

On the role of social clusters in the transmission of infectious diseases

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Abstract. *We introduce a spatial stochastic model for the spread of tuberculosis and HIV. We have three parameters: the size of the social cluster for each individual and the infection rates within and outside the social cluster. We show that when the infection rate from outside the cluster is low (this is presumably the case for tuberculosis and HIV) then an epidemic is possible only if the typical social cluster and the within infection rate are large enough. These results may be important in formulating new hypotheses for the transmission of TB and HIV.*

1. Introduction. It is suspected that some infectious diseases can only spread in populations where people are grouped in clusters in which individuals have repeated and sustained contacts. This is the case for HIV and also for tuberculosis. In this paper we will concentrate on tuberculosis. This disease has been known for thousands of years and is associated with the emergence of urban civilizations and hence high concentration of population (see Ayvazian (1993)). Tuberculosis is a major world wide concern. This is due to the appearance of multi drug resistant strains and the fact that the HIV pandemic favors the appearance of TB; see the report of the Open Society Institute (1999). Transmission of tuberculosis is thought to occur mostly through sustained and repeated contact with an infected person. Many such cases have been documented, see for instance Lincoln (1965) and Raffalli et al. (1996). Several cases were documented in schools where a teacher or a student have infected other students. In most cases infection occurred after sustained and repeated contacts. In the case of tuberculosis we may think of individuals with whom we spend several hours a week in the same room (car or bus) as part of our social cluster. Most documented tuberculosis cases come from within the infected individuals' own clusters. However, it is clear that the infection needs to have originated somewhere else. That is, 'casual' transmission must be possible as well. There are a few documented cases of those as well. For instance, Raffalli et al. (1996) mentions a Dutch rock band that had two infected musicians and which is thought to have infected hundreds of people with tuberculosis during their concerts.

In this paper we analyze mathematical models that incorporate social clusters. The idea of dividing a population in groups and consider different infection rates within and between groups goes back to at least Rushton and Mautner (1955). Sattenspiel (1989) reviews a number of such deterministic models and provides extensive references. We focus

AMS 1991 subject classifications: 60K35

Key words and phrases: infectious diseases, tuberculosis, sexually transmitted diseases, clusters, spatial stochastic model

on different questions than the papers above. In particular we are interested in understanding the role and interplay of several parameters in the appearance of an epidemic. We consider very simple minded models with only three parameters: the cluster size, the casual transmission rate and the within cluster transmission rate. We introduce a spatial stochastic model for which we have the following results. If the casual infection rate is high enough then there is a chance for an epidemic independently of the cluster size and the within cluster infection rate. In the case of tuberculosis the casual infection rate is probably not very high. For low or intermediate casual infection rate we show that an epidemic is possible only if the cluster size is large enough. However, in the case of low or intermediate casual infection rate, even if the cluster size is large enough the epidemic may occur only if the within cluster infection rate is large enough. We believe that these results shed some light about the interplay between the different parameters. In particular, the model suggests that given a low casual infection rate the cluster size and the within cluster infection rate are determinant.

2. The spatial stochastic model. Our model evolves on the square lattice \mathbf{Z}^d , typically $d = 2$ but our results hold in any dimension $d \geq 1$. At each site x in the square lattice \mathbf{Z}^d there is a cluster of N individuals. We denote our stochastic process by η_t . For a site x in \mathbf{Z}^d $\eta_t(x) = i$ means that there are i individuals (among the N individuals in the cluster at x) that are infected, i is an integer between 0 and N . Thus, each site has $N + 1$ possible states. We assume that an individual may get infected from within his cluster or from outside his cluster. The latter type of infection will also be called casual infection. The parameters λ and ϕ are the infection rates for infection from outside and from within the cluster, respectively. We use the notation $y \sim x$ to indicate that site y is one of the $2d$ nearest neighbors of site x . Assume that the model is in configuration η then the state at a given site x changes according to the following transition rates:

$$\begin{aligned}
 0 &\rightarrow 1 \text{ at rate } \lambda \sum_{y \sim x} \eta(y) \\
 i &\rightarrow i + 1 \text{ at rate } i\phi \text{ for } 1 \leq i \leq N - 1 \\
 i &\rightarrow 0 \text{ at rate } 1 \text{ for } 1 \leq i \leq N
 \end{aligned}$$

In words, the model depends on three parameters λ , ϕ and N . We start the process with a single infected individual in the cluster at the origin of \mathbf{Z}^d . Infected individuals may infect individuals in their cluster at rate ϕ . Infected individuals may also infect individuals in one of the $2d$ neighboring sites at rate λ provided the target site has no infected individuals yet. The idea is that once a site is infected infections within the site are a lot more likely than additional infections from the outside so we neglect the latter. This hypothesis simplifies the mathematics and the model. Moreover, we have checked that allowing casual infections in an already infected site does not change the qualitative behavior of the model. Finally, all the infected individuals in a cluster are simultaneously replaced by healthy individuals at rate 1. This applies in particular to places where there is a good tracking system of infectious diseases and once an infected individual is discovered its social cluster is rapidly tracked down.

We will start by stating a technical result that is crucial in our analysis.

Proposition 1. *Let η_t be our spatial stochastic process with parameters λ , ϕ and N and whose initial configuration is such that all the individuals are healthy except for one infected individual in the cluster at the origin of \mathbf{Z}^d . Let $|\eta_t|$ be the total number of infected individuals at time t . The probability*

$$P(|\eta_t| \geq 1, \text{ for all } t \geq 0)$$

is increasing in λ , ϕ and N .

That is, the probability that there will be infected individuals at all times increases the parameters λ , ϕ and N .

Given N and ϕ we define the critical value

$$\lambda_c(N, \phi) = \inf\{\lambda > 0 : P(|\eta_t| \geq 1, \text{ for all } t \geq 0) > 0\}.$$

By Proposition 1, one sees that if $\lambda < \lambda_c(N, \phi)$ then the infection eventually dies out in the population while if $\lambda > \lambda_c(N, \phi)$ there is a positive probability of an epidemic. That is, there is a positive probability that there will be infected individuals at all times.

The model with $N = 1$ is well known and is called the basic contact process. It is a model with just two states: 0 and 1 and it evolves as follows

$$\begin{aligned} 0 &\rightarrow 1 \text{ at rate } \lambda \sum_{y \sim x} \eta(y) \\ 1 &\rightarrow 0 \text{ at rate } 1 \end{aligned}$$

In the case $N = 1$ the value of ϕ is irrelevant, hence the critical value $\lambda_c(1, \phi)$ does not depend on ϕ and we will denote it by $\lambda_c(1)$. It is known that in any $d \geq 1$ the critical value $\lambda_c(1)$ is in $(\frac{1}{2d-1}, \frac{2}{d})$ but precise rigorous bounds are difficult to obtain. See, for instance, Liggett (1999) for more on the contact process. We now have enough notation to state our main result.

Theorem 1.

(a) *if $\lambda > \lambda_c(1)$ then there is positive probability for an epidemic for models with parameters (λ, N, ϕ) for all $N \geq 1$ and all $\phi \geq 0$.*

(b) *if $N\lambda < \lambda_c(1)$ there can be no epidemic for models with parameters (λ, N, ϕ) for all $\phi \geq 0$.*

(c) *if $N\lambda > \lambda_c(1)$ but $\lambda < \lambda_c(1)$ then there is a critical value ϕ_c , in $(0, \infty)$, depending on λ and N such that if $\phi < \phi_c$ there is no epidemic for the model with parameters (λ, N, ϕ) while if $\phi > \phi_c$ there is a positive probability for an epidemic for the model with parameters (λ, N, ϕ) .*

Thus, (a) tells us that if the casual rate λ is large enough there is a positive probability for an epidemic independently of the cluster size N and the cluster infection rate ϕ . From (b) and (c) we see that if $\lambda < \lambda_c(1)$ there is critical cluster size: if $N > \lambda_c(1)/\lambda$ an epidemic is possible while if $N < \lambda_c(1)/\lambda$ no epidemic can take place. Finally, when $\lambda < \lambda_c(1)$ and N is above the critical cluster size $\lambda_c(1)/\lambda$ then an epidemic can happen only if the within cluster infection rate ϕ is large enough.

3. The mean field model

We now introduce the mean field model corresponding to our spatial stochastic model. Let u_i be the density of individuals whose cluster has i infected individuals for $0 \leq i \leq N$. In particular, $u_0 + u_1 + \dots + u_N = 1$. Assuming that the contacts are through homogeneous mixing we get the following mean field model.

$$\begin{aligned} u'_0 &= \sum_{i=1}^N u_i - \sum_{i=1}^N i\lambda u_i u_0 \\ u'_1 &= \lambda u_1 u_0 + 2\lambda u_2 u_0 + 3\lambda u_3 u_0 + \dots + N\lambda u_N u_0 - u_1 - \phi u_1 \\ u'_i &= (i-1)\phi u_{i-1} - u_i - i\phi u_i \text{ for } 2 \leq i \leq N-1 \\ u'_N &= (N-1)\phi u_{N-1} - u_N \end{aligned}$$

We have the disease free equilibrium $u_0 = 1$ and $u_i = 0$ for all $i \geq 1$. The first part of our analysis involves only the first differential equation. First note that

$$u'_0 = \sum_{i=1}^N u_i - \sum_{i=1}^N i\lambda u_i u_0 \leq \sum_{i=1}^N u_i - \sum_{i=1}^N \lambda u_i u_0 = 1 - u_0 - \lambda u_0(1 - u_0) = (1 - u_0)(1 - \lambda u_0).$$

Note that if $\lambda > 1$ and u_0 is in $(1/\lambda, 1)$ then $u'_0 < 0$. That is, the disease free equilibrium is not stable and an epidemic is possible for all values of N and ϕ .

Next, observe that

$$u'_0 = \sum_{i=1}^N u_i - \sum_{i=1}^N i\lambda u_i u_0 \geq \sum_{i=1}^N u_i - \sum_{i=1}^N N\lambda u_i u_0 = (1 - u_0)(1 - N\lambda u_0).$$

If $\lambda N < 1$ and $u_0 < 1$ then $u'_0 > 0$. That is, the disease free equilibrium is stable and no epidemic can take place.

The analysis above shows that the behavior of the mean-field model is qualitatively the same as the behavior the spatial stochastic model as described in Theorem 1 (a) and (b).

We now turn to the case where $N\lambda > 1$. From the system of differential equations above we see that if (u_0, u_1, \dots, u_N) is an equilibrium then

$$u_i = \frac{(i-1)\phi}{i\phi + 1} u_{i-1} \text{ for } 2 \leq i \leq N-1.$$

Hence,

$$(1) \quad u_i = \frac{(i-1)!\phi^{i-1}}{(i\phi + 1)((i-1)\phi + 1) \dots (2\phi + 1)} u_1 \text{ for } 2 \leq i \leq N-1.$$

From the last equation in the system of differential equations above we have

$$u_N = (N-1)\phi u_{N-1}.$$

Letting $i = N - 1$ in (1) we get

$$u_N = \frac{(N - 1)! \phi^{N-1}}{((N - 1)\phi + 1) \dots (2\phi + 1)} u_1.$$

We now use the last expression and (1) in the second equation of the system of differential equations above to get

$$u_0 = \frac{1 + \phi}{\lambda} \frac{1}{1 + \sum_{i=2}^{N-1} \frac{i! \phi^{i-1}}{(i\phi+1)\dots(2\phi+1)} + \frac{N! \phi^{N-1}}{((N-1)\phi+1)\dots(2\phi+1)}}.$$

As we let ϕ go to infinity it is not difficult to see that u_0 converges to $\frac{1}{\lambda N}$. Thus, if $\lambda N > 1$ we can pick ϕ so that an equilibrium giving positive density to the infected states is possible. In other words, we have for the mean-field model a result similar to what we have for the spatial stochastic model in Theorem 1 (c).

4. Discussion. There has been interest in the recent literature on the relative importance of the cluster size, the within cluster infection rate and the casual infection rate in the transmission of infectious diseases, in particular for tuberculosis, see Aparicio et al. (2000) and Raffalli et al. (1996). In this paper we obtain the following results for a spatial stochastic model. If the casual infection rate is above a certain threshold denoted by $\lambda_c(1)$ then there is a chance for an epidemic even for a cluster size of 1 and a within cluster infection rate of 0. If the casual infection rate is below $\lambda_c(1)$ then an epidemic is possible only if the cluster size is large enough. Even if the cluster size is large enough then an epidemic is possible only if the within cluster infection rate is large enough when the casual infection rate is below $\lambda_c(1)$. Note that some of the results just described (in particular, the existence of a critical cluster size) were already implicit for the deterministic model of Aparicio et al. (2000).

The main example we have in mind is the transmission of tuberculosis. However, we believe that this model is also interesting in the context of sexually transmitted diseases. It has been conjectured, for instance, that the transmission of HIV in the gay communities of the U.S.A is caused by a core of very sexually active individuals that are customers of bathhouses, see Thompson (1984) and Rotello (1997) for instance. One may think that such an individual is part of a huge cluster and even if the casual infection rate (that is, the infection rate from a very active cluster into another cluster) is low our results show that an epidemic may occur provided the within cluster infection rate is high enough. Of course, in reality all clusters have not the same size and it would be interesting to analyze a model with variable (maybe random) cluster size to see whether our results still hold.

Our model is too simple to yield useful quantitative results. Transmission of infectious diseases depend on many more than 3 parameters. Transmission of tuberculosis is particularly challenging. It is believed that the vast majority of infected individuals never develop the disease and are never infectious. However, some do and some do more than once and the discussion is still on to decide whether the reinfection is usually exogenous or endogenous, see Feng et al. (2000) and Styblo (1991). These questions are now even more important with many people simultaneously infected by HIV and TB since HIV infected

individuals are a lot more likely to develop TB. Models like ours are useful in getting a qualitative understanding of the basic transmission mechanisms. We believe that a lot must be understood about these basic transmission mechanisms before one can confidently look at models with more parameters.

In our spatial model there is very little mixing (individuals interact with their nearest neighbors only) while in the mean-field model every individual mixes with everybody else. The fact that the qualitative behavior is the same for both models shows that our results are probably robust and do not depend on the specific mixing conditions of the models. In particular, we expect finite range spatial models to behave like our nearest neighbor spatial model.

5. Proofs

Proof of Proposition 1.

It is useful to have some idea on how to construct our process η_t . The construction we have in mind here is based on appropriate families of Poisson processes. Each site has Poisson processes corresponding to the different transition rates. For instance, each site x , for each integer i between 1 and $N - 1$, has a Poisson process with rate $i\phi$. At each occurrence of the Poisson process with rate $i\phi$, if site x is in state i then it goes to state $i + 1$. By defining appropriate families of Poisson processes we get a process with the prescribed transition rates. This type of construction is usually referred to as the graphical construction. For more details see p 32 in Liggett (1999), for instance. One of the nicest features of this construction is that it allows us to construct several processes on the same probability space. For instance, if we are interested in comparing two epidemic models η_t^1 with $\phi = \phi_1$ and η_t^2 with $\phi_2 > \phi_1$, then we may define at each site Poisson processes with rates $i\phi_2$ to construct η_t^2 . We may use the same Poisson processes to get Poisson processes with rates $i\phi_1$ by doing the following. Every time there is an occurrence for η_t^2 it is an occurrence for η_t^1 with probability ϕ_1/ϕ_2 . This gives us a process η_t^1 with the appropriate rates. Moreover, one sees that by constructing the two processes in the same probability space the process with ϕ_1 must have less infected individuals at every site than the process with ϕ_2 . The same type of idea may be used to compare processes with different N and different λ . One only needs to check that no transition can make the process with the lower parameter have more infected individuals than the process with the higher parameter at any given site, provided one starts with the same configuration for both processes. A crucial feature of the model that ensures this monotonicity property is the constant transition rate for $i \rightarrow 0$ for all states $i \geq 1$ (actually, any transition rate which is decreasing in i would work). This completes the proof of Proposition 1.

Proof of Theorem 1. (a)

Due to Proposition 1 the critical parameter $\lambda_c(N, \phi)$ is decreasing in N and ϕ . Thus,

$$\lambda_c(N, \phi) \leq \lambda_c(1, \phi) = \lambda_c(1).$$

Since we assume that $\lambda > \lambda_c(1)$ we have $\lambda > \lambda_c(N, \phi)$ for any N and ϕ . By definition of $\lambda_c(N, \phi)$ we have that $P(|\eta_t| \geq 1, \text{ for all } t \geq 0) > 0$. Hence, there is a positive probability

of an epidemic for all models with parameters (λ, N, ϕ) such that $\lambda > \lambda_c(1)$. This completes the proof of Theorem 1 (a).

Proof of Theorem 1 (b)

Consider the model with $\phi = \infty$. In this case as soon there is one infected in the cluster all the cluster is infected. So each site has only two possible states: 0 and N . The transition rates are given by:

$$0 \rightarrow N \text{ at rate } \lambda \sum_{y \sim x} \eta(y)$$

$$N \rightarrow 0 \text{ at rate } 1$$

Observe that $\eta(y)$ can only be 0 or N . So the transition from 0 to N occurs at λN times the number of nearest sites that are infected. Therefore, the model above is a contact process with birth rate λN . If $\lambda N < \lambda_c(1)$ this contact process dies out. By Proposition 1 the model with parameters (λ, N, ∞) has more infected individuals than the model with parameters (λ, N, ϕ) for any $\phi \geq 0$. Since there can be no epidemic for the first model the same is true for the second model. This completes the proof of Theorem 1 (b).

Sketch of the proof of Theorem 1 (c)

We are given λ and N such that $N\lambda > \lambda_c(1)$ and $\lambda < \lambda_c(1)$. We will proceed in two steps. Our first step will be to show that there is $\phi_1 > 0$ such that if $\phi < \phi_1$ then no epidemic can take place for the system with (λ, N, ϕ) . In our second step we will show that there is $\phi_2 < \infty$ such if $\phi > \phi_2$ then an epidemic may take place for the system with (λ, N, ϕ) . Due to the monotonicity of this process this will imply the existence of a critical value $\phi_c(\lambda, N)$ in $[\phi_1, \phi_2]$ such that if $\phi < \phi_c$ no epidemic can happen while if $\phi > \phi_c$ an epidemic may happen.

This proof is done under the assumption $d = 2$, in order to avoid more cumbersome notation.

We start the first step of the proof by defining two space–time regions:

$$\mathcal{A} = [-2L, 2L]^2 \times [0, 2L], \quad \mathcal{B} = [-L, L]^2 \times [L, 2L]$$

where L is an integer to be chosen later. Define \mathcal{C} to be part of the ‘boundary’ of the box \mathcal{A} :

$$\mathcal{C} = \left\{ (m, n, t) \in \mathcal{A} : |m| = 2L \text{ or } |n| = 2L \text{ or } t = 0 \right\}$$

We will compare the spatial stochastic model η_t to a certain dependent percolation process on the set $\mathcal{L} = \mathbf{Z}^2 \times \mathbf{Z}_+$, where $\mathbf{Z}_+ = \{0, 1, 2, \dots\}$. We say that the site (k, m, n) in \mathcal{L} is *wet* if there exist no infected sites in the (smaller) box $(kL, mL, nL) + \mathcal{B}$ *regardless of the states of sites in the boundary $(kL, mL, nL) + \mathcal{C}$* . In other words, we want no infection path going from the boundary of the larger box into the smaller box. Note that the event $\{(k, m, n) \text{ is wet}\}$ depends only on the existence (or not) of paths of infection within \mathcal{A} . We require this uniformity on the states of the boundary in order to ensure that the percolation process in \mathcal{L} , although dependent, has an interaction with only finite range. Sites which are not wet are called *dry*.

Given $\lambda < \lambda_c(1)$ we will show that there is an integer L and a real number ϕ_1 such that

$$P((k, m, n) \text{ is wet}) \geq 1 - \epsilon \text{ if } \phi < \phi_1.$$

We start by showing the above property when $\phi = 0$. Then, using a continuity argument, we will deduce that the inequality remains true for small ϕ . By translation-invariance, it suffices to consider the site $(0, 0, 0) \in \mathcal{L}$.

Note that if $\phi = 0$ then each site may be in only 2 states: 0 and 1. The process η_t becomes a contact process with birth rate λ . If there is a 1 in \mathcal{B} it must have originated in $[-2L, 2L]^2 \times 0$ or at a later time from one of the sides of the box \mathcal{A} . In the former case the infection must have survived at least L before it reaches \mathcal{B} while in the latter case it must have a radius of at least L . Note that since $\lambda < \lambda_c(1)$ η_t is a subcritical contact process if $\phi = 0$. Bezuidenhout and Grimmett (1991) have shown, for the subcritical contact process, that the probability that an infection lasts L or more is less than $Ce^{-\gamma L}$ where C and γ are strictly positive constants and that the probability that an infection has a radius of L is less than $Ce^{-\gamma L}$. Therefore, we have

$$P((0, 0, 0) \text{ is wet}) \geq 1 - 8L(4L + 1)e^{-\gamma L} - (4L + 1)^2 e^{-\gamma L}.$$

By taking L large enough the probability above may be made larger than $1 - \epsilon/2$. We have

$$P((0, 0, 0) \text{ is wet}) \geq 1 - \epsilon/2 \text{ if } \phi = 0.$$

Since \mathcal{A} is a finite box there is $\phi_1 > 0$ such that with probability at least $1 - \epsilon/2$ there are no occurrences of Poisson processes with rate ϕ_1 inside \mathcal{A} . Therefore, we get

$$P((0, 0, 0) \text{ is wet}) \geq 1 - \epsilon \text{ if } \phi < \phi_1.$$

This shows that even if all sites on the boundary of \mathcal{A} are in state N by taking ϕ sufficiently small η_t will be a subcritical contact process inside \mathcal{A} and with high probability there will be no infected site in the smaller box \mathcal{B} .

We may define a percolation process on \mathcal{L} for which the probability that a given site be wet is $1 - \epsilon$. Since $\epsilon > 0$ can be taken arbitrarily small, it is not difficult to see that the probability of a path of length n of dry sites in the percolation process decreases exponentially fast with n , see (8.2) in Berg et al. (1998). The crucial point is that an infected site for η_t corresponds with a path of dry sites in the percolation process. By using this comparison it is possible to show that for any fixed site, after a finite random time, there will never be an infection at that site if $\phi < \phi_1$ for η_t . See the proof of Theorem 4.4 in Berg et al. (1998) for more details. This completes the first step of our proof of Theorem 1 (c).

We now deal with the second step of the proof. In this step too, we will compare our spatial stochastic model to a simple oriented percolation model. Let e_1 be the vector $(1, 0)$ and

$$\begin{aligned} \mathcal{L} &= \{(m, n) \in \mathbf{Z}^2 : m + n \text{ is even}\} \\ B &= (-4L, 4L)^2 \times [0, T] \quad B_{m,n} = (2mLe_1, nT) + B \end{aligned}$$

$$I = [-L, L]^2 \quad I_m = 2mLe_1 + I$$

$$C = [-k, k]^2$$

where L and T are parameters to be chosen later and k is the largest integer smaller than \sqrt{L} . We declare $(m, n) \in \mathcal{L}$ wet if the process restricted to $B_{m,n}$ and starting with a translate of C in I_m full of infected sites at time nT , is such that there is a translate of C in I_{m-1} and a translate of C in I_{m+1} both full of infected sites at time $(n+1)T$. In other words, we declare (m, m) wet if a square of side $2k+1$ full of infected sites in I_m at time nT is reproduced at time $(n+1)T$ in both I_{m-1} and I_{m+1} . We will now show that by choosing L and T sufficiently large the probability of a site (m, n) being wet can be made arbitrarily close to 1. By translation invariance, it is enough to show this for the site $(0,0)$. We start by setting $\phi = \infty$ in the box B . As noted above in the proof of Theorem 1 (b) the process η_t with $\phi = \infty$ is a contact process with birth rate λN . Each site may be in one of 2 states: 0 and N . Since we are assuming that $\lambda N > \lambda_c(1)$, this is a supercritical contact process. By taking k and therefore L large enough one can show that the probability for a supercritical contact process with k initial infected sites to survive forever can be made arbitrarily close to 1. See for instance (1.9) in p 36 in Liggett (1999). The Shape Theorem (see p 128 in Liggett (1999)) states that a supercritical contact process that does not die out spreads out at a constant speed. Thus, if the sites in state N do not die out, they should get to the boundary of B by time aL , where $a > 0$ depends on the linear speed of the infection. Moreover, the Shape Theorem also guarantees that the infected sites are distributed according to the upper stationary distribution of the contact process. Since this distribution is ergodic (see Proposition 2.16 p 143 in Liggett (1985)) there will be translates of C in I_1 and I_{-1} at time $L + aL$ with high probability. This shows that for any $\epsilon > 0$ we may pick $T = L + aL$ and L large enough so that

$$P((0,0) \text{ is wet}) > 1 - \epsilon \text{ for } \phi = \infty.$$

Since B is a finite space time box, we may pick ϕ_2 large enough so that

$$P((0,0) \text{ is wet}) > 1 - 2\epsilon \text{ for } \phi > \phi_2.$$

We may take $\epsilon > 0$ small enough so that there is an infinite cluster of wet sites in \mathcal{L} with positive probability. If we start the process η_t with at least one infected individual then there is a positive probability of having a square C of sites in state N somewhere at time 1. We then start the construction above and there is a positive probability of an infinite cluster of wet sites. This in turn guarantees that there will be infected sites at all times and completes the proof of our second step and of Theorem 1 (c).

Acknowledgements. We thank two anonymous referees for catching a mistake in a previous version of this paper that had far reaching consequences . We thank two other referees for their suggestions.

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