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A threshold area ratio of organic to conventional agriculture causes recurrent pathogen outbreaks in organic agriculture

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ABSTRACT

Conventional agriculture uses herbicides, pesticides, and chemical fertilizers that have the potential to pollute the surrounding land, air and water. Organic agriculture tries to avoid using these and promotes an environmentally friendly approach to agriculture. Instead of relying on herbicides, pesticides and chemical fertilizers, organic agriculture promotes a whole system approach to managing weeds, pests and nutrients, while regulating permitted amendments. In this paper, we consider the effect of increasing the total area of agricultural land under organic practices, against a background of conventional agriculture. We hypothesized that at a regional scale, organic agriculture plots benefit from existing in a background of conventional agriculture, that maintains low levels of pathogens through pesticide applications. We model pathogen dispersal with a diffusive logistic equation in which the growth/death rate is spatially heterogeneous. We find that if the ratio of the organic plots to conventional plots remains below a certain threshold l_c, the pest population is kept small. Above this threshold, the pest population in the organic plots grows rapidly. In this case, the area in organic agriculture will act as a source of pest to the surrounding region, and will always infect organic plots as they become more closely spaced. Repeated localized epidemics of pest outbreaks threaten global food security by reducing crop yields and increasing price volatility. We recommend that regional estimates of this threshold are necessary to manage the growth of organic agriculture region by region.

1. Introduction

A rapidly growing agricultural system is organic agriculture. This system has its origins in concerns over the accumulation of synthetic chemicals and the use of synthetic fertilizers in conventional agriculture, with harmful consequences to the environment (Trewavas, 2001; Ramesh et al., 2005; Carvalho, 2006; Hobbs, 2007; Badgley et al., 2007). Organic farming (Lampkin, 2007; Tamm, 2001) is gaining popularity in Europe, south America, north America, Japan and Australia among consumers and producers (OECD, 2008; Yussefi, 2004). These studies report an increase of 60% in global acreage under organic agriculture between 2000 and 2004, and an average annual growth rate of about 20%, although it reaches 50% in Turkey (Sayin et al., 2004). Initially supplied by numerous small operations, more recently large suppliers to international distributors have engaged in providing organic-labeled produce (Raynolds, 2004; Brand, 2006). Produce with an organic label meet the criteria that certify it was produced without applications of pesticides, herbicides, chemical fertilizers and free of genetically modified organisms, as governed by national or regional legislation. Consumers are drawn to these produce for two principal reasons. First, health conscious consumers perceive organic produce to be healthier (Woese et al., 1997; Yiridoe et al., 2005) and safer as they do not contain trace amounts of chemicals that are potentially hazardous to human health (Barceló and Hennion, 1997; Rivas et al., 1997; Sharpe, 1999). Second, organic produce are believed to be environmentally friendly, because organic agriculture avoids using chemicals that are perceived to be environmentally harmful to soil, freshwater, ground water and the air (Carvalho et al., 1997; Taylor et al., 2003; Chernyak et al., 1996). As many of these chemicals are not immediately biodegradable they can persist in the environment and bioaccumulate through the food web into many non-target species, including humans (Nhan et al., 1999; Carvalho, 2005). Two general concerns with organic agriculture are regularly raised. The first concerns food safety issues, the second is food security issues (Carvalho, 2006; Perfecto and Badgley, 2007). One would assume organic produce, having been produced without the application of pesticides would be safe to consume. However, there are regular recent cases of consumer illnesses and cross-border product recalls caused by contaminated organic produce, (US-FDA, accessed 31st March 2010). Thus, it is no longer possible to assume that organic produce are de facto safer than conventional produce. More significantly, the concern with food security is more difficult to address. Compared to

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conventional output, organic agriculture tends to produce statistically significant lower yields under intensive agriculture, due to decreased germination success and loss to disease, among other issues (Borlaug, 2000; Trewavas, 2002; Smil, 2000; Green et al., 2004). Can organically produced crops provide food security, while being more susceptible to yield fluctuations caused by pathogens and pests (Trewavas, 2001; Perfecto and Badgley, 2007)? The issue continues to be debated but it is clear that organic agriculture is more expensive (Ramesh et al., 2005). This becomes a more serious issue as more land under conventional agriculture is brought into organic production (Badgley et al., 2007).

In an agricultural landscape, organic farms operate against a background of conventional agriculture that maintains pathogen load and pest levels low. Intuitively, as the number of plots in organic agriculture increases they become closer together. Thus, as the number of plots in organic agriculture continues to increase, the number of refugia for pests and pathogens is postulated to increase. Therefore, it is worth considering whether disease outbreaks could become more frequent as the ratio of total agricultural land under organic farming increases relative to the area under conventional agriculture. We hypothesize that, in any given region, organic agriculture benefits from the conventional agriculture landscape which provides a low pathogen background. We further hypothesize that as organic agriculture plots become more frequent in the landscape, the likelihood of pathogen outbreaks increases. We addressed this question mathematically in one and two dimensions. The results show the existence of a bifurcation point above a threshold ratio of organic to conventionally farmed area, above which infections will always occur.

2. Theory and calculations

Typically, different mathematical modeling approaches are used to model agricultural pathogen dispersal at different scales (Maanen and Xu, 2003; Kuparinen et al., 2007; Viljanen-Rollinson et al., 2007). Many of the pests we wish to consider are spread by winds which have a prevalent direction. However, if we consider appropriate time and spatial scales, we can consider the direction of the wind to be close to uniformly random. On this scale, random diffusion is a reasonable assumption (Maanen and Xu, 2003; Kuparinen et al., 2007; Viljanen-Rollinson et al., 2007). For regional or descriptive models, one could include the effect of a dominant wind direction by adding an advective term. We focused on wind dispersed foliar pathogens and assumed that in organic plots pathogen control was less effective than in conventional plots.

In constructing the model, we make the following assumptions:

1. At the regional scale pathogens spread in a manner consistent with random diffusion.
2. In the absence of pesticide, the pathogen population is non-zero and can be modeled by a logistic growth model.
3. The addition of pesticide causes negative growth rate of pathogens and at sufficient concentration, it causes the population to die out.
4. In organic plots, positive growth rate of pathogens occurs because plot management does not independently prevent pathogen outbreak.

We now define the variables and parameters used in the model:

- p — The fraction of the maximal pest population
- D — The diffusivity of the pest
- ν — The rate of pest growth in the absence of pesticides
- μ — The death rate due to the presence of pesticide. We assume $\mu > \nu$ (assumption 3 of our model)
- l — The size of a farm plot devoted to organic practices
- and L — Size of the entire area

In one dimension, the model is then given by,

$$\frac{\partial p}{\partial t} = D\frac{\partial^2 p}{\partial x^2} + \nu p(1-p) - h_l(x)\mu p, \; 0 < x < L, \tag{1}$$

$$\frac{\partial p}{\partial x}(0) = \frac{\partial p}{\partial x}(L) = 0 \tag{2}$$

where,

$$h_l(x) = \begin{cases} 0, 0 < x < l \\ 1, l < x < L \end{cases}. \tag{3}$$

Here, $\frac{\partial^2 p}{\partial x^2}$ is the second partial derivative of p with respect to x and $\frac{\partial p}{\partial t}$ is the partial derivative of p with respect to time. This boundary condition allows us to consider infinite domains in which organic and conventional farms are interspersed periodically. By varying the value of l, we can examine the effects of varying the percentage of farmland devoted to organic methods. Note that related models were also considered in other contexts (Ludwig et al., 1979; Shigesada et al., 1986; Cantrel and Cosner, 1989; Berestycki et al., 2005) among others. Refer to the Appendix for the mathematical proof.

3. Results

The main result is that if l is sufficiently small, the pest population becomes extinct throughout the organic and conventional farms. However as the total area under organic farming l is increased, there exists a critical domain size of l_c such that if $l > l_c$ then there will be growth of pest in the organic farm which will then act as a source of pest to the neighboring agricultural region. In this case, the density of the pest is most concentrated within the organic area and decreases away from it (see Appendix A). The value of l_c is the smallest positive root of

$$\propto \tan(\propto l_c) = \beta \tanh(\beta(L-l_c)) \tag{4}$$

where $\alpha = \sqrt{v/D}$ and $\beta = \sqrt{(u-v)/D}$.

In two dimensions the result is similar: as shown in the Appendix, pathogen outbreak will always occur if the area under organic farming is sufficiently large. For the special case when the organic area has a circular shape of radius l inside a larger conventionally farmed area of radius L, the critical threshold value l_c is given by

$$\alpha\frac{J_1(\alpha l_c)}{J_0(\alpha l_c)} = \beta\frac{K_1(\beta(l_c-L))}{K_0(\beta(l_c-L))}, \tag{5}$$

where J_i, K_i are Bessel functions of order i (Abramowitz and Stegun, 1964).

Mathematically, this behavior corresponds to a bifurcation of the zero steady state as l is increased past l_c. An example of this phenomenon is illustrated in Fig. 1 for $D=1$, $\mu=4$, $\nu=1$ and $L=10$. By numerically solving Eq. (4) we then find that $l_c=1.047$. Fig. 1(a) shows $p(0)$ as a function of l. Note that a pest outbreak solution $p>0$ bifurcates from the point $l=l_c$ as l is increased. The corresponding equilibrium profiles $p(x)$ are illustrated (Fig. 1b). Since there is a very large range of pest diffusivity in one direction, expressed from 10 m day^{-1} to 10,000 m day^{-1}, we plot the critical organic plot area l_c versus the logarithm of pest diffusivity (Fig. 2). From Eq. (4), it is clear that as $D\rightarrow\infty, l_c\rightarrow L\frac{\mu}{\mu+\nu}$. Therefore, from this simple relationship, one can estimate the critical ratio of organic to conventional farmed area for rapidly dispersing pathogens. A diversity of hypothetical scenarios demonstrates the critical threshold of organic to conventional area varies depending on the parameters (Table 1). In addition, this model can be refined further using local or regional scale models that contain more parameters if regional data exists.

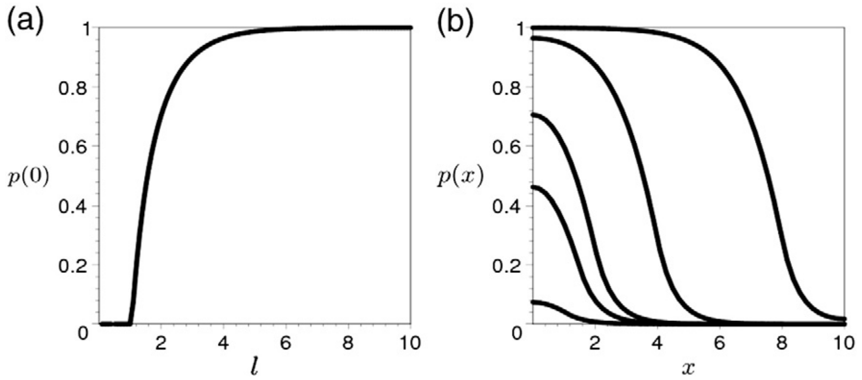

Fig. 1. (a) Plot of the fraction of the farmland dedicated to organic farming against fraction of maximal pest population abundance with $D=1$, $L=10$, $v=1$, $\mu=4$ and l as given. If less than 10% of the farmland is dedicated to organic practices the pest population abundance remains minimal. Past this limit, the pest population grows rapidly to maximal. This agrees with the prediction given by Eq. (4) of $l_c=1.047$. (b) The graph of the pest population profile for various values of l. From bottom to top, $l=1.1,1.5,2,4,8$. Other parameters are as in (a). Symbols: D is pest diffusivity, L is the total farmed area, l is the area under organic farming, l_c is the critical area of organic to conventional farmed area as described in Eqs. (4) and (5), v is the rate of pest growth in the absence of pesticide, and μ is the death rate due to pesticides.

Our first hypothesis that organic agriculture benefits from the background of conventional agriculture in the landscape is supported by the model calculations. When there are few organic plots, or when the total area of organic to conventional agriculture is low, pathogen outbreaks occur but do not spread into an epidemic. Our second hypothesis is also supported, that as organic plots become more frequent or when the total area of organic to conventional agriculture is above a threshold, pathogen outbreaks are more likely to occur and spread into an epidemic to all organic plots.

4. Discussion

4.1. Interpretation of the model

The results show that above a threshold ratio of organic to conventionally farmed land area, pathogen outbreaks will always occur. This is possible because each organic plot acts as a refugia for pathogens, against the dominant land area under conventional agriculture. When organic plots are sparse and far apart from each other, infections will tend to be mostly through autodeposition (same field infections). But as the abundance of plots increase the ratio of autodeposition to allodeposition (new field infections across distance) decreases. The conventional plots, although regularly infected from conventional and organic plots, reduce or inhibit pathogen spread and viability through the application of pesticides. However, this increases the cost of production for conventional fields, that have to apply pesticides more often as the abundance of organic plots increase in their midst to counter pathogen spread from the untreated organic plots.

4.2. Pathogen control

The National List of Allowed and Permissible Substances in organic agriculture (Electronic Code of Federal Regulation – Agriculture – Part 205 National Organic Program) lists and prescribes chemicals usage for pathogen control. A search of the effectiveness of pesticides used in organic agriculture demonstrated a dearth of published data in refereed journals. Instead the literature relies on a combination of mild pathogen level management (disease resistant plant varieties, crop rotations, integrated pest management, treatments from pre-industrial agriculture period, manual removal of diseased plants) (Barker, 2010). In the pre-industrial period crops were regularly devastated by epidemics that swept through Europe with consequences on human nutrition and food security (for example Ordishe, 1987; Millardet, 1885; Gennadios, 1889).

4.3. Pathogen dispersal distance

Plant pathogens tend to be viruses, bacteria, fungal spores or fungal hyphae, nematodes or arthropods, thus representing a range of size and weight of the dispersal particle. Dispersal occurs through rain drops, wind, arthropod dispersal, carried by a vector such as an insect or through an infected host (seed, pollen, insect) (Viljanen-Rollinson et al., 2007; Chen and Feng, 2006; Togashi and Jikumaru, 2007) as well as anthropogenic transport. Dispersal of spore, pollen, seed or arthropods is usually described by diffusion based analytical models (Cantrell and Cosner, 1993), persistent random walk (Bicout and Sache, 2003), logistic and Gompertz models (Maanen and Xu, 2003; Shaw et al., 2006; Pethybridge et al., 2005; Bergua et al., 2008), Lagrange puff models with Gaussian or non-Gaussian turbulence as in atmospheric pollution studies (Pfender et al., 2006; Kuparinen et al., 2007). Epidemic progress can be modeled with general disease models developed from data (Maanen and Xu, 2003). Field data confirm that spore dispersal and deposition events are randomly distributed because spores arrive both from allodeposition, autodeposition and accumulation in the field from previous years (Bicout and Sache, 2003; Zhang et al., 2005; Pfender et al., 2006; Pethybridge et al., 2005; Schmale et al., 2005; Roslin et al., 2007).

The dispersal distance is affected by weight of the infective particle and the time scale, so that with longer time duration the particles reach further but become less abundant with distance (Kuparinen et al., 2007). Thus the infection severity decreases with distance as particle density decreases. The epidemic progress is affected, in addition to weight, time scale and infection severity, by day-to-day weather changes, temperature, crop growth phase, pathogen life history and landscape characteristics (Pivonia and Yang, 2006). Regional disease progress models can be constructed for the most important pathogens, with more or less parameters, to predict and manage epidemics locally.

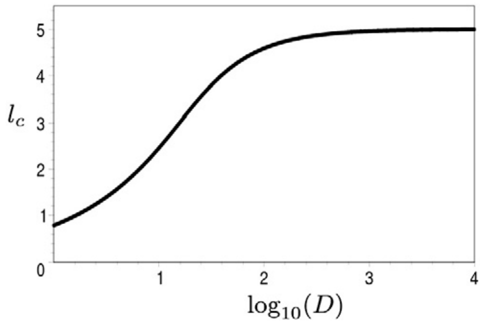

Fig. 2. Plot of l_c versus log$_{10}$ for the case $\mu=2$, $v=1$ and $L=10$. See Fig. 1 for symbol legend.

Table 1
Critical values l_c for various hypothetical pathogen parameter values in one dimension. Here the total length of the agriculture area is 1, so the critical length can be thought of as a fraction of the area of organic to conventional agriculture. The model was run for a range of diffusivity representing length per unit time, with three values of net growth rate and two values of net death rate from the logistic equation as individuals per unit time. See Fig. 1 for symbols legend.

Diffusivity	Growth rate	Death rate	Calculated critical threshold l_c
D = 10	$\nu = 0.1$	$\mu = 0.2$	= 0.0906
		$\mu = 0.4$	= 0.2296
	$\nu = 1$	$\mu = 2$	= 0.2364
		$\mu = 4$	= 0.4782
	$\nu = 10$	$\mu = 15$	= 0.3097
		$\mu = 25$	= 0.5503
D = 100	$\nu = 0.1$	$\mu = 0.2$	= 0.0306
		$\mu = 0.4$	= 0.0865
	$\nu = 1$	$\mu = 2$	= 0.0906
		$\mu = 4$	= 0.2296
	$\nu = 10$	$\mu = 15$	= 0.1350
		$\mu = 25$	= 0.3160
D = 1000	$\nu = 0.1$	$\mu = 0.2$	= 0.0099
		$\mu = 0.4$	= 0.0291
	$\nu = 1$	$\mu = 2$	= 0.0306
		$\mu = 4$	= 0.0865
	$\nu = 10$	$\mu = 15$	= 0.0475
		$\mu = 30$	= 0.1300

From Table 1, one can estimate which parameters are likely to affect pathogen growth the most, considering regional wind patterns and pathogen species. However, an unpredictable stochastic element exists through long-distance wind dispersal. Vertical wind puffs carry a fraction of infective particles into the upper parts of the atmospheric boundary layer (mixed layer). This atmospheric pathway is responsible for long-distance and inter-continental dispersal hundreds to thousands of kilometers from the source (Viljanen-Rollinson et al., 2007; Zeng and Luo, 2006; Brown and Hovmøller, 2002; Isard et al., 2005). Increased frequency of outbreaks in one region can therefore cause repeated long-distance infections through air currents as demonstrated in these papers.

Regionally, organic plot aggregation or total area can be maintained below a problematic threshold. The effective organic plot frequency can be further reduced by maintaining crop rotations so that plots growing the same crops remain further apart. This situation is analogous to plant population fragmentation studies that have considered the opposite scenario. Plant pollination studies showed that as a species becomes more fragmented or less dense, the declining population size reaches a threshold value for extinction likelihood, beyond which it will go extinct as the dispersing pollen fails to reach the plants (Ghazoul, 2005) (This is true for non self-fertilizing species). The species rarefaction affects both pollen dispersal success and insect foraging efficiency in insect pollinated plants (analogous to vector or host dispersal of pathogens) (Ghazoul, 2005). The existence of this threshold has plant conservation implications when selecting size and frequency of protected areas. The issue was discussed ecologically (Janzen, 1983, 1986) and, then, mathematically (Cantrell and Cosner, 1993) confirming the existence of parameter values for which extinction will always occur even in protected areas, and conditions under which co-existence of competitor plants is possible. For plants in parks or protected areas, growth rate, park area, plant species density, and population fragmentation are important parameters affecting dispersal and plant establishment. Parks that are too small suffer from what ecologists call an edge effect, and called a buffer zone by Cantrell and Cosner (1993). In the situation studied by these authors, the park is located inside a background of agriculture or secondary successional habitat, but the species invasion is from the background into the protected area, the inverse situation from our analysis here with pathogens. This issue is relevant here as organic agriculture provides refuge habitats without pesticides for pollinators and beneficial insects to disperse from, which can benefit conventional plots. However, whether these insects can be abundant enough to be useful depends in turn on the ratio of the area of the organic agriculture refuge to the area under conventional agriculture.

5. Conclusions and perspectives

We showed that as the ratio of the area under organic agriculture increases relative to the area under conventional agriculture, there is a threshold ratio indicated by a mathematical bifurcation, above which organic plots always become infected with pathogens. This also increases the pathogen infection rate on the surrounding area under conventional practice. The value of the threshold will vary with the pathogen, environmental factors, field management related variables and abiotic parameters. Compared to conventional agriculture, lower crop yield, reduced germination rate and plant growth, and higher loss to disease are the three principal arguments levied at organic agriculture. These arguments raise both doubt and concern that world food supply security can be maintained through establishment and expansion of organic agriculture alone. However, one ought not ignore the valid serious criticisms raised against conventional methods, that stimulated organic agriculture (Ramesh et al., 2005; Hobbs, 2007; Badgley et al., 2007). Clearly, the soil erosion, organic matter depletion, reduced water and nutrient retention, and bioaccumulation of toxic agricultural pollutants, at their current levels are unsustainable. There is a fertile middle-ground of agricultural practices that provide sustainable alternatives to conventional methods. These include conservation agriculture (no-tillage methods), reduced tillage, better management of irrigation water resources, crop rotations, integrated pest management, and reduced applications of fertilizer, pesticides, and herbicides through better soil organic matter management (Rotz et al., 2005; Rosset and Altieri, 1997; Lal, 2007; Lal et al., 2007; Smith et al., 2008; Hobbs, 2007; Ye et al., 2002). In addition, returning to locally adapted crop varieties, at least for cereals, shows potential in increasing yields under sustainable or organic management (Mason and Spaner, 2006). The current trend of increasing the area under organic farming is itself unsustainable, but reactionary in response to consumer demand. Although there is a place for organic produce in society (Ramesh et al., 2005), realistically, they must remain confined to small productions or the luxury end of the market if we are serious about addressing food security issues, until published evidence accumulates that methods used by the organic agriculture sector are effective.

Acknowledgments

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Appendix A. Mathematical analysis of the model

We show the existence of the critical domain size l_c. In either one or two dimension, the model can be written as

$$\frac{\partial u}{\partial t} = D\Delta u + g(x)u - \nu u^2, x \in \Omega, \partial_n u = 0, x \in \partial\Omega \tag{6}$$

where Ω is either a one or two-dimensional bounded domain and

$$g(x) = \begin{cases} \nu, |x| < l \\ \nu - \mu < 0, |x| > l \end{cases}$$

The main conclusion is the following.

Theorem. There exists a critical domain size l_c such that:

1. If $l < l_c$, then for any initial conditions $p(x,0) = p_i(x) \geq 0$, we have $p(x,t) \to 0$ as $t \to \infty$. That is, infestation does not occur.
2. When $l > l_c$, there exists a unique, positive steady state $p_o(x) > 0$ such that for any non-zero initial conditions $p(x,0) = p_i(x) > 0$, we

have $p(x,t) \to p_o(x) > 0$ as $t \to \infty$. That is, an outbreak will occur and the pest profile will settle to $p_o(x) > 0$ after some time.

3. When $l > l_c$ and in the case when the domain Ω is a disk (in one or two dimensions), the outbreak profile p_0 is radially symmetric, $p_0(x) = p_0(|x|)$ and is decreasing in $|x|$.

Proof. It was shown in Berestycki et al. (2005) that the instability of the zero solution is a necessary and sufficient condition for the oubreak solution to occur. The global stability and uniqueness of $p_0(x)$ were also proven there. Therefore to show 1. and 2., it remains to show that there exists a unique l_c such that the zero solution is stable if $l < l_c$ and is unstable if $l > l_c$. Linearizing around $p = 0$, we obtain the eigenvalue problem

$$\lambda\varphi = D\Delta\varphi + g\varphi, \partial_n\varphi = 0, x \in \partial\,\Omega. \tag{7}$$

Using the Raylegh–Ritz quotient, the biggest eigenvalue of Eq. (7) satisfies the

$$\lambda = \ max \frac{\int\limits_{\Omega} -|\nabla\phi|^2 + g\phi^2 dx}{\int\limits_{\Omega} \phi^2 dx} \tag{8}$$

where the maximum is taken over all C^1 functions with $\partial_n\phi = 0$ on $\partial\Omega$. Since g is an increasing function of l for a fixed x, it follows that λ is an increasing function of l. When $l = 0, g < 0$ and from Eq. (8) it follows that $\lambda < 0$. On the other hand, when $l = L$, we have $g > 0$ and using $\phi = 1$ as a test function, it immediately follows that $\lambda > 0$. Therefore there exists a unique l_c such that $\lambda = 0$ when $l = l_c$; $\lambda < 0$ when $l < l_c$ and $\lambda > 0$ when $l > l_c$.

Next we prove the monotonicity of p_o for a radially symmetric case. Note first that by the uniqueness of p_o, it must be symmetric (otherwise rotating p_o yields another solution). Therefore the outbreak profile p_o satisfies

$$\frac{\partial^2 p}{\partial r^2} + \frac{n-1}{r}\frac{\partial p}{\partial r} + (g-p)0 = 0, p(0) = 0, p(L) = 0, p(r) > 0 \tag{9}$$

where $n = 1$ if Ω is one-dimensional or $n = 2$ for a disk. Differentiating we obtain

$$\frac{\partial^3 p}{\partial r^3} - \frac{n-1}{r^2}\frac{\partial p}{\partial r} + \frac{n-1}{r}\frac{\partial^2 p}{\partial r^2} + g(x)p + (g-2p)\frac{\partial p}{\partial r} = 0. \tag{10}$$

Suppose that $\frac{\partial p}{\partial r} > 0$ at some point inside (0,L). Then let $r_0 \in (0,L)$ be the point where $\frac{\partial p}{\partial r}$ attains its maximum, so that $\frac{\partial^2 p}{\partial r^2}(r_0) = 0, \frac{\partial^3 p}{\partial r^3}(r_0) < 0$. Then from Eq. (10) we have $g(r_0) - 2p(r_0) \geq 0$. Conversely from Eq. (9) we also have $g(r_0) - p(r_0) \leq 0$. But this implies $p(r_0) \geq 2p(r_0)$ which is a contradiction since $p > 0$ by assumption. This concludes the proof.

In one dimension, when $\Omega = (0,L)$, Eq. (7) can be written as

$$\begin{cases} \lambda\phi = D\dfrac{d^2\phi}{dx^2} + \nu\phi = 0, 0 < x < l \\ \lambda\phi = D\dfrac{d^2\phi}{dx^2} - (\mu-\nu)\phi = 0, l < x < L \\ \phi'(0) = 0, \phi'(L) = 0, \phi(l^-) = \phi(l^+), \phi(L) = 0, \phi'(l^-) = \phi'(l^+) \end{cases}.$$

Setting $\lambda = 0, l = l_c$, we find that l_c satisfies Eq. (4). When Ω is a two-dimensional ball of radius L, the formula (5) is similarly computed.

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