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REVIEW

Influence of atmospheric and climatic change on plant–pathogen interactions

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Atmospheric change studies conducted in free air concentration enrichment (FACE) systems and open-topped chambers have increased understanding of how factors, such as rising CO_2 and O_3 levels, impact the development of plant disease epidemics. Using these systems, plant scientists have been able to study host/pathogen systems under real-world conditions where variations in multiple environmental parameters impact disease outcomes. Results from these studies are useful for evaluating earlier predictions on plant responses to climate-change parameters and the resulting impacts on plant disease epidemics. Some of these predictions have been verified, whilst others have yet to be tested. Significant interactions among climate-change parameters are highlighting the importance of conducting studies under real-world conditions. The development of molecular and gene expression tools is allowing the fine scale mechanisms responsible for the observed reactions to be determined, and should increase the ability to predict plant disease outcomes under future climatic conditions.

Keywords: carbon dioxide (CO_2), FACE, microarray, ozone (O_3), plant disease, signal transduction

Introduction

With increasing concern about effects of global climate change, plant biologists have devoted greater effort toward studying the impact of various aspects of climate change on the development of plant disease epidemics and the specific mechanisms altering the interactions between plant pathogens and their hosts under field conditions. Plant disease expression results from a three-way interaction of a susceptible host plant, a virulent pathogen and an environment suitable for disease development; referred to as the disease triangle. Changes in environmental conditions are known to exacerbate plant disease symptoms (Boyer, 1995; McElrone *et al.*, 2001) and are implicated in 44% of new disease emergence (Anderson *et al.*, 2004b). Thus, the altered environmental conditions associated with climatic change (e.g. temperature regimes, atmospheric chemistry and drought) have the potential to alter the incidence and severity of plant disease epidemics and disease pressures on natural and crop plant systems, as well as to reshape the co-evolutionary relationships between plants and pathogens (Chakraborty, 2005; Burdon *et al.*, 2006; Fargette *et al.*, 2006; Ziska & Runion, 2007; Crowl *et al.*, 2008).

Previous reviews (Manning & Tiedemann, 1995; Garrett *et al.*, 2006; Ziska & Runion, 2007) have summarized research on the effects of predicted changes in environmental conditions on the development of plant diseases and disease epidemics. This article will build on, rather than re-summarize the information presented in those reviews, by relating information obtained from recently published studies to the trends established by earlier research. Free air concentration enrichment (FACE) facilities, established over the past two decades, have enabled researchers to study plant/pathogen responses to changing atmospheric conditions under field conditions. In this way, interactions with other biotic and abiotic environmental variables have been examined under field conditions. In addition, advances in techniques for monitoring responses at the molecular and gene regulation level are making it possible to more precisely determine how alterations in atmospheric conditions result in changes in disease expression. Plant development and stress responses, while genetically programmed, are also influenced by environmental conditions. Plants adjust to environmental challenges by tightly and differentially regulating their transcriptomes (Baker *et al.*, 1997; Cushman & Bohnert, 2000; Chen

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et al., 2002; Yamaguchi-Shinozaki & Shinozaki, 2006). These alterations in molecular mechanisms determine a plant's ability to respond to internal and external signals and to adjust to changing conditions. More accurate determination of responses on scales from the molecule to the field are allowing scientists to better understand, predict and prepare for the effects of global climate change on plant disease systems.

Plant pathogens vary in the level of host specificity and in the degree of physiological interactions they have with their plant hosts, depending on their mode of infection, and climate-change factors may affect these various pathosystems differently (Runion *et al.*, 1994; Ziska & Runion, 2007). Necrotrophic pathogens, which derive nutrients from killed host tissues, have somewhat limited interactions with the active metabolism of host cells (Schumann & D'Arcy, 2006). Thus, abiotic factors that cause or accelerate tissue necrosis, such as elevated O_3 levels, may favour infection by these types of pathogens. Conversely, biotrophic pathogens, also known as obligate parasites, have extended periods of physiological interaction with their hosts, as they derive nutrients from living cells (Schumann & D'Arcy, 2006). Therefore, the factors that alter plant growth, such as elevated levels of CO_2, may also alter the colonization of host tissues by biotrophic pathogens through changes in host physiology.

Environmental changes can also have direct effects on pathogens, as well as host plants. Pathogen survival in the absence of a host (e.g. overwintering and over-summering) can be affected by temperature, and the processes of propagule production and germination and host infection are often regulated by temperature and moisture conditions (Colhoun, 1973, 1979). Slightly warmer air temperatures, more frequent rainfall events, or initiation of infections earlier in the season can result in more damaging epidemics (Campbell & Madden, 1990). Even changes in the host can alter the microenvironment experienced by the pathogen, as canopy density and structure can affect parameters of temperature, moisture and UV light levels at infection sites. Host responses to environmental changes, such as development of thicker wax layers on leaves or changes in stomatal densities, could also impact the process of infection. After infections are established, temperatures and plant water potentials affect the rate of colonization of host tissues, the production of new inoculum and the expression of symptoms by the host (Colhoun, 1973, 1979; Campbell & Madden, 1990). These types of factors affect both biotrophic and necrotrophic pathogens, and the ability of the pathogens to cause diseases, as do changes resulting in stimulation or suppression of host defence responses.

Some pathogens (e.g. *Xylella fastidiosa*) are able to cause disease on a wide range of host plants belonging to different genera and plant families (Hopkins, 1989), whilst other pathogens can only infect a narrow range of closely related host biotypes (Agrios, 2005). Similarly, host defence reactions can range from non-specific stress-induced responses to very specific reactions based on the presence of individual genes in both the host and pathogen; the gene-for-gene interaction as described by Flor (1971). Climate-change factors also may differentially effect these interactions, as inducement of general defence responses may affect non-specific interactions more than specific ones. However, many host defence responses are tied together through multiple cascading pathways, resulting in similar defence responses to different forms of induction.

Elevated CO_2 and O_3 effects on host plants

Concentrations of CO_2 and tropospheric O_3 have increased markedly since the inception of the industrial revolution, and they will continue to climb well into the 21st century. By 2050, CO_2 concentrations are expected to double the pre-industrial levels, whilst O_3 concentrations are increasing by as much as 2·5% annually and are expected to reach a global mean of >60 nmol mol^{-1} (Prather *et al.*, 2001; Vingarzan, 2004; Solomon *et al.*, 2007). Even today, O_3 can episodically reach levels as high as 100 nmol mol^{-1} (Gard *et al.*, 1998; Houghton *et al.*, 2001). It is well documented that both elevated CO_2 and O_3 alter plant function, but in opposite ways. In general, photosynthetic capacity, water-use efficiency, growth and yield are positively affected by elevated CO_2, but negatively affected by elevated O_3 across a wide range of study species.

Manning & Tiedemann (1995) summarized the responses of plants to future atmospheric conditions and suggested a variety of plant responses that would alter host–pathogen interactions under these scenarios. Since their review, researchers have continued to assess the impact of atmospheric change on plant disease, including expanded studies under realistic growing conditions at FACE facilities. In the last 8 years, numerous meta-analytical studies have also quantified the intensity and consistency of plant responses to elevated CO_2 and O_3 studied singularly. We reviewed and summarized these meta-analyses and other recent FACE studies (Table 1) to determine if plant responses likely to impact host–pathogen interactions (as suggested by Manning & Tiedemann, 1995) actually change in a manner consistent with these predictions across a wide variety of host plants.

Summarized plant responses suggest that whilst stomatal number was generally unaffected by either CO_2 or O_3, stomatal opening was strongly and consistently restricted by physiology (i.e. stomatal conductance, g_s) and by wax occlusion under elevated CO_2 and/or O_3 conditions (Table 1). Stomatal-invading pathogens would encounter smaller infection sites and lower humidity on the leaf surface. Greater quantities of leaf surface wax and alterations in its composition consistently occurred under elevated CO_2 and/or O_3 conditions (Table 1); such changes would likely alter host–pathogen communication/interactions at the molecular scale prior to infection. Enhanced photosynthetic efficiency under elevated CO_2 provided additional carbohydrate supply that strongly and consistently resulted in increased starch and sugar

Table 1 Physiological responses of plants to elevated CO_2 and/or O_3 and predicted changes in pathosystems based on these responses

Altered plant response	Elevated CO_2[a]	Elevated O_3[a]	Elevated $O_2 \times O_3$[a]	Predicted effect on disease	References[b]
Foliar characters and physiology					
Stomatal density	↔	↔ – ↑	↔	Alter infection of stomatal invading pathogens	19, 20
Wax amount/composition	↑	↑↑	↑↑	Alter surface interactions and infection of leaf invading pathogens	12, 13, 15, 18, 19, 22
Wax occlusion of stomata	↑	↑↑	↑↑	Alter surface interactions and infection of leaf invading pathogens	12, 13, 15, 18, 19, 22
g_s[c]	↓↓↓ – ↓↓	↓↓↓ – ↓	↓↓↓ – ↓↓	Alter infection of stomatal invading pathogens	1, 2, 5–7, 13, 16, 17, 26
A[d]	↑↑	↓↓↓ – ↓↓	↓ – ↔	Alter carbohydrate accumulation; alter growth and sporulation of biotrophic pathogens	1, 2, 5, 6, 12, 13, 16, 17, 26
Sugar	↑↑ – ↑↑↑	↓↓	↑↑	Alter growth of sugar dependent pathogens	1, 2, 5, 6, 9, 13, 16, 24
Starch	↑↑↑ –↑↑↑↑	↓↓↓ – ↔	↑↑↑	Alter growth of sugar dependent pathogens	1, 2, 5, 6, 9, 13, 16, 24, 26
Leaf [N]	↓	↔	↓ – ↔	Alter nutritional quality of host tissue	1, 2, 9, 13, 14, 16, 17, 23, 24, 26
Leaf phenolics	↓ – ↑↑	↑ – ↑↑↑	↔ – ↑	Alter nutritional quality of host tissue	10, 13, 14, 23, 24
Necrotic lesions on leaves	NA	↑↑↑	↔ – ↑	Alter no. infection sites for necrotrophic pathogens	12, 13
Aboveground biomass accumulation and yield					
Plant height	↑	↓	↔	Alter canopy environment – humidity, airflow, light levels affecting growth and sporulation of pathogens	1, 2, 5, 6, 13, 16, 26
Leaf number	↑	↔	↔	Alter amount host tissue and canopy environment	1, 2, 13, 16
Leaf area	↑↑↑	↓↓↓ – ↓	↔	Alter amount host tissue and canopy environment	1, 2, 13, 16, 26
Leaf area index	↔ – ↑	↓	↔	Alter canopy environment	1, 2, 13, 16
Shoot biomass	↑↑	↓↓↓	↓↓ – ↔	Alter amount host tissue, canopy environment, and over-wintering potential	1, 2, 4, 5, 6, 8, 13, 16, 26
Total biomass	↑↑ – ↑↑↑	↓↓	↔	Alter amount host tissue and canopy environment	1, 2, 13, 26
Crop yield	↑↑	↓↓↓ – ↓	↓↓ – ↔	Alter amount host tissue	1, 2, 5, 6, 8, 16
Ripening and senescence	↓	↑	↔	Alter infection period and colonization by necrotrophic pathogens	3, 13
Belowground responses					
Root biomass	↑↑↑	↓↓↓ – ↓	↓ – ↔	Alter amount host tissue	2, 4, 5, 6, 8, 11, 13, 16, 25, 26
Root exudation	↑↑↑	NA	NA	Alter pathogen and antagonist activity	19

[a]Arrow direction: ↑, positive response; ↓, negative response; ↔, no significant response; arrow number: one arrow <10%, two arrows 10–20%, three arrows 20–50%, four arrows >50%; arrow number applies to both positive and negative responses.

[b]1. Ainsworth & Long, 2005; 2. Ainsworth *et al.*, 2002; 3. Castro *et al.*, 2009; 4. de Graaff *et al.*, 2006; 5. Feng *et al.*, 2008; 6. Feng *et al.*, 2009; 7. Field *et al.*, 1995; 8.Grantz *et al.*, 2006; 9. Hamilton *et al.*, 2005; 10. Hartley *et al.*, 2000; 11. Jackson *et al.*, 2009; 12. Karnosky *et al.*, 1999; 13. Karnosky *et al.*, 2003; 14. Knepp *et al.*, 2005; 15. Mankovska *et al.*, 2005; 16. Morgan *et al.*, 2003; 17. Noormets *et al.*, 2010; 18. Oksanen *et al.*, 2001; 19. Percy *et al.*, 2002, 2003; 20. Phillips *et al.*, 2009; 21. Reid *et al.*, 2003; 22. Riikonen *et al.*, 2010; 23. Stiling & Cornelissen, 2007; 24. Valkama *et al.*, 2007; 25. Wang & Taub, 2010; 26. Wittig *et al.*, 2009.

[c]g_s = stomatal conductance maximum, measured as maximum the rate of passage of water vapour or carbon dioxide through the stomata.

[d]A = leaf CO_2 assimilation (photosynthetic rate).

levels in leaf tissue (Table 1). Despite an offsetting, opposite response under elevated O_3, where starch and sugar levels decrease with impaired photosynthesis, starch and sugars still increase under a combination of elevated CO_2 and O_3 (Table 1). Enhanced sugar content of leaves under this condition should improve the status of sugar-dependent pathogens. In general, leaf N and phenolic compound levels exhibit non-significant or minimal changes under a combination of elevated CO_2 and O_3 (Table 1).

Above- and belowground biomass accumulation exhibits strong and consistent increases under elevated CO_2 and decreases under elevated O_3 conditions (Table 1). Increases in leaf number, leaf area, canopy size and density under elevated CO_2 can alter canopy temperature and microclimate humidity that should promote fungal diseases. The negative effects of elevated O_3 on plant physiology, growth and yield are primarily linked to decreased photosynthetic capacity caused by damage to the photosynthetic biochemical machinery that result in visible lesions, decreased leaf longevity and premature leaf senescence (Sandermann, 1996; Reichenauer *et al.*, 1998; Miller *et al.*, 1999; Long & Naidu, 2002; Morgan *et al.*, 2004). However, when elevated CO_2 and O_3 are considered in combination, the opposite responses offset for most of the parameters associated with biomass accumulation. Plant height, leaf area, leaf area index, total biomass, ripening and senescence show no significant responses under the combination of conditions, whilst responses of shoot and root biomass and crop yield can be negative but dampened under these conditions (Table 1). In some species inhibition of leaf development and longevity is amplified as O_3 elicits a characteristic stress response in the plant, such as the emission of ethylene and jasmonate, which tend to have negative impacts on leaf development and longevity (Long & Naidu, 2002; Vuorinen *et al.*, 2004). This response may contribute to the continued negative effects under elevated CO_2 and O_3 in combination for some plant parameters (e.g. crop yield and shoot biomass; Table 1).

Elevated CO_2 and O_3 effects on plant–pathogen interactions from recent research

Recent FACE and open-top-chamber (OTC) studies involving plant diseases continue to support earlier findings that show responses to elevated CO_2 levels vary with the host–pathogen system. In some cases predictions of higher disease levels resulting from increased plant growth have been verified, especially for necrotrophic

Table 2 Summary of recent studies assessing the effects of elevated atmospheric CO_2 on plant pathosystems

Effect on disease severity/disease system	Effects on host	References
Lower disease severity or incidence		
Downy mildew of soybean	No changes in stomatal densities; hypothesized changes in g_s; some changes in cuticular wax structure	Eastburn *et al.*, 2010
Late blight of potato	No effect on plant growth or canopy structure; increased levels of β-1-3 glucanases	Plessl *et al.*, 2007
Phyllosticta leaf spot of maple	No change in stomatal density; reduced g_s; altered leaf chemistry; reduced nutritive quality of leaf tissue (elevated C:N ratios)	McElrone *et al.*, 2005
Increased disease severity or incidence		
Powdery mildew of *Arabidopsis*	More stomata on resistant varieties and fewer on susceptible varieties; resistant varieties become more susceptible	Lake & Wade, 2009
Brown spot of soybean	Increased plant height and canopy density	Eastburn *et al.*, 2010
Rice blast	Lower silicon concentrations in leaf tissues under elevated CO_2	Kobayashi *et al.*, 2006
Sheath blight of rice	Increased number of tillers per plant under elevated CO_2	Kobayashi *et al.*, 2006
Crown rot of wheat	Increased plant biomass under elevated CO_2	Melloy *et al.*, 2010
Cercospora leaf spot of redbud and sweet gum	No changes in host chemistry	McElrone *et al.*, 2010
No effect on disease severity or incidence		
Leaf rust on aspen	Increased plant growth and some difference in cuticular wax deposition under elevated CO_2	Karnosky *et al.*, 2002; Percy *et al.*, 2002, 2003
Pyrenopeziza betulicola on silver birch	Reduced stomatal conductance under elevated CO_2, but no changes stomatal density or stomatal index	Riikonen *et al.*, 2008

pathogens. However, there are examples of both necrotrophic and biotrophic pathogens showing lower disease levels, increased disease levels, or no effect of increased concentrations of CO_2 (Table 2).

Studies published in the 1930s showed that CO_2 concentrations can have an effect on several rust diseases of cereal crops. Infections by *Puccinia coronata* on oats, *Puccinia dispersa* on rye, and *Puccinia graminis* f.sp *tritici* and *Puccinia recondita* f.sp *tritici* on wheat were all enhanced by concentrations of CO_2 in the 0·5–0·75% range (Gassner & Straib, 1930; *sensu* Manning & Tiedemann, 1995). However, a more recent study on leaf rust (*Melampsora medusae* f. sp. *tremuloidae*) on aspen trees growing in a FACE system found that elevated levels of CO_2 (560 p.p.m.) had no effect on rust incidence (percentage of leaves infected) and severity (Karnosky *et al.*, 2002; Percy *et al.*, 2002, 2003). The observed impact of elevated CO_2 concentrations on rusts, a group of biotrophic pathogens, remains somewhat mixed.

In a 3-year experiment with soybeans at the SoyFACE facility in Illinois, severity levels of soybean downy mildew, caused by *Peronospora manshurica*, another biotroph, were found to be lower on plants grown under conditions of elevated CO_2 than on those grown with ambient levels of CO_2 (Eastburn *et al.*, 2010). Because this pathogen infects through stomata, there was speculation that elevated CO_2 levels might lower the number of stomates per unit leaf area, thus providing fewer infection sites. However, analysis of leaf surfaces showed no apparent changes in stomatal density associated with atmospheric treatment. There were some changes in the structure of cuticular waxes on the leaves, with leaves on plants grown in a high-CO_2 environment showing a smoother structure than that on leaves from other treatments, although the amount of wax was not changed. Higher CO_2 levels may have reduced the amount of time that stomata were open, which may have reduced the level of infection.

Lake & Wade (2009) found that infection of *Arabidopsis thaliana* by the biotrophic pathogen *Erysiphe cichoracearum* resulted in fewer stomata produced on the infected leaf surfaces, and this effect was enhanced in environments with elevated levels of CO_2. Elevated CO_2 levels were also associated with increased colony establishment by the powdery mildew pathogen. However, this study showed differential responses of resistant and susceptible host ecotypes to elevated CO_2, with stomatal densities (number of stomata per mm^2) of resistant types increasing in response to high CO_2 levels, whilst the densities decreased on susceptible ecotypes. These results suggest that resistant ecotypes may become more susceptible to infection in enhanced CO_2 environments. Thus, once again, there are mixed effects of enhanced CO_2 levels on the development of diseases caused by biotrophic pathogens.

In the soybean FACE study, plants were also evaluated for the development of the necrotroph, *Septoria glycines*, the causal agent of brown spot, over the course of the growing season (Eastburn *et al.*, 2010). Infection by this pathogen begins on the lower-most leaves, and the disease progresses upward through the canopy under conditions of high humidity and extended periods of leaf wetness. Soybean plants grown in the elevated CO_2 environment grew faster than those in the ambient plots; their canopies closed earlier in the season, and the canopies were denser throughout the season. As a result, brown spot progressed up through the canopy more rapidly in the CO_2-enriched plots than it did in the ambient plots.

In a FACE study on rice, Kobayashi *et al.* (2006) evaluated the effects of elevated CO_2 on the development of rice blast on experimental inoculations and natural infections of sheath blight in the plots. Rice plants grown under elevated CO_2 conditions showed an increased susceptibility to the leaf blast phase of the disease in two of the three years of the study. The number of lesions was greater on plants grown in the CO_2-enriched plots compared to ambient conditions. By contrast, elevated CO_2 levels had little to no effect on incidence levels of the panicle blast phase of the disease. Levels of silicon (Si) in the leaf tissues were lower in the CO_2-enriched plots, and the authors speculated that increased leaf blast severity resulted from these reduced leaf Si levels. Incidence levels of naturally occurring sheath blight were higher in the CO_2-enriched plots as well. Plants in the CO_2-enriched plots had greater numbers of tillers per hill, which increased the likelihood of disease spread between more densely packed neighbouring plants. Another FACE study looking at crown rot on wheat found that elevated CO_2 levels resulted in increased plant biomass, as well as increased biomass of the fungal pathogen, *Fusarium pseudograminearum*, and increased stem browning in some situations (Melloy *et al.*, 2010). So, the results from several FACE studies have shown that elevated CO_2 levels fostered greater plant growth, and that growth led to increased disease levels of necrotrophic pathogens.

In climate-chamber-based experiments, atmospheric conditions were evaluated for their effects on the severity of symptoms of late blight on potato, caused by *Phytophthora infestans,* and colonization of tissue by the pathogen, as measured by PCR (Plessl *et al.*, 2007). In this study, potato plants were exposed to CO_2 concentrations of 400 or 700 p.p.m., and O_3 at a fifth, one or two times ambient concentration. Elevated CO_2 reduced the levels of leaf necrosis caused by late blight, and that was linked to increased β-1,3-glucanase activity, which plays a role in disease resistance, in the leaves under elevated CO_2. However, these responses were mediated by increasing concentrations of O_3. The greatest levels of resistance were found under elevated CO_2 and one-fifth the ambient concentration of O_3. As O_3 levels increased to one or two times the ambient level, the beneficial effects of CO_2 decreased. Higher levels of starch and soluble sugars were found in the leaves, and lower levels of starch were found in the tubers under elevated level of O_3, indicating that altered nutrient quality of the leaves and translocation within the plant can impact disease progression under these conditions.

McElrone *et al.* (2005) assessed how elevated CO_2 affects a foliar fungal pathogen, *Phyllosticta minima*, of red maple (*Acer rubrum*) growing in the understory at the Duke FACE experiment in Durham, North Carolina. Surveys of *A. rubrum* saplings from 2002–2004 revealed that elevated CO_2 significantly reduced disease incidence, with fewer saplings and leaves infected per plant in the three consecutive years. Elevated CO_2 also significantly reduced disease severity (i.e. mean lesion area) in infected plants in all years. Scanning electron micrographs (SEM) verified that conidial germ tubes of *P. minima* infect *A. rubrum* leaves by entering through the stomata. Stomatal conductance was reduced by 21–36% under elevated CO_2, providing smaller openings for infecting germ tubes. Reduced disease severity under elevated CO_2 was probably the result of altered leaf chemistry and reduced nutritive quality; elevated CO_2 reduced leaf N and increased the C:N ratio, total phenolics, and tannins.

In contrast to the effects of elevated CO_2, the tissue damage resulting from exposure to elevated O_3 levels might be expected to favour colonization by necrotrophic and opportunistic pathogens. However, more open canopies from decreased plant growth, decreased stomatal openings, or induction of plant defence mechanisms could also act to inhibit infection and disease development, and recent FACE and OTC studies have found mixed effects of O_3 on plant diseases (Table 3).

In the FACE study on soybeans, the effect of elevated O_3 levels on downy mildew, a biotrophic pathogen, were found to lower severity levels of downy mildew (Eastburn *et al.*, 2010), but the effects were not as great or consistent as the reductions seen with elevated CO_2 levels. Again, there was speculation that changes in stomatal opening might play a role in the infection process, as no differences in stomatal densities were found to be associated with the elevated O_3 treatment.

In this same study, elevated O_3 levels had no significant effect on the incidence or severity of septoria brown spot, caused by a necrotrophic pathogen, over a 3-year period (Eastburn *et al.*, 2010). The soybean plants exposed to elevated O_3 were shorter and had a sparser canopy compared to ambient conditions. However, the O_3 levels had little measurable impact on the observed severity levels of brown spot, measured as height of symptoms in the canopy. Thus, the severity of downy mildew, caused by the biotrophic pathogen *P. manshurica,* was reduced with elevated O_3 concentrations, but the severity of brown spot, caused by the necrotrophic pathogen *Septoria glycines*, was not affected by the same treatment.

Systemic infection of soybean plants by *Soybean mosaic virus* (SMV) was also slowed when plants were exposed to elevated levels of O_3, in a different study at the SoyFACE site (Bilgin *et al.*, 2008). High-throughput gene expression analysis showed that the transcript levels of several defence genes increased in response to elevated O_3 levels, and this induced a nonspecific transient defence response against SMV.

European beech (*Fagus sylvatica*) and Norway spruce (*Picea abies*) showed contrasting reactions to their exposure to elevated O_3 concentrations and the root pathogen *Phytophthora citricola* (Luedemann *et al.*, 2009), a necrotrophic pathogen. Exposure of beech trees to both

Table 3 Summary of recent studies assessing the effects of elevated O_3 on plant pathosystems

Effect on disease severity/disease system	Effects on host	Reference
Lower disease severity		
Downy mildew of soybean	Lower canopy density; reduced plant height; possible reduction in g_s	Eastburn *et al.*, 2010
Soybean mosaic	Increased expression of defence genes induced a nonspecific transient defence response against *Soybean mosaic virus*	Bilgin *et al.*, 2008
Phytophthora citricola on European beech	Reduced photosynthesis and nitrogen uptake resulting in reduced leaf and root biomass	Luedemann *et al.*, 2009
Increased disease severity		
Late blight of potato	Increased starch in leaves and reduced starch in tubers under elevated O_3; elevated O_3 negated beneficial effects of elevated CO_2	Plessl *et al.*, 2007
P. citricola on Norway spruce	Minimal reduction of root and shoot biomass under elevated O_3; greater root loss with exposure to both the pathogen and O_3	Luedemann *et al.*, 2009
Pyrenopeziza betulicola on silver birch	Longer guard cell length with elevated O_3 correlated with slightly higher infection density (lesions per area); otherwise no effect on disease	Riikonen *et al.*, 2008
No effect on disease severity		
Brown spot of soybean	Decreased plant height and reduced canopy density, but no effect on disease development	Eastburn *et al.*, 2010

elevated O_3 and *P. citricola* resulted in lower root mass. However, elevated O_3 levels reduced the impact of the pathogen. Reduced photosynthesis, nitrogen uptake, and leaf and root biomass are thought to be directly related to the reduced disease susceptibility of the trees grown in high-O_3 environments. In contrast, Norway spruce is less susceptible to the effects of elevated O_3; spruce tree root biomass was lowest on spruce trees exposed to both elevated O_3 and the pathogen. The authors of this study used these examples to support their hypothesis that when exposure to elevated O_3 shifts the growth-differentiation balance from growth to stress defence activities, the plants become more resistant to certain diseases.

Effects of atmospheric change on plant–pathogen interactions at the molecular level

The dynamics of plant–pathogen interactions are affected by the changes in gene expression (Garrett *et al.*, 2006). Variation in the plant transcriptome may result in changes in the stress response that can shape a plant's ecological interactions. The biological processes altered by abiotic stress have an impact on the defence–growth tradeoff that plants face whenever there is a pathogen attack (Table 4).

To determine the response of various plant species (*A. thaliana*, soybean, *Medicago truncatula*) to elevated levels of O_3, several studies have performed high-throughput transcript analysis (Li *et al.*, 2006; Tosti *et al.*, 2006; Bilgin *et al.*, 2008; Casteel *et al.*, 2008; Puckette *et al.*, 2009; O'Neill *et al.*, 2010). Both with acute exposure to high concentrations of O_3 and chronic exposure at moderately elevated levels, the transcripts of defence-related genes increased; this pattern held for both pathogenesis-related genes (*PR-1*, *PR-2*, *PR-5*, *PR-10*) and enriched disease susceptibility 1, (*EDS1*). Oxidative bursts caused by acute O_3 treatments also affect antagonistic and synergistic interaction between the plant growth regulators ethylene (ET), salicylic acid (SA) and jasmonic acid (JA) and abscisic acid (ABA). Phytohormone-activated defence and reactive oxygen species (ROS) scavenging gene transcripts increased in response to O_3 because of the oxidative stress O_3 induced upon entering the apoplast (Schraudner *et al.*, 1996; Puckette *et al.*, 2009). As part of defence responses, the phenylpropanoid biosynthesis genes such as phenylalanine ammonia-lyase (*PAL*), 4-coumarate:CoA ligase (*4CL*), chalcone synthase (*CHS*), chalcone reductase (*CHR*), chalcone isomerase (*CHI*), isoflavone synthase (*IFS*), isoflavone reductase (*IFR1*), and 2-hydroxyisoflavanone dehydratase (*HID*) were also activated (Bilgin *et al.*, 2008; Casteel *et al.*, 2008).

Elevated CO_2 alters the phenylpropanoid profile as well. However, unlike elevated O_3, elevated CO_2 induces production of scopolin and 4- and 5-*O*-caffeoyl-D-quinic acid (CGA). The increase in these compounds elevates resistance against *Tobacco mosaic virus* (TMV) and the fungus *Cercospora nicotianae* in tobacco (Shadle *et al.*, 2003; Matros *et al.*, 2006). The accumulation of scopolin around the necrotic tissue in an incompatible interaction suggests a role as an antimicrobial compound from which the effective aglycone is released to prevent further spread of the pathogen (Van Etten *et al.*, 1989; La Camera *et al.*, 2004). The major carbon-based secondary compound CGA may be one of the key defence agents against *Potato virus X* in tobacco because neither the SA concentration nor the *PR-1* transcript levels were increased by elevated CO_2 treatment (Matros *et al.*, 2006).

Table 4 Dynamics of biological processes [positive (↑) or negative (↓)] based on transcriptional changes in response to elements of global climate change; table generated from functional annotations provided in various microarray data

	Abiotic stress		
Biological process	O_3	CO_2	Drought
ROS scavenging network	↑	↑	↑
Senescence	↑	↓	↑
Defence	↑	↑	↑
Flavonoid biosynthesis	↑	↑	↑
Photosynthesis	↓	↑	↓
Photorespiration	↓	↑	↓
Amino acid and protein metabolism	↓	↑	↓
Phytohormonal control	↑	↑	↑
Starch and sucrose metabolism	↓	↑	↓
Carbohydrate metabolism	↓	↑	↓
References	Kangasjarvi *et al.*, 1994, 2005; Moeder *et al.*, 2002; Grimmig *et al.*, 2003; Gadjev *et al.*, 2006; Tosti *et al.*, 2006; Bilgin *et al.*, 2008; Casteel *et al.*, 2008; Kontunen-Soppela *et al.*, 2010	Taylor *et al.*, 2005; Ainsworth *et al.*, 2006; De Souza *et al.*, 2008; Cseke *et al.*, 2009; Fukayama *et al.*, 2009; Leakey *et al.*, 2009	Rabbani *et al.*, 2003; Rook *et al.*, 2006; Watkinson *et al.*, 2006; Huang *et al.*, 2008; Jain & Chattopadhyay, 2010; Zheng *et al.*, 2010

Table 5 Selection of soybean transcripts differentially regulated by *Soybean mosaic virus* (SMV) infection, elevated O_3 treatment and combination of SMV and O_3 at 8 and 72 h post-infection (h.p.i.); shaded values present the 72-h.p.i. results; data from Bilgin *et al.*, 2008

	Transcript fold change (log_2), FDR *P*-value<0·01			
Gene description	Affymetrix ID	SMV	O_3	SMV + O_3
Defence-related genes				
PR1a precursor	GmaAffx.36484.1.S1_s_at	–	–	1·59
PR10 (Stress-induced protein SAM22)	Gma.6999.3.S1_s_at	2·79	1·38	3·33
EDS1 putative	GmaAffx.80842.1.S1_at	–	1·00	1·64
Disease resistance gene (RPP13-like)	GmaAffx.57686.1.S1_at	–	–	1·54
Disease resistance gene (RPM1)	GmaAffx.67437.1.S1_at	–	–	1·75
TIR-NBS disease resistance-like protein	GmaAffx.92922.1.S1_at	–	–	1·73
Beta-1,3-glucanase 3	Gma.7852.1.S1_at	–	–	1·14
Chitinase class I	Gma.2833.1.S1_s_at	–	–	1·0
SRG1 (senescence related gene1) oxidoreductase	Gma.8240.1.S1_at	–	1·52	1·20
Oxidative stress-related genes				
Alternative oxidase	Gma.1439.1.S1_at	–	–	1·96
Ascorbate oxidase precursor	GmaAffx.47157.1.A1_at	–	1·26	2·26
Peroxidase	GmaAffx.44031.1.S1_at	–	1·55	1·01
Peroxidase 4 precursor	Gma.5629.1.S1_at	1·51	–	2·04
GST 12	Gma.2730.1.S1_at	–	–	1·55
Ethylene signallin- related genes				
ERF-like protein	Gma.12330.1.S1_s_at	–	–	1·26
ERF-like protein	Gma.12330.2.S1_s_at	–	–	1·12
ATERF5	Gma.15943.2.S1_at	–	–	–
ATERF5	Gma.5293.1.S1_a_at	–	–	–
Transcription factor EIL1	GmaAffx.13724.1.S1_s_at	1·98	1·84	1·68
Transcription factor EIL1	GmaAffx.93218.1.S1_at	1·85	1·75	1·64
Transcription factor EIL2	Gma.2347.2.S1_at	2·35	2·13	2·18
EBF1 (EIN3-binding F box 1)	GmaAffx.54144.1.S1_at	2·79	2·67	2·64
Jasmonic acid signalling-related genes				
Lipoxygenase LOX1	GmaAffx.45573.1.S1_at	2·27	2·09	1·78
Lipoxygenase (AtLox2)	Gma.2382.1.S1_s_at	–	–	1·72
12-oxophytodienoate reductase	GmaAffx.6142.1.S1_at	1·16	–	–
NAC transcription factors				
NAC domain protein NAC1	GmaAffx.32328.1.A1_at	–	1·90	−1·44
NAC domain protein NAC2	Gma.4774.2.S1_at	−1·53	1·47	−1·33
NAC domain protein NAC3	GmaAffx.57970.1.S1_at	−1·61	2·27	−2·16
NAC domain protein NAC3	Gma.1649.1.A1_at	–	2·18	−1·34
NAC domain protein NAC4	Gma.5331.1.S1_at	−1·23	2·84	−1·79
NAC family protein	GmaAffx.90028.1.S1_s_at	−1·64	1·59	−1·22
NAC family protein	Gma.5519.1.S1_at	−1·60	1·63	−1·33
Putative NAC domain protein	Gma.5519.3.S1_x_at	−1·07	–	–

Many preformed and induced anti-pathogen defence systems involve activation of the same or overlapping signaling pathways with abiotic stresses (Kariola *et al.*, 2006; Lee *et al.*, 2006a,b; Sohn *et al.*, 2006; Jung *et al.*, 2007). The stress-induced plant response may induce defence responses and enhance tolerance for a second stress factor. The early stages of stress induced signal transduction pathways may involve specificity against the stress. However, downstream events involving stress adaptation may share common genes and nodes in biotic and abiotic stress responses. Long-term elevated O_3 treatment of soybeans altered many defence-related gene transcripts (Table 5). The highest gene expression change was observed when the plants were exposed to both stresses at the same time. Both O_3 treatment and SMV infection increased the transcript levels of various *PR* genes, ethylene-signalling genes and decreased the *NAC* (*NAM*, *ATAF* and *CUC*) transcription factors, which mainly function in plant growth and development (Xie *et al.*, 2000; Kim *et al.*, 2006; Zhong *et al.*, 2006). This example supports the use of common genes and signalling pathways in response to abiotic and biotic stresses (Table 5).

In particular, plant defence responses to pathogen infection and elevated O_3 share many commonalities (Singh *et al.*, 2002; Mittler, 2006). Biphasic ROS accumulation was observed during plant incompatible defence responses such as non-host and *R*-gene-mediated resistance against pathogens. The ROS accumulation was associated with programmed cell death (PCD) as part of the hypersensitive response (HR) (Tenhaken *et al.*,

1995; Thordal-Christensen *et al.*, 1997; Dat *et al.*, 2003). The oxidative burst generated at the early stages of HR resembles oxidative stress caused by O_3. Acute O_3 (150–300 nmol mol^{-1} for 4–6 h) induces HR-like localized cell death in plants, similar to the *R*-gene-mediated host resistance response (Rao & Davis, 1999; Overmyer *et al.*, 2005). Upon pathogen attack, extracellular H_2O_2 is produced at the site of infection at concentrations that vary by plant species and developmental stage. H_2O_2 accumulation can serve as a diffusible signal-transducing molecule in response to pathogen infection (Foyer & Harbinson, 1997) and was considered an antimicrobial agent. The oxidative burst caused by elevated O_3 elicits defence signalling pathways similar to pathogen defence. The antagonistic and synergistic interactions between the plant growth regulators JA, SA, ET and ABA are also affected by oxidative bursts caused by acute O_3 treatments. Ozone reacts primarily with apoplastic fluids and cell membranes, which causes changes in the lipid composition and increases the production of linoleic acid (Mudd, 1998). The biosynthesis of JA triggered by excess linoleic acid may attenuate SA-dependent HR and cell death (Rao *et al.*, 2000a,b). On the other hand, functional SA-signaling pathways are required for O_3-induced ET biosynthesis and induction of HR-like cell death in *A. thaliana* (Rao *et al.*, 2002). An increase in the O_3-induced biosynthesis of ET production boosts the biosynthesis of ABA, which regulates processes such as glucose signalling and stomatal conductance in plants (Leon & Sheen, 2003; Ahlfors *et al.*, 2004). The similarities in the induction of defence signalling pathways can affect the plant defence response and the incidence and severity of plant diseases caused by biotic agents.

Drought and temperature effects on plant–pathogen interactions

Evidence for global climate warming is clear and now widely accepted, and models predict that altered precipitation regimes will accompany the increased temperature in many regions (Solomon *et al.*, 2007). Drought and temperature stresses resulting from climate change will directly affect plant growth and survival, as well as plant disease development, as it is closely tied to these parameters (e.g. leaf wetness duration, relative humidity, soil water potential) (Colhoun, 1973, 1979). These environmental factors can affect pathogens directly by altering spore germination and hyphal growth rates or by affecting the rate of inoculum production (Schnathorst & Mathre, 1966; Huber & Gillespie, 1992). Moisture and temperature can also impact disease development by affecting the susceptibility of the host to infection. Reviews by Boyer (1995) and Schoeneweiss (1975) have illustrated that a predisposition to disease is often observed in host plants experiencing soil water deficits, and more recent work on other pathosystems has revealed similar increases in disease expression under water stress conditions (McElrone *et al.*, 2001; Suleman *et al.*, 2001). Boyer (1995) proposed the following two mechanisms to explain how water stress increases the susceptibility of plants to attack by pathogens: (i) reduced photosynthate production induced by drought eliminates the plant's ability to produce defensive compounds; or (ii) plant growth is reduced without reducing the pathogen's ability to reproduce, thus allowing further progression and increased symptom severity in the host.

Conversely, many plant diseases are less severe under low moisture conditions. Notably, plant diseases caused by some oomycetes e.g. *Phytophthora* and *Pythium* spp. and the downy mildew fungi, are less severe when moisture levels are low (Duniway, 1983; Agrios, 2005), and many foliar diseases require extended periods of leaf wetness for infection to take place (Huber & Gillespie, 1992). For soilborne, root-infecting diseases, low soil moisture levels can restrict root growth, thus reducing the chances that a root system will come into contact with propagules of soilborne pathogens and lowering the incidence of infection (Huisman, 1982; Gilligan, 1983).

Plant responses to drought and temperature stress have been shown to impact other stress responses, including responses to pathogens. Drought stress activates the ABA-responsive signalling pathway and alters response to biotic stresses. ABA mediates long-distance signalling within the plant and stomatal closure (Zhang *et al.*, 2006). Changes in endogenous ABA levels affect SA-, JA- and ET-related defence responses (Kariola *et al.*, 2006; Asselbergh *et al.*, 2008; Zavala *et al.*, 2009). In *Arabidopsis*, the *Early Responsive to Dehydration 15* gene (*ERD15*) is rapidly induced in response to drought and pathogen infection (Kariola *et al.*, 2006). The overexpression and silencing of the *ERD15* gene not only affected abiotic stress tolerance but also disease resistance.

ABA has a negative effect on SA accumulation and SA-induced defence responses to pathogen infection. Incompatible interaction of *Arabidopsis* and *Pseudomonas syringae* is prevented by exogenous ABA treatment (Mohr & Cahill, 2007). Tomato ABA-deficient mutant high-throughput gene expression analysis showed increase in the transcript levels of SA-inducible *Pathogenesis Related-1* (*PR-1*) gene, in compatible and incompatible defence responses against *Botrytis cinerea* (Asselbergh *et al.*, 2007). Exogenous ABA application down-regulated SA-inducible *Pathogenesis Related-2* gene (*PR-2*/β-1,3-glucanase) in tobacco cell cultures (Rezzonico *et al.*, 1998). Although ABA suppresses SA-induced defence responses, ABA-induced stomatal closure is part of SA-induced innate immunity to bacteria (Melotto *et al.*, 2006; Underwood *et al.*, 2007). Upon recognition of plant pathogenic and non-pathogenic bacteria, ABA-induced stomatal closure occurs. As part of the infection strategy, *P. syringae* pv. *tomato* virulence factor coronatine mimics JA and inhibits stomatal closure to enter internal leaf tissue (Melotto *et al.*, 2006).

The interaction between SA and ABA can be explained by the effect of ABA on JA/ET biosynthesis and signalling. It has been suggested that ABA induces

JA biosynthesis and indirectly suppresses SA-induced defence (Adie *et al.*, 2007). Another indirect effect is ABA-induced callose formation. Callose deposition blocks SA-inducible defence responses (Nishimura *et al.*, 2003). Also, ABA suppresses phenylpropanoid (PAL) activity and ROS production, which are the major defence processes against pathogens (Ward *et al.*, 1989; McDonald & Cahill, 1999; Audenaert *et al.*, 2002; Asselbergh *et al.*, 2007, 2008). ABA has both negative and positive effects on JA and ET signalling, and any alteration in its biosynthesis may affect the plant defence response to pathogens. Exogenous ABA treatment suppressed JA/ET-induced gene expression, such as *PDF1.2* (plant defensin 1.2), *CHI* (chitinase) and *HEL* (hevein-like/PR-4). These genes were up-regulated in an ABA-deficient mutant and resistance was stronger against *Fusarium oxysporum* (Anderson *et al.*, 2004a; Mauch-Mani & Mauch, 2005). These examples show that drought can dramatically affect plant defence responses against pathogens.

Climate change scenarios predict a 1–3·5°C increase in global average temperature and a 20% change in precipitation regimes for many regions by 2099 (Solomon *et al.*, 2007). The altered air temperatures are predicted to significantly impact plant disease systems (Chakraborty *et al.*, 2000b; Chakraborty, 2001). Estimates of the amounts of downy mildew likely to develop on grapevine grown in northern Italy in the years 2030, 2050 and 2080, were determined based on the IPCC predictions of climatic conditions in those years (Salinari *et al.*, 2006). Estimated climatic conditions were used in a disease prediction model, and the levels of downy mildew were predicted to increase in a climate with increased temperatures and decreased levels of rainfall. Even though the pathogen is dependent on leaf wetness for infection, the increase in temperature more than compensated for the reduction in rainfall, in part because infections would initiate earlier in the growing season, allowing more time for epidemics to develop. Other recent modelling studies have reported similar patterns for most pathosystems, with increasing temperatures consistently resulting in greater occurrence of pathogens and disease development, but in many cases being counterbalanced by decreased occurrence of pathogens and disease development under lower precipitation conditions (Desprez-Loustau *et al.*, 2007).

Resistance responses to two viral pathogens were found to be reduced as temperatures increased from 22 to 28 or 30°C. One set of evaluations was carried out on the leaves of tobacco plants (*Nicotiana benthamiana*) infiltrated with *Agrobacterium tumefaciens* strains containing the *Rx* gene for *Potato virus X* (PVX) resistance and its elicitor, the PVX coat protein. Another set of tobacco leaves were infiltrated with *A. tumefaciens* strains containing the N gene for TMV resistance and its elicitor, the TMV helicase fragment (p50). In both cases HR reactions developed rapidly on plants incubated at 22°C, but not on plants incubated at 28 or 30°C (Wang *et al.*, 2009). Thus, the resistant plants may become partially susceptible to these viruses as the temperature increases.

In genotype trials of wheat conducted in Bangladesh, India and Nepal, yield and plant susceptibility to spot blotch, caused by the fungus *Cochliobolus sativus*, were evaluated over a 6-year period. Over the course of that study, regional temperatures gradually increased. These higher temperatures, especially the night-time temperatures in March, were positively correlated with an overall increase in spot blotch severity and reductions in yield (Sharma *et al.*, 2007). The warmer night-time temperatures in March allowed for the initiation of epidemics of spot blotch earlier in the growing season, resulting in greater yield losses. These results led the authors to suggest that global warming is already causing crop yield losses as a result of elevated disease severity on wheat in Southeast Asia.

Increasing temperatures from 22 to 28°C reduced the effectiveness of both basal and R-gene mediated resistance in *A. thaliana* when challenged with virulent and avirulent strains of *P. syringae* pv. *tomato* respectively (Wang *et al.*, 2009). Increased levels of symptom expression were found to be the result of changes in the defence responses associated with the host–pathogen interaction, rather than just an increase in pathogen growth at the higher temperature. Thus, increasing temperatures may result in an increase in disease as a result of reductions in host resistance responses.

Knowledge gaps

Atmospheric change and resultant climatic shifts do not occur in isolation and may intensify in the future. However, only a few studies have attempted to evaluate the combined effects of multiple climate factors. Over the three seasons of the SoyFACE experiment, Eastburn *et al.* (2010) showed that although severity levels of two foliar diseases (downy mildew and brown spot) were affected by changes in CO_2 and O_3 in some years, the effects varied with seasonal temperature and rainfall patterns. McElrone *et al.* (2010) also looked at the interactive effects of CO_2, rainfall and temperature on foliar diseases of redbud (*Cercis canadensis*) and sweetgum (*Liquidambar styraciflua*) trees. Climatic data varied considerably between the 5 years and altered disease expression with greater incidence and severity for both tree species in years with above-average rainfall. Disease incidence for sweetgum decreased significantly in years with above-average temperatures, and when significant changes did occur, disease incidence and severity always increased under elevated CO_2. However, increased disease severity resulting from elevated CO_2 was offset by higher photosynthetic efficiency in the remaining undamaged leaf tissues (McElrone *et al.*, 2010).

Focusing on only one factor, such as CO_2, may result in conclusions that are not applicable over a range of different environments and conditions (see also Wang & Taub, 2010). The importance of studying host and pathogen responses under combined stress conditions is well illus-

trated by the offsetting effects of concurrent elevated CO_2 and O_3 on host plant responses (Table 1). Even in studies where interactions are investigated, data may not allow for a rigorous evaluation of the interactions. One article noted, "There were too few observations of the interaction of O_3 with elevated CO_2 and drought to conclusively project how these climate change factors will alter tree responses to O_3" (Wittig *et al.*, 2009). Studies designed to look at multiple environmental factors have found interesting interactions. For example, a study looking at the effects of elevated CO_2, nitrogen application and water supply on powdery mildew on wheat showed that levels of disease severity were reduced in the elevated-CO_2 environment. However, mildew levels increased with increasing leaf nitrogen concentrations and were reduced with increased leaf water content (Thompson *et al.*, 1993). In fact, the authors assert that the result of this study showed that the reason that mildew levels were lower in the elevated-CO_2 environment was that the elevated CO_2 levels caused the nitrogen levels in the leaf tissue to decline and the water content to increase, and that the water content factor had the greatest influence on mildew severity.

Estimates of the effects of climate change on plant disease epidemics based on climate models often focus on a few factors, such as temperature and precipitation. However, their conclusions often do not account for any offsetting interactions as a result of atmospheric change. The earlier described study in which increased severity levels of downy mildew of grape were predicted for northwestern Italy based on climate-model-based predictions of temperature and rainfall (Salinari *et al.*, 2006) did not factor in disease responses to levels of CO_2 and O_3 predicted for the same time period. However, the soybean FACE experiment found lower levels of downy mildew resulting from increased atmospheric levels of CO_2 and O_3. Which of these factors will prove to be more influential has yet to be determined, but some recent studies suggest that climatic impacts resulting from atmospheric change will be more important drivers of disease responses (e.g. McElrone *et al.*, 2010). More studies that include several climatic factors, including atmospheric composition, temperature and moisture levels, need to be undertaken to improve our understanding of how the interaction of these different factors will impact the development of plant disease epidemics. Modelling efforts provide an excellent opportunity to simulate multiple factors simultaneously (Jeger & Pautasso, 2008). Mechanistic studies can be used to clearly identify the change in disease to help find some unifying themes. Molecular analyses may help with these inquiries.

Despite the recent flurry of studies evaluating foliar disease response to climate change conditions, little work has been done to evaluate the effect these environmental changes will have on soilborne, root-infecting plant pathogens and the diseases they cause. A recent review article (French *et al.*, 2009) summarizes several studies that have looked at the effects of elevated atmospheric levels of CO_2 on general microbial activity in soil and the rhizosphere, showing changes in respiration rates, enzyme activity levels and community structure, but studies on soilborne pathogen systems need to be conducted as well (see Pritchard, 2011).

Conclusions

Climate-change studies over the past decade have increased understanding of how factors such as rising CO_2 and O_3 levels will impact the development of plant disease epidemics. The establishment of FACE experiments has allowed plant scientists to study host–pathogen systems under real-world conditions where variations in multiple environmental parameters impact disease outcomes. The development of molecular and gene expression tools has also allowed us to go beyond speculation of the fine-scale mechanisms responsible for the observed reactions.

In 1995, Manning & Tiedemann predicted how plant hosts would respond to elevated CO_2 levels and how these responses would, in turn, affect disease development. Several of these predictions have been verified for some host–pathogen systems. Increases in plant biomass and canopy density resulting from elevated CO_2 levels have been found to be related to higher levels of some diseases, especially those caused by necrotrophic pathogens. Reduced stomatal opening also appears to be associated with lower infection rates with some diseases. Other predicted plant responses, such as increased crop residues resulting in increased rates of pathogen survival, or accelerated ripening and senescence promoting necrotrophic pathogens, have yet to be adequately studied.

The mixed responses to changing climatic conditions observed in pre-2000 studies have continued to occur with more recent investigations. The FACE and growth-chamber studies continue to demonstrate the generally positive effects of elevated CO_2 and negative effects of elevated O_3 on plant growth parameters. However, disease responses continue to vary with pathogen and host, sometimes varying between disease-resistant and susceptible biotypes of the same plant host.

Some of the published research has included investigations on the interactions of multiple environmental parameters, and in many cases the benefits of elevated CO_2 are offset by the negative effects of elevated O_3 levels. In some cases, plant disease responses to one environmental parameter change significantly when another parameter is introduced. However, despite the call for more research over 10 years ago, we still lack ample data assessing the interactive effects of various climate-change factors. Recent multi-year studies (e.g. McElrone *et al.*, 2010) have enabled researchers to assess the interactive effects that natural variation in air temperature and precipitation regimes can have with changing atmospheric chemistry (see also suggestions of Jeger & Pautasso, 2008). In the future, factorial studies should be incorporated into FACE facilities, with subplots that restrict precipitation (e.g. rainout treatments) and simulate increasing temperature as predicted by climate-change

models (e.g. warming lamps) to evaluate these effects on a shorter term. Such studies also would allow researchers to determine the cumulative effects of these treatments over time and their effects on interannual pathogen population dynamics, similar to those described by Chakraborty *et al.* (2000a). Some of these studies have already been initiated, and results should be available in the next few years.

Progress in understanding the effects of climate change on foliar pathogens, seen over the past 10 years, has not been matched by progress in understanding the effects on soilborne pathogen systems. A few FACE studies have found no significant effects of elevated CO_2 or O_3 on soilborne diseases. The nature of these pathogens, and the fact that they infect root systems, makes data acquisition more difficult, especially in multidisciplinary experiments where destructive sampling may not be appreciated by other researchers using the same sets of plants. However, root-system health is just as important as the health of the aboveground plant parts, and more attention needs to be directed towards these pathogen systems.

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