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Key words: diversity, fluctuation niche, hydrological niche, life history, niche, nutrients, species coexistence, sum of exceedance value.

The nexus of host and pathogen phenology: understanding the disease triangle with climate change

We have observed a remarkable increase in large-scale, *sudden* onset of decline (unknown causes) and known disease (bacterial, fungal, viral) outbreaks in the last few decades, with more predicted globally (Ganley *et al.*, 2009). Increases in temperature, changes in the timing and effectiveness of precipitation, the change in the frequency and intensity of other, catastrophic events (e.g. windthrows, tornadoes, bark beetle outbreaks) and invasions of both native and exotic pathogens have thrown unlikely combinations of host plants, plant pathogens and environmental variability together with unpredicted outcomes. A recent canker outbreak in *Alnus tenuifolia* in interior Alaska, associated with the hot, dry summer of 2004 (Ruess *et al.*, 2009), has refocused attention on the role of temperature and drought in canker incidence (Schoeneweiss, 1975). In this issue of *New Phytologist*, Rohrs-Richey *et al.* (pp. 295–307) open a new line of research in host–pathogen relationships with clarity: an experimental test of the intersection of the phenology of host susceptibility (*Alnus fruticosa*), the life cycle of the pathogen (*Valsa melanodiscus*) and environmental variability (temperature, drought).

'For the first time, Rohrs-Richey et al. *quantified the reduction in transpiration directly attributable to a stem canker.'*

Alnus is a circumpolar, dominant, deciduous broadleaf shrub in the boreal biome. It is the key genus responsible for nitrogen (N) fixation in floodplains of interior Alaska (Ruess *et al.*, 2009). *Alnus* is an important food source for insect herbivores, microtines, rodents and ungulates. Warming at high latitudes, especially interior continental regions, has been twofold the global average (IPCC, 2007). Increased temperatures (both winter and summer) will favor an increase in deciduous woody species, but will probably increase plant drought stress (Chapin *et al.*, 2010).

Within this context, Rohrs-Richey *et al.* conducted two tests of the role of drought stress and host phenology in disease development. The first test was imposed at the peak of growth and the second test was imposed in late summer. At peak growth, significant plant drought stress developed additionally in the low-water treatment as a result of high temperatures and high evaporative demand. In late summer, temperatures and evaporative demand were much lower, and plant drought stress was more moderate than in the first test. Although canker development was greater for isolate 2 in the low-water treatment in the first test, disease severity was greater in the second test when conditions were probably more conducive to pathogen growth and spread within the stem (especially laterally). In the second test, there were significant differences in the severity of disease development between the two isolates, which in the first test were apparent only on a single date (day 60 after inoculation) in the low-water treatment for isolate 2. For the first time, Rohrs-Richey *et al.* quantified the reduction in transpiration to be directly attributable to a stem canker.

This set of experiments demonstrates the importance of the intersection, and the interaction of host plant phenology (and the plant capacity for resistance, tolerance or defense) and the coincidence of optimal environmental conditions for pathogen growth. Their results challenge the role of drought stress in host susceptibility to cankers. Other studies have demonstrated the role of plant host phenology in susceptibility to disease. Active cambial tissue was correlated to successful infection (*Quercus agrifolia* to *Phytophthora ramorum*; Dodd *et al.*, 2008). The timing of lignification after inoculation in peach canker disease was retrospectively analyzed (*Prunus persica* vs *Cytospora*; Biggs, 1997). The role of host phenology was also highlighted in Shaw (2009, pp. S8).

The experimental design in Rohrs-Richey *et al.* was constructed to test the two hypotheses of host susceptibility: plant stress-induced predisposition or through multiple stressor effects on host physiology and thus susceptibility. In this glasshouse experiment, plants in the low-water treatment were not 'predisposed' to pathogens. Inoculation efficiency (successful inoculation/total inoculation) was slightly greater (92% vs 87%), as was ramet mortality attributable to *V. melanodiscus* (1% vs 8%) in test 2 where the overall degree of plant drought stress was moderate (chronic) vs acute in test 1. Disease severity increased with lower overall plant drought stress, but in late summer, the potential for lowered plant defense capacity was untested. There was a slight reduction in the proportion of inoculated ramets that produced callus (70% vs 63%) in late summer, suggesting at least down-regulated morphological defense (e.g. reduced cambial activity in late summer). The authors report some evidence for and against the multiple-stressors hypothesis. The evidence against the multiple-stressors hypothesis was a general depression in maximum photosynthesis: drought stress plus disease was not additive or synergistic. The evidence in support included a steeper quantum efficiency (greater carbon gain per mol of light received) and half the light-saturated photosynthetic rate accompanying plant drought stress plus disease. However, alder was less drought-stressed than perhaps phenologically challenged: the environmental conditions were more favorable to pathogen growth in late summer and were perhaps coupled with the plant host's incapacity to respond (resist, tolerate and defend) to the pathogen in late summer. The pathogen is responsive over a short time-period: weeks to months. They are flexible organisms, particularly adapted to a changing environment. Plant phenological patterns are somewhat flexible within a growing season and somewhat plastic from generation to generation. However, photoperiodism will not be altered by climatic change. Plant programmed response to photoperiod is likely to require many, many generations, even with strong selection such as increased disease incidence. The authors have clarified a new approach to host–pathogen relationships: the nexus between the phenology of the host plant, the life history of the pathogen and the environmental conditions favorable for pathogen development.

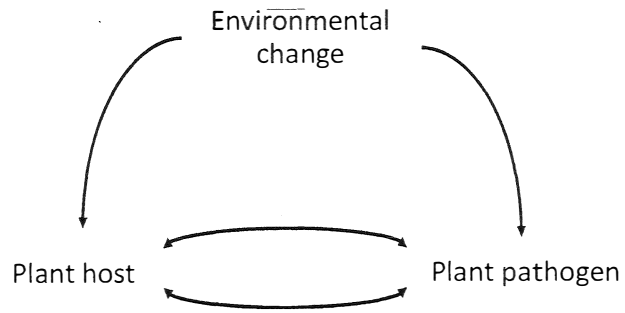

Fig. 1 Modification of the environment–host–pathogen triangle (Stevens, 1960), indicating the importance of interactions between the physiology of the host and the capability of successful inoculation and growth of the pathogen under changing environmental conditions. Important components of environmental change include elevated temperature and greater duration of favorable conditions for plant and pathogen growth; change in atmospheric chemistry; and changes in precipitation patterns, including higher frequency, duration and intensity of drought. Some of the attributes of the plant host that are relevant under changing environmental conditions include earlier onset of bud break and delayed onset of senescence; the potential for earlier and different magnitudes of peak growth; changes in tissue quantity and quality; and the potential for changes in the timing and type of responses to pathogens (plant capacity for resistance, tolerance, and mechanical and chemical defense). It is unclear for most species how environmental cues for phenology (e.g. degree days of warming, photoperiod) may limit the capacity of long-lived plants to synchronize plant growth with new environmental conditions *in situ*. Some of the attributes of the plant pathogen that are relevant under changing environmental conditions include the timing of optimal temperatures for growth relative to optimal moisture conditions, the nutritional status of the plant host (the 'media') and the changes in the cell structure that permit or slow pathogen growth.

Climate change-induced effects on co-occurring factors

Warmer temperatures may lead to higher-water-content snow and to warmer snow that is heavier and remains *in situ* rather than being redistributed at high latitudes. Heavier snow may increase mechanical damage to underlying vegetation. Mechanical damage results in more wounding with subsequent pathogen entry (e.g. *Cupressus macrocarpa* vs *Seiridium cardinal*; Hood *et al.*, 2009). The greater amplitude of temperature changes at high latitudes results in greater freeze–thaw cycles, which also result in greater likelihood of fungal, bacterial or viral entry points (*Betula alba* vs *Botryosphaeria dothidea*; Crist & Schoeneweiss, 1975). Because there is more, heavier snow, patterns of herbivory change: there may be more under-snow herbivory and wounding of stems. Increased temperatures at higher latitudes translate to greater survival rate of beetles, which are vector pathogens. Insect herbivory on stems also increases entry points for pathogens (*Chamaecyparis obtuse* vs *Epinotia granitalis*; Yamada *et al.*, 2000; *Pinus radiata* vs *Aphrophora canadensis*; Storer *et al.*, 1998). Conversely, canker presence increases attack by beetles, and in the case of *P. radiata* in urban ecosystems, an increase in invasive beetle attack (*Dendroctonus valens*; Storer *et al.*, 2002). Higher temperatures increase the abundance of insects already *in situ*; they alter insect herbivores by permitting either new insect species, or new fungal, bacterial, or viral associates of already occurring insects, to move northward. It is these new associates that may have the greatest potential for future sources and magnitude of mortality (Lu *et al.*, 2010).

Implications at the landscape level

An increase in disease is almost always correlated to reduction in biomass and, in alder, to a reduction in N-fixing capabilities (Ruess *et al.*, 2009). Alder occurs along rivers, and their N inputs through N fixation dominate freshwater N dynamics. Dissolved inorganic nitrogen (DIN) and dissolved organic nitrogen (DON) are the primary controllers of freshwater ecosystem productivity and food chains. The recent reduction in alder as a result of canker outbreak in 2004 was estimated to have reduced terrestrial N inputs by 30% (Ruess *et al.*, 2009).

The complexities of interactions in climate–host–disease–insect interactions are particularly difficult to predict, in part because relatively little is known about wildland vs agronomic cankers, the specificity of the attacks (host–pathogen), the diversity of the pathogens themselves (fungal, bacterial or viral) and the likelihood for climate-induced stress, stress-increased susceptibility to pathogen entry and pathogen-induced increases in susceptibility to insects or stress-enhancing effects (original or new stresses). Rohrs-Richey *et al.* open a new line of research in host–pathogen relationships: an intact-plant, experimental test of the intersection and inter-relationships of the phenology of host susceptibility, the life cycle of the pathogen and environmental variability (Fig. 1). Coupling host phenology and patterns of pathogen development will probably permit a more mechanistic approach to model the effects of climate change on disease and insect dynamics at the landscape level.

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Key words: *Alnus*, climate change, disease incidence, pathosystems, taiga, *Valsa*.

Letters

MODIS Enhanced Vegetation Index data do not show greening of Amazon forests during the 2005 drought

Introduction

In a recent Research review, Asner & Alencar (2010), while discussing Samanta *et al.*'s (2010) report on greenness changes of Amazon forests during the drought of 2005, write 'They found little evidence for green-up during the 2005 drought, and showed that the original work of Saleska *et al.* (2007) was irreproducible' without providing details. The purpose of this letter is to inform the readers of the substantive arguments regarding the irreproducibility of the results of Saleska *et al.* (2007), through providing some new results.

Amazonian forests store some 100 billion tons of carbon in woody biomass (Malhi *et al.*, 2006). Their 'dieback' from reduced precipitation in a progressively warming future climate, as suggested by some studies (e.g. Cox *et al.*, 2004; Salazar *et al.*, 2007; Huntingford *et al.*, 2008), can further accelerate global climate change via carbon release (Cox *et al.*, 2000). In the current climatic regime itself, major droughts – such as those associated with the 1983 and 1998 El Nino events – have served as natural experiments of prolonged moisture stress that enhanced tree mortality and forest flammability (Nepstad *et al.*, 2004, 2007). However, there are conflicting reports of forest response to the more recent drought of 2005, which was different from the previous El Nino southern oscillation (ENSO)-related droughts in that it intensified during the dry season as opposed to the wet season, and mainly affected southwestern Amazonia but not the central and eastern parts (Marengo *et al.*, 2008). On the one hand, there are reports of higher tree mortality and reduced growth from field studies (Phillips *et al.*, 2009) and enhanced biomass burning (Aragao *et al.*, 2007), while, on the other hand, satellite-based measurements showed extensive forest 'green-up' during this drought (Saleska *et al.*, 2007). Thus, reconciling these opposing reports has been the goal of several studies (Anderson *et al.*, 2010; Samanta *et al.*, 2010).

Saleska *et al.* (2007) reported that Amazon forests had greened-up during the drought of 2005 based on analysis of 2000–2006 July to September (JAS) collection 4 (C4) Enhanced Vegetation Index (EVI) data from the Moderate Resolution Imaging Spectroradiometer (MODIS) sensor aboard the NASA Terra satellite. The C4 MODIS data sets were superseded by the current collection 5 (C5) data sets and deleted at NASA data centers. Samanta *et al.* (2010) analyzed both C5 (2000–2008 JAS) and C4 (2000–2005 JAS) EVI data sets – the C4 data were provided by K. Didan, an author listed in Saleska *et al.* (2007), but 2006 JAS data and all C4 pixel level quality flags were missing. All other data used in this research are freely available from the Land Processes Distributed Active Archive Center (https://lpdaac.usgs.gov/).

Irreproducibility

Samanta *et al.* (2010) concluded that the results published in Saleska *et al.* (2007) are not reproducible for the following reasons.

- The greening patterns in Fig. 1(b) of Saleska *et al.* (2007) cannot be reproduced with the current C5 EVI data, irrespective of whether or not the data are screened for cloud and aerosol contamination (Fig. 1c,d in Samanta *et al.* (2010),