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Linking Plant Disease Risk and Precipitation Drivers: A Dynamical Systems Framework

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abstract: Plant pathogens often respond sensitively to changes in their environmental conditions and consequently represent a potentially important ecological response to global change. Although several studies have considered the effects of increased temperature and CO2 concentrations on plant pathogen risk, the effects of changing precipitation regimes have drawn less attention. Many classes of plant pathogen, however, are sensitive to changes in the water potential of their local environment. This study applied existing ecohydrological frameworks to connect precipitation, soil, and host properties with scenarios of pathogen risk, focusing on two water-sensitive pathogens: *Phytophthora cinnamomi* and *Botryosphaeria dothidea*. Simple models were developed to link the dynamics of these pathogens to water potentials. Model results demonstrated that the risk of host plants being colonized by the pathogens varied sensitively with soil and climate. The model was used to predict the distribution of *Phytophthora* in Western Australia and the severity of disease in horticultural blueberry trials with variable irrigation rates, illustrating potential applications of the framework. Extending the modeling framework to include spatial variation in hydrology, epidemic progression, and feedbacks between pathogens and soil moisture conditions may be needed to reproduce detailed spatial patterns of disease. At regional scales, the proposed modeling approach provides a tractable framework for coupling climatic drivers to ecosystem response while accounting for the probabilistic and variable nature of disease.

Keywords: ecohydrology, plant pathology, soil water potential, multiple stressors, dichotomous Markov noise, *Phytophthora cinnamomi*.

Introduction

Plant pathogens are important agents of disturbance, generating significant economic and environmental impacts in association with agricultural crop damage and plant mortality events. Plant pathogen epidemiology is often directly influenced by local edaphic and climatic factors (Agrios 2005) and may respond sensitively to relatively small changes in the local environment (Tapsoba and Wilson 1983; Maxwell et al. 1997; Mayek-Perez et al. 2002; Waugh et al. 2003; Thompson et al. 2010). The potential for altered pathogen ranges and epidemiology under future climates is considered to be a significant risk associated with global change (Coakley et al. 1999; Chakraborty et al. 2000; Boland et al. 2004; Garrett et al. 2006; Ibanez et al. 2006). Most studies examining the risks of altered pathogen behavior under future climate scenarios have focused on the role of temperature (Chakraborty 2005; Garrett et al. 2006; Thompson et al. 2010) and, in some cases, elevated atmospheric carbon dioxide concentrations (Pangga et al. 2004; Melloy et al. 2010) in altering pathogen dynamics. By contrast, there have been relatively few investigations of the potential for altered precipitation regimes to influence pathogen dynamics under future climate scenarios (Desprez-Loustau et al. 2006), despite the importance of water for the spread, survival, and disease potential of many plant pathogens.

Plant Pathogens in a Changing Rainfall Environment

An extensive body of research links water availability, and specifically ambient soil moisture or plant water potentials, to the dynamics of plant pathogens (Crist and Schoeneweiss 1975; Malajczuk and Theodorum 1979; Dickenson and Wheeler 1981; Madar et al. 1989; Boyer 1995; Schober and Zadoks 1999; Suleman et al. 2001; Desprez-Loustau et al. 2006; Ferrin and Stanghellini 2006). Many pathogens have a nonlinear response to changes in the hydration of their environment, which is manifest in terms of pathogen growth rates, survival, disease intensity, and reproductive behavior. The mechanisms that underlie this sensitivity may include changes in plant resistance to disease under waterlogged or drought conditions (Crist and Schoene-
sensitivity to water potentials. The first are the upon two relatively well-studied classes of pathogen with the epidemiology of plant pathogens, so the study focuses deals with the dynamics of soil moisture and plant water potentials to develop a simple theoretical framework to link precipitation, soil properties, and host species properties with disease risk. The focus is on “nonaggressive” species, as the pathogens are most likely to display a sensitive response to altered climate or precipitation dynamics.

It is notoriously difficult to make generalizations about the epidemiology of plant pathogens, so the study focuses upon two relatively well-studied classes of pathogen with sensitivity to water potentials. The first are the Phytophthora water molds, and in particular Phytophthora cinnamomi (Pc). Phytophthora species are soil-borne pathogens that destroy plant fine root systems, starving plants of water and nutrients and ultimately causing death. As a contrasting example, stem canker pathogens, exemplified by Botryosphaeria dothidea (Bd) are considered. Canker-causing organisms enter host plants through wounds and then grow in the stem, colonizing the sapwood and ultimately girdling and killing their hosts. The main distinction between the pathogens here, however, is that Pc disease is promoted by wet conditions and inhibited in dry soils, while Bd mostly causes disease in drought-stressed plants. A brief review of the biology and water sensitivity of both species follows.

**Biology of Phytophthora Root-Rots**

*Phytophthora cinnamomi* is a mycelial oomycete (or “water mold”), which occurs either as a saprophyte or a root parasite infecting the fine roots of woody plants (Weste and Marks 1987; Harham 2005). Pc is highly destructive to susceptible plants, and is listed as a key threat to biodiversity in Australia (Environmental Protection and Biodiversity Conservation Act 1999). Pc is also associated with tree mortality and loss of commercial timber production in Hawaii, Europe, and North America (Newhook and Podger 1972; Hwang and Ko 1978; Balci and Halmschlager 2003; Judelson and Blanco 2005; Benson et al. 2006; Jönsen 2006). Pc is most problematic in wet soil conditions, which increase the growth of fungal mycelia, the rate of production, survival, motility of fungal propagules, and the infection potential of the pathogen (Hwang and Ko 1978; MacDonald and Duniwai 1978; Nesbitt et al. 1978; Malajczuk and Theodorou 1979; Benjamin and Newhook 1981). By comparison, dry conditions can prevent or reverse Pc spread (Tippett et al. 1989). Pc is also sensitive to water potentials within host plants, with extension of fungal lesions and the progression of root mortality varying with water potential (Malajczuk and Theodorou 1979; Tippett et al. 1987). In sterile soil, water potentials exceeding −3,000 kPa are needed for pathogen growth (see fig. 1, supplemental material, available online), while disease symptoms within, for example, susceptible *Eucalyptus marginata*, are suppressed at phloem water potentials of less than −900 kPa (Tippett et al. 1987); these thresholds vary with soil type, fertility, and the host species (Sterne et al. 1977a, 1977b). Pc cannot survive long periods of freezing temperatures (Bergot et al. 2004) and tends to be absent from rich soils with a diverse microflora community, apparently due to predation on the pathogen by amoebae and other soil organisms (Weste and Marks 1987). There is ongoing debate as to how Pc infection leads to plant mortality (Weste and Marks 1987; Davidson and Tay 1995; Maurel et al. 2001) and regarding the causes of the large variability in host susceptibility observed in many ecosystems (Weste and Marks 1987). The framework here will not explicitly address temperature variation or soil fertility and assumes that water stress is the dominant factor leading to mortality of hosts infected with Pc. It provides a tool for linking Pc dynamics to ecohydrologic variation but is not a comprehensive framework for interrogating Pc disease risk.

**Biology of Botryosphaeria Stem Cankers**

*Botryosphaeria dothidea* is a fungal pathogen that generates canker diseases in woody stems. Bd spreads through the vascular tissues of infected plants, causing bleeding necrosis, blocking xylem pit membranes, increasing cavitation risk, disrupting water and photosynthate transfer through the stem, and reducing hydraulic conductance of the plant (Crist and Schoeneweiss 1975; Madar et al. 1990; Vannini and Valentini 1994). The prevalence and severity of Bd increases with drought stress in a wide range of species (Pusey 1989; Brooks and Ferrin 1994; Smith et al. 1994; Rayachhetry et al. 1996; Ma et al. 2001). The most detailed studies of Bd sensitivity to water potentials examined the disease behavior in white birch (Crist and Schoeneweiss 1975). Growth of Bd stem cankers in white birch was inhibited when water potentials in the trees were maintained at over −1200 kPa, while water-stressed plants...
with potentials of less than $-1200$ kPa developed disease symptoms. Removing water stress resulted in callus formation and decline of the pathogen, suggesting that the canker infections were reversible over some range of severity (Crist and Schoeneweiss 1975). The water potential sensitivity of Bd seems to be related to both a reduction in host resistance when stressed, as suggested by histological studies (McPartland and Schoeneweiss 1984), and to the intrinsic properties of Bd, as suggested by petri-dish studies (Ma et al. 2001). As with Pc, Bd epidemiology is not solely a function of drought stress in plants. Although Bd causes disease in water-stressed hosts, the pathogen requires wet conditions to spread (Ahimera et al. 2004), and the disease gains entry into plants through wounding (Rayachhetry et al. 1996; Rolshausen et al. 2010). Studies of Bd have not explored how extensive the canker can become before disease becomes irreversible. It is assumed here that Bd infection is present, if cryptic, in unstressed trees. Removal of water stress—if maintained indefinitely—is assumed to result in a complete recovery of the stem. These assumptions, although undoubtedly simplifications, provide a tractable starting point for developing a modeling framework.

Model Development

The model framework focuses on developing a simple model of pathogen dynamics and their effects on the stress experienced by the host plant and coupling it to existing ecohydrological models, as illustrated in figure 1. The pathogen growth rates are assumed to depend deterministically on the water potentials within the host plant, which can be directly related to soil water potentials ($\Psi$). Because these water potentials are driven by soil moisture availability, which fluctuates in time as a Markov process (Rodriguez-Iturbe et al. 1999), the pathogen growth rates themselves are stochastic. For both Pc and Bd, the dependence of the growth rates on the water potentials is similar to a step function, meaning that these fluctuations are dichotomous in nature: either the pathogen grows at its maximum rate, or the water availability is unfavorable and the pathogens grow at a minimum rate (which may be
zero). The critical water potential around which these fluctuations occur is termed $\Psi_c$. The probability density function (PDF) of processes forced by dichotomous Markov noise may be analytically determined using a solution presented by Horsthemske and Lefever (1984), provided that the statistics of the Markovian fluctuations (i.e., the soil moisture fluctuations) are known. To determine these statistics we draw on existing ecohydrological theories that predict the probability density function of soil moisture and its “crossing properties,” that is, the frequencies of fluctuations in soil moisture around a given point. The existing theory allows the soil moisture PDF to be analytically predicted as a function of soil properties, rainfall statistics and evaporative demand. It therefore provides a mechanism to specify the fluctuations in the state of the pathogen purely as a function of (known) soil and climate properties. The role of existing theory in forging this link is shown conceptually in figure 1. Because this theory has already been extensively reported in the literature (Rodriguez-Iturbe et al. 1999; Laio et al. 2001) and is quite mathematically detailed, we do not reproduce it here.

**Pathogen Model**

To generate a simple model of Pc infection in a plant root system, several assumptions are adopted. Firstly, we assume that there is a maximum root volume $v_{\text{max}}$, that can be sustained by the host tree. A completely healthy tree has a root volume $v$ equal to $v_{\text{max}}$, with no infection in any of its roots. As Pc colonizes the host’s root zone it impedes the function of and ultimately kills infected roots, so that the extent of the impeded roots, $b$, lies between 0 and $v_{\text{max}}$. In a host with resistance to Pc, new, uninfected roots regrow to replace dead or infected roots Newhook and Podger 1972; Weste and Taylor 1987. This has the effect of reducing the proportion of $v_{\text{max}}$ impeded by Pc. The growth rate of Pc through the root volume reaches a maximum value in soils with water potentials greater than $\Psi_c$. In soils with water potentials below $\Psi_c$, the rate of root regeneration is assumed to exceed the rate at which infection expands, so that the infected root volume shrinks overall.

These assumptions are an approximation to the real dynamics of Pc infections and host recovery, the complete description of which requires a coupled model of both the pathogen growth and root turnover. With appropriate parameter choices, however, the Pc dynamics predicted by such a coupled model can be reasonably approximated by a single expression for $b$, based on the assumptions above. This point is justified in the supplemental material. The simplified expression takes the form

$$\frac{db}{dt} = b(r(\Psi) - m) \quad \text{for } b < v_{\text{max}},$$

$$= \min \left[b(r(\Psi) - m), 0\right] \quad \text{for } b = v_{\text{max}},$$

where

$$r(\Psi) = r_{\text{max}} \quad \text{for } \Psi \geq \Psi_c,$$

$$= r_{\text{min}} \quad \text{for } \Psi < \Psi_c.$$

Here $b$ [m$^3$] represents the volumetric extent of the host’s root impairment due to both the spread of Pc and mortality of infected roots. Term $b$ is bounded between 0 and the maximum rooting volume $v_{\text{max}}$, $r(\Psi)$ is the rate at which the pathogen extent increases throughout the host root system. Term $m$ is the rate at which root regeneration reduces the proportion of the root system that is impaired by the pathogen activity. This rate is assumed to be constant regardless of host, soil water, or pathogen status, meaning that the total production of new roots is greatest when the pathogen has caused the most impairment. This approximation is most reasonable for woody species with large reserves of photosynthate that could be deployed to restore the root-shoot ratio. The growth rate of the fungal mycelia $r$ (day$^{-1}$) is dependent on the plant water potential, so that $r(\Psi) = r_{\text{max}}$ when $\Psi > \Psi_c$, and $r(\Psi) = r_{\text{min}}$ otherwise. That is, the growth response of Pc to water potential is approximated as a step function around a critical plant water potential value $\Psi_c$, a reasonable approximation to experimental observations (Malajczuk and Theodorou 1979) (see fig. 1 in supplemental material). Observations also indicate a reduction in Pc growth rates under permanently saturated conditions (Malajczuk and Theodorou 1979). Saturation rarely persists in most soils, so this reduction is neglected. As outlined in the supplemental material the plant water potential, soil water potential, and soil water content are readily interconvertible using the Brooks-Corey equation and an Ohm’s law analogy for flow in the soil-plant system (Brooks and Corey 1964; Tyree and Ewers 1991).

The dynamics described by equation (1) are simple. Under wet soil conditions the pathogen spreads at its maximum rate $r_{\text{max}}$, colonizing the root zone. The extent of its proportional colonization is reduced by the production of new roots at rate $m$. If the soil becomes sufficiently dry, $r = r_{\text{max}} < m$, and the root zone of the hosts recovers. These linear dynamics prevail unless the extent of the pathogen infection increases so that $b \approx v_{\text{max}}$, in which case further growth of the pathogen does not result in further damage to the root zone of the host; that is, $b$ is bounded by 0 and $v_{\text{max}}$.

The piecewise functional form in equation (1), while intuitive, makes further analysis awkward. A single-equation expression that maintains the $db/dt = 0$ condition at

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b = \nu_{\text{max}} can be formed by adjusting equation (1) with the nonlinear term \((b/\nu_{\text{max}})^n\):

\[
\frac{db}{dt} = b(r(\Psi) - m) - \left(\frac{b}{\nu_{\text{max}}}\right)^n \tag{3}
\]

where, as \(n \to \infty\), the steady state of equation (3), \(b^* \to \nu_{\text{max}}\). The advantage of this nonlinear form is the rate at which the growth rate of the pathogen approaches zero as \(b\) approaches \(\nu_{\text{max}}\) can be made arbitrarily fast by appropriate selection of \(n\). The cutoff represents a maximum pathogen extent from the perspective of the infected plant (the pathogen cannot infect more than all of the plants roots) but not necessarily a point where pathogen growth should slow dramatically. In what follows \(n = 100\), which causes the \(b\) growth dynamics to closely approximate a piecewise function of a constant growth rate for small \(b\) and a growth rate of 0 when \(b \approx \nu_{\text{max}}\). In practice, a finite value of \(n\) results in the steady state value of \(b\) to be slightly less than \(\nu_{\text{max}}\). For \(n = 100\), this steady state value deviates from \(\nu_{\text{max}}\) by approximately 1%. This deviation can be made smaller by increasing \(n\). Similarly, when \(r = r_{\text{min}}\) and \(b \approx \nu_{\text{max}}\) this formulation causes the rate of pathogen decline to be slightly overestimated while \((b/\nu_{\text{max}})^n\) is comparable in magnitude to \(r_{\text{min}} - m\). This overestimation occurs only for \(b\) within 1% of the maximum value of \(\nu_{\text{max}}\) and has no significant impact on the predicted dynamics. The simplicity of the expression in equation (3), however, is extremely valuable.

Equation (3) can be nondimensionalized by taking \(B = b(\nu_{\text{max}})\) \(t' = tm\), \(R_{\text{max}} = r_{\text{max}}/m\) and \(R_{\text{min}} = r_{\text{min}}/m\), so that

\[
\frac{dB}{dt'} = B(R(\Psi) - 1 - B^n),
\]

\[
R(\Psi) = R_{\text{max}} \text{ for } \Psi > \Psi_{b}, \tag{4}
\]

\[
= R_{\text{min}} \text{ for } \Psi \leq \Psi_{b}.
\]

A very similar set of arguments can be made regarding stem canker dynamics, except that the canker growth is limited by the finite cross-sectional area of the stem and the soil moisture dynamics are imposed on the host resistance rather than the pathogen growth. The sensitivity of the growth rates to the water potentials is inverted for Bd compared to Pc, that is (again in nondimensional terms),

\[
\frac{dC}{dr} = C(1 - K(\Psi) - C^n),
\]

\[
K(\Psi) = K_{\text{max}} \text{ for } \Psi > \Psi_s, \tag{5}
\]

\[
= K_{\text{min}} \text{ for } \Psi \leq \Psi_s,
\]

where \(K\) is a nondimensional mortality rate for the canker induced by host resistance, \(C\) is the canker area extent nondimensionalized by the stem area \(A_s\), and \(r\) is time nondimensionalized against a fixed canker growth rate.

### Dichotomous Markov Noise and the PDF of the Pathogen Extent

Having derived simple nondimensional expressions for the pathogen extent (eqq. [4], [5]), we can use the probabilistic dynamics of soil moisture and the theory of dichotomous Markov noise to derive an approximate probability density function for the pathogen extent. Equations (4) and (5) satisfy several criteria that are necessary to apply the solution methodology. First, the equations are stochastic ordinary differential equations multiplicatively driven by a Markovian random process (soil moisture variations; see Rodriguez-Iturbe et al. 1999; Rodriguez-Iturbe and Porporato 2004). Although the driving process can adopt many values, its effect on the pathogen dynamics is to introduce dichotomous switching around the soil moisture threshold (e.g., \(R\) and \(K\) adopt only two values). Secondly, a steady state condition exists for each of the two potential values of \(R\) and \(K\). Finally, the steady state conditions differ for \(R_{\text{min}}\) and \(R_{\text{max}}\) and for \(K_{\text{min}}\) and \(K_{\text{max}}\), and bound the plausible range of values (0 to 1) of the state variables.

These conditions allow the analytical solution of Horstemke and Lefever (1984) to be applied to determine the steady state PDF of the pathogen conditions \(B\) and \(C\). This analytical solution for the PDF depends on the probability of switching around the soil moisture threshold and the form and parameters of equations (4) and (5). To apply the analytical solution, the governing equations are first reexpressed in terms of a stochastic “switch” \(\Delta\) that can adopt values of 0 or 1, depending on the soil moisture. For the Pc case,

\[
\frac{dB}{dt'} = g(B) + h(B)\Delta, \tag{6}
\]

\[
g(B) = (R_{\text{min}} - 1 - B^n)B, \quad h(B) = (R_{\text{max}} - R_{\text{min}})B.
\]

That is, if \(\Delta = 1\) the pathogen will grow, and if \(\Delta = 0\) the host resistance will prevail. The \(\Delta\) must be conditioned on the value of the plant water potential—or equivalently on the soil moisture—as follows:

\[
\Delta(\Psi) = \frac{\Delta_D = 0}{\Delta_W = 1} \text{ for } \Psi \leq \Psi_s, \tag{7}
\]

Here the subscripts \(D\) and \(W\) refer to “dry” and “wet” conditions and are included for clarity. To compute the PDF of \(B\), the derivations given in Muneepeerakul et al.
(2007) and Horstemke and Lefever (1984) are followed. The switching behavior of $\Delta$ can be characterized by two frequencies, $f^+$ and $f^-$, which represent the frequency of switching across the soil moisture threshold from dry to wet ($\Delta_n$ to $\Delta_w$) and from wet to dry ($\Delta_w$ to $\Delta_n$). The probability associated with being in either of the moisture states can be expressed as

$$p(\Delta_w) = \frac{f^+}{\gamma^*},$$

$$p(\Delta_n) = \frac{f^-}{\gamma^*},$$

(8)

where $\gamma^* = f^+ + f^-$. The mean value ($E(\Delta)$) of $\Delta$ is

$$E(\Delta) = \frac{1}{\gamma^*} (f^+ \times \Delta_w + f^- \times \Delta_n).$$

(9)

The connection to the soil moisture PDF and the rainfall statistics lies in $\gamma^*$ and $\gamma$. These values are determined ($W$) and the “loss function” (i.e., the sum of evaporation and transpiration) for a given soil moisture $\rho(\Psi)$ (Porporato et al. 2001):

$$f^+ = p(\Psi^*)p(\Psi^c).$$

(10)

The computation of $p(\Psi^*)$ and $p(\Psi^c)$ as a function of the soil and climate properties is detailed in the supplemental material. Given $f^+$, the frequency of downcrossing can be computed from the probability $p^+$, where $p^+ = p(\Psi^* > \Psi^c)$. This is computed as $p^+ = 1 - \text{CDF}(\Psi^c)$, where CDF indicates the cumulative distribution function of the soil moisture. Similarly, the probability of the water potential being less than the critical value is $p^- = \text{CDF}(\Psi^c)$.

The steady state PDF of the variable $B$ can be computed as

$$p(B) = \frac{h(B)}{(g(B) + \Delta_n h(B))(g(B) + \Delta_w h(B))} \times \exp\left[-\gamma^* \int \frac{g(z) + E(\Delta)h(z)}{(g(z) + \Delta_w h(z))(g(z) + \Delta_n h(z))} dz\right].$$

(11)

Substituting equations (7) and (9) into equation (11) and integrating, the PDF is obtained as

$$p(B) = -N'(R_{\text{max}} - R_{\text{min}})B^{n}(1 - R_{\text{max}} + B^n)^{n'},$$

(12)

where

$$a_1 = \left[1 + \frac{f^+}{R_{\text{min}} - 1} + \frac{f^-}{R_{\text{max}} - 1}\right]^{-1},$$

$$a_2 = \frac{f^-}{n(R_{\text{max}} - 1)},$$

$$a_3 = \frac{f^+}{n(R_{\text{min}} - 1)},$$

(13)

for $B \in [0, 1]$ and $N'$ is a normalization constant chosen such that $\int p(B) dB = 1$.

Term $B$ is defined on the region $B \in [0, 1]$. This analytical solution reproduces numerical simulations of the pathogen growth and plant recovery well, including the location of the mode(s) of the distributions at the extremes of $B = 0$ or $B = 1$ associated with the presence of the steady states (see fig. 2 in the supplemental material). The strength of the bimodality increases as $f^+$ and $f^-$ decrease, meaning that the duration of excursions above or below increases relative to the timescales of pathogen growth or loss. Less of the PDF mass is concentrated at the steady states as the $f^-$ and $f^+$ increase, because soil moisture fluctuations tend to reverse net changes in pathogen biomass rapidly, providing a stabilizing influence. Eventually (with the appropriate parameter choice), the PDF collapses to a single mode.

The expectation of $B$ can be computed analytically as

$$\bar{B} = \frac{1}{1 + a_i} (R_{\text{max}} - 2)^{\nu_i} [(2 - R_{\text{max}})(R_{\text{min}} - 1)]^{\nu_i} \times (R_{\text{max}} - R_{\text{min}})(R_{\text{min}} - 2)^{\nu_i} \times ((R_{\text{min}} - 2)(R_{\text{max}} - 1))^{\nu_i} \times A\left(1 + \frac{1}{n}, -a_j; -a_j; 1 + a_i + n, 1, R_{\text{max}} - 1, R_{\text{min}} - 1\right),$$

(14)

where $a_i$, $a_j$, and $a_k$ adopt the values given in equation (13), and $A$ is the Appell hypergeometric function of the first type (Appell 1925), defined as

$$A(\xi; \mu; \nu; x, y) = \sum_{i=0}^{\infty} \sum_{j=0}^{\infty} \xi^{i+j} \mu^{i} \nu^{j} x^i y^j.$$

(15)

The same derivation can be applied to the Bd canker dynamics described by equation (5). Since the host resistance rather than the growth rate is conditioned on the soil threshold for the Bd case, none of the other definitions of $\Delta$ or the probabilities need to be altered. The equations are

$$\frac{dC}{dt} = g(C) + h(C)\Delta,$$

$$g(C) = (1 - K_{\text{min}} - C^n)C,$$

$$h(C) = (K_{\text{min}} - K_{\text{max}})C.$$
with $\Delta$ defined as in equation (7) and $C$ representing the dimensionless extent of the stem canker. Repeating the analysis in equations (8)–(11), the PDF for the stem canker disease is obtained as

$$p(C) = -N(K_{\max} - K_{\min})C^d \times (C^* + K_{\max} - 1)^d(C^* + K_{\max} - 1)^d,$$

where

$$a_i^* = \frac{f^*}{K_{\max} - 1} + \frac{f^-}{K_{\max} - 1} - 1,$$

$$a_i^* = \frac{f^-}{n(1 - K_{\max})} - 1,$$

$$a_i^* = \frac{f^-}{n(1 - K_{\min})} - 1,$$

and the expectation of the PDF is:

$$\mathbb{E} = \frac{K_{\min}(K_{\max}/(K_{\max} - 1))^{d}((K_{\max} - K_{\max})K_{\min}((K_{\max} - 1))^{d}}{2 + a_i^*},$$

$$\times \left(1 + a_i^*; 1, -a_i^*; 2 + a_i^* + n, \frac{1}{1 - K_{\max}}, 1 - K_{max} \right).$$

$$\mathbb{E} = \frac{K_{\min}(K_{\max}/(K_{\max} - 1))^{d}((K_{\max} - K_{\max})K_{\min}((K_{\max} - 1))^{d}}{2 + a_i^*},$$

$$\times \left(1 + a_i^*; 1, -a_i^*; 2 + a_i^* + n, \frac{1}{1 - K_{\max}}, 1 - K_{max} \right).$$

The random variables $B$ (e.g., normalized extension of the infection) and $s$ (e.g., relative soil moisture) are assumed to be independent (see supplemental material for validation of this assumption). Given that $B$ and $s$ are independent, the expected value of the PDF of the joint water and pathogen stress may be computed from the PDFs of $\{[s^* - s]/(s^* - s_s)\}_s$ (given in Porporato et al. 2001) and the PDF of $B$ computed above, as

$$\gamma(s) = B^o + (1 - B^o)\left(\frac{s - s_o}{s - s_s}\right).$$

$$\gamma(s) = B^o + (1 - B^o)\left(\frac{s - s_o}{s - s_s}\right).$$
Equation (21) quantifies the joint effect of pathogen and water stress in a mean-field sense.

**Parameterization**

Parameterization of the model requires estimates of pathogen growth rates during both expansion and reduction phases of growth. For Pc, the approach taken is to estimate a reasonable value for the maximum and minimum growth rates from laboratory data and then to treat the host resistance as a variable parameter. For Bd, the baseline growth rate, the “growth rate minus \( K_{\text{min}} \)” value is taken from greenhouse data, and the maximum host resistance \( K_{\text{max}} \) is treated as a variable.

The Pc growth rate data used are data describing mycelial extension obtained by Malajczuk and Theodorou (1979), who determined mycelial growth over a 10-day period from a fixed initial disk (assumed to have an initial length of 0.5 mm) introduced into 1.3-mm diameter tubing. Assuming uniform hyphal density and exponential growth, the maximum rate of mycelial growth was 0.46 \( \text{day}^{-1} \). Under dry conditions, the minimum finite growth rate measured for Pc was 0.07 day \( ^{-1} \). Hosts with low resistance were assumed to impose a pathogen loss rate of 0.1 day \( ^{-1} \), and hosts with high resistance were assumed to impose a pathogen loss rate of 0.4 day \( ^{-1} \). These parameters exclude potential biochemical resistance in some plants, which may result in uniformly lower growth rates.

Bd growth rates in the absence of the host can be determined from experiments conducted by Ma et al. (2001) by fitting an exponential profile to colony diameter values obtained when growing Bd from 5-mm-diameter plugs on potato starch media. A growth rate of 2.4 day \( ^{-1} \) was estimated from these data. Bd growth rates within plants can be obtained from Crist and Schoeneweiss (1975) in the same manner. The resulting value for the growth rate within the plant is 0.26 day \( ^{-1} \), suggesting that the minimum host resistance may be estimated as \( \approx 2.1 \) day \( ^{-1} \). We assume that a slowly recovering host has a maximum resistance of 2.6 day \( ^{-1} \), while a rapidly recovering host has a maximum resistance of 3 day \( ^{-1} \). These data were used to develop the parameterizations in Table 1.

**Model Implementation and Testing**

The model was used to explore the sensitivity of the pathogen response and resulting host stress to climatic variations, soil texture, and depth. The model was then applied to three well-characterized case studies. Two relate to *Phytophthora cinnamomi* disease in Western Australia. The first case study explored the large-scale variations in Pc range (at scales of approximately 500 \( \text{km} \times 500 \) \( \text{km} \)), while the second case study attempted to reproduce small-scale spatial variations in observed Pc disease in the forested Wungong water catchment (scales of approximately 5 \( \text{km} \times 5 \) \( \text{km} \)). The third case study uses the model to estimate the relative severity of *Phytophthora cinnamomi* disease in terms of the root characteristics of infected Blueberry (*Vaccinium sp.*) under differing irrigation regimes.

**Sensitivity Analysis.** The model was initially applied for Pc and Bd to explore the mean pathogen extent and mean host stress for a range of reasonable rainfall statistics (mean storm depth \( \alpha \in (0.1, 3) \) cm, and mean storm frequency \( \lambda \in (0.01, 0.3) \) days \( ^{-1} \)). The soil was assumed to be a loamy sand with a 30-cm rooting zone. Soil parameters were taken from the Clapp and Hornberger (1978) data set. The pathogen and host parameters used are given in Table 1, and both the low and high resistance cases were simulated. The peak evapotranspiration was taken as 0.45 cm/day, and bare soil evaporation was assumed to be negligible. To explore the effects of soil texture and depth, these analyses were repeated for Pc using a fixed, intermediate rainfall case with \( \alpha = 1.3 \) cm and \( \lambda = 0.3 \) day \( ^{-1} \). The soil texture was varied through the five classes given by Clapp and Hornberger (1978): sand, sandy loam, loamy sand, sandy clay loam, and clay while maintaining the soil depth at 30 cm. The soil depth was then varied from 10- to 120- cm depth on a sandy loam. The resulting PDFs and means of Pc extent were recorded.

**Case Study 1: Phytophthora cinnamomi Range in Western Australia.** One advantage of the analytical framework is that it can be coupled to large-scale data sets of climate...
and soils to make regional predictions. To illustrate the model’s performance at these scales we used it to estimate the range of Pc in Western Australia. Pc was first known as “Jarrah dieback” disease in Western Australia in the 1920s (because Pc most obviously affected the timber species Jarrah, Eucalyptus marginata), and was positively identified as *Phytophthora cinnamomi* in 1965 (Weste and Marks 1987). Although there have been active quarantine efforts in Western Australia, this long disease history has allowed sufficient time for the disease to become established over a wide range in the southwest of the state. Southwest Western Australia is characterized by a strong decreasing rainfall gradient from coastal to inland areas, mirrored by a strongly increasing gradient in evaporative demand from the south coast into the interior. The southwest region has a diversity of soil types and depths. Native vegetation is highly susceptible to Pc disease, with the period of greatest disease risk occurring in the spring, when warming temperatures and moderate rainfall support Pc spread (Tregonning and Fagg 1984; Tippett et al. 1989).

Large-scale soil data for Western Australia are available from the Commonwealth Science and Industry Research Organization (2012) ASRIS data set, while the Australian Bureau of Meteorology provides gridded spatial mean monthly rainfall and mean monthly rain days data (Australian Government Bureau of Meteorology 2007, 2009). The rainfall data used were for the Australian spring period (defined as the months of September, October, and November). We note that although rainfall statistics can vary on a month-by-month basis, assuming fixed statistics on seasonal timescales produces a good approximation to the soil moisture PDFs (Rodriguez-Iturbe and Porporato 2004). The mean rainfall depth \( \alpha \) (cm) was computed by dividing the cumulative mean spring rainfall for each pixel by the cumulative number of rain-days (the mean number of days with rainfall depths of greater than 1 mm recorded). The frequency of the rainfall events (\( \lambda \) days\(^{-1} \)) was taken as the mean number of rain-days divided by the 90 days in the spring period. Mapped average areal evapotranspiration estimates (representative of the spring period) are available as image files (Australian Government Bureau of Meteorology 2005) and were manually digitized. Note that the Western Australian spring follows a cool, rainy winter. Winter rainfall statistics are similar to those in the spring, but evapotranspiration rates are lower. This may lead to an underestimation of the spring soil moisture and thus the pathogen risk during the seasonal transition.

Soil data are available to describe the clay content and soil depth of the A1, A2, B1, B2, and C horizons. The active soil layer was defined as the soils above the first occurrence of a heavy clay (\( \% \text{ clay of } > 45\% \)). The mean soil texture was estimated based on the depth-weighted average of the clay content in each layer. The mean percentage clay content was then used to classify the soils into the soil textural categories of Clapp and Hornberger (1978) to parameterize the soil moisture PDF model. The pathogen growth rates used correspond to the “susceptible” Pc case from table 1. For each approximately 7 × 7-km pixel, the expected value of the Pc severity, \( \Bar{B} \), was computed. We assumed that there would be a proportionality between \( \Bar{B} \) and the observation of population-scale disease at a site. Field and experimental trials indicate that established disease reduces the efficiency of the hydraulic apparatus (stomatal and stem conductances) of *Eucalyptus* species by 40%–60% (Dawson and Weste 1984; Crombie and Tippett 1990). This suggests that aboveground observation of disease symptoms might be most likely where \( \Bar{B} \approx 0.5 \). Accordingly we compared the model output to the mapped disease range of Pc. Maps indicating the contemporary range of Pc in Western Australia were obtained from the Center for *Phytophthora* Science and Management, and locations of Pc digitized for comparison with the model results.

**Case Study 2: Phytophthora cinnamomi Distribution in the Wungong Catchment.** At large scales, the model results reflect both soil and climatic variation. At small scales, climatic variation is relatively minor, and the roles of variable soil depth and texture should become more apparent. To explore the model performance at small scales, it was applied to a region of the Wungong Catchment (located approximately 62 km southeast of the city of Perth, Western Australia). Pc outbreaks in this catchment in the late 1960s were monitored using aerial photography and ground surveys (Batini and Hopkins 1972), allowing publication of a map depicting the presence of Pc disease in the Jarrah Forest over a 5 × 5-km area at a time when approximately 30% of the area was affected by visible Pc symptoms. This snapshot of the progressing Pc epidemic indicates highlights complex spatial patterns in disease emergence which appear to track soil type changes, stream courses, and roads. Climate data for the study region were taken from mapped rainfall averages for late spring, giving \( \lambda = 0.15 \), and a mean 8.5 cm of rainfall (\( \alpha = 0.63 \) cm). Potential evaporation was approximately 40 cm over the same period. Soil data were taken from the ASRIS data set and were processed in the same way as for the large-scale case to generate spatially variable maps of soil depth and textural properties. Again we assumed that the regions of disease establishment should approximately track regions in which likely disease severity was greatest.

**Case Study 3: Phytophthora cinnamomi Severity with Varying Irrigation in “Duke” Cultivars of Blueberry Vaccinium sp.** The previous case studies treat disease severity as a proxy for disease emergence at landscape scales. To address
disease severity directly we applied the model to a Pc outbreak associated with drip-irrigation of "Duke" cultivars of blueberry Vaccinium sp. reported by Bryla and Linderman (2007). Irrigation occurs three times a week at three rates, set to provide 50%, 100%, and 150% of the estimated transpiration demand ($E_{\text{max}}$, 358 mm over the 5-month growing season). Rainfall occurred additionally to the irrigation. To account for the combined effect of rainfall and irrigation water input, we forced the model with a value of $\lambda$ that was derived from the sum of rainfall and irrigation water. In the absence of a time series of irrigation plus rainfall events, we estimated $\lambda$ based on a synthetic time series of observed rainfall and regular irrigation using rainfall records from Corvallis, Oregon (Pacific Northwest Cooperative Agricultural Weather Network 2012). These data suggested that $\lambda_{\text{in situ}}$, for the summer is around 1/13 days$^{-1}$, approximately a rain day every 2 weeks. The frequency of water input from the synthetic time series was $\approx 3.3/7$ days. This value of $\lambda$, with $\alpha$ computed for the combined rainfall and infiltration volumes, was used for the simulation. The active soil depth was 0.3 m, set by the peak rooting depth observed in the study, in a loam soil. In horticultural settings, Pc disease often remains inactive at high water potentials—often as great as or exceeding field capacity (Sterne et al. 1977a, 1977b). We therefore chose to treat $\psi$ as a calibration parameter. “Duke” cultivars are known to be susceptible to Pc (compared to other blueberry varietals), and therefore, the “susceptible host” growth parameters in table 1 were used. Mean soil moisture conditions were reported for the different irrigation rates (lumped across three irrigation types): drip irrigation consistently lead to a 3% higher volumetric water contents than the other irrigation methods used (sprinklers and microsprayers), and we adjusted the reported mean soil moisture accordingly. Root volume data were reported using a root vigor classification, ranging from 5 (where roots were large and vigorous, extending to a maximum root zone extent of 0.3 m) to 1 (roots that barely extended beyond the original root ball, estimated as approximately 5 gallons in volume based on standard pot sizes for the 2-year transplants, used to initiate the study). Normalizing the estimated root ball volume by the maximum volume, $\psi v_{\text{max}}$ ranged from 16% to 100%. This range was converted to pathogen extent $B$ (by taking $B = 1 - \psi v_{\text{max}}$) and then divided into four ranges: 0%–21% (vigor of 4–5); 21%–42% (vigor of 3–4); 42%–63% (vigor of 2–3); 63%–84% (vigor 1–2). The different irrigation rates lead to different root vigors: between 3 and 4 for the lowest irrigation rate, between 2 and 3 for irrigation at 100% of $E_{\text{max}}$, and between 1 and 2 for irrigation at 150% of $E_{\text{max}}$. We adjusted $\psi c$, so that the highest irrigation rate lead to $B \approx 70\%$, and computed the PDFs of soil moisture, the pathogen extent $B$, and the expected values of each of these distributions. We note that the authors also isolated Pythium species of root fungi from the blueberries; however, the relationship between Pythium and root vigor was not statistically significant, and Pythium was also found in individuals with otherwise intact root systems. The alterations in the root system extent are therefore attributed to the action of Phytophthora.

Results and Discussion

Sensitivity Analysis

The response of the pathogen extents and stress in the hosts is shown in figure 2 as a function of variable rainfall statistics. Sharp threshold transitions in pathogen risk and extent are obvious, lying along contours in the $\alpha$, $\lambda$ space. The contours of mean $B$ and $C$ risk approximately track contours of the mean rainfall rate (given by $\alpha \lambda$). This reflects that the timescales of disease development remain relatively long compared to the soil moisture fluctuations, allowing the pathogen response to integrate over the climatic variability. The host response mediated the transition between favorable and unfavorable conditions for Pc and Bd, with this transition being more abrupt for susceptible hosts. Altering host resistance had only a small impact on the predicted prevalence of stem canker disease but led to a large increase in the prevalence of Pc. This presumably reflects that very dry conditions were needed to completely inhibit Pc when $\psi = -3,000$ kPa, so that host resistance played an important role across a wide range of climates. Climate was more generally limiting for Bd. When a combination of pathogen and drought stress was considered it was evident that Bd did not greatly exacerbate the static stress conditions experienced by its hosts: water stress was the dominant factor in determining the host stress. By contrast, drought and pathogen stress acted synergistically in the case of Pc and defined a restricted climatic space in which stress on the plants was minimized. The sensitivity of the pathogen extent to soil texture and soil depth were also explored with respect to Pc. Pc risk increased from $B = 0.1$ to $B = 0.7$ as soil texture was altered from sand to clay while holding rainfall constant ($\alpha = 1.3$ and $\lambda = 0.3$). Increasing soil depth ($Z_r$) tended to increase the Pc risk, but the relative increase in risk per increment in depth declined as the soil depths increased. For instance, doubling soil depth from 10 to 20 cm lead to a fivefold increase in the mean Pc extent (from $B = 0.1$ to $B = 0.5$). Doubling soil depth from 60 to 120 cm under the same conditions, however, caused only an ~8% increase in $B$ (from $B = 0.72$ to $B = 0.78$). These dynamics were largely due to the likelihood of the shallow soils drying out between rainfall events, a dynamic that effectively prevents Pc establishment. The risk of drying
Figure 2: Expected values of the pathogen extent and the resulting plant stress for varying rainfall frequencies ($\lambda$ day$^{-1}$) and mean depths ($\alpha$ cm), using the parameters given in Table 1 assuming a 30-cm root zone in loamy sand soils and $E_{\text{max}}$ is 0.45 cm. The top row shows the pathogen extents for Botryosphaeria dothidea (Bd) (C) in hosts with low susceptibility (i) and high susceptibility (ii), and Phytophthora cinnamomi (Pc) extents (B) in hosts with low and high susceptibility (iii, iv). The bottom row shows the corresponding host stress ($\zeta$) for the pathogen behavior in the panel above it. The stress was assumed to be nonlinear in the pathogen extent, with $q_1 = q_2 = 3$. The color scales are identical for all panels.

The soils decline rapidly as $Z$ increases from 10 cm, but decreases only slightly with increasing $Z$ for deep soils, so that Pc risk depends most sensitively on depth when $Z$ is small.

Case Study 1: Phytophthora cinnamomi Range in Western Australia
A comparison between the predicted Pc severity and observations of Pc disease in the southwest of Western Australia is shown in Figure 3. The results indicate that, in natural (susceptible) vegetation and subject to spring rainfall conditions, Pc would establish and persist in coastal regions throughout the southern and southwestern part of the state. This range declines sharply about 100 km inland and in the northern extent of the modeled range. The model predictions have an excellent correspondence with the reported current range of Pc in Western Australia. While soil properties cause minor differences in the predicted Pc severity at small scales, the large scale trends strongly reflect the gradients in rainfall and potential evapotranspiration in Western Australia. It is likely that this analysis has overstated the absolute value of the Pc risk in the interior of the state: changing vegetation type and lower winter minimum temperatures (which may prevent long-term establishment of Pc) have not been included in the model and would be likely to reduce Pc risks in these areas.

Case Study 2: Phytophthora cinnamomi Distribution in the Wungong Catchment
A comparison between the predicted Pc presence and observations of Pc disease in the Wungong catchment (centered on 32°24’50.37”S, 116°3’3.90”E) is shown in Figure 4. Interpretation of these results needs to account for the fact that the data represent a snapshot of a dynamically progressing epidemic: thus the lack of observations of Pc even in high-risk areas is likely to be associated with the migration of the disease front, rather than indicating a “failure” of the model. The spatial features shown in Figure 4, however, can be interpreted with the assistance of the model.

During winter and early spring, the risk of Pc was uniformly high ($\overline{B} \rightarrow 1$) across the Wungong Catchment. However, during the late spring period (when temperatures warm and Pc growth peaks), the predicted Pc severity was $\overline{B} \approx 0.8$ on deep clay loams (“high-risk areas”), and $\overline{B} \approx
Figure 3: Predicted Phytophthora cinnamomi (Pc) extent (B) in Western Australia assuming sensitive vegetation and forcing the soil moisture model with spring rainfall and evapotranspiration. Circles indicate reported current range of Pc in Western Australia, as reported by the Centre for Phytophthora Science and Management, http://www.cpsm.murdoch.edu.au/.

0.4 on the shallower sandy loams ("low-risk areas"). When these soil regions were compared with the mapped disease extent, we found that Pc infection was observed on 28% of the low-risk area and on 53% of the high-risk area. That is, Pc occurred in low-risk regions at approximately half the rate at which it occurred in high-risk regions. This suggests that while Pc can become established in both soil types, there may have been an overall tendency for greater disease establishment in regions with finer-textured, deep soils. Appealingly, the proportionality in the rate of occurrence is comparable to the proportionality in B between the soil types. A strong association between Pc and the location of roads was visually evident when they are plotted together. However, proximity to roads alone did not explain any patterns in Pc risk through space (the Pc risk is approximately constant at around 40% at all distances from a road). However, when segregated by soil type, distance from a road affected Pc risk. In particular, the data show a large and monotonic decline in Pc risk on low-risk soils, from around 40% adjacent to roads to 10% at distances >200 m from roads. The elevation of Pc risk on low-risk soils near roads may reflect the significance of roads as dispersal corridors for disease and potentially the role of road drainage in locally elevating soil moisture conditions. The effect of the realistic increments of water that could be shed to the surrounding area was analyzed with a simple sensitivity analysis: a localized 10% increase in water availability (\( \alpha \)) on low-risk soils increases the predicted Pc risk by 30%. A 50% increase in water availability on low-risk soils would result in the risk of Pc being equivalent to that of high-risk soils receiving only rainfall. The majority of Pc infection established in low-risk sites was associated with either proximity to Pc infection within a high-risk location or to a roadway, with the exception of the region labeled A (fig. 4). Location A is identified by Batini and Hopkins (1972) as a swampy region. We suspect that a perched water table (a hydrological elaboration not accounted for in the simple one-dimensional water balance used) may help provide locally suitable conditions for Pc at this location. Note also that the scale of soil mapping is coarser than the scale on which Pc outbreaks are mapped and that the location of transitions between soil types should therefore be interpreted with some caution. A final consideration is that some of the simplifying assumptions made in the model could result in a conservative prediction of disease occurrence. For instance, the probabilistic model assumes that there is no feedback between the pathogen condition and the soil moisture dynamics. Since Pc in particular directly affects the hydraulic system of the host plant, this assumption may be problematic in some cases (see supplemental material for a numerical investigation).

Phytophthora cinnamomi Severity with Varying Irrigation in “Duke” Cultivars of Blueberry Vaccinium sp.

The model output associated with this case study is shown in figure 5. Panel A shows the predicted PDF of soil moisture associated with the three irrigation regimes. The predicted PDFs of the pathogen extent are shown in panel B. A clear change in the pathogen behavior is evident between...
Conclusion

The potential for global change to exacerbate the economic, environmental, and social problems associated with plant pathogens is widely acknowledged (Coakley et al. 1999; Chakraborty et al. 2000; Boland et al. 2004; Garrett et al. 2006; Ibanez et al. 2006), as is the importance of resolving the major uncertainties associated with the feedbacks between climatic change and ecosystem changes (Lloyd and Farquhar 2008). Widespread outbreaks of plant disease are clearly one of the mechanisms (along with fire, herbivory, and drought; Allen et al. 2010) that have the potential to result in widespread, rapid, and potentially...
irreversible changes to ecosystems. Developing frameworks that can relate climatic drivers to the risks of such mortality episodes remains an outstanding step in coupling these feedbacks to broader climate change predictions.

The framework presented here provides one approach to coupling physical and biological factors that relate to plant disease risk and impacts on individual trees. It addresses interactions of soil, water, evaporative demand, host, and pathogen properties to generate estimates of disease risk, assuming the pathogen is already present in the host system. Although the one-dimensional nature of the framework means it cannot be used to predict the spatial progression of disease, it appears to predict long-term pathogen ranges effectively based on rainfall, soil, and evaporation limitations alone. As such it is a useful complement to existing approaches that have primarily considered temperature as a control on pathogen ranges (Berg et al. 2004). The framework appears capable of generating predictions of Pc severity as a function of cultural practices in managed conditions with minimal calibration. The model also proved useful as a tool to interpret high-resolution observations of spatial patterns of disease spread. Numerous avenues for future research were identified at these intermediate scales, however, including the value of linking models of pathogen spread to local edaphic conditions, potentially modeling such conditions using high-resolution distributed hydrological models to capture the complexities of water redistribution in sites with shallow water tables or significant riparian areas and the need to further explore the potential for dynamic feedbacks between pathogen impacts and biophysical conditions. Batini and Hopkins (1972), for instance, report that loss of canopy in diseased areas increased soil temperatures and accelerated rates of disease spread. They observed an approximately 10% increase in stream flow from the Wungong Catchment during the period of Pc spread, which they attributed to the disease. These findings, although anecdotal, illustrate the potential for disease to alter the energy and water balances of infected sites, leading to watershed-scale impacts. These feedbacks, and the implications of plant disease for ecosystem function as well as ecological change, remain open for exploration. They would form a particularly interesting comparison with similar natural disturbances that “turn off” canopy transpiration without disturbing soils (and are thus distinct from fire, landslide events, or clear cutting). Such disturbances include pine beetle outbreaks (Brooks et al. 2010) and climate-driven tree mortality events (Guardiola-Clarabantte et al. 2011). These disturbances offer natural experiments with which to explore the dependence of landscape-scale biophysical processes on canopy transpiration and thus the potential land surface feedbacks of climatically induced herbivory, disease, or mortality events.

Although the analytical model developed here required a number of process simplifications, it retains the advantage of producing analytical results that can be applied to large, spatially distributed data sets. It is eminently suitable for coupling with regional-scale climate models. Given the outstanding challenges associated with incorporating ecosystem scale feedbacks into earth system modeling (Lloyd and Farquhar 2008), frameworks of this kind—which make simplifying but justifiable assumptions—provide an approach for synthesizing the complexities of soil, hydrologic, and ecological dynamics into tractable outcomes. Although important theoretical challenges such as linking metrics of plant stress to quantitative predictions of mortality risk remain, stochastic approaches may offer useful avenues for incorporating ecosystem change in large-scale modeling efforts.

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