Modelling control of epidemics spreading by long-range interactions

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We have studied the spread of epidemics characterized by a mixture of local and non-local interactions. The infection spreads on a two-dimensional lattice with the fixed nearest neighbour connections. In addition, long-range dynamical links are formed by moving agents (vectors). Vectors perform random walks, with step length distributed according to a thick-tail distribution. Two distributions are considered in this paper, an α -stable distribution describing self-similar vector movement, yet characterized by an infinite variance and an exponential power characterized by a large but finite variance. Such long-range interactions are hard to track and make control of epidemics very difficult. We also allowed for cryptic infection, whereby an infected individual on the lattice can be infectious prior to showing any symptoms of infection or disease. To account for such cryptic spread, we considered a control strategy in which not only detected, i.e. symptomatic, individuals but also all individuals within a certain control neighbourhood are treated upon the detection of disease. We show that it is possible to eradicate the disease by using such purely local control measures, even in the presence of long-range jumps. In particular, we show that the success of local control and the choice of the optimal strategy depend in a non-trivial way on the dispersal patterns of the vectors. By characterizing these patterns using the stability index of the α -stable distribution to change the power-law behaviour or the exponent characterizing the decay of an exponential power distribution, we show that infection can be successfully contained using relatively small control neighbourhoods for two limiting cases for long-distance dispersal and for vectors that are much more limited in their dispersal range.

Keywords: epidemiological modelling; disease spread; stochastic modelling; epidemiological control; dispersal patterns

1. INTRODUCTION

Human, animal and plant epidemics often spread on complicated networks characterized by a mixture of local and non-local interactions. These links reflect complicated structures of geographical, biological, social and economical interactions among individuals and are often very difficult to identify and control. This is particularly true for long-range transmission events and yet those are often the major factors behind the global and devastating spread of epidemics. Long-range transmissions have been implicated in the control of recent major epidemics of human (notably SARS (Dye & Gay 2003)) and animal (foot-and-mouth disease (Keeling *et al.* 2001)) diseases. Numerous, well-documented examples are also accumulating for plant disease, the primary focus of this paper, in which

hosts remain stationary but inoculum of the pathogen is dispersed over long distances by wind and vectors, including humans. Examples include Dutch elm disease (Swinton & Gilligan 1996), citrus canker (Gottwald *et al.* 2001), sudden oak death (Rizzo *et al.* 2002) and rhizomania of sugar beet (Stacey *et al.* 2004). The problem of tracking linkages itself is an important issue (Dybiec 2008). Whereas failure to identify and account for connections among individuals can significantly reduce the efficiency of disease control strategies, the cost of tracking long-range interactions can consume large amounts of resources that could otherwise be used for the treatment of infected individuals (Kleczkowski *et al.* 2006). It follows that a balance must be found in allocating resources between identification of links and control actions.

Epidemiological models can be used to assist in the development of detection and control strategies that aim at the reduction or eradication of the infectious

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agent while minimizing costs (Dybiec *et al.* 2004; Forster & Gilligan 2007). We have previously studied an epidemiological model within a small-world framework, involving a mixture of local and non-local interactions (Dybiec *et al.* 2004, 2005; Kleczkowski *et al.* 2006). We have shown that it is possible to control the spread of disease in such systems by a mixture of preventive and responsive treatment strategies that act locally, analogous to ring vaccination. We have also shown that the effectiveness of such control measures could be significantly improved if some long-range links in a small-world model are tracked down and removed (Kleczkowski *et al.* 2006). However, in the previous model, the network topology was fixed for the duration of the epidemic, although the long-range component was allowed to vary between realizations. This assumption is clearly not true for many epidemic systems and, in particular, the non-local part is often associated with highly intermittent and variable processes. For example, the spread of the fungus *Ophiostoma novo-ulmi* that causes Dutch elm disease might involve rare, unpredictable and ephemeral events caused by the movement of the bark beetle vector (Swinton & Gilligan 1996). Similar long-distance events may occur with human vectors moving through the landscape, either by migrant workers and contractors' machinery for diseases such as rhizomania (Stacey *et al.* 2004) or in the postulated case for sudden oak death on the boots of hikers (Rizzo *et al.* 2005). It is therefore not reasonable to assume that long-range links are fixed, but rather we need to consider dynamical realizations of such events.

In this paper we consider epidemics that spread not only via the fixed local nearest-neighbour links, but also via dynamical random interactions. The latter could be modelled in a number of ways. For example, in a small-world model (Dybiec *et al.* 2004), randomly placed links join two hosts regardless of the distance between them. In this paper, we use two more realistic models describing the distance a vector can move among hosts. We apply two families of thick-tailed, probability densities to describe the dispersal patterns of vectors, an α -stable density and the exponential power density. Each of these gives rise to the dispersal patterns with isolated daughter foci, while differing in the underlying dispersal mechanisms. α -Stable distributions encompass a range of cases, controlled by a single parameter, that extend from a dynamical small-world model with occasional long-distance movement to essentially local spread. The distributions are self-similar and so can be used to simulate vector movements that are similar on all length scales (Janicki & Weron 1994), in which frequently occurring smaller moves are interspersed by more rarely occurring longer jumps. This behaviour is characteristic of a Lévy flight (Shlesinger *et al.* 1995; Klafter *et al.* 1996; Sims *et al.* 2008), a type of a random walk in which each step is chosen from an α -stable distribution. Lévy flight models have been proposed for the trajectories of many vectors, including humans (Viswanathan *et al.* 1996; Boyer *et al.* 2004; Hufnagel *et al.* 2004; Brockmann *et al.* 2006; Reynolds 2006a) for which various mechanistic interpretations for foraging behaviour have been advanced (Viswanathan *et al.*

1999; Bartumeus *et al.* 2005; Reynolds 2006a,b,c; Reynolds & Frye 2007) with some degree of controversy (Edwards *et al.* 2007). In our approach, vectors perform a random walk with increments drawn from an α -stable distribution (as for that in a Lévy flight), but each step of the random walk corresponds to a single update of the epidemic model. In contrast to a Lévy walk (a continuous-time version of a Lévy flight (cf. Janicki & Weron 1994)), our model assumes that the vectors jump instantaneously to the new location where they might interact with hosts. It is important to note that, in this paper designed to analyse strategies for local control of disease with short- and long-distance dispersal, the α -stable distribution is used as a convenient and simple mechanism to simulate a self-similar random-jump process. We test the robustness of our results for the α -stable distribution, by repeating the key analyses for a second distribution for long-range movements, the exponential power distribution (Haynes 1974), which has a finite variance for jump increments.

We first present the model formulation and define the total cost of epidemic spread and control. We assume that control can only be affected locally, but possible long-range spread cannot be traced and prevented owing to its ephemeral nature. In §3, we study the sensitivity of the cost to the choice of the particular control strategy and, in particular, the control radius as defined in Dybiec *et al.* (2005) and Kleczkowski *et al.* (2006). We show that, despite uncertainties about the long-range spread, it is still possible to halt the epidemic outbreak using purely local methods. The model is primarily motivated by plant epidemics spreading in a population of static hosts, but it can be generalized to include vector-mediated diseases of humans and animals.

2. MODEL

The model has three basic elements: (i) individual behaviour, (ii) topology of local spread, and (iii) dynamics of moving agents (vectors) that are responsible for non-local transmission of infection. For the basic model structure, we assume a regular lattice topology with periodic boundary conditions without any additional shortcuts.

Each site is occupied by one individual that can be in one of five states: susceptible **S**; infected **I**; detected **D**; spontaneously recovered **R**; and treated (vaccinated) **V** state (Anderson & May 1991; Dybiec *et al.* 2004, 2005). Initially, all nodes on a two-dimensional 100×100 regular lattice (with periodic boundary conditions) are occupied by susceptible individuals. A small proportion of the nodes (10, i.e. 0.1% of all nodes) are then changed to infectious symptomatic individuals, reflecting the state of the system at the initial stage of an epidemic when the authorities are first made aware of it. Each susceptible individual in state **S** can be infected with probability p by any infectious or detected individual in its immediate neighbourhood (including the four nearest neighbours) or by a contagious (pathogen carrying) vector (for details see below). The dual nature of the force of infection reflects either two different modes of spread (direct and vector) or models

a dispersal that is characterized by clearly separated time and spatial scales.

Following a successful infection, an infected and pre-symptomatic individual (**I**) can become detected (and move to the detected class **D**) with probability q (which controls the duration of the cryptically infectious period). Spontaneous recovery can follow, with probability r , upon which the detected individual moves to the recovered class **R**. Alternatively, it can trigger a control measure (with probability v), in which all individuals within the control neighbourhood (including the individual that has triggered it) move to the treated class **V**. The control neighbourhood is characterized by a radius of vaccination z : z represents a maximal order of the control neighbourhood, constructed in an iterative way starting from an individual that triggered control measures and following its first-order neighbours, second-order neighbours, up to order z . Thus, the control action is applied locally, but extends beyond the nearest neighbours. The main objective of the extension of the neighbourhood ($z \geq 1$) is to treat all pre-symptomatic but already infectious individuals (and not only symptomatic individuals) before they can spread infection further. Individuals in class **R** and **V** can neither move back to the susceptible class nor become reinfected. Further details of the model and spatial structure of the disease and control neighbourhoods are given in Dybiec *et al.* (2005) and Kleczkowski *et al.* (2006). The total number of nodes is denoted by N and the number of susceptible nodes by S , infected by I , detected by D , recovered by R and treated by V .

The system is assumed to be homogeneous; all individuals are characterized by identical parameters including details of the treatment strategy. The treatment is triggered by individuals in the detected class but encompasses all individuals in the treatment neighbourhood independently of their status. No further tests are performed, for example to establish whether there are any infected individuals just outside the treatment range. While this is clearly an oversimplification, it faithfully represents a situation in which the detection of pre-symptomatic individuals is very difficult if at all possible.

In addition to the local force of infection, any individual in the susceptible class **S** can be infected with probability p by one of the moving contagious (pathogen carrying) agents (vectors). In this paper, we assume that vectors perform a random-jump process with properties that are unrelated to the position or the state of individuals located at lattice nodes. Thus, the vectors do not actively search for either infected or susceptible individuals, but the infectious process is subsidiary to the dispersal process. In order to distinguish between the local spread of the infection by a node-to-node contact, we assume that the vector-mediated spread is highly non-local. At each time step, each vector jumps from one location to another, with a distance that is independent of whether it is contagious or not and is independently generated along both axes. When a vector jumps to a new location, it interacts with a single host at the nearest node to its final location only. The random-jump process is assumed to occur

independently of the epidemiological status of the hosts and/or vectors. Periodic boundary conditions are assumed for both local interactions and vector random-jump process.

We use two families of probability distributions to describe the jump process. Firstly, we apply α -stable distributions with a characteristic function given by equation (2.1) (Feller 1968; Janicki & Weron 1994). The α -stable densities belong to a family of unimodal probability density functions (Janicki & Weron 1994). Here, we restrict our considerations to symmetric distributions only that are described by the characteristic function (Feller 1968; Janicki & Weron 1994),

$$\phi(k) = \exp[ik\mu - \sigma^\alpha |k|^\alpha]. \quad (2.1)$$

In equation (2.1), the most important parameter is the stability index of the distribution, α ($\alpha \in (0, 2]$), which describes the power-law behaviour, i.e. for $\alpha < 2$ with large x , $p(x) \propto |x|^{-(1+\alpha)}$. Two remaining parameters are μ , the location parameter, and σ , the scale parameter. In the asymptotic case of $\alpha = 2$, any stable distribution is equivalent to the Gaussian distribution with mean μ and variance $2\sigma^2$ (see equation (2.1)). The random numbers distributed according to the characteristic function given by equation (2.1) are generated using the Janicki–Weron algorithm (Janicki & Weron 1994; Weron 1996).

The choice of α -stable increments for the random-jump process leads to an infinite variance for the distribution of individual jumps. We also use an alternative approach in which the increments for each jump are drawn from the exponential power distribution given by the probability density function

$$p(x) = \frac{1}{2a\Gamma(1 + 1/b)} \exp\left[-\left|\frac{x}{a}\right|^b\right], \quad (2.2)$$

where a is the scale parameter and b is the exponent characterizing the tail of the distribution. In contrast to the α -stable distribution, the distribution (2.2) is of the thick-tail type but characterized by finite mean and variance. For $b = 2$, the exponential power distributions are equivalent to the Gaussian one. Similar to the α -stable distribution, the ‘tail-shape’ parameter b can be used to study the effect the thick-tail property of the dispersal has on the choice of the optimal control strategy. Small values of α and b correspond to a long-range dispersal in which vectors tend to spend most of the time taking small steps, but occasionally a very long step is made. By contrast, large values of α and b correspond to a short-range dispersal pattern in which vectors tend to move in relatively small steps.

Initially, all vectors are in a non-contagious state. A vector becomes contagious after each contact with an infectious or detected individual. Following this contact, if the vector encounters a susceptible individual, it will pass on the infection with probability p . In order to model a finite infectious time for the vector, we assume that it can only infect a given number of susceptible individuals in a series of encounters. This number is generated from a uniform distribution with the maximal value of 50. This number is fixed for every vector and it is measured since the last contact with an

infectious or detected individual. The total number of vectors ($\#Vec$) is fixed but the numbers of vectors in contagious (C) and non-contagious (NC) classes vary over time.

During the simulation, transition probabilities were calculated and the state of the system updated at each step, with a fixed (and small) time increment. The simulation loop was performed as long as $I(t) + D(t) + C(t) > 0$. For a given set of parameters, 100 realizations were simulated. From the simulations, sample quantities characterizing epidemics and their variability were estimated.

The main objective of this paper is to identify a control strategy that not only stops the epidemic as quickly as possible, but also does this at the smallest possible cost. These two objectives are conflicting and so we need to define a measure that allows us to find an optimal compromise between treating all individuals in the population (thus minimizing the cost of infection) and treating none at all (thus minimizing the cost of the treatment). We define a severity index, $X \equiv R(\infty) + V(\infty)$, a linear combination of the severity of an outbreak at the end of an epidemic measured by the number of affected individuals (R) and the costs of treating them (the number of treated individuals, V). The parameters have been chosen to represent the ‘worst-case’ scenario in which infection and disease quickly spread through the system when no controls are applied (except when p is very small). The recovery parameter r is chosen to be small so that $X \approx V(\infty)$ except when the treatment is very limited (small z , very small values of q and v).

In the simulations, we varied all parameters except the recovery probability, $r=0.01$ (giving $R(\infty)/V(\infty) \leq 0.016$). The treatment range for z (order of the treatment neighbourhood) varied between 1 and 16. The numbers of vectors ($\#Vec$) that can spread infection from one place to another varied between 0 and 500 by 50. The value of the stability index, α , characterizing the Lévy flights was changed from $\alpha=0.1$ to $\alpha=2.0$. The exponent b in equation (2.2) was altered from $b=0.1$ to $b=2.0$. Values of other parameters were adjusted to $\sigma=1$ and $a=1$. However, we have also tested the influence of these scale parameters on the eradication strategies.

3. RESULTS

We first show the results obtained for α -stable increments (Lévy flights) in the random-jump process describing the dynamics of vectors and subsequently relax the assumption of a self-similar nature of the dispersal by comparing the results with those obtained for the exponential power distribution. The severity of epidemics in the absence of vectors is characterized by two distinct regions (figure 1). For small values of the control radius, z , the local control is ineffective as it does not catch a large enough proportion of pre-symptomatic individuals, leading to the value of $X \approx R(\infty) \approx N$. This reflects our choice of parameters, particularly r , whereby, in the absence of control, the epidemic spreads rapidly and encompasses the whole population. For large values of the control radius, z ,

Figure 1. The normalized severity of the epidemics, $X = [V(\infty) + R(\infty)]/100\%$, for the probability of detection, $q=0.5$, and the probability of treatment, $v=0.5$. The number of vectors is (a) 0, (b) 50 and (c) 100; stability index $\alpha=0.5$. Dots indicate optimal strategies.

there is overkill in control and resources are ‘wasted’ on vaccination of susceptible individuals. The two regions are separated by a ‘valley’ in which the particular control strategy is close to the optimal one (denoted by dots). For each value of z , we can then identify an optimal value z_c corresponding to the minimum value of $X = X_c$ and we analyse those two characteristics later in the paper. For purely local spread, the valley is very deep: if the control strategy is chosen correctly, the spread of the epidemic can be halted very quickly and at a small cost. This is reflected by relatively small values of z_c and X_c for the case without vectors in figure 2.

Addition of vectors (figure 1) causes the surface to move up (higher values of X) and to become more shallow, as we introduce an additional source of infection. More interestingly, in the large- z limit, the severity X does not change much when the control radius, z , is increased. In this region, we are able to stop the epidemic with relatively few control events

Figure 2. (a) The normalized severity, X_c , corresponding to the optimal control radius and (b) optimal radius of vaccination, as functions of the number, $\#Vec$, of moving vectors. Other simulation parameters: $q=0.5$, $v=0.5$. The α -stable distributions are characterized by $\alpha=(i) 0.5$, (ii) 1.0, (iii) 1.5, (iv) 2.0. Different symbols correspond to different infection probabilities (from top to bottom), $p=0.9$ (circles), $p=0.5$ (squares) and $p=0.1$ (triangles). Error bars represent the standard deviation of the sample.

independently of the actual value of z . An increase in z causes an increase in X whereby the treatment is wasted on healthy individuals. However, we do not treat vectors, and when they are introduced they are able to spread the infection further and so more treatment events are necessary to control the spread of disease. The relative independence of X on z in the large- z limit means, in addition, that the estimators of the optimal radius of control, z_c , and the corresponding severity, X_c , are less reliable in this limit. This reflects the large variability among different realizations with the effect increasing when the number of vectors increases.

An increase in the number of vectors results in a monotonic increase in both the optimal radius, z_c , and the corresponding severity, X_c (figure 2), with large fluctuations associated with z_c . The presence of vectors that are able to forage far ($\alpha=0.5$) leads to a rapid increase in epidemic size, even at the optimal choice of z_c .

Thus, the addition of even very few Lévy fliers to the system makes the local control very ineffective (compare, for example, the values of z_c and X_c for no vectors and for 100 vectors, i.e. for 1% of the number of nodes). This is corroborated by the increase in the severity as the range of the Lévy flights increases while α decreases. The change of α from 0.5 to 1.5 corresponds to a decrease in the probability of very long jumps being made by individual vectors.

The dependence of X_c on disease-specific parameters is shown in more detail in figure 3 and of z_c in figure 4. The severity of the epidemics, X_c , and the critical control radius, z_c , increase monotonically with probability of spread, p , although there is a large amount of variation in z_c without much change in X_c . Once p increases from 0.1 to 0.5, it is already difficult to stop the epidemic other than by treating almost all individuals and so the corresponding z_c is very large. Increases in the probability of detection, q , and the speed with which the treatment is

Figure 3. The severity of the epidemic at the optimal strategy, X_c , as a function of the probability of infection, p . The α -stable distributions are characterized by (a,b) $\alpha=0.5$ and (c,d) $\alpha=1.5$. Various symbols correspond to different numbers of vectors (from top to bottom), 500 (circles), 250 (squares) and 50 (triangles). The detection and treatment probabilities are (a,c) $q=0.1$ and $v=0.1$ and (b,d) $q=0.9$ and $v=0.9$.

Figure 4. The optimal value of z_c corresponding to epidemic severity presented in figure 3 as a function of the probability of infection, p . The α -stable distributions are characterized by (a,b) $\alpha=0.5$ and (c,d) $\alpha=1.5$. Various symbols correspond to different numbers of vectors (from top to bottom), 500 (circles), 250 (squares) and 50 (triangles). The detection and treatment probabilities are (a,c) $q=0.1$ and $v=0.1$ and (b,d) $q=0.9$ and $v=0.9$.

applied, v , reduce the optimal radius (figure 4) and the corresponding size of the epidemic (figure 3).

To explore further the role of dispersal of vectors in disease spread and control, we studied the dependence of the severity of the epidemic at the optimal strategy, X_c , and optimal radius of vaccination, z_c , as functions of the characteristic exponent α for various numbers of vectors ($\#Vec=150$ and 500 ; figure 5). As expected, the decrease in the range of vectors (increasing stability index α) leads to a monotonic decrease in epidemic

severity, X_c . However, an interesting effect occurs for the optimal range of vaccination, z_c . Efficient vaccination ($v=0.9$) together with moderate incubation time and infection probability (e.g. $q=0.1$ with $p>0.5$) leads to non-monotonic behaviour of z_c as a function of the parameter characterizing the dispersal patterns of vectors, α (figure 5).

The non-monotonic behaviour is also robust with respect to the changes in the character of spread for vectors. Choosing equation (2.2) as a probability

Figure 5. (a) Sample symmetric α -stable densities for $\alpha=2.0$ (solid line), 1.5 (long dashed line), 1.0 (dashed line), 0.5 (dotted line) with $\sigma=1$ and (b) severity of the epidemics at the optimal strategy, X_c , and optimal radius of vaccination, z_c , as a function of the stability index α . Various symbols correspond to different infection probabilities (from top to bottom), $p=0.9$ (circles), $p=0.5$ (squares) and $p=0.1$ (triangles). The detection and treatment probabilities are $q=0.1$ and $v=0.9$. The number of vectors is (i) $\#Vec=150$, (ii) $\#Vec=500$.

density function for the increments in the random-jump process, we obtain qualitatively the same results as for the α -stable increments (figures 5 and 6). Decreasing the chances of very long-range jumps for vectors (increasing α or b) reduces the cost of epidemics in terms of both control and infection costs. Interestingly, the transition between epidemics that are very costly to control (large X_c) and those that can be stopped at a relatively low cost (small X_c) is quite sharp ($b \sim 0.8-1$). The transition region is also associated with a rapid increase in the optimal range of vaccination. This suggests that for the vectors that spend most of the time locally except for occasional very long jumps (small α for the α -stable distribution or small b for the exponential power distribution), local control measures are optimal but need to be applied repeatedly leading to a high cost of treatment. For vectors that spread locally (large α , large b), only few local control measures are necessary to stop the epidemics.

We also assessed the robustness of our results with respect to the selected key assumptions of the model. In particular, periodic boundary conditions were used in our simulations to avoid problems with truncating long-tail distributions of jumps. In this approach, periodic boundary conditions serve as an approximation to an effectively infinite system and the validity

of the results can therefore be tested by simulating systems within a wide range of sizes. The key result of our paper, the non-monotonic behaviour of z_c as a function of α (or b), is robust to the changes in system size ($N=10^4-10^5$) and numbers of vectors ($\#Vec=50-10^4$) and is unaffected if N and $\#Vec$ scale together. An increase in the system size with a fixed number of vectors decreases the relative severity of epidemics, X . This is caused by the fact that for larger systems it is harder for moving vectors to encounter a pathogen (and become contagious). The severity of the epidemics can be increased by increasing the number of vectors and/or by increasing the maximal number of individuals that can be infected by a single contagious (pathogen carrying) vector.

In our simulations, we assumed that the scale parameter is $\sigma=1$. Simulations were also performed for larger values of the scale parameter, up to $\sigma=10$, and the results were found to be consistent with the results for $\sigma=1$ for a broad range of the exponent α (and the parameter b for the exponential power distribution) and the number of vectors $\#Vec$. Therefore, it can be concluded that the interesting behaviour of the model studied is determined by the tails of the distributions for jump length, which in turn are determined by the stability index α (or by the exponent b ; figures 5 and 6).

Figure 6. (a) Sample exponential power distributions for $b=2.0$ (solid line), 1.5 (long dashed line), 1.0 (dashed line), 0.5 (dotted line) with $a=1$ and (b) severity of the epidemics at the optimal strategy, X_c , and optimal radius of vaccination, z_c , as a function of the exponent b . Various symbols correspond to different infection probabilities (from top to bottom), $p=0.9$ (circles), $p=0.5$ (squares) and $p=0.1$ (triangles). The detection and treatment probabilities are $q=0.1$ and $v=0.9$. The number of vectors is (i) $\#Vec=150$, (ii) $\#Vec=500$.

The central parts of distributions, which are determined by the scale parameter σ , play a different role from the tails of the distributions. In particular, an increase in the scale parameter σ increases the severity of epidemics for large values of the stability index α , making the non-monotonic effect less pronounced. Similar results can be obtained for the exponential power distribution by increasing the value of b .

4. SUMMARY AND CONCLUSIONS

A successful mathematical model for disease control should assist in identifying those processes that govern the size of the potential epidemics and propose efficient ways to limit the size of the outbreaks within practical and budget constraints. In this paper, we have looked at the potential of local treatment strategies to control the spread of diseases that are characterized by a mixture of local (individual-to-individual) and non-local interactions, mediated by vector movements. In contrast with most former epidemic control models, we do not assume that long-range links are fixed, but that vectors capable of transmitting a pathogen follow random flights. The choice of Lévy flights allows us not only to introduce dynamical—and therefore more realistic—network interactions, but also it provides an additional parameter, α , that we can use to model

different behaviours of vectors and particularly their dispersal range. At one extreme, small values of α mean that vectors can jump very large distances, making the model equivalent to a dynamical small-world model. For large values of α , the movement of vectors is very limited, approaching the local spread case. Figure 5 shows that the success of local control and the choice of the optimal strategy depend, in a non-trivial way, on the dispersal patterns of the vectors. Thus, we have shown that disease could be successfully contained using relatively small control neighbourhoods for both limiting cases, for vectors that disperse far (small α) and for those that are much more limited in their range (large α). We propose the following explanation for this behaviour. In our model, vectors explore a landscape that is dominated by local spread and local control. Vectors can only become infected (pathogen carrying) if they encounter infectious hosts. They can also infect only a limited number of susceptible hosts after becoming infective. The major long-distance role of vectors is in the creation of new foci by the transmission of the pathogen, with local multiplication being represented by the nearest-neighbour transmission. The creation of new foci is determined largely by the tails of the dispersal distribution, and is governed by the parameter α . For small α , the probability of large jumps by vectors is increased (relative to large α),

leading to a new focus that can be effectively removed by local control (small z_c) if the vector is infectious or to wasted epidemiological opportunities if the vector is not infectious. It follows that, for large jumps, it may be difficult for the vector to become infectious while new foci are efficiently controlled, so further reducing the scope for infection of vectors. In the opposite limit, for large α , vector dispersal is largely restricted to local spread, so reinforcing the local dynamics of the epidemic. Even though vectors are now more likely to become infectious, they are also confined to the regions that are saturated with infection and control so reducing the probability of encountering a susceptible host. It follows again that small z_c may be effective in controlling the spread of infection. For intermediate values of α , vectors can still perform long jumps so giving rise to new foci, but the frequency of shorter jumps ensures that the vectors are more likely to be infectious than for small α and so it is necessary to expand vaccination (i.e. z_c) to a larger zone. The same explanation holds in the situation when the dispersal pattern of vectors is described by the exponential power distribution which, as the α -stable density, is of the thick-tailed type.

We have focused on the inspection of the non-local dynamical spread of epidemics and its effects on the efficiency of local eradication strategies. In our approach, vectors perform random jumps with lengths determined by one of two thick-tailed distributions and the position after the jump determines their potential for spreading disease. The resulting spatial random process depends neither on whether the source or the target host is infected or not, nor on the distance covered. This stands in contrast to, for example, many Lévy search processes in which vectors might actively search for hosts and then stop. Consequently, we have used α -stable densities as an approximate way of simulating the self-similar character of dispersal patterns of vectors, rather than invoking a particular mechanism involved in creating this particular pattern. While Lévy flights and Lévy walks (Janicki & Weron 1994; Shlesinger *et al.* 1995) are believed to play a special role in random search strategies (Viswanathan *et al.* 1996, 1999; Bartumeus *et al.* 2002), there are some controversies (Edwards *et al.* 2007) as to whether or not the observed patterns of dispersing animals are really of the Lévy flight type. Some criticism centres on the infinite variance associated with the α -stable densities. In practical applications, there is always a cut-off due to a finite size or the number of dispersal steps that a vector can perform. For example, the finite number of steps in a realizable, finitely long Lévy flight leads to the situation when the effective distribution is restricted to the finite domain with the segment-length variance scaling with the number of jumps, M , as $M^{2/\alpha-1}$ (for $\alpha < 2$; Bouchaud & Georges 1990). In our approach, we do not impose explicitly any cut-off on the number or size of steps in the vector movement. The assumed shape of the jump length distribution however does not account for the results that are supported for the exponential power as well as the α -stable distribution. We have also checked our results by varying the system size and found that they were consistent across a wide

range of sizes, meaning that the observed phenomenon (of a non-monotonic change in z_c with dispersal parameter) is not related to our assumption about the boundary conditions.

We have used a very simple criterion for choosing an optimal strategy, comprising the total cost of an epidemic including the cost of infection, $R(\infty)$, and of treatment, $V(\infty)$. The choice of the functional form for the severity index has been dictated by its simplicity, ease of calculation and clear interpretation. We have used two main characteristics of an 'optimal' strategy. For a given set of parameters, we varied the radius of the control neighbourhood to obtain z_c corresponding to the lowest value of the severity index X . Both z_c (the optimal strategy) and the corresponding value of X_c (the total cost resulting from the optimal strategy) were used to characterize the strategy. An alternative approach, often taken in practice, is to compute the number of individuals that are prevented from being infected. However, in contrast to our choice of X , this approach does not take into account the costs associated with either infection or treatment.

The eradication of vectors is difficult in practice. However, our results suggest that the prevention of vector spread is crucial for an effective control of epidemics. Even though we have shown that purely local measures can eradicate disease that spreads by vectors, the cost of such control rapidly increases with the numbers of vectors. In contrast to purely local spread where a small increase in the size of the control neighbourhood can significantly reduce the severity (Dybiec *et al.* 2004), there appears to be no threshold associated with the density of vectors. Thus, we note that it is essential to control the vectors completely. The alternative strategy is to reduce the mobility of vectors and in particular the range of their spread (figure 2). For example, in order to control the outbreaks of rhizomania efficiently, it is imperative to reduce the transport of infested soil on farm machinery and to impose strict control over soil disposal at sugar beet factories (Stacey *et al.* 2004). The combined use of responsive and preventive actions can lead to a decrease in the severity of epidemics. The other key issue determining the efficiency of the optimal strategy is the real-time detection of new cases (detection probability, q) and real-time response to observed cases (vaccination probability, v). It should also be stressed that in this paper we have looked at the optimal local strategies with respect to the number of neighbours treated in response to the appearance of an individual showing symptoms. The optimal strategy in our understanding is identified as the one leading to the lowest combined cost, involving a cost of treatment (represented by $V(\infty)$) and the potential cost of not treating infected individuals (represented by $R(\infty)$). This cost, although minimal for a given set of parameters, can still be very high and other control strategies (including preventive blanket vaccination) can be more cost-effective (Kleczkowski *et al.* 2006).

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