THE ROYAL SOCIETY

Received 26 June 2003
Accepted 5 August 2003
Published online 16 October 2003

Contact tracing and disease control

Ken T. D. Eames[1] **and Matt J. Keeling**[2,3*]

[1]*Department of Zoology, University of Cambridge, Downing Street, Cambridge CB2 3EJ, UK*
[2]*Department of Biological Sciences, and* [3]*Mathematics Institute, University of Warwick, Gibbet Hill Road, Coventry CV4 7AL, UK*

Contact tracing, followed by treatment or isolation, is a key control measure in the battle against infectious diseases. It is an extreme form of locally targeted control, and as such has the potential to be highly efficient when dealing with low numbers of cases. For this reason it is frequently used to combat sexually transmitted diseases and new invading pathogens. Accurate modelling of contact tracing requires explicit information about the disease-transmission pathways from each individual, and hence the network of contacts. Here, pairwise-approximation methods and full stochastic simulations are used to investigate the utility of contact tracing. A simple relationship is found between the efficiency of contact tracing necessary for eradication and the basic reproductive ratio of the disease. This holds for a wide variety of realistic situations including heterogeneous networks containing core-groups or super-spreaders, and asymptomatic individuals. Clustering (transitivity) within the transmission network is found to destroy the relationship, requiring lower efficiency than predicted.

Keywords: tracing efficiency; transmission networks; pairwise approximations; stochastic simulation

1. INTRODUCTION

A range of control measures, from prophylactic vaccination (Fine & Clarkson 1983; McLean & Anderson 1988; Anderson & May 1992) to pre-emptive culling (Woolhouse & Donaldson 2001; Keeling *et al.* 2001), have been successfully used to combat the spread of disease or even eradicate infection entirely (Nokes & Swinton 1997; Bazin 2000). It is now well accepted that targeting such control measures, rather than applying them randomly, leads to an often dramatic increase in efficiency (Hethcote & Yorke 1984; Kault 1995). Contact tracing is an extreme from of targeted control, where the potential next-generation cases are the primary focus. Contact tracing has proved to be a highly successful strategy when the number of infectious cases is low. As such, contact tracing is used in the control of sexually transmitted diseases (STDs; Clarke 1998; FitzGerald *et al.* 1998; Macke & Maher 1999); it is the recommended policy for tackling outbreaks of new or re-emerging infections such as the recent severe acute respiratory syndrome (SARS) epidemic (Donnelly *et al.* 2003); and it is the standard tool for eliminating minor outbreaks in the latter stages of disease eradication. Spatially explicit contact tracing, in the form of either ring-vaccination or ring-culling, is also commonly used for a variety of livestock infections (Müller *et al.* 2000*b*; Woolhouse & Donaldson 2001; Haydon *et al.* 2003). With the recent increase in STD prevalence, the perceived risk from bio-terrorism and the aim of global polio eradication, the public-health importance of an understanding of contact tracing has never been greater.

Contact tracing is fundamentally linked to the individual-level spread of infection and, in particular, the network of potential transmission routes. Networks are a particularly important concept in human disease modelling, where social interactions can take place over a wide range of distances and where geographical distance alone is an inadequate measure of infection risk. For this reason we concentrate on human diseases in this paper. While some models have been developed to consider the role of contact tracing in randomly interacting populations (Müller *et al.* 2000*a*; Hyman *et al.* 2003), only network-based models that consider transmission pathways (Halloran *et al.* 2002; Huerta & Tsimring 2002) and the associated pairwise equations can provide an accurate mechanistic understanding of the structured nature of human interactions. Here, we use detailed pairwise equations (Eames & Keeling 2002) to gain an analytical insight into contact tracing both for STDs, such as gonorrhoea and chlamydia (which are described by a susceptible–infected–susceptible (SIS) framework), and for airborne infections, such as SARS, smallpox, polio and measles (which follow the susceptible–infected–recovered/dead paradigm). Finally, stochastic individual-based network simulations are used to validate the pairwise predictions and allow us to ascertain the effect of locally clustered networks on contact tracing.

Pairwise correlation models offer a robust mechanism to capture spatial effects by formulating equations for the number of connected pairs rather than just the number of individuals, and hence link individual-level behaviour to population-level dynamics. They have been shown to be in close agreement with full stochastic network simulations (Eames & Keeling 2002), and have been used to study a range of ecological and epidemiological problems (Keeling *et al.* 1997; Van Baalen & Rand 1998; Keeling 1999; Ferguson *et al.* 2001), in particular STDs, where pairs of connected individuals (sexual partnerships) are naturally specified (Bauch & Rand 2000; Ferguson & Garnett 2000; Eames & Keeling 2002). The pairwise approach has several key advantages over more complex simulations: most importantly, it provides an analytical framework, can be readily parameterized with available data (Eames & Keeling 2002) and generates results based on simple

*Author for correspondence (m.j.keeling@warwick.ac.uk).

properties of the transmission network. These advantages are used here to develop robust and generic results.

2. CONTACT-TRACING MODEL

Many STDs broadly conform to the SIS paradigm (Anderson & May 1992) whereby individuals enter sexually active life as susceptible (S), and infected individuals (I) return to the susceptible class following treatment. Treatment comes from two distinct sources, either the patient seeking medical assistance once symptoms develop, or the tracking of infection within the population by following potential transmission routes (sexual partnerships). Such contact tracing is an important public-health tool (Clarke 1998; FitzGerald *et al.* 1998; Macke & Maher 1999), especially when the disease may be asymptomatic (but still infectious), as it provides the only means by which such individuals can be easily identified. Contact tracing is incorporated into the model design using an SITS framework, where the treatment class (T) is short-lived and rapidly 'transmitted', mimicking the relatively quick speed at which tracing spreads across the network. From an ecological perspective we can view contact tracing as hyperparasitism (Mills & Gutierrez 1996; Sullivan & Völkl 1999), spreading through the same network as the disease, but affecting only those individuals that are already infected. Tracing can also be considered when individuals acquire immunity from further infection; this adds a recovered (R) state, giving rise to an SITR model. Throughout, it is assumed that while in the treatment class an individual can no longer transmit infection; this may be the result either of treatment or of isolation, whether behavioural (such as avoiding risky sexual practices) or physical (e.g. isolation wards).

We denote by $[S]$, $[I]$ and $[T]$ the numbers of susceptible and infected individuals, and the number in the treatment class, respectively. The population dynamics of each of these classes is determined by the partners to whom they are connected. Hence, to formulate the equations we let $[AB]$ refer to the number of partnerships within the network involving an individual of status A and another of status B (Keeling *et al.* 1997). The differential equations that govern the numbers in the individual classes are

$$[\dot{S}] = -\tau[SI] + a[T]$$
$$[\dot{I}] = \tau[SI] - g[I] - c[IT]$$
$$[\dot{T}] = g[I] + c[IT] - a[T],$$

where recovery occurs only after seeking medical advice, which initiates the contact-tracing process. Here, τ is the rate of spread of infection across a partnership, g is the recovery rate in the absence of tracing, c is the rate of contact tracing and a is the rate at which individuals move out of the treatment class. As indicated above, further interpretations are possible: we might consider g to be an isolation rate, for instance.

To solve these equations the behaviour of pairs of connected individuals (partnerships) also needs to be modelled. These pair equations in principle need information about triples (and triple equations would need information about higher-order terms); however, we can close the system by approximating these triples in terms of pairs and singles (see electronic Appendix A available on The Royal Society's Publications Web site). We define the infectivity of the disease, r, as the rate of transmission across a contact multiplied by the infectious period, $r = \tau/g$, and similarly the tracing and treatment level, t, is the rate that treatment 'spreads' multiplied by the treatment time, $t = c/a$. The probability that infection passes across a contact is $\tau/(g + \tau) = r/(1 + r)$, and the probability that treatment is passed on is $t/(1 + t)$. We refer to this latter quantity, which represents the proportion of contacts that are traced, as the 'tracing efficiency'.

3. CONTACT TRACING IN HOMOGENEOUS NETWORKS

When all infectious individuals display symptoms and all individuals have exactly n contacts to whom they can pass infection, analytical results allow an insight into eradication. By looking at the parameter values at which equilibrium prevalence and the initial growth of infection are zero, we can derive the level of tracing required to eliminate infection. In the SIS scenario, the critical level of tracing and treatment needed to eradicate infection is given by

$$t_c = \frac{rn^2 + 1 - 2rn - 2n + \sqrt{r^2n^4 + 2rn^2 + 1 - 4r^2n^2(n-1)}}{2(n-1)} \tag{3.1}$$

and hence $t_c/(1 + t_c)$ is the proportion of contacts that needs to be successfully traced and treated, i.e. the critical tracing efficiency. Figure 1*a* shows how the critical tracing efficiency dramatically increases with both the transmission probability of the disease and the number of contacts.

Applying the pairwise approximation to SIR-type infections results in a critical level of tracing with a much simpler form

$$t_c = \frac{rn(n-2) - n}{n-1}. \tag{3.2}$$

Figure 1*b* shows the associated critical tracing efficiency for diseases of this sort. SIR infections always require less effort to control, although the difference between SIR and SIS models becomes negligible as the number of contacts, n, becomes large—only where n is small are figures 1*a* and 1*b* appreciably different. In both models (most clearly from equation (3.2)), the critical level of contact tracing increases almost linearly with both the infectivity of the disease, r, and the number of contacts, n. We can relate this result to more standard epidemiological measures. The basic reproductive ratio, R_0, is defined as the expected number of secondary cases produced by an infectious individual in a totally susceptible population, and is calculated from the early asymptotic growth rate of the epidemic (Diekmann *et al.* 1990). For an SIR disease on a network, pairwise models show that the basic reproductive ratio is given by $R_0 = (n - 2)r$ (Keeling 1999). Hence, when the number of contacts, n, is large, equation (3.2) implies that

$$\text{critical tracing efficiency, } \frac{t_c}{t_c + 1} \approx 1 - \frac{1}{R_0}, \tag{3.3}$$

although this is a slight underestimate (figure 1*c*). For example, if $R_0 = 4$, around 75% of contacts must be traced to control infection. The same relationship also holds for

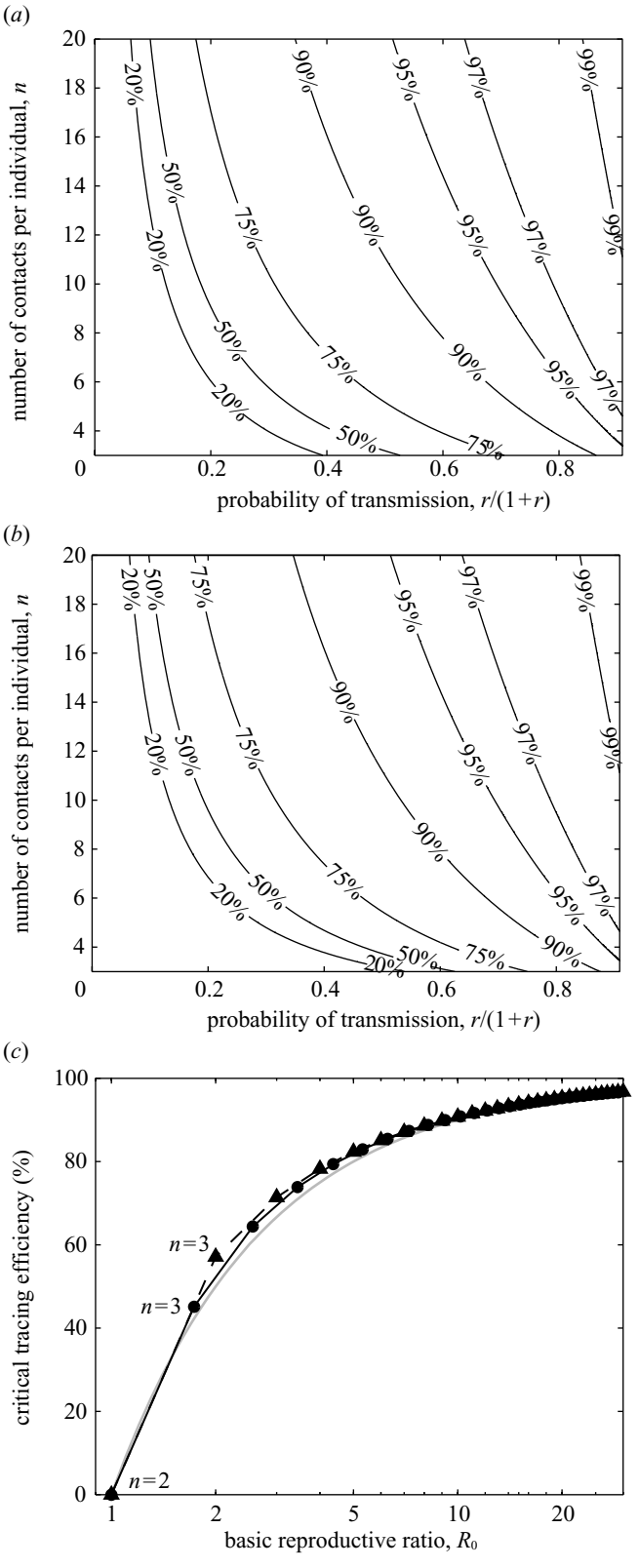


Figure 1. The effects of contact tracing on the dynamics of disease spread through networks; results from the pairwise model. (*a*) The critical tracing efficiency necessary to control an SIS-type epidemic in an unstructured network where everyone has an equal number of contacts. (*b*) The critical tracing efficiency for an SIR-type disease in a similar network. (*c*) Comparison between the simple prediction and results for the SIS and SIR pairwise disease models 3.1 and 3.2. Grey line indicates the theoretical level $(1 - 1/R_0)$; circles indicate the SIS model, and triangles indicate the SIR model. The probability of transmission is fixed at 0.5 (hence $r = 1$), while the number of contacts is varied to vary R_0; similar results are found for other levels of transmission.

SIS-type infections (equation (3.1)), despite the more complicated forms of t_c and R_0 (Eames & Keeling 2002). Surprisingly, this approximation is in direct agreement with the threshold level of random vaccination needed to eradicate infection (Anderson & May 1992) and agrees with that predicted using unstructured partnership models (Müller *et al.* 2000*a*). Thus, while the presence of strong local correlations, captured by the pairwise models, has a significant impact on the two emergent properties of R_0 and the critical tracing level, the relationship between these two properties remains largely unchanged.

It should be noted that we have implicitly assumed that all contacts are known and therefore theoretically traceable. However, if a proportion, p, of contacts are unknown (and therefore untraceable), then, to maintain the same average level of tracing, the tracing efficiency for the remaining known contacts must be increased to

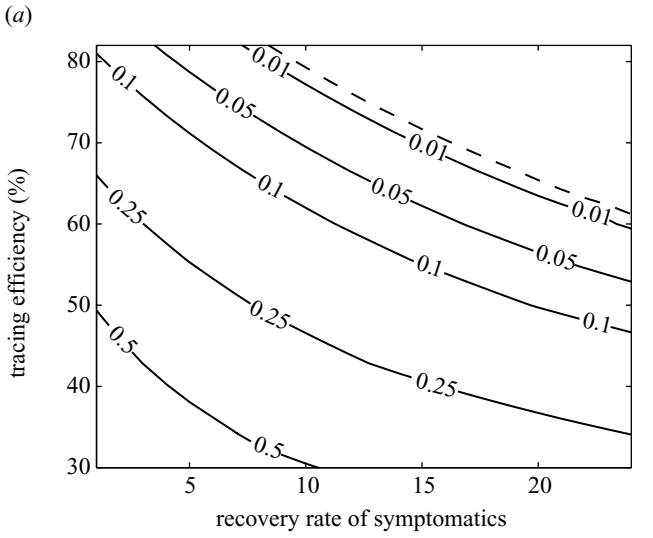
$$\text{critical tracing efficiency} \approx \frac{1}{1-p}\left(1 - \frac{1}{R_0}\right).$$

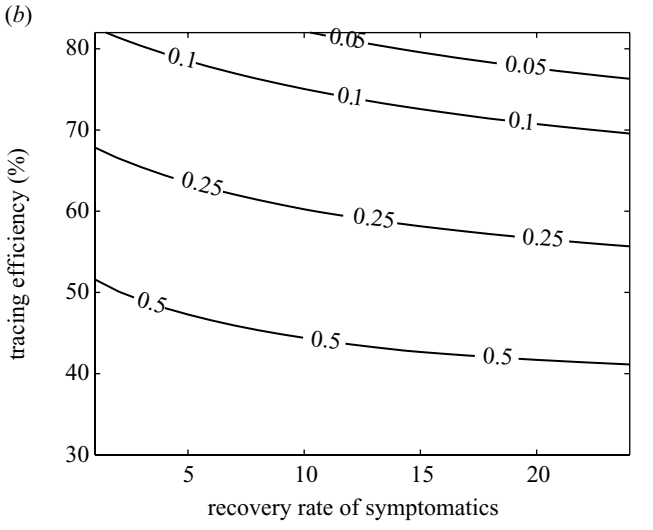
Thus, control of diseases with large reproductive ratios requires not only a high level of contact tracing but also comprehensive knowledge of the potential transmission routes.

4. CONTROL IN SEXUAL MIXING NETWORKS

We demonstrate how the pairwise model may be extended by introducing two more realistic features of STD transmission. First, a heterogeneous network is used so that individuals have differing numbers of contacts, reflecting the important role of super-spreaders or core-groups in the transmission and persistence of infection. This adaptation is appropriate for a wide range of infections. We use a network based on observed social networks (Klovdahl *et al.* 1977), but parameterizations from other networks produce comparable results. Second, a proportion of infected individuals are assumed to be asymptomatic: these individuals seldom seek treatment and consequently have a much longer infectious period (see electronic Appendix A). Figure 2*a*,*b* shows the equilibrium prevalence of infection as control measures are varied. The axes reflect the effort expended on each of the two major control mechanisms: identification of those individuals with symptoms (x-axis) and the contact-tracing efficiency (y-axis). When the proportion of new cases that are asymptomatic is larger (figure 2*b*) the importance of contact tracing increases. As asymptomatics are rarely detected or treated, it may be impossible to eradicate the infection without contact tracing even if symptomatic individuals are identified and treated immediately. Contact tracing is essential for eradication when each asymptomatic case is expected to give rise to at least one more asymptomatic case, i.e. when

(basic reproductive ratio of asymptomatics)
$\times$ (probability of asymptomatic infection) > 1.





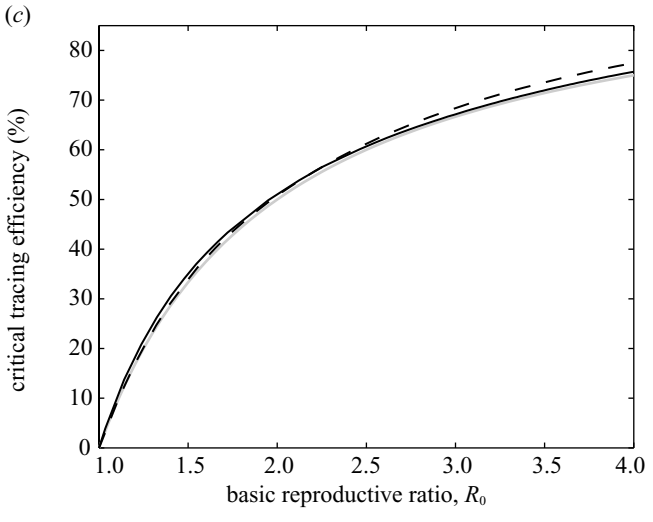


Figure 2. Prevalence of infection using the pairwise model for an STD in a realistic heterogeneous network. Asymptomatic individuals, who primarily receive treatment through tracing, are included. Both symptomatics and asymptomatics transmit infection across a partnership at rate $\tau = 12$; asymptomatics recover very slowly at the rate $g_a = 0.1$, while the recovery rate of symptomatics, g_s, is varied to mimic changes in the speed of case identification. All timescales are considered to be in years, and parameters match plausible values for STDs. The proportions of the new infections assumed to be asymptomatic are (*a*) 30%, also showing critical tracing efficiency (dashed line) and (*b*) 70%. (*c*) Comparison between the simple prediction (grey line, $1 - 1/R_0$) and results for these more realistic pairwise models (solid line, 30% asymptomatic; dashed line, 70% asymptomatic); R_0 is varied by altering the transmission rate, τ. In the example shown here, a network from Klovdahl *et al.* (1977) is used.

Because asymptomatics may subsequently cause symptomatic cases, contact tracing can potentially identify and treat such individuals, dramatically reducing the force of the infection. The prevalence of infection in the population largely determines the optimal balance between the identification of symptomatic individuals and contact tracing. Contact tracing is favoured when infection is globally rare but locally common within connected areas of the network.

The extinction thresholds for the situations shown in figure 2*a*,*b* can be redrawn in terms of R_0 (figure 2*c*). Despite the additional levels of complexity that have been included in the model, the simple relationship critical tracing efficiency $\approx 1 - 1/R_0$ still holds. This degree of robustness is remarkable and reflects a fundamental link between the spread of infection and the spread of control.

5. THE EFFECTS OF CLUSTERING IN TRANSMISSION NETWORKS

While pairwise models provide an excellent approximation to the network of sexual contacts involved in the spread of STDs (Eames & Keeling 2002), they are less accurate for airborne diseases where there is a high level of clustering (transitivity) within the network, so that connected individuals are likely to share common contacts. We therefore investigate the role of contact tracing and treatment in full stochastic simulations of disease transmission on a variety of computer-generated networks (Read & Keeling 2003; figure 3). Not only does this provide much more flexibility in the network structure, but it allows us to address many more applied issues. For the simplest situation, where every individual has exactly the same number of neighbours, figure 3*a* (solid line) shows the tracing efficiency needed to eradicate infection. When the network is highly clustered the necessary level of tracing decreases; this is to be expected as R_0 is generally reduced in clustered networks owing to the self-limitation of the epidemic as the local density of susceptibles is depleted. When the number of contacts is heterogeneous and the clustering low (dashed line), the associated value of R_0 is increased, as predicted from both standard theory (Anderson & May 1992) and pairwise approximations (Eames & Keeling 2002), and this necessitates an increase in the efficiency of contact tracing.

Once again, these results can be redrawn in terms of R_0 (figure 3*b*), although here there is a clear deviation from the standard pattern. Where there is no clustering within the network, stochastic simulations confirm the analytically derived threshold of $1 - 1/R_0$. However, clustering

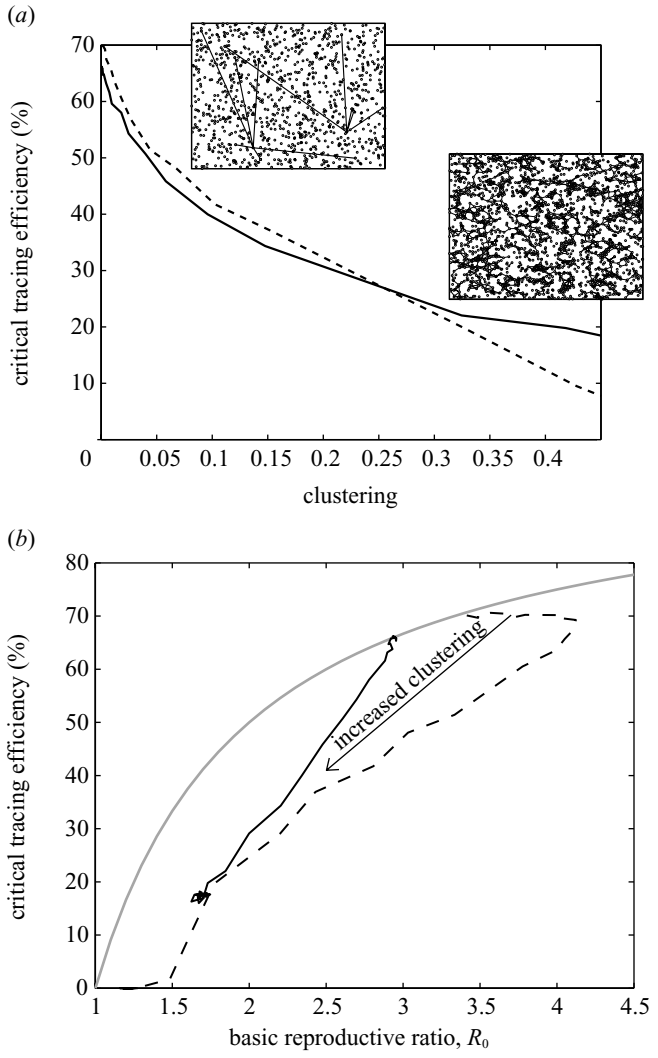


Figure 3. Average results from 500 stochastic simulations on computer-generated networks, focusing on the effects of clustering. Ten thousand individuals are positioned at random on a two-dimensional landscape, and connections are formed with a probability given by a Gaussian kernel. The clustering (the ratio of triangles to all triples in the network; Keeling 1999) of the network is determined by the kernel, with highly clustered configurations corresponding to a narrow kernel (Read & Keeling 2003). The infectivity is set at $r = 1$, so the probability of infection being transmitted across a contact is 0.5. (*a*) The critical tracing efficiency necessary for control is seen to decrease as the clustering increases. Results for two distinct types of network are shown: a homogeneous network in which everyone has exactly five contacts (solid line), and a heterogeneous network in which the number of contacts per individual varies but the average number of contacts remains five (dashed line). Also shown are examples of a random and a clustered network: for the random network the connections from two individuals are shown, whereas for the clustered network it is feasible to display all the connections. (*b*) The results from graph (*a*) shown with respect to the basic reproductive ratio, R_0. Clustering causes a substantial deviation away from the simple theoretical ideal ($1 - 1/R_0$, grey line), such that lower tracing efficiency is sufficient. When clustering is equal to zero, the theoretical threshold applies, but as clustering increases, the threshold moves away from the expected curve. For the heterogeneous network, small amounts of clustering actually increase R_0 owing to aggregation of high-risk individuals with many partners.

acts very differently on the values of R_0 and the critical tracing efficiency. In general, R_0 decreases with clustering owing to increased self-limitation, although in the heterogeneous network (dashed line) small amounts of clustering create interconnected core-groups, which promote spread, and therefore R_0 initially increases. While the critical tracing efficiency responds to changes in R_0, clustering causes an additional reduction as individuals can be traced via a variety of routes owing to multiple partners being infected. Thus, contact tracing performs far better than predicted in highly clustered environments, and may be more effective against the clustered spread of airborne diseases than expected from work with STDs.

6. DISCUSSION

The analytical and simulation results presented have obvious public-health implications. For STDs, because most of the population have few sexual partners (Johnson *et al.* 1994), tracing is a very efficient means of identifying infected individuals, and hence reducing the number of cases or even eradicating the disease. Contact tracing provides a method of targeted control whereby intervention is automatically focused on the subset of the population most likely to be at risk of infection, i.e. partners of infected individuals. The benefits of contact tracing are even more pronounced when asymptomatic infections can linger unchecked in the population. For airborne infections, where the average number of neighbours and the basic reproductive ratio are larger, contact tracing has to be far more efficient and generally far more rapid.

Robust approximation models and stochastic simulations have shown that in the absence of clustering (when contacts occur independently) the necessary tracing efficiency can be estimated from the basic reproductive ratio (see equation (3.3)). This simple relationship is robust to a number of modifications that increase the realism of the epidemic models. Thus, while spatial correlations, network heterogeneity and asymptomatic infections can dramatically modify the epidemic dynamics (Keeling *et al.* 1997; Keeling 1999; Eames & Keeling 2002), the relationship between the emergent properties of R_0 and the critical tracing efficiency is conserved. This basic relationship can therefore be expected to hold for a wide range of STDs and real-world transmission scenarios. However, the more clustered (transitive) nature of airborne or social transmission networks breaks this relationship—although it still provides a useful upper bound. For the airborne spread of re-emerging diseases, where contact tracing is a recommended control strategy, an informed tracing policy requires an estimation of the degree of clustering within the transmission network. So far this element of transmission networks has not been comprehensively investigated, although diary-based studies of individual contacts may provide results (Edmunds *et al.* 1997).

The critical tracing efficiency derived for homogeneous networks shows that the level of contact tracing required depends on the number of contacts. This suggests that, in a realistic heterogeneous network, prioritizing tracing from those individuals with many contacts might further reduce the healthcare burden. Such targeted tracing may be a

valuable tool in future control strategies, and merits deeper investigation with the techniques discussed here.

There are several distinct scenarios under which contact tracing may not be an effective control strategy. In high-risk groups, with many possible transmission routes and a high incidence of infection, random screening may be a more efficient tool. Also, for airborne infections there may be a significant fraction of contacts that are untraceable; in such cases it may be impossible to achieve the necessary threshold, and additional control measures would need to be applied. If the tracing process is significantly slower than the infection process then, no matter how large a proportion of contacts is eventually traced, it will be impossible to keep pace with the epidemic; there is a trade-off between tracing speed and tracing efficiency. Finally, we note that, once above the critical level of contact tracing, the number of cases decreases through time, as the effective reproductive ratio, R, is less than 1. However, when this rate of decline is not sufficiently rapid (R is close to 1) the epidemic may be long-lived and many subsequent cases can arise. Hence, the theoretical threshold, which reduces the reproductive ratio to below 1, acts as a lower bound to the tracing efficiency that should be sought. In practice, much higher tracing efficiency may be needed to reduce the epidemic size quickly to a minimal level. Highly efficient contact tracing may place fewer demands on medical resources as a rapidly curtailed outbreak will involve fewer individuals needing to be traced and treated.

The models presented here represent highly simplified versions of real infection processes, but serve to indicate how contact tracing may be effective. More complex disease dynamics may alter the balance between different intervention methods. For example, diseases that are infectious before they become symptomatic will be harder to control through contact tracing: by the time index cases are identified, the epidemic will have spread some distance through the network. However, the ability of contact tracing to identify asymptomatic individuals would prove beneficial in such situations. The efficacy of contact tracing, therefore, is dependent not only on constraints or resources, but also on the disease itself.

This research was supported by the MRC (K.T.D.E.), the BBSRC (M.J.K.), the Wellcome Trust (M.J.K.) and The Royal Society (M.J.K.). The authors thank two anonymous referees for their thoughtful comments.

REFERENCES

Anderson, R. M. & May, R. M. 1992 *Infectious diseases of humans*. Oxford University Press.

Bauch, C. & Rand, D. A. 2000 A moment closure model for sexually transmitted disease transmission through a concurrent partnership network. *Proc. R. Soc. Lond.* B **267**, 2019–2027. (DOI 10.1098/rspb.2000.1244.)

Bazin, H. H. 2000 *The eradication of smallpox: Edward Jenner and the first and only eradication of a human infectious disease*. London: Academic.

Clarke, J. 1998 Contact tracing for chlamydia: data on effectiveness. *Int. J. STD AIDS* **9**, 187–191.

Diekmann, O., Heesterbeek, J. A. P. & Metz, J. A. J. 1990 On the definition and the computation of the basic reproduction ratio R_0, in models for infectious diseases in heterogeneous populations. *J. Math. Biol.* **28**, 365–382.

Donnelly, C. A. (and 18 others) 2003 Epidemiological determinants of spread of causal agent of severe acute respiratory syndrome in Hong Kong. *Lancet* **361**, 1761–1766.

Eames, K. T. D. & Keeling, M. J. 2002 Modeling dynamic and network heterogeneities in the spread of sexually transmitted disease. *Proc. Natl Acad. Sci. USA* **99**, 13 330–13 335.

Edmunds, W. J., O'Callaghan, C. J. & Nokes, D. J. 1997 Who mixes with whom? A method to determine the contact patterns of adults that may lead to the spread of airborne infections. *Proc. R. Soc. Lond.* B **264**, 949–957. (DOI 10.1098/rspb.1997.0131.)

Ferguson, N. M. & Garnett, G. P. 2000 More realistic models of sexually transmitted disease transmission dynamics: sexual partnership networks, pair models, and moment closure. *Sex. Transm. Dis.* **27**, 600–609.

Ferguson, N. M., Donnelly, C. A. & Anderson, R. M. 2001 The foot-and-mouth epidemic in Great Britain: pattern of spread and impact of interventions. *Science* **292**, 1155–1160.

Fine, P. E. M. & Clarkson, J. A. 1983 Measles in England and Wales. 3. Assessing published predictions of the impact of vaccination on incidence. *Int. J. Epidemiol.* **12**, 332–339.

FitzGerald, M. R., Thirlby, D. & Bedford, C. A. 1998 The outcome of contact tracing for gonorrhoea in the United Kingdom. *Int. J. STD AIDS* **9**, 657–660.

Halloran, M. E., Longini, I. M., Nizam, A. & Yang, Y. 2002 Containing bioterrorist smallpox. *Science* **298**, 1428–1432.

Haydon, D. T., Chase-Topping, M., Shaw, D. J., Matthews, L., Friar, J. K., Wilesmith, J. & Woolhouse, M. E. J. 2003 The construction and analysis of epidemic trees with reference to the 2001 UK foot-and-mouth outbreak. *Proc. R. Soc. Lond.* B **270**, 121–127. (DOI 10.1098/rspb.2002.2191.)

Hethcote, H. W. & Yorke, J. A. 1984 *Gonorrhea transmission dynamics and control.* Springer Lecture Notes in Biomathematics no. 56. Berlin: Springer.

Huerta, R. & Tsimring, L. S. 2002 Contact tracing and epidemics control in social networks. *Phys. Rev.* E **66**, article number 056115.

Hyman, J. M., Li, J. & Stanley, E. A. 2003 Modeling the impact of random screening and contact tracing in reducing the spread of HIV. *Math. Biosci.* **181**, 17–54.

Johnson, A. M., Wadsworth, J., Wellings, K. & Field, J. 1994 *Sexual attitudes and lifestyles*. Oxford: Blackwell Scientific.

Kault, D. A. 1995 Modeling AIDS reduction strategies. *Int. J. Epidemiol.* **24**, 188–197.

Keeling, M. J. 1999 The effects of local spatial structure on epidemiological invasions. *Proc. R. Soc. Lond.* B **266**, 859–867. (DOI 10.1098/rspb.1999.0716.)

Keeling, M. J., Rand, D. A. & Morris, A. J. 1997 Correlation models for childhood epidemics. *Proc. R. Soc. Lond.* B **264**, 1149–1156. (DOI 10.1098/rspb.1997.0159.)

Keeling, M. J., Woolhouse, M. E. J., Shaw, D. J., Matthews, L., Chase-Topping, M., Haydon, D. T., Cornell, S. J., Kappey, J., Wilesmith, J. & Grenfell, B. T. 2001 Dynamics of the 2001 UK foot and mouth epidemic: stochastic dispersal in a heterogeneous landscape. *Science* **294**, 813–817.

Klovdahl, A. S., Dhofier, Z., Oddy, G., O'Hara, J., Stoutjesdijk, S. & Whish, A. 1977 Social networks in an urban area: first Canberra study. *Aust. N. Z. J. Sociol.* **13**, 169–172.

Macke, B. A. & Maher, J. E. 1999 Partner notification in the United States: an evidence-based review. *Am. J. Prev. Med.* **17**, 230–242.

McLean, A. R. & Anderson, R. M. 1988 Measles in developing countries. 2. The predicted impact of mass vaccination. *Epidemiol. Infect.* **100**, 419–442.

Mills, N. J. & Gutierrez, A. P. 1996 Prospective modeling in biological control: an analysis of the dynamics of heteronomous hyperparasitism. *J. Appl. Ecol.* **33**, 1379–1394.

Müller, J., Kretzschmar, M. & Dietz, K. 2000*a* Contact tracing in stochastic and deterministic epidemic models. *Math. Biosci.* **164**, 39–64.

Müller, J., Schönfisch, B. & Kirkilionis, M. 2000*b* Ring vaccination. *J. Math. Biol.* **41**, 143–171.

Nokes, D. J. & Swinton, J. 1997 Vaccination in pulses: a strategy for global eradication of measles and polio. *Trends Microbiol.* **5**, 14–19.

Read, J. M. & Keeling, M. J. 2003 Disease evolution on networks: the role of contact structure. *Proc. R. Soc. Lond.* B **270**, 699–708. (DOI 10.1098/rspb.2002.2305.)

Sullivan, D. J. & Völkl, W. 1999 Hyperparasitism: multitrophic ecology and behavior. *A. Rev. Entomol.* **44**, 291–315.

Van Baalen, M. & Rand, D. A. 1998 The evolution of altruism in viscous populations. *J. Theor. Biol.* **193**, 631–648.

Woolhouse, M. & Donaldson, A. 2001 Managing foot-and-mouth. *Nature* **410**, 515–516.

As this paper exceeds the maximum length normally permitted, the authors have agreed to contribute to production costs.

Visit http://www.pubs.royalsoc.ac.uk to see an electronic appendix to this paper.