Organic agriculture cannot replace conventional agriculture

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In this paper, we consider the effect of increasing the area of agricultural land under organic practices. We assumed that organic agriculture does not have effective means of pathogen control. 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 is below a certain threshold, the pest population is kept small. Above this threshold, the pest population in the organic plot grows rapidly. In this case, the organic plot will act as a source of pest to the surrounding regions, and will always infect organic plots as they become more closely spaced.

Introduction

Organic farming [1, 2] is gaining in popularity in Europe, south America, north America, Japan and Australia among consumers and producers [3, 4]. These studies report an increase of 60% in global acreage under organic agriculture between 2000-2004, and an average annual growth rate of about 20%, although it reaches 50% in Turkey [5]. Initially supplied by numerous small operations, more recently large suppliers to international distributors have engaged in providing organic-labeled produce [6, 7]. Produce with an organic label meet 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 [8, 9] and safer as they do not contain trace amounts of chemicals that are potentially hazardous to human health [10, 11, 12]. Second, organic produce are believed to be environmentally friendly, because organic agriculture avoids using environmentally harmful chemicals that pollute soil, freshwater, ground water and the air [13, 14, 15]. 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 [16, 17]. Two general concerns with organic agriculture are regularly raised. The first concerns food safety issues, the other food security issues [18]. One would assume organic produce, having been produced without application of pesticides would be safe to consume. However, there have been recent cases of consumer illnesses and cross-border product recalls caused by contaminated organic produce, (US-FDA, accessed 20th August 2008). 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 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 [19, 20, 21, 22]. Can organically produced crops provide food security, while being more susceptible to yield fluctuations caused by pathogens and pests? The issue continues to be debated but it is clear that organic agriculture is more expensive [23]. This becomes a more serious issue as more land under conventional agriculture is brought into organic production [24]. Intuitively, organic agriculture mimics a pre-industrial agriculture period that did not have adequate defense against crop pathogens and pests. Crops were regularly devastated by epidemics that swept through Europe with consequences on human nutrition and food security (for example [25, 26, 27]). Currently, organic farms operate against a background of conventional agriculture that maintains pathogen load and pest levels low. However, as the area under organic agriculture continues to increase rapidly, the number of refugia for pests and pathogens are postulated to increase. Therefore, it is worth considering whether disease outbreaks could become more frequent as the ratio of agricultural land under organic farming increases relative to the area under conventional farming. We addressed this question mathematically in one and two dimensions, and show the existence of a bifurcation point above a threshold ratio of organic to conventionally farmed area, above which infections will always occur.

Mathematical Model

In constructing the model, we make the following assumptions:

1. The pest spread in a manner consistent with random diffusion.
2. In the absence of pesticide, the pest population is non-zero and can be modelled by a logistic growth model.
3. The addition of the pesticide causes the pest population to die out.

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. For regional or descriptive models one could include the effect of a dominant wind direction by adding an advective term.

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
- $v$: The rate of pest growth in the absence of pesticides

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\( \mu \) - The death rate due to the presence of pesticide. We assume \( \mu > \nu \) (assumption 3 of our model)
\( \ell \) - The size of a farm plot devoted to organic practices.
\( L \) - Size of the entire plot.

In one dimension, the model is then given by,

\[
p_t = D p_{xx} + \nu p (1 - p) - h(x) p, 0 < x < L,
\]
\[
p(0) = 0, \quad p(L) = 0,
\]
where,

\[
h(x) = \begin{cases} 
0 & 0 < x \leq \ell \\
1 & \ell < x < L 
\end{cases}
\]

and \( p_{xx} \) is the second partial derivative of \( p \) with respect to \( x \) and \( p_x \) is the partial derivative of \( p \) with respect to \( x \).

This boundary condition allows us to consider infinite domains in which organic and conventional farms are interspersed periodically. By varying the value of \( \ell \), we can examine the effects of varying the percentage of farmland devoted to organic methods. Note that related models were also considered in [28, 29, 30, 61] among others.

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

\[
\alpha \tan(\alpha l_c) = \beta \tanh(\beta (L - l_c)),
\]
where \( \alpha = \sqrt{\mu} \) and \( \beta = \sqrt{\nu \mu} \).

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

\[
\alpha J_1(\alpha l_c) = \beta K_1(\beta (L - l_c)) / K_0(\beta (L - l_c)),
\]
where \( J_i, K_i \) are Bessel functions of order \( i \) (see [31]).

Mathematically, this behaviour corresponds to a bifurcation of the zero steady state as \( l \) is increased past \( l_c \). An example of this phenomenon is illustrated in Figure 1. Take \( D = 1, \mu = 4, \nu = 1 \) and \( L = 10 \). By numerically solving (4) we then find that \( l_c = 1 \). Figure ?? 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 shown in Figure ???. Since there is a very large range of pest diffusivity, expressed from 10 m \( \text{day}^{-1} \) to 10 000 m \( \text{day}^{-1} \), we plot the critical organic plot area \( l_c \) versus the logarithm of pest diffusivity in Figure 2. From (4), it is clear that as \( D \to \infty, \ell_c \to \frac{\mu}{\nu} \). Therefore from this simple relationship one can estimate the critical ratio of organic to conventional farm area for rapidly dispersing pathogens. In addition this model can be refined further using local or regional scale models that contain more parameters.

**Discussion**

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 alldodeposition (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.

**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. Dispersion occurs through rain drops, wind, arthropod dispersal, carried by a vector such as an insect or through an infected host (seed, pollen, insect) [32, 33, 34] as well as anthropogenic transport. Dispersion of spore, pollen, seed or arthropods is usually described by diffusion based analytical models [35], persistent random walk [36], logistic and Gompertz models [37, 38, 39, 40], Lagrange puff models with gaussian or non gaussian turbulence as in atmospheric pollution studies [41, 42]. Epidemic progress can be modeled with general disease models developed from data [37]. Field data confirm that spore dispersal and deposition events are randomly distributed because spores arrive both from alldodeposition, autodeposition and accumulation in the field from previous years [36, 43, 41, 39, 44, 45].

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 [42]. 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 [46]. Regional disease progress models can be constructed with more or less parameters to predict and manage epidemics locally. However, an unpredictable stochastic element exists through long-distance wind dispersal. Vertical wind puffs carries 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 [32, 47, 48, 49]. Increased frequency of outbreaks in one region can cause repeated long-distance infections through air currents.

Regionally, organic plot aggregation or density can be maintained low to remain 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 problem. 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 (This is true for non self-fertilizing species) [50]. The species rarefaction affects both pollen dispersal success and insect foraging efficiency in insect pollinated plants (analogous to vector or host dispersal of pathogens) [50]. The existence of this threshold has plant conservation implications when selecting size and frequency...
of protected areas. The issue was raised ecologically [51, 52] and then mathematically [35] 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[35]. In this situation, 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 reverse situation from our analysis here with pathogens.

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 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 organic agriculture alone. However, one ought not ignore the valid serious criticisms raised against conventional methods, that stimulated or-

Appendix: Mathematical analysis of the model

We now show the existence of the critical domain size \( l_c \). In either one or two dimension, the model (1) can be written as

\[
u_t = D \Delta u + g(x)u - \nu u^2, \quad x \in \Omega; \quad \partial_n u = 0, \quad x \in \partial \Omega \quad [6]
\]

where \( \Omega \) is either a one or two-dimensional bounded domain and

\[g(x) = \begin{cases} v > 0, & |x| < l \\ v - \mu < 0, & l < |x| \end{cases} \]

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 \( p(x, 0) = p_0(x) \geq 0 \), 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 \( p_0(x) > 0 \) such that for any non-zero initial conditions \( p(x, 0) = p_0(x) > 0 \), we have \( p(x, t) \to p_0(x) > 0 \) as \( t \to \infty \). That is, an outbreak will occur and the perturbed profile will settle to \( p_0(x) > 0 \) after some time.
3. When \( l > l_c \) and in the case when the domain \( \Omega \) 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 [61] that the instability of the zero solution is necessary and sufficient condition for the outbreak solution to occur. The global stability and uniqueness of \( p_0(x) \) was 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

\[
\lambda \phi = D \Delta \phi + g \phi, \quad x \in \Omega; \quad \partial_n \phi = 0, \quad x \in \partial \Omega. \quad [7]
\]

Using the Rayleigh-Ritz quotient, the biggest eigenvalue of (7) satisfies the

\[
\lambda = \max \left\{ \frac{\int_{\Omega} - |\nabla \phi|^2 + g \phi^2 \, dx}{\int_{\Omega} \phi^2 \, dx} \right\} \quad [8]
\]

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 \( \lambda \) is an increasing function of \( l \). When \( l = 0 \), \( g < 0 \) and from (8) it follows that \( \lambda < 0 \). On the other hand, when \( l = L \), we have \( g \geq 0 \) 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 \( p_0 \) for a radially symmetric case. Note first that by uniqueness of \( p_0 \), it must be symmetric (otherwise rotating \( p_0 \) yields another solution). Therefore the outbreak profile \( p_0 \) satisfies

\[
p_{rr} + \frac{n - 1}{r} p_r + (g - p) p = 0; \quad p'(0) = 0, \quad p'(L) = 0, \quad p(r) > 0. \quad [9]
\]

where \( n = 1 \) if \( \Omega \) is one-dimensional or \( n = 2 \) for a disk. Differentiating we obtain

\[
p_{rrr} - \frac{n - 1}{r^2} p_r + \frac{n - 1}{r} p_{rr} + g'(x) p + (g - 2p) p_r = 0. \quad [10]
\]

Suppose that \( p_r > 0 \) at some point inside \((0, L)\). Let then \( r_0 \in (0, L) \) be the point where \( p_r \) attains its maximum, so that \( p_r(r_0) = 0 \), \( p_{rr}(r_0) < 0 \). Then from (10) we have \( g(r_0) - 2p(r_0) > 0 \). On the other hand from (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 \( \Omega = (0, L) \), equation (7) can be written as

\[
\begin{cases}
\lambda \phi = D \phi_{xx} + (\nu - \nu \nu) \phi = 0, \quad 0 < x < l \\
\lambda \phi = D \phi_{xx} - (\nu - \nu \nu) \phi = 0, \quad l < x < L \\
\phi'(0) = 0, \quad \phi'(L) = 0; \quad \phi'(l) = \phi(l^+), \quad \phi'(l^-) = \phi(l^-).
\end{cases}
\]

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

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Fig. 1. (a) Plot of the fraction of the farmland dedicated to organic procedures vs fraction of maximal pest population with $D = 1$, $L = 10$, $\nu = 1$, $\mu = 4$ and $\ell$ as given. If less than 10% of the farmland is dedicated to organic practices the pest population remains minimal. Past this limit, the pest population grows rapidly to maximal. This agrees with the prediction given by (4) of $\ell_c = 1.047$. (b) The graph of the pest population profile for various values of $\ell$. From bottom to top, $\ell = 1, 1.1, 1.5, 2, 4, 8$. Other parameters are as in (a).

Fig. 2. Plot of $\ell_c$ versus $\log_{10}(D)$ for the case $\mu = 2$, $\nu = 1$ and $L = 10$. 