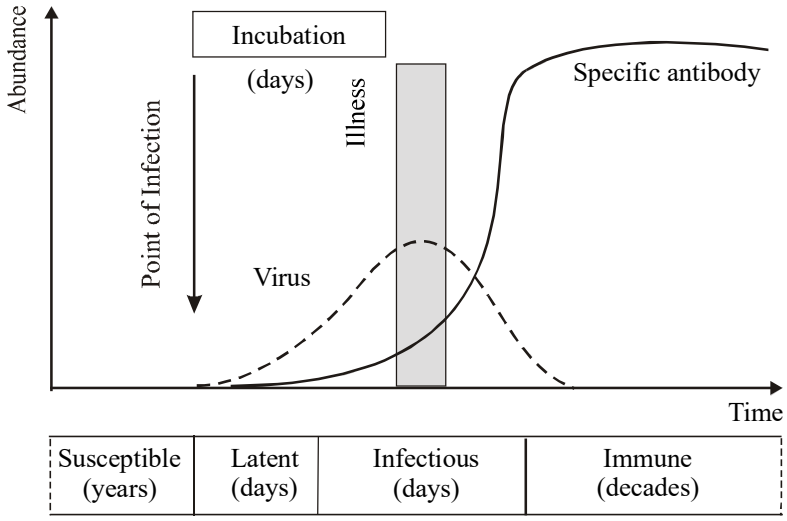
4.3 CONTAGION IN EPIDEMIOLOGY

4.3.1 Clarifying the Notions

The biology of infectious diseases (Anderson and May 1991, p. 13) divides the population which can become host to microparasites into the following classes of individuals: susceptible (uninfected), infected (latent, infectious) and recovered/immune. The latent period is defined as the average period of time from the point of infection to the point when an individual becomes infectious to others, the infectious period denotes the average period over which an infected person is infectious to others, and the period from the point of infection to the appearance of symptoms of disease is termed the incubation period (Anderson and Nokes 1997, p. 692). The duration of symptoms of disease, as depicted in Figure 4.1, is 'not necessarily synchronous with the period during which an infected host is infectious to susceptible individuals' (Anderson and May 1991, p. 14).

Figure 4.1 The development of an infectious disease



Source: Anderson and Nokes (1997, p. 692).

Surprisingly, 'contagion' is not a key term in epidemiology: instead, the equivalent notion is 'transmission'; the so-called 'transmission coefficient' determines the rate at which 'new infections arise as a consequence of mixing between the susceptible and infected individuals' (ibid.). The so-called 'force

of infection' is the *probability that a given susceptible host will become infected* (ibid., p. 63).

Note also that indirect transmission exists; in this case, 'the parasite passes through one or more species of intermediate hosts in order to complete its life cycle' (ibid., p. 22). There is an average stay of the microparasites in the infected class who recover and survive the disease and there is also an average stay of the virus among the class of immune individuals. During recovery, viral abundance decays to zero/very low levels and antibodies specific to viral antigens rise to high levels. 'Recovered hosts are almost invariably fully immune to further infection in the case of viral parasites' (ibid., p. 31). However, the duration of human immunity is not lifelong in the majority of cases. This is why ex-immunes become by definition newly susceptible.

A major issue in the framework of spreading epidemic diseases is the effective reproductive rate of a parasite (F); a parasite must have a basic reproductive rate (F_0), that is, the average number of successful offspring it is capable of producing, of $F_0 > 1$. In a human environment, F_0 is more precisely defined as the 'average number of secondary infections produced when one infected individual is introduced into a host population where everyone is susceptible' (ibid., p. 17). In equilibrium, the effective rate will be just one and, in particular, this is known as the 'endemic equilibrium' (Anderson and Nokes 1996, p. 238):

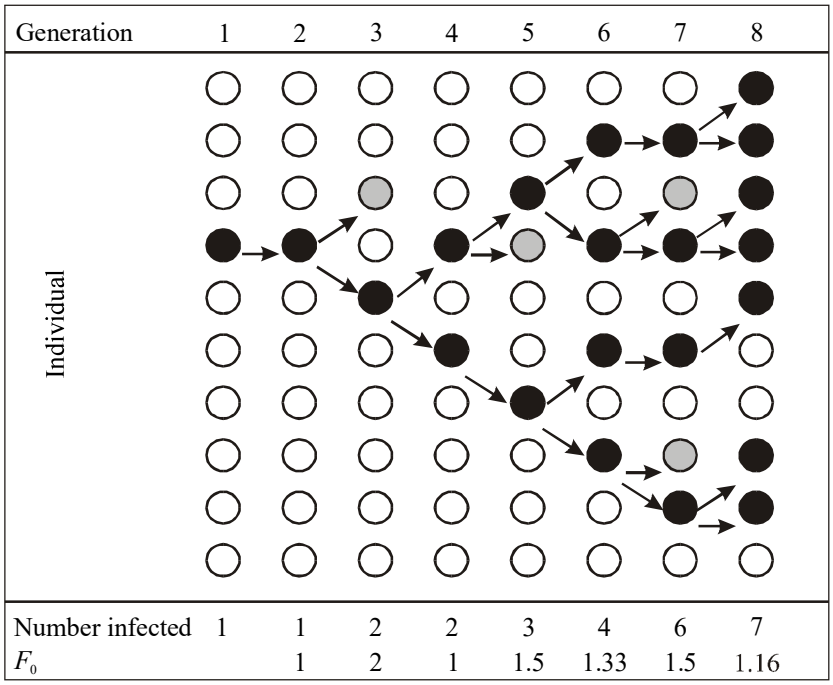
$$F = F_0 s^* = 1 \tag{4.1}$$

where s^* is the fraction of the host population that is susceptible in equilibrium (Anderson and May 1991, p. 17).[8] If the prevalence or incidence of infection is stable through time, the effective reproductive rate F must equal unity in value; this is a situation in which each primary case gives rise, on average, to a single secondary infectious individual (Anderson and Nokes 1997, p. 700). This result is important for the economic modelling of infection and contagion in economics as it points to the possibility of restriction he analysis to a two-country or two-investor perspective.

Let us explore this phenomenon in more detail. On invasion, the vast majority of hosts are susceptible, and hence provided $F_0 > 1$, the epidemic expands as illustrated in Figure 4.2.

> However, as the epidemic progresses, more and more of the contacts made by an infected host are either immune or already infected. As the effective reproductive number declines, and eventually at equilibrium it settles to the value of unity where each infected person generates an average of one secondary infection. (Anderson 1998, p. 33)

Figure 4.2 The expansion process of an epidemic



Source: Anderson (1998, p. 33).

Note that in periods 1 and 2 (4 and 5) the associated transmission from an individual 4 (6) to an individual 4 (7) is of the 'latent domino effect' nature; the same applies to individual 2 in periods 6 and 7. However, 'real domino effects' apply only when we find a chain: this is the case with individuals 6 (period 6 = start), 6, 5 (period 8 = end). 'Pure contagion', that is, situations where for each infected individual $F_0 > 1$, are better represented by all remaining cases of transmission depicted in Figure 4.2! Hence, we may conclude that domino effects are only a partial aspect of contagion! F_0 can be approximated by the following formula:

$$F_0 = \beta ST \tag{4.2}$$

that is the number of susceptibles present with which the primary case can come into contact (S), multiplied by the length of time that the primary case is infectious to others, T, multiplied by the transmission coefficient, β. Hence, we achieve:

$$F = \beta STs^* = 1 \tag{4.3}$$

$$s^* = 1/\beta ST. \tag{4.4}$$

There is a critical level for S such that F_0 is set at one and the broad reproductive rate is lower than necessary for an epidemic expansion.

$$S_T = 1/\beta T. \tag{4.5}$$

In this case, if the density of susceptibles can be reduced to less than S_T in value, an eradication of the virus (by a mass vaccination for instance) becomes feasible (Anderson and Nokes 1997, pp. 700–701).

4.3.2 A Simple Model of Contagious Disease

Consider a stable population of size N which consists of:

$$N = M + S + H + Y + Z \tag{4.6}$$

where M is the number of infants with maternally derived immunity, S is the number of susceptibles, H is the number of infected, but not yet infectious individuals, Y is the number of infectious individuals and Z is the number of immunes. Figure 4.3 shows in a flow chart how these variables are related to one another.

It is assumed that the net birth rate is equal to the natural per capita mortality rate (μ) so that the number of births is μN; δ is the per capita rate of movement out of class M; β is the rate of transmission that defines the probability of contact and infection transfer between a susceptible and an infectious person, σ defines the per capita rate of leaving the latent class; γ is the per capita rate of leaving the infectious class and (not included in Figure 4.3) τ is the per capita disease-induced death rate (Anderson and May 1991, p. 58; Anderson and Nokes 1997, p. 693). With this notation, one may define the following system of differential equations:

$$dM/dt = \mu N - (\delta + \mu)M \tag{4.7}$$

$$dS/dt = \delta M - (\beta Y + \mu)S \tag{4.8}$$

$$dH/dt = \beta SY - (\sigma + \mu)H \tag{4.9}$$

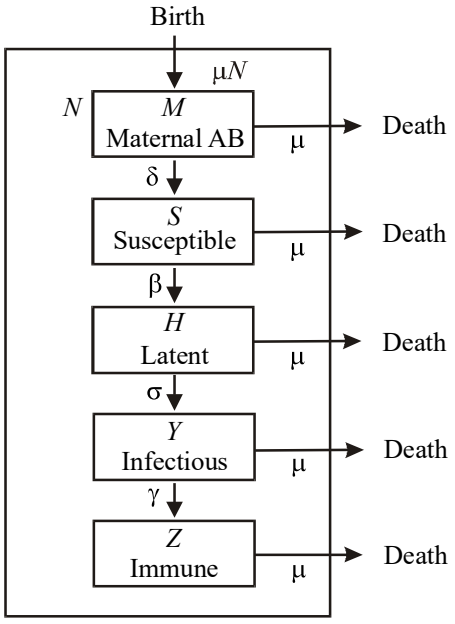
$$dY/dt = \sigma H - (\tau + \gamma + \mu)Y \tag{4.10}$$

$$dZ/dt = \gamma Y - \mu Z. \tag{4.11}$$

The net rate of infection βSY, is approximated by a relationship proportional (β) to the density of susceptibles (S) multiplied by the density of infectious individuals, Y (Anderson and Nokes 1997, p. 694). The equations (4.7) to (4.11) constitute a simple model of infection transmission; the equilibrium properties of this system ('endemic equilibrium') can be examined by setting the time derivatives equal to zero, 'that is such that there are assumed to be no further changes in the number of individuals within each infection class because the flows into and out of any one category are equal' (ibid., p. 695):

$$M^* = \frac{\mu}{\delta + \mu} N \tag{4.12}$$

Figure 4.3 Different classes within a population affected by an infectious disease



Source: Anderson and Nokes (1997, p. 604).

$$S^* = \frac{(\sigma + \mu)(\gamma + \tau + \mu)}{\beta\sigma} \tag{4.13}$$

$$H^* = \frac{(\gamma + \alpha + \mu)}{\sigma} Y^* \tag{4.14}$$

$$Y^* = \frac{(\delta M^* - \mu S^*)}{\beta S^*} \tag{4.15}$$

$$Z^* = \frac{\gamma}{\mu} Y^*. \tag{4.16}$$

The interpretation of the steady-state solutions is straightforward. From these ten equations, at least six, (4.8) and (4.13), (4.10) and (4.15), and (4.11) and (4.16) deserve our special interest, as we have to establish a link to 'contagion' in economics later on. Hence, in the following, we shall focus on a reduced set of equations which are able to show how an epidemic arises, but also when it may die out. If we disregard the infants with derived immunity, equation (4.8) simplifies to:

$$dS/dt = \mu N - (\beta Y + \mu)S. \tag{4.17}$$

If we also neglect τ and the distinction between latent and infectious individuals, we achieve:

$$dY/dt = \beta SY - (\gamma + \mu)Y. \tag{4.18}$$

Finally, the immunes are modelled as before:

$$dZ/dt = \gamma Y - \mu Z. \tag{4.19}$$

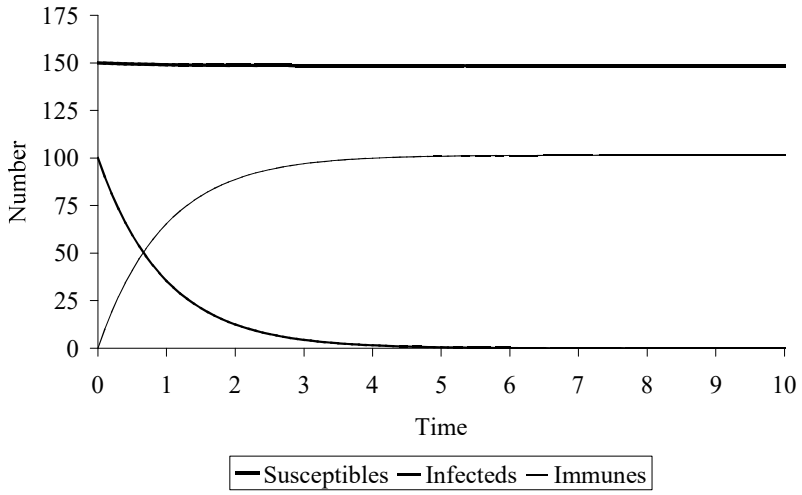
Total population now consists of:

$$N = S + Y + Z. \tag{4.20}$$

With this reduced set of equations we can now easily simulate three scenarios: (i) no epidemic, (ii) transitory epidemic and (iii) persistence (latent epidemic)! Consider in the first case (i) a population which is totally susceptible, but not affected by mortality (then μ is zero in all of the above equations) where we introduce a few infecteds. The epidemic will not occur if the basic reproductive rate is less than one ($F_0 = 0.005$) and the density of

susceptibles does not reach the critical threshold value ($S_T = 10{,}000$). See Figure 4.4 for a simulation of this first case.

Figure 4.4 The no-epidemic case



Notes: At time $t = 1$: $S = 150$, $Y = 100$, $Z = 0$, $\beta = 0.0001$, $\gamma = 1$, $F_0 = 0.015$, $S_T = 10{,}000$.

Sources: Anderson and Nokes (1997, p. 702); own calculations.

The epidemic will, however, occur (ii) if the basic reproductive rate is greater than or equal to one ($F_0 = 5$) and the density of susceptibles exceeds the threshold value ($S_T = 100$). But, even in the outbreak of an epidemic case, 'as time progresses, the density of susceptibles will decline, until the effective reproductive rate F is less than unity (that is, the number of susceptibles falls below the threshold ($S_T = 1/\beta T$)) and the infection dies out' (Anderson and Nokes 1997, p. 701). For a simulation of this second case, see Figure 4.5.

For an epidemic to become persistent (latent epidemic), one of two things must happen. In the first example for (iii), we shall limit our scope to the case where[9] susceptibles are *continually introduced into the population by births* at a net rate of μN, where μ is the per capita birth rate and at the same time the rate of mortality, so that total population is maintained at a constant level. The infection will persist in the population, provided that $F_0 \geq 1$ (Anderson and Nokes 1997, pp. 701–2). Let us calculate the *endemic equilibrium* implied by looking at the steady-state values of equations (4.17), (4.18) and (4.19):

$$\begin{aligned} 0 &= \mu N - (\beta Y^* + \mu) S^* \\ \beta S^* Y^* &= \mu (N - S^*) \\ \frac{S^* Y^*}{S_T} &= \frac{\mu}{\gamma} (N - S^*). \end{aligned} \tag{4.21}$$

In equilibrium (4.18) turns into

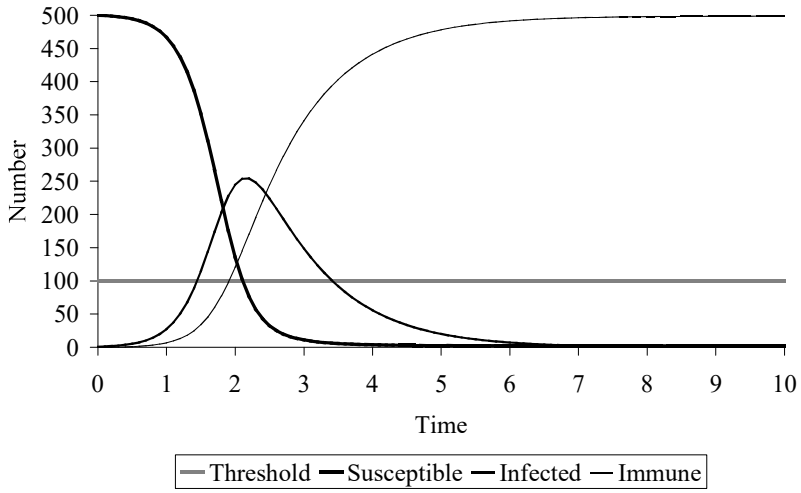
$$\begin{aligned} 0 &= \beta S^* Y^* - (\gamma + \mu) Y^* \\ (\gamma + \mu) Y^* &= \beta S^* Y^* \\ \frac{(\gamma + \mu)}{\gamma} Y^* &= \frac{S^* Y^*}{S_T}. \end{aligned} \tag{4.22}$$

Introducing (4.21) gives:

$$Y^* = \frac{\mu}{(\mu + \gamma)} (N - S^*). \tag{4.23}$$

The steady-state solution for the immunes is straightforward:

Figure 4.5 The transitory epidemic case



Notes: At time $t = 1$: $S = 500$, $Y = 1$, $Z = 0$, $\beta = 0.01$, $\gamma = 1$, $F_0 = 5$, $S_T = 100$.

Sources: Anderson and Nokes (1997, p. 702); own calculations.

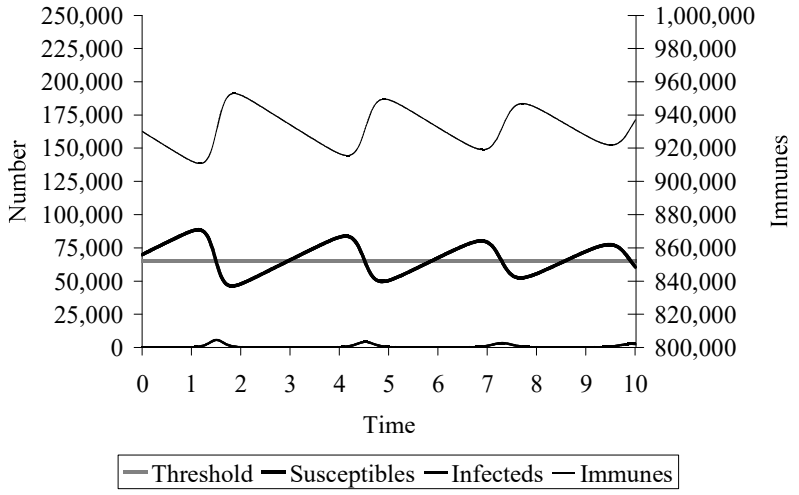
$$Z^* = \frac{\gamma}{\mu} Y^* . \tag{4.24}$$

As in equilibrium $S^* = S_T$, (4.23) gives:

$$Y^* = \frac{\mu}{(\mu + \gamma)} (N - S_T) . \tag{4.25}$$

The stationary solutions, S^*, Y^*, Z^* define the set of variables in the endemic equilibrium. A simulation of this first example for an infection which persists in a community (iii) is provided by Figure 4.6.

Figure 4.6 Persistence of an infection (renewal due to births)



Notes: The conditions for the persistence of an infection in a community when renewal of susceptibles is due to births are at time $t = 1$: $S = 70{,}000$; $Y = 1$; $Z = 930{,}000$; $\beta = 0.0004$; $\gamma = 26$; $\mu = 0.02$; $\varepsilon = 0$; $F_0 = \beta N/(\mu+\gamma) \approx 15.37$; $S_T = 65{,}050$.

Sources: Anderson and Nokes (1997, p. 702); own calculations.

The second example for (iii) abstracts from new births and from mortality ($\mu = 0$), but rather concentrates on the possibility that immunity is of short duration with $1/\varepsilon$ as the average duration of immunity and hence ε as the rate at which immunes 'regain' susceptibility. The respective equations then change into:

$$dS/dt = -\beta YS + \varepsilon Z \tag{4.26}$$

$$dY/dt = \beta SY - \gamma Y \tag{4.27}$$

$$dZ/dt = \gamma Y - \varepsilon Z. \tag{4.28}$$

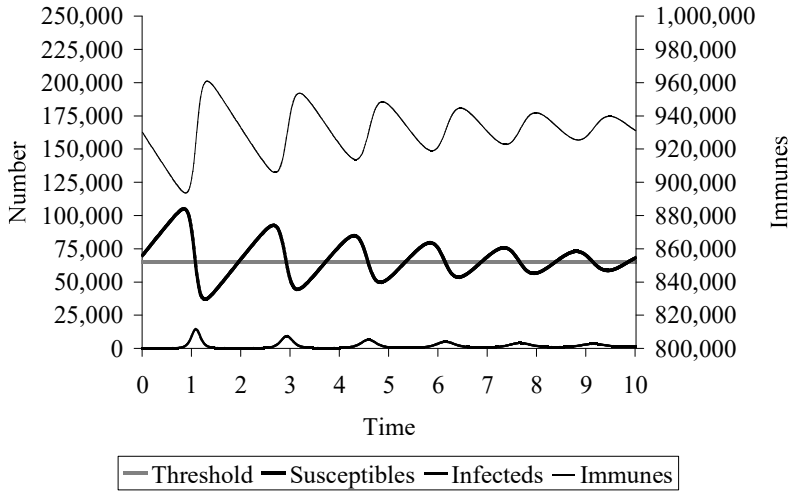
The endemic equilibrium values now are:

$$Y^* = \frac{\varepsilon}{(\varepsilon + \gamma)}(N - S_T) \tag{4.29}$$

$$Z^* = \frac{\gamma}{\varepsilon} Y^*. \tag{4.30}$$

A simulation of this second case of (iii) is found in Figure 4.7.

Figure 4.7 Persistence of an infection (renewal due to waning immunity)



Notes: The conditions for the persistence of an infection in a community when renewal of susceptibles is due to waning immunity are at time $t = 1$: $S = 70{,}000$; $Y = 1$; $Z = 930{,}000$; $\beta = 0.0004$; $\gamma = 26$; $\mu = 0.05$; $\varepsilon = 0.05$; $F_0 = \beta N/(\mu+\gamma) \approx 15.37$; $S_T = 65{,}050$.

Sources: Anderson and Nokes (1997, p. 702); own calculations.

4.3.3 Illness and Recovery in a Two-Agent-(Dis)Equilibrium Setting

A better understanding of contagion is provided when we concentrate on the *differences* with regard to a composite *m* of main medical indicators –

temperature, blood pressure, pulse frequency and blood composition (density of red blood cells, number of infected cells, percentage of infected cells and so on, see Anderson 1994, p. 474) of, say, two hypothetical patients *i*, *j*. When two individuals of comparable characteristics (age, size and so on) are both healthy, key medical indicators for the functioning of their organisms tend to show little difference (*first equilibrium*):

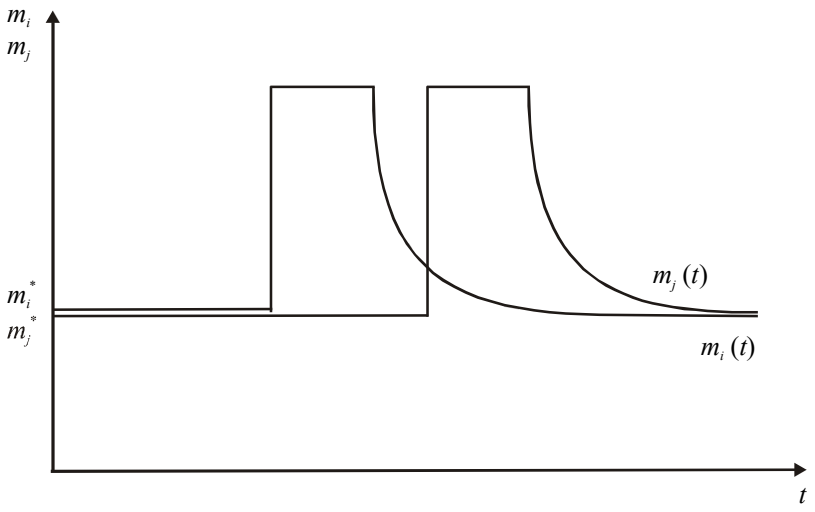
$$m_i^* \cong m_j^* . \tag{4.31}$$

If one of the two individuals suffers an infection, the difference in these indicators will shoot up quickly:

$$m_i(t) \neq m_j(t) . \tag{4.32}$$

Contagion will have been effective if we observe a strong tendency of those indicators to pursue a similar though lagged pattern in the following, as depicted in Figure 4.8. After recovery of both individuals has begun, the indicators tend to normalize. In the hopefully new, *second equilibrium* the indicators of both individuals will resemble each other as they did at the time of the *first equilibrium*. Note that 'illness' is treated as a temporary all-time-high scenario for the respective indicators $m_i(t)$, $m_j(t)$.

Figure 4.8 Illness and recovery in a two-patient setting

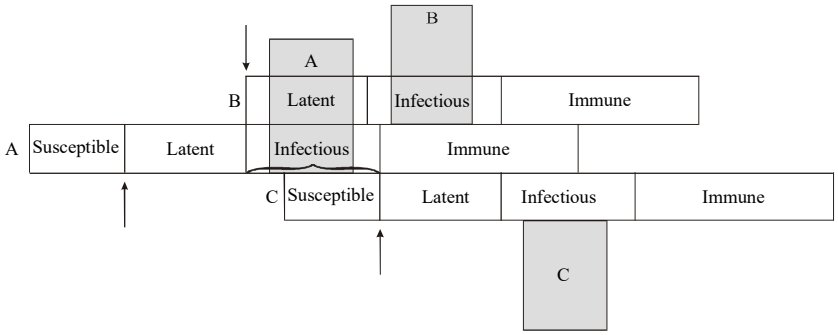


Source: Own compilation.

4.3.4 How to Deal with the Identification Problem?

With the help of the sequence depicted in Figure 4.9, which is nothing but a three-individual overlapping time profile of an infection, we may try to avoid the so-called 'identification problem' in epidemiology: 'when differential vulnerability to an unobserved common shock[10] reflects unobserved characteristics, we may get what looks like true contagion, since a crisis in one country will be followed by a crisis in another, with no apparent explanation than the original crisis itself' (Drazen 1999, p. 5).

Figure 4.9 The overlapping time profile of an infection (three individuals)



Source: Own compilation.

Individual A can – as the example was designed – at most transmit the disease to B and C; however, we cannot be sure a priori if individual C was infected by A or by B! When time-series data were available, we would have to apply Granger causality tests. Notice that when B (A) Granger causes the infection of C (only B), we have 'latent domino effects' (see above).

A problem with 'contagion' in economics, however, is that we have to identify the time durations within each of the identified classes in Figure 4.1 and 4.9! If we think about contagion among investors in the sense of herding (see above), for instance, it is most likely that we have to deal with much shorter time intervals.

4.3.5 What about Exogenous Immunization and other Means of Prevention?

So far so good; doctors of medicine *and* economists would be delighted if they could rely on the endogenous process of immunization described above. However, in many cases, severe infections require means of prevention and

exogenous immunization. Note that exogenous immunization (either active or passive) is one of the possible prevention instruments. Other means of prevention against infection from microparasites among individuals are hygiene, quality controls of water and air, the removal and recycling of dust, and food hygiene.

A synonymous term for exogenous immunization is vaccination; to eradicate an infection by mass vaccination it is indispensable that the proportion of the population successfully immunised, v, exceeds a critical value v_C, where:

$$v_C = 1 - 1/S_0 \tag{4.33}$$

so that too few susceptibles remain to perpetuate transmission, that is:

$$S < S_T. \tag{4.34}$$

Therefore, the larger the value F_0, the higher the coverage (v_C) needed to eliminate infection, *ceteris paribus*!

4.4 A REINTERPRETATION OF 'CONTAGION' AS A TERM IN ECONOMICS

4.4.1 The Insights Gained from Epidemiology

A major lesson that contagion literature in economics can learn from epidemiology is first, to differentiate painstakingly between infections without epidemic (i), transitory epidemic (ii), and epidemic oscillations around an endemic equilibrium (iii). Second, it should be clear that the first case is not of much relevance for economics, whereas the second and the third are of considerable relevance. Third, it seems to me, however, that there is to the best of my knowledge not a single contribution in the contagion literature which distinguishes – as there should be – between cases (ii) and (iii)!

4.4.2 Towards a Useful Definition

First of all: what is (are) the disease(s) we are talking about? We are interested in economic crises triggered by financial market and exchange rate crises, the latter defined 'broadly to include not only devaluations but also successful defence of a peg that involves substantial increases in interest rates and losses of reserves' (Masson 1998, p. 4).