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Review

Impacts of Global Change on Diseases of Agricultural Crops and Forest Trees

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Abstract

The fourth assessment report of the Intergovernmental Panel on Climate Change projects rising levels of greenhouse gas and global temperature. The well-known dependence of plant diseases on weather has long been exploited for predicting epidemics and to time applications of control measures for tactical disease management. Fingerprints of inter-annual climatic variation on pathogens have recently been shown in literature linking pathogen abundance to atmospheric composition. Past reviews have dealt with impacts of changing atmospheric composition and climate on diseases, regional or country-wide assessments of climate change impacts and impacts on specific disease/pathogen or pathogen groups. All agree on paucity of knowledge prompting a need to generate new empirical data on host–pathogen biology under a changing climate. Focused on experimental research, the purpose of this review is to summarize published and unpublished studies on plant pathogens and diseases in free-air CO_2 enrichment (FACE) facilities and open top chambers and other current non-FACE research to offer a summary of future research needs and opportunities. Critical review of recent literature on the influence of elevated CO_2 and O_3 on agriculture and forestry species forms a major part of the treatise. Summaries of unpublished or ongoing experimental research on plant pathogens from FACE studies are included as a catalogue of work in this neglected area. The catalogue and knowledge gaps are intended as a resource for workers initiating research in this area as well as the general scientific community grappling with the design and scope of next generation of FACE facilities.

Keywords: FACE, Climate change, Plant pathogens, Plant disease, CO_2, Ozone

Review Methodology: Existing recent reviews on the subject have served as a useful input. More recent literature has been searched using the Web of Science, Scopus, CAB Abstracts and the CSIRO electronic journals collection. Search terms used: CO_2 and plant disease, CO_2 and plant pathogen, climate change and plant disease; climate change and plant pathogen. Published and unpublished information from researchers in the field has been accessed through personal communication.

Atmospheric Composition and Global Climate are Changing

Global change encompasses all changes in climate, land, oceans, atmospheric composition and chemistry, and ecological systems that influence the global environment. It is a comprehensive view of our changing planet and includes climate change. The interactions between atmosphere, hydrosphere, cryosphere and biosphere as driven by solar radiation make our earth's climate. A part of the radiation reaching the earth is absorbed to heat up the earth's surface and some is radiated back to space. The oceans, covering over 70% of the earth's surface, absorb solar energy; while snow and ice reflect some 60–90% of the solar energy. The reflected radiation is trapped by radiatively active water vapour, CO_2, CH_4, N_2O and O_3 in the atmosphere, acting like the glass of a greenhouse that warms the earth's surface. The longevity and radiative efficiency of these greenhouse gases determine their global-warming potential. CO_2 has the highest radiative forcing, at 1.66 W/m^2, followed by CH_4 at 0.48 W/m^2 and some of the perflourinated compounds have the longest lifetimes (summarized in [1]). Perfluorocarbons are by-products of aluminium smelting, uranium enrichment and semiconductor manufacturing. Their global-warming potential is 6500–9200 times that of CO_2. Vehicular and industrial emissions and other processes raise the concentration of greenhouse gas, and large-scale destruction of vegetation cover reduces CO_2 sequestration; both processes increase radiative forcing and cause global warming.

The fourth assessment report of the Intergovernmental Panel on Climate Change [1] now provides comprehensive analysis, among others, of changes in greenhouse gas composition and global temperature using multiple lines of evidence. However, despite strong scientific evidence for global warming, uncertainties surrounding future greenhouse gas emissions and difficulties in determining patterns of climate change at a smaller scale from global estimates have persisted. Here, we outline a brief summary of issues relevant to this review and direct readers to the IPCC assessment for detailed analyses.

The global atmospheric CO_2 concentration has increased from about 280 ppm since the pre-industrial times (1750) to 379 ppm in 2005. This concentration exceeds the 180–300 ppm range observed from air pockets trapped within ice cores for over the last 650,000 years. Atmospheric concentration of other greenhouse gases including N_2O and CH_4 has also increased since pre-industrial times. The combined radiative forcing resulting from increases in greenhouse gases has led to a warming of the globe. For example, CO_2 radiative forcing has increased by 20% from 1995 to 2005 [1].

Although there is uncertainty surrounding the projection of future warming and other changes in climate, since 1990, when the first IPCC report was published, actual increases in the global temperature per decade have been within the range of projected increase of between 0.15 and 0.3°C per decade. The fourth IPCC assessment report projects a 0.2°C warming per decade for the next two decades for a range of IPCC emission scenarios originally outlined in IPCC [2]. Other changes in climate include rising sea level, shrinking of glaciers and increased rainfall in the middle and high latitudes of the Northern Hemisphere but a decrease over the sub-tropics.

History shows that the impacts of climate change have been a lot more far-reaching than simply adding to the challenges of producing food, fibre, timber and animal feed for the burgeoning human population. Changes in climate, namely temperature fluctuations, have influenced the frequency of war and population changes, according to an analysis of paleo-climatic data [3]. Impeded agricultural production as a result of cooling has brought about a series of social problems including price inflation, outbreak of war and famine with subsequent declines in human populations. For agriculture and forestry industries, pests and diseases routinely lower production and quality despite significant gains in the development of improved plant varieties and farming practices [4]. How the adaptive capacity of agriculture and forestry industries would be affected by shifting disease dynamics resulting from the changing climate is worthy of consideration for social, economic and political reasons.

Fingerprints of Global Change on Pathogen and Disease Dynamics

There are numerous disease-forecasting models that use short-term weather data for tactical disease management, but very few models/studies link disease prevalence or severity to long-term climate data. Trawling for example of plant diseases driven by multiyear climatic cycles is an essential first step to find fingerprints of slow, decadal global change on disease dynamics. Links between the El Niño-Southern Oscillation (ENSO) and wheat scab (*Fusarium graminearum*) prevalence in China and between ENSO and wheat stripe rust in China and stem rust in the USA [5] serve as examples.

As emphasized by Jeger and Pautasso [6], the availability of long-term data sets is a prerequisite for finding fingerprints of inter-annual climatic variation on plant diseases. The long-term Broadbalk experiment on wheat at Rothamsted in the UK is an excellent example of long-term data and archived plant samples since 1844. Two recent works have used this resource spanning over 160 years, applying polymerase chain reaction methods, for demonstrating a link between fluctuations in two wheat pathogens *Phaeosphaeria nodorum* and *Mycosphaerella graminicola* and changes in spring rainfall, summer temperature and SO_2 emission [7, 8]. Long-term variation in DNA content of these two pathogens in leaf and grain samples were determined by weather factors occurring over a period longer than the growing season. In a recent study that used datasets spanning 69 years Hannukkala *et al.* [9] has linked the early occurrence and increased frequency of potato late blight epidemics in Finland to the effects of climate change and a lack of rotation crops. Fungicide use in Finland increased 4-fold from 1980 to 2002 because of more frequent and earlier epidemics, although the aggressiveness of *Phytophthora infestans* isolates only had a minor effect on the onset of epidemics after 1991. Using datasets covering only 40–50 years, Woods *et al.* [10] have found a strong relationship between increase in summer rainfall and *Dothistroma septosporum* needle blight of *Pinus contorta* var. *latifolia* in north-western British Columbia, Canada. Outside of these examples, there are few standardized, continuous datasets of suitable quality that can be used for fingerprint analysis.

Purpose of this Review

Given their impact on agriculture and their dependence on weather and other environmental factors, not surprisingly, plant diseases under climate change has been the subject of several reviews, with Burdon *et al.* [11], Garrett *et al.* [12] and Kliejunas et al. [13] being the most recent. Reviews have dealt with the impact of changing atmospheric CO_2, O_3 and UV-B on plant diseases [14]; biospheric and climate change influence on pests and diseases [15–21]; regional or country-wide assessments of climate change impacts [15, 22–24]; and impact assessment for specific diseases/pathogens or pathogen groups [25–32]. Overall, research on climate change and plant disease has followed two major routes [6]: (a) experimental work that deals with one or few elements of weather or atmospheric constituents in controlled conditions; and (b) models predicting how projected changes in climate and/or atmospheric composition will alter distribution, prevalence, severity and management. Many of the reviews have excellent summaries of both experimental and modelling studies and that material will not be repeated here.

Plant disease control of agricultural crops relies heavily on the use of host plant resistance and plant protection chemicals, while agronomic and other physical and biological measures contribute to the efficacy of management, especially through application of decision support tools such as integrated pest management. Molecular biology offers an exciting array of opportunities to augment traditional plant breeding and to transfer novel genes from alien species through genetic engineering [33]. If host plant resistance is to remain at the frontline of the battle against devastating diseases like rusts, strategic experimental studies must be made in environments that mimic future climate and atmospheric composition. Free-air CO_2 enrichment (FACE) studies offer one such strategy for developing genetic and transgenic solutions and testing their effectiveness under changing pathogen virulence spectrum resulting from microevolution. In the last 10 years there have been several FACE sites established with species ranging from annual crops to perennial tree species and pastures, and pest and disease studies are included in a handful of these. Some sites have O_3 enrichment in addition to CO_2. The aim of this review is to summarize published and unpublished studies on plant pathogens and diseases in FACE facilities and open top chambers and other current/recent non-FACE research to offer a summary of future research needs and opportunities.

FACE Experiments

FACE experiments allow for the study of elevated atmospheric CO_2 on agricultural crops, forest trees and plant ecosystems grown under natural conditions without enclosure. FACE facilities have been developed and deployed from those on the scale of 1–2 m for low-stature crops or ecosystems [34, 35] to those of 10–20 m diameter for field crop evaluation [36, 37], to those for young-to medium-aged forest stands [34, 38, 39] and finally to mature trees of basically any size [40]. Recently, Ainsworth and Long [41] have reviewed and summarized FACE research in 12 FACE facilities, some established since 1993, where crops such as soybean (SoyFACE, Illinois, USA), rice (Rice FACE, Shizukuishi, Japan), grapes (Rapolano Mid FACE, Chianti Region, Italy), C3 and C4 crops (Maricopa FACE, Arizona, USA); forestry species including aspen (Aspen FACE, Wisconsin, USA), loblolly pine (FACTS 1, Duke Forest, North Carolina, USA), poplar (POPFACE, Viterbo, Italy), sweetgum (Oak Ridge, Tennessee, USA); and ecosystems such as natural prairies grassland (BioCON, Minnesota, USA), managed grassland (SwissFACE, Eschikon, Switzerland), desert (Nevada desert, USA) and managed pasture (Pasture FACE, Bulls, New Zealand) have been studied. However, most field studies on the effects of elevated CO_2 have been done in open-top chambers (OTCs) [42], and OTCs have long been known to modify the environment by altering light intensity, relative humidity, wind speed and direction, and other environmental factors. This is particularly disconcerting for studies of disease occurrence when the chambers may interfere with the dispersal of natural inoculum or alter the plant's susceptibility to a given pathogen.

Ozone Predisposition to Disease and Pests

Ozone (O_3) is a secondary pollutant that is increasing downwind of major metropolitan areas around the world [43, 44]. It is a highly phytotoxic pollutant that decreases carbon assimilation of O_3-sensitive plants through direct effects on photosynthesis, leaf area and leaf area duration [45–47].

It has long been known that ozone can also alter plant responses to biotic diseases [48–52]. Effects of O_3 on pathogen interactions are variable depending on the timing of the exposure to both O_3 and the pathogen, the O_3 concentration, the stage of plant development, predisposing factors and environmental conditions [52]. The occurrence of co-occurring elevated atmospheric CO_2 can also alter the $O_3 \times$ disease interactions [53, 54]. Thus, it is not surprising that $O_3 \times$ biotic disease interactions have ranged from significant enhancement for diseases such as powdery mildew (*Erysiphe graminis*), leaf spot disease (*Septoria nodorum*) and spot blotch (*Bipolaris sorokiniana*) on wheat flag leaves exposed to O_3 [55], tan spot fungus (*Pyrenophora tritici-repentis*) on wheat [56] and other root diseases experiments in southern California, USA [57] and elsewhere [58] to decreases or no impact with other diseases. The occurrence of mycorrhizal and non-mycorrhizal root-infecting fungi [59], powdery mildew (*Sphaerotheca fuliginea*) on cucumber [60], spot blotch on barley and fescue [61] and leaf rust (*Puccinia recondita*) on wheat [53] have been shown to decrease with O_3. Finally, at least one study has shown no interaction of O_3 and pathogen occurrence in wheat [62].

Rapidly growing trees such as aspen, hybrid poplars and birch are particularly sensitive to O_3. In addition to slowing growth, O_3 has long been known to predispose trees to pathogens [14, 48, 50]. Probably the earliest work on O_3 predisposition of trees to pathogenic fungi was that of increased susceptibility of ponderosa and Jeffrey pines to the root rot fungus *Fomes annosus* and other root diseases in experiments in the San Bernardino mountains in southern California, USA where O_3 levels were some of the highest anywhere in the world [57, 63]. However, field surveys in California were not able to confirm this relationship [64], possibly due to the patchy disease incidence [65] or high levels of O_3 needed for the effect to occur [66]. Ozone-induced predisposition of poplars to both *Marssonina* leafspot disease [67] and *Melampsora* rust [68–70] is well known. More recently, O_3-induced predisposition of oak leaves to attack by *Diploidia corticola* and *Biscogniauxia mediterranea* has been reported by Paoletti *et al.* [71]. Luedemann *et al.* [72] and Pritsch *et al.* [58] found a significant predisposition effect of O_3 on susceptibility of spruce, but not of beech, to the root pathogen *Phytophthora citricola*. The proposed mechanisms of O_3-induced predisposition of tree have ranged from inhibition of defence compounds ([45]; Figure 1) to alterations of the leaf surface [69, 70] Figure 2). Just as with agricultural crops, not all interactions of pathogens and O_3 for trees have resulted in increased fungal occurrence as no interactive effects have been reported [75] and O_3 sometimes suppresses fungal occurrence [76].

Elevated CO_2 Effects on Plant Diseases

There is extensive literature on effects of high CO_2 on host anatomy, morphology, phenology and physiology (reviewed by Ainsworth and Long [41]). Combining this with the scant literature on the influence of high CO_2 on pathogen life cycle and disease epidemiology, Chakraborty [16] outlined two main driving forces influencing pathogen and disease development. In the short term, disease severity levels are determined by the opposing effects of enhanced host resistance that slows host invasion versus enlarged plant canopy that offers more infection sites and produces a microclimate conducive to disease development. More importantly, in the long term, higher pathogen fecundity, inoculum trapping by an enlarged plant canopy and a higher number of infection cycles interact with the effects of enhanced host plant resistance to determine host–pathogen adaptation. This can accelerate the rate of host-pathogen microevolution [77]. Many of the findings on pathogens at elevated CO_2 come from growth chamber studies, and data from FACE studies are just starting to emerge. Whether findings from controlled environment studies mimic results obtained from more realistic field studies such as within FACE facilities is yet to be determined. This is mainly because of a paucity of FACE studies examining host–pathogen interactions. The following sections deal with the recent handful of studies of plant pathogens from FACE facilities.

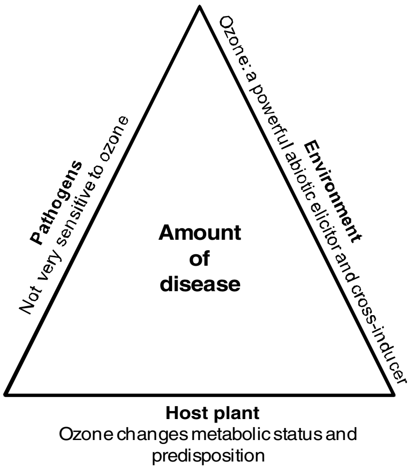

Figure 1 Role of ozone in plant disease. The classic triangle depicting the amount of plant disease as dependent on the host plant, the pathogen and environmental factors is modified for including the ozone effects described in the text (modified from [45])

Diseases of Agricultural Crops and Global Change

Rice Diseases

Using the 'Rice FACE' facility in Shizukuishi in northern Japan, Kobayashi *et al.* [78] studied the effect of 200–280 ppm above-ambient CO_2 on rice blast and sheath blight disease over three seasons. Severity of leaf blast (*Magnaporthe oryzae*) was consistently higher at elevated CO_2 in all three years assessed at two different stages of rice growth, possibly because of the reduced silicon content of leaves. In contrast, severity of panicle blast caused by the same pathogen was not consistently higher at high CO_2 levels. The incidence of rice plants naturally infected with sheath blight (*Rhizoctonia solani*) was generally higher at elevated CO_2 concentrations under high nitrogen levels, but this trend was not apparent for sheath blight severity. The higher incidence was attributed to the increased chance of fungal sclerotia coming in contact with leaf sheaths as a result of increased tillering.

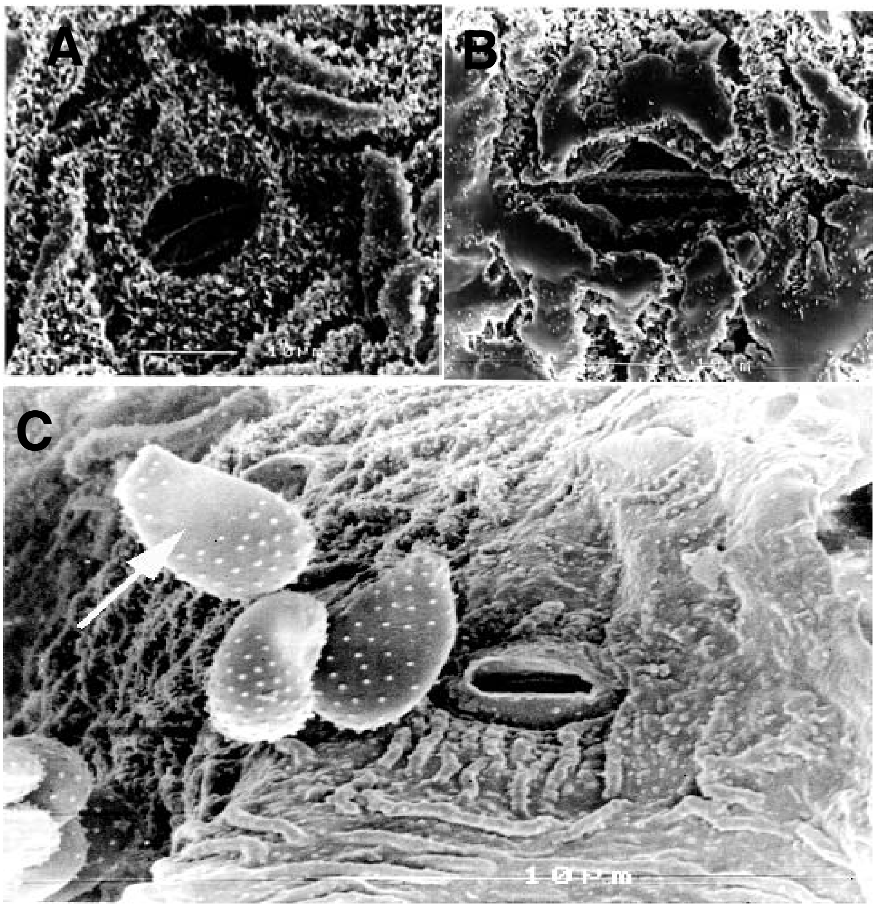

Figure 2 Alteration of the leaf surface of aspen clone 259 by O_3 is shown in A. On the left is an SEM of the lower leaf surface waxes and stomate of clone 259 under low O_3 conditions. B. On the right is an SEM of the same clone grown under 1.5 x ambient O_3. Note the difference in wax structure (modified from [69]) These structural changes have been verified with examination of hundreds of leaves from several aspen clones over the past 6 years with controlled O_3 exposures [70, 73] and with observations along a natural O_3 gradient [74]. C. Aspen leaf rust spores shown. The equatorial smooth band (see arrow) that is characteristic of *Melampsora medusae* is shown

Wheat Diseases

In 2007 the Australian Grains FACE (AGFACE) facility at Horsham in Victoria http://www.jcci.unimelb.edu.au/FACE/FACEOutline.htm was established for studying changes in wheat agronomy at CO_2 concentrations projected under the IPCC emission scenario A1B [2]. With eight 12 m diameter rings, each at 550 ppm or ambient CO_2, the main trial is a factorial of two CO_2 levels, two wheat varieties, two times of sowing and two levels of water supply in four replicates. Small areas of this trial have been used for studying pathogen biology and host–pathogen interaction for the biotrophic stripe rust (*Puccinia striiformis*), necrotrophic crown rot (*Fusarium pseudograminearum*) and BYDV (*Barley yellow dwarf virus*). In addition, naturally occurring levels of soil-borne pathogens *Heterodera avenae*, *R. solani*, *Gaeumannomyces graminis* var. *tritici*, *Pratylenchus neglectus* and *Pratylenchus thornei*, *F. pseudograminearum* and *Fusarium culmorum* and *Bipolaris* spp. were quantified from the soil using PCR-based diagnostics from each FACE ring both prior to sowing and post harvesting.

For the stripe rust study, a susceptible (H45) and a partially resistant (Janz) cultivar have been used for monitoring disease progress and components of pathogen life cycle including incubation period, latent period and fecundity following natural infection of plants. For crown rot, a bread wheat breeding line with partial resistance (2–49) and susceptible durum wheat (Tamaroi) were monitored for disease severity and pathogen life cycle traits after artificial inoculation at sowing. Stubble and soil samples were collected for monitoring pathogen survival and fitness. A natural infection of yellow dwarf viruses (BYDV and cereal yellow dwarf virus, CYDV) was used for determining disease severity and the main serotypes of BYDV and CYDV on one variety (Yitpi). There was good correlation between visual symptoms of YDVs and positive serotype tests, with the PAV strain of BYDV, the most frequent of at least three other YDV strains. A culture of the dominant strain and a colony of its main vector, *Rhopalosiphum padi* have been established for future inoculation studies.

Data on all pathogens from 2007 [79] will form a part of a larger dataset to be collected from this ongoing study and trends are expected after 2–3 wheat seasons.

Forest Diseases and Global Change

Apart from significant economic impact on agriculture and forestry industries, pathogens can influence the structure and dynamics of plant communities and populations in natural ecosystems [11, 80, 81]. Forests cover some 43% of the Earth's surface and account for some 70% of terrestrial net primary production (NPP) and carbon storage [82]. Whether or not these carbon stocks will increase or decrease under global change is largely dependent on interacting impacts of insects, diseases and fire [83]. Among the key global change drivers that are expected to impact forest trees over the next century are the predicted doubling of atmospheric CO_2 [1] and the related warming of the Earth's surface by 1.4–5.8°C [1]. Other key global change drivers include nitrogen deposition, and increasing levels of tropospheric O_3. These global change drivers can have effects either directly on the tree hosts, on the pathogens directly, or on the vectors of these pathogens [84]. Further complicating this situation is the fact that the impacts of these global change drivers can be very different when these drivers co-occur. The major concerns with tree diseases and global change are: (1) the key global change drivers may predispose otherwise healthy forest trees to pathogenic attack, as demonstrated by Capretti and Battisti [85]; and (2) major range shifts of pathogens into forests that have not previously been exposed to a given disease. Of course, climate change itself can have adverse effects, including reduced growth and increased mortality, even without insects or diseases being involved [86, 87].

Range Shifts

Pest species of forest trees moving pole-ward and upward on mountains have been proposed as a consequence of global warming [88]. One documented example of this happening is the increased occurrence of *Cronartium ribicola* in the Rocky Mountains, associated with the range expansion of the bark beetle which transmits the disease [89]. The recent outbreak of *Dothistroma* needle blight in lodgepole plantations in northwestern British Columbia is suspected to be related to observed warming in the region [10]. In Europe, there is a concern that pathogens of the Mediterranean or of tropical origin will extend their geographic range to Europe in response to climate change [90]. Recent modelling studies suggest large-range expansions are possible for the oak pathogen *Phytophthora cinnamomi* [25] as well as other important forest pathogens [27]. There is also concern that the pine wilt (*Bursaphelenchus xylophilus*) disease may be able to expand its range into northern Asia [91, 92] because of the capability of the pine sawyer host to survive in warming winters.

Elevated CO_2 Effect on Forest Diseases

In contrast to other global change drivers, it has not been possible yet to study the effects of elevated atmospheric CO_2 on forest tree disease occurrence *in situ*. While the levels of atmospheric CO_2 have increased some 30% over the past century [1], this has occurred at the same time that considerable warming has occurred and there has been increasing nitrogen deposition making it difficult to sort out direct or indirect effects of elevated atmospheric CO_2 on forest tree/pathogen interactions. For this reason, the development of FACE systems allowing realistic forest conditions has been important for the study of interactions of elevated atmospheric CO_2 and forest/pathogen systems. However, altering CO_2 on its own is not representative of future conditions where different factors such as temperature and soil nutrients will interact with elevated CO_2. A number of FACE systems have been developed for controlling CO_2 levels in forest canopies, but the most common have used the infrastructure-intense Brookhaven National laboratory design [38], the pure-CO_2 tube ejection system, which has considerably less obtrusive infrastructure [93], and the Web-FACE system for exposing tall trees in mature forests [40]. These systems have also been altered to allow for fumigation of O_3 alone [94] or for O_3 in combination with CO_2 [95].

Using the 'Aspen FACE' in Wisconsin, the co-occurring impacts of elevated CO_2 and O_3 were examined for the occurrence of *Melampsora* leaf rust caused by the fungus *Melampsora medusae* on aspen [69, 70]; Figure 3). A 3–5 fold increase in *Melampsora* rust occurrence was seen under elevated O_3, alone, with O_3-sensitive aspen clones showing a higher increase than O_3-tolerant clones [69]. The effects were attributed to alterations of the leaf surface waxes, which allowed leaf surfaces to be more wettable and thus, more conducive to *Melampsora* leaf rust spore germination [69, 70]. Similar effects on the leaf surface waxes were also seen along a natural O_3 gradient [73, 74]. Co-occurring-elevated CO_2 dampened the O_3-induced increase in rust occurrence, possibly because of the increased vigour of these trees compared to those growing in O_3 alone.

More recently, Karnosky's team has shown that elevated CO_2 decreases the occurrence of *Hypoxylon* canker, a deadly disease killing 1–2% of the aspen in the Lake District of USA annually [96] and caused by the fungus *Hypoxylon mammatum* on trembling aspen regardless of O_3 exposure (Figure 4). While we do not know why elevated CO_2 reduced *Hypoxylon* canker, we do know that elevated CO_2 decreases water stress by

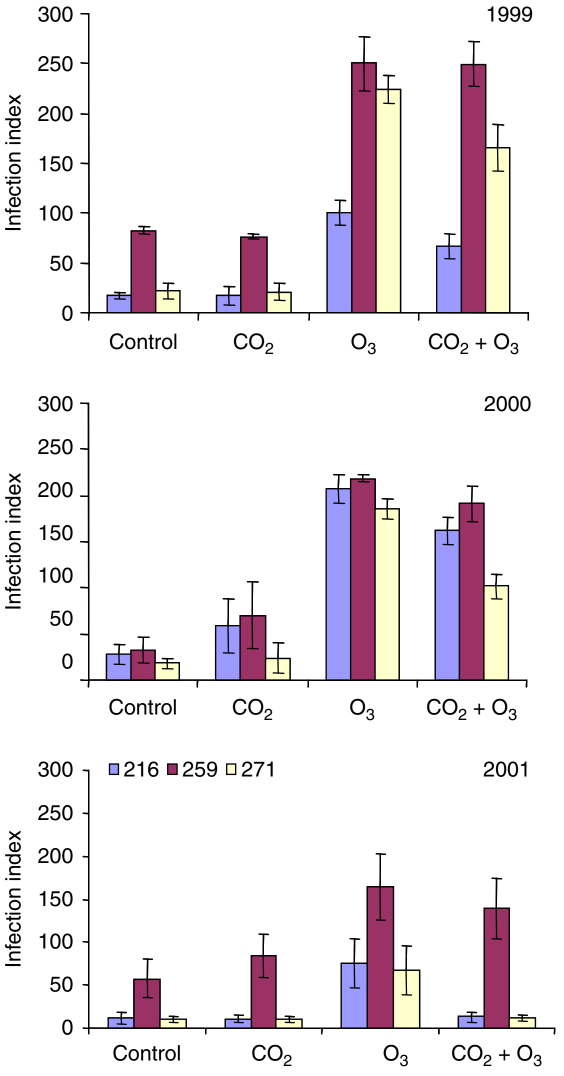

Figure 3 Aspen leaf rust infection index of three aspen clones (216, 259 and 271) at the Aspen FACE experiment for 1999, 2000 and 2001. The infection index was developed by multiplying the percentage of leaves infected per tree×the severity of the infection (1=1–20% of the leaf area covered with urediniospores, 2=21–40%, 3=41–60%, 4=61–80%, and 5=81–100%). Significantly higher (3–5×) levels of rust occurrence were seen each year under elevated O_3, regardless of the CO_2 treatment. In the lowest rust infection year (2001), the strong clonal differences in O_3 predisposition can be seen as clone 259 (O_3 sensitive) was more affected by rust in O_3 treatments than were the two O_3 tolerant clones (216 and 271) (modified from [69])

causing decreased stomatal conductance. *Hypoxylon* canker is known to be stress-induced [98, 99] and we have commonly measured decreased stomatal conductance and decreased water stress under elevated CO_2 at Aspen FACE [100]. Elevated CO_2 may have reduced water stress during the numerous dry summers over the past five years.

Elevated CO_2 has also significantly decreased the occurrence of a fungal pathogen, *Phyllosticta minima*, on red maple in the 'Duke Forest' FACE experiment near Raleigh, NC, USA [101]. Reduced disease severity was likely to be the result of altered leaf chemistry and decreased nutritive quality as elevated CO_2 reduced leaf N by 20% and increased the C:N ratio by 20%. Furthermore, the stomatal conductance under elevated CO_2 was reduced by 21–36%, suggesting decreased stomatal aperture may have also contributed to the reduced disease successes. Similarly, elevated CO_2 was shown to decrease the area of oak leaves infected with the same *Phyllosticta* fungus [102].

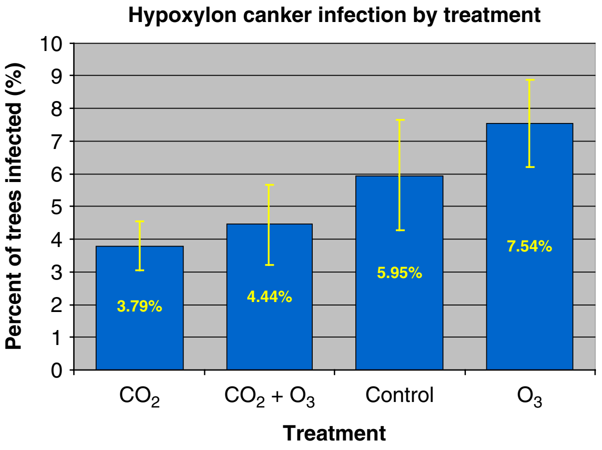

Figure 4 *Hypoxylon* canker occurrence on trembling aspen across treatments at the Aspen FACE experiment as evaluated in 2005. *Hypoxylon* canker occurrence was significantly less under elevated CO_2, regardless of O_3 exposure. The treatments for were summarized this experiment in Karnosky *et al.* [97]

The complexity of elevated atmospheric CO_2/pathogen interactions is illustrated by the 'POPFACE' experiment in Viterbo, Italy, where Scarascia-Mugnozza *et al.* [103] found that while elevated CO_2 did not affect disease intensity or incubation period of *Marssonina* species or PMV virus, an increased attack by sooty mold fungi occurred under elevated CO_2. This latter effect was probably because of the increase in the sucking insects that favoured diseased plants. Recently, Fleischmann *et al.* [104] reported that the interaction of nitrogen was important for the response of beech seedlings to the root pathogen *P. citricola* grown under elevated CO_2. Highest susceptibility of beech to the fungus occurred for seedlings grown under high CO_2 and high nitrogen.

FACE Studies on Diseases of other Plants

Mitchell *et al.* [105] considered the effects on disease load in tallgrass prairie of plant diversity, nitrogen deposition and increased atmospheric CO_2. The higher pathogen loads on C_3 species in lower diversity plots made it possible to detect the increased load resulting from elevated CO_2. While there was a trend toward higher pathogen loads with CO_2 enrichment in the higher diversity plots, the trend was not statistically significant. CO_2 enrichment did not increase pathogen load for C_4 species or forbs. The longer leaf longevity from CO_2 enrichment, observed in C_3 species but not the other species, may explain in part why the pathogen load of C_3 species responded more strongly to CO_2 enrichment.

Other grassland research on the changing structure of microbial populations in rhizosphere soil under FACE, including ones from the Swiss FACE [106, 107] and other research on soil microbiology that does not deal directly with plant pathogens or diseases are not discussed in this review.

Using the BioCON (Biodiversity, Carbon dioxide and Nitrogen effects on ecosystem functioning) facility, Strengbom and Reich [108] examined the incidence of leaf spot disease and its relationship to photosynthesis in the herb *Solidago rigida* grown in monoculture under ambient or elevated (560 ppm) CO_2 with two levels of N. Incidence was lower under elevated CO_2 and enriched N treatments compared to ambient conditions. The disease reduced leaf photosynthetic rate by more than 50% in all treatments, irrespective of severity, as measured by the leaf area covered in lesions. This finding highlights the need for considering disease and other biotic constraints in making projections of yield advantage under the so-called CO_2 fertilization effect'.

Other Recent Research on Global Change and Plant Diseases

Changes in Range and Severity

A number of recently published papers have dealt with the development and use of models for predicting changes in distribution, prevalence, survival, severity and management of plant pathogens under projected changes in climate and/or atmospheric composition. Many have used outputs from one or more General Circulation Models (GCM) in making predictions on range. Bergot *et al.* [25] and Marçais *et al.* [109] combined ARPEGE, a GCM, with a physiologically based model of the oak pathogen *P. cinnamomi* for predicting a range expansion in France for the pathogen from an increased winter survival. Using outputs from five GCM, Ghini *et al.* [28] have predicted increased number of generations in Brazil for the coffee races of the nematode *Meloidogyne incognita*. These authors have also predicted the risk of black sigatoka in banana for Brazil [110]. Salinari *et al.* [111] used output from two GCMs corresponding to the SRES-A2 emissions scenario from present to 2100 to show increases in grapevine downy mildew (*Plasmopara viticola*) in each decade and two more fungicide sprays were necessary for achieving the current level of protection. Combining a weather-based forecasting model for *Leptosphaeria maculans* on brassicas with HadRM3 model, Evans *et al.* [112] have predicted an increase in both the range and severity of *Phoma* stem canker in the UK. Archibald and Brown [113] also predict an increase in the spread and severity of *D. septosporum* (red band needle blight) in the UK, if increases in temperature continue as predicted. Salinari *et al.* [114] have used an empirical statistical model to study downy mildew outbreaks on grapevine under climate change.

Changes in range of both vegetation and pests can also occur with altitude because of global warming. Until recently, the potential impact of invasive forest pathogens on alpine treeline dynamics has not been considered [115]. The exotic fungal pathogen white pine blister rust, caused by the fungus *C. ribicola*, threatens the continued existence of whitebark pine (*Pinus albicaulis*) in the northern Rocky Mountains of North America [116]. The role of climate change is as yet undetermined in this host–parasite interaction but at the very least the occurrence of exotic pathogens can greatly confound treeline responses to global change [115].

Mechanism of Host–pathogen Interaction

Recent studies on the physiological, biochemical and molecular mechanisms of host–pathogen interaction under high CO_2 and/or other elements of global change are very encouraging, although mechanisms such as reduced stomatal opening in high CO_2 reducing pathogen infection and changes in leaf chemistry [101, 117–120] have been well known. Heat and drought stress may either predispose plants to pathogen infection [121] or induce defence cascades for enhancing host plant resistance. Hence, separating mechanisms of drought or water stress and biotic stress from abiotic stress from plant pathogens is complicated as there may be common genes and gene networks triggered by signals from both stress. For instance, Luo *et al.* [122] reported that 42 expressed sequenced tags (EST) from several functional categories were up-regulated under both *Aspergillus parasiticus* challenge and drought stress. Of these, 25 genes were commonly expressed in both stress treatments. In another study, the expression of a transcription factor LtWRKY21, a key regulator of signalling networks in *Larrea tridentata* was enhanced by high CO_2, water deficit, high salinity and wounding [123]. Similarly, high CO_2 and O_3 have interactive effects on disease resistance. In spring barley resistance against *Drechslera teres* increased at double-ambient O_3 with significantly higher activities of $\beta-1$-3-glucanase and chitinase, but this effect is less obvious at 700 ppm than at 400 ppm CO_2 [124].

Increased understanding of interacting mechanisms and crosstalk of hormone, biotic and abiotic stress including elevated CO_2 and O_3 signalling pathways are essential for ultimately breeding/generating plants with increased adaptation to global change. Unfortunately, understanding of mechanisms from controlled environments often does not coincide with processes that occur in the field under a more complex environment. Miyazaki *et al.* [125] have examined transcript abundance and changes due to exposures to high CO_2 and O_3 in the growth chamber and at the 'SoyFACE' using the model plant *Arabidopsis thaliana*. Most changes in gene expression were between growth chamber and ambient field conditions, compared with high versus low CO_2 or O_3 contrasts.

Other recent studies on elevated CO_2 have explored the role of secondary metabolites with obvious links to disease resistance. In tobacco grown at elevated CO_2 secondary metabolites such as phenylpropanoids and the major carbon-rich compounds and coumarin scopoline and scopoletins are significantly increased to increase the level of resistance against potato virus Y [126], but this accumulation is dependent on the availability of nitrogen. Soybean cultivars that differ in resistance to *Phytophthora sojae* behave differently when exposed to high CO_2 [127]. Exposure to high CO_2 stimulates production of the phytoalexin glycceollin in a resistant variety but not in a susceptible variety. These host–pathogen studies on physiology and gene expression are a step in the right direction, but the generally complex nature of the interaction does not readily point to simple 'one size fits all' solutions and the paucity of knowledge has to be overcome before any generalization.

Gaps in Knowledge

Pathogen impacts are yet to be integrated with crop models that project the fate of agriculture and forestry industry or natural ecosystems under a changing climate. Even the most recent commentaries on food security, advocating priorities for climate change adaptation needs [128, 129], continue to ignore impacts of pest and diseases on agricultural production and quality. Process-based models linking key elements of pathogen/disease cycle to crop models [130] would be more appropriate in projecting climate change impacts.

Realistic assessments of climate change impacts on host–pathogen interactions are still scarce and there are only a handful of FACE studies. Despite being interesting from a pathogen life-cycle point of view, realistic effects of high CO_2 need to be considered in the context of rising temperature and other changes projected under a changing climate. For instance, even if a rise in CO_2 does not make any significant impact on pathogen development or disease severity, these effects may be stimulated when considered together with a rise in temperature [119]. While climatic factors can be relatively easily studied in controlled environments, combining temperature and CO_2 effects in FACE studies remains a challenge. Extrapolating trends based on findings from discrete CO_2 levels is another drawback of current studies and the use of CO_2 gradients in the field needs consideration.

One of the most important challenges for FACE research will be incorporating results of necessarily small-scale experiments in larger scale predictions. Understanding all the interactions, thresholds and positive feedbacks in systems will be critical for making this possible [131]. In some circumstances, interactions between higher CO_2 concentrations and the introduction of invasive plant, pathogen or vector species may result in unexpectedly large or small effects of CO_2 change [132]. Pathogen reproduction may increase rapidly if populations rise above a threshold in response to new environments [134]. Positive feedback loops may result in lower management efficiencies for IPM systems when regional inoculum loads are higher.

Soil microbial communities, both pathogenic and non-pathogenic, are important for the complete study of climate change effects [134]. While studies of CO_2 effects on disease have emphasized specific pathosystems to date, new tools will allow the characterization of much more complete microbial communities. More efficient sequencing technologies allow thousands of microbial taxonomic units to be studied simultaneously from soil [135] or plant samples. Functional gene arrays support the analysis of thousands of microbial functions simultaneously [136]. As genes supporting pathogenicity are better understood, functional arrays can be designed to study shifts in their presence and activity in microbial communities in response to CO_2 change.

In cases where higher disease risk results from climate change, the impact will not be only through potential crop loss. Methods for disease management may also influence other ecosystem services provided by agricultural systems [137]. For example, if disease management requires pesticide applications or additional tillage, these activities will have their own impacts on ecosystem services that may be difficult to quantify (Cheatham *et al.*, personal communication). Likewise, plant disease may influence carbon sequestration. Knowledge of these impacts will also be important for fully evaluating CO_2 effects.

Long-term studies on epidemiology, host–pathogen population genetics and evolution under a changing climate in realistic FACE environments are strikingly absent from the literature. A sole study in controlled environment has raised the spectre of accelerated pathogen microevolution as a consequence of increased fecundity and infection cycles under high CO_2 [77]. In cultivated agriculture and forestry species, plant improvement specialists would need to consider ways of exploiting changes at high CO_2 that lead to enhanced resistance for fortifying crops against pathogens potentially evolving at a rapid rate. The time for pre-emptive selection and breeding for developing adapted varieties is now. Yet, host plant adaptation under elevated CO_2 has never been examined. Genetic improvements would need to rely both on traditional selection/breeding and genetic transformation routes. Selection for naturally occurring resistance among germplasm has to happen in FACE facilities. Similar approaches are needed for the transgenic route and recent studies on secondary metabolites [126, 127] and gene expression [123, 125] at elevated CO_2 are a step in the right direction.

Climate change will alter the suitability of crops and other plants for certain locations. If crops continue to be grown for economic reasons, such as existing infrastructure for irrigation storage and transportation, poor health and chronic stress from marginal climates would increase susceptibility to pathogens. If host species migrate to new areas, pathogens are likely to follow, but changing temperature may alter the balance between pathogen species. Changes in temperature and other climatic factors may activate some sleeper pathogen species while others may cease to be economically important. This may apply equally to endemic and exotic pathogens. Often minor changes in climatic factors such as temperature can tip the balance in favour of an exotic species for gaining a bridgehead following its inadvertent introduction. Unusual disease outbreaks could also occur from sporadic weather events. Import risk analysis has to consider future climate scenarios for assessing and evaluating incursion potential of exotic pest and pathogens.

The topic of climate change and plant diseases has not yet sparked widespread interest among plant pathologists and other plant protection specialists, with the exception of entomologists. This may be partly because of the well-understood genetic basis of host-pathogen specificity, where many of the crop disease management options have come from resistant varieties and the lack of a clear-cut concept of individual/populations for fungal pathogens. There is optimism that challenges imposed by climate change can be largely solved by the development and deployment of well-adapted resistant varieties, developed using the traditional or transgenic route [12]. Consequently, ecological principles and approaches have not been widely applied in plant pathology. However, there has been a steady stream of new research and reviews in the area with the most recent meeting focusing on potato [138]. In contrast, insect–host relationships are often not specific to the same extent and well-defined insect populations easily lend themselves to studies using ecological approaches. Despite renewed community interest in climate change, plant protection professionals have not embraced this area and there are no dedicated graduate level courses to train and motivate future generations. Professional societies have a role to play in fostering and promoting opportunities in the science and policy issues to future generations.

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References

1. Intergovernmental Panel on Climate Change. Summary for Policymakers. In: Solomon S, Qin D, Manning M, Chen Z, Marquis M, Averyt KB, *et al.*, editors. Climate Change 2007: The Physical Science Basis. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge University Press, New York, NY, USA; 2007.
2. Intergovernmental Panel on Climate Change. Special Report. Emission Scenarios. Summary for Policymakers 2000. Available from: URL: http://www.ipcc.ch/pdf/special-reports/spm/sres-en.pdf.
3. Zhang DD, Brecke P, Lee HF, He Y, Zhang J. Global climate change, war, and population decline in recent human history. Proceedings of National Academy of Sciences of the USA 2007;104:19214–9.
4. Oerke EC, Dehne HW, Schonbeck F, Weber A. Crop production and crop protection, Estimated Losses in Major Food and Cash Crops. Elsevier, Amsterdam, The Netherlands; 1994.
5. Scherm H, Yang XB. Interannual variations in wheat rust development in China and the United States in relation to the El Niño/Southern Oscillation. Phytopathology 1995;85:970–6.
6. Jeger MJ, Pautasso M. Plant disease and global change – the importance of long-term data sets. New Phytologist 2008;177:8–11.
7. Bearchell SJ, Fraaije BA, Shaw MW, Fitt BD. Wheat archive links long-term fungal pathogen population dynamics to air pollution. Proceedings of the National Academy of Sciences of the USA 2005;102:5438–42.
8. Shaw MW, Bearchell SJ, Fitt BDL, Fraaije BA. Long-term relationships between environment and abundance in wheat of *Phaeosphaeria nodorum* and *Mycosphaerella graminicola*. New Phytologist 2008;177:229–38.
9. Hannukkala AO, Kaukoranta T, Lehtinen A, Rahkonen A. Late-blight epidemics on potato in Finland, 1933–2002; increased and earlier occurrence of epidemics associated with climate change and lack of rotation. Plant Pathology 2007;56:167–76.
10. Woods A, Coates DK, Hamann A. Is an unprecedented Dothistroma needle blight epidemic related to climate change? Bioscience 2005;55:761–9.
11. Burdon JJ, Thrall PH, Ericson L. The current and future dynamics of disease in plant communities. Annual Review of Phytopathology 2006;44:19–39.
12. Garrett KA, Dendy SP, Frank EE, Rouse MN, Travers SE. Climate change effects on plant disease: genomes to ecosystems. Annual Review of Phytopathology 2006;44: 489–509.
13. Kliejunas JT, Geils B, Glaeser JM, Goheen EM, Hennon P, Kim M-S, *et al.* Climate and forest diseases of western north America: a literature review; 2008. Available from: URL: http://www.fs.fed.us/psw/topics/climate_change/forest_disease/Final.Forpathandcc.lit.jk.022208.pdf.
14. Manning WJ, von Tiedemann A. Climate change: potential effects of increased atmospheric carbon dioxide (CO_2), ozone (O_3), and ultraviolet-B (UVB) radiation on plant diseases. Environmental Pollution 1995;88:219–45.

15. Boland GJ, Melzer MS, Hopkin A, Higgins V, Nassuth A. Climate change and plant diseases in Ontario. Canadian Journal of Plant Pathology 2004;26:335–50.
16. Chakraborty S. Potential impact of climate change on plant–pathogen interactions. Australasian Plant Pathology 2005;34:443–8.
17. Coakley SM. Biospheric change: will it matter in plant pathology? Canadian Journal of Plant Pathology 1995;17:147–53.
18. Coakley SM, Scherm H. Plant disease in a changing global environment. Aspects of Applied Biology 1996;45:227–37.
19. Coakley SM, Scherm H, Chakraborty S. Climate change and plant disease management. Annual Review of Phytopathology 1999;37:399–426.
20. Paoletti E, Bytnerowicz A, Andersen C, Augustaitis A, Ferretti M, *et al.* Impacts of air pollution and climate change on forest ecosystems – emerging research needs. The Scientific World Journal 2007a;7:1–8.
21. Sutherst RW, Baker RHA, Coakley SM, Harrington R, Kriticos DJ, Scherm H. Pests under global change – Meeting your future landlords? In: Canadell JJ, Pataki DE, Pitelka LF, editors.Terrestrial Ecosystems in a Changing World. Springer, Berlin; 2006. p. 211–26.
22. Atkinson D. Global climate change, its implication for crop protection. British Crop Protection Council Monograph No. 56. British Crop Protection Council, Surrey, UK; 1993.
23. Chakraborty S, Murray GM, Magarey PA, Yonow T, O'Brien R, Croft BJ, *et al.* Potential impact of climate change on plant diseases of economic significance to Australia. Australasian Plant Pathology 1998;27:15–35.
24. Prestidge RA, Pottinger RP. The Impact of Climate Change on Pests, Diseases, Weeds and Beneficial Organisms Present in New Zealand Agricultural and Horticultural Systems. MAF Technology, Ruakura Agricultural Centre, Hamilton, NZ; 1990.
25. Bergot M, Cloppet E, Pérnaud V, Déqué M, Marçais B, Desprez-Loustau M. Simulation of potential range expansion of oak disease caused by *Phytophthora cinnamomi* under climate change. Global Change Biology 2004;10:1539–52.
26. Brasier CM, Scott JK. European oak decline and global warming a theoretical assessment with special reference to the activity of *Phytophthora cinnamomi*. EPPO Bulletin 1994;24:221–32.
27. Desprez-Loustau M-L, Robin C, Reynaud G, Déqué M, Badeau V, Piou D, *et al.* Simulating the effects of a climate-change scenario on the geographical range and activity of forest-pathogenic fungi. Canadian Journal of Plant Pathology 2007;29:101–20.
28. Ghini R, Hamada E, Pedro MJ, Marengo JA, Gonçalves RRV. Risk analysis of climate change on coffee nematodes and leaf miner in Brazil. Pesquisa Agropecuária Brasileira 2008;43:187–95.
29. Hijmans RJ, Grunwald NJ, van Haren RJF, MacKerron DKL, Scherm H. Potato late blight simulation for global change research. Global Initiative on Late blight Newsletter 2000;12:1–3.
30. Kaukoranta T. Impact of global warming on potato late blight: risk, yield loss and control. Agricultural and Food Science in Finland 1996;5:311–27.
31. Luo Y, TeBeest DO, Teng PS, Fabellar NG. Simulation studies on risk analysis of rice blast epidemics associated with global climate in several Asian countries. Journal of Biogeography 1995;22:673–8.
32. Sharma RC, Duveiller E, Ortiz-Ferrara G. Progress and challenge towards reducing wheat spot blotch threat in the Eastern Gangetic Plains of South Asia: is climate change already taking its toll? Field Crops Research 2007;103:109–18.
33. Huang J, Pray C, Rozelle S. Enhancing the crops to feed the poor. Nature 2002;418:678–84.
34. Miglietta F, Hoosbeek MR, Foot J, Gigon F, Hassinen A, Heijmans M, *et al.* Spatial and temporal performance of the miniface (free-air CO_2 enrichment) system on bog ecosystems in northern and central Europe. Environmental Monitoring and Assessment 2001;66:107–27.
35. Hovenden MJ, Miglietta F, Zaldei A, Vander Schoor JK, Wills KE, Newton PCD. The TasFACE climate-change impacts experiment: design and performance of combined elevated CO_2 and temperature enhancement in a native Tasmanian grassland. Australian Journal of Botany 2006;54:1–10.
36. Kimball BA, Pinter Jr PJ, Garcia RL, LaMorte RL, Wall GW, Hunsaker DJ, *et al.* Productivity and water use of wheat under free-air CO_2 enrichment. Global Change Biology 1996;1:429–42.
37. Bernacchi CJ, Morgan PB, Ort DR, Long SP. The growth of soybean under free air [CO_2] enrichment (FACE) stimulates photosynthesis while decreasing in vivo Rubisco capacity. Planta 2005;220:434–46.
38. Hendrey GR, Ellsworth DS, Lewin KF, Nagy J. A free-air enrichment system for exposing tall forest vegetation to elevated atmospheric CO_2. Global Change Biology 1999;5:293–309.
39. Karnosky DF, Gielen G, Ceulemans R, Schlesinger WH, Norby RJ, Oksanen E, *et al.* Face systems for studying the impacts of greenhouse gases on forest ecosystems. In: Karnosky DF, Scarascia-Mugnozza G, Ceulemans R, Innes J, editors. The Impact of Carbon Dioxide and Other Greenhouse Gases on Forest Ecosystems. CABI Publishing: New York, NY, USA; 2001. p. 297–324.
40. Pepin S, Körner C. Web-FACE: a new canopy free-air CO_2 enrichment system for tall trees in mature forests. Oecologia 2002;133:1–9.
41. Ainsworth EA, Long SP. What have we learned from 15 years of free-air CO_2 enrichment (FACE)? A meta-analytic review of the responses of photosynthesis, canopy properties and plant production to rising CO_2. New Phytologist 2005;165:351–372.
42. Long SP, Ainsworth EA, Rogers A, Ort DR. Rising atmospheric carbon dioxide: Plants FACE the future. Annual Review of Plant Biology 2004;55:591–628.
43. Intergovernmental Panel on Climate Change. A Report of Working Group I of the Intergovernmental Panel on Climate Change. 2001. Available from: URL: http://www.ipcc.ch/
44. Vingarzan R. A review of surface ozone background levels and trends. Atmospheric Environment 2004;38: 3431–42.
45. Sandermann Jr H. Ozone and plant health. Annual Review of Phytopathology 1996;34:347–66.

46. Krupa S, McGrath MT, Andersen CP, Booker FL, Burkey KO, Chappelka BI, *et al.* Ambient ozone and plant health. Plant Disease 2000;85:4–12.

47. Karnosky DF, Skelly JM, Percy KE, Chappelka AH. Perspectives regarding 50 years of research on effects of tropospheric ozone air pollution on US forests. Environmental Pollution 2007a;147:489–506.

48. Heagle AS. Interactions between air pollutants and plant parasites. Annual Review of Phytopathology 1973;11: 365–85.

49. Manning WJ. Interactions between air pollutants and fungal, bacterial and viral plant pathogens. Environmental Pollution 1975;9:87–90.

50. Laurence JA. Effects of air pollutants on plant-pathogen interactions. Journal of Plant Diseases and Protection 1981;8:156–72.

51. Sandermann Jr H. Ozone/biotic disease interactions: molecular biomarkers as a new experimental tool. Environmental Pollution 2000;108:327–32.

52. Fuhrer J. Agroecosystem responses to combinations of elevated CO_2, ozone, and global climate change. Agriculture, Ecosystems and Environment 2003;97:1–20.

53. von Tiedemann A, Firsching KH. Interactive effects of elevated ozone and carbon dioxide on growth and yield of leaf rust-infected versus non-infected wheat. Environmental Pollution 2000;108:357–63.

54. Plessl M, Elstner EF, Rennenberg H, Habermeyer J, Heiser I. Influence of elevated CO_2 and ozone concentrations on late blight resistance and growth of potato plants. Environmental and Experimental Botany 2007;60:447–57.

55. von Tiedemann A, Weigel H, Jäger HJ. Effects of open-top chamber fumigations with ozone on 3 fungal leaf diseases of wheat and the mycoflora of the phyllosphere. Environmental Pollution 1991;72:205–24.

56. Sah DN, von Tiedemann A, Fehrmann H. Effects of ozone exposure on resistance of wheat genotypes to *Pyrenophora tritici-repentis*. Journal of Phytopathology 1993;138:249–56.

57. Fenn ME, Dunn PH, Wilborn R. Black stain root disease in ozone-stressed ponderosa pine. Plant Disease 1990;74:426–30.

58. Pritsch K, Luedemann G, Matyssek R, Hartmann A, Schloter M, Scherb H, *et al.* Mycorrhizosphere responsiveness to atmospheric ozone and inoculation with *Phytopthora citricola* in a phytotron experiment with spruce/beech mixed cultures. Plant Biology 2005;7:718–27.

59. Bonello P, Heller W, Sandermann Jr H. Ozone effects on root-disease susceptibility and defence responses in mycorrhizal and non-mycorrhizal seedlings of Scots pine (*Pinus sylvestris* L.). New Phytologist 1993;124:653–63.

60. Khan MR, Khan MW. Effects of intermittent ozone exposures on powdery mildew of cucumber. Environmental and Experimental Botany 1999;42:163–71.

61. Plazek A, Hura K, Rapacz M, Zur I. The influence of ozone fumigation on metabolic efficiency and plant resistance to fungal pathogens. Journal of Applied Botany 2001;75: 8–13.

62. Pfleeger TG, da Luz MA, Mundt CC. Lack of a synergistic interaction between ozone and wheat leaf rust in wheat swards. Environmental and Experimental Botany 1999;41:195–207.

63. James RL, Cobb Jr FW, Miller PR, Parmeter Jr JR. Effects of oxidant air pollution on susceptibility of pine roots to *Fomes annosus*. Phytopathology 1980;70:560–3.

64. Pronos J, Merrill L, Dahlsten D. Insects and pathogens in a pollution-stressed forest. In: Miller PR, McBride JR, editors. Oxidant Air Pollution Impacts on the Montane Forests of Southern California: A Case Study of the San Bernardino Mountains. Volume 134. Ecological Studies, Springer-Verlag, New York, USA; 1999. p. 317–37.

65. Fenn ME, Poth MA, Bytnerowicz A, Sickman JO, Takemoto BK. Effects of ozone, nitrogen deposition, and other stressors on montane ecosystems in the Sierra Nevada. Developments in Environmental Science 2003;1:111–55.

66. James RL, Cobb Jr FW, Miller PR, Parmeter Jr JR. Effects of ozone on sporulation, spore germination and growth of *Fomes annosus*. Phytopathology 1982;72:1205–8.

67. Beare JA, Archer SA, Bell JNB. *Marssonina* leafspot disease of poplar under elevated ozone: pre-fumigated host and in vitro studies. Environmental Pollution 1999a;105: 409–17.

68. Beare JA, Archer SA, Bell JNB. Effects of *Melampsora* leaf rust disease and chronic ozone exposure on poplar. Environmental Pollution 1999b;105:419–26.

69. Karnosky DF, Percy KE, Xiang B, Callan B, Noormets A, Mankovska B, *et al.* Interacting elevated CO_2 and tropospheric O_3 predisposes aspen (*Populus tremuloides* Michx.) to infection by rust (*Melampsora medusae* f. sp. *tremuloidae*). Global Change Biology 2002;8:329–38.

70. Percy KE, Awmack CS, Lindroth RL, Kubiske ME, Kopper BJ, Isebrands JG, *et al.* Altered performance of forest pests under CO_2- and O_3-enriched atmospheres. Nature 2002;420:403–7.

71. Paoletti E, Naselmi N, Franceschini A. Pre-exposure to ozone predisposes oak leaves to attacks by *Diploida corticola* and *Biscogniauxia mediterranea*. The Scientific World Journal 2007b;7:222–30.

72. Luedemann G, Matyssek R, Fleischmann F, Grams TEE. Acclimation to ozone affects host/pathogen interaction and competitiveness for nitrogen in juvenile *Fagus sylvatica* and *Picea abies* trees infected with trees infected with *Phytophthora citricola*. Plant Biology 2005;7:640–9.

73. Mankovska B, Percy KE, Karnosky DF. Impacts of greenhouse gases on epicuticular waxes of *Populus tremuloides* Michx.: results from an open-air exposure and a natural O_3 gradient. Environmental Pollution 2005; 137:580–6.

74. Karnosky DF, Mankovska B, Percy K, Dickson RE, Podila GK, Sober J, *et al.* Effects of tropospheric O_3 on trembling aspen and interaction with CO_2: results from an O_3-gradient and a FACE experiment. Journal of Water, Air and Soil Pollution 1999;116:311–22.

75. Pearce RB, McLeod AR. Effects of sulphur dioxide and ozone on stilbenes and disease resistance in spruce trees from the Liphook Forest Fumigation Project. Plant, Cell and Environment 1995;18(3):303–7.

76. von Sury R, Flückiger W. Effects of air pollution and water stress on leaf blight and twig cankers of London planes [*Platanus*×*acerifolia* (Ait.) Willd.] caused by *Apiognomonia*

veneta (Sacc. & Speg.) Höhn. New Phytologist 1991;118:397–405.

77. Chakraborty S, Datta S. How will plant pathogens adapt to host plant resistance at elevated CO_2 under a changing climate? New Phytologist 2003;159:733–42.
78. Kobayashi T, Ishiguro K, Nakajima T, Kim HY, Okada M, Kobayashi K. Effects of elevated atmospheric CO_2 concentration on the infection of rice blast and sheath blight. Phytopathology 2006;96:425–31.
79. Luck J, Hollaway G, Freeman A, Spackman M, Chakraborty S. Examining the effects of elevated CO_2 on key pathogens of wheat. Proceedings of the 9th International Congress of Plant Pathology; 24–29 August 2008; Turin, Italy; 2008.
80. Klironomos JN. Feedback with soil biota contributes to plant rarity and invasiveness in communities. Nature 2002;417:67–70.
81. Strengbom J, Nordin A, Nasholm T, Ericson L. Parasitic fungus mediates change in nitrogen-exposed boreal forest vegetation. Journal of Ecology 2002;90:61–7.
82. Melillo JM, McGuire DA, Kicklighter DW, Moore B, Vorosmarty CJ, Scholl AL. Global climate change and terrestrial net primary production. Nature 1993;363: 234–40.
83. Kurz WA, Apps MJ. A 70-year retrospective analysis of carbon fluxes in the Canadian forest sector. Ecological Applications 1999;9:526–47.
84. Kirilenko AP, Sedjo RA. Climate change impacts on forestry. Proceedings National Academy of Sciences of the USA 2007;104:19697–702.
85. Capretti P, Battisti A. Water stress and insect defoliation promote the colonization of *Quercus cerris* by the fungus *Biscogniauxia mediterranea*. Forest Pathology 2007;37:129–35.
86. Van Mantgem PJ, Stephenson NL. Apparent climatically induced increase of tree mortality rates in a temperate forest. Ecology Letters 2007;10:909–16.
87. Reich PB, Oleksyn J. Climate warming will reduce growth and survival of Scots pine except in the far north. Ecology Letters 2008;11:588–97.
88. Parmesan C. Ecological and evolutionary responses to recent climate change. Annual Review of Ecology, Evolution and Systematics 2006;37:637–9.
89. Logan JA, Regniere J, Powell JA. Assessing the impacts of global warming on forest pest dynamics. Frontiers in Ecology Environment 2003;1:130–7.
90. Desprez-Loustau M-L, Marcais B, Nageleisen L-M, Piou D, Vannini A. Interactive effects of drought and pathogens in forest trees. Annual Forest Science 2006;63:597–612.
91. Kawai M, Shoda-Kagaya E, Maehara T, Zhou Z, Lian C, Iwata R, *et al.* Genetic structure of pine sawyer *Monochamus alternatus* (Coleoptera: Cerambycidae) populations in northeast Asia: consequences of the spread of pine wilt disease. Environmental Entomology 2006;35: 569–79.
92. Zhang Z-C, Sun J-H, Huang B-X, Zhang F, Ouyang H. Study of the pine wilt disease occurrence and its range expansion pattern based on GIS. Agriculture and Life Science 2006;32:551–6.
93. Miglietta F, Peressotti A, Primo Vaccari F, Zaldei A, De Angelis P, Scarascia-Mugnozza G. Free-air CO_2 enrichment (FACE) of a poplar plantation: the POPFACE fumigation system. New Phytologist 2001;150:465–76.
94. Werner H, Fabian P. Free-air fumigation of mature trees – a novel system for controlled ozone enrichment in grown-up beech and spruce canopies. Environmental Science and Pollution Research 2002;9:117–21.
95. Karnosky DF, Werner H, Holopainen T, Percy K, Oksanen T, Oksanen E, *et al.* Free-air exposure systems to scale up ozone research to mature trees. Plant Biology 2007b;9:181–90.
96. Schipper Jr AL, Anderson RL. How to identify Hypoxylon canker of aspen. USDA Forest Service North Central Forest Experiment Station; 1976.
97. Karnosky DF, Pregitzer KS, Zak DR, Kubiske ME, Hendrey GR, Weinstein D, *et al.* Scaling ozone responses of forest trees to the ecosystem level in a changing climate. Plant, Cell and Environment 2005;28:965–81.
98. Manion PD, Griffin HD. Sixty-five years of research on hypoxylon canker of aspen. Plant Diseases 1986;70:803–8.
99. Frey BR, Lieffers VJ, Hogg EH, Landhäusser SM. Predicting landscape patterns of aspen dieback: mechanisms and knowledge gaps. Canadian Journal of Forestry Research 2004;34:1379–90.
100. Darbah JNT. Impacts of elevated atmospheric CO_2 and/or O_3 on carbon gain and reproductive capacity in northern forest ecosystems [PhD Thesis]. Michigan Technological University, Houghton, MI; 2007.
101. McElrone AJ, Reid CD, Hoye KA, Hart E, Jackson RB. Elevated CO_2 reduces disease incidence and severity of a red maple fungal pathogen via changes in host physiology and leaf chemistry. Global Change Biology 2005;11:1828–36.
102. Aldea M, Hamilton JG, Resti JP, Zangerl AR, Berenbaum MR, Frank TD, *et al.* Comparison of photosynthetic damage from arthropod herbivory and pathogen infection in understory hardwood saplings. Oecologia 2006;149: 221–32.
103. Scarascia-Mugnozza G, De Angelis P, Sabatti M, Calfapietra C, Miglietta F, Raines C, *et al.* Global change and agro-forest ecosystems: adaptation and mitigation in a FACE experiment on a poplar plantation. Plant Biosystems 2005;139: 255–64.
104. Fleischmann F, Raidl S, OBwald WF. Changes in susceptibility of beech (*Fagus sylvatica*) saplings towards *Phytophthora citricola* under the influence of elevated CO_2 and nitrogen fertilization. Tree Physiology; in press 2008.
105. Mitchell CE, Reich PB, Tilman D, Groth JV. Effects of elevated CO_2, nitrogen deposition, and decreased species diversity on foliar fungal plant disease. Global Change Biology 2003;9:438–51.
106. Marilley L, Hartwig UA, Aragno M. Influence of an elevated atmospheric CO_2 content on soil and rhizosphere bacterial communities beneath *Lolium perenne* and *Trifolium repens* under field conditions. Microbial Ecology 1999;38:39–49.
107. Tarnawski S, Hamelin J, Jossi M, Aragno M, Fromin N. Phenotypic structure of Pseudomonas populations is altered under elevated pCO_2 in the rhizosphere of

perennial grasses. Soil Biology and Biochemistry 2006;38:1193–201.

108. Strengbom J, Reich PB. Elevated [CO_2] and increased N supply reduce leaf disease and related photosynthetic impacts on *Solidago rigida*. Oecologia 2006;149:519–25.

109. Marçais B, Bergot M, Pérarnaud V, Levy A, Desprez-Loustau M-L. Prediction and mapping of winter temperature on the development of *Phytopthora cinnamomi*-induced cankers on red and preduncolate oak in France. Phytopathology 2004;94:826–31.

110. Ghini R, Hamada E, Gonçalves RRV, Gasparotto L, Pereira JCR. Risk analysis of climate change on black sigatoka in Brazil. Proceedings of the 9th International Congress of Plant Pathology; 24–29 August 2008; Turin, Italy; 2008.

111. Salinari F, Giosuè S, Tubiello FN, Rettori A, Rossi V, Spanna F, *et al.* Downy mildew (*Plasmopara viticola*) epidemics on grapevine under climate change. Global Change Biology 2007a;12:1299–307.

112. Evans N, Baierl A, Semenov MA, Gladders P, Fitt BDL. Range and severity of a plant disease increased by global warming. Journal of Royal Society Interface 2008;5:525–31.

113. Archibald S, Brown A. The relationship between climate and the incidence of red band needle blight in the East Anglia Forest District, Britain. Proceedings of the 9th International Congress of Plant Pathology; 24–29 August 2008; Turin, Italy; 2008.

114. Salinari F, Giosuè S, Rossi V, Tubiello FN, Rosenzweig C, Gullino ML. Downy mildew outbreaks on grapevine under climate change: elaboration and application of an empirical-statistical model. EPPO Bulletin 2007b; 37:317–26.

115. Tomback DF, Resler LM. Invasive pathogens at alpine treeline: consequences for treeline dynamics. Physical Geography 2007;28:397–418.

116. Resler LM, Tomback DF. Blister rust prevalence in krummholz whitebark pine: Implications for treeline dynamics, northern Rocky Mountains, Montana, U.S.A. Arctic, Antarctic and Alpine Research 2008;40:161–70.

117. Hassan IA, Bell JNB, Marshall FM. Effects of ozone-protectant chemicals on physiology and growth of Egyptian clover grown in open-top chambers and at a rural site in Egypt. Proceedings of the 9th International Congress of Plant Pathology; 24–29 August 2008; Turin, Italy; 2008.

118. Hibberd JM, Whitbread R, Farrar JF. Effect of 700 μmol mol^{-1} CO_2 and infection with powdery mildew on the growth and carbon partitioning of barley. New Phytologist 1996;134:309–15.

119. Liu JZ, Titone P, Garibaldi A, Gullino ML. Effects of elevated CO_2 and temperature on infection of zucchini by powdery mildew. Proceedings of the 9th International Congress of Plant Pathology; 24–29 August 2008; Turin, Italy; 2008.

120. Titone P, Gullino ML, Garibaldi A. Effect of global change on powdery mildew-grapevine pathosystems: study under controlled environment. Proceedings of the 9th International Congress of Plant Pathology; 24–29 August 2008; Turin, Italy; 2008.

121. Green S, Ray D, MacAskill GA. Risks to Sitka spruce and other forest tree species in Scotland due to drought and fungal disease. Proceedings of the 9th International Congress of Plant Pathology; 24–29 August 2008; Turin, Italy; 2008.

122. Luo M, Liang XQ, Dang P, Holbrook CC, Bausher MG, Lee RD, *et al.* Microarray-based screening of differentially expressed genes in peanut in response to *Aspergillus parasiticus* infection and drought stress. Plant Science 2005;169:695–703.

123. Zou X, Shen QJ, Neuman D. An ABA inducible WRKY gene integrates responses of creosote bush (*Larrea tridentata*) to elevated CO_2 and abiotic stresses. Plant Science 2007;172:997–1004.

124. Pless M, Heller W, Payer H-D, Elstner EF, Habermeyer J, Heiser I. Growth parameters and resistance against *Drechslera teres* of spring barley (*Hordeum vulgare* L. cv. Scarlett) grown at elevated ozone and carbon dioxide concentrations. Plant Biology 2005;7:694–705.

125. Miyazaki S, Fredricksen M, Hollis KC, Poroyko V, Shepley D, Galbraith DW, *et al.* Transcript expression profiles of *Arabidopsis thaliana* grown under controlled conditions and open-air elevated concentrations of CO_2 and of O_3. Field Crops Research 2004;90:47–59.

126. Matros A, Amme S, Kettig B, Buck-Sorlin GH, Sonnewald U, Mock HP. Growth at elevated CO_2 concentrations leads to modified profiles of secondary metabolites in tobacco cv. SamsunNN and to increased resistance against infection with potato virus Y. Plant Cell and Environment 2006;29: 126–37.

127. Braga MR, Aidar MPM, Marabes MA, de Godoy RL. Effects of elevated CO_2 on the phytoalexin production of two soybean cultivars differing in the resistance to stem canker disease. Environmental and Experimental Botany 2006;58:85–92.

128. Lobell DB, Burke MB, Tebaldi C, Mastrandrea MD, Falcon WP, Naylor RL. Prioritizing Climate change adaptation needs for food security in 2030 Science 2008;319:607–10.

129. Schmidhuber J, Tubiello FN. Global food security under climate change Proceedings National Academy of Science of the USA 2007;104:19703–8.

130. White NA, Chakraborty S, Murray G. A linked process-based model to study the interaction between *Puccinia striiformis* and wheat. Proceedings of the Fourth International Crop Sciences Congress; 26 September–1 October 2004, Brisbane, Australia; 2004. Available from: URL: http://www.cropscience.org.au/icsc2004/poster/2/8/1242_white.htm (accessed 21 July 2008).

131. Garrett KA. Climate change and plant disease risk. In: Global climate change and extreme weather events: understanding the contributions to infectious disease emergence. Washington, DC: The National Academics Press; 2008. In press.

132. Kao-Kniffin J, Balser TC. Elevated CO_2 differentially alters belowground plant and soil microbial community structure in reed canary grass-invaded experimental wetlands. Soil Biology and Biochemistry 2007;39:517–25.

133. Otten W, Bailey DJ, Gilligan CA. Empirical evidence of spatial thresholds to control invasion of fungal parasites and saprotrophs. New Phytologist 2004;163:125–32.

134. Waldrop MP, Firestone MK. Response of microbial community composition and function to soil climate change. Microbial Ecology 2006;52:716–24.

135. Roesch LF, Fulthorpe RR, Riva A, Casella G, Hadwin AKM, Kent AD, *et al.* Pyrosequencing enumerates and contrasts soil microbial diversity. ISME Journal 2007; 1:283–90.

136. He ZL, Gentry TJ, Schadt CW, Wu LY, Liebich J, Chong SC, *et al.* GeoChip: a comprehensive microarray for investigating biogeochemical, ecological and environmental processes. ISME Journal 2007;1:67–77.

137. Millennium Ecosystem Assessment. Ecosystems and Human Well-being: Synthesis. Island Press, Washington, DC, USA; 2005.

138. Hannukkala A, Segerstedt M. editors. New and old pathogens of potato in changing climate. Proceedings of the EAPR Pathology section seminar, 2–6 July 2007, Hattula, Finland, Agrifood Research Working papers 142. 2007. Available from: URL: http://www.mtt.fi/mtts/pdf/mtts142.pdf