The dynamics of two-stage contagion

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Abstract

We explore simple models aimed at the study of social contagion, in which contagion proceeds through two stages. When coupled with demographic turnover, we show that two-stage contagion leads to nonlinear phenomena which are not present in the basic ‘classical’ models of mathematical epidemiology. These include: bistability, critical transitions, endogenous oscillations, and excitability, suggesting that contagion models with stages could account for some aspects of the complex dynamics encountered in social life. These phenomena, and the bifurcations involved, are studied by a combination of analytical and numerical means.

1 Introduction

The notion of social contagion has been gaining increasing prominence, with accumulating empirical evidence of its importance for many aspects of our lives, from political mobilization, spread of ideas and innovations, and psychological well-being, to substance abuse, crime and violence, obesity, financial panics, and mass psychogenic illness [8, 12, 22, 36].

Contagion phenomena have traditionally been mathematically modelled in the field of infectious disease epidemiology [29, 38], and it is natural to apply the tools developed in this field to social contagion, as is indeed being done by researchers from diverse fields [6, 33, 40, 43]. Models for various instances of social contagion have been developed, including smoking [7], drug use [47], bulimia [19], political activism [28, 39], spread of rumors [16], crime [17], language dynamics [37], organized religions [23], diffusion of new products and technologies [2, 18] or ideas in a scientific community [3], among many more examples.

It is important to address the various aspects in which social contagions differ from biological contagions at the level of the individual, and the consequences of these differences for the dynamics of these phenomena at the population level. In elucidating this micro-macro link, mathematical modelling plays an important role, as it allows us to study the population-level patterns emerging from different contagion mechanisms, which are often far from intuitively obvious, and which may be quite different from those familiar from the study of classical models for the transmission of infectious diseases.

One important way in which social contagion mechanisms differ from those of
biological contagion is that while infection with a pathogen is a discrete event occurring upon contact of a susceptible individual with an infectious individual, social contagion may require transitions through several stages, with each such transition dependent on contact with ‘infectives’. This is a central tenet of Rogers’ Diffusion of Innovations theory [41], which studies the spread of innovations - including, for example, technologies, products, ideas, cultural practices, health-related behaviors, and more. Rogers’ theory posits that the adoption of an innovation, at the individual level, involves a series of five stages in which the individual comes to learn about the innovation, assess it, and finally adopt it. The transition rate from one stage to another is modulated by the interpersonal influence of other individuals in one’s social network, and hence depends on the number of people who have already adopted the innovation. As another example from the social sciences, Klandermans and Oegema [30] analyse the process of becoming a participant in a social movement as consisting of four stages: becoming part of the mobilization potential, becoming the target of mobilization attempts, becoming motivated to participate, and overcoming barriers to participation. A closely related idea is that of complex contagion [8], referring to social contagions which require sustained or repeated contacts with adopters in order to spread.

To incorporate the idea of stages of contagion into mathematical models, one can divide the population into classes, each of which consists of individuals at a certain stage in the adoption process, with movement among the classes due to contact with adopters. Models of this type have been proposed studied in several works [4, 10, 11, 13, 14, 21, 26, 27, 32], and in section 1.2 we will briefly describe and compare these with the model studied here.

Our aim here is to make a detailed study of the dynamics of a two-stage contagion model, which, unlike in nearly all previous works, incorporates the process of demographic turnover - recruitment and departure of individuals from the population. This may be due to births and deaths or, if considering a contagion spreading in particular institutional settings or age groups, individuals entering and leaving the institution, or maturing into and out of the relevant age group. Our model is thus a two-stage analog of the classical SIR model with demographic turnover [29, 35]. We will show that stages of contagion, in conjunction with demographic turnover, lead to new and sometimes surprising dynamical phenomena, which are not present in the basic one-stage models familiar in mathematical epidemiology. The interesting behaviors we observe in our simple model suggest a possible generative mechanism for some of the complex phenomena observed in the social world: alternative stable states, discontinuous transitions, critical mass effects, and periodic cycles.

Since we wish to highlight the fact that stages of contagion lead to novel phenomena in the simplest of models, we resist the temptation to generalize by incorporating more mechanisms or more stages of contagion. The simplicity of the present models also provides the advantage that we can go quite far in characterizing their dynamics analytically, in different parameter regimes, using standard tools of stability analysis of equilibria. Some aspects of the dynamics, however, will be studied numerically, and obtaining full mathematical proofs for some of our conclusions from these simulations seems like a challenging task for the future. Any phenomena present in our minimal model will also occur
**1.1 The models**

We assume that the population is divided into three classes: class $S_0$ consists of 'naive' individuals who have not been exposed to the innovation, class $S_1$ consists of 'informed' individuals who have encountered the innovation but have not yet adopted it, and class $A$ consists of adopters. We choose the terminology of 'adoption of innovations' for convenience; the models could just as well describe potential supporters of a social movement ($S_0$), actual supporters ($S_1$), and activists ($A$), or many other examples of social contagion.

The mechanisms involved in the models are:

- Adopters randomly encounter other individuals, at a rate of $\beta$ effective contacts per unit time.
- Upon effective contact with an adopter, a naive individual adopts the innovation with probability $p$ ($0 < p < 1$), and otherwise becomes informed.
- An informed individual adopts the innovation upon encounter with an adopter.
- Demographic turnover (recruitment into and departure from the relevant population, for example through births and deaths) occurs at per capita rate $\mu$, so that $\mu^{-1}$ is the mean residence time of an individual in the population (e.g., the life expectancy). Since recruitment and departure rates are assumed equal, we are assuming a constant population size. It is also assumed that individuals entering the population are naive (class $S_0$).

Denoting the fraction of the population in each of the three classes at time $t$ by $S_0(t)$, $S_1(t)$, $A(t)$ (so that $S_0(t) + S_1(t) + A(t) = 1$), the above assumptions, represented also in the diagram of figure [1], translate, in the standard way [29, 35], into the following differential equations:

\[ S'_0 = \mu - \beta S_0 A - \mu S_0, \]
\[ S'_1 = (1 - p)\beta S_0 A - \beta S_1 A - \mu S_1, \]
\[ A' = \beta[p S_0 + S_1] A - \mu A. \]

\[ a \text{ ffortiori} \] in more elaborate models, for some parameter values, and such models might give rise to further interesting dynamics not present in the two-stage model, and deserve to be studied further.

In the remainder of this section we introduce the two models to be studied, one in which an individual’s adoption of an innovation is permanent and one in which it is temporary, and make a comparison with previous models incorporating two-stage contagion. The permanent adoption model will be studied in section 2 and the temporary adoption model, which leads to a richer variety of possible dynamical behaviors, will be studied in section 3. In section 4 we will recap the novel features that the two-stage contagion models have in comparison with their classical one-stage counterparts, and discuss their significance.
This is the permanent adoption model. In our second model, the temporary adoption model, we assume that adopters abandon the innovation (or activists in a social movement become ‘burned out’) at a constant per capita rate $\gamma$, so that the mean duration of adoption is $\gamma^{-1}$. It is further assumed that those who have abandoned the innovation will not re-adopt it. This adds an additional class of removed individuals $R$ (see figure 2), and changes the model equations to

\begin{align}
S_0' &= \mu - \beta S_0 A - \mu S_0, \quad (4) \\
S_1' &= (1 - p)\beta S_0 A - \beta S_1 A - \mu S_1, \quad (5) \\
A' &= \beta[pS_0 + S_1] A - (\gamma + \mu)A, \quad (6) \\
R' &= \gamma A - \mu R. \quad (7)
\end{align}

It should be stressed here that a crucial feature of the two-stage contagion models is that both transitions between stages depend on contagion. A model in which the transition from stage $S_1$ to stage $A$ were a spontaneous one, that is occurring at a constant per capita rate, would be equivalent to the standard SEIR model \cite{29, 38}, and would not generate any of the interesting behaviors that are our focus here.

### 1.2 Previous work on two-stage contagion models

We briefly survey previous work on two-stage contagion models of which we are aware, and compare the models which have been investigated with the model studied here.
In the statistical physics literature, a two-stage contagion model was introduced in \cite{26, 27} under the name Extended General Epidemic Process (EGEP). Like the model considered here, this model includes two stages, with contagion leading either directly to adoption ($A$) or to moving to a ‘weakened’ stage ($S_1$), which upon further contagion can lead to adoption, and with permanent removal of adopters to a recovered stage (temporary adoption). An equivalent model is introduced in \cite{9}, as a way to describe co-infection with two pathogens, under some symmetry assumptions. A similar model was introduced in \cite{32}, with the aim of describing innovations (with permanent adoption) and fads (with temporary adoption), and including an arbitrary number of stages, though without direct movement of naive individuals into the adopter class. The models above do not include demographic turnover, so that there is no mechanism for the renewal of the naive population, and they therefore generate transient epidemics rather than endemic states (except for the permanent adoption model of \cite{32}, in which the entire population eventually adopts). Their investigation thus centers on the size of the epidemic, that is the total number of individuals who become infected before it fades. Of prime interest here is the fact that, in contrast with the one-stage epidemic model, in the two-stage model one has, under certain conditions, a discontinuous (‘first order’ in the language of statistical physics) dependence of the total size of the epidemic on the contact parameter. This has led to interest in the statistical physics community, and several works investigate the two-stage contagion process on lattices and random graphs \cite{4, 10, 11, 13, 21}.

In \cite{46}, which develops models inspired by Rogers’ Diffusion of Innovation Theory \cite{41}, mechanisms inducing renewal of the naive population are introduced, so that an endemic contagion becomes possible, as in this work. However, there are also some significant differences between the assumptions in \cite{46} and in our models, which we now detail.

In the first model of \cite{46}, it is assumed that adoption is temporary, but the adopters who abandon the innovation return to the naive class so that they may re-adopt (this is analogous to the SIS model in epidemiology). In addition, informed individuals (class $S_1$) move back to the naive stage (‘forgetting’) at a constant per capita rate. These two flows are the mechanism providing renewal of the pool of naive individuals – the model does not incorporate demographic turnover. By contrast, in our model we do not allow individuals to move back into the naive stage, and the renewal of the naive pool is provided by demographic turnover. This is not a trivial difference, as may be seen from the fact that the model of \cite{46} reduces to a two-dimensional one, whereas our temporary adoption model is essentially three dimensional and cannot be reduced to a two-dimensional one. The appropriateness of one set of assumptions or the other (or some different combination of assumptions) is dependent on the precise nature of the contagion phenomena involved, as well as on the relevant time scale. One can construct a larger model including both models, but in order to understand the specific contribution of each mechanism there is much advantage in investigating simple models highlighting that specific mechanism.

The models of \cite{46} also included the mechanisms of ‘spontaneous’ transition of naive individuals to the informed stage and of informed individuals to adoption stage, at constant per capita rates, as a way to model the effects of mass-media. Indeed for some of the results in \cite{46} it was assumed that the mass-media
effects are strong relative to the contagion effect. Here we treat the opposite extreme of pure contagion, without adding the media effects, as in the previous works on the EGEP model. Since contagion effects are nonlinear while media effects are linear, some of the interesting nonlinear phenomena which follow from the contagion effects are muted or cancelled when media effects are sufficiently strong. When media effects are present but sufficiently weak, behavior will be similar to the pure-contagion case, by structural stability. On the other hand, the model of [46] does not include direct contagion of naive individuals into the adopter class. Here we do include this mechanism (controlled by the parameter $p$), as in the EGEP model, which is in fact essential for the generation of some of the more interesting phenomena, such as the periodic oscillations.

A further element which is introduced in the first model of [46] is the possibility of a nonlinear dependence of the per-capita transition rate from the informed class ($S_1$) to the adopter class on the number of adopters, that is replacing the term $\beta AS$ by a term of the form $g(A)S$, where $g$ is a nonlinear function. It is shown that choosing $g(A)$ to be quadratic leads to bistability. In our models we show that bistability occurs even without this additional mechanism, and we will not introduce such nonlinear dependence.

The second model of [46] introduces a time delay to model the intermediate evaluation stage, as well as demographic turnover, though not the direct contagion of naive individuals into the adoption stage. The time delay makes the stability analysis of equilibria considerably more difficult, but an interesting consequence of this delay is that it gives rise to periodic oscillations for some parameter values. Note that in our model with temporary adoption, we will show that periodic oscillations arise even in the absence of a delay. We will not consider delays in this work.

To conclude this short survey, we mention the work [14], which contains rigorous results on a stochastic two-stage contagion model on a lattice with return of adopters to the naive stage. In this work we restrict ourselves to mean-field deterministic models.

2 The permanent adoption model

In this section we analyze the dynamics of the permanent adoption model. It will be useful to define the dimensionless contact parameter

$$\delta = \frac{\beta}{\mu},$$

which plays a role similar to the basic reproductive number $R_0$ in epidemiological models: since an adopter spends an average time $\mu^{-1}$ in the adopter compartment before leaving the population, $\delta$ is the average total number of effective encounters that an adopter has.

We note that since $S_0 + S_1 + A = 1$, we can eliminate one of the variables, say $S_1$, by substituting

$$S_1 = 1 - S_0 - A,$$

(8)
and reformulate the model equations (1)–(3) as a two-dimensional system

\[ S_0' = f(S_0, A) = \mu - \beta S_0 A - \mu S_0, \]
\[ A' = g(S_0, A) = \beta [1 - (1 - p) S_0 - A] A - \mu A. \]

This defines a flow in the invariant region of the phase plane given by

\[ S_0 \geq 0, \ A \geq 0, \ S_0 + A_0 \leq 1. \]

We can use the Bendixson-Dulac criterion \[44\] to verify that this system does not have limit cycles. Indeed

\[ \frac{\partial}{\partial S_0} \left( \frac{1}{A} f(S_0, A) \right) + \frac{\partial}{\partial A} \left( \frac{1}{A} g(S_0, A) \right) = -2 \beta - \frac{\mu}{A} < 0. \]

Since this is a two dimensional system in a bounded region, the Poincaré-Bendixon theorem \[44\] implies that

**Proposition 1.** Every trajectory of the system (9), (10) approaches an equilibrium point as \( t \to \infty. \)

We therefore now study that equilibria of the model, which correspond to solutions of the algebraic equations

\[ \mu - \beta S_0 A - \mu S_0 = 0, \]
\[ \beta [1 - (1 - p) S_0 - A] A - \mu A = 0. \]

From (11) we have

\[ S_0 = \frac{1}{\delta A + 1}. \]

From (12) we have

\[ A = 0 \ \text{or} \ (1 - p) S_0 + A = 1 - \delta^{-1}. \]

If \( A = 0 \) then we obtain the contagion-free equilibrium

\[ E_0 : (S_0, S_1, A) = (1, 0, 0). \]

Assuming \( A \neq 0 \), and substituting (13) into (14) gives

\[ \frac{1 - p}{\delta A + 1} + A = 1 - \delta^{-1}, \]

which may be rewritten as a quadratic equation and solved to give

\[ A_{1,2} = \frac{1}{2} \left[ 1 - 2\delta^{-1} \pm \sqrt{1 - 4(1 - p)\delta^{-1}} \right]. \]

These solutions will correspond to endemic equilibria if and only if they are real and positive. We will denote by \( E_1 \) the equilibrium corresponding to \( A = A_1 \), with the other components given by (13), (8), and by \( E_2 \) the equilibrium corresponding to \( A_2 \). We now determine the conditions on the parameters under which these equilibria exist.
\( A, A_2 \) are real if and only if
\[
\delta \geq 4(1 - p). \tag{16}
\]

Assuming now that \((16)\) holds, we have
\[
A_1 > 0 \iff \sqrt{1 - 4(1 - p)\delta^{-1}} > 2\delta^{-1} - 1 \iff \delta > 2 \text{ or } \frac{1}{p} < \delta \leq 2. \tag{17}
\]

Noting that \(\frac{1}{p} \geq 4(1 - p)\), we get
\[
A_1 > 0 \iff \delta \geq \begin{cases} 4(1 - p) & \text{when } p \leq \frac{1}{2}, \\ \frac{1}{p} & \text{when } p > \frac{1}{2}. \end{cases}
\]

For the equilibrium \(E_2\), we have, assuming \((16)\) holds,
\[
A_2 > 0 \iff \sqrt{1 - 4(1 - p)d^{-1}} < 1 - 2d^{-1} \iff 2 < \delta < \frac{1}{p}, \tag{18}
\]
so that
\[
A_2 > 0 \iff p < \frac{1}{2} \text{ and } 4(1 - p) < \delta < \frac{1}{p}.
\]

To determine the stability of the equilibria, we linearize (9) around an equilibrium, obtaining the Jacobian matrix
\[
J = \begin{pmatrix} -\beta A - \mu & -\beta S_0 \\ -\beta(1 - p)A & \beta[1 - (1 - p)S_0] - 2\beta A - \mu \end{pmatrix}
\]

For the contagion-free equilibrium \(E_0\) we have
\[
J = \begin{pmatrix} -\mu & -\beta \\ 0 & \beta p - \mu \end{pmatrix},
\]
with eigenvalues \(-\mu, \beta p - \mu\), and we conclude that \(E_0\) will be stable when both of these eigenvalues are negative, that is when \(\delta p < 1\), and unstable if \(\delta p > 1\).

For the endemic equilibria \(E_1, E_2\) we have, using \((14)\)
\[
J = \begin{pmatrix} -\beta A - \mu & -\beta S_0 \\ -\beta(1 - p)A & \beta[1 - (1 - p)S_0] - 2\beta A - \mu \end{pmatrix}
\]
so that
\[
\text{tr}(J) = -2\beta A - \mu < 0, \quad \det(J) = \beta^2 A[2A + 2\delta^{-1} - 1],
\]
hence the equilibrium is stable if and only if \(\det(J) > 0\), that is iff
\[
2(A + \delta^{-1}) > 1 \tag{19}
\]
Using the explicit solutions \((15)\) we have that \((19)\) is equivalent to
\[
1 \pm \sqrt{1 - 4(1 - p)d^{-1}} > 1,
\]
which holds if and only if the + sign is taken. We conclude that \(E_1\) is stable whenever it exists, while \(E_2\) is always unstable whenever it exists.

We summarize the results of the preceding analysis.
Proposition 2. (I) If $p < \frac{1}{2}$ then:

- For $\delta < 4(1-p)$ there are no endemic equilibria, and the contagion-free equilibrium $E_0$ is stable.
- For $4(1-p) < \delta < \frac{1}{p}$ there are two endemic equilibria $E_1, E_2$, with $E_1$ stable and $E_2$ unstable, and the contagion-free equilibrium $E_0$ is stable.
- For $\delta \geq \frac{1}{p}$ there is a unique endemic equilibrium $E_1$, which is stable, and the contagion-free equilibrium $E_0$ is unstable.

(II) If $p \geq \frac{1}{2}$ then:

- For $\delta \leq \frac{1}{p}$ there is no endemic equilibrium, and the contagion-free equilibrium $E_0$ is stable.
- For $\delta > \frac{1}{p}$ there is a unique endemic equilibrium $E_1$, and the contagion-free equilibrium $E_0$ is unstable.

We now discuss the interesting dynamical consequences of the preceding results.

When $p \geq \frac{1}{2}$ (see figure 3 right), the behavior is similar to that familiar from a standard one-stage contagion model – the SI model with demographic turnover: if contact rate is low ($\delta < \frac{1}{p}$) then contagion cannot spread (the contagion-free equilibrium is stable, and there is no endemic equilibrium), and as $\delta$ crosses the invasion threshold $\delta = \frac{1}{p}$ the contagion-free equilibrium loses stability and an endemic equilibrium is born, so that contagion is established. This transition is a continuous one: for values of $\delta$ slightly above the threshold, the fraction of adopters $A_1$ is small, and it increases as $\delta$ increases.

Things are more interesting in the case $p < \frac{1}{2}$ (see figure 3 left), since in this case we have a critical transition at the endemicity threshold $\delta = 4(1-p)$, in which two endemic equilibria $E_1, E_2$ appear ("out of the blue"), so that contagion can establish at the level $A_1$ corresponding to the stable equilibrium $E_1$. At the endemicity threshold $\delta = 4(1-p)$ we have

$$A_1 = \frac{1}{2} \cdot \left[ 1 - \frac{1}{2(1-p)} \right] > 0$$

so that as this threshold is crossed the level of contagion can jump from 0 to a positive value. Note however that even above the endemicity threshold, the contagion-free equilibrium remains stable until the invasion threshold $\delta = \frac{1}{p}$ (which is larger) is crossed. This means that for values of $\delta$ between these two thresholds we have bistability - contagion may establish, or not, depending on whether the initial conditions belong to the basin of attraction of $E_0$ or of $E_1$. This is demonstrated in figure 4, in which we show the solution $A(t)$ for parameter values $p = 0.1, \delta = 5$, for two initial conditions: when the initial fraction of adopters is 4% the contagion is extinguished, while for an initial fraction 5% contagion is established, with equilibrium value of $A_1 = 56.5\%$ of the population. Thus under essentially the same conditions - that is the same parameter values and only slightly different initial conditions, the system may achieve radically different outcomes.
The critical transition displayed by this model has important implications with regard to changes in outcomes under variation of its parameters. If we assume that initially we are near the contagion-free equilibrium $E_0$, with $\delta < 4(1-p)$, and slowly increase the contact parameter $\delta$ (e.g. by increasing the contact rate $\beta$) then, as the endemicity threshold $\delta = 4(1-p)$ is crossed we will still be in the basin of attraction of the $E_0$, so that the population will remain contagion-free. This will continue until the invasion threshold $\delta = \frac{1}{p}$ is reached, at which point $E_0$ loses stability, and then we will observe an even more dramatic jump to the value $A_1$ corresponding to $\delta = \frac{1}{p}$, that is to

$$A_1 = \frac{1}{2} \cdot \left[ 1 - 2p + \sqrt{1 - 4(1-p)p} \right].$$

On the other hand, if initially $\delta > \frac{1}{p}$ and we are near the endemic equilibrium $E_+$ and slowly decrease $\delta$, then we will remain near the endemic equilibrium even as $\delta$ drops below the invasion threshold and the contagion-free equilibrium becomes stable. This will continue until the endemicity threshold $\delta = 4(1-p)$ is reached, at which point the endemic equilibria disappear and we will jump from the value given by (20) to a contagion-free state. We thus have the phenomenon of hysteresis in which discontinuous transitions occur at different values of the parameter, depending on whether it is increased or decreased.

In the extreme case $p = 0$ (no direct adoption by naive individuals), there is no invasion threshold and the contagion-free equilibrium is stable for all $\delta$. In this two endemic equilibria are born when $\delta = 4$, and we have bistability for all $\delta > 4$.

To conclude our discussion of the permanent adoption model, it is of interest to consider the dependence of the sizes of the different population fractions at the stable equilibrium $E_1$ on the contact parameter $\delta$. $A = A_1$ is given by (15),

$$A_1 = \frac{1}{2} \cdot \left[ 1 - 2p + \sqrt{1 - 4(1-p)p} \right].$$
Figure 4: Fraction of adopters $A(t)$, for $p = 0.1$, $\mu = 0.05$, $\beta = 0.25$ (so $\delta = 5$), for initial conditions: $A(0) = A_0$, $S_0(0) = 1 - A_0$, $S_1(0) = 0$, $A_0 = 0.04$, $A_0 = 0.05$.

Figure 5: Fraction of informed individuals $S_1$ at the stable equilibrium $E_1$, as a function of $\delta$, for $p = 0.4$ (left) and for $p = 0.6$ (right).

and by (8),(13), we have

$$S_0 = \frac{1 - \sqrt{1 - 4(1 - p)\delta^{-1}}}{2(1 - p)}, \quad S_1 = \delta^{-1} - \frac{p(1 - \sqrt{1 - 4(1 - p)\delta^{-1}})}{2(1 - p)}.$$  

While it is immediate that $S_0$ is monotone decreasing and $A$ is monotone increasing as a function $\delta$, the fraction $S_1$ of individuals at the intermediate stage is not monotone in $\delta$, as shown in figure 5. Indeed it is easy to calculate that the fraction $S_1$ is maximized at $\delta = \frac{4}{p}\frac{1}{\Gamma}$, attaining the value $S_1 = \frac{1 - p}{4}$.

3 The temporary adoption model

We now move to the analysis of the two-stage contagion model with temporary adoption, which displays a richer repertoire of dynamic behaviors. As for the
previous model, it will be useful to define the non-dimensional parameter

\[ \delta = \frac{\beta}{\gamma + \mu}, \]

which can again be interpreted as the mean number of effective contacts that an adopter makes during the period of adoption, so that it will be called the contact parameter.

3.1 Analysis of equilibria

3.1.1 Existence of equilibria

Equilibria of the model are given by solutions, with non-negative components, of the equations obtained by equating the derivatives in (4)-(7) to zero:

\[ \mu - \beta S_0 A - \mu S_0 = 0, \]
\[ (1-p)\beta S_0 A - \beta S_1 A - \mu S_1 = 0, \]
\[ \beta A[pS_0 + S_1] - (\gamma + \mu)A = 0, \]
\[ \gamma A - \mu R = 0. \]

We now analyze the solutions of this algebraic system.

From (21), (22), (24) we have

\[ S_0 = \frac{1}{\delta(\frac{\beta}{\mu} + 1)A + 1}, \quad S_1 = (1-p) \cdot \frac{\delta(\frac{\gamma}{\mu} + 1)A}{(\delta(\frac{\beta}{\mu} + 1)A + 1)^2}, \quad R = \frac{\gamma}{\mu} \cdot A. \]  

From (23) we have that either \( A = 0 \) or

\[ pS_0 + S_1 = \delta^{-1}. \]

In the case \( A = 0 \) we obtain the contagion-free equilibrium

\[ E_0 : \quad A = 0, \quad S_0 = 1, \quad S_1 = 0, \quad R = 0. \]

In the case \( A \neq 0 \) we have, substituting (25) into (26),

\[ p \cdot \frac{1}{(\frac{\beta}{\mu} + 1)A + \delta^{-1}} + (1-p) \cdot \frac{\delta(\frac{\gamma}{\mu} + 1)A}{((\frac{\beta}{\mu} + 1)A + \delta^{-1})^2} = 1, \]

which is equivalent to a quadratic equation with solutions

\[ A_{1,2} = \frac{1}{2} \cdot \left(1 + \frac{\gamma}{\mu}\right)^{-1} \left[1 - 2\delta^{-1} \pm \sqrt{1 - 4(1-p)\delta^{-1}}\right]. \]

The expression (28) is identical to the expression (15) for the permanent adoption model, apart from the multiplicative factor \( \left(1 + \frac{\gamma}{\mu}\right)^{-1} \) (indeed when \( \gamma = 0 \) the temporary adoption model degenerates to the permanent adoption model). Therefore the analysis of the conditions for existence of equilibria is the same in both cases, and we obtain:
Proposition 3. (I) If \( p < \frac{1}{2} \) then:

- For \( \delta < 4(1-p) \) there are no endemic equilibria,
- For \( 4(1-p) \leq \delta < \frac{1}{p} \) there are two endemic equilibria \( E_1, E_2 \) (which coincide when \( \delta = 4(1-p) \)).
- For \( \delta \geq \frac{1}{p} \) there is a unique endemic equilibrium \( E_1 \).

(II) If \( p \geq \frac{1}{2} \) then:

- For \( \delta \leq \frac{1}{p} \) there is no endemic equilibrium,
- For \( \delta > \frac{1}{p} \) there is a unique endemic equilibrium \( E_1 \).

However, the conditions for stability of the equilibria, which to a large extent determine the dynamics of the model, are quite different from those for the permanent adoption model, and we turn to these next.

3.1.2 Stability of equilibria

To investigate stability of the equilibria, we examine the linearization of the system around an equilibrium \([44]\). In fact since \( R \) does not appear in the first three equations, and is determined by the other variables by \( R = 1 - S_0 - S_1 - A \), it suffices to consider \([1]-[6]\). Linearization of this system around an equilibrium \((S_0, S_1, A)\) gives the Jacobian matrix

\[
J = \begin{pmatrix}
-\beta A - \mu & 0 & -\beta S_0 \\
(1-p)\beta A & -\beta A - \mu & (1-p)\beta S_0 - \beta S_1 \\
p\beta A & p\beta A & \beta[pS_0 + S_1] - \gamma - \mu
\end{pmatrix}.
\]

Beginning with the contagion-free equilibrium \([27]\), we have

\[
J = \begin{pmatrix}
-\mu & 0 & -\beta \\
0 & -\mu & (1-p)\beta \\
0 & 0 & \beta p - \gamma - \mu
\end{pmatrix}
\]

With eigenvalues \( \lambda_1 = \lambda_2 = -\mu \) and \( \lambda_3 = \beta p - \gamma - \mu \), so that we obtain

**Proposition 4.** The contagion-free equilibrium \( E_0 \) is stable if and only if \( \delta < \frac{1}{p} \).

Moving to the endemic equilibria, we compute the characteristic polynomial of \( J \), and substitute the expressions \([25]\) for \( S_0, S_1 \) at the equilibrium, to obtain

\[
P(\lambda) = \lambda^3 + a_2\lambda^2 + a_1\lambda + a_0,
\]

where

\[
a_0 = \beta\mu \left( (1-\delta^{-1})(\beta A + \mu) + (1-p)\mu \left( \frac{\mu}{\beta A + \mu} - 2 \right) \right),
\]

\[
a_1 = (\beta A + \mu)^2 + (p-\delta^{-1})\beta\mu
\]

(29)
\[ a_2 = 2(\beta A + \mu). \] (32)

By the Routh-Hurwitz stability criterion [44], the equilibrium is stable when:
\[ a_0 > 0, \ a_2 > 0, \ a_1 a_2 > a_0. \]

The condition \( a_2 > 0 \) holds automatically. To check the condition \( a_0 > 0 \), we substitute (28) into (30) and obtain (with a + sign for \( A_1 \) and a − sign for \( A_2 \)):
\[
a_0 = \frac{1}{2} \beta \mu^2 \delta \left( \sqrt{1 - 4(1 - p)\delta^{-1}} \right) \sqrt{1 - 4(1 - p)\delta^{-1}}, \tag{33}
\]
whence
\[ a_0 > 0 \iff \sqrt{1 - 4(1 - p)\delta^{-1}} + (1 - 2\delta^{-1}) > 0. \]

For \( A = A_1 \) (+ sign), the last condition always holds, while for \( A = A_2 \) it is equivalent to
\[
\sqrt{1 - 4(1 - p)\delta^{-1}} > 1 - 2\delta^{-1} \iff \delta > \frac{1}{p},
\]
but under the last condition \( A_1 \) is the unique endemic equilibrium, so we conclude that the condition \( a_0 > 0 \) never holds for \( A = A_2 \).

We have therefore shown that

**Proposition 5.** The equilibrium \( E_2 \), when it exists, is unstable.

By the above we have that \( A = A_1 \) will be stable if and only if \( a_1 a_2 > a_0 \), and we proceed to check when this condition holds. Substituting \( A = A_1 \) as given by (28) into (30)-(32) we calculate
\[
a_1 a_2 - a_0 = \frac{1}{4} \mu^3 \delta^3 \left[ 1 + \sqrt{1 - 4(1 - p)\delta^{-1}} \right]^3 + \beta \mu^2 \delta (p - \delta^{-1}) \left[ 1 + \sqrt{1 - 4(1 - p)\delta^{-1}} \right],
\]
so that \( a_1 a_2 > a_0 \) is equivalent to
\[
\frac{\mu}{\gamma + \mu} \delta^2 \left[ 1 + \sqrt{1 - 4(1 - p)\delta^{-1}} \right]^3 > 2(1 - 2p) \cdot \left( (\delta - 2) + \delta \sqrt{1 - 4(1 - p)\delta^{-1}} \right). \tag{34}
\]

We note that if \( p \geq \frac{1}{2} \) then (34) automatically holds, since the left hand side is positive and the right hand side is non-positive. If \( p < \frac{1}{2} \) then (34) is equivalent to:
\[
\frac{\gamma}{\mu} < F(\delta) \equiv \frac{\delta^2}{2(1 - 2p)} \cdot \left[ 1 + \sqrt{1 - 4(1 - p)\delta^{-1}} \right]^3 + (\delta - 2) + \delta \sqrt{1 - 4(1 - p)\delta^{-1}} - 1. \tag{35}
\]

We therefore conclude that

**Proposition 6.** (i) If \( p \geq \frac{1}{2} \) then the endemic equilibrium \( E_1 \) is stable.

(ii) If \( p < \frac{1}{2} \) then the endemic equilibrium \( E_1 \) is stable if \( \frac{\gamma}{\mu} < F(\delta) \) and unstable if \( \frac{\gamma}{\mu} > F(\delta) \), where \( F(\delta) \) is defined in (35).
Since the function $F(\delta)$ plays an important role, we wish to understand the shape of its graph, assuming $p < \frac{1}{2}$. This function is defined for $\delta > 4(1-p)$, and we have

$$F(4(1-p)) = \left(\frac{1}{1-2p} + 1\right)^2 - 1, \quad \lim_{\delta \to \infty} F(\delta) = +\infty.$$ 

Differentiating $F$, we find that

$$F'(\delta) > 0 \iff \frac{1}{2} (\delta - 2) \sqrt{1 - 4(1-p)\delta^{-1}} > 2 - p - \frac{1-p}{\delta} - \frac{\delta}{2},$$

which, using some elementary algebra, gives

**Proposition 7.** (i) If $p < \frac{1}{4}$ then $F(\delta)$ is monotone increasing for all $\delta \geq 4(1-p)$.

(ii) If $\frac{1}{4} \leq p < \frac{1}{2}$ then $F(\delta)$ is decreasing for $4(1-p) \leq \delta < 1 + \frac{1}{\sqrt{p}}$ and increasing for $\delta > 1 + \frac{1}{\sqrt{p}}$.

### 3.2 The phase diagram: dynamics of the model

We now synthesize our previous result, to obtain a picture of the dependence of the model dynamics on the parameters.

We first note that when $p \geq \frac{1}{2}$, the behavior is rather simple: for $\delta < \frac{1}{p}$ we have only the contagion-free equilibrium $E_0$, which is stable. At $\delta = \frac{1}{p}$ the contagion-free equilibrium loses stability and a stable endemic equilibrium $E_1$ arises in a continuous transition. This behavior is similar to that observed in the standard SIR model with demographic turnover [29, 38].

In the case $p < \frac{1}{2}$ we observe new phenomena. To understand these, we divide the plane of parameters $(\delta, \gamma, \mu)$ into five regions, each of which corresponds to different properties of the equilibria, as determined in section 3.1. These regions are shown in figure 6 for the case $p = 0.15$ and $p = 0.4$ (the interval between the endemicity threshold and the invasion threshold is much narrower in the latter case, so we choose a different scale on the $\delta$-axis). The qualitative difference in appearance between the two cases stems from the fact that the function $F(\delta)$, which defines the boundary $\gamma(\mu) = F(\delta)$ between regions II and V and between region III and IV is monotone increasing for when $p < \frac{1}{2}$ and has a minimum when $p > \frac{1}{2}$ (proposition 7).

We now study the dynamics for each of these regions in turn, using both the analytical results regarding the equilibria and their stability obtained above and numerical simulations.

#### 3.2.1 Region I: No contagion

In region I, defined by the condition

$$\delta < 4(1-p),$$

15
the contagion-free equilibrium $E_0$ is stable, and there exist no endemic equilibria (propositions 3,4). When the parameters are in this region the contagion will not spread.

### 3.2.2 Region II: Bistability

In region II, given by the conditions

$$4(1 - p) < \delta < \frac{1}{p}, \quad \frac{\gamma}{\mu} < F(\delta),$$

the contagion-free equilibrium $E_0$ is stable, but there exist also two endemic equilibria $E_1, E_2$, with $E_1$ stable and $E_2$ unstable (propositions 5,5,6). Thus in this region we have bistability - the contagion may persist or not, depending on the initial conditions. This is demonstrated in figure 7 (left), in which, when the initial fraction of adopters is 3% the contagion dies out, while if the initial fraction of adopters is 4% the contagion persists and approaches an endemic equilibrium.

Let us note that in this region the coefficients of the characteristic polynomial (29) of the linearization at $E_2$ satisfy $a_0 < 0$, $a_2 > 0$. $a_0 < 0$ implies that the product of eigenvalues is positive, so there are either three positive eigenvalues, one positive eigenvalue and two negative eigenvalues, or one positive eigenvalue and two complex conjugate eigenvalues. But $a_2 > 0$ implies that the sum of all eigenvalues is negative, ruling out the possibility of three positive eigenvalues, and implying that if there are two complex conjugate eigenvalues then their real part must be negative. Therefore we conclude that $E_2$ is a saddle with a one-dimensional unstable manifold.
Figure 7: Dynamics for parameter values in region II. Left: Solution $A(t)$ for parameters $p = 0.15, \mu = 0.05, \delta = 3.7, \gamma = 5$. Initial conditions are $A(0) = A_0$, $S(0) = 1 - A_0$, $S_1 = R = 0$, with $A_0 = 0.04, A = 0.03$. Right: Some trajectories in the phase space, for the same parameter values, and initial conditions $A(0) = A_0$, $S(0) = 1 - A_0$, $S_1 = R = 0$ with $A_0 = 0.01, 0.02, 0.03, 0.04, 0.05$. This demonstrates the bistability of contagion-free and the endemic equilibrium $E_1$, when parameters are in region II. The endemic equilibria are shown in blue.

3.2.3 Region III: Endemic equilibrium or bistability of equilibrium and limit cycle

In region III, given by the conditions

$$\delta > \frac{1}{p}, \quad \frac{\gamma}{\mu} < F(\delta),$$

the contagion-free equilibrium $E_0$ is unstable, and there exists a unique endemic equilibrium $E_1$, which is stable (propositions 3.6). Thus in this region contagion will not fade out, and it can persist at the stable endemic equilibrium. The simulations show that this is indeed the case for most parameter values in region III, as illustrated in figure 8. However we also find a small range of parameter values in region III, near its boundary with region IV, for which a stable limit cycle coexists with the stable equilibrium $E_1$. For these parameter values we have bistability of the endemic equilibrium and a limit cycle, so that contagion can persist either at a constant or at periodically varying prevalence, depending on initial conditions. This phenomenon will be explained in section 3.3.3 when we discuss the Hopf bifurcation at the boundary of regions III and IV.

3.2.4 Region IV: Endogenous oscillations

In region IV, given by the conditions

$$\delta > \frac{1}{p}, \quad \frac{\gamma}{\mu} > F(\delta),$$
the contagion-free equilibrium $E_0$ is unstable, and there exists a unique endemic equilibrium $E_1$, but it too is unstable (propositions 3–6). Indeed in this region we have that the coefficients of the characteristic polynomial (29) of the linearization at $E_1$ satisfy: $a_0 > 0$, $a_2 > 0$, $a_1 a_2 < a_0$. The fact that $a_1 a_2 < a_0$ implies that at least one eigenvalue of the linearization has a positive real part. $a_0 > 0$ implies that the product of eigenvalues is negative, so that there is exactly one real negative eigenvalue, and the other eigenvalues are either both positive or complex conjugate with a positive real part. In both cases we conclude that the unstable manifold of $E_1$ is two-dimensional.

In view of the fact that the contagion-free equilibrium is unstable, we know that the contagion cannot fade out. On the other hand, since both equilibria $E_0$, $E_1$ are unstable, the system cannot stabilize at an equilibrium, and we conclude that the contagion must persist in a non-stationary regime. Simulations show that contagion persists as a limit cycle, leading to sustained oscillations, representing repeated epidemic cycles. This is demonstrated in figure 9. In this example $\delta = 8$, the population turnover time is $\mu^{-1} = 20$ years and the mean duration of adoption is $\gamma^{-1} = 1$ year, and periodic oscillations have a period of around 29 years are obtained, with the fraction of adopters varying between less than 0.2% to nearly 25% of the population. The period of oscillations varies widely with the parameters. In figure 10 we display the oscillations where we have reduced the contact parameter to $\delta = 7$ (by reducing $\beta$), keeping the same population turnover time and mean duration of adoption. The period of oscillations is now approximately 128 years.

It should be noted that although the stability analysis of the equilibrium points showed that contagion must persist in a non-stationary state, it does not follow automatically that this must be a periodic one - indeed it is known that a three dimensional system can also exhibit quasi-periodic and chaotic behaviors. However, our simulations for various parameter values in region IV have not revealed any non-stationary dynamics other than a limit cycle. Verifying this
3.2.5 Region V: excitability

In region V, given by the conditions

\[ 4(1 - p) < \delta < \frac{1}{p}, \quad \gamma > \frac{2}{\mu} \]

the contagion-free equilibrium \( E_0 \) is stable, and there exist two endemic equilibria \( E_1, E_2 \), both of which are unstable (propositions 3.5.6). Thus in this region the contagion cannot persist in the form of an endemic equilibrium. A priori one might think that contagion might persist in the form of endogenous oscillations (as is the case for region IV), but in fact the simulations show that this is not the case, and in fact generic trajectories converge to the contagion-free equilibrium as \( t \to \infty \). However, here we observe a different phenomenon (see figure 3.2.5):
trajectories in phase space, starting at points near the contagion-free equilibrium, make a large excursion away from this equilibrium and then back to it. This means that contagion spreads as a large epidemic, and then disappears. This is quite different from the behavior in region I, where contagion disappears without spreading. This phenomenon is known as *excitability*, and it is familiar in the field of neuroscience \[25\]. To understand the underlying reason for it, we need to look at the unstable manifold of the equilibrium $E_2$. Note that, as is the case for region II, since the coefficients of the characteristic polynomial of the linearization at $E_2$ satisfy $a_0 < 0, a_2 > 0$ we have that this unstable manifold is one-dimensional. When we plot this unstable manifold (by numerically solving the system, taking initial values very close to $E_2$), see figure 3.2.5, we observe that its two ends connect $E_2$ to the stable contagion-free equilibrium $E_0$, forming a heteroclinic cycle. This cycle attracts nearby trajectories and is responsible for the excitability phenomenon. As we will see further on, the heteroclinic cycle above can be understood as a ‘residue’ of the limit cycle which exists when parameters are in region IV, arising from it through a homoclinic bifurcation.

### 3.3 Bifurcations

Having characterized the model dynamics in each of the five regions of the parameter plane, it is interesting to understand the transitions that occur when the boundaries from one region to the other are crossed. Each such crossing corresponds to a bifurcation involving one of the equilibrium points. We will examine the six transitions that can occur when the parameters vary along a generic curve in the parameter plane. To illustrate this, we take $p = 0.15$ (the corresponding phase diagram is in figure 5 left), choose three horizontal lines in the $(\delta, \gamma)$-plane, $(\gamma = 4, 10, 20)$ and examine the bifurcations along these lines, as they cross regions. The $A$-values of the equilibria, as well the range of
Figure 12: Heteroclinic loop involving the stable contagion-free equilibrium $E_0$ and the unstable endemic equilibrium $E_2$, for parameters $p = 0.15, \mu = 0.05, \delta = 5, \frac{2}{\mu} = 20$ (equilibrium $E_1$ is also plotted, but not involved).

$A$-values for the limit cycles, are plotted in figures 13,14,16 using the numerical continuation package MATCONT [15]. The investigation to follow will reveal that there are also global bifurcations which occur at interior points of some of the regions, and not only at their boundaries.

3.3.1 $I \to II$ and $I \to III$: fold bifurcations

When the line $\delta = 4(1 - p)$ (the endemicity threshold) is crossed from left to right, we have a fold (also known as limit-point) bifurcation [35] in which the two endemic equilbria $E_1, E_2$ appear. In the case of crossing from region $I$ to region $II$ (that is when the point of crossing satisfies $\frac{2}{\mu} < F(\delta)$, as is the case in figure 13), the equilibrium $E_1$ is stable and $E_2$ is a saddle with a one-dimensional unstable manifold - this is also known as a saddle-node bifurcation. In the case of crossing from $I$ to $III$ (as is the case in figure 14 and 16) both $E_1$ and $E_2$ are saddles, with $E_1$ having a two-dimensional unstable manifold and $E_2$ having a one-dimensional unstable manifold.

3.3.2 $II \to III$: transcritical bifurcation

When we cross the invasion threshold $\delta = \frac{1}{p}$ from region $II$ to region $III$, a transcritical bifurcation occurs whereby the unstable endemic equilibrium $E_1$ merges with the stable contagion-free equilibrium $E_0$ and then disappears (its $A$ component becomes negative), and $E_0$ becomes unstable.
3.3.3 $IV \rightarrow III$: Hopf bifurcation

When we cross from region $III$ to region $IV$, along the curve $\frac{\gamma}{\mu} = F(\delta)$, the unique endemic equilibrium $E_1$ loses stability as two eigenvalues of the linearization around $E_1$ move from the left to right-hand side of the complex plane, leading to the birth of a limit cycle through a Hopf bifurcation \cite{35}. For the case $\frac{\gamma}{\mu} = 20$, this occurs at $\delta = 8.14$, and the bifurcating limit cycle can be observed in figure 14. By taking a closer look at the neighborhood of the bifurcation point (figure 14 right), we find that the bifurcation is of subcritical type: as $\delta$ increases beyond the critical value $\delta = 8.14$, a branch of unstable limit cycles is born out of the equilibrium point (which changes from unstable to stable). At $\delta = 8.184$, this branch of limit cycles folds back and becomes stable, and this generates the limit cycles which characterize the dynamics in region $IV$. This means that for $8.14 < \delta < 8.184$ - parameter values for which we are in region $III$, there exist both a stable endemic equilibrium and a stable limit cycle, and in addition there is an unstable limit cycle - thus we have bistability of periodic and stationary behavior. It also implies that the transition from a stable endemic equilibrium to periodic behavior as $\delta$ decreases, so that we move from region $III$ to region $IV$, occurs in a discontinuous manner - when $E_1$ loses stability the stable limit cycle which characterizes the dynamics is a large one.
Figure 15: Saddle-node homoclinic bifurcation as $\delta$ decreases, for $p = 0.15$, $\gamma = 20$.

### 3.3.4 IV $\rightarrow$ V: Saddle-node homoclinic bifurcation

Referring to figure 14 (left) we see that when $\delta$ is reduced beyond the value $\delta = \frac{1}{p} = \frac{1}{0.15} = 6.666$, so that we move from region IV to region V, the limit cycle disappears, and we would like to understand the type of bifurcation involved. We recall (section 3.2.5) that when parameter values are in region V the dynamics is characterized by excitability, stemming from the a heteroclinic loop connecting the unstable equilibrium $E_2$ to the stable contagion-free equilibrium $E_0$. The transition from the limit cycle to the heteroclinic loop occurs through a saddle-node homoclinic bifurcation [35], see figure 15: as $\delta$ approaches $\frac{1}{0.15}$ from above, part of the limit cycle approaches closer to the contagion-free equilibrium $E_0$, and when $\delta = \frac{1}{0.15}$ the limit cycle touches $E_0$, thus forming a homoclinic (note that this implies that as $\delta$ approaches the critical value, the period of the limit cycle approaches infinity). As soon as $\delta < \frac{1}{0.15}$, the unstable endemic equilibrium $E_2$ bifurcates out of $E_0$, which now becomes stable, and the heteroclinic loop $E_2 \rightarrow E_0 \rightarrow E_2$ is formed (note that the other unstable equilibrium $E_1$ is not involved in this bifurcation). The limit cycle has thus been replaced by a heteroclinic loop, so that oscillations have been replaced by excitability.

### 3.3.5 V $\rightarrow$ II: Hopf bifurcation of an unstable limit cycle

When we move from region V to region II across the boundary defined by the curve $\frac{1}{\bar{p}} = F(\delta)$, the endemic equilibrium $E_1$ becomes stable, as two eigenvalues
of its linearization cross from the right to the left halves of the complex plane. This is accompanied, as expected, by a Hopf bifurcation, in which an unstable limit cycle emerges from $E_1$. This can be seen in Figure 16 where we take $\gamma = 10$ and the transition from region $V$ to region $II$ occurs at $\delta = 4.752$. In this figure we also see that the unstable limit cycle which is born at $\delta = 4.752$ disappears at $\delta = 4.953$. A closer examination reveals a global bifurcation involving also the one-dimensional unstable manifold of $E_2$. For values of $\delta$ below the bifurcation value $\delta = 4.953$ (figure 17 left), this unstable manifold forms a homoclinic loop with $E_0$. At the bifurcation the unstable limit cycle collides with the unstable equilibrium $E_2$, forming a homoclinic orbit, which then vanishes, and the two parts of the unstable manifold of $E_2$ now connect to $E_0$ and to $E_1$ (figure 17 right).

Figure 16: Left: Equilibria and limit cycles in dependence on $\delta$, when $p = 0.15$, $\mu = 10$. Right: A closer look at values of the parameter $\delta$ near the Hopf bifurcation point.

Figure 17: For $p = 0.15$, $\mu = 10$, unstable manifold of $E_2$ before ($\delta = 4.9$, left) and after ($\delta = 5.1$, right) the homoclinic bifurcation.
4 Discussion

We have shown that simple two stage contagion models with demographic turnover generate interesting nonlinear effects, which do not arise in their ‘classical’ one-stage counterparts (SI and SIR models [29, 38]). We now summarize these effects in a non-technical way, so as to highlight the qualitative conclusions that can be drawn regarding conditions under which different types of dynamics are obtained, and consider their significance for the behavior of contagion at the population level. We expect that the broad features described below will be robust, in the sense that they will hold also under various modifications of the model.

(1) When the probability of adoption on first encounter is sufficiently high (\( p \geq 1/2 \)), the behavior of the two-stage contagion models is much like that of a one-stage model: there exists an invasion threshold \( \delta = 1/2 \) such that when contagion is weak (i.e. the contact parameter \( \delta \) satisfies \( \delta \leq 1/p \)) the contagion cannot persist, while for \( \delta \) above this threshold the contagion persists and approaches an endemic equilibrium. Moreover, the transition from non-contagion to contagion is continuous, in the sense that when the contact parameter is slightly above the threshold, the extent of contagion (\( A \)) at equilibrium will be small.

When the probability of adoption on first encounter is sufficiently low (\( p < 1/2 \)), new qualitative features emerge. In this case the dynamical behavior of the model depends on two factors: the contact parameter \( \delta \), and the duration of the adoption period relative to the average residence time of an individual in the population. The different phenomena which occur, depending on these two factors, are summarized below.

(2) When the contact paramater is below the endemicity threshold (\( \delta < 4(1-p) \)), contagion will not spread in the population.

(3) When the contact parameter is above the invasion threshold (\( \delta > 1/p \)), we observe different behaviors, depending on the duration of adoption:

(i) If the mean duration of adoption (\( \gamma^{-1} \)) is sufficiently long relative to the mean residence time of individuals in the population (\( \mu^{-1} \)) – including the extreme case \( \gamma = 0 \) which corresponds to the model with permanent adoption, the contagion becomes established at a constant level (a stable endemic equilibrium), regardless of the initial number of adopters.

(ii) If the mean duration of adoption is sufficiently short (that is \( \gamma \) is sufficiently large), sustained periodic oscillations occur - corresponding to cyclic epidemics of contagion. These oscillations are an emergent phenomenon at the population level, arising from interactions among individuals, each of which displays no cyclic behavior - recall that in this model each individual who abandons the innovation does not re-adopt it. We therefore have a mechanism for the endogenous generation of periodic fads and fashions. We note that a quite different mechanism capable of generating periodic fashions, based on ‘snobs’ and ‘followers’, is modelled in [1, 5]. We note that endogenous oscillations do not occur in the basic models of mathematical epidemiology, and some special mechanisms are required to generate such oscillations, the most prominent being temporary immunity with delay [24, 48]. Our results show that two-stage contagion is an-
other mechanism which induces periodic oscillations, and it appears that it is among the simplest mechanisms producing this effect.

(iii) In a narrow intermediate range of values of the mean duration of adoption, endemic equilibrium and periodic oscillations are both stable, so that contagion will be maintained either at a constant level or in the form of cyclic epidemics, depending on initial conditions.

(4) When the contact parameter is above the endemicity threshold but below the invasion threshold \((4(1-p) < \delta < \frac{1}{p})\), we observe different behaviors, depending on the duration of adoption:

(i) If the mean duration of adoption \((\gamma^{-1})\) is sufficiently long relative to the mean residence time of individuals in the population \((\mu^{-1})\) (including in the limit \(\gamma = 0\), corresponding to permanent adoption), we have bistability of the contagion-free and endemic equilibria, (‘alternative stable states’ [22]), giving rise to a ‘critical mass’ threshold, so that contagion will only ‘catch’ if sufficiently many individuals adopt it at the start. An important implication of this bistability is that eradication of an established contagion will require reducing the contact parameter to a much lower value than the invasion threshold. For example, if \(p = 0.1\), then the invasion threshold is \(\delta = \frac{1}{p} = 10\), but eradicating an existing endemic contagion will require reducing \(\delta\) below \(\delta = 4(1-p) = 3.6\).

Related to this is the discontinuous transition which occurs under a varying contact parameter: when the contagion-free state is de-stabilized as \(\delta\) crosses the invasion threshold from below, a jump from no contagion to a high level of contagion will occur. Similarly, if a contagion is already established and the contact parameter is reduced until it reaches the endemicity threshold, a large contagion will disappear without warning. This type of ‘critical transition’ or ‘regime shift’ phenomenon [22] can provide an explanation for rapid opinion shifts and dramatic behavioral changes which can arise under minor changes in external conditions [34, 45]. Under this explanation, the discontinuous transition is a collective effect arising from the interactions among individuals - the behavior of individuals changes only in a gradual way as the contact parameter varies.

The bistability and hysteresis effects described above do not occur in the basic ‘one stage’ epidemiological models, in which transition from the contagion-free state to endemicity is continuous. However such effects do occur in some more elaborate models, and are known under the term ‘backward bifurcation’. Various epidemiological mechanisms are known to induce backward bifurcations, e.g. exogenous reinfection of latently infected individuals, imperfect vaccination, and risk structure [20]. Our results show that contagion with stages is another mechanism which generates backward bifurcation.

(ii) If the mean duration of adoption is sufficiently short (that is \(\frac{\gamma}{\mu}\) is sufficiently large), then contagion cannot become endemic, but we observe the phenomenon of excitability: starting with a small fraction of initial adopters, a large epidemic develops before the contagion fades. This contrasts with one-stage contagion models, in which, below the invasion threshold, the number of adopters will always decrease, whatever its initial value. Thus the two-stage model can account for large contagion epidemics which do not become endemic, despite the renewal of the susceptible population provided by the demographic turnover.
Developing the mathematical theory of social contagion requires classifying relevant mechanisms at the micro-level, and exploring their dynamical consequences at the population level using mathematical modelling. Simple models, like the one considered here, show that a combination of basic mechanisms (here, two-stage contagion and demographic turnover) can give rise to rich phenomena, suggestive of some of the complexities found in social systems. As always, the fact that a mathematical model can produce a phenomenon which is reminiscent of a real-world one is far from proof that the mechanisms described by the model are those responsible for the real effect, but it does constitute a proof-of-principle that the mechanisms involved are capable of producing the effect [31]. It would be of great interest to attempt a direct validation of a two-stage (or multi-stage) contagion model by fitting it to empirical data.

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