# Dynamic and game theory of infectious disease stigmas

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## Abstract

Stigmas are a primal phenomena, ubiquitous in human societies past and present. Some evolutionary anthropologists have argued that stigmatization in response to disease is an adaptive behavior because stigmatization may help people and communities reduce the risks they face from infectious diseases and increase reproductive success. On the other hand, some cultural anthropologists and social critics argue that stigmatization has strong negative impacts on community health. One recent analysis resolved this conflict by hypothesizing that stigmas had individual and group-evolutionary benefits but are now maladaptive because of an intervening societal transitions.

Here, we present the first quantitative theory of infectious disease stigmatization. Using a four-compartment model of stigmatization against a chronic disease, we show a stigma ratio, being the ratio of net transmissions by stigmatized people to net transmissions by unstigmatized people, predicts the impact of stigmatization on lifetime infection risk. When stigmatized people are segregated from the rest of the population and there are no alternative interventions that reduce transmission, stigmatization can reduce prevalence and infection risk. When stigmas do not lead to segregation but do discourage behavior change and reduce access to medical interventions, stigmatization acts to increases the lifetime risk of infection in the community. We further show that fear of stigmas can create policy resistance to healthcare access. The societal consequences of fear are worse when effective medical treatment is available. We conclude that stigma's can be adaptive, but good healthcare and leaky ostracism can make stigmas against chronic infectious disease maladaptive, and that the deprecation of stigmas is a natural transition in the modern urban societies.

Key words: stigma; ostracism; infectious disease dynamics; evolution; cultural evolution theory; group membership; math modelling; Lyapunov function; population game

# 1 Introduction

Disease has been one of the strongest evolutionary pressures on *Homo sapiens* over the duration of our species' existence, and indeed on all mammalian species. Bacteria, fungi, protozoa, worms, and all manner of parasites have caused the untimely deaths of tens of billions of our potential ancestors, driving us to evolve a complex immune system that learns about, adapts to, and remembers the infections we suffer over our lives (Frank, 2002; Kuby, 1994). This same evolutionary pressure has also been operating on human instinct for millennia, creating a parallel system of behavioral disease resistance (Schaller *et al.*, 2015) including hygiene (Curtis & Biran, 2001) and stigmatization (Kurzban & Leary, 2001). However, behavioural resistance's particulars have important dynamic consequences that differ from those of the immune system (Reluga & Medlock, 2007). While hygiene continues to be universally valued, stigmatization is now contentious.

Stigmas are defined as simplified, standardized images of the profound disgrace of a particular social group shared by a community at large (Smith, 2007). But stigmas mark more than difference. They designate someone as profoundly devalued, discredited, and disgraced (Goffman, 1963). Among humans, stigmas are socialized: community members need to be able to recognize those who pose threats to their group and to limit this stigmatized group's access to community resources and future interactions. Consequently, humans developed messages to meet these goals. To stimulate stigma-related processes (creating beliefs, and inspiring actions to isolate and remove the labeled, and to socialize others), these messages need to include content that quickly gains attention, encourages grouping and stereotyping, and provides reasons and emotional motivation to engage in devaluation and discrimination (Smith, 2011).

Despite receiving research attention since the 1960's (Goffman, 1963), stigmas remain a poorly understood component of public health. Anecdotal accounts of the impacts of stigma, such at those from the 2014 West African Ebola epidemic (Karamouzian & Hategekimana, 2014), are indeed compelling. But while authors frequently claim strong negative impacts of stigma's on health, there is limited direct testing and quantification is limited and sometimes ambiguous (Weiss, 2008; Courtwright & Turner, 2010). The difficulties faced in the study of stigma are rooted in the nature of the phenomena itself – people don't like to talk about stigmas and since stigmas are perceived rather than real, we can not test for them as we would a virus or parasite. In addition, studies of stigma are not readily transferable from place to place because of their interdependence with the social and cultural contexts where they occur (Weiss, 2008). There are a wide variety of indirect studies of disease stigmas, including laboratory psychological studies (Bishop *et al.*, 1991; Green *et al.*, 2010; Murray & Schaller, 2011), media reporting studies (Kinsman, 2012), database searches (Churcher, 2013), and victim reports (Hewlett & Amola, 2003). Unfortunately, all of these indirect approaches have serious shortcomings for quantifying the net impacts of communicable disease stigmas on public health.

Still, there is good evidence that stigmas pose major barriers to disease prevention, control, and treatment. This is particularly well-studied in the case of HIV, a chronic viral infection for which we have treatments but no cure (UNAIDS, 2004). Surveys document a fear of future stigmatization associated with HIV testing (Herek *et al.*, 2003). In a survey of a set of rural Pennsylvania MSM communities, stigma was found to be positively correlated with higher rates of risky sexual behavior (Preston *et al.*, 2007). Based on survey results of an HIV positive community in Los Angeles, California, perceived stigma from a health care provider was related to reduced access to care among HIV-infected people (Kinsler *et al.*, 2007). In a North Carolina, HIV positive patients had difficulty adhering to their treatment regimes because they feared taking ART doses in public would revealing their HIV status (Golin *et al.*, 2002).

Although stigmas can be a significant barrier to health, they are ubiquitous in human societies, geographically and historically (Smith, 2007). Related phenomena appear in other social species as well, including ants (Hughes & Cremer, 2007; Wilson, 2000), frog tadpoles (Kiesecker *et al.*, 1999), spiny lobsters (Behringer *et al.*, 2011), and chimpanzees (Goodall, 1986). Scholars have proposed several hypotheses to explain this ubiquity of stigmas despite their costs. Some scholars (Neuberg *et al.*, 2000) argue that stigmas defend group functioning. Others feel stigmas against infectious diseases may be a side effect of the evolution of discriminate sociality (Kurzban & Leary, 2001) or conversely that stigmas in general are spill-over consequences of the evolution of disease-avoidance behavior (Park, 2003; Oaten *et al.*, 2011). Infectious diseases capitalize on the social nature of groups, by spreading from one person to another (Smith & Hughes, 2014). For social species who depend on each other for survival, stigmas may have functioned to protect uninfected members and the group, by identifying infected members and ostracizing them from the group.

For most of human history, particularly during the paleolithic period, humans lived in small, family-based groups (bands), with infrequent intergroup interactions (typically for mating). For an infected person in a band, ostracism meant swift and certain death due to the infection, lack of resources and vulnerability to predators (Wilson, 2000; Smith & Hughes, 2014). For the band, ostracism stopped the spread of infection and eliminated a reservoir for future infections. This evolutionary advantage of stigmatization to the band was strongest in pre-urban times (3000 years ago and earlier), when ostracized members would not be brought into other bands.

Over the past three millennia, many conditions for the human species have changed – the population has grown, transportation facilitated widespread contact between humans, and medical practices provided treatments for infectious diseases, all of which have implications for slowly evolving behaviors (Schaller *et al.*, 2015). Smith & Hughes (2014) explained how these new conditions could eliminate any fitness advantage provided by stigmas, and, in fact, damage public health. For example, ostracism from one band no longer guarantees segregation and isolation from others. In addition, humans are noted for their sensitivity to social rejection, with initial reactions to rejection registering

like physical pain (Eisenberger *et al.*, 2003). The social cost can be so high that people will keep suspected infections secret and avoid interacting with medical systems altogether, which eliminates their ability to access medical treatments for the infection (Smith, 2011). Stigmas have been found to create similar barriers to health care access for mental illness (Corrigan *et al.*, 2014).

However, to understand how stigmatization transitions from an advantageous to a maladaptive behavior, we have to quantify the impacts of each of these changes in behavior. When do stigmatization's benefits from infectious disease risk reduction stop offsetting it's social costs? Conceptual models and theories of stigma have been proposed previously (Bos *et al.*, 2013; Oaten *et al.*, 2011; Weiss, 2008), but can not reconcile competing effects. Kalichman *et al.* (2006) presents a regression model for correlates of sexual risk behavior, including stigma, in relation to HIV infection. And within the evolutionary ecology literature, there is a good development of disease avoidance theory in the context mate selection for animals (Hamilton, 1990) and humans (Tybur & Gangestad, 2011). But we have not found any dynamic mechanistic models that allow one to investigate the evolutionary trade-offs and health impacts of stigma.

In this paper, we propose a theory based on a 4-compartment model for the interactions between stigmatization and infectious disease prevalence (Sec. 2). Per Occam's razor, this theory is the simplest we could construct that captures basic features of stigmatization and can reconcile the competing perspectives described above. Our theory projects disease prevalence and community size under steady-state conditions using demographic parameters, infection risks, and behavior (Sec. 3). We use this model to determine conditions when stigmatization hurts individuals and public health, and show how the value of stigmas to community welfare can reverse as society develops (Sec. 4). This is followed by a population game that shows how infected people should rationally respond to stigmatization and the consequent implications for improving community welfare (Sec. 5). We conclude with a summary of our results and a discussion of model limitations and directions for future study (Sec. 6).

## 2 Methods

We begin with a high-level overview of our theory. This is followed by a detailed technical (but slightly redundant) model description in Section 2.1. The model itself is formulated as a compartmental differential equation system that is derived as a mean-field limit from a Markov-chain description of the events in an individual's life. The Markov-chain description of the individual scale is also needed for the calculation of payoffs in our population game in Section 5.1.

Infectious disease stigmas have several complications that make their quantitative theory challenging. While infection is a biological state that can be empirically tested using Koch's postulates (Evans, 1995), a stigma is imposed on an individual by their neighbors and is not a testable property

of the individual. A statement that a person is stigmatized is a communication about the perception of that person by the community, rather than a statement about the person themselves. Moving a stigmatized person to a naive community may relieve them from experiencing stigmatization, at least temporarily. In addition, the assignment of infectious disease stigmas is imperfect. Humans rely on physical (often visual) and behavioral marks to identify targets for stigmatization (Park, 2003), even though infected hosts are often able to spread disease to others before or even without showing symptoms (Koelle & Wald, 2000). Other conditions having generic symptoms overlapping the specific disease can be mis-attributed. More insidiously, stigma criteria can drift because of people exploiting the situation for personal gain.

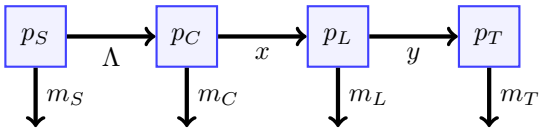


**Figure 1. State-transitions for stigmatization.** Here, $p_S$ denotes the probability of being in susceptible state, $p_C$ is the probability of being in the cryptically infected state, $p_L$ is the probability of being in the labelled state, and $p_T$ is the probability of begin in the stigmatized state. See Table 2 for parameter definitions and values.

While there are inherent difficulties in the compartmental modelling of stigmas, the dynamic analysis of stigmas can still provide useful insight. Our dynamic model is summarized in Figure 1, and studied mathematically in 2.1. The mathematical structure is closely related to the classical SEIR model (Hethcote, 2000). A person's state is categorized among four mutually-exclusive possibilities: susceptible ($S$), cryptically infected ($C$), infected and labelled ($L$), and infected and stigmatized ($T$). Note that, with the exception of the susceptible state, these names are intentionally different from the standard states of SEIR theory to emphasize their different interpretations. Susceptible people are at risk of infection when they interact with either other infected community members or an external/environmental disease reservoir. Infections are chronic and people remain infected for the rest of their lives, as in several current and historical diseases that have generated stigmas. Infected people interact with susceptible people in different ways, depending on whether the community is ignorant of their infection (cryptically infected), or has labelled or stigmatized them. Cryptically infected individuals interact with other people normally and can transmit disease. But when the community becomes aware of the diagnosis, identifies the symptoms, or observes behavior change of a cryptically infected person, that person transitions to a labelled state – our math assumes that gossip of this sort permeates a community quickly. Once people are labelled, they can access doctors, hospitals, and other medical resources publicly. However, the labelled can still transmit disease and are at risk for stigmatization by their community. Stigmas result in ostracism and reduced contacts with susceptibles. Stigmatized people may (or may not) experience higher removal and mortality rates because of stress and reduced access to healthcare. Since there is no consistent and

reliable means by which to remove a stigma (Smith & Hughes, 2014), stigmas persist indefinitely in our model, although parameter estimates can reflect a variety of coping mechanisms like escape, concealment, or acceptance, with variable success and consequences. Because we have no data with which to parameterize the alternative hypotheses, we have assumed stigma's are never mis-assigned to susceptible people.

For simplicity and illustration, we assume the infection pressure $\Lambda$, being the per-capita rate of disease acquisition by susceptibles, obeys the generalized mass-action law $\Lambda := \beta\left([C] + [L]\sigma + [T]\eta\right) + \epsilon$, where $\beta$ is the per-capita transmission rate by those cryptically infected, $\sigma$ is the relative infectiousness of the labelled, $\eta$ is the relative infectiousness of the stigmatized, $\epsilon$ is the background infection hazard created by disease reservoirs, and the square brackets are used to denote the total density of people of each type. The instantaneous infection risk for a person is $\Lambda dt$, and at steady-state, the cumulative probability that an individual becomes infected over their lifetime (a.k.a. lifetime risk) $\mathcal{K} := \Lambda/(\Lambda + m_S)$. For demographics, people immigrate into the community as susceptible at a known rate, and are removed from each of the four states at per-capita rates characteristic of each.

Our model can be easily applied to predict prevalences, community sizes, and lifetime infection risks, based on parameter estimates. We have extended the model to allow for population heterogeneity, as well as transition rates that are plastic functions of human behavior (see 5.1 and 5.2).

## 2.1 Model Details

In this section, we will describe in detail the mathematics of our model. The structure is an elementary variation of classical SEIR theory, which can be thought of as a special case of Kermack–McKendrick's more general age-of-infection theory (Kermack & McKendrick, 1927; Hethcote, 2000). However, to complete the payoff calculations in our population-game analysis in Section 5, we will also need an individual-scale version that describes the stochastic events in one person's life. Specifically, the model simultaneously combines a classical reaction-network with a 4-state Markov chain – an approach we've previously used successfully in epidemic game theory (Reluga & Galvani, 2011). The 4-state Markov chain describes the changes in an individual's disease state. The reaction-network is a system of nonlinear ordinary differential equations for disease prevalence in a large well-mixed population. We will formulate our model in a fully dynamic sense for clarity, but only steady-state calculations are needed to obtain our results. Definitions of our variables are provided in Table 1, while definitions of our parameters and estimates for model scenarios are shown in Table 2.

Transitions between states are governed by a continuous-time Markov chain summarized in Figure 1. Each person in our model is categorized in one of four possible states: susceptible, cryptically infected, infected and labelled, or infected and stigmatized. Pick a person who enters the community at time $t_0$; the probabilities that that person is in each of these states is represented respectively by $p_S(t;t_0)$, $p_C(t;t_0)$, $p_L(t;t_0)$, and $p_T(t;t_0)$, where $t$ represents time, measured in

**Table 1.** State variables used in our model. Without brackets, the variable is a probability, but with brackets, the variable is a density (people per unit area in the community).

| Symbol | Meaning |
|---|---|
| $t$ | Time measured in years |
| $p_S$ | Probability of being susceptible |
| $p_C$ | Probability of being cryptically infected |
| $p_L$ | Probability of being labelled with infection |
| $p_T$ | Probability of begin stigmatized with infection |
| $[S]$ | Density of susceptible people |
| $[C]$ | Density of cryptically infected people |
| $[L]$ | Density of people labelled with infection |
| $[T]$ | Density of people stigmatized with infection |
| $[P]$ | Density of infected people ($[C]+[L]+[T]$) |

years. A person can also leave the system because of death. Each individual is initially susceptible, so $p_S(t_0;t_0) = 1$ while $p_C(t_0;t_0) = p_L(t_0;t_0) = p_T(t_0;t_0) = 0$. A susceptible person can become cryptically infected (rate $\Lambda(t)$), a cryptically infected person can become labelled (rate $x$), and a labelled person can be stigmatized (rate $y$). Death rates may depend on the person's state $(m_S, m_C, m_L, m_T)$. In matrix form, the probabilities of being in each state change according to the matrix equation

$$\begin{bmatrix} \mathrm{d}p_S/\mathrm{d}t \\ \mathrm{d}p_C/\mathrm{d}t \\ \mathrm{d}p_L/\mathrm{d}t \\ \mathrm{d}p_T/\mathrm{d}t \end{bmatrix} = \begin{bmatrix} -m_S-\Lambda & 0 & 0 & 0 \\ \Lambda & -m_C-x & 0 & 0 \\ 0 & x & -m_L-y & 0 \\ 0 & 0 & y & -m_T \end{bmatrix} \begin{bmatrix} p_S \\ p_C \\ p_L \\ p_T \end{bmatrix} \tag{1}$$

where the infection pressure $\Lambda$ may change over time.

We use a standard nonlinear reaction network to project the ensemble population dynamics created by these individuals interactions (see Figure 2). Our reaction network is an elementary extension of classic compartmental models for epidemic dynamics (Hethcote & Levin, 1989; Hethcote, 2000; Brauer, 2008). It describes the change in community state over time $t$ under the assumption

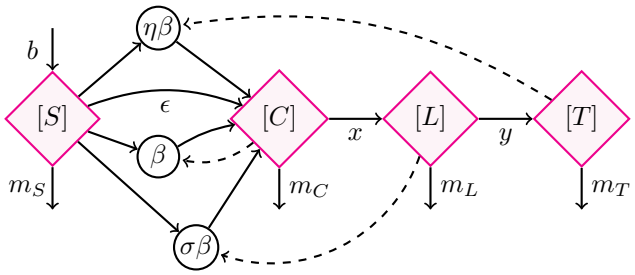


**Figure 2. States and reaction network of the population dynamics of our model.** Diamonds are population state variables, and circles are mass-action reaction nodes. Edges between states are linear reactions. Dashed edges represent reactants that are not consumed. See Tables 1 and 2 for variable and parameter definitions and values.

**Table 2.** Parameters used in our model, their meanings, and the estimated values used in our 3 scenarios. To facilitate plot comparisons, we choose the transmission rate $\beta$ so that in the absence of stigmatization or labelling ($y = x = 0$), the basic reproductive ratio $\mathscr{R}_0 = 2$. The bottom entries are derived parameters calculated from the parameter values above.

| Meaning | Parameter | Scenario 0 (Non-infectious) | Scenario 1 (Pre-Urban) | Scenario 2 (Modern) |
|---|---|---|---|---|
| Immigration rate of susceptible people | $b$ | 2 | 2 | 200 |
| Death rate while susceptible | $m_S$ | 0.02 | 0.02 | 0.02 |
| Death rate while cryptically infected | $m_C$ | 0.02 | 0.10 | 0.10 |
| Death rate while labelled | $m_L$ | 0.02 | 0.10 | 0.04 |
| Death rate while stigmatized | $m_T$ | 0.20 | 0.20 | 0.04 |
| Rate infected become labelled | $x$ | 12.0 | 1.00 | 1.00 |
| Rate labelled become stigmatized | $y$ | - | - | - |
| Background infection hazard | $\epsilon$ | $2 \times 10^{-2}$ | $10^{-3}$ | $10^{-5}$ |
| Transmission rate when cryptically infected | $\beta$ | 0 | $2 \times 10^{-3}$ | $2 \times 10^{-5}$ |
| Relative transmission rate once labelled | $\sigma$ | na | 1.0 | 0.1 |
| Relative transmission rate once stigmatized | $\eta$ | na | 0.01 | 0.5 |
| Income while susceptible | $u_S$ | na | 1 | 1 |
| Income while cryptically infected | $u_C$ | na | 0.8 | 0.8 |
| Income while labelled | $u_L$ | na | 1. | 1. |
| Income while stigmatized | $u_T$ | na | 0. | 0. |
| Baseline community size | $N$ | 100 | 100 | 10,000 |
| Lifetime infection risk | $\mathcal{K}$ | 1/2 | - | - |
| Stigma ratio | $\mathcal{Z}$ | na | 0.005 | 5 |
| Stigma threshold for behavior change | $y^*$ | na | 0.025 | 0.085 |

that the population is strongly mixed, and all individuals have equal frequencies of contact with each other. Two new states are added to the classical compartmental epidemic model of chronic infectious disease transmission to count when people have been labelled and stigmatized. We modify the notation slightly from standard practice for readability. Let $[S](t)$ be the density of susceptible people without infection. Let $[C](t)$ be the density of infected people whose state has not yet been publicly identified. These cryptically infected people may or may not know their own disease state – the important thing is that the public at large assumes they are uninfected. Let $[L](t)$ be the density of people who are infected and have had their infection publicly identified, and let $[T](t)$ be the density of infected people who have been stigmatized for their infected state. To avoid complications associated with demographic transients, we assume a constant immigration rate of susceptible people ($b$). Over time, susceptible individuals may become infected through exposure to people in any of the infected compartments. We approximate the infection pressure $\Lambda$ as a linear function of the density of infected individuals.

$$\Lambda := \beta\left([C] + [L]\sigma + [T]\eta\right) + \epsilon, \tag{2}$$

where $\epsilon$ can be interpreted as the background risk of infection from an environmental reservoir, $\beta$ is the baseline transmission rate for cryptically infected people, and $\sigma$ and $\eta$ are relative transmission

rates compared to the cryptically infected for labelled and stigmatized categories respectively. This linear approximation is equivalent to the mass-action hypothesis in a strongly-mixed community with an environmental disease reservoir, but is also good approximation near equilibrium other hypothetical forms of the infection pressure. Infected people become labelled at a constant rate, labelled people become stigmatized at a constant rate. This leads to a system of 4 ordinary differential equations with one additional algebraic constraint:

$$\frac{d[S]}{dt} = b - [S]m_S - [S]\Lambda, \tag{3a}$$
$$\frac{d[C]}{dt} = [S]\Lambda - [C]m_C - [C]x, \tag{3b}$$
$$\frac{d[L]}{dt} = [C]x - [L]m_L - [L]y, \tag{3c}$$
$$\frac{d[T]}{dt} = [L]y - [T]m_T, \tag{3d}$$
$$\Lambda = \beta\left([C] + [L]\sigma + [T]\eta\right) + \epsilon. \tag{3e}$$

Note that contact-reduction through stigmatization is a refinement of social distancing (Reluga, 2013). While social-distancing is applied equally to all contacts, stigmatizing selectively reduces contacts with specific subpopulations.

## 3 Steady-state Analysis

We will now study the steady-state properties System 3. Readers not concerned with the math details may wish to skip to Section 4. The methods we will use are entirely standard and may well be special cases of existing epidemic model analyses. When there is no external risk of infection ($\epsilon = 0$), our model is a special case of the general sequential-compartment SIR models analyzed by Guo & Li (2006), and shown to have a unique endemic steady-state that attracts all positive initial conditions. A generalized Lyapunov function is presented in Appendix A that shows the same global asymptotic stability holds when source terms are present ($\epsilon > 0$).

To understand the effect of stigmatization, let's walk through the standard analysis. In the absence of disease, the baseline community size at steady-state is $N := b/m_S$. The basic reproductive ratio, which determines the persistence of disease when background infection risks from reservoirs are small ($\epsilon \approx 0$) is

$$\mathscr{R}_0 := \frac{b\beta}{m_S}\left[\frac{1}{m_C + x} + \frac{\sigma x}{(m_C + x)\, m_L}\left(\frac{m_L}{m_L + y}\right) + \frac{\eta x}{m_T\,(m_C + x)}\left(\frac{y}{m_L + y}\right)\right]. \tag{4}$$

When there is no stigmatization ($y = 0$),

$$\mathscr{R}_0 = \frac{b\beta\,(m_L + \sigma x)}{m_S m_L\,(m_C + x)}, \tag{5}$$

while if stigmatization is instant ($y = \infty$),

$$\mathscr{R}_0 = \frac{b\beta\,(m_T + \eta x)}{m_S m_T\,(m_C + x)}. \tag{6}$$

We can see from inspection of Eq. (4) that varying the stigmatization rate $y$ monotonely interpolates between these two extremes.

We determine steady-state community sizes and disease prevalence by setting time-derivatives in System (3) to zero and deriving a quadratic equilibrium equation for infection pressure $\Lambda^*$, namely,

$$\Lambda^* = \frac{\Lambda^* m_S \mathscr{R}_0}{\Lambda^* + m_S} + \epsilon. \tag{7}$$

As long as there is some environmental reservoir seeding infection ($\epsilon > 0$), there is a single steady-state infection pressure $\Lambda^* \in (0, \beta b/m_S + \epsilon)$ which is monotone increasing with both the basic reproductive ratio ($\mathscr{R}_0$) and the reservoir pressure ($\epsilon$). The steady-state lifetime risk (the probability of a person becoming sick over their lifetime)

$$\mathcal{K} := \frac{\Lambda^*}{\Lambda^* + m_S}. \tag{8}$$

The steady-state densities $[S]^*$, $[C]^*$, $[L]^*$, and $[T]^*$ can then all be calculated using linear algebra. The gross steady-state population density is $[S]^* + [C]^* + [L]^* + [T]^*$, while the prevalence is $[P]^* := [C]^* + [L]^* + [T]^*$.

Stigmatization's influence on risk, infection pressure, and prevalence can be assessed through differentiation. The infection pressure depends on stigmatization only through the basic reproductive ratio, according to Eq. (7). Differentiating the reproductive ratio, we find

$$\frac{\mathrm{d}\mathscr{R}_0}{\mathrm{d}y} = \frac{b\beta x\,(\eta m_L - m_T \sigma)}{m_S m_T\,(m_C + x)\,(m_L + y)^2} \tag{9}$$

Only the $\eta m_L - m_T\sigma$ term in the numerator can change sign. Thus, the impact of stigmatization can be summarized by defining a dimensionless stigma ratio

$$\mathcal{Z} := \left(\frac{m_L}{\sigma\beta}\right)\left(\frac{\eta\beta}{m_T}\right) \tag{10}$$

which is the ratio of the average cumulative transmission of a labelled but unstigmatized individual, and the average cumulative transmission of a stigmatized individual. Then the sensitivity of the

basic reproduction number

$$\frac{\mathrm{d}\mathscr{R}_0}{\mathrm{d}y} = \frac{b\beta x\sigma m_T\left(\mathcal{Z}-1\right)}{m_S m_T\left(m_C+x\right)\left(m_L+y\right)^2} \tag{11}$$

While the $\beta$'s cancel each other out in Eq. (10), we have left them in to clarify the biological interpretation. The important terms in the stigma ratio are the relative transmission rates of labeled verses stigmatized, and the death rates of labeled verses stigmatized. If $\mathcal{Z}=1$, then basic reproductive ratio is independent of the rate of stigmatization. If $\mathcal{Z}>1$, then more stigmatization increases the reproductive ratio, but if $\mathcal{Z}<1$, then more stigmatization decreases the reproductive ratio. Thus, the relationship between the stigmatization rate and the basic reproductive ratio is monotone, with a sign determined by the stigma ratio. Since the infection pressure is a monotone function of the basic reproductive ratio, the same holds for it – if $\mathcal{Z}>1$, faster stigmatization increases the infection risk, but if $\mathcal{Z}<1$, faster stigmatization decreases the infection risk.

The stigma ratio $\mathcal{Z}$ depends on the mortality rates $m_L$ and $m_T$ which can be altered by medical treatment and state-correlated stresses. The value of the stigma ratio also depends on the relative transmission rates $\sigma$ (for the labelled) and $\eta$ (for the stigmatized), which incorporate changes in contact rates and changes the probability of infection per contact. Changes in contact rate and infectiousness can be attributed to differences in medical treatment and behavior because of social state. For example, the stigmatized might try to escape social stresses by avoiding medical treatment and beneficial behaviors that reveal their disease state, leading to a relative increase in $\eta$ over $\sigma$, so the stigma ratio $\mathcal{Z}$ will be large. On the other hand, if the stigmatized are ostracized (reducing $\eta$) and have lower survivorship (increasing $m_T$), then the stigma ratio will be small. Other parameters may change the amplitude of stigmatization's effect, according to Eq. (9), but they can not change its direction. Parameters are only summary statistics; the responses to stigmatization will be heterogeneous, with some people conforming to social norms while others rebel against the same norms, so in practice we have to average over the effected population to estimate the stigma ratio.

The stigma ratio does not itself tell the whole story. The effect of stigmatization on prevalence is mathematically more complicated. The steady-state disease prevalence $[P]^*$ has the matrix formula

$$[P]^* = \begin{bmatrix} 0 \\ 1 \\ 1 \\ 1 \end{bmatrix}^T \begin{bmatrix} -m_S-\Lambda & 0 & 0 & 0 \\ \Lambda & -m_C-x & 0 & 0 \\ 0 & x & -m_L-y & 0 \\ 0 & 0 & y & -m_T \end{bmatrix}^{-1} \begin{bmatrix} b \\ 0 \\ 0 \\ 0 \end{bmatrix} \tag{12}$$

wherer $\Lambda$ is the solution of Eq. (7). To see how the rate of stigmatization impacts prevalance, we

differentiating prevalence and find the prevalence's sensitivity to stigmatization

$$\frac{\mathrm{d}[P]^*}{\mathrm{d}y} = \frac{\partial[P]^*}{\partial y} + \frac{\partial[P]^*}{\partial \Lambda}\frac{\partial \Lambda}{\partial \mathscr{R}_0}\frac{\mathrm{d}\mathscr{R}_0}{\mathrm{d}y} \tag{13a}$$

$$\text{where} \quad \frac{\partial[P]^*}{\partial y} = \frac{\Lambda b x \left(m_L - m_T\right)}{m_T \left(\Lambda + m_S\right)\left(m_C + x\right)\left(m_L + y\right)^2}, \tag{13b}$$

$$\frac{\partial[P]^*}{\partial \Lambda} = \frac{b m_S \left(m_L m_T + m_T x + m_T y + x y\right)}{m_T \left(\Lambda + m_S\right)^2 \left(m_C + x\right)\left(m_L + y\right)}, \tag{13c}$$

$$\frac{\partial \Lambda}{\partial \mathscr{R}_0} = \frac{\Lambda m_S \left(\Lambda + m_S\right)}{(\Lambda + m_S)^2 - \mathscr{R}_0 m_S^2} = \frac{\Lambda^2 m_S}{\Lambda^2 + \epsilon m_S} \tag{13d}$$

and $\mathrm{d}\mathscr{R}_0/\mathrm{d}y$ is given in Eq. (11). From inspection, we can tell that $\partial[P]^*/\partial\Lambda > 0$ and $\partial\Lambda/\partial\mathscr{R}_0 \geq 0$, Since stigmatization is never expected to improve life-expectancy ($\mu_L \leq \mu_T$), $\partial[P]^*/\partial y \leq 0$. It then follows that whenever the stigma ratio $\mathcal{Z} < 1$, faster stigmatization reduces the basic reproductive number ($\partial\mathscr{R}_0/\partial y \leq 0$), and thus will reduce prevalence. On the other hand, if stigmatization has no effect on mortality ($\mu_L = \mu_T$) and the stigma ratio is large ($\mathcal{Z} > 1$) then faster stigmatization increases prevalence.

## 4 Scenario comparison

We can now explore how stigmatization impacts the prevalence, community size, and life-time risk of infection. A detailed model analysis is performed in 3, including sensitivity analyses for each parameter. For illustration, we consider three different scenarios (see Fig. 3). **Scenario 0 (Non-infectious)**: For purposes of comparison to the next two scenarios, suppose the infection pressure is independent of the number of those already sick, individuals with the disease die sooner, and the disease-state is easily identified in others. Then strong stigmatization rates reduce prevalence because of the high mortality among the stigmatized. Stigmatization correspondingly reduces the community size and is consequently costly to the group (although benefits from selection for disease resistance may kick-in over longer time scales). **Scenario 1 (Pre-Urban)**: Now, suppose the infection pressure is primary from contact with those infected, a community is so small a stigma is inescapable, stigma increases a person's mortality risk several fold, and there is no effective medical treatment or prophylaxis against transmission. Then cultural norms of stigmatization will decrease prevalence and enlarge a community. **Scenario 2 (Modern)**: But if we are considering a large urban community where labelled and stigmatized individuals are diagnosed to receive treatment which prolongs their lives relative to un-diagnosed individuals, labelled individuals can take prophylactic actions that reduce transmission, and stigmatized individuals can partially escape ostracism, then stigmatization will increase prevalence and reduce community size. Specific parameter values for Scenarios 0, 1, and 2 are given in Table 2.

Comparing Scenarios 0 and 1 (Fig. 3), we see that under our theory, the major benefits of

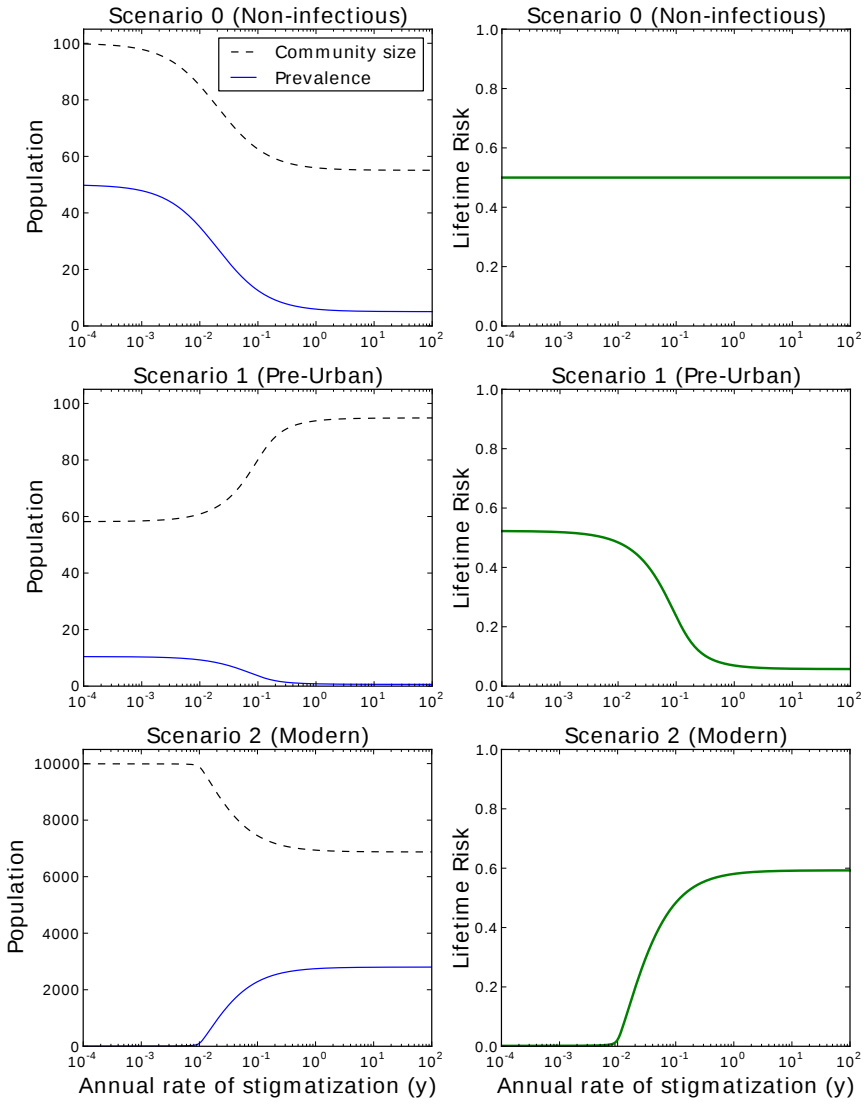


**Figure 3. Comparison of stigma's impact on prevalence and risk.** In Scenario 0 (top), stigmas applied to a non-transmissible disease reduce both prevalence and community size. In Scenario 1 (middle), where no treatment is available and stigmatization is inescapable, increasing rates of stigmatization reduces prevalence and allows for larger communities. In Scenario 2 (bottom), where treatment is available and stigmatization can be escaped, stigmatization increases prevalence, leading to reduced community size. See Table 2 for parameter values. Note: stronger stigmas never increase both community size and prevalence simultaneously.

stigmatization for a community appears only for transmissible diseases. For non-transmissible infections, stigmatization reduces prevalence and population size, but not lifetime risk. Thus, there is no externally imposed cost from a case of sickness on susceptibles, and thus no selective pressure favoring stigmatization. In general, stigmatization of others should only have evolutionary benefit to a group when those others impose some form of external fitness burden, *e.g.* an externality, to borrow a term from economics. For example, physical traits like blindness, paralysis, and senility can impose burdens on those who must care for the stricken. These externalities could also select for stigmatization, even when the condition is not transmissible. But in our model, externalities only occur through transmission.

Comparison of Scenarios 1 and 2 shows that the impacts of stigmatization against transmissible diseases are not universally good or bad, but depend on the specifics of the disease, environment, and medical development of a community. These results are consistent with the arguments of Smith & Hughes (2014). The impact of stigmatization can be summarized by defining a dimensionless stigma ratio $\mathcal{Z}$ which represents the ratio of the average cumulative transmission of a stigmatized individual to that of a labelled but unstigmatized individual. The relationship between the stigmatization rate and the lifetime infection risk ($\mathcal{K}$) is monotone, with the stigma ratio determining the derivative's sign – if $\mathcal{Z} < 1$, then more stigmatization decreases lifetime risk, but if $\mathcal{Z} > 1$, then more stigmatization increases lifetime risk (see Section 3). The general conditions for the impact of stigmatization can be obtained through differentiation of the reproductive ratio (see Section 3), and holds in both homogeneous and heterogeneous populations.

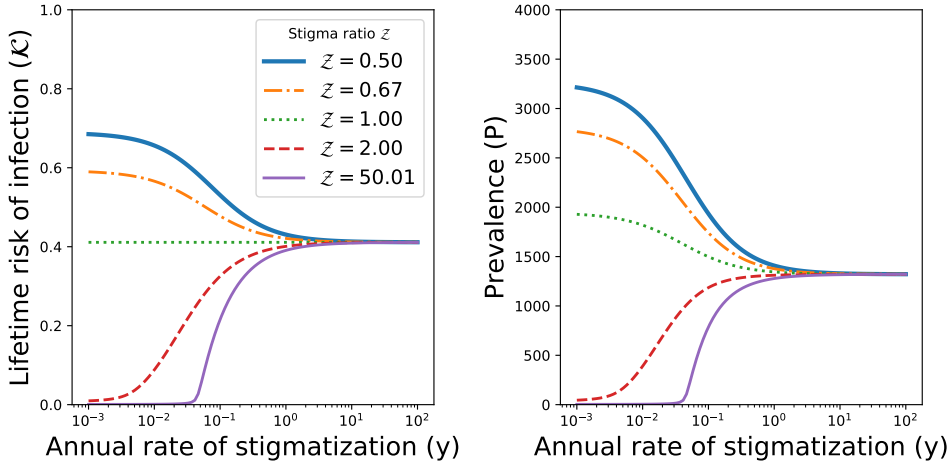


**Figure 4. Stigma ratios.** As the stigma ratio ($\mathcal{Z}$) decreases, the benefits of stigmatization are diminished and reversed. For the lifetime risk $\mathcal{K}$, this change occurs exactly when $\mathcal{Z} = 1$. Prevalence also responds this way, but a smaller value of the stigma ratio is needed before stigmatization increases prevalence, and the affect of the stigmatization rate may not be monotone for an intermediate interval of values. Parameters from Scenario 2, but with stigma ratio increase caused by a decrease in the relative infectiousness of labelled individuals $\sigma$ when $\mu_T = 0.06$ (Table 2).

The relative infectiousness of labelled individuals plays a crucial role in determining the impact of stigmatization (see Fig. 4). If individuals who have been identified as infected receive good medical treatment and counselling, the chance that they further transmit infection can be greatly reduced, and pre-empt the benefits of stigmatization. On the other hand, if labelled individuals maintain relatively high transmission rates, stigmatization remains an effective method of reducing infection pressure by reducing contact rates.

# 5 Labelling game

We now consider a heterogeneous population game where individuals can influence public knowledge about their infection status in response to cultural norms of stigmatization.

## 5.1 Behavior feedbacks

The rate with which cryptically infected individuals are willing to seek out medical help and be "out" about their condition can depend on the rate of stigmatization and it's consequences. In communities where stigmatization is rapid and harsh, low rates of diagnosis and labelling ($x$ small) may prevent people from taking advantage of effective treatments and educational programs even when available.

A person's behavior and the implied choice of a labelling rate can be thought of as an optimization problem. We postulate that individuals acting rationally will choose their labelling rate $x$ so as to maximize the payoff of their actions. Using Markov chain theory (see Appendix B), we can reasonably argue based on System 1 that the expected payoff of labelling at rate $x$ in a community that stigmatizes at rate $y$ is

$$\mathcal{U}(x,y,\Lambda) := \frac{1}{\Lambda+h+m_S}\left(u_S + \frac{\Lambda}{h+m_C+x}\left(u_C + \frac{x}{h+m_L+y}\left(u_L + \frac{yu_T}{h+m_T}\right)\right)\right) \tag{14}$$

where $u_i$ represents the income (value per time) of residing in state $i$ and $h$ is the rate discounting of future returns relative to the present. The best labelling rate

$$x_{\text{best}}(y,\Lambda) := \underset{x}{\text{argmax}}\ \mathcal{U}(x,y,\Lambda). \tag{15}$$

However, the payoff on the infection pressure $\Lambda$, and the infection pressure depends on the typical labelling rate $\overline{x}$ in the community ($\Lambda(\overline{x})$). So the payoff to the individual depends not just on their own choice of labelling rate, but also on the choices of others. Thus, the payoff function $\mathcal{U}(x,y,\Lambda(\overline{x}))$ defines a population game, where we take the infection pressure to be its steady-state value. We call this game the "labelling game".

The main solution concept of interest here for our labelling game is a pure Nash equilibrium. A pure Nash equilibrium $x_{\text{nash}}$ of a population game is any strategy that is a best reply when everybody

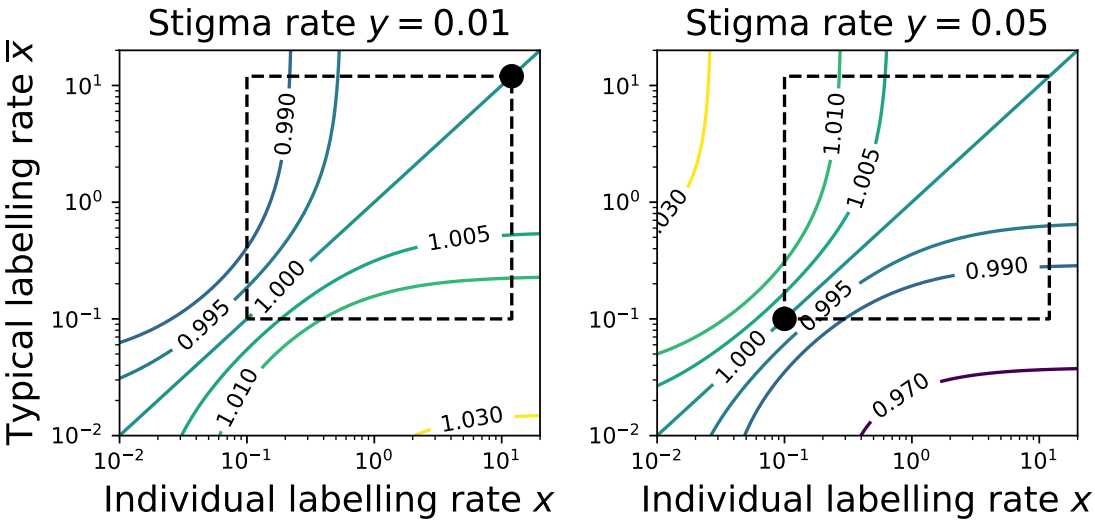


**Figure 5.** Example contour plots of the relative expected payoff $U(x, \Lambda(\overline{x}))/U(\overline{x}, \Lambda(\overline{x}))$ as a function of the individual's labelling rate $x$ and the typical labelling rate $\overline{x}$. Dashed boxes are the boundaries of feasible labelling rates. When stigmatization is slow ($y < y^*$, left), faster labelling is always better for the individual, and the Nash equilibrium (dot) maximizes the labelling rate. When stigmatization is fast ($y > y^*$, right), slower labelling is always better for the individual, and the Nash equilibrium (dot) minimizes the labelling rate. Parameters from Scenario 1 (Table 2).

else is using the same strategy, solving

$$x_{\text{nash}} \in x_{\text{best}}(y, \Lambda(x_{\text{nash}})). \tag{16}$$

This expected payoff is a hyperbolic function of the labelling rate $x$, so the best labelling rate a person can pick will be either the smallest ($x_{\min} > 0$) or largest ($x_{\max}$) value possible, but never something in between. By differentiation, we find

$$x_{\text{best}}(y, \Lambda) := x_{\min} + (x_{\max} - x_{\min})\mathrm{H}\left(\frac{y}{m_L + h}\left(\frac{u_T}{m_T + h} - \frac{u_C}{m_C + h}\right) + \left(\frac{u_L}{m_L + h} - \frac{u_C}{m_C + h}\right)\right) \tag{17}$$

where H() represents Heaviside's step correspondence.

We see by inspection that even though the payoff depends on the infection pressure, the best labelling rate is independent of the infection pressure ( $x_{\text{best}}(y, \Lambda) = x_{\text{best}}(y)$) in the labelling game. This implies that the Nash equilibrium $x_{\text{nash}}(y) = x_{\text{best}}(y)$ (see Figure 5).

Let us now consider how the best labelling rate choice $x_{\text{best}}(y)$ depends on the stigmatization

rate $y$. There is a threshold stigmatization rate

$$y^* := (m_L + h) \left( \frac{\dfrac{u_L}{m_L + h} - \dfrac{u_C}{m_C + h}}{\dfrac{u_C}{m_C + h} - \dfrac{u_T}{m_T + h}} \right) \tag{18}$$

where the switch between fast and slow labelling occurs. Suppose we assume, as would commonly be the case, that the stigma state is less valuable than the labelled state ($u_L/(m_L+h) > u_T/(m_T+h)$). If it is also worse to be cryptically infected than labelled or stigmatized, ($u_T/(m_T+h) > u_C/(m_C+h)$), then it's best to maximize the labelling rate ($x_{\text{best}}(y) = x_{\max}$). If cryptic infection is better than stigma and labelling ($u_C/(m_C + h) > u_L/(m_L + h)$), then it's best to minimize the labelling rate ($x_{\text{best}}(y) = x_{\min}$). If cryptic infection's payoff falls between the payoffs of stigmatization and labelling, the best behavior depends on the risk of stigmatization, and slow labelling is optimal only if the stigmatization rate is large enough.

## 5.2 Heterogeneous populations

Community populations typically have some degree of heterogeneity, such that different individuals will have different infection risks and different payoff functions. We would like our theory to be able to take known heterogeneities into account.

Epidemic theories that account for population heterogeneity are more complicated than the homogeneous theory we have presented above, as they account for differences in health, age, and contact rate (Lindquist *et al.*, 2010; Inaba, 2006; Nol *et al.*, 2009). For an initial model of stigmatization in a heterogeneous population, we suggest all individuals face the same risk, but may differ in all other parameters. Specifically, individuals are distributed over a type-space $\Omega$ according to a measure $\mu(\omega; y)$ which varies according to the stigmatization rate $y$. Then dynamically,

$$\frac{d[S_\omega]}{dt} = b - [S_\omega]m_S - [S_\omega]\Lambda, \tag{19a}$$

$$\frac{d[C_\omega]}{dt} = [S_\omega]\Lambda - [C_\omega]m_C - [C_\omega]x, \tag{19b}$$

$$\frac{d[L_\omega]}{dt} = [C_\omega]x - [L_\omega]m_L - [L_\omega]y, \tag{19c}$$

$$\frac{d[Z_\omega]}{dt} = [L_\omega]y - [Z_\omega]m_Z, \tag{19d}$$

$$\Lambda = \epsilon + \beta \int_{\omega \in \Omega} \left( [C_\omega] + [L_\omega]\sigma + [Z_\omega]\eta \right) \mathrm{d}\mu(\omega; y), \tag{19e}$$

where the parameter values $(\sigma, \eta, u_S, u_C, u_L, u_Z)$ are implicitly treated as independent functions of the type $\omega \in \Omega$. This model is strongly mixing, so infection pressure $\Lambda$ and corresponding lifetime

risk of infection $\mathcal{K}$ are the same for all individuals. The analysis will be more complicated when infection pressure varies among subpopulations, although variation will likely need to be large to have an observable effect (Reluga, 2009).

The steady-state infection pressure is the unique positive solution of the quadratic equation

$$\Lambda = \frac{\Lambda m_S \mathscr{R}_0(y)}{\Lambda + m_S} + \epsilon \tag{20a}$$

$$\text{where} \quad \mathscr{R}_0(y) = \frac{b\beta}{m_S(m_C + x)} \int_{\omega \in \Omega} \left(1 + \frac{\sigma x}{(m_L + y)} + \frac{\eta x y}{m_Z\,(m_L + y)}\right) \mathrm{d}\mu(\omega; y) \tag{20b}$$

with $\mathscr{R}_0(y)$ being the expected basic reproductive ratio for a heterogeneous population. For small reservoir pressures ($\epsilon \to 0$) and $\mathscr{R}_0 > 1$,

$$\Lambda \approx m_S(\mathscr{R}_0(y) - 1) + \epsilon \frac{\mathscr{R}_0(y)}{\mathscr{R}_0(y) - 1} + O(\epsilon^2). \tag{21}$$

From this approximation, the sensitivity of the lifetime risk to changes in the stigmatization rate

$$\frac{\mathrm{d}\,\mathcal{K}}{\mathrm{d}y} = \frac{1}{\mathscr{R}_0^2} \frac{\mathrm{d}\mathscr{R}_0}{\mathrm{d}y} \left[1 - \frac{\epsilon}{m_S} \frac{(2\mathscr{R}(y) - 1)}{(\mathscr{R}(y) - 1)^2} + O\left(\epsilon^2\right)\right] \tag{22}$$

Thus, to minimize lifetime risk in a community without environmental reservoirs, it suffices to study the effects of stigmatization on the basic reproductive ratio $\mathscr{R}_0$. Our expression for the stigma ratio $\mathcal{Z}$ can be derived from this in the special case of a homogeneous population.

When the population is weakly heterogeneous, the threshold for switching between maximal and minimal labelling rates will be approximately Galton (a.k.a. lognormally) distributed around the homogeneous switching threshold given in Eq. (17), where the expected values of the population distributions for each parameter are used in place of the deterministic values of the homogeneous population. It follows that the fraction of the population for whom it is best to maximize labeling ($x_{\text{best}}(\omega; y) = x_{\max}$) will be approximated by an error function

$$F(\omega; y) := \frac{1}{2}\left(1 - \operatorname{erf}\left(\frac{\ln y - \ln \widetilde{y}}{\delta\sqrt{2}}\right)\right) \tag{23}$$

where $\widetilde{y}$ is the median labelling cutoff and $\delta$ is the standard deviation of the log of $y$. The mean labelling rate in the population is then

$$\overline{x} = x_{\max} F(\omega; y) + x_{\min}(1 - F(\omega; y)). \tag{24}$$

### 5.3 Policy resistance

These observations can be rephrased in terms of the systems-dynamics concepts of policy resistance and policy reinforcement (Sterman, 2006; Li *et al.*, 2016). When people acting in their own self-interest counter-act a community policy, we call it "policy resistance". When people's actions reinforce and amplify a policy, we call it "policy reinforcement".

Suppose cultural norms of stigmatization evolve to maximize community welfare $\mathcal{W}(\overline{x}, y)$. At equilibrium, we expect the typical labelling rate to be at Nash equilibrium, so the community welfare will be $\mathcal{W}(x_{\text{nash}}(y), y)$. In their weak differential forms, policy reinforcement and policy resistance are as follows. Stigmatization exhibits policy reinforcement if and only if

$$\left(\frac{\partial W}{\partial \overline{x}}\frac{\partial x_{\text{nash}}}{\partial y}\right)\left(\frac{\partial W}{\partial y}\right) > 0. \tag{25}$$

On the other hand, stigmatization exhibits policy resistance when

$$\left(\frac{\partial W}{\partial \overline{x}}\frac{\partial x_{\text{nash}}}{\partial y}\right)\left(\frac{\partial W}{\partial y}\right) < 0. \tag{26}$$

Intuitively equivalent – though slightly more complicated – definitions are available when $x_{\text{nash}}$ shows jumps as here.

The importance of policy resistant feedbacks in heterogeneous and homogeneous populations is illustrated by the numerical results shown in Fig. 6. Define the community welfare as the typical expected time to infection ( $\mathcal{W}(\overline{x}, y) := 1/\mathcal{K}(\overline{x}, y)$ ). When individuals have control of their own labelling rate and choose it to maximize their personal payoff, high stigmatization rates which would otherwise benefit the community by reducing lifetime infection risk actually can backfire and significantly increase infection risk relative to community norms where there is no stigmatization. In such cases, both the best and worst stigmatization policies may occur at intermediate stigmatization rates. We leave detailed sensitivity analysis of alternative distributions to future studies where the choice can be empirically informed. In Scenario 1, we always observe policy resistance at the threshold stigmatization rate $y^*$ (see Fig. 7). In Scenario 2, we observe policy reinforcement, at the threshold stigmatization rate $y^*$, although policy resistance is also possible under different value parameters $u_i$ (see Fig. 8).

## 6 Discussion

Here, we have presented a model of the arguments previously made by Smith & Hughes (2014). Our analysis shows stigmas switch from evolutionarily beneficial to detrimental when they stop reducing risk, a threshold quantified in terms of the stigma ratio $\mathcal{Z}$. Our formulas can clarify the rhetorical and policy arguments over the use of stigma as a tool to manage public health in this and other

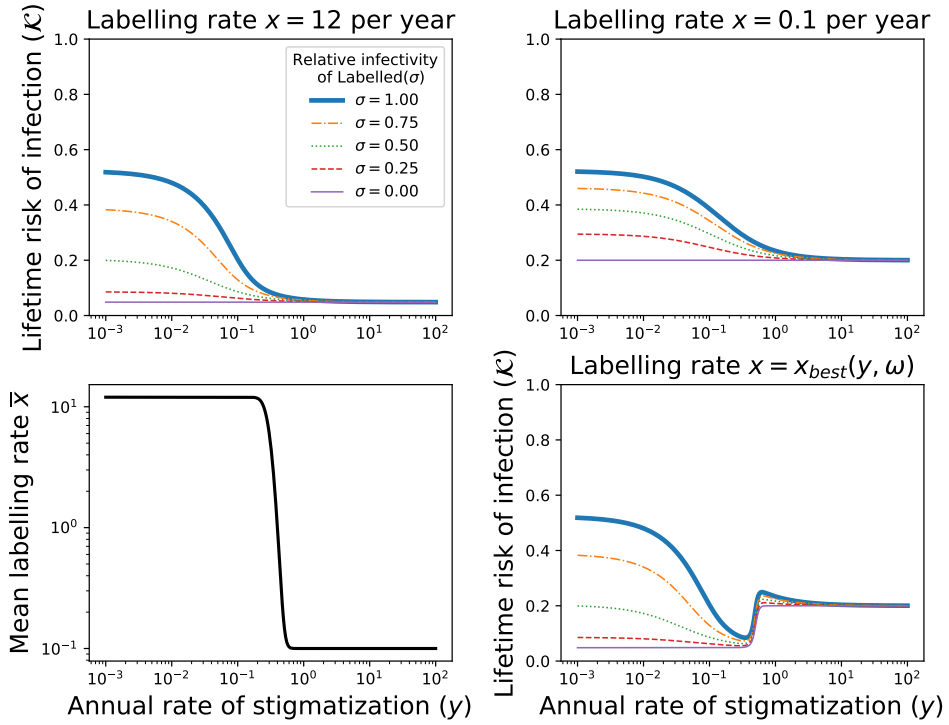


**Figure 6. Stigma can backfire into policy resistance.** In situations like Scenario 1 where faster stigmatization uniformly leads to lower risk when the labelling rate is fixed (top row), cryptically infected individuals can reverse the benefits of stigmatization by manipulating their labelling rate in their own self-interest and concealing their infection state. So individual control of labelling rates (bottom left) changes the collective optimal stigmatization rate (bottom right). For low relative infectiousness ($\sigma = 0$), the community is best off minimizing stigmatization, while for intermediate and large relative infectiousness, the best stigmatization rate is about 0.3 per year. Parameters from Scenario 1 (Table 2), with $x_{\text{best}} \in (0.1, 12)$ per year while the distribution of preferences is parameterized with median cutoff $\widetilde{y} = 0.3$ and width $\delta = 0.2$.

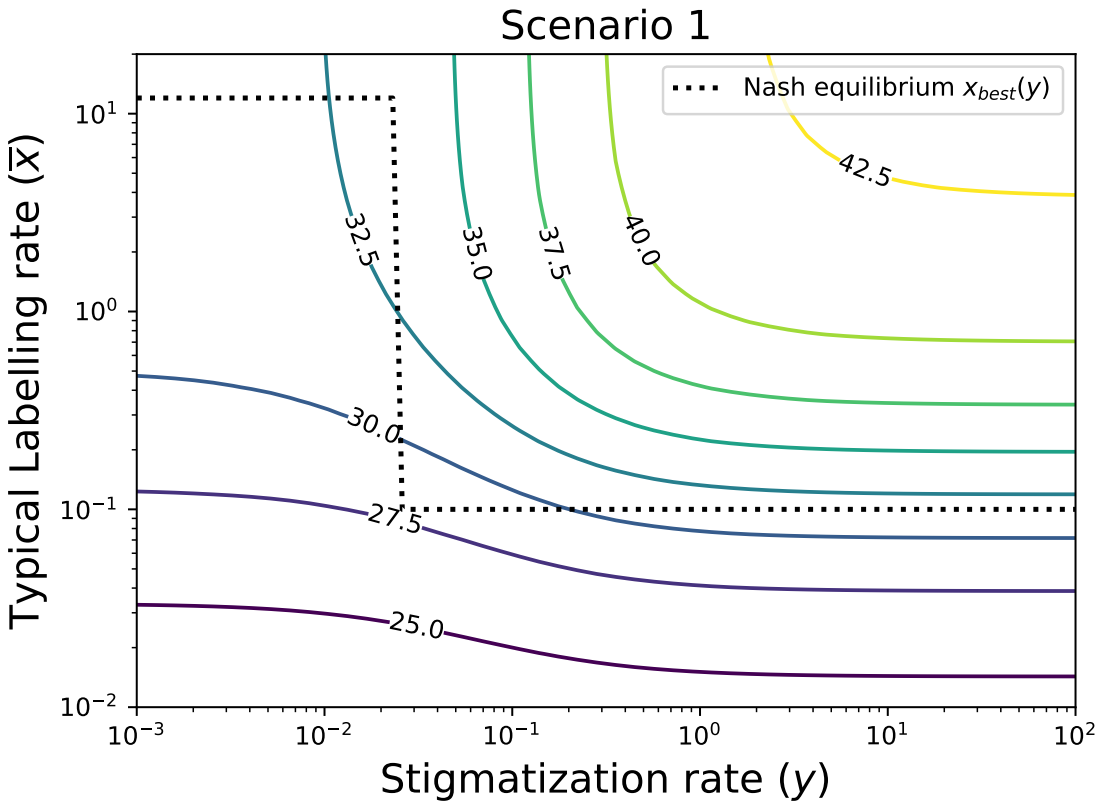


**Figure 7.** Contour plot of Scenario 1's community welfare $\mathcal{W}(\overline{x}, y) = 1/\mathcal{K}(\overline{x}, y)$ as a function of the stigmatization rate $y$ and the typical labelling rate $\overline{x}$ in a homogeneous community. Faster stigmatization and faster labelling both increase the community welfare, but only labelling rates on the dotted line are Nash equilibria. Because of the shape of the Nash equilibrium, the system suffers policy resistance when stigmatization rates are increased past the threshold stigmatization rate $y^* \approx 0.03$.

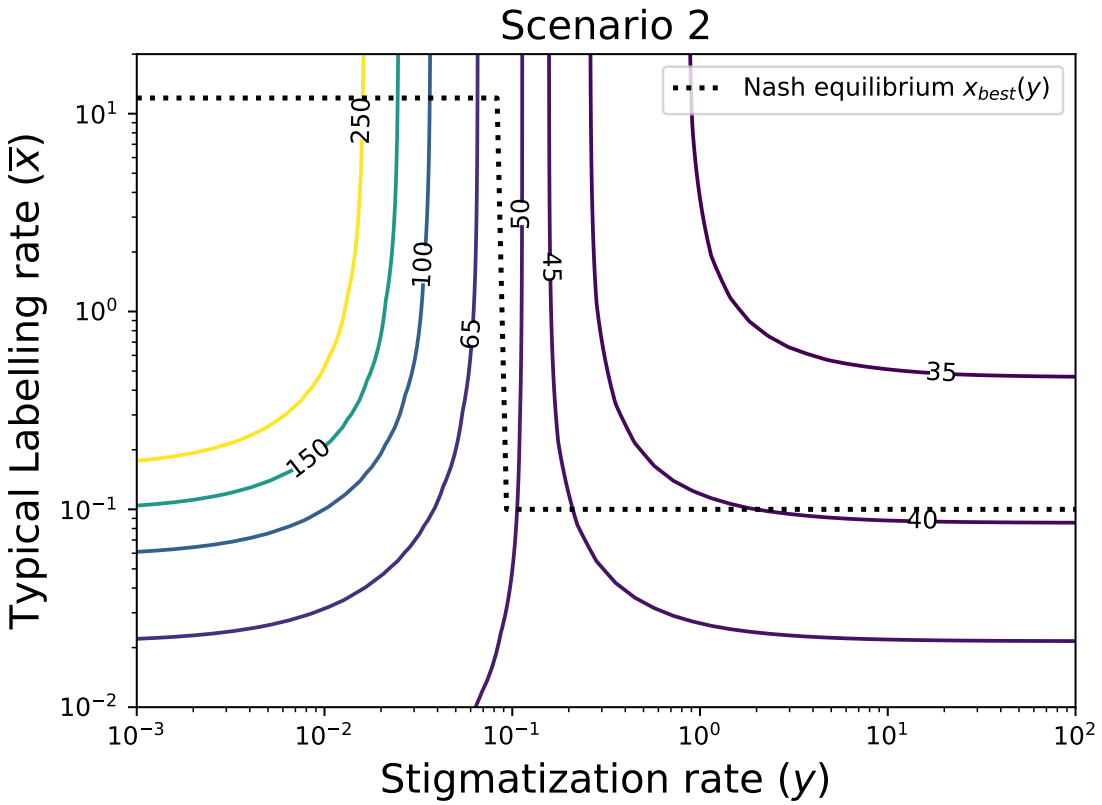


**Figure 8.** Contour plot of Scenario 2's community welfare $\mathcal{W}(\overline{x}, y) = 1/\mathcal{K}(\overline{x}, y)$ as a function of the stigmatization rate $y$ and the typical labelling rate $\overline{x}$ in a homogeneous community. Unilateral decreases in stigmatization improve community welfare, but the effect of a unilateral decrease in labelling depends on the stigmatization rate. Reducing the stigmatization rate past the threshold stigmatization rate $y^* = 0.085$ results in policy reinforcement. Alternatively, policy resistance could appear under this scenario if the threshold stigmatization rate were larger (*i.e.* $y^* \approx 1$).

health contexts (Vartanian & Smyth, 2013). Rather than falling back on unstated prior beliefs that bias discussions of stigmatization, the relative merits of stigmatization can now be directly estimated from empirical data using the formulas we supply. This will help us move toward an evidence-based dialog about the reasons for stigmatization and the best approaches available for coping with it and the underlying infectious disease.

Shifts of the stigma ratio $\mathcal{Z}$ from high to low values over the centuries may well be a marker of a transition into the modern era. Populations have become concentrated in urban areas, while advances in public health have removed the threats from pestilences like cholera, tuberculosis, smallpox, and plague that beset early cities. However, this transition is mediated by the institutional, cultural, demographic, and Darwinian components of societal evolution, the last two of which progress more slowly than the first two. The different time-scales create a tension within societies as some people struggle to reconcile their instincts with reason and social norms. Migrations that bridge gradients in social norms between urban and rural communities may add to this tension. Modern outbreaks for stigmatization in response to new epidemics like HIV (Bishop *et al.*, 1991) and Ebola (Davtyan *et al.*, 2014) may well be symptoms of this tension – the initial lack of ameliorating public health interventions and established social norms leaves people to revert to instinctual behavioral responses.

Instinct and social norms are not independent, but rather interact during development in complex ways to shape individual and community behavior, particularly through the emotion of disgust (Herz, 2012). We're excited about the future explorations of how these nuances may effect the potential for infectious disease stigmatization.

Of course, our stigma ratio theory does not account for all possibilities. We hope future work will be able to directly model the rich variety of events and behaviors that follow stigmatization. To the best of our understanding, these can have strong dependences on population composition, culture, environment, and the particular infectious disease, and further study will be needed to extend our theory beyond it's current scope. Movement in response spatial variation in risk may pose an interesting counterpoint, as it can flip the agency of stigmatization. Nor have we tried to account for costs associated with the common false-positive application or conflation of stigmas, or any possible side-effects – this is a topic for further research. Similarly, it would be great to be able to perform the accountings above when we relax our assumption that knowledge of stigmas is communicated instantly and universally without objection or conflict. And there may well be additional knock-on effects to public health or social structure. For applications, our theory should probably be extended by merging demographic components of population structure, including age, gender, and race.

Our measures of group success and community welfare have only been group size and lifetime risk, and thus can be interpreted under either group selection (Sober & Wilson, 1998) or cultural (Richerson & Boyd, 2006) theories of community evolution. We have not attempted to address the more complicated impacts of disease and stigma on group function or productivity. Selective

pressures for stigmatization may be offset in part by kin-selection in the scope of family units – nursing and care of sick relatives improves the survival and reproductive success of ones own genes. Infectious disease prevalence can promote sociality (Bonds *et al.*, 2005). Such behavior is, in fact, well documented (Parsons, 1975), and we may expect more effects to emerge from larger-scale structures in social networks. However, sometimes kin-relationships accelerate stigmatization as family members attempt to avoid "courtesy stigma" (Goffman, 1963; Smith, 2011), complicating the matter further.

Our analysis has only considered cases of chronic infectious diseases. While we expect the same factors to be at play in a case of an epidemic of an acute immunizing infection, dynamic aspects of the epidemic and stigmatization will become very important, as the infection hazards will change relatively quickly, ultimately receding entirely, while stigmas may persist long after the epidemic ends, and have negative impacts on both individuals and communities. Kin and neighbor care-giving will continue to be complicating. Our general conclusion should still hold: stigmas will help or hurt individuals and communities, depending on the quality of health care and the effectiveness of stigmatization at reducing risk. But any benefits are likely to be short term, and to disappear as the epidemic wains. However, scenario-specific analyses will be needed to make sense of all the moving parts.

In conclusion, we have provided a quantitative theory of how stigmatization of infected individuals can affect community health, and shown that while stigmatization may have had benefits historically, this may well not be the case in dense modern societies. We hope our theory will prove useful in futures studies of infectious disease stigmas and will provide a foundation for future mathematical and computational studies.

## 7 Acknowledgements

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# A Global stability

Assume the background infection hazard $\epsilon > 0$. Define a function

$$\Psi([S],[C],[L],[Z]) := \begin{bmatrix} 1 \\ \frac{[S]^*}{[C]^*} \\ [S]^* \frac{\epsilon+\beta(\sigma[L]^*+\eta[Z]^*)}{x[C]^*} \\ [S]^* \frac{\epsilon+\beta\eta[Z]^*}{y[L]^*} \end{bmatrix}^T \begin{bmatrix} \frac{[S]}{[S]^*} - \ln\left(\frac{[S]}{[S]^*}\right) - 1 \\ \frac{[C]}{[C]^*} - \ln\left(\frac{[C]}{[C]^*}\right) - 1 \\ \frac{[L]}{[L]^*} - \ln\left(\frac{[L]}{[L]^*}\right) - 1 \\ \frac{[Z]}{[Z]^*} - \ln\left(\frac{[Z]}{[Z]^*}\right) - 1 \end{bmatrix}. \tag{27}$$

The form and weights of this function were suggested by C. McCluskey (McCluskey, 2010, and personnel communication), and will be addressed by him in future publications. For convenient reference, we provide a lemma that is well-known to experts.

**Lemma 1.** *Given $n$ finite positive real numbers $u_1, u_2, \dots u_n$ such that $u_1 u_2 \cdots u_n = 1$,*

$$\sum_{i=1}^{n} u_i \geq n.$$

*Proof.* The result follows immediately from the arithmetic-geometric mean inequality

$$\frac{1}{n}\sum_{i=1}^{n} u_i \geq \left(\prod_{i=1}^{n} u_i\right)^{1/n}.$$

□

Now, the reason we have defined $\Psi$.

**Theorem 2.** *The function $\Psi([S],[C],[L],[Z])$ is a Lyapunov function for non-negative solutions of System (3).*

*Proof.* It is trivial to check that $\Psi$ is a non-negative convex function and that $\Psi([S]^*, [C]^*, [L]^*, [Z]^*) = 0$ is it's global minimum. Using a change of variables to eliminate the birth and death parameters (except $m_S$), we can show that along any solution of System (3) with non-negative initial condition,

$$\frac{dV}{dt} = -(\beta[C]^* + m_S)\frac{(s-1)^2}{2} - \beta\sigma[L]^*\left(\frac{c}{\ell} + \frac{\ell s}{c} + \frac{1}{s} - 3\right) - \beta\eta[Z]^*\left(\frac{c}{\ell} + \frac{\ell}{z} + \frac{sz}{c} + \frac{1}{s} - 4\right) - \epsilon\left(z + \frac{\ell}{z} + \frac{c}{\ell} + \frac{s}{c} + \frac{1}{s} - 5\right) \quad (28)$$

where $s = [S]/[S]^*$, $c = [C]/[C]^*$, $\ell = [L]/[L]^*$, and $z = [Z]/[Z]^*$. By the Lemma above and the non-negativity of all parameters and steady-states, it follows that $dV/dt \leq 0$ with equality holding only at the steady-state solution. □

Thus, under the standard theory of Lyapunov functions, the unique positive steady-state is globally asymptotically attracting for all biological parameter values. A short symbolic algebra implementation of the proof, including gory details of steady-state analysis, is available on request.

# B Calculation of discounted expected payoff

The estimation of expected payoff value for an individual whose state evolves according to a Markov chain is the topic of Markov decision process theory (Howard, 1960), and has been used more generally in prior publications Reluga & Galvani (2011); Reluga *et al.* (2007). However, for our stigma theory, where the changes in an individual's state must follow a specific sequence, we can greatly simplify our explanation of the payoff calculation.

Suppose that a person accumulates income at rate $u_i$ when residing in state $i$. Then the expected payoff for entering state $i$ would be the income for the state times the expected residence time in that state. Let's name the expected residence time of state $i$ as $\tau_i$, so that the expected payoff for entering state $i$ is $u_i\tau_i$. Note that in our theory, there are no loops among states, so once an individual leaves state $i$, they never return to it again.

But there is a chance that an individual never reaches state $i$, and this is something for which we must account. Since all individuals are treated without distinction and have the same initial state, the chance of eventually reaching state $i$ can be described with a single probability $\phi_i$. It follows that the total expected payoff for an individual starting in the susceptible state $\mathcal{U}$ is given as as the sum of the chances of reaching each state times the expected payoff for entering said state. Specifically, for the susceptible ($S$), cryptically infected ($C$), labelled ($L$), and stigmatized ($Z$) states,

$$\mathcal{U} = \sum_{i\in\{S,C,L,Z\}} \phi_i u_i \tau_i. \quad (29)$$

For the Markov chain described by System (1), where all transition rate are constant, the expected residence times are the reciprocals of the exit rates, or

$$\left[\tau_S, \quad \tau_C, \quad \tau_L, \quad \tau_Z\right] = \left[\tfrac{1}{m_S+\Lambda}, \quad \tfrac{1}{m_C+x}, \quad \tfrac{1}{m_L+y}, \quad \tfrac{1}{m_Z}\right] \tag{30}$$

The probabilities of reaching each state, rather than dying, are

$$\left[\phi_S, \quad \phi_C, \quad \phi_L, \quad \phi_Z\right] = \left[1, \quad \tfrac{\Lambda}{m_S+\Lambda}, \quad \left(\tfrac{\Lambda}{m_S+\Lambda}\right)\left(\tfrac{x}{m_C+x}\right), \quad \left(\tfrac{\Lambda}{m_S+\Lambda}\right)\left(\tfrac{x}{m_C+x}\right)\left(\tfrac{y}{m_L+y}\right)\right]. \tag{31}$$

If follows from Eq. (29) that without discounting ($h = 0$), the expected payoff for an individual is

$$\mathcal{U}(h=0) = \frac{1}{m_S+\Lambda}\left(u_S + \frac{\Lambda}{m_C+x}\left(u_C + \frac{x}{m_L+y}\left(u_L + \frac{yu_Z}{m_Z}\right)\right)\right). \tag{32}$$

If future income at time $t$ is discounted relative to current income by fraction $e^{-ht}$, it is a standard result of Markov decision process theory that all the same analysis holds, but with excess death rate $h$, such that the discounted expected payoff

$$\mathcal{U} = \frac{1}{h+m_S+\Lambda}\left(u_S + \frac{\Lambda}{h+m_C+x}\left(u_C + \frac{x}{h+m_L+y}\left(u_L + \frac{yu_Z}{h+m_Z}\right)\right)\right). \tag{33}$$

To close, we should emphasize that while the expected utility is a convenient model for decision making that captures the important qualitative properties of interest, the payoff is a random variable, and other models such as risk-seeking or risk-averse behaviors could be considered, particularly for applications where parameter estimates are being derived from field data.