29 Geiser, A.G. *et al.* (1993) *Proc. Natl. Acad. Sci. U. S. A.* 90, 9944–9948
30 Vodovotz, Y. *et al.* (1996) *J. Exp. Med.* 183, 2337–2342
31 Ho, A.S. *et al.* (1993) *Proc. Natl. Acad. Sci. U. S. A.* 90, 11267–11271
32 Rees, R.J.W. and D' Arcy Hart, P. (1960) *Br. J. Exp. Pathol.* 42, 83–88
33 Murray, P.J. *et al.* (1997) *J. Immunol.* 158, 315–324
34 Kuhn, R. *et al.* (1993) *Cell* 75, 263–274
35 North, R.J. (1998) *Clin. Exp. Immunol.* 113, 55–58
36 Toossi, Z. and Ellner, J.J. (1998) *Clin. Immunol. Immunopathol.* 87, 107–114
37 Toossi, Z. *et al.* (1995) *J. Immunol.* 154, 465–473
38 Hernandez-Pando, R. *et al.* (1997) *Immunology* 90, 607–617

Perspective: human contact patterns and the spread of airborne infectious diseases

Jacco Wallinga, W. John Edmunds and Mirjam Kretzschmar

Networks of social contacts channel the transmission of airborne infections. Emerging insights from fields of science as diverse as mathematics, population biology and the social sciences are beginning to reveal how the contact pattern of the hosts determines the spread and evolution of airborne infectious agents.

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The spread of infectious agents, such as tuberculosis bacilli, measles viruses and influenza viruses, through human populations is of immediate concern for public health. One of the most obvious features of these infectious agents is their transmission from person to person. The contact patterns of the host population determine who is at a high risk of contracting an infection. Contact patterns therefore provide the information necessary to focus preventative measures at specific groups in a population and can help to determine the efficacy of prevention programmes. In addition, they influence the properties of the pathogens themselves by shaping the environment in which they evolve. For a complete understanding of the distribution and abundance of a pathogen in its host population, one should take account of the effects of the contact patterns of the host population. In this review, we will discuss the new insights and perspectives on contact patterns and their consequences for the design of public health policy.

Modelling the spread of infection

Until about two decades ago, essentially all the mathematical modelling of the spread of airborne infections was based on the assumption that the host population was homogeneously mixed. In such a population, each individual has an equal chance of contacting anyone else in the population, and the probability of contacting any person in the population is independent of whether or not that person has been contacted before. Despite the simplicity of the notion of homogeneous mixing, the predictions based on this assumption often resemble the observed epidemiological patterns in a situation where an infection is widespread (e.g. measles in the pre-vaccination era). In low-prevalence situations, however, the aim of modelling has shifted towards gaining a better understanding of local outbreaks of infection in smaller communities and of rarer pathogens, including new pathogenic strains. In these cases, a more sophisticated description of contact patterns is required.

Contact patterns and their consequences have been a major focus of research on sexually transmitted diseases[1–4]. The exact nature of the relationship between contact patterns and the spread of airborne infections, however, has only recently begun to be investigated, as a result of simultaneous advances in several fields. For example, quantitative studies in social-networks research have recently begun to characterize relevant network structures, and mathematical modelling is shedding new light on how specific aspects of contact patterns can alter the spread of infectious diseases and the evolution of the infective agents[5]. In addition, advances in molecular epidemiology have

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provided tools for tracing the spread of different strains of infectious agents at the level of individual contacts.

Age-specific contacts

Many diseases, such as measles and rubella, primarily affect children. For childhood diseases, age is obviously an important variable that indicates the risk of acquiring and transmitting the disease[6–8]. Modelling studies of these diseases that aim to assess optimum vaccination coverage and age of vaccination require an accurate estimate of contact rates within, and between, age categories. Usually, the age-specific contact rates are indirectly derived from age-specific force-of-infection estimates[8]. Although widely used, this indirect technique is rather unsatisfactory because it is based on assumptions about age-specific contact rates that are difficult to verify (see Ref. 9).

Contact rates can be estimated directly if the relevant 'at-risk' events for an infection can be defined and quantified. One such at-risk event might be a conversation because, if two people are close enough to converse with each other, they are probably close enough to pass on at least some infections. This assumption has been used to measure directly the contact patterns that could lead to the spread of airborne infections[9]. Participants in the study (mainly university staff and students) were asked to record the people they had conversations with in diaries. The results showed that contacts sufficient for transmission of infection are highly structured according to age (Fig. 1a). The mean age of contacts increased with the age of participants, with the older participants having more contact with older adults, a larger variability in the age of their contacts and a tendency to mix with older children than the younger participants. Other sociological studies have also shown that contact patterns tend to be assortative (like-with-like) with respect to age[10]. However, in these studies, the definition of contacts usually includes telephone conversations and letters, which have little bearing on the spread of infectious diseases.

In addition to these sociological techniques, molecular epidemiology provides the opportunity to study the contact patterns of infection in great detail (Box 1). In one three-year study in The Netherlands[11], DNA fingerprints of *Mycobacterium tuberculosis* isolates were collected from patients. From 4241 isolates, 81 clusters of two people were identified that had isolates with identical fingerprints. Within each cluster of two, there was a strong positive correlation with age (Fig. 1b), suggesting that sources

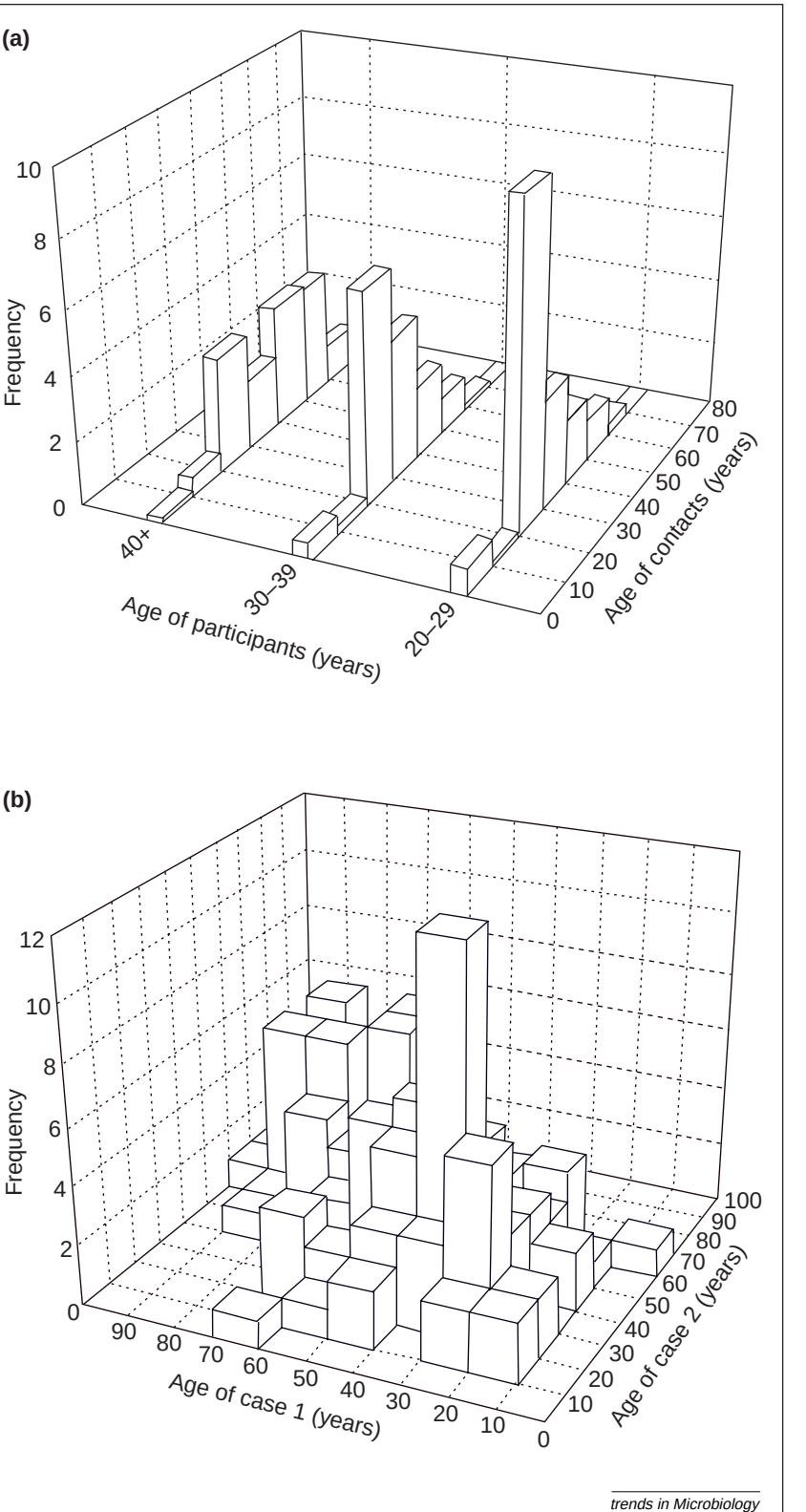

Fig. 1. (a) Age-specific contact frequencies, as measured directly. Frequency of contact was measured as the number of two-way conversations per participant per day. Notice that the participants are grouped in broader age classes than their contacts (modified from Ref. 9). **(b)** The age-specific transmission of tuberculosis, as observed in clusters of two patients with identical *Mycobacterium tuberculosis* DNA fingerprints. The frequency of transmission was measured as the number of people involved in clusters recorded in The Netherlands between January 1993 and December 1996 (modified from Ref. 11).

Box 1. Estimating the basic reproduction ratio

The techniques for direct estimation of contact rates can also be used to estimate the basic reproduction ratio of a pathogen, R_0, which is defined as the average number of secondary cases that a single case causes in an entirely susceptible population[a]. If $R_0 > 1$, an infection can spread once introduced. It has been suggested that the value of the threshold parameter for airborne diseases could be very high[b]; an airborne infectious disease with an infectious period of only one day that can be transmitted during a conversation could have an R_0 of up to 17. Using DNA fingerprinting, it is possible to estimate that the average number of secondary cases of infectious tuberculosis caused directly or indirectly through recent transmission by a single source case is 0.26 (Ref. c). As this estimate does not account for endogenous reactivation and relapses, it should be considered a lower bound to the value of this threshold parameter for tuberculosis.

Knowledge of R_0 is extremely valuable for planning prevention strategies as it gives some indication of the effort required to reach a certain prevention goal. For example, if vaccination is intended to eliminate a pathogen from a population, one can estimate the vaccination coverage needed to reach that aim. If elimination has already been achieved and the infection occurs only in localized outbreaks, the aim of vaccination is often to minimize the size of those outbreaks. The value of R_0 can provide information about the expected size of an outbreak. It is then necessary to take into account more detail of local contact patterns in order to calculate R_0.

References
a Diekmann, O., Heesterbeek, J.A.P. and Metz, J.A.J. (1990) *J. Math. Biol.* 28, 365–382
b Edmunds, W.J., O'Callaghan, C.J. and Nokes, D.J. (1997) *Proc. R. Soc. London B Biol. Sci.* 264, 949–957
c Borgdorff, M.W. *et al.* (1998) *Am. J. Epidemiol.* 147, 187–195

Box 2. Fur trappers and 'Spanish 'flu'

Unravelling the epidemiological processes that lie behind the spread of infection is possible where we have detailed information on the precise movements of an infected index case. This information is only available in rare situations. One example is the 'Spanish 'flu' pandemic that hit communities of fur trappers in the central Canadian subarctic in 1918 and 1919. Church records show the dramatic consequences for these communities: at Norway House in central Manitoba, 107 people died out of a population of ~750 and 23% of all casualties were from seven out of 237 families. The majority of the families (167 out of 237) were uninfected but all, or nearly all, of the members of the seven most severely affected families died.

Sattenspiel and Herring[a] found that much of the differential impact of the epidemic among families derived from the unusual community structure of the Canadian fur trappers. The trappers lived in winter hunting groups, consisting of at least two married couples and their children, at fur-trapping locations along the major rivers of the region. When enough furs were available, or when they needed more supplies, one or two men from the group would travel to the trading post at Norway House.

Ball *et al.*[b] showed that, in a population with such a contact pattern, with low contact rates between groups and frequent contact within groups, it is likely that an infectious disease will generate markedly heterogeneous incidence patterns. Thus, the settlement structure and mobility patterns of the Canadian fur trappers, with groups widely scattered across the landscape and low rates of mobility connecting the groups, could have been an important cause of the marked heterogeneity in mortality.

References
a Sattenspiel, L. and Herring, D.A. (1998) *Hum. Biol.* 70, 91–115
b Ball, F., Mollison, D. and Scalia-Tomba, G. (1997) *Ann. Appl. Prob.* 7, 46–89

of tuberculosis tend to transmit infection to people close to their own age.

Global versus local contacts

Another important form of heterogeneity in human contact patterns is the fact that most of our contacts tend to be in a limited social group. An extreme example of such a population structure is provided by fur trappers in the Canadian subarctic during the early part of this century. They lived along trapping outposts in groups of one or two families, with group members rarely making contact with others outside their group (Ref. 12; Box 2). Other societies are likely to be less rigidly structured, although important social groupings, such as households, school classes or cities, remain. The key idea here is that the population consists of groups that have frequent contacts within groups (local contacts) and relatively rare contacts between groups (global contacts). Although this idea is not new, going back more than 40 years[13], the exact role of global and local contacts in determining epidemic patterns has only recently become an area of active research.

An obvious way of investigating the role of global and local contacts is to separate the population into smaller subpopulations, each with its own independent transmission dynamics but with some transmission between them[14]; this is termed a metapopulation or patch model[15,16]. Computer simulations of such models show that the subpopulation dynamics can become desynchronized, such that when infection has faded out in some patches it may still be present in others, allowing reintroduction into the infection-free patches. The relatively few global contacts that transmit the infection between subpopulations can prevent global fade-outs, making it more difficult to eliminate the disease.

If a few global contacts are critical to the persistence of infections what is the role, if any, of local contacts? Several authors[17–19] have developed mathematical frameworks to analyse the transmission of infectious disease in a population where mixing takes place on the local level within small groups like households, and on the global level between random individuals in the population. Ball *et al.*[17] have shown that transmission within local groups amplifies the total epidemic compared with homogeneous mixing with global contacts only. This has consequences for the design of vaccination strategies: when the population consists of different groups and each individual makes local and global contacts, the aim of vaccination should be to reduce the mean size of local outbreaks. Neglecting this social structure in the design of vaccination strategies might actually lead to underestimating the coverage needed to eliminate the infection from the population[17–19].

Contact networks

Instead of viewing a population as a community of social groups, it can be viewed more generally as a network in which individuals are the nodes and their contacts are the links. In one of the few studies of

sociological network data in an epidemiological context (other than that of sexually transmitted diseases) Rapoport and Yuan[20] predicted the spread of an infectious disease through a population of pupils in a junior high school in the USA. They assumed that infected pupils can only spread a disease to their best and second-best friends, which might approximate the sort of contact required to spread meningococcal meningitis. They found that, compared with a network model where individuals make the same number of contacts at random, the epidemic spreads slower in the long-term and reaches fewer pupils. This result shows how social ties can act as a constraint on the spread of infection.

As it is very difficult, if not impossible, to map entire contact networks, attention has been focused on the role of the parameters that characterize networks. In the social sciences, there is a relatively long history of characterizing social networks in human populations[21]. Three network characteristics have emerged from this work that may have an important bearing on the spread of infectious diseases – the average number of contacts, the network's transitivity and the characteristic path length.

Average number of contacts

The average number of people contacted per person indicates how many secondary cases might potentially acquire the infection from one index case. It has been observed that the average number of daily contacts is 17 (based on a group from an English university[9]). In the study by de Sola Pool and Kochen[10], the participants had an average of 23 daily contacts (including telephone conversations and letters) although the average total over 100 days was only 360. A limited number of people were contacted regularly and the remainder were contacted very infrequently[10]. This pattern has been confirmed and it has been shown that the frequency with which people are contacted depends not only on the period over which the contacts are recorded but also on the degree of intimacy of contacts (W.J. Edmunds, unpublished).

Transitivity

Transitivity is the average proportion of secondary contacts that also contact each other (i.e. how many of your friends are also friends of each other[21]) and is a measure of how clustered a network is. Clearly, the degree of clustering has important implications for constraining the spread of infections through a network of contacts[22]. Measuring transitivity directly is difficult but it was estimated by de Sola Pool and Kochen[10] that any person contacted by one of their study participants knew between 8% and 36% of the other people contacted by that participant. Again, the exact value will depend on the period over which contacts are recorded and the degree of intimacy of the contact.

Characteristic path length

The characteristic path length of a network is defined as the average number of contacts in the shortest route between two individuals in the network, with the average taken over all possible pairs of individuals[23]. The number of individuals contacted and the transitivity describe the local structure of the contact network, whereas the characteristic path length describes the global network structure. Using computer simulations, Watts and Strogatz[23] have shown that the spread of infection through a contact network is determined, to a large extent, by the network's global properties, particularly the characteristic path length. To our knowledge, no estimates of the value of this parameter exist for contact networks in relation to the spread of airborne infections. A few global contacts could determine the overall dynamics of an infection spreading through a network and greatly influence the characteristic path length without affecting the transitivity[23].

Contact networks and the evolution of pathogens

In the preceding sections, we have discussed how the structure of a host contact network affects the spread of an infectious agent. As the reproductive success of a pathogen depends on its growth within hosts and its ability to be transmitted between hosts, it is not unrealistic to think that different contact structures might select for different pathogenic traits. A good example of how a highly virulent strain could be selected for by a specific contact pattern is the 1918 'Spanish' influenza epidemic (Box 2). This epidemic could have originated in the trenches in World War 1 (Ref. 24). Infectious soldiers who fell ill in the trenches were removed and replaced, thus increasing the number of contacts between an infected individual and susceptible individuals. Ewald[25] hypothesized that this specific contact pattern selected for highly virulent influenza strains. To what extent can we expect contact patterns to affect not only the epidemiology but also the evolution of infectious agents?

Of particular interest here is the evolution of virulence (the capacity of a pathogen to damage a host by causing morbidity or increasing host mortality), because it involves interplay between the within-host dynamics of a pathogen and the between-host transmission of strains, where the latter depends strongly on the contact patterns in the host population[26]. Although within-host competition selects for higher virulence, the host–pathogen dynamics will evolve to form a compromise between pathogen reproduction and host survival[27,28].

If the population mixes homogeneously, there is no obvious relationship between the number of contacts of an infectious host and the evolution of virulence[29]. However, if the host population does not mix homogeneously, pathogenic strains with different virulence levels may coexist, even if there is complete cross-immunity between strains. For a host population with a highly spatially structured contact network, computer simulations have shown that less-virulent pathogenic strains can invade a host population already exploited by a more-virulent strain[30]. Using a more analytical approach, Van Baalen[29] studied a host population in which each host contacted a limited

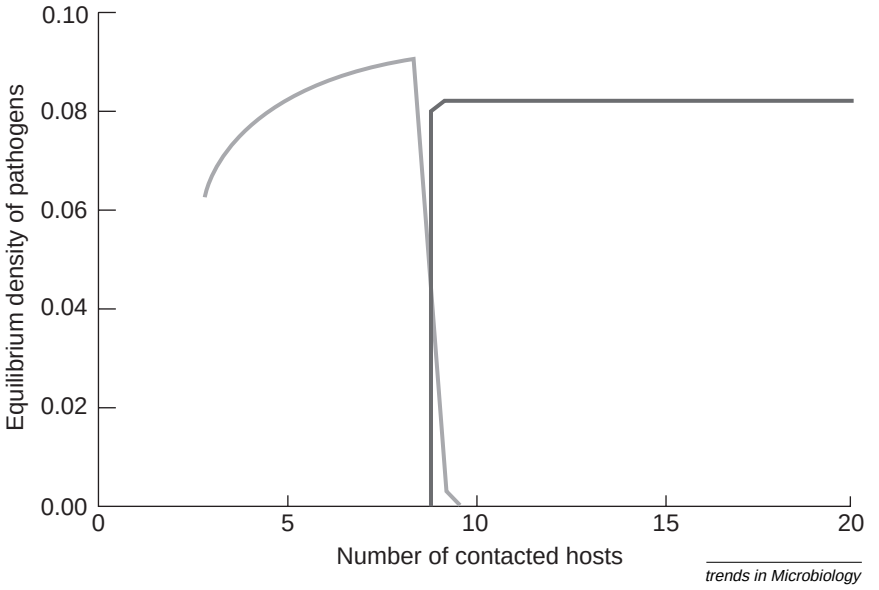

Fig. 2. The theoretical effect of gregariousness of the host on the equilibrium abundance of virulent pathogens (dark grey line) or avirulent pathogens (light grey line). Each host has contact with a limited number of different other hosts shown on the *x*-axis. It is assumed that the infectious period of a host is longer for an avirulent pathogen than for a virulent pathogen. If each host contacts only a small number of other hosts, avirulent pathogens are abundant; if each host contacts a large number of other hosts, virulent pathogens are abundant. Whether virulent or avirulent pathogens are favoured by natural selection depends on the contact pattern of the host (modified from Ref. 29).

number of other hosts. It was found that when each host contacts many other hosts, the virulent strain is more abundant; when each host contacts only a few neighbouring hosts, the less-virulent strain is more abundant (Fig. 2). In this case, a more-gregarious contact pattern selects for a more-virulent pathogen.

Using a model for a spatially structured population, Rand *et al.*[31] studied the evolution of virulence and transmissibility of pathogens. They found that the selective pressure on pathogens is reduced greatly in a population with a clear spatial contact pattern compared with a homogeneously mixing population. Evolution is slower and there is more resistance to invasion by mutant pathogens. Also, there is an upper limit for transmissibility above which the pathogen cannot maintain itself in the population. This is a consequence of the depletion of susceptible individuals in the neighbourhood of an infected host. If susceptible individuals do not move into the local neighbourhood quickly enough (i.e. if the contact pattern is locally very stable) the infectious host cannot produce any more secondary infections.

These results underline the long-term impact that changes in host contact patterns can have on the properties of its pathogens. They show how the threat of (re)emerging infections is connected with increasing mobility and decreasing rigidity of spatial structure in human contact patterns. They also bring a fresh perspective to public health policy by providing an insight into how to use host contact patterns in the design of prevention programmes. If public health measures were to influence human contact patterns in the right way, they could lead to the evolution of less-virulent or less-transmissible pathogens[25].

Questions for future research

- For any particular infectious disease, what types of contact allow the transmission of infection (e.g. sharing a room, speaking, coughing or sneezing) and what is the structure of the network for that type of contact?
- How complete does a sample of a given contact network need to be in order to reconstruct the spread of disease through this network by, for example, using DNA fingerprints of the infectious agent?
- Is it possible for airborne infectious diseases to identify a core group of spreaders, as with sexually transmitted diseases? If so, can this disease be controlled by treating only a core group of spreaders, or should everyone be treated?
- When do preventative measures favour less-virulent strains over more-virulent strains?

Conclusions

Contact patterns are an important part of the environment of infectious agents. They channel the spread of infectious diseases through the host population and shape the evolutionary response of pathogen populations. The assumption of homogeneous mixing is often sufficient to obtain general insights. However, a better knowledge of contact patterns is necessary for a more accurate estimate of the effect of vaccination on future disease incidence, particularly in the era of high vaccine coverage when infections are often confined to isolated outbreaks. More detailed knowledge of human contact patterns could lead to studies that shed light on the long-term evolutionary consequences of public health policies.

The exact structure of the contact patterns in the general population is, to a large extent, still unknown. Recent observations of structural network characteristics[9,11] suggest which modelling assumptions and parameter values best describe the spread of infectious disease through a population. Recent theoretical insights into contact patterns[17,23] point to those structural characteristics that should be observed: the number of contacts per person, the transitivity and the characteristic path length. A combination of theoretical and empirical research is needed to increase our understanding of the impact of human contact networks on the spread and evolution of infectious agents, and to assess the implications of this for the planning of public health policy. This is especially important as global human contact patterns have changed radically in the past few decades. The long-term effects of the social changes on the abundance and evolution of airborne infectious diseases are only beginning to be investigated.

Acknowledgements
We thank Lisa Sattenspiel for useful comments. The idea for this review came from the workshop 'Modelling of Contact Structure and the Spread of Infectious Diseases', supported by the NWO via the Dutch Priority Programme 'Non-Linear Systems in Population Dynamics'. The workshop took place in Bilthoven, The Netherlands, 24–25 September 1998.

References

1 Anderson, R.M., Gupta, S. and Ng, W. (1990) *J. AIDS* 3, 417–429
2 Kretzschmar, M. and Morris, M. (1996) *Math. Biosci.* 133, 165–195
3 Ghani, A.C., Swinton, J. and Garnett, G.P. (1997) *Sex. Transm. Dis.* 24, 45–56
4 Morris, M. (1997) *AIDS* 11 (Suppl. A), S209–S216
5 Levin, S.A. *et al.* (1997) *Science* 275, 334–343
6 Schenzle, D. (1984) *IMA J. Math. Appl. Med. Biol.* 1, 169–191
7 Anderson, R.M. and May, R.M. (1991) *Infectious Diseases of Humans: Dynamics and Control*, Oxford University Press
8 Anderson, R.M. and May, R.M. (1985) *J. Hyg.* 94, 365–436
9 Edmunds, W.J., O'Callaghan, C.J. and Nokes, D.J. (1997) *Proc. R. Soc. London B Biol. Sci.* 264, 949–957
10 de Sola Pool, I. and Kochen, M. (1978) *Soc. Networks* 1, 5–51
11 Borgdorff, M.W. *et al.* (1999) *Int. J. Tuberc. Lung Dis.* 3, 202–206
12 Sattenspiel, L. and Herring, D.A. (1998) *Hum. Biol.* 70, 91–115
13 Rushton, S. and Mautner, A.J. (1955) *Biometrika* 42, 126–132
14 Sattenspiel, L. (1987) *Hum. Biol.* 59, 411–438
15 Keeling, M.J. (1997) *Trends Microbiol.* 5, 513–518
16 Grenfell, B. and Harwood, J. (1997) *Trends Ecol. Evol.* 12, 395–399
17 Ball, F., Mollison, D. and Scalia-Tomba, G. (1997) *Ann. Appl. Prob.* 7, 46–89
18 Becker, N.G. and Dietz, K. (1995) *Math. Biosci.* 127, 207–219
19 Becker, N.G. and Utev, S. (1998) *Math. Biosci.* 147, 23–39
20 Rapoport, A. and Yuan, Y. (1989) in *The Small World* (Kochen, M., ed.), pp. 327–348, Ablex Publishing, New Jersey, USA
21 Wasserman, S. and Faust, K. (1994) *Social Network Analysis: Methods and Applications*, Cambridge University Press
22 Keeling, M.J., Rand, D.A. and Morris, A.J. (1997) *Proc. R. Soc. London B Biol. Sci.* 264, 1149–1156
23 Watts, D.J. and Strogatz, S.H. (1998) *Nature* 393, 440–442
24 Crosby, A.W. (1989) *America's Forgotten Pandemic: The Influenza of 1918*, Cambridge University Press
25 Ewald, P.W. (1994) *Evolution of Infectious Disease*, Oxford University Press
26 Lipsitch, M. and Moxon, E.R. (1997) *Trends Microbiol.* 5, 31–37
27 Van Baalen, M. and Sabelis, M.W. (1995) *Trends Microbiol.* 3, 414–416
28 Levin, B.R. (1996) *Emerg. Infect. Dis.* 2, 93–102
29 Van Baalen, M. in *Virulence Management: The Adaptive Dynamics of Pathogen–Host Interactions* (Dieckmann, U. *et al.*, eds), Cambridge University Press (in press)
30 Claessen, D. and De Roos, A.M. (1995) *Oikos* 74, 401–413
31 Rand, D.A., Keeling, M. and Wilson, H.B. (1995) *Proc. R. Soc. London B Biol. Sci.* 259, 55–63

The influence of dsRNA viruses on the biology of plant pathogenic fungi

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Double-stranded RNA viruses are ubiquitous in fungi. They are non-infective and, like most prokaryotic plasmids, are only transmitted to compatible strains via cell fusion. Most are cryptic, but some with an established phenotype, such as the hypoviruses of the chestnut-blight fungus, have been studied for their potential as biological control agents of fungi.

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Viruses are among the variety of cytoplasmic genetic elements that has been identified in fungi. From our current knowledge, it appears that, at least in one way, they have evolved differently from viruses of other organisms in that they lack infectivity yet are still maintained in their hosts. The most common fungal viruses have double-stranded (ds) RNA genomes and are recognized as viruses even though some, such as the Hypoviridae, lack protein capsids[1]. The acceptance of these genetic elements as viruses is not without controversy, because of their lack of infectivity. These elements are transmitted in fungi in a similar manner to the transmission of plasmids in prokaryotes and, therefore, never leave a cytoplasmic environment. The biology of dsRNA fungal viruses is interesting, both because they represent an unusual adaptation of a parasite to its host and because of the variety of phenotypes that has been found to be associated with characterized dsRNAs.

dsRNA elements are ubiquitous in fungi, but only those associated with an obvious phenotype have been studied in detail. Four taxonomic families have been created based on these studies: the Totiviridae, the Partitiviridae, the Narnaviridae and the Hypoviridae[1,2]. Other fungal viruses are known that clearly do not belong to these families and it would appear, therefore, that we have only just begun to unearth the diversity of fungal viruses. However, we expect few surprises regarding the structure of any new viruses, as they appear to

 PII: S0966-842X(99)01568-1