

The transmission and persistence of 'urban legends': sociological application of age-structured epidemic models.

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(Working paper — comments welcome.)

Abstract

This paper describes two related epidemic models of rumor transmission in an age-structured population. Rumors share with communicable disease certain basic aspects, which means that formal models of epidemics may be applied to the transmission of rumors. The results show that rumors may become entrenched very quickly and persist for a long time, even when skeptics are modeled to take an active role in trying to convince others that the rumor is false. This is a macrophe-nomeon, because individuals eventually cease to believe the rumor, but are replaced by new recruits. This replacement of former believers by new ones is an aspect of all the models, but the approach to stability is quicker, and involves smaller chance of extinction, in the model where skeptics actively try to counter the rumor, as opposed to the model where interest is naturally lost by believers. Skeptics hurt their own cause. The result shows that including age, or a variable for which age is a proxy (e.g. experience), can improve model fidelity and yield important insights.

Keywords: Rumors—mathematical models; rumors—age-structure; rumors—persistence.

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1 Introduction

Word-of-mouth spread of news and rumors is the simplest form of mass diffusion of information. Although rumor-spreading can be abetted by technology, the essence of rumors is person-to-person contact. The large-scale dynamics of rumor spread and persistence are, however, poorly understood. Why are some rumors short-lived, while others never seem to die? This paper addresses this question by comparing two models of the spread of a special class of rumors called ‘urban legends’—persistent, usually non-verifiable, short tales spread by word-of-mouth or by cognate means (e.g., electronic mail). The persistence of urban legends is the key factor of interest here. I take persistence to be what sets urban legends apart from rumors more generally, which may disappear almost as soon as they arise.

Urban legends abound. Three examples are: (1) Spider eggs are an ingredient of a certain brand of soft chewing gum. This rumor was rampant among children in the United States in the late 1970s and early 1980s when soft chewing gum, in this case *Bubble Yum* brand, became more popular among children than hard chewing gum.¹ (2) The actor from a well-known television commercial died from a lethal combination of candy and *Coca-cola*. According to this rumor, the child actor who played the character Mikey in commercials for *Life* breakfast cereal died because he ate *Pop-rocks* and drank *Coca-cola* at the same time.² (*Pop-rocks* are a candy that contain bicarbonate of soda, which make a popping sound in the mouth.) (3) Upon requesting the cookie recipe at the café of an upscale department

¹See <http://www.topsecretrecipes.com/sleuth/legends/legend2.htm>.

²See <http://www.snopes2.com/horrors/freakish/poprocks.htm>.

store, a patron was presented with the recipe—for which he was billed \$200. The patron takes revenge by emailing the recipe, gratis, to all his friends and requests them to do the same. This is the canonical example of an email hoax or rumor, and some variant of the cookie recipe legend is almost certainly still in circulation.³ All three of these urban legends are well-documented in the popular literature and have been experienced by the author. Example web pages have been provided, and the interested reader can find more such pages by doing a standard Internet search.

The remarkable persistence of rumors is a macro-phenomenon, not necessarily a micro- one; rumors keep spreading even after their original adherents become skeptical. The answer to why specific urban legends keep spreading is not that more-and-more people believe the legend. As with other social phenomena, the overall system does not mimic the behavior of a single, idealized, actor (for an overview, see, for example, Schelling 1978, Coleman 1990). This paper uses mathematical models to explore the properties of rumor propagation where data collection is problematic. Incorporating age-structure into the models yields insights about how rumors can persist at the population level despite the fact that individuals may cease to believe the rumor after a certain period. And drawing on the deep, empirically tested, literature on mathematical models in epidemiology helps insure that the assumptions made about population mixing are reasonable ones.

³See <http://tutor.kilnar.com/hoax/myth/cookie.html>.

2 Rumors and Epidemics

The spread of rumors is analogous to the spread of an epidemic infectious disease. The similarity between epidemic models and rumor models is obvious, and long-recognized in both social science and epidemiology literature (see, e.g., Coleman 1964: 46, Cane 1966, Dietz 1967, Bartholomew 1967, Frauenthal 1980). Shibutani’s landmark study of rumors (1966) identifies rumors as a type of “behavioral contagion”.

There are two main strands of mathematical modeling literature in epidemiology. The first strand concentrates on the mathematics of epidemics, and seeks analytical solutions. In this context, the term ‘epidemic’ includes a wide variety of stochastic processes and deterministic models, some of which bear little relation to real biological or social phenomena. The second strand concentrates on epidemiology *per se* and its real-world relevance. The archetypal work in the first strand is Bailey’s *The mathematical theory of infectious diseases and its applications* (1975); in the second strand a good example is *Infectious diseases of humans: Dynamics and control* by Anderson and May (1992). In these two overlapping branches of the literature the goal is essentially either mathematical or epidemiological. In the former case, numerical solutions are beside the point, and in the latter case, they are often necessary to arrive at a conclusion.

The models introduced in this paper have four states, are nonlinear, and are explicit in age and time—such complications necessitate the use of a computer for numerical solution, and thus place this work, at least nominally, in the second tradition of epidemic models. Age plays an important

role in the rumors I investigate. The young are more credulous than the old, at least according to the assumptions set out here. The first age-structured epidemic model, of a hypothetical disease, was by Hoppensteadt (1974), and the more applied strand of the modeling literature has been strongly influenced by this work, particularly because age is a key factor in vaccine-preventable diseases.⁴ The simultaneous inclusion of age and time makes the models difficult to solve analytically.

Before discussing the model specifics, I review briefly the affinity between epidemic models and rumor diffusion models. Measles is the representative infectious disease for the purposes of the present discussion. Measles is highly contagious, and is spread by infected-to-susceptible contact (specifically, through airborne transmission of the measles virus). Rumors are also highly contagious: what differentiates rumors from other pieces of information is that the possessor of a rumor has an irresistible urge to tell others. Dunbar (1996) proposes that human language itself arose out of an inherent need to gossip. While this hypothesis is clearly speculative, it underscores the fact that rumor transmission is one of the most natural forms of social communication.

There are two types of immunity to rumors. Call the first type ‘skepticism’: a skeptic does not accept the rumor as true, neither the first time she hears it, nor after repeated exposure. The second type of immunity is ‘acquired immunity’: after being infected with the rumor for a certain

⁴Schenzle (1984) was the first to study an age-specific model of measles transmission. McLean and Anderson (1988a,b) applied such models to developing countries, where measles remains an important cause of death. And Eichner, Zehnder and Dietz (1996) applied detailed German data on measles to a sophisticated model incorporating many aspects of transmission.

length of time, the rumor carrier comes to believe that she has been duped, and ceases to believe the rumor. Belief in a rumor and desire to spread the rumor are here taken to be identical, though in practice belief may persist even after the burning desire to spread a new rumor wanes. The contact spread of pathogens and the contact spread of rumors is analogous. Skepticism plays the same role in rumor spread that vaccination plays in measles epidemiology. Acquired immunity is analogous across the two domains.

In two respects, the measles–rumors analogy breaks down. Measles has a latent (i.e. infected but pre-contagious) period which is unlike most rumors; with rumors, there is no distinction between infection and contagiousness. Measles involves recovery (or death) within a few weeks of initial infection, whereas some rumors may be believed for years. These differences are easy to deal with from the modeling perspective. In the present model, an individual is in a state of believing the rumor or not; qualitative aspects of rumor transmission—e.g., consideration that rumors tend to change content as they are spread (cf. Buckner 1965), or that rumor ambiguity affects transmission (cf. Allport and Postman 1946: 502)—are therefore omitted.

3 Model I: an Epidemic Model

3.1 Model description

The present model is a system of four partial differential equations in age and time (eqns. 1–4). This is a modified version of the classic three-state SIR (susceptible, infected, recovered/immune) epidemic model of Kermack and McKendrick (see Murray 1993), the dynamics of which are similar to

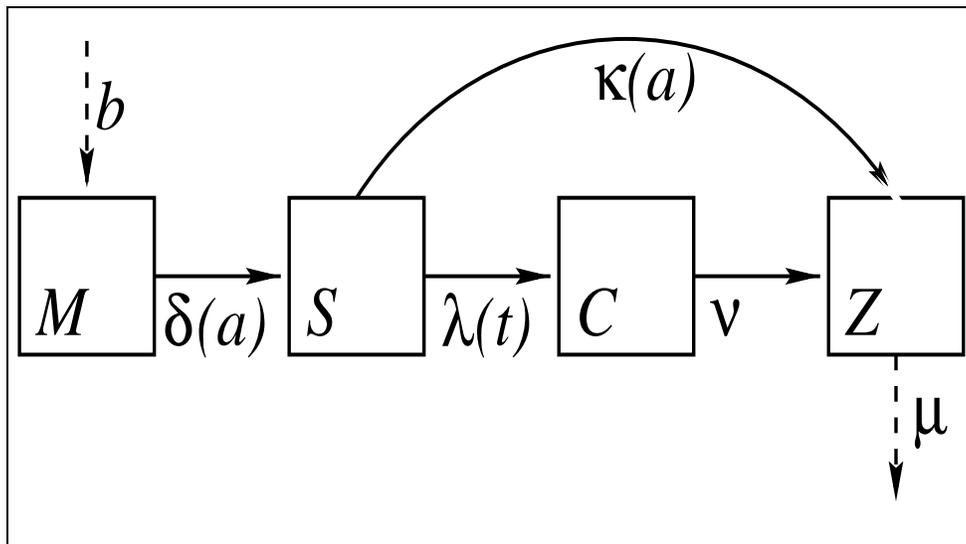


Figure 1: Model schematic. Model states boxed. Boundary conditions shown with dashed lines, model parameters shown with solid lines. The boundary condition b represents a birth rate and μ represents a mortality rate. In the present version, births=deaths, and the life table is rectangular, so $\mu = 0$ for all ages except the oldest age, ω . Mortality occurs in all states, but the population at the oldest age is primarily in state Z , so for clarity μ is shown only there. Other symbols as discussed in the text. The nonlinearity of the model comes from the key parameter $\lambda(t) = \beta \cdot C(t)/N$.

the familiar Lotka-Volterra predator-prey systems. The additional state in the present model is those who do not understand the rumor, which from the point of view of transmission is the same as being immune, except that it is mostly a very young group; the simulated population is ‘born’ into this group. The population itself is at equilibrium in size and in age structure (i.e. what demographers call stationary), and has a rectangular life table. Births and deaths are treated as boundary conditions.

The corresponding system of difference equations is solved numerically.⁵ This numerical solution can also be thought of as a deterministic macrosimulation of the rumor dynamics; macro- because the program does not keep track of simulated individuals, only of flows between stocks, and there are no integer constraints on these flows. Progression through the states of the model is age-related, but not completely determined by age, which makes it worthwhile to include age as well as time in the model equations. If the model states perfectly determined age, or if there were no relation between age and the stages of the model, then ordinary differential equations in time could be used effectively.

The concept of exponential decay plays an important role in models of this type. Constant rates—implying exponential decay—are the simplest decrements to include in differential equations, so they are attractive provided there is good realism in their use. In the epidemiology modeling literature, constant rates within age-stratified models have proven to be good matches to available data.

⁵Using a computer program written by the author in Pascal. Euler’s method is used, with 52 iterations taken to be one model ‘year’ of age/time.

The population at age zero is all in group M^6 , which they leave with age-specific rate $\delta(a)$, a delayed exponential decay into the susceptible group, S (see eqn. 5). There are two modes of exit from susceptibility: infection and skepticism. The susceptible population becomes skeptical or immune (denoted Z because it is the final class, or absorbing state, of the model) with the age-specific skepticism rate $\kappa(a)$, also a delayed exponential decay (see eqn. 6). The motivation for these rates is that up to age ζ all children are too young to be able to understand the rumor, and above this age there is rapid (exponential) recruitment into the susceptible class, as the children become more able to communicate and to understand stories (cf. eqn. 5). Similarly, below age ξ it is assumed that no child is savvy enough to be skeptical of a rumor, but above that age some children will not believe everything they are told (cf. eqn. 6).

The rate between susceptible and infected is the force of infection, $\lambda(t)$, and varies over time but not by age. The population is assumed to mix with itself equally by age. Although children mix mostly with other children during the day, they spread rumors to their older siblings and to their parents at home in the evening, and *vice versa*. Note that class M and class Z are inert from the point of view of rumor transmission: these classes neither transmit nor receive the rumor. So the assumption of uniform population mixing does *not* mean that, e.g., a rumor about chewing gum is as likely to be transmitted to an adult as to a school-aged child. The adult may be told the rumor, but she is, in all likelihood, immune, and will not accept it.

The force of infection is the most important parameter in the model:

⁶In the measles literature, this group is ‘protected by maternal antibodies’, hence M .

its variation over time drives the rise and fall of rumor epidemics, and it is the source of the model nonlinearity. The force of infection makes the model nonlinear because the rate between states S and C depends on C : $\lambda(t) = \beta \cdot C(t)/N$, where $C(t)$ is the entire rumor-infected population (all ages) and N is the total population in all ages and classes. The assumption of mixing is what makes the model a mass-action model in the language of mathematical epidemiology, which in turn borrowed the phrase from chemistry. Like molecules in a test-tube, people are mixing with each other constantly. Suppressing age, the net transmission from eqn. 3 is:

$$\lambda(t)S(t)dt = \beta \frac{C(t)}{N} S(t)dt$$

or, the population of susceptibles multiplied by the proportion contagious in the entire population, multiplied by a mixing parameter, β . The probability that a susceptible person will mix with a contagious person, conditional on the susceptible contacting *any* other person, is simply $C(t)/N$. The constant β captures both population mixing (i.e. the number of contacts between susceptible people and others in the population per unit time per susceptible person), and the probability that transmission will occur, conditional on contact (i.e. that the rumor will be spoken). Thus, $\lambda(t)S(t)dt = \beta S(t)(C(t)/N)dt$ provides a mass-action model of rumor transmission.

Note that as constructed here, mass-action models are concerned with proportions, not numbers. The total population size, N , simply acts as a scale factor. Density dependence—absolute numbers affecting model dy-

Parameter	Signifies	Value
$\delta(a)$	net rate $M \rightarrow S$	see eqn. 5
ζ	minimum age $M \rightarrow S$	156 weeks
$\tilde{\delta}$	rate $M \rightarrow S, a \geq \zeta$	0.0064 week ⁻¹
$\kappa(a)$	net rate $S \rightarrow Z$	see eqn. 6
ξ	minimum age $S \rightarrow Z$	312 weeks
$\tilde{\kappa}$	rate $M \rightarrow S, a \geq \xi$	0.0014 week ⁻¹
$\lambda(t)$	force of transmission	$\beta \cdot C(t)/N$
β	mass-action constant	1.0097 week ⁻¹
λ^*	used to calibrate β	0.0012 week ⁻¹
ν	recovery rate	0.2 week ⁻¹
N	population size	100,000
ω	oldest age	40 years
$C(t)$	total contagious	$\int_0^\omega C(a, t) da$

Table 1: Summary of parameter values.

namics—gives rise to another class of models, considered in different settings by (e.g.) Mayhew and Levinger (1976) and de Jong, Diekmann and Heesterbeek (1995).

The β parameter reflects population mixing, and therefore sets the stage for how quickly or slowly the rumor propagates. The value of β is assigned by a multiple-equilibrium process. The model is first run at length with zero rumor transmission, but with all other forces in effect. This initializes the population with the correct number of M , S , and Z at each age for the population without rumor transmission, but subject to the other transition rates of eqns. 1–4. Call this population the ‘starting equilibrium’. Next,

ask: what would be the mean age of infection with the rumor if it were spread with a constant rate of infection? That is, suppose that the population is at an equilibrium such that $\lambda(t)$ does not change over time; such equilibria (of disease transmission) are observed in pre-vaccination populations. The younger the mean age of infection, the more contagious the rumor in question. With a candidate value for mean age of infection, $\langle a \rangle$, the approximate corresponding fixed force of transmission, λ^* , is also known. Compensating for the period up to age ζ when there is no susceptibility, it follows from calculus that $\lambda^* \approx (\langle a \rangle - \zeta)^{-1}$. The result would only be exact in a population where $\tilde{\delta}$ is very large and $\kappa(a) = 0$ for all a , but it is a good approximation for the present purpose. I then run the model with full rumor transmission, but with $\lambda(t) \equiv \lambda^*$; from this simulation, β can be back-calculated as $N \cdot (\lambda^*) / C(t)$. The simulation is stopped when β reaches an equilibrium value, β^* , which is taken to be the ‘natural’ β for endemic rumor transmission with mean age of infection $\langle a \rangle$.

This way of setting β by adjusting the mean age of infection and running the model until equilibrium, is simply a way of assigning a meaningful value to β by using the commonsense notion of the mean age of infection under equilibrium conditions. Using this technique, β can be calibrated to a realistic value without recourse to either trial-and-error or advanced mathematics.

In the runs of the model, the population is reset to its starting equilibrium. A handful ($n = 3$) of rumor-infected people are placed in the population at age 312 weeks, and β fixed at β^* , with $\lambda(t)$ now free to change. The only other model parameter is the constant ν , which is the rate of recovery,

or the rate of acquired immunity. The resulting dynamics are described below. Table 1 summarizes all the model parameters.

The rates in the model are not duration-specific (i.e. the transition rates vary by age, and by time, but not by duration in a given state beyond that specified by the combination of age and time). The delayed exponential decay represented by eqn. 5 is a duration-specific effect, because there is a minimum time of residence in state M before transition to state S can occur. But this is a coincidence with an age-specific effect, since the whole population up to age ζ is in class M . Duration-specific effects themselves are an ill-defined concept in a compartmental model (as these models are sometimes called, after the compartments of figure 1), because the program keeps track of stocks, not simulated individuals. However, given the complexity of the model, with age-specific effects, and transition rates that depend on the state of the model and thus vary over time, the omission of duration-specific effects does not do violence to any essential aspects of the rumor dynamics.

3.2 Model equations

$$\frac{\partial M}{\partial a} + \frac{\partial M}{\partial t} = -\delta(a)M(a, t) \quad (1)$$

$$\frac{\partial S}{\partial a} + \frac{\partial S}{\partial t} = \delta(a)M(a, t) - [\lambda(t) + \kappa(a)] S(a, t) \quad (2)$$

$$\frac{\partial C}{\partial a} + \frac{\partial C}{\partial t} = \lambda(t)S(a, t) - \nu C(a, t) \quad (3)$$

$$\frac{\partial Z}{\partial a} + \frac{\partial Z}{\partial t} = \kappa(a)S(a, t) + \nu C(a, t) \quad (4)$$

where:

a, t : age, time

M : too young to understand rumor

S : susceptible to rumor
 C : infected with rumor, contagious
 Z : immune to rumor (absorbing state)

and:

$$\delta(a) = \begin{cases} 0 & a < \zeta \\ \tilde{\delta} & a \geq \zeta \end{cases} \quad (5)$$

$$\kappa(a) = \begin{cases} 0 & a < \xi \\ \tilde{\kappa} & a \geq \xi \end{cases} \quad (6)$$

Mortality and fertility are left out of the above equations. They are boundary conditions, not part of the differential equations *per se*. This version of the model assumes a rectangular life table (no mortality except at the oldest age, ω), which is an acceptable approximation for developed countries. For developing countries, a mortality parameter $\mu(a)$ would have to be added to the model equations.

The following transition matrix is equivalent to the model equations (1–4). The equations are analogous to von Foerster equations (Keyfitz 1985: 139-140), while the transition matrix is analogous to the Leslie matrix.⁷ Figure 1 is a flowchart diagram of the system, equivalent to either the equations

⁷In the present case of a multi-state population projection, I find the von Foerster equations preferable, though this is a question of taste. The v.F. equations imply the aging of the Lexis diagram and concentrate on the action of the state changes. The transition matrix shown here is not a true Leslie matrix because aging is suppressed. Multi-state Leslie matrices with explicit aging are large and unwieldy; the v.F. equations are more compact.

or the transition matrix.

$$\begin{pmatrix} 1 - \delta(a) & 0 & 0 & 0 \\ \delta(a) & 1 - \lambda(t) - \kappa(a) & 0 & 0 \\ 0 & \lambda(t) & 1 - \nu & 0 \\ 0 & \kappa(a) & \nu & 1 \end{pmatrix}$$

3.3 Epidemic Model Results

Figures 2–6 show the model results. Two runs of the model are considered, the first under conditions summarized in table 1 (call this ‘case 1’), and the second when the recovery rate ν has been decreased to 0.04, which also has the effect of changing β to 0.20208 (‘case 2’). By decreasing the recovery rate, more contagious people can accumulate at any given time, but because β also changes in response to the change in ν , the result is not necessarily much larger epidemics as measured by the height of the peak (cf. figure 3). That β changes when ν does is a consequence of the equilibrium method for calculating β , described above.

Figure 2 shows the susceptible population at the end of the starting equilibrium discussed above. This is the result of the model with no rumor transmission, so the dynamics leading to the initial conditions are linear. This initial population does not change when rumor parameters change. Note that the susceptible population is mostly children and teenagers, but there are a non-negligible number of adults susceptible to the rumor. Tweaking the model parameters can change the shape of this curve; for instance a higher value of $\tilde{\kappa}$ would decrease the proportion of adults. Given that this

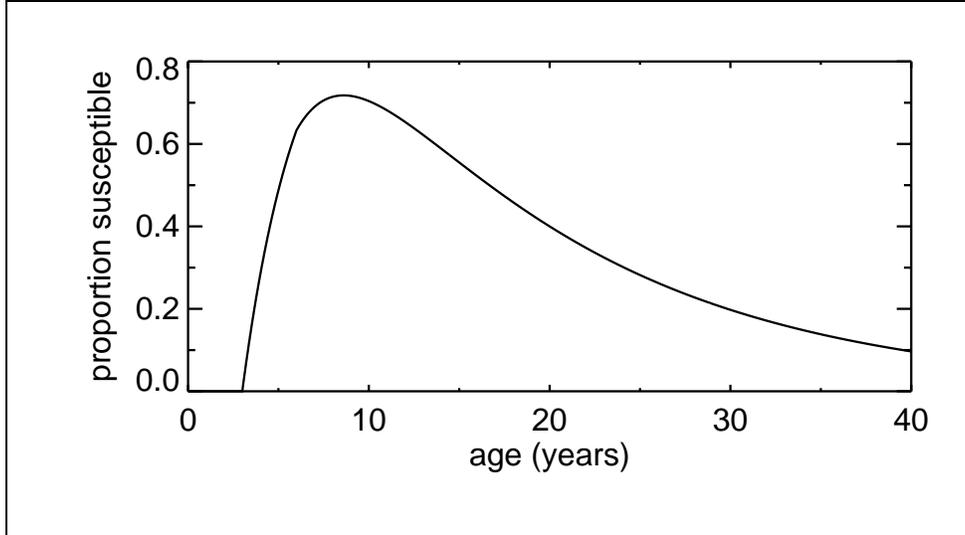


Figure 2: The initial condition of the model: proportion susceptible by age. This distribution represents the equilibrium state of the model when $\lambda(t) \equiv 0$.

is a hypothetical rumor, the curve in figure 2 is acceptable.

The dynamics of the model with rumor epidemics are shown in figure 3. The initial outbreak—when the rumor is new to all members of the population, old and young—is the most intense. But more people infected means, before too long, more becoming skeptical, and the epidemic burns itself out. In case 2, the rumor prevalence drops well below 1% of the population, but never crashes to an extremely low number as in case 1. Over time, many of these skeptics die, and new cohorts of susceptibles are born. After about fifteen years, there are enough susceptibles to sustain transmission with a reproductive rate (R —the number of daughter infections caused by each infection) greater than unity, and a new epidemic occurs (see Anderson and May 1992 for a detailed discussion of the reproductive rate). As Granovetter (1978: 1420) notes, rumor spreading is a threshold phenomenon. Here,

$R > 1$ is the threshold necessary to sustain an epidemic.

The second epidemic is less intense than the first—the oldest in the population still remember the first time that rumor went around, and they don't believe it—but it lasts longer before burning out. The rumor is never re-introduced explicitly. It persists at low rates in the population, which can have two interpretations. The first is that there are some diehards who won't let the story rest, and who find the occasional recruit, even after a recent epidemic has wiped out most susceptibles in the population. The real problem with this interpretation is that persistent adherents to the rumor are not an explicit part of the model—the recovery rate ν applies to everyone. The second interpretation is extinction. The simulation uses decimals, and a number that is well below unity can be taken to mean zero. In this interpretation, the second epidemic must follow a re-introduction of the rumor. Historical measles epidemics in island populations followed the pattern of epidemic, extinction, and re-introduction (Cliff, Haggett and Smallman-Raynor, 1993).

Later epidemics become more diffuse and the inter-epidemic level becomes higher. The long-run trend of such dynamics is stable transmission, such that there is never an epidemic, but a constant number of people who believe the rumor at any given time. This type of model shows how an epidemic-to-endemic path can occur, such that an urban legend can go from being a new “crazy story” that burns out quickly, only to be resurrected later, and eventually believed by a constant fraction of the population. The long-run approach to stability is seen more clearly in case 2 because it has higher inter-epidemic levels (figure 3). Using this model to explain the per-

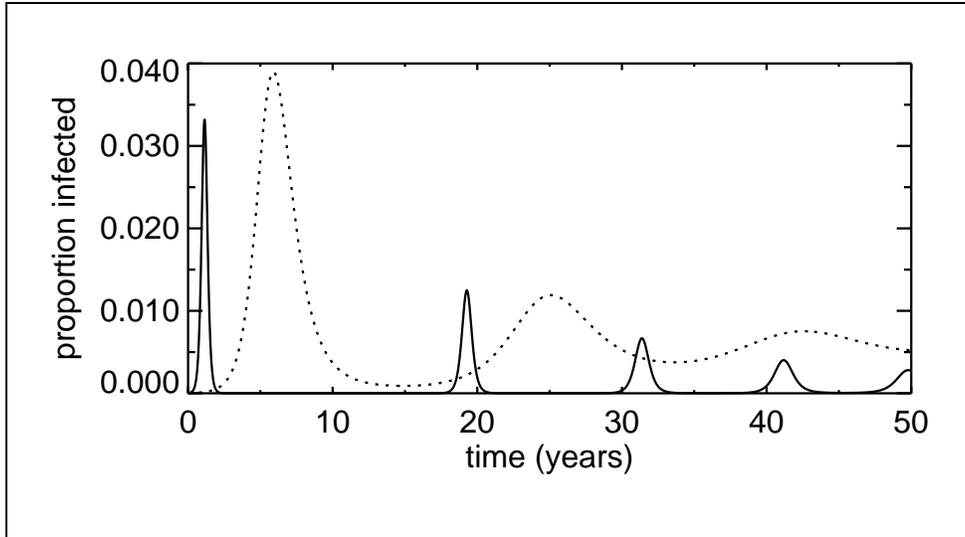


Figure 3: Epidemic curve. The initial outbreak is the most intense, but as the epidemics decrease in intensity, the rumor tends towards endemicity, with wider peaks and higher inter-epidemic baselines. Solid curve: case 1; dotted curve: case 2 (see text).

sistence of urban legends is viable, but it requires tolerance of the epidemic-to-endemic cycles, with the possibility of re-introduction as noted to keep the rumor going between the first few cycles.

Epidemic curves plot either proportion infected or prevalence (absolute numbers infected) over time. Only current infections are counted, not those who ever believed the rumor. This is an important distinction between epidemic models and the large body of innovation diffusion literature, where the well-worn result is a logistic or S-shaped adoption curve (Dodd 1952a,b, Rogers 1995). Adoption is analogous to ever having believed the rumor, the plot of which would indeed be S-shaped for any given epidemic peak.

The age-structured epidemic model is useful not only because it generates more realistic dynamics, but because it allows us to break down these

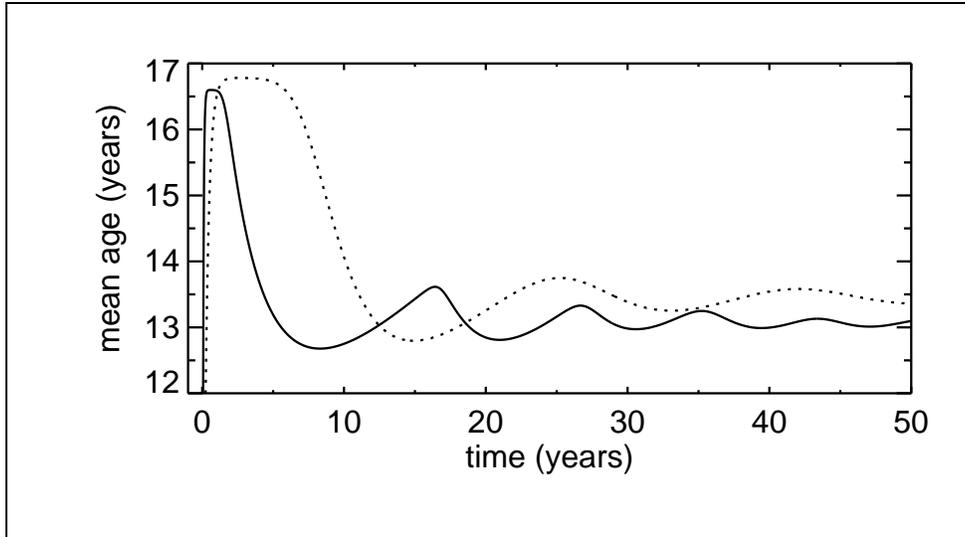


Figure 4: Epidemic curve—mean age of infection. During outbreaks, the mean age of infection increases. Solid curve: case 1; dotted curve: case 2 (see text).

dynamics by age. Figure 4 shows the average age of those infected over time. The average age of infection increases during the epidemics. There is an interesting point here about gullibility: it depends on time as well as age. The same rumor may be believed by different age groups, depending on whether or not the population is in an epidemic state or an inter-epidemic state. For example, an older teenager may note smugly that a certain rumor is only believed by younger children (the average age of believers is below 13 after the first or second epidemic in figure 4), when in fact the *same* rumor had an average age over 16 in its first appearance. The age at which a group is collectively too wise to believe a rumor depends as much on cohort experience as it does on any inherent skepticism that comes with age. The standard deviation of the age distribution of believers is plotted in figure 5; note that this tends toward a limit along with the mean.

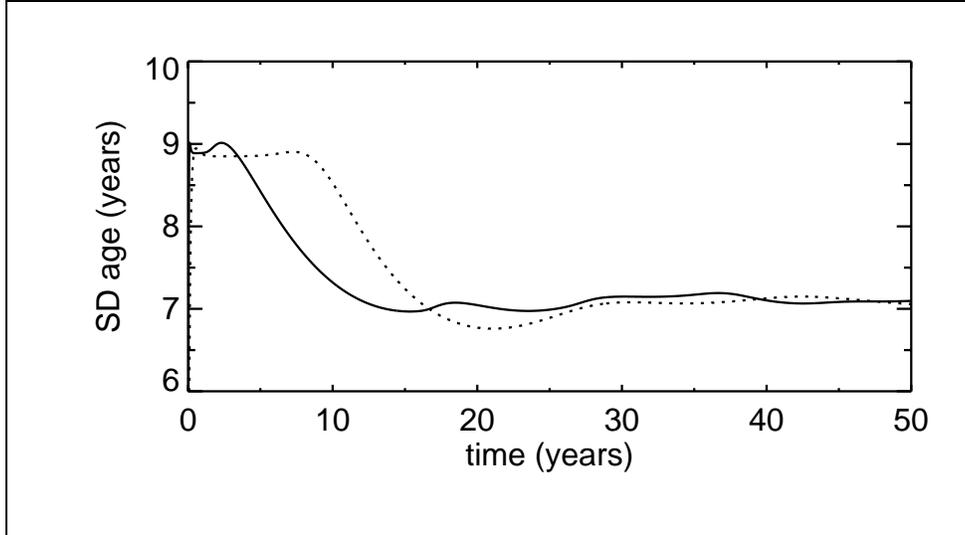


Figure 5: Epidemic curve—standard deviation of age of infection. Solid curve: case 1; dotted curve: case 2 (see text).

Figure 6 represents a Lexis surface of the rumor epidemics (case 2). The Cartesian plane is age and time, and the wire-frame surface is the prevalence of the rumor (i.e. number infected). This surface provides an illustration of why the mean age of infection gets older during an epidemic. The first (leftmost) epidemic in figure 6 is the most peaked, and has a heavy tail at older ages (age increases front-to-back). The surface of the second epidemic brings out visually the point about aging and susceptibility. The age distribution of the second epidemic is markedly younger, and unlike the first epidemic, there is a drop-off in the surface above age 20. Cohorts, aging one year in age for each year of time, move diagonally (45°) up the age-time plane. Older people became infected in the first epidemic, and hence are no longer susceptible. Thus, where the heavy tail of the second epidemic *would* be, lie the people who were in the height of the first epidemic peak

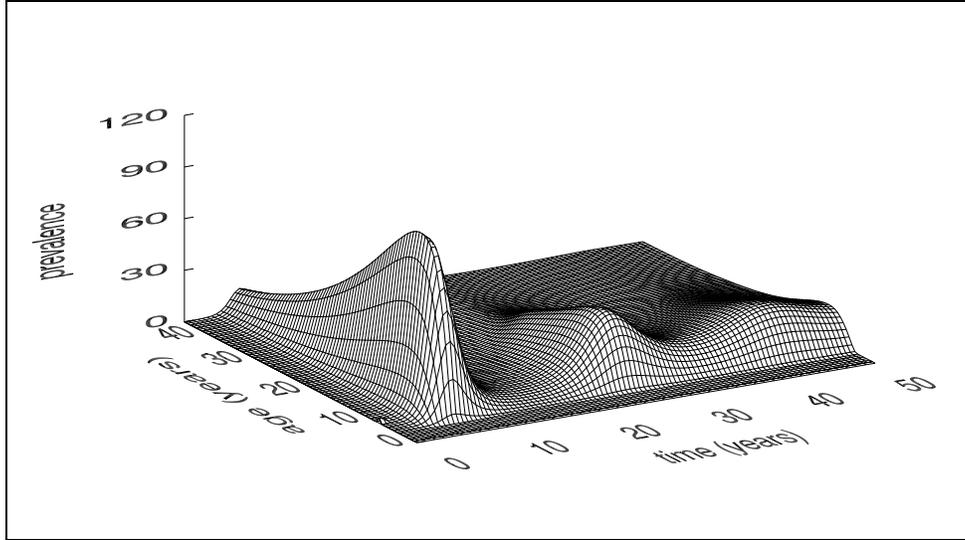


Figure 6: Lexis surface, rumor epidemics (case 2). The bottom Cartesian plane is age and time, and the wire-frame surface is the prevalence (number infected). Note the younger age distribution of the second and third epidemics compared to the first.

(and are therefore immune). Epidemics not only consume their own supply of susceptibles, and thus burn out, but they also clip the magnitude of later epidemics by converting young people to skeptics, who become immune for life. In this way, each epidemic has a slightly younger age distribution than the previous epidemic. Stable equilibrium is achieved when the recruitment of new (young) susceptibles balances those becoming skeptical. Thus the mean age and standard deviation are lower at equilibrium than during the early epidemics (figures 4 and 5).

4 Model II: Autocatalytic Skepticism

The epidemic model of rumor transmission shows how rumors can become durable, following a period of epidemics. This section develops a modified

version of the model which has some advantages over the epidemic model.

4.1 Model description

The modified model is mostly the same as the first one, and only the differences will be touched upon. Suppose, unlike measles, infection with a rumor does not decay with some constant rate ν . After all, if someone believes a rumor in the first place, why should she spontaneously stop believing the rumor? Suppose instead that the rumor is believed indefinitely until it is challenged through contact with skeptics. Replace the constant ν with the force of skepticism, $\nu(t)$, which is motivated as follows.

The parameter β represents a combination of population mixing and the probability of successful rumor spread, conditional on contact between a susceptible and a contagious individual. Call this probability of successful rumor transmission p , such that

$$\beta = p\tilde{\beta}.$$

In the model framework, β is already known, so with some value for p the pure mixing parameter $\tilde{\beta}$ can be calculated.

Assume that the rate of mixing between susceptibles and contagious is the same as between contagious and skeptical. This assumption is very sensible unless the contagious can differentiate, ex-ante, between skeptics and susceptibles, and seek out the latter, which seems far-fetched. The

force of skepticism is then defined analogously to the force of infection $\lambda(t)$:

$$\nu(t) = \gamma \frac{Z(t)}{N}$$

where:

$$\gamma = q\tilde{\beta}.$$

Since $\tilde{\beta}$ represents population mixing, q represents the probability that a contagious person will become a skeptic, conditional on contact with a skeptic. Thus, skeptics transmit their immunity to the contagious in the same fashion that the contagious transmit the rumor to the susceptible. This is autocatalytic because it is a self-reinforcing process where the force of skepticism increases with the proportion of skeptics. I assumed $p = 0.8$, and for q I test a case where those believing the rumor are fairly ready to change their mind ($q = 0.3$), and a case where those believing the rumor are loathe to be skeptical ($q = 0.01$). Note that a low value for q does not imply high gullibility at the population level; it means that, conditional on falling for the rumor in the first place, there is reluctance to be convinced the rumor is false. All other equations and parameters of the model and procedures of implementation remain unchanged.

4.2 Autocatalytic Model Results

When q is moderately large, that is, when there is some willingness among rumor carriers to concede that the rumor is false, the autocatalytic model behaves a lot like the previous result (figure 7). The solid curve of figure 7 ($q = 0.3$) would be about the same height as the curves in figure 3 if it

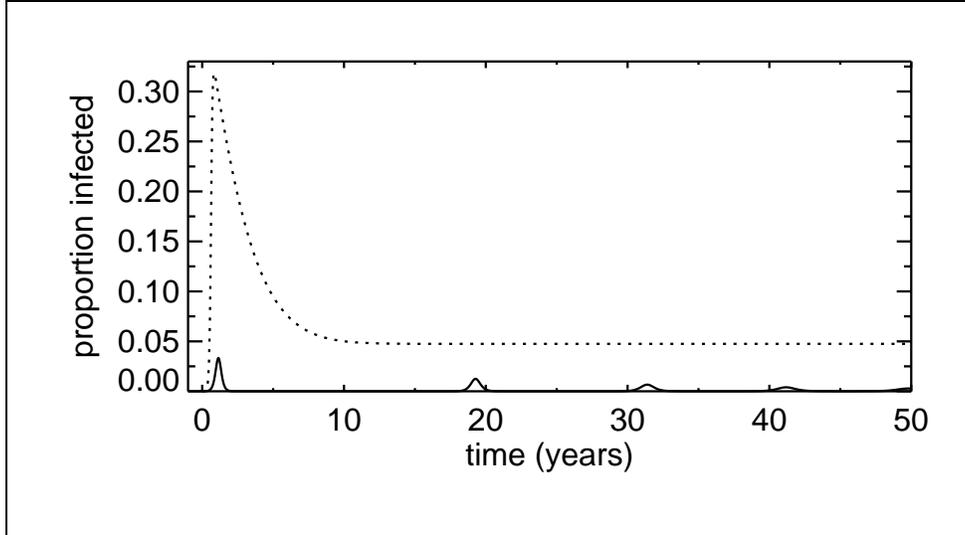


Figure 7: Epidemic curve, autocatalytic-skeptic model. Solid curve: $q = 0.3$; dotted curve: $q = 0.01$ (see text).

were plotted on the same axes. However, when the resistance to skeptics is high (i.e., q is very low), the rumor has a huge initial epidemic of 30% of the population, followed by rapid and cycleless convergence to a stable equilibrium. Figures 8 and 9 are illustrative. The mean age and standard deviation of age when $q = 0.3$ is approximately the same as in the epidemic models. When there is convergence ($q = 0.01$), there is a much younger mean age of infection and much smaller standard deviation of age of infection than the other models. Figure 10 explains this younger and tighter age distribution of infecteds: after the initial epidemic, only a narrow rib of young people continue to believe the rumor. As they age, they become skeptical, but are replaced by new adherents to the rumor. Figure 11 plots the equilibrium age distribution from figure 10.

The autocatalytic case leading to quick convergence is worth examining

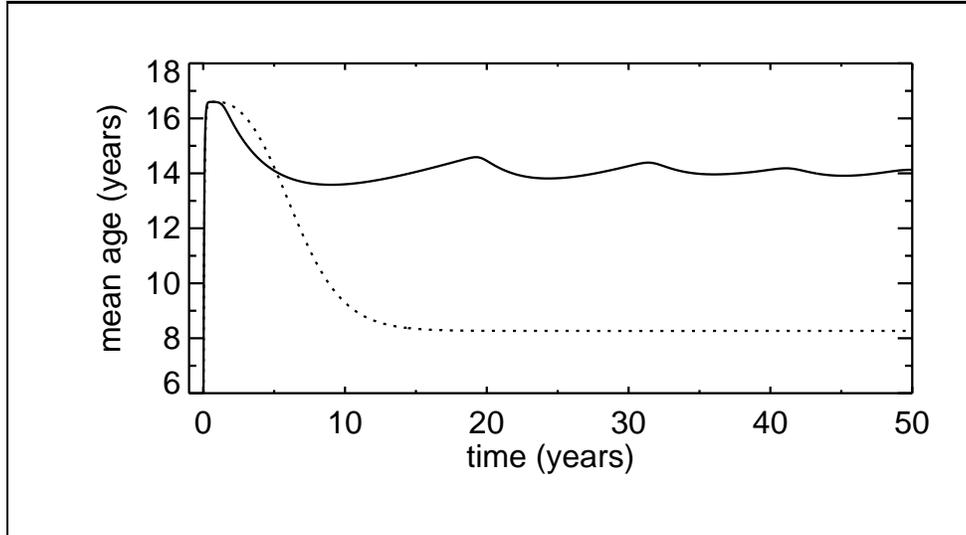


Figure 8: Epidemic curve—mean age of infection, autocatalytic-skeptic model. During outbreaks, the mean age of infection increases. Solid curve: $q = 0.3$; dotted curve: $q = 0.01$ (see text).

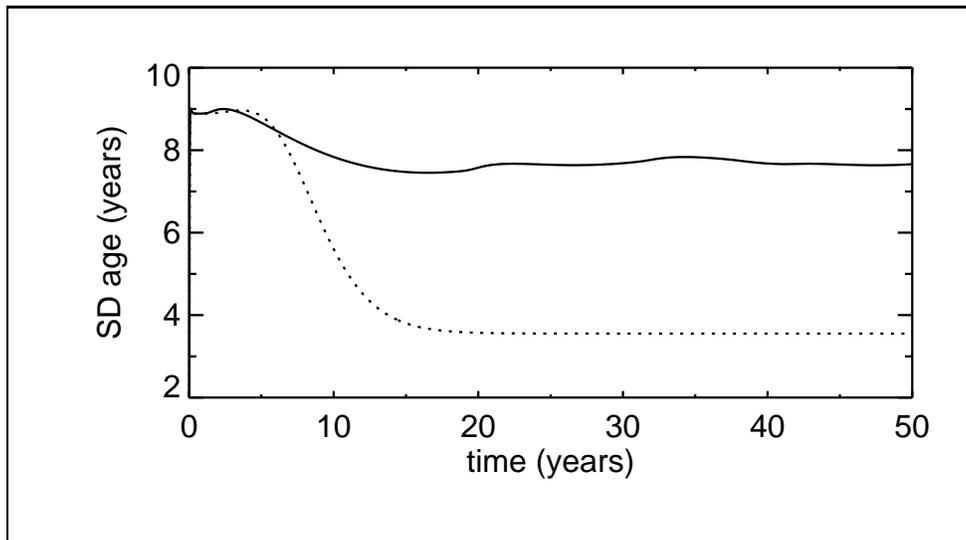


Figure 9: Epidemic curve—standard deviation of age of infection, autocatalytic-skeptic model. Solid curve: $q = 0.3$; dotted curve: $q = 0.01$ (see text).

more closely. The initial epidemic in this case is large, but that is not a mystery: there is reluctance to become skeptical, so larger numbers believing the rumor accumulate at any given time. And the age distribution of the initial epidemic is not markedly different between figures 6 and 10. The reluctance to give up belief in the rumor is not absolute—as the equilibrium distribution (figure 11) shows, almost nobody above age 20 believes the rumor. But the reluctance to change one’s mind is large enough to provide convergence: again, looking at figure 10, where the first epidemic peak would descend to an inter-epidemic interval, the infected population instead declines slightly and plateaus. Because more infected people means more rumor-spreading, the young are recruited into the rumor almost as soon as they are capable of understanding it. The result is a push-and-pull between rumor spreaders and skeptics. The elevated number infected increase recruitment, but the unlike in the first model, skeptics ensure that as time goes on, fewer-and-fewer of the infected remain so, and thus the age tail in figure 11 is not heavy (i.e. compared to the initial outbreak). In the end, there is a perfect balance between the opposing forces.

Figures 6 and 10, by showing age and time and rumor prevalence, yield insights that help understand the more simple epidemic curves of proportion versus time. In this case, understanding the stable equilibrium of rumor transmission without examining age would be shortsighted. It is the constant supply of young susceptibles—provided by births—that drives the equilibrium. Ironically, when skeptics discourage others from believing the rumor, the rumor itself just becomes more entrenched in the population and the mean age of infection shifts to a lower equilibrium value (more suscep-

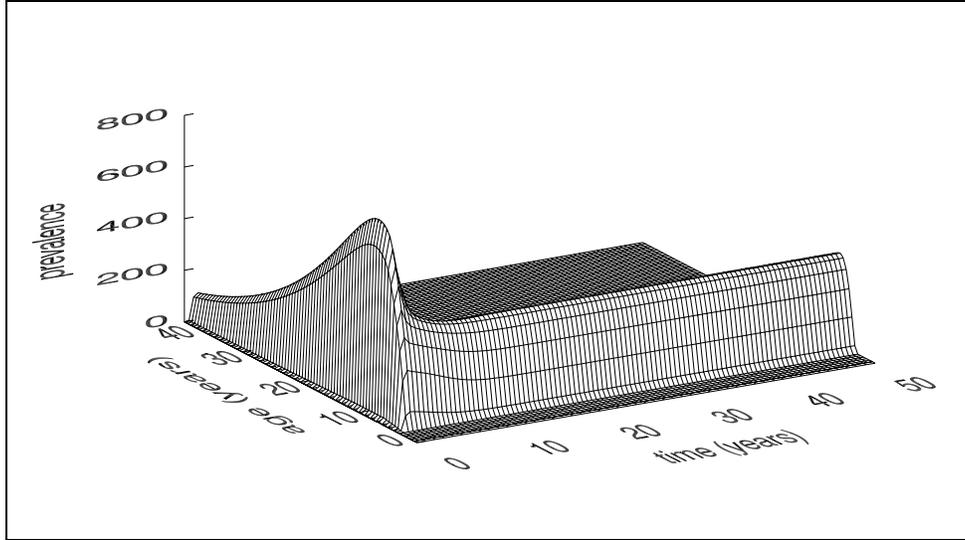


Figure 10: Lexis surface, rumor epidemic ($q = 0.01$). See text.

tibles are to be found at younger ages). Given an equilibrium prevalence of around 5% of the population, the rumor is clearly alive and well despite (indeed, perversely, because of) the efforts of the skeptics.

5 Conclusion

The ‘exploding *Pop-rocks*’ legend persisted for *years*, and indeed may still be in circulation. As noted, the ‘cookie recipe’ legend is thought still to persist. These are not a true accounts, but given enough time, stories, true or false, seem to get a life of their own—think of the story of George Washington confessing to cutting down the cherry tree. More generally, however, rumors are no laughing matter. Rumors, even falsifiable ones, can force actors to change their behavior. One example from business: Procter & Gamble, the consumer-products company, conscious of its corporate reputation, removed

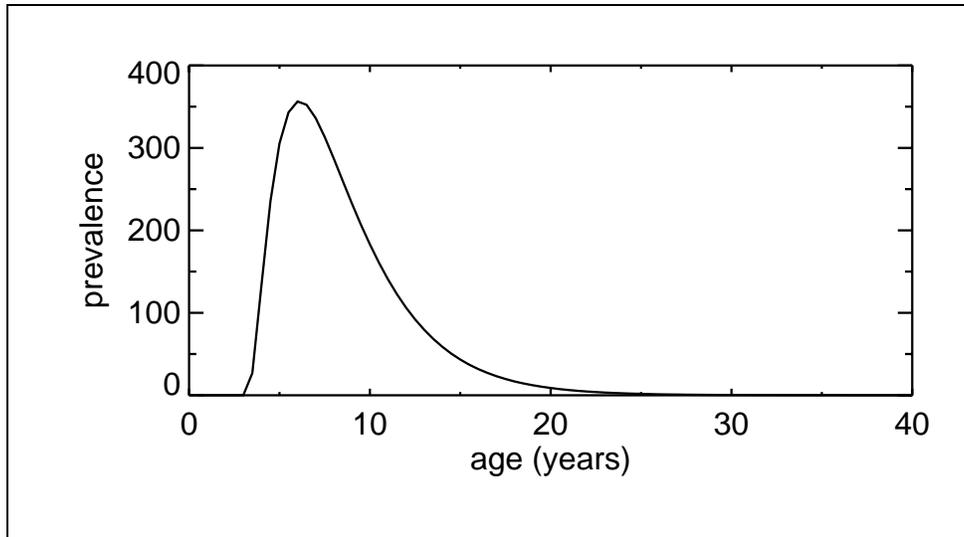


Figure 11: Equilibrium distribution in age of figure 10.

the characteristic moon-and-stars device from its products because of rumors it was a satanic symbol (Kapferer 1990). Rumors are closely related to diffusion of new ideas, collective behavior phenomena such as bank panics and riots, fads and fashions, drug use and crime (Wilson 1985), and other important social phenomena.

Quantitative data collection in this area would be difficult at best. To collect survey data, stratified by age, over many years, about knowledge and belief of a specific rumor, would be a daunting task. Design of such a study would be dogged by issues of accurately distinguishing belief in the rumor from knowledge of it. There would be the contaminating effect of the survey instrument itself (especially if the rumor is plausible). Not to mention garden variety sample size problems if the rumor is more obscure. This is why comparing alternative theories through modeling can be a profitable exercise.

The ‘data’, broadly construed, that the present models address is the fact that some rumors are known to have staying power that cannot be explained by tenacious persistence of belief among individuals. The *Pop-rocks* rumor has the look-and-feel of a rumor spread among youth; it is less likely a topic of conversation at the office watercooler. But the rumor circulated long enough that its original adherents were already grown up while it was still in circulation. These observations are not sufficient to validate quantitative aspects of the results, especially the simulated Lexis surfaces. Nonetheless, they are social data that can be explained, and the model results are congruent with these observations. Models such as the one in this paper allow us to experiment in ways that are impossible in the real world. Being able to borrow techniques from epidemic modeling guides realistic assumption-making about population mixing, because mathematical epidemic models have been validated many times with empirical data.

The first two rumors discussed in the introduction were specifically chosen for their youthful character. Though the models are generalizable to any age group, as implemented here they show rumor spread among the young. As in other aspects of social life, age in these models acts as a proxy for experience (Ryder 1965). The cookie recipe legend is an illustration of this, because susceptibility to an electronic mail hoax seems to decline with the amount of time someone has been using electronic mail.

Henry Rosovsky (1990: 251) describes a similar phenomenon in the arena of management, specifically of running a university:

As a group, students have short memories, and the same issues arise year after year as new student leaders reach the limelight.

New leaders will, with considerable regularity, accuse administrators of not being responsive to their demands, even though a clearly negative reply was given annually for the past decade.

Note that he draws a distinction, as does the present work, between individual student memory and collective student memory. What is clearly at work in the case described by Rosovsky is that new students—one fourth of the undergraduate population—are recruited each year. These are like newly-born ‘susceptibles’ in the current framework, and their grievance is analogous to the rumor. What is important in the collective persistence of the students seeking redress is not their individual tenacity, but the significant annual turnover in the college population. The students’ (in)experience, not their age, is what is at issue. Stock traders’ susceptibility to rumors in the market is another example where number of years of experience is more relevant than age.

As shown by the student grievances, the persistence of any idea can be thought of like a rumor, whether or not the idea would be called a ‘rumor’ in everyday parlance. Thus there are specific differences—but no clear boundary—between the study of persistent rumors, as in the present work, and the study of collective memory (e.g. as conceived by Halbwachs [1992]). In the present work, I deal with beliefs that persist in a population despite the fact that original carriers have ceased to believe. In the case of collective memory, older cohorts (generations) do not necessarily lose their belief. But in the long-run, everyone is mortal, and collective memory phenomena depend on the transmission of ideas from older ‘infecteds’ to younger ‘susceptibles’.

In summary, this paper motivated two simple age-structured nonlinear models of urban legends, and the most rapid path to endemicity (persistence) occurs when skeptics play an active role in trying to suppress a rumor, a process I label ‘autocatalysis’. This is counterintuitive, since autocatalysis of skepticism should suppress rumors. Rapid convergence to a stable equilibrium, without intervening epidemic cycles, occurs under autocatalysis when those who believe a rumor are reluctant to give up their belief. Reluctance to change one’s mind about a rumor can occur when there is no dispositive evidence that the rumor is false, so rumors about distant events may be more persistent than local rumors. When skeptics try to stop a rumor from spreading further, the nature of the dynamics changes from epidemic cycles to endemic transmission; skeptics actions are at cross-purposes to their intentions. These results are only possible when age is included in the model, because age and time combine to make the cohort concept meaningful, and it is the influx of younger cohorts that is important here. Viewed another way, the paradox of long-lived rumors is a micro-macro disconnect. Individuals do not believe the rumor for a particularly long time, yet the rumor persists in the population seemingly unabated for years. These models show that in an age-stratified population, the paradox is much easier to understand. That this micro-macro phenomenon can be explained by age effects suggests strongly that age structure should be incorporated into mathematical models whenever practical.

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