.6.925)
- Pugh, S. D. 2001 Service with a smile: emotional contagion in the service encounter. *Acad. Manage. J.* **1018**–1027. (doi:10.2307/3069445)
- Radloff, L. S. 1977 The CES-D scale: a self-report depression scale for research in the general population. *Appl. Psychol. Meas.* **1**, 385. (doi:10.1177/014662167700100306)

- Roberts, R. E. 1980 Reliability of the CES-D scale in different ethnic contexts. *Psychiatr. Res.* **2**, 125–134. (doi:10.1016/0165-1781(80)90069-4)
- Rush, A. J. 2007 *Handbook of psychiatric measures*. Washington, DC: American Psychiatric Publication.
- Sacerdote, B. 2001 Peer effects with random assignment: results for dartmouth roommates. *Q. J. Econ.* **116**, 681–704. (doi:10.1162/00335530151144131)
- Smith, K. P. & Christakis, N. A. 2008. Social networks and health. *Annu. Rev. Sociol.* **34**, 405–429. (doi:10.1146/annurev.soc.34.040507.134601)
- Tarnita, C. E., Antal, T., Ohtsuki, H. & Nowak, M. A. 2009a Evolutionary dynamics in set structured population. *Proc. Natl Acad. Sci. USA* **106**, 8601–8604. (doi:10.1073/pnas.0903019106)
- Tarnita, C. E., Ohtsuki, H., Antal, T., Fu, F. & Nowak, M. A. 2009b Strategy selection in structured populations. *J. Theor. Biol.* **259**, 570–581. (doi:10.1016/j.jtbi.2009.03.035)
- Ueno, K. 2005 The effects of friendship networks on adolescent depressive symptoms. *Soc. Sci. Res.* **34**, 484–510. (doi:10.1016/j.ssresearch.2004.03.002)
- Uzzi, B. 1996 The sources and consequences of embeddedness for the economic performance of organizations: the network effect. *Am. Sociol. Rev.* **61**, 674–698. (doi:10.2307/2096399)
- Van Kleef, G. A., De Dreu, C. K. W. & Manstead, A. S. R. 2004 The interpersonal effects of anger and happiness in negotiations. *J. Pers. Soc. Psychol.* **86**, 57–76. (doi:10.1037/0022-3514.86.1.57)