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REVIEW PAPER

CLIMATE CHANGE: POTENTIAL EFFECTS OF INCREASED ATMOSPHERIC CARBON DIOXIDE (CO_2), OZONE (O_3), AND ULTRAVIOLET-B (UV-B) RADIATION ON PLANT DISEASES

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Abstract

Continued world population growth results in increased emission of gases from agriculture, combustion of fossil fuels, and industrial processes. This causes changes in the chemical composition of the atmosphere. Evidence is emerging that increased solar ultraviolet-B (UV-B) radiation is reaching the earth's atmosphere, due to stratospheric ozone depletion. Carbon dioxide (CO_2), ozone (O_3) and UV-B are individual climate change factors that have direct biological effects on plants. Such effects may directly or indirectly affect the incidence and severity of plant diseases, caused by biotic agents. Carbon dioxide may increase plant canopy size and density, resulting in a greater biomass of high nutritional quality, combined with a much higher microclimate relative humidity. This would be likely to promote plant diseases such as rusts, powdery mildews, leaf spots and blights. Inoculum potential from greater overwintering crop debris would also be increased. Ozone is likely to have adverse effects on plant growth. Necrotrophic pathogens may colonize plants weakened by O_3 at an accelerated rate, while obligate biotroph infections may be lessened. Ozone is unlikely to have direct adverse effects on fungal pathogens. Ozone effects on plant diseases are host plant mediated. The principal effects of increased UV-B on plant diseases would be via alterations in host plants. Increased flavonoids could lead to increased diseased resistance. Reduced net photosynthesis and premature ripening and senescence could result in a decrease in diseases caused by biotrophs and an increase in those caused by necrotrophs. Microbial plant pathogens are less likely to be adversely affected by CO_2, O_3 and UV-B than are their corresponding host plants. Changes in host plants may result in expectable alterations of disease incidence, depending on host plant growth stages and type of pathogen. Given the importance of plant diseases in world food and fiber production, it is essential to begin studying the effects of increased CO_2, O_3 and UV-B (and other climate change factors) on plant diseases. We know very little about the actual impacts of climate change factors on disease epidemiology. Epidemiologists should be encouraged to consider CO_2, O_3 and UV-B as factors in their field studies.

INTRODUCTION

World population continues to grow, resulting in significant increases in urban development and agricultural, economic and industrial activities. Deforestation and habitat destruction are accelerating rapidly to accommodate the need for open space to support increasing population growth. Accompanying this are increased emissions of gases from agriculture, combustion of fossil fuels, and industrial processes. This has resulted in changes in the chemical composition of the atmosphere.

Concern about increased emission of gases into the atmosphere focuses on the possible or potential effects of accumulation of these gases above levels that can be tolerated and balanced by the self-regulating processes and dynamics of the atmosphere. Elevated concentrations of individual gases may have direct biological effects, or taken as a whole, they may also influence the earth's climate by causing changes in the atmospheric re-radiative effect (the 'greenhouse effect') resulting in atmospheric warming, global temperature increases, and changes in wind events and precipitation patterns.

An holistic perspective of climate change is presented in Fig. 1. It has been well documented that changes have occurred in the chemical composition of the atmosphere. Increases in radiatively active gases, such as carbon dioxide (CO_2), chlorofluorocarbons (CFCs), methane (CH_4), nitrous oxide (N_2O) and water vapor (H_2O), together with ozone (O_3) at pollutant concentrations, have been widely reported and summarized

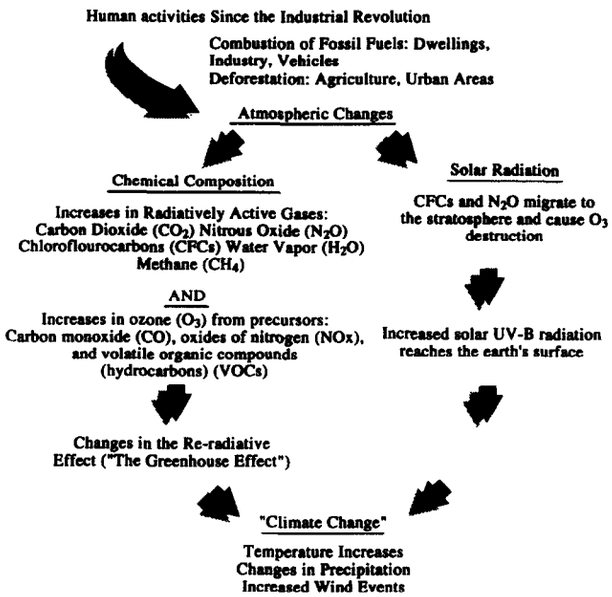

Fig. 1. An holistic perspective of climate change (Enquete Commission, 1992; Krupa & Kickert, 1989).

(Bishof *et al.*, 1985; Krupa & Kickert, 1989; Enquete Commission, 1992).

Evidence is emerging that increased solar ultraviolet-B (UV-B) radiation is reaching the earth's atmosphere, due to stratospheric O_3 depletion (Blumthaler & Ambach, 1990; Crutzen, 1992; Seckmeyer & McKenzie, 1992; Kerr & McElroy, 1993). While the magnitude of the increase in this narrow wavelength of solar radiation is still a matter of controversy, it may have potentially important biological effects.

There is considerable speculation and controversy regarding whether known changes in the chemical composition of the atmosphere have already, or will later, cause general climate change, as expressed in temperature increases (global warming) and associated changes in precipitation patterns (Lindzen, 1994). Incomplete data and problems with methods complicate the development and validation of climate change models needed to prove the assumption of cause/effect between increases in radiatively active gases and temperature increases. In view of this uncertainty, attempts to predict the biological significance of over-all climate change would be completely speculative.

Individual climate change factors, such as CO_2, O_3 and UV-B, are known to have direct biological effects on cultivated and native plants (Guderian *et al.*, 1985; Krupa & Manning, 1988; Bazzaz, 1990; Ashmore & Bell, 1991; Baker & Allen, 1994; Rogers *et al.*, 1994; Runeckles & Krupa, 1994). A considerable amount of research has been conducted on the individual effects of these factors on biomass formation and/or crop yields. It has also been demonstrated that CO_2, O_3 and UV-B can indirectly affect the incidence and severity of plant diseases, caused by biotic agents (Dowding, 1988; Manning & Keane, 1988; Colls & Unsworth, 1992; Rogers *et al.*, 1994; Runeckles & Krupa, 1994). By affecting plant or

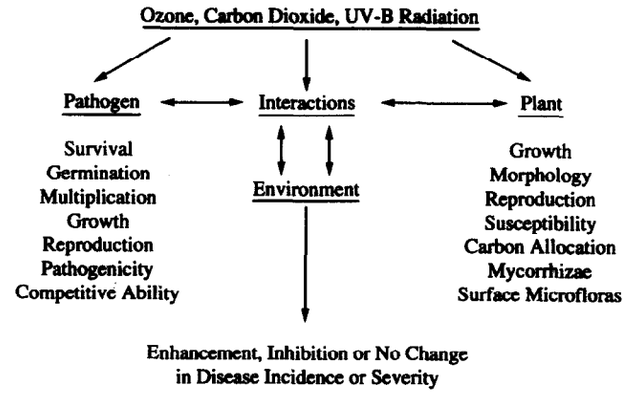

Fig. 2. Changes in disease incidence and severity due to CO_2, O_3 or UV-B and by affecting plant or pathogen.

pathogen (or both), CO_2, O_3 or UV-B may enhance, inhibit, or not change disease incidence or severity (Fig. 2).

An examination of important fungal diseases of wheat (*Triticum aestivum* L.) in North America and Europe (Table 1) indicates that the potential for yield losses due to biotic pathogens greatly exceeds that attributed to elevated O_3 effects alone. In view of what we know about ozone/pathogen interactions, however, it is quite likely that yield losses attributed to biotic agents alone, also reflect key predisposing indirect effects of O_3 on disease incidence as well (Manning *et al.*, 1969; Dohmen, 1988; Manning & Keane, 1988; Tiedemann, 1992*a,b*).

Carbon dioxide, ozone and ultraviolet-B radiation are increasing in the troposphere. Our purpose is to examine what is known about interactions of CO_2, O_3 and UV-B with plants and pathogens in relation to plant diseases and to consider the potential for change in disease incidence and severity if these climate change factors continue to increase.

Table 1. Yield reducing potential of fungal diseases of wheat (*Triticum aestivum* L.) compared to yield reductions reported to be due to elevated concentrations of ozone, adjusted for cultivar, site, and yearly variation.

Fungal pathogens	Disease	Maximum yield losses in the field (%)
Pseudocercosporella herpotrichoides	Eyespot (stem base)	10–20[a]
Fusarium species	Foot rot	10
Fusarium species	Leaf spots, ear scab	60
Septoria nodorum	Leaf and glume blotch	50–65
Septoria tritici	Speckled leaf blotch	10–30
Erysiphe graminis	Powdery mildew	10–20
Puccinia striiformis	Stripe rust	30–100
Puccinia recondita	Leaf rust	10–20
Puccinia graminis	Stem rust	10–100
Elevated ozone		5–15[b]

[a]Sources: European Handbook of Plant Diseases, 1988; Prillwitz, 1983.
[b]Data from OTC-studies in the USA (NCLAN 1980–88) and the European Open-Top Chamber Program (1984–91) (Heck *et al.*, 1988; Skarby *et al.*, 1992).

CARBON DIOXIDE

Tropospheric CO_2 concentrations are projected to increase from 355 ppm (v/v) to 710 ppm, by the year 2050. There is an enormous literature on the beneficial effects of elevated CO_2 concentrations on biomass production, probably due to increased water use efficiency (Cure, 1986; Bazzaz, 1990; Berntson & Woodward, 1992; Baker & Allen, 1994; Rogers *et al.*, 1994). Much less is known about CO_2 effects on the incidence and severity of biotic diseases of plants.

A summary of the effects of elevated concentrations of CO_2 on plant pathogenic bacteria and aerial and soilborne fungi is given in Table 2. These studies were conducted with cultures on agar plates or in liquids in sealed containers. Some of the soil fungi were also examined in non-sterilized and sterilized sand or soils.

Wells (1974) provided the one report on plant pathogenic *Erwinia* spp. and *Pseudomonas fluorescens*. No inhibitory effect was observed on cell growth in liquid culture from 0·03–3% CO_2.

Work with soil fungi has been of interest as the air in normal soils is normally enriched with CO_2 and may have as much as 6–18% CO_2 content, depending on organic matter decomposition, microbial and root respiration, and other factors (Papavizas & Davey, 1962).

Most soil-inhabiting fungi tolerate more than 10- or 20-fold increases in atmospheric CO_2 concentration. Some typical soil-borne plant pathogens like species of *Phytophthora, Aphanomyces, Sclerotium* and different pathotypes of *Fusarium oxysporum* have been found to be well adapted to and even multiply better at high CO_2 and low O_2 levels. This also seems to be a specific character of *Geotrichum candidum*. Stimulation of growth by carbon dioxide has been attributed to CO_2 fixation by the fungi. Carbon dioxide can be used as additional C-source by some fungi and incorporated into organic acids, like oxaloacetic acid, fumaric or citric acid, thus entering the Krebs cycle to be utilized for energy supply and growth (Tabak & Cooke, 1968; Wells & Uota, 1970). Isolates of *Rhizoctonia solani* and *Pythium irregulare* were inhibited by CO_2 concentrations exceeding 5–10%. Griffin and Nair (1968), however, reported that an isolate of *Sclerotium rolfsii* had reduced mycelial growth at near ambient CO_2.

There are fewer reports of elevated CO_2 effects on aerial fungi. *Rhizopus stolonifer, Cladosporium herbarum, Botrytis cinerea, Aspergillus niger* and *Alternaria tenuis* were inhibited at CO_2 concentrations exceeding 5–10%. Within current atmospheric ranges of CO_2, Cotty (1987), Smart *et al.* (1968) and Svircev *et al.* (1984) found inhibition of several species of *Alternaria* and *Peronospora hyoscyani* f. sp. *tabacina*.

Some attention has been paid to the effects of elevated CO_2 on nematodes in soil. Freckman *et al.* (1991) exposed cores of prairie soils to elevated CO_2 concentrations, but found no appreciable effects on numbers of nematodes or species composition. Rhizosphere numbers of nematodes tended to decrease in the root-zone soil of cotton plants exposed to elevated CO_2 (Runion *et al.*, 1993).

Most of the studies summarized in Table 2 involved the use of greatly elevated CO_2 concentrations. They have value as possible predictions of higher CO_2 effects, but they are not very relevant to current-level CO_2 effects. It could possibly be concluded from them, however, that increases in CO_2 from 0·03–0·07% will probably have no direct effects on most pathogens and may even be slightly stimulatory.

Much more interesting, but also much rarer, are studies on the CO_2-disease relationship, particularly if we consider that few researchers have worked with reasonable concentrations. Information reported in the literature from 1930 until today is summarized in Table 3. Several interesting studies on soilborne, above-ground and storage diseases, under the influence of elevated CO_2, have been published.

Soilborne diseases have been studied either by fumigating the soil containing inoculum or by incubating plants in enriched CO_2 atmospheres while growing in infested soil. In most cases, however, experiments were made with realistic CO_2 concentrations only for soil air composition, but far too high compared with the atmospheric CO_2. Carbon dioxide favors soilborne infections by *Fusarium* spp., especially the incitant of snow mold of cereals, and the members of the *F. oxysporum* group. In a study by Volk from 1931, soil containing basidiospores of *Ustilago hordei* (cause of covered smut of barley) was fumigated with CO_2 enriched air. The number of infected barley seedlings grown in that soil were significantly greater than those from soil fumigated with normal air. In a similar experiment, snow mold infection of rye seedlings grown in CO_2 fumigated sand increased to 33% compared to 19% without enrichment. High concentrations of CO_2 reduced seedling attack by *Rhizoctonia solani* (Papavizas & Davey, 1962), which corresponds well with the CO_2 sensitivity of that fungus. Other root diseases caused by *Pythium splendens* or *Thielaviopsis basicola* on poinsettia were not affected by elevated CO_2 atmospheres in the greenhouse (Zornbach & Schickedanz, 1987).

Undoubtedly, the prevalent effect of a global rise of CO_2 on biotic diseases will be exerted via changes in the physiological and morphological status of the host plant. This means that altering the predisposition of the plant will presumably be the predominant impact of a rise in CO_2 levels on the occurrence of biotic plant diseases. This may not only be expected from the much greater effects a CO_2 doubling evidently has on the growth of plants than on pathogens, but is also supported by some investigations with above-ground diseases.

One of the most comprehensive investigations conducted so far, however, originates from the early thirties. This outstanding study was published in German by Gassner and Straib in 1930 and dealt with effects of increasing atmospheric CO_2 concentrations on various rust diseases of cereals. The objective of those experiments was related to the potential effect of the carbohydrate status of cereals on susceptibility to rust

Table 2. Effects of elevated concentrations of carbon dioxide (CO_2) on growth and development of phytopathogenic bacteria and fungi

Organisms	Exposure systems	Range of CO_2 concentrations resulting in			Remarks on effects	Reference
		Inhibition	No effect	Stimulation		
Bacteria						
Enwinia atroseptica	Liquid	> 3%	0·03–3%	—	Measured in air with 21% oxygen	Wells, 1974
E. carotovora						
Pseudomonas fluorescens		> 10%	0·03–3%	—		
Fungi						
Aerial						
Alternaria cassiae, A. crassa	Agar	> 0·04%	—	—	Effects refer to sporulation	Smart *et al.*, 1968
A. brassicae, A. citri., A. carthami	Agar	> 0·117%	—	—	Enrichment by sealing petri dlshes, 83–100% inhibition of sporulation at 0·23%	Cotty, 1987
A. cucumerina, A. macrospora, A. porri, A. raphani, A. tagetica						
Alternaria tenuis	Liquid	> 10–16%	0·03–32%	—	Mycelial growth inhibited at 10% or more, no effect on spore germination up to 32%	Wells & Uota, 1970 Mitchell & Mitchell, 1973
Aphanomyces euteiches	Liquid	15%	—	5%	Effects on oospore production	
Aspergillus niger	Liquid	> 5%	Up to 5%	—	Effects refer to % conidial germination	Svircev, *et al.*, 1984
Botrytis cinerea	Liquid	> 4–8%	—	—	Spore germination inhibited at 4% or more, mycelial growth inhibited	Wells & Uota, 1970
B. cinerea	Agar	> 1·3%	Up to 1·3%	—	Refers to effects on conidial germination	Svircev *et al.*, 1984
Cladosporium fulvum	Agar	—	Up to 5%	—	No effects on conidial germination	Volk, 1931
C. herbarum	Liquid	> 4–8%	—	—	Spore germination inhibited at 4% or greater	Wells & Uota, 1970
Peronospora hyoscyami	Agar	> 0·8%	0·5–0·8%	—	Refers to effects on sporangial germination	Svircev *et al.*, 1984
Rhizopus stolonifer	Liquid	> 4–8%	—	—	Mycelial growth inhibited above 10%, spore germination inhibited at 4% or more	Wells & Uota, 1970
Soilborne						
Fusarium oxysporum	Infested					
F. sp. *cucumerinum*	Loam/sand	—	—	4%	Substantial stimulation of growth in soil	Stover & Freiberg, 1958
F. sp. *lycopersici*		—	—	4%		
F. sp. *nicotianae*		—	—	4%		
F. *oxysporum*	Agar	80–100% completely 5–60% gradually	0·3–10%	—	Inhibitory effects on mycelial growth	Toler *et al.*, 1965
F. tracheilphilum						
F. roseum	Liquid	> 10–16%	—	Up to 4–10%	Mycelial growth and sporulation stimulated	Wells & Uota, 1970
F. solani f. *pisi*	Liquid	> 5–15%	—	—	Effects on mycelial growth	Mitchell & Mitchell, 1973
Geotrichum candidum	Liquid	3–30%	—	1%	Growth stimulation in low O_2 atmosphere	Wells & Spalding, 1975
G. candidum	Agar	—	—	Enriched	Stimulation of hyphal growth and arthrospore germination	Robinson & Thompson, 1982
Phytophthora parasitica nicotianae	Soil Agar	> 15%	0·03–5%		High CO_2 and low O_2 tolerated	Dukes & Apple, 1965
Phytophthora megasperma	Liquid	15%	5%	—	Effects on oospore production	Mitchell & Mitchell, 1973
P. capsici. P. citrophthora	Liquid	> 5–15%	—	Up to 5%	Effects on mycelial growth (at atmospheric O_2)	Mitchell & Zentmyer, 1971
P. palmivora	or solid	5%	Up to 5%			
P. capsici, P. citrophthora, P. palmivora, P. cinnamomi, P. megasperma	Liquid or solid	5–15%	—	—	Reduction of oospores and sporangia (at atmospheric O_2)	Mitchell & Zentmyer, 1971
P. drechsleri, P. parasitica						
Pythium irregulare	Liquid	> 5%	—	—	Effects on oospore production	Mitchell & Mitchell, 1973
Rhizoctonia solani	Agar tube	20%	0·5–2·5%	Up to 0·5%	Aerial isolates more sensitive than subterranean strains	Durbin, 1959
R. solani	Unsterilized soil	> 10%	—	—	Colonization of organic baits inhibited	Papavizas & Davey, 1962
R solani	Liquid	> 5–15%	—	—	Effects on mycelial growth	Mitchell & Mitchell, 1973
Sclerotinia minor	Agar	> 8%	—	—	Effects on mycelial growth & sclerotial formation	Imolehin & Grogan, 1980
		> 20%	0·03–20%		Effects on sclerotial germination	
Sclerotium rolfsii	Liquid	> 5–15%	—	—	Effects on mycelial growth	Mitchell & Mitchell, 1973
S. rolfsii	Agar	> 0·03%	—	—	Effects on mycelial growth	Griffin & Nair, 1968
		> 5%	—	0·03–3·3%	Effects on formation of sclerotia	
S. rolfsii	Cellophane	> 3–5%	—	0·5–2·5%	Effects on mycelial growth	Kritzman *et al.*, 1977
	Agar culture	0·3%	—	—	Suppression of sclerotial formation,	
S. rolfsii	Agar	20%	0·5–9%	—	Effects on sclerotial germination	Punja & Jenkins, 1984

diseases. They inoculated wheat, rye and oat plants with several rusts and then exposed the plants to 0·03, 0·15, 0·3, 0·75, 1·5, 4·5 and 6% CO_2. Exposures were continued until the outbreak of rust pustules. The result was a substantial promotion of the different rust diseases within a range of 0·15 to 0·75% CO_2 where the fastest and strongest development of uredia was observed (Table 3). Optimal concentrations of atmospheric carbon dioxide for growth of stem rust and stripe rust on wheat were slightly higher (0·3–0·75%) than for crown rust on oats, leaf rust on rye and wheat (0·15–0·5%). Concentrations of 3–7·5% CO_2 had no visible harmful effects on uredospore germination and early development of germ tubes of the rust fungi tested.

In another early and remarkable study, Volk (1931) exposed tomato plants inoculated with *Cladosporium fulvum* and maize plants inoculated with *Ustilago maydis* to concentrations of CO_2 increasing from ambient to 0·5 and 5%. At 0·5% the disease symptoms in both systems developed earlier, spread more readily and sporulation was more intense than in air containing normal levels of CO_2. At 5% both plant growth and disease were inhibited. Enrichment of the air with CO_2 had no effect on infection of lettuce leaf disks by *Sclerotinia minor* and of cyclamen plants by *Botrytis cinerea*, while powdery mildew on roses was reduced (Table 3).

An early study on CO_2 effects on stored potato tubers and infections by *Alernanari solani* was published by Klaus in 1943. Tuber infections were not affected by concentrations of up to 12% CO_2 in the storage room, while mycelial growth of the fungus in pure culture was inhibited at 5% CO_2. Other experiments have demonstrated a reduction of several fruit or flower rots when storing the material in controlled atmospheres with extremely high concentrations of carbon dioxide. These studies, however, provide no information valuable in an estimation of climate change effects on plant diseases.

While not regarded as plant pathogens, endomycorrhizal fungi (vesicular–arbuscular or VA fungi) are commonly associated with the roots of many herbaceous plants and a few tree species. Ectomycorrhizal fungi form mycorrhizae with roots of many trees and woody perennial plants. Mycorrhizae provide many benefits to plants including protecting roots from toxins and root pathogens and increasing uptake of water and minerals (Ruehle & Marx, 1974; Dighton & Jansen, 1991). Any above-ground factor that affects photosynthesis and carbon allocation will affect incidence and vitality of mycorrhizae (Andersen & Rygiewicz, 1991).

Elevated CO_2 is expected to cause increases in plant root volumes and lengths (Stulen & den Hertog, 1993; Rogers *et al.*, 1994). Increased root biomass could result in an increase in mycorrhizae. For ectomycorrhizae, this could mean an increase in the number of morphological types (morphotypes) associated with a root system and a corresponding increase in fruitbody production (Dighton & Jansen, 1991). This has been demonstrated with shortleaf pine and white oak seedlings (Norby *et al.*, 1987; O'Neill *et al.*, 1987).

Root surfaces (rhizoplanes) and the narrow zones of soil around roots (rhizospheres) greatly influence the activities of the soil microflora and microfauna. Curl and Truelove (1986) consider that 90% of the microbial population of the soil is found in the rhizosphere. Plant-mediated effects on rhizodeposition (release of dead cells, mucilages, exudates, etc.) from plant roots will have corresponding effects on rhizoplane and rhizosphere microfloras. This could lead to an increase in growth-promoting microorganisms or an increase in root diseases. Increases in mycorrhizae may influence root disease incidence. We know so very little about the influence of elevated CO_2 levels on plant/root/rhizosphere/mycorrhiza/pathogen interactions.

A summary of reported effects of elevated CO_2 concentrations on biotic plant diseases is given in Table 4. Disease enhancement occurred for four of nine reported diseases caused by necrotrophic fungi. Six of seven cases of reported diseases were enhanced where biotrophic fungi were the causal agents. There are too few reported incidents to draw many trends or conclusions, but there are clear indications that considerably more work needs to be done in this area. CO_2 concentrations will continue to increase and we need to know more about elevated CO_2 effects on disease incidence and severity.

While not much is known about elevated CO_2 concentrations on plant diseases, we do know considerably more about the effects of increased CO_2 on plants. Using this information, we could postulate potential effects on disease incidence and epidemiology. A summary of major plant responses to elevated CO_2 and corresponding potential effects on the incidence and epidemiology of plant diseases is given in Table 5.

An increase in plant canopy size and density would mean the availability of greater biomass of high nutritional quality, combined with a much higher microclimate humidity. This will likely promote plant diseases, such as rusts, powdery mildews, leaf spots and blights. The inoculum potential of necrotrophs, from greater overwintering crop debris, would be appreciably increased.

OZONE

Increases in carbon monoxide (CO), oxides of nitrogen (NO_x) and volatile organic compounds (VOCs) will continue to cause increases in tropospheric O_3 (Fig. 1). In the northeastern United States in 1993 there were widespread violations of the US standard for O_3 (120 ppb for one hour) (NESCAUM, 1993). Ozone is also becoming a problem in eastern European countries, such as Poland (Bytnerowicz *et al.*, 1993). Significant increases in surface ozone within the last 100 years is confirmed by long-term measurements in Germany and France (Feister & Warmbt, 1987; Volz & Kley, 1988). Ashmore and Bell (1991), Krupa and Kickert (1989) and Penkett (1988) provide complete reviews of increases in tropospheric O_3 and its potential role in climate change. Chameides *et al.* (1994) estimate 10–35% of the world's grain producing areas are already

Table 3. Effects of elevated concentrations of CO_2 on plant diseases

Pathogen	Host plant	Exposure system	Doses	Effects of CO_2 on disease parameters	Reference
Soilborne diseases					
Fusarium nivale	Rye	Fumigated sand	CO_2 enriched air	Increased number of infected seedlings	Volk, 1931
Ustilago hordei	Barley	Fumigated, unsterile soil	CO_2 enched air	Increased number of smutted ears	Volk, 1931
Fusarium oxysporum f. sp. *cyclaminis*	Cyclamen	Greenhouse chambers	0·18%	Earlier and more severe disease symptoms	Zornbach & Schickedanz, 1987
Pythium splendens	Poinsettia	Greenhouse chambers	0·18%	No differences in infection severity compared to control	Zornbach & Schickedanz, 1987
Thielaviopsis basicola		Greenhouse chambers	0·18%		
Fusarium sp.	Wheat	Enriched atmosphere	2–8%	Strong enhancement of snow mold	Gaeumann, 1951
Rhizoctonia solani	Radish, sugar beet	Fumigated, unsterile soil	10, 20, 30%	Inhibition of emergence due to soil infestation strongly reduced	Papavizas & Davey, 1962
Above-ground diseases					
Botrytis cinerea	Cyclamen	Greenhouse chambers	0·18%	No effects on severity of shoot infection	Zornbach & Schickedanz, 1987
Sphaerotheca pannosa	Roses	Greenhouse chambers	0·18%	Reduced infection and sporulation	
Cladosporium fulvum	Tomato	Chambers,	0·2, 05 and	Enhancement of disease and sporulation at 0·5%	Volk, 1931
Ustilago maydis	Maize	controlled fumigation	5%	inhibition of disease at 5%	Volk, 1931
Puccinia striiformis	Wheat	Glass cuvettes in the greenhouse	0·03,0·15,0·3 0·75,1·5,4·5 & 6%	Most rapid and intense pustule formation at 0·3-0·75%, inhibition above 1·5%	Gassner & Straib, 1930 Gassner & Straib, 1930
P. coronata	Oats	Glass cuvettes in the greenhouse	0·03,0·15,0·3 0·75,1·5,4·5 & 6%	Optimum 0·15–0·5%, inhibition above 4·5%	Gassner & Straib, 1930
P. dispersa	Rye	Glass cuvettes in the greenhouse	0·03,0·15,0·3 0·75,1·5,4·5 & 6%	Optimum 0·15–0·75%, inhibition above 4·5%	Gassner & Straib, 1930
P. graminis tritici	Wheat	Glass cuvettes in the greenhouse	0·03,0·15,0·3 0·75,1·5,4·5 & 6%	Optimum 0·3–0·75%, inhibition above 6%	Gassner & Straib, 1930
P. recondita tritici	Wheat	Glass cuvettes in the g reenhouse	0·03,0·15,0·3 0·75,1·5,4·5 & 6%	Optimum 0·15–0·3%, inhibition above 6%	Gassner & Straib, 1930
Sclerotinia minor	Lettuce	Leaf disks in petri plates	2·1–20·2%	No effect on infection and sclerotial formation up to 8·5% CO_2; both parameters reduced above that value (tested at normal oxygen level)	Imolehin & Grogan, 1980
Storage diseases					
Alternaria solani	Potato (tubers)	Fumigated glass cuvette	12%	No effect on tuber infections	Klaus, 1943
Botrytis cinerea	Roses	Climate chamber	10, 20, 30%	Substantial reduction of flower rot	Phillips, 1985
Botrytis cinerea *Rhizopus stolonifer*	Strawberries	Climate chamber	10, 20, 30%	Reduced decay at 10% CO_2 or more	Couey & Wells, 1970
Fusarium sp. *solani* *Alternaria solani* *Botrytis cinerea* *Penicillium* spp. *Rhizopus* spp. *Sclerotinia sclerotorum*	Tomato (fruits)	Fumigated storage chamber	3%	Reduction of all tomato rots except *A. tenuis* and *Fusarium* spp.,the latter being strongly favored in the CO_2 enriched atmosphere (oxygen conc. was 3%)	Lockhart *et al.*, 1969
Pseudomonas marginialis	Tomato (fruits)	Fumigated glass cuvette	2–10%	Strong reduction of soft rot under low oxygen concentrations	Ibe, 1983

Table 4. Summary of reported effects of elevated carbon dioxide concentrations on plant diseases caused by fungi

Pathogen Group	Total number of diseases reported	Number of cases reported where diseases were		
		Enhanced	Not affected	Reduced
Necrotic fungi	9	4	4	1
Biotrophic fungi	7	6	—	1

Literature reviewed from 1930–93.

Table 5. Potential effects of CO_2 on plant diseases extrapolated from the major responses of plants to a CO_2 doubling in the atmosphere

Major plant responses on elevated carbon dioxide concentrations	Potential effects on the incidence and epidemiology of plant diseases
Shoot responses	
Increased biomass production (increased number of branches, shoots, tillers, leaves, flowers and fruits)	Increased mass of utilizable host tissue for pathogens on stems and leaves
Increased carbohydrate content	Promoted growth of sugar-dependent pathogens (i.e. rusts, powdery mildews)
Increased canopy density and height	Promoted growth, sporulation and spread of most leaf infecting fungi requiring high air humidity but not rain (rusts, powdery mildews, leaf necrotrophs)
Increased mass of crop residues	Improved conditions for necrotrophic pathogens overwintering in/on plant residues
Reduced opening of stomates	Inhibition of stomata-invading pathogens (rusts, downy mildews, some necrotrophs)
Accelerated ripening and senescence, shortened growth period	Reduced infection period for biotrophic pathogens (rusts, powdery mildews), promotion of necrotrophic pathogens
Root responses	
Increased root biomass and dry weight, root length, and root/total shoot weight ratio	Increased proportion of host tissue utilizable for mycorrhizal fungi/or soil-inhabiting pathogens; increased compensation in root mass for loss to pathogens
Increased root exudation	Stimulation of pathogenic *and* antagonistic (plant growth promoting) microflora in the rhizosphere

Table 6. Effects of virus infections on ozone sensitivity of plants

Virus	Host plant	Ozone dose	Effects on ozone sensitivity	References
Alfalfa mosaic	Pinto bean	0·25 ppm/4 hr	Partial projection from ozone injury	Davis & Smith, 1976
Bean common mosaic	Pinto bean	0·25 ppm for 4 hr	Partial protection from ozone injury	Davis & Smith, 1974
Peanut stunt	White clover	0·038-0·097 ppm for 25–29 in OTC(12 h/d)	Neither reduction nor increase of ozone injury	Heagle *et al.*, 1992
Tobacco etch	Tobacco	0·25 ppm for 4 h	Protection against ozone injury	Moyer & Smith, 1975
	Tobacco	0·05–0·40 ppm for 3 h/d	Less ozone-induced growth suppression	Reinert *et al.*, 1988
Tobacco mosaic	Tobacco	0·30–0·40 ppm for 3-6 h	Suppression of ozone injury symptoms	Brennan & Leone, 1969
	Tobacco	ambient air (field)	60% less ozone injury	Bisessar & Temple, 1977
	Bean	0·35-0·40 ppm for 4 h	Systemic nature of virus protection against ozone: partial protection of non-inoculated primary leaves	Vargo *et al.*, 1978
	Pinto bean	0·25 ppm for 4 h	Systemic induction of resistance	Davis & Smith, 1976
Tobacco streak	Tobacco	0·30 ppm for 3 h or 3 + 3h	Increased susceptibility to ozone injury	Reinert & Gooding, 1978
Tobacco vein mottle	Tobacco	5 d/w for 3 weeks	Additive growth suppression effects on 2 of 3 cultivars	Reinert *et al.*, 1988
Tomato ring-spot, tobacco ring-spot	Pinto bean	0·25 ppm for 4 h	Partial protection from ozone injury	Davis & Smith, 1976

Table 7. Effects of ozone on bacterial diseases of plants

Bacterium	Host plant	Ozone dose chronic/acute	Pre/post-inoculation ozone exposure	Effects on disease severity	References
Pseudomonas glycinea	Soybean	Acute	Pre- or post-inoculation	Reduced number of lesions	Laurence & Wood, 1978*a*
Xanthomonas alfalfae	Alfalfa	Acute	Pre-inoculation	Reduced bacterial infection severity	Howell & Graham, 1977
Xanthomonas fragariae	Wild strawberry	Acute	Pre- or post-inoculation	Reduced number of lesions	Laurence & Wood, 1978*b*
Xanthomonas phaseoli	White bean	Acute (field)	Pre- and post-inoculation	No effect, i.e. no protection against bacterial blight	Temple & Bisessar, 1979

exposed to O_3 concentrations that may reduce yields. Failure to reduce NO_x (and VOC) emissions now, and in the future, could result in O_3 concentrations three times the present levels by the year 2025. While this would directly affect plant growth and productivity, effects on plant disease incidence are not known. Examination of reports from current and past literature, however, may provide some indications of future effects.

Virus-infected plants are usually partially or completely protected from ozone injury (Table 6). This has been mainly observed in bean and tobacco, both in growth chambers and in the field. There are also a few examples for additive effects of virus infections and ozone injury and one case where no virus-ozone interaction occurred (Heagle *et al.*, 1992).

The predisposing effects of ozone on the severity of bacterial diseases have been described in four studies (Table 7). In three of them, the pre-inoculation treatment with ozone, reduced the severity of the following bacterial infections, while one field study did not find any changes in the development of the bacterial symptoms (Temple & Bisessar, 1979). It is noteworthy to mention that in all these studies ozone exposure had caused acute injury to the leaves before bacterial infections occurred. This might have triggered an efficient induced resistance in the plant which is well-known from many other host-pathogen studies.

Few publications deal with the protective effects of bacterial or fungal infections in relation to ozone injury. Two earlier reports parallel with virus studies in demonstrating induction of partial ozone resistance of infected plants. (Table 8). Furthermore, similar protective effects have also been demonstrated on wheat infected with the stem rust fungus (Heagle & Key, 1973*a,b*), on broad beans infected with *Botrytis cinerea* (Magdycz & Manning, 1973) and on peas infected with powdery mildew *Erysiphe polygoni* f sp. *pisi* (Rusch & Laurence, 1993).

There are many publications regarding fungal diseases and ozone. This reflects the high significance fungi have as plant pathogens, their worldwide distribution, high economic importance and huge multitude of host plants. Unquestionably, ozone alters the host plant which in turn may affect its susceptibility to fungal pathogens. Necrotrophic and obligate biotrophs, however, prefer quite different types of predisposition of their host plants. Whereas the first infect and grow better on weakened host tissue, the latter generally are adapted to (and depend on) healthy plants. This is the reason why most researchers have assumed that ozone levels, adverse for the plant, would be also adverse for obligate biotrophs, but favorable for necrotrophs. It is therefore reasonable to look at the two groups separately. When we do this we see that there are some interesting exceptions to that general conclusion.

Earlier work on leaf diseases intensively dealt with the effects of ozone on *Botrytis* species on several crop plants like potato, geranium, onion and bean (Table 9). Manning *et al.* (1969*a*) first reported a new disease syndrome on potato leaves which required O_3 injury as a predisposing factor for leaf blight, caused by *Botrytis cinerea*. Similar studies were conducted with *Alternaria solani* on potato (Bisessar, 1982; Holley *et al.*, 1985). The general observation was an enhancement of infection rates or disease severity due to availability of the ozone-induced lesions, which were thought to serve as

Table 8. Effects of bacterial and fungal infections on ozone sensitivity of plants

Pathogen	Host plant	Ozone dose	Effects on ozone sensitivity	References
Bacteria				
Xanthomonas alfalfae	Alfalfa	0·20 for 4 h	Decreased sensitivity to ozone	Howell & Graham, 1977
Xanthomonas phaseoli	White bean	Ambient air (field)	Decreased susceptibility to ozone injury	Temple & Bisessar, 1979
Fungi				
*Puccinia graminis f.*sp. *tritici*	Wheat	0·24 ppm for 6 h 3 or 4 d	Decreased ozone injury in the substomatal mesophyll areas	Heagle & Key, 1973*b*
Botrytis cinerea	Broad bean	0·20 ppm for 8 h	Noninjured (protected) haloes around fungal lesions	Magdycz & Manning, 1973

Table 9. Effects of ozone on above-ground diseases of plants caused by necrotrophic fungi

Fungus	Host plant	Ozone injury visible	Ozone exposure pre-/post-inoculation exposure technique	Effects of ozone on disease severity	References
Alternaria solani	Potato	Yes	Pre-/post inoculation field	Enhanced colonization of ozone-injured sites of leaves	Bisessar, 1982
	Potato	Yes	Pre-/post-inoculation field	Ozone injuries are used as infection courts	Holley *et al.*, 1985
Ascochyta sp., *Gerlachia nivalis* *Drechslera sorokiniana*	Wheat	No	Pre-inoculation CC	Increased percentage of diseased leaf area; effects cultivar-dependent	Fehrmann *et al.*, 1986
Botrytis cinerea	Potato	Yes	Pre-inoculation GH	Enhanced infection only on ozone- injured leaves	Manning *et al.*, 1969*a*
	Geranium	Yes	Pre-inoculation GH	Enhanced infection only on ozone- injured leaves	Manning *et al.*, 1969*b*
	Poinsettia	Yes (leaves), No (bracts)	Post-inoculation GH	No effect on disease incidence, on ozone-injured leaves or on non-injured bracts	Manning *et al.*, 1972
	Geranium	Yes	Post-inoculation CC	Adverse effects on fungal growth on host plant	Krause & Weidensaul 1978
	Grapevine	No	Pre-inoculation CC	Number of lesions unaffected or reduced on ozone-treated plants	Tiedemann & Ferhmann, 1986; Tiedemann *et al.*, 1990
	French beans	Yes	Pre-inoculation CC	Lesion number correlated with percentage ozone injury, reduced infection at medium ozone dosages in the presence of inorganic phosphate	Leone &Tonneijck, 1990; Tonneijck, 1994
B. cinerea *B. squamosa*	Onion	Yes/no	Pre/post-inoculation CC	*B. cinerea* infection enhanced both by chronic and acute exposure; only by acute exposure; *B. squamosa* only by acute exposure; no enhancement of lesion formation by of ozone	Rist & Lorbeer, 1984*a*
B. cinerea, B. allii *B. squamosa*	Onion	Yes	Pre- and post-moculation OTC	Enhanced infection via ozone-induced wounds and due to leakage	Wukasch & Hofstra, 1977*a,b*
Drechslera teres *D. sorokiniana*	Barley	No	Pre-inoculation CC	Increased percentage of diseased leaf area; effects cultivar-dependent	Fehrmann *et al.*, 1986
Fusarium subglutinans	Loblolly pine	No	Post-inoculation OTC	2·5 × ambient O_3 exposure resulted in bigger cankers on susceptible half-sib families	Casey & Kelly, 1994
Helminthosporium maydis	Maize	No	Pre and/or post-inoculation CC	No effects on lesion sizes; sporulation enhanced after preinoculative exposure to medium doses of ozone, but reduced on plants exposed to ozone after inoculation	Heagle, 1976
Lophodermium sp.	Pine	Yes	Pre/post-inoculation	Increased severity of fungal needle blight	Alvarado & Bauer, 1991
Septoria nodorum.	Wheat	No	Pre-inoculation CC	Increased percentage of diseased leaf area, shortened duration of latent period	Tiedemann, 1992*a*
Septoria nodorum *D. sorokiniang*	Wheat	Yes/no	Pre-inoculation OTC	Increased percentage of diseased leaf area, not dependent on acute leaf damage	Tiedemann *et al.*, 1991
Septoria musiva	Poplar	—	Pre/post inoculation OTC	Increased stem canker formation and dying-off	Woodbury *et al.*, 1993

GH = greenhouse chamber; cc = climate chamber; OTC = open-top chamber.

infection courts for the fungus. More recent work on cereals has shown that acute ozone injury is not required for the enhancement of several leaf spot fungi such as *Drechslera* species, *Gerlachia nivalis*, *Ascochyta* sp. or *Septoria nodorum* (Fehrmann *et al.*, 1986). There are, however, some cases where ozone appeared to have no effects on foliar diseases. This was true for *Helminthosporium maydis* on maize (Heagle, 1977), *Marssonina brunnea* on eastern cottonwood (Coleman *et al.*, 1987*a*), and *Botrytis cinerea* on grapes (Tiedemann & Fehrmann, 1986; Tiedemann *et al.*, 1990). The work with grapes is the only study where *Botrytis* was applied to exposed leaves not previously visibly injured by ozone. Interestingly, in this case ozone induced resistance against *Botrytis*. Other studies which have applied post-inoculation fumigations with elevated doses of ozone are difficult to evaluate, as the direct inhibition of pathogens cannot be separated from effects on plant susceptibility.

Unlike CO_2, O_3 does not penetrate the soil surface (Turner *et al.*, 1973). Ozone does affect photosynthesis (Reich & Amundson, 1985) which has indirect effects on root growth, senescence, and health via changes in carbon allocation patterns (Cooley & Manning, 1987). Changes in carbon allocation can affect root growth and senescence. Manning *et al.* (1971*b*) investigated the effects of chronic exposure to O_3 on the rhizoplane mycoflora of pinto bean roots over a 28-day period. They found that O_3 accelerated shoot and root senescence and increased the rate of colonization of roots and hypocotyls by several normal decay fungi. None of the fungi isolated were pathogenic to pinto bean roots in the absence of ozone stress.

Below-ground diseases have been studied in post-inoculation exposure experiments (plants growing in artificially-infested soils) or in forest stands at polluted vs less polluted sites (Table 10). Any direct effect of ozone on the pathogen was prevented through the absorptive capabilities of the soil medium (Turner *et al.*, 1973). Root rot diseases caused by *Heterobasidium annosum* were enhanced on ponderosa pine by ozone injury (James *et al.*, 1980, 1982). Black stain disease, caused by *Leptographium wagneri* var. *ponderosum*, increased on roots of pondersoa pine trees exposed to ozone (Fenn *et al.*, 1990). Root infections of white pine by *Verticicladiella procera* were increased in ozone-stressed white pine (Skelly *et al.*, 1983). *Fusarium* wilt was delayed on tomato and not influenced on soybean or cabbage as a result of exposure of the plants to ozone (Manning *et al.*, 1971*a*; Manning & Vardaro, 1976; Damicone, *et al.*, 1987). Brown root rot of tomato, caused by *Pyrenochaeta lycopersici* increased in roots of plants exposed to O_3 (Manning & Vardaro, 1974).

Ozone has been shown to reduce endomycorrhizae on citrange and tomato, but not on soybean (Table 11). Variable results have been obtained for O_3/ectomycorrhizae interactions (Table 11). Using a natural mixture of loblolly pine ectomycorrhizal fungi in a natural soil, Meier and coworkers (1990) did, however, find a linear dose/response relationship between O_3 concentrations and intensity of mycorrhizae formed. Much more needs to be done to determine the influence of foliar O_3 injury on mycorrhizae.

A general enhancement of diseases caused by necrotrophic fungi due to plant exposure to ozone, though found in the majority of cases studied, cannot always be assumed. Moreover, factors like timing of exposure in relation to infection, presence or absence of acute injury caused by O_3 before inoculation, and the specific host-parasite relationship itself are likely to play an important role in determining the direction of predisposing effects. If acute ozone injury precedes inoculation, the results are different than when a host plant only experiences nonvisible physiological alterations before inoculation.

The effects of ozone on fungal diseases of plants caused by obligate biotrophs are summarized in Table 12. Barley powdery mildew was found to be relatively ozone tolerant and only inhibited by elevated ozone doses acting during germ-tube growth. Post-inoculation exposure of powdery mildew already established on the plant increased the size of colonies on barley and the severity of the disease on wheat. Pre-inoculative exposure of wheat to ozone, however, was inhibitive for mildew growth. Rusch and Laurence (1993) have recently demonstrated that pea powdery mildew (*Erysiphe polygoni* f. sp. *pisi*) infections can suppress foliar ozone injury. Ozone exposure before and after inoculation with *E. polygoni* f. sp. *pisi* conidia, reduced powdery mildew on pea leaves.

Studies reporting the direct effects of ozone on pathogens are summarized in Table 13. Most of the in-vitro studies with fungi in culture involved exposures to very high O_3 concentrations and were often conducted under conditions not conducive to O_3 uptake. For both in-vitro and plant surface studies, the effects were strongly influenced by the stage of development of the fungus, the amount of ozone applied, and by the species of fungus involved. The species influence evidently is largely determined by spore morphology and pigmentation for which there is a great diversity among fungi. Moisture status also appeared to be a key factor determining fungal sensitivity to ozone. In an early in-vitro study on ozone sensitivity of 14 heterotrophic fungi, dry spores or spores immersed in a liquid medium were more or less insensitive even if exposed to 1 ppm ozone for 6 h. However, sensitivity significantly increased when spores were exposed under moist conditions on a layer of agar. At higher doses of ozone (0·5–1·0 ppm), there was a tendency of greater tolerance of pigmented and multicellular spores, compared to thin-walled hyaline spores, for which inhibition began at 0·25 ppm after 4–6 h. No species was inhibited by ozone at doses of 0·1 ppm or less (Hibben & Stotzky, 1969) (Table 13).

The significance of pigmentation for ozone sensitivity of fungi is supported by a study reported by Treshow *et al.* (1969). They demonstrated that the two pigmented fungi *Helminthosporium sativum* and *Alternaria oleraceae* were not affected by exposure to 0·1, 0·4 or

Table 10. Effects of ozone on below-ground diseases of plants caused by necrotrophic fungi

Fungus	Host plant	Ozone injury visible	Ozone exposure pre-/post-inoculation exposure techniques	Effects of ozone on disease severity	References
Fusarium oxysporum	Soybean	No	Post-inoculation GH	No effect of ozone on root and hypocotyl rot severity	Damicone *et al.*, 1987
F. oxysporum f. sp. *conglutinans*	Cabbage	No	Post-inoculation GH	No effect on wilt	Manning, *et al.*, 1971*a*
F. oxysporum f. sp. *lycopersici*	Tomato	No	Post-inoculation GH	Wilt symptoms delayed by ozone exposure	Manning & Vardaro, 1976
Heterobasidion annosum (Fomes annosus)	Ponderosa pine	Yes	Pre/post inoculation/forest	Correlation of root collar colonization with oxidant injury	James *et al.*, 1982
	Ponderosa & Jeffrey pine	Yes	Post-inoculation GH	Ozone increased root infections No effect of ozone-induced foliar damage or root infection	James *et al.*, 1980
	White pine	Yes	Pre/post inoculation forest		Leininger *et al.*, 1990
Leptographium wagneri var *ponderosum*	Ponderosa pine	Yes	Post-inoculation OTC	Increased development of black stain disease on roots of fumigated trees	Fenn, 1990
Pyrenochaeta lycopersici	Tomato	Yes	Post-inoculation GH	Increased brown root rot disease	Manning & Vardaro, 1974
Verticicladiella procera	White pine	Yes	Natural forest	Increased root disease with ozone stress	Skelly *et al.*, 1983

GH = greenhouse (chamber); CC = Climate chamber; OTC = open-top chamber.

Table 11. Effects of elevated concentrations of ozone on formation of mycorrhizae by endomycorrhizal (vesicular–arbuscular (VA)) and ectomycorrhizal fungi

Fungi	Host plant	Exposure systems & ozone concentrations	Ozone effects on mycorrhizae	References
Endomycorrhizal fungi				
Glomus fasciculatus	Citrange	0·15–0·30 ppm GH	Mycorrhizae reduced, chlamydospores reduced	McCool *et al.*, 1979
	Tomato	do	do	McCool & Menge, 1983
Glomus geosporum	Soybean	0·079–0·250 ppm OTC	No inhibition at 0·079 ppm — fewer mycorrhizae and chlamdospores at 0·250 ppm	Brewer & Heagle, 1983
Ectomycorrhizal fungi				
Pisolithus tinctorius	Loblolly pine	0·070 ppm GH	No effect on mycorrhizae, protection from O_3 injury	Mahoney *et al.*, 1985
	White birch	0·06–0·08 ppm 12 weeks, GH	No effects on mycorrhizae	Keane & Manning, 1988
	Pitch pine	0–0·200 ppm 13 weeks, GH	Decrease in mycorrhizae abe 0·050 ppm	McQuattie & Schier, 1992
Natural mixture of ectomycorrhizal fungi in soils	Loblolly pine	0–0·150 ppm GH	Linear dose/response relationship between O_3 concentration and intensity of mycorrhizal formed	Meier *et al.*, 1990
	White pine Red oak	0·02–0·14 ppm 0·02–0·07 ppm GH, 5 days/week	Declines in mycorrhizae above 50 ppb O_3 low doses stimulated mycorrhizae, higher caused declines	Reich *et al.*, 1985, 1986
	Norway spruce	0·05 or 0·10 ppm O_3	No effects attributed to O_3	Blaschke & Weiss, 1990
	Loblolly pine	Ambient and 2 × ambient OTC, three years	2 × ambient reduced mycorrhizae, no consistent pattern in morphotype changes	Edwards & Kelly, 1992

GH = greenhouse chambers, OTC = open-top chamber.

Table 12. Effects of ozone on fungal diseases of plants caused by obligate biotrophs

Fungus	Host plant	Ozone injury visible	Ozone exposure pre-/post-inoculation exposure technique	Effects of ozone on disease severity	References
Rusts					
Melampsora medusae	Cottonwood	Yes	Pre-inoculation OTC	Reduced uredia production in leaf disk and whole-plant assay, not associated with leaf injury	Coleman *et al.*, 1987*b*
Puccinia coronata	Oats	No	Post-inoculation CC	Reduced growth of uredia	Heagle, 1970
Puccinia graminis f. sp. *tritici*	Wheat	Yes	Post-inoculation, pre-inoculation CC	Decreased hyphal growth and uredio spore production on ozone-injured leaves, no inhibition of infection on non-injured leaves after exposure	Heagle & Key, 1973*a*
Puccinia recordita f. sp. *tritici*	Wheat	No	Pre-inoculation CC	Increased number of pustules and urediospores at or past shooting stage only.	Tiedemann, 1992*b*
	Wheat	No	Pre-inoculation CC	Decreased number of rust pustules	Dohmen, 1987
Tranzschelia prun-spinosae	Peach	No	Simultaneously OTC	Enhanced spread of disease	Badiani *et al.*, 1992
Uromyces phaseoli	Bush bean	Yes	Post-inoculation CC	Increased number of pustules and inoculum, decreased pustule size	Resh & Runeckles, 1973
Powdery Mildews					
Erysiphe graminis f. sp. *hordei*	Barley	Yes	Post-inoculation CC	Reduced rate of infection if exposed during incubation; enhanced colony size when infection is established	Heagle & Strickland, 1972

Table 13. Effects of elevated concentrations of ozone on the growth and development of fungi *in vitro* and on the plant surface

Organisms	Exposed stage	Range of ozone concentrations exhibiting			Remarks on effects	References
		Inhibition	No effect	Stimulation		
In-vitro						
Alternaria oleraceae	Colony on plate	—	0·6 ppm	0·1–0·6 ppm	No growth effects, sporulation strongly stimulated	Treshow *et al.*, 1969
Alternaria solani	Filter paper	0·1 ppm/4h 1·0 ppm/2h	—	—	Conidiophores reversibly damaged	Rich & Tomlinson, 1968
Botrytis cinerea	Agar plates	0·15 ppm/ 3 × 7h	—	—	Mycelial growth reversibly inhibited	Tiedemann & Fehrmann, 1986
Colletotrichum lagenarium	Colony on plate	>0·1 ppm	—	—	Effects on radial growth	Treshow *et al.*, 1969
Helminthosporium sativum	Colony on plate	—	0·6 ppm	—	No effects on growth or sporulation	Treshow *et al.*, 1969
Heterobasidion annosum	Colony on plate	>0·05 ppm >0·1 ppm	— —	— —	Conidial production inhibited Hyphal growth and conidial germination	James *et al.*, 1982
Spore germination effects						
Chaetomium sp.	Spores on agar	—	1·0 ppm	—	Effects measured as percentage of germinated spores	Hibben & Stotzky, 1969
Stemphylium spp.	for 1 to 6 h	—	1·0 ppm	—		
Alternaria sp.	"	—	1·0 ppm			
Trichoderma viride	"	0·5–1·0 ppm	—	0·1–0·25 ppm	"	
Aspergillus terreus	"	0·5–1·0 ppm	—	0·1–0·25 ppm	"	
A. niger	"	0·5–1·0 ppm	—	—	"	
Penicillium sp	"	0·5–1·0 ppm	—	0·1–0·25 ppm	"	
Botrytis allii	"	0·5–1·0 ppm	—	—	"	
Rhizopus stolonifer	"	0·5–1·0 ppm	—	0·1–0·25 ppm	"	
Fusarium oxysporum	"	>0·25 ppm	—	—	"	
Colletotrichum lagenarium	"	>0·25 ppm	—	—	Effects measured as percentage of germinated spores	
Verticillium dahliae	"	>0·25 ppm	—	0·1–0·25 ppm	"	
V. albo-atrum	"	>0·25 ppm	—	—	"	
On the plant						
Botrytis cinerea	Poinsettia	—	0·15–0·45ppm	—	No effects on germination and infection	Manning *et al.*, 1972
	Geranium	0·15	—	—	Hyphal growth, germination and infection inhibited	Krause & Weidensaul, 1978
	Pinto bean	—	—	0·06-0·08 ppm	Increased isolation of *B. cinerea* from leaf surfaces and O_3-caused lesions	Manning, 1976
Erysiphe graminis f. sp. *hordra*	Barley	0·1–0·3 ppm 0·1–0·3 ppm	0·1–0·3 ppm	—	No effect on germination and vegetative growth, reduced infection of germ tubes	Heagle & Strickland, 1972
Microsphaera alni	Lilac	—	0·9–1·0 ppm	—	Germination of conidia not affected	Hibben & Taylor, 1975
Puccinia coronata	Oats	—	0·2 ppm	—	No effect on ability to germinate and infect the host	Heagle, 1970
P. graminis f. sp. *tritici*	Wheat	—	0·2 ppm	—		Heaglel & Key, 1973

0·6 ppm ozone for 4 h whereas this completely suppressed growth and sporulation of *Colletotrichum lindemuthianum*. In another study, ozone at 0·1 ppm for 4 h or 1·0 ppm for 2 h led to swellings or collapses of the apical cells of growing conidiophores of *Alternaria solani*. However, it is noticeable that after removal of the pollutant stress, conidiophores recovered rapidly and sporulated normally (Rich & Tomlinson, 1986).

Botrytis cinerea exposed to 0·15 ppm ozone 7 h daily for three days was significantly reduced in its mycelial growth but regained growth comparable to the control after one additional day in filtered air (Tiedemann & Fehrmann, 1986). Ozone at or above 0·05 ppm reduced conidial production of *Fomes* (*Heterobasidium*) *annosus*, whereas hyphal growth and conidial germination were affected only above 0·1 ppm (James *et al.*, 1982).

In investigating the direct effects of ozone on fungi on plants the problem is that effects due to alterations of the plant cannot be ruled out completely. Powdery mildew on barley was relatively tolerant even when exposed to high doses of ozone (0·1–0·3 ppm) for 24 h. Growth of vegetative hyphae after establishment of infections was not affected and was even slightly stimulated by exposure to ozone. Germ tubes, exposed to the same levels of ozone failed to induce infections at the same rate as in filtered air (Heagle & Strickland, 1972). Germination of conidia of the lilac powdery mildew fungus (*Microspharea alni*) was essentially not affected even by the extremely high doses of ozone of 0·9–1·0 ppm for 1–6 h (Hibben & Taylor, 1975) (Table 12).

Rust fungi on cereals have also been found to be rather insensitive to elevated doses of ozone. Both the urediospores of crown rust on oats and stem rust on wheat exposed to maximum doses of 0·2 ppm ozone were not affected in their ability to germinate or to infect non-exposed plants (Heagle, 1970; Heagle & Key, 1973*a,b*). Exposure of conidia of *Botrytis cinerea* on poinsettia plants to 0·15–0·45 ppm ozone for 4 h did not affect germination and infection by the fungus (Manning *et al.*, 1972), whereas hyphal growth, conidial germination and infection ability of the fungus were largely inhibited on geranium plants exposed to 0·15 ppm ozone for two 6-h periods (Krause & Weidensaul, 1978).

Obligate biotrophs have been extensively studied on above-ground plant parts. Experimental results suggest a picture opposite to that of necrotrophs. However as stated above, the experimental design must be considered thoroughly when judging and comparing different results. The majority of investigations were conducted with various species of rust fungi. The results vary largely, however, and this is partly explainable by differing experimental conditions. Leaf rust on wheat was enhanced on mature plants and inhibited on young plants (three-leaf stage). Inhibition of disease progress occurred also with stem rust on wheat and crown rust on oats, if the exposure happened after inoculation and/or caused injury to the leaves (Table 12). Other studies have shown that rust on bush beans was increased even if exposure took place after inoculation and there was acute injury to the leaves. In a recent study conducted in open-top chambers in Italy leaf rust on peach trees was significantly enhanced during a chronic and long-term ozone fumigation (Table 12). Powdery mildews have received less attention, and there have been no studies on the effects of ozone on downy mildews.

Manning (1976) and Rist and Lorbeer (1981) have reviewed the effects of O_3 on plant leaf surfaces and leaf surface microflora. Using washed leaf discs and leaf surface prints, Manning (1976) found increased numbers of fungi, especially *Botrytis cinerea*, associated with pinto bean leaf surfaces exposed to O_3 (0–28 days). Results obtained suggested that symptoms of O_3 in pinto bean leaves may be due to cell death caused by O_3 plus enhanced growth of microorganisms. Little is known about the microbiology of foliar O_3 injury symptoms.

Fungi, *per se*, appear to be largely tolerant of ozone levels experienced at present in most areas of the world. Significant effects were only found if growing hyphae or germ tubes were exposed and/or doses of ozone were applied which are far above realistic levels. Moreover, some examples have demonstrated the rapid recovery of fungi after exposure to ozone. It should also be considered that the growth of sensitive hyphal structures normally requires conditions of high relative air humidity, i.e. wet, cloudy or rainy days. There is a strongly negative correlation between rainfall or relative air humidity and photochemical ozone generation in the atmosphere (Guicherit & van Dop, 1977). On wet days that are appropriate for vegetative growth of fungi on plant surfaces, ozone levels are usually low. Consequently, a coincidence of biologically harmful ozone concentrations in nature and germinating spores or actively growing mycelium seems to be largely excluded. Thus, for several reasons, direct effects of ozone on fungal pathogens does not appear to be of great importance.

Table 14 summarizes the reported effects of elevated O_3 concentrations on biotic plant diseases. Bacteria as

Table 14. Summary of reported effects of elevated ozone concentrations on biotic plant diseases

Pathogen group	Total	Number of cases with acute ozone injury on leaves	Number of cases reported where diseases were		
			Enhanced	Not affected	Reduced
Bacteria	4	4	—	1	3
Necrotrophic fungi	30	14	19	7	4
Biotrophic fungi	15	7	6	2	7

Literature reviewed from 1969–93.

pathogens have not been studied extensively in O_3/plant/pathogen interactions. Most of the attention has been given to fungi, especially necrotrophic fungi. As with biotrophic fungi, variable results have been reported. We don't know enough about interactions between ambient concentrations of O_3 and pathogens to draw general conclusions.

UV-B RADIATION

Ultraviolet radiation in the wavelength 280–320 nm is called UV-B radiation. It is considered to have biological effects that are harmful. The extent of increase of UV-B radiation in the atmosphere, due to tropospheric ozone depletion, is debatable (Krupa & Kickert, 1989; Penkett, 1989; Blumthaler & Ambach, 1990; Seckmeyer & McKenzie, 1992; Kerr & McElroy, 1993; Runeckles & Krupa, 1994). Quaite and coworkers (1992) have also predicted that increases in UV-B radiation will have smaller biological effects, using an absolute action spectrum for DNA damage due to UV-B to intact alfalfa seedlings. More seems to be unknown than known about possible effects of increases of UV-B in the atmosphere.

Numerous experiments have been conducted on the effects of increased UV-B on crops and natural vegetation (Teramura, 1983; Krupa & Kickert, 1989; Tevini & Teramura, 1989; Runeckles & Krupa, 1994). There is also a considerable literature on the effects of UV-light on fungal spore germination, hyphal growth and sporulation (Ensminger, 1993). There are only a few reports, however, on the potential effects of UV-B on the occurrence and severity of plant diseases.

Photobiological studies with fungi go back to the early thirties (Dillon-Weston, 1931). In those earlier studies the light quality was rarely reported precisely in wavelengths, but only as light colors and generally, the specific UV-B band of the spectrum was not specifically tested. More precise studies on light effects on fungi growing in-vitro began in the sixties. The main objective of those investigations was the improvement of identification methods for seedborne fungi, or to induce sporulation of fungi.

The great significance of light, especially in the near-ultraviolet band (NUV), for fungal sporulation was recognized very early and confirmed in numerous studies, of which only the most important ones will be reviewed here. It is important to be aware of the fact that all these 'near UV-light studies' have examined only the effects of wavelengths mainly between 310 and 400 nm, the longer wavelength part of the UV-B spectrum. Most studies indicate, however, that main effects on sporulation were due to wavelengths below 350 nm.

Leach (1962) exposed agar cultures of 34 species of mainly phytopathogenic fungi, such as *Ascochyta pisi, Botrytis cinerea, Fusarium oxysporum, Ophiobolus graminis, Phoma* spp. or *Verticillium albo-atrum* to near-ultraviolet (NUV) light (320–400 nm) and measured effects on sporulation. Some species like *Helminthosporium oryzae* required a period of darkness after NUV irradiation to complete the formation of mature spores. *H. sativum* and *Kabatiella caulivora* sporulated as well in darkness as under NUV irradiation. In a further study 'direct sporulators' were distinguishable from 'constant temperature sporulators'. The former (*Alternaria dauci, A. tomato, Stemphylium botryosum*) had two distinct phases of photosporogenesis, a phase of NUV-light induced formation of conidiophores followed by a terminal phase of sporulation which was strongly inhibited by NUV and blue light and required darkness. Species belonging to the latter category such as *Fusarium nivale, Cercosporella herpotrichoides* and *Helminthosporium catenarium* also formed mature spores when exposed under continuous NUV light (Leach, 1967).

A strong increase in sporulation was also found in cultures of *Alternaria tomato* following induction by monochromatic light with wavelength below 340 nm. Blue light between 390 and 515 nm, however, completely inhibited sporulation (Aragaki, 1962). Such opposing effects of inductive UV radiation and inhibitory blue light on fungal sporulation has been demonstrated in numerous investigations (Honda *et al.*, 1968). Under white light the inhibitory effect of blue light may nullify the inductive action of UV-light, as has been found for UV-dependent fungi like *Alternatia dauci, A. porri* and *A. solani*, which did not sporulate under continuous white light, though it contained UV-light. In other fungi such as *Alternaria brassicae, Botrytis squamosa*, and *Stemphylium botryosum* sporulation was not affected by blue light if total illumination included the NUV spectrum. Those fungi equally sporulated under white and NUV light (Sasaki & Honda, 1985).

Induction of sporulation by NUV light (< 340 nm) and inhibition by blue light (360–530 nm) has also been described more recently for *Alternaria cichorii* (Vakalounakis & Christias, 1981) and *A. tomato* (Kumagai, 1982). A marked induction of sporulation was also demonstrated for *Botrytis cinerea* which produced a maximum of conidia when exposed to light with a lower wavelength cutoff between 305 and 240 nm (Hite, 1973).

In a more comprehensive investigation conducted by Schlosser (1970), the inductive effect of NUV light on sporulation was tested on 75 different species belonging to the ascomycetes and fungi imperfecti. Daily 9-h light exposures stimulated sporulation in 62 species, most of them obligately needed light for the initiation of spore formation and some also sporulated in the dark, however, at a markedly lower extent. Thus, in the large majority of isolates examined, intermittent exposure to NUV-light either initiated or at least increased sporulation. In none of the tested 417 fungal strains any inhibition of sporulation by light exposure was detected.

Normal daylight contains NUV wavelengths in its spectrum and it has been demonstrated that photosporogenetic effects of daylight are roughly equal to the pure NUV effects, unless inhibitory effects of blue light occur, and may be offset if the near-UV band is filtered out (Leach, 1962, 1971). Evidently, fungi use intense

Table 15. Effects of UV-B radiation on biotic plant diseases

Pathogen	Host plant	Exposure systems	Range of wavelengths	Effects of UV-B on diseases	References
Potato virus S (PVS)	*Chenopodium quinoa*	Filtered sunlamps, greenhouse	290–320 nm, 6 h	Partial or complete virus inactivation	Semeniuk & Goth, 1980
Botrytis cinerea	Cucumber, tomato	UV-absorbing vinyl films compared with UV-transmissive films	300–390 nm, sunlight continuously during daytime	Strong increase in number of diseased tomato and cucumber fruits under UV-transmissive vinyl films compared to UV-absorbing films	Honda *et al.*, 1977
Sclerotinia sclerotiorum	Eggplant, cucumber	"	"	Strong increase of apothecia formation and of blighting under UV-transmissive films	Honda & Yunoki, 1977
Alternaria dauci, *A. porri, A. solani.* *Botrytis squamosa*	Certain greenhouse vegetables	" " "	" " "	Significantly enhanced disease development under the UV-transmissive films	Sasaki & Honda, 1985
Colletotrichum lagenarium	Cucumber	Filtered sunlamps	280–320 nm	Decrease of percentage leaf area infected	Carns *et al.*, 1978 (in Ovens & Krizek, 1980)
Cladosporium sp. *Alternaria* sp.	Tomato	"	"	No effect on disease severity	
Diplocarpon rosae	Roses	Detached leaflets in large petri dishes growth room	280–320 nm, 6 h	Decrease of infection; no effects on infectivity of exposed spores; high sensitivity of germ tubes	Semeniuk & Stewart, 1981
Puccinia coronata *Uromyces phaseoli* *Erysiphe graminis*	Oats Beans Wheat	Irradiated growth chamber	286–320 nm	Decrease of disease severity	Esser, 1980 (in Orth *et al.*, 1990)
Colletotrichum lagenarium. *Cladosporium cucumerinum*	Cucumber	Filtered sunlamps, greenhouse	280–320 nm, 7h/d, 1–12 days	Increased severity of both diseases by preinfectional UV-B treatments	Orth *et al.*, 1990
Cercospora beticola	Sugar beets	Filtered lamps, chamber	280–320 nm, 4 h/d, 40 days	More-than-additive damaging effects of UV-B exposure and infection	Panagopoulos *et al.*, 1992
Puccinia recondita f. sp. *tritici*	Wheat	Filtered sunlamps	280–320 nm	Rust infections increased on rust-sensitive wheat cultivar.	Biggs *et al.*, 1984

Major plant responses to enhanced UV-B radiation	Effects on disease incidence and epidemiology
Morphological	
Stunted growth Increased branching Increased size of leaves	Improved microclimate conditions for bacterial or fungal infections, and for the buildup of epidemics
Physiological	
Reduced net photosynthesis	Reduction of "High-sugar-disease" (rusts, mildews)
Increased production of photoactive phenolics (flavonoids)	Potential of antibiotic action against pathogens
Premature ripening and senescence	Shortened infection period for biotrophs and prolonged infection period for necrotrophs
Increased contents of soluble proteins, decreased contents of membrane lipids	Improved availability of host nutrients, enhanced growth of necrotrophic pathogens

Fig. 3. Potential impacts of major alterations of plant morphology and physiology induced by enhanced UV-B radiation on biotic plant diseases.

short-wave light as an alarm signal to initiate reproduction. In an ecophysiological sense this seems to be highly reasonable as NUV irradiance is almost consistently associated with dry and thus inconvenient conditions for a continuous vegetative growth. On the other hand, light in the NUV band obviously may be directly harmful if sensitive fungal stages are exposed, such as germinating conidia (Owens & Krizek, 1980); uredospores (Maddison & Manners, 1972, 1973) or thin-walled and non-pigmented ascospores (Caesar & Pearson, 1983). Thus, the fungus protects itself both from drying out and from being damaged by irradiation, when it sporulates readily in the presence of NUV light. Newly-formed spores, especially when they contain melanin pigments in their outer cell wall layers, generally provide an efficient means for fungal survival from adverse conditions associated with intense light influx (Bell & Wheeler, 1986).

Considering the stimulating effects of near-UV-light on fungal reproduction, investigations in the seventies, mainly in Japan, were initiated to evaluate the usefulness of UV-absorbing plastic films as covers of greenhouses to reduce the disease potential of certain foliar pathogens of vegetables. This research now provides one of the most important data bases for evaluating the potential effects of increased UV-B radiation on plant diseases. Those studies in greenhouses (Table 15) compared disease development under UV-absorbing vinyl films (lower cutoff at 390 nm) with that under a standard acetate film which transmitted light down to a wavelength of 300 nm. In all those studies, the use of UV-absorbing films consistently reduced the severity of diseases caused on several vegetable crops by *Sclerotinia sclerotiorum*, *Botrytis cinerea*, *B. squamosa*, or species of *Alternaria*. The high effectiveness of UV-absorbing films in disease reduction, which generally attained 90%, led to suggestions of using such films in commercial greenhouses as a measure of integrated disease control (Honda *et al.*, 1977; Honda & Yunoki, 1977; Sasaki & Honda, 1985). The main effect of filtering out the UV-A/B wavelengths was a marked reduction of sporulation, which in turn decreased the potential of secondary infections.

Increases in disease severity due to enhanced UV-B radiation were found on cucumber inoculated with *Colletotrichum lagenarium* (anthracnose) or *Cladosporum cucumerinum* (scab). However, it is noticeable that predisposition for both diseases only occurred following a preinfectional light treatment of the plants and not, if the exposure happened just after inoculation (Orth *et al.*, 1990). This is the only study which clearly demonstrates UV-B effects on the susceptibility of the host plant, whereas in all the other studies light exposure was continued during infection and disease development, and thus they do not allow differention between the effects on the plant and those on the pathogen (Fig. 3).

On roses, a four-fold increase of UV-B radiation between 6 and 18 h post-inoculation reduced the severity of blackspot disease caused by *Diplocarpon rosae*, which was mainly attributed to harmful effects on the conidium germ tubes (Semeniuk & Stewart, 1981). In a recent study, damaging effects of UV-B treatment and inoculation with *Cercospora beticola* on plant dry weight and chlorophyll content were more-than-additive on sugar beets (Panagopoulus *et al.*, 1992).

Biggs and coworkers (1984) considered the effects of cultivar resistance, days after planting, and simulated percentage ozone reduction (and hence increasing incidence of UV-B) on wheat rust (*Puccinia recondita* f. sp. *tritici*) incidence. Increasing UV-B incidence had little effect on rust incidence for leaves of the resistant cultivar (Florida 301), but resulted in increases in rust infections on leaves of the rust-susceptible cultivar 'Red Hart'.

Our literature survey has shown that enhanced UV-B radiation may increase or decrease severity of biotic diseases, or have no effect. A serious comparison of such contradictory results, however, is not possible. The ranges of light qualities, light intensities and exposures used, were too large and too variable as were the experimental designs and time courses applied to really combine the available data into a general conclusion on the mode of action of UV-B light. Nevertheless,

Table 16. Summary of reported effects of increasing UV-B radiation on biotic plant diseases

Pathogen group	Total number of diseases reported	Number of cases reported where diseases were		
		Enhanced	Not affected	Reduced
Necrotic fungi	13	9	2	2
Biotrophic fungi	4	1	—	3

Literature reviewed from 1970–93.

numerous in-vitro studies undoubtedly have shown that the majority of fungi use or require UV-light as a stimulant for spore formation. Studies in Japan with UV-absorbing films additionally have demonstrated that this effect does have significant consequences for the extent of disease development, since it strongly influences the inoculum production on infected plants. Thus, one might suggest, that an increase in solar UV-B radiation would be likely to promote sporulation of pathogenic fungi in a way, which substantially could enhance the buildup of epidemics. However, it is important to consider that normal daylight already contains a sufficient UV-portion to stimulate sporulation of light dependent fungi. Provided that this stimulation is mainly a qualitative effect of specific wavelengths, a slight quantitative increase in that narrow wavelength band, as is expected due to a proceeding depletion of the ozone layer, is not likely to substantially influence the life cycle of pathogenic fungi in the field. This would only occur if a notable shifting in the wavelength spectrum of the sunlight takes place, adding completely new wavelength bands with strongly inductive properties, but this is not expected.

Enhanced UV-B radiation requires clear skies and will be only biologically efficient when it reaches the target organism directly. Fungi, however, preferentially grow when the sky is cloudy and thereby are mainly active on shaded parts of the plant or in non-irradiated angles of the ecosystem. Pathogenic fungi in particular are additionally protected while growing partly or completely within the host tissue which may efficiently filter harmful UV-radiation. The phylloplane and phyllosphere mycoflora, however, would have less protection.

Like CO_2 and O_3, the principal effects of increased UV-B on plant diseases would be via alterations in host plants. Major morphological alterations for virtually every plant species consist of stunted growth, increases in branching and number of leaves and reduced leaf size (Teramura, 1983; Krupa & Kickert, 1989; Tevini & Teramura, 1989; Barnes *et al.*, 1990; Runeckles & Krupa, 1994) (Table 15).

Physiological effects were not consistent for all plant species. In general, enhanced UV-B reduced net photosynthesis, induced the production of flavonoids, accelerated ripening and reproduction (Ziska *et al.*, 1992), increased leaf soluble proteins and decreased membrane lipids (Teramura, 1983). Flavonoids evidently play an important role as inducible protectants of plants growing under high UV-irradiation (Caldwell *et al.*, 1983; Tevini *et al.*, 1991; Gislefoss *et al.*, 1992). Flavonoids are a rather diverse group of secondary compounds which occur in many plant species and may exert growth inhibitory effects in plants. They may affect auxin synthesis and thus reduce plant height. Though a direct relation to resistance against pathogens has not been described, it cannot be excluded that a UV-B induced production of phenolic compounds would give rise also to the production of plant metabolites with an antibiotic activity against pathogens. This could enhance the resistance of the plant to biotic diseases. The potential consequences of major physiological effects of increased UV-B radiation on biotic diseases are listed in Fig. 3.

We know little about increased UV-B effects on biotic plant diseases (Table 16). There are no reports on interactions with bacteria. Most attention (as with O_3), has been paid to potential interactions with necrotrophic fungi. In most, but not all, cases disease was enhanced. It is not possible to conclude much about trends from the few studies reported.

MECHANISMS THAT MIGHT EXPLAIN THE EFFECTS OF CO_2, O_3 AND UV-B RADIATION ON THE OCCURRENCE OF PLANT DISEASES

We know a great deal about the individual effects of CO_2, O_3 and to a lesser extent UV-B on plants. Ozone is known to affect the incidence and course of plant diseases. Much less is known about the effects of CO_2 and UV-B. The mechanisms of how diseases are enhanced or inhibited by these climate change factors, however, remain largely unexplained. Here we consider nine ways in which observed alterations in plant/pathogen interactions could be accomplished.

Induction of necrotic lesions

Studies of field-grown onions (Wukash & Hofstra, 1977; Rist & Lorbeer, 1984*b*) and potatoes (Manning *et al.*, 1969*b*; Holley *et al.*, 1985) have shown that elevated ozone levels may cause acute injury on leaves and that this injury may be used by fungal pathogens *Botrytis cinerea* and *Alternaria solani* to invade plant leaves.

Acute O_3 injury favors infection by certain necrotrophic or weak pathogens. Cultivars and species of plants that are quite sensitive to O_3 will experience both chronic and acute O_3 injury. At present, most other plants will suffer at most only chronic injury. As O_3 concentrations increase, however, necrotic lesions may become more common on many plant cultivars and species thus increasing the importance of acute injury as an infection court.

Changes in cuticle integrity

Changes in cuticular wax structure have been shown mainly on different tree species exposed to elevated ozone concentrations (Fink, 1988). Experiments with beeches and Norway spruce have indicated that notable changes in the physical properties of cuticles occur only if ozone concentrations exceed ambient. At lower O_3 concentrations, there were no changes in permeability for water (Kerstiens & Lendzian, 1989; Barnes & Brown, 1990). Slight reductions of wax production rates, but no significant changes of thickness of the cuticular wax layer, were found on red spruce, after exposure to moderate ozone concentrations (Percy *et al.*, 1992).

At present, our knowledge is too limited to estimate the real significance of cuticle alterations for recognition and infection mechanisms of plant pathogens. It does not appear likely that slight changes in the structure and amount of epicuticular waxes would greatly influence the occurrence or dissemination of pathogens.

Enhancement of membrane permeability and leakage

The result of exposure to ozone generally is an enhancement of membrane permeability and thus of leakage (Evans & Ting, 1973; Heath & Castillo, 1988). The enhanced loss and efflux of soluble substances from plant tissues essentially alters growth conditions for leaf pathogens and phyllopane microorganisms. Leakage of ions and carbohydrates was measured on onion leaves subjected to elevated ozone stress. This promoted the growth and infectivity of *Botrytis cinerea* and increased the severity of infections (Rist & Lorbeer, 1984*a*).

Cytological studies with *Septoria nodorum* on wheat fumigated with ozone, demonstrated an accelerated growth of the fungus when compared with plants grown in filtered air (Tiedemann & Firsching, 1992). Wheat leaves exposed to ozone, but without visible injury, had increased permeability for soluble carbohydrates, proteins, amino acids and inorganic salts. Leaking fractions from ozone-treated wheat leaves stimulated the germination of conidia of *S. nodorum* (Tiedemann & Phahler, 1994).

Control of membrane permeability is a basic requirement for a normal working plant. Bacterial and fungal pathogens have developed specific mechanisms to disturb the membrane function of their host plants in order to improve their opportunities to cause disease. The main tools they use are toxins which specifically induce leakage of infected plant tissue. Atmospheric factors like ozone or UV-B radiation may act in the same way, with membranes already altered before visible injury occurs. Therefore, it is likely, that latent changes in the membrane status of plants is among the significant mechanisms of global O_3 and UV-B impacts on plant diseases.

Induction of antibiotic substances

Induction of antibiotic metabolites in plants by abiotic stresses has long been known. Ozone may change the activity of phenylalanine ammonilyase (PAL) and thus the status and productivity of the whole phenylpropanoid pathway. The products of this pathway include compounds like flavorioids, isoflavonoids, coumarios, stilbenes, lignins and other phenols. All of these compounds can be involved in disease resistance (Ebel, 1986).

Flavonoid synthesis was enhanced by ozone exposure of several plant species such as soybeans (Keen & Taylor, 1975), alfalfa (Hurwitz *et al.*, 1979), and snap bean (Rubin *et al.*, 1983). Stilbene synthesis was markedly enhanced in ozone-exposed pine seedlings leading to strong increase of pinosylvin contents of primary needles (Rosemann *et al.*, 1991). Similar effects are known with UV-light, which induced the production of resveratrol, precursor of the viniferins, in grapevines (Langcake & Pryce, 1977) and hydrovyphaseollin in soybeans (Bridge & Klarman, 1972). Recent research has revealed the protective role of flavonoids in plants against harmful UV-B irradiation and the inductivity of such phenols by UV-light has been demonstrated as well (Caldwell *et al.*, 1983; Tevini *et al.*, 1991; Gislefoss *et al.*, 1992).

There are many indications of significant alterations of secondary metabolites, mainly of phenolic nature, in plants exposed to enhanced ozone or UV-light stresses. This undoubtedly may substantially affect the resistance of plants to pathogens. There are no clear reports which prove this kind of linkage between these atmospheric factors and the severity of biotic diseases. The role of changes of secondary metabolism of plants in a changing environment in relation to disease cannot be determined unless further research is conducted.

Changes in surface microfloras

The direct effects of CO_2, O_3 and UV-B on leaf microflora have not been extensively investigated (Rist & Lorbeer, 1981; Manning, 1976). Qualitative changes were noted in the surface fungi of pinto bean leaves exposed to O_3 in relation to length of exposure. *Botrytis cinerea* isolations increased in frequency when visible injury occurred (Manning, 1976). There were, however, no notable qualitative or quantitative changes in the phyllosphere mycoflora of wheat (Tiedemann *et al.*, 1991) or several tree species (Fenn *et al.*, 1989), when the plants were exposed to concentrations of ozone that did not cause visible foliar injury.

We know very little about the indirect effects of CO_2, O_3 or UV-B on roots, especially under natural conditions (Colls & Unsworth, 1992), let alone much about the indirect effects of pollutants on root surface microfloras. Manning and coworkers (1971*b*) studied fungal colonization of pinto bean roots in relation to O_3 exposure and plant age. Ozone exposure accelerated colonization of normal senescence fungi, such as *Fusarium oxysporum*.

Alteration of stomatal function

Effects of O_3 on stomatal reactions have been described, but the results are quite contradictory. Both

enhancement and inhibition of the stomatal aperture were found (Winner *et al.*, 1988). Stomatal function is strongly influenced by CO_2. Elevated CO_2 will decrease or close stomatal apertures (Rogers *et al.*, 1994).

Weidensaul and Darling (1979) supposed that infections on pines by needle pathogens were inhibited through an ozone-induced closure of the stomata. However, the investigators did not directly prove this assumption. The question remains open, if a closed stoma really would hinder the invasion of a pathogen which is specialized in this way of infection (e.g. rust fungi). Moreover, the stomatal hypothesis would only explain alterations in the behavior of stoma-invading parasites, but not for the many pathogens penetrating the cuticle. Plants are also dependent on the gas exchange performed through the stomata. Thus, the stomatal mechanism apparently plays a minor role in the interaction between the atmosphere and biotic diseases.

Changes in canopy structure

Both CO_2, O_3 and radiation in the UV wavelength band influence the biomass production of crop plants. Carbon dioxide and UV-B radiation may cause a denser canopy structure since more biomass would be accumulated per unit area (CO_2) and the more would be produced by plants reduced in height (UV-B). Consequently, microclimate in the canopy will potentially be moister and provide improved conditions for disease development and spread of pathogens. Similar secondary effects on the occurrence of plant diseases are known from fertilizer experiments, where nitrogen may have comparable effects on biomass accumulation. Thus, an expected more vigorous growth due to elevated carbon dioxide level will certainly aggravate problems with pathogens. Growth reduction by ozone and UV-B radiation will partly or completely counteract this effect. The overall effect of all three factors will depend on the relative importance of each of them, which is not exactly predictable at present. Model experiments could help to better estimate the total effects of the factor combination, not only in terms of plant growth but also in relation to disease susceptibility.

Changes in carbon allocation

Ozone is known to change carbon allocation in plants (McCool & Menge, 1983; Cooley & Manning, 1987; Runeckles & Krupa, 1994). Repair and compensatory growth by leaves and shoots can shift allocation of carbohydrates away from root sinks. This can adversely affect endo- and ectomycorrhizae (Andersen & Rygiewicz, 1991) and result in a deterioration of root health and invasion by root pathogens (James *et al.*, 1980). Increased UV-B would probably have the same effects as elevated O_3 concentrations.

Elevated CO_2 levels would increase root biomass (Rogers *et al.*, 1994) and presumably increase mycorrhizae and decrease root disease incidence. Increased root biomass would also allow more root area loss from root disease pathogens, without significant effects on plant growth and yield.

Induction of premature senescence

Plant resistance to pathogens is largely determined by their growth stages. Disease resistance changes markedly as plants mature and begin to senesce. Mature plants are often more resistant to rust and mildew diseases and more sensitive to necrotrophic pathogens. Latent infections also develop into full disease syndromes as plants age and mature.

Ozone and UV-B are known to promote maturity and senescence in plants (Runeckles & Krupa, 1994). Long-term exposure can reduce the length of plant life cycles and change their sensitivity to pathogens. This would be important for necrotrophic pathogens and latent infections. Carbon dioxide, at elevated concentrations, promotes biomass production by C3 plants. This too is dependent on plant growth stages and does not continue indefinitely (Baker & Allen, 1994). Elevated CO_2 may delay plant maturity and senescence and increase susceptibility to biotrophic pathogens.

CONCLUSIONS

Elevated concentrations of CO_2, O_3 and increased incidence of UV-B radiation may have direct effects on plant pathogens, such as survival, growth rate, and

PLANTS		PATHOGENS	
CO_2	considerable stimulation of growth and biomass accumulation by C3 plants	CO_2	significant effects mainly above 2–5%
O_3	considerable effects on photosynthesis, carbon allocation, secondary metabolism and yield	O_3	significant effects only at conc. above 100–250 ppb; coincidence with sensitive stages low
UV-B	considerable effects on shoot morphology and leaf area	UV-B	significant effects on sporulation, but quantitive effects of slight increases of normal daylight level not proved; coincidence with sensitive stages low
	Sensitivity relatively high		Sensitivity relatively low
	Adaptability medium (UV-B, CO_2) to low (O_3)		Adaptability generally high

Fig. 4. Comparative effects of CO_2, O_3 and UV-B on plants and pathogens.

reproduction. These effects are summarized in Fig. 4. It appears unlikely, however, that pathogens, *per se*, will be significantly directly affected by present or future levels of CO_2, O_3 and UV-B or in relation to changes in disease incidence. The rationale for this generalization can be summarized as follows:

1. The two non-continuously acting factors ozone and UV-B radiation are more or less completely non-coincident with infection periods of most pathogens. The sensitivity of microorganisms to increases of both factors within the present or expected range can be considered low.
2. The continuously acting carbon dioxide level is coincident with all stages of the pathogen's life cycle, however, it is very unlikely to exert any substantial effects, neither adverse nor favorable, on pathogens within the range of an expected doubling of the present atmospheric partial pressure.

From this we conclude that microbial plant pathogens are less likely to be adversely affected by increased CO_2, O_3 and UV-B than are their corresponding host plants and hence changes of host plants mainly trigger any expectable alteration of disease incidence.

The literature clearly shows that sensitive higher plants are directly and indirectly affected by CO_2, O_3 and UV-B. These effects are summarized in Fig. 4. All of the effects listed there will have some effect on the incidence and severity of plant diseases caused by biotic pathogens. Effects of enhanced UV-B radiation on (altered) disease occurrence and changes in disease epidemiology are summarized in Fig. 3. We know very little about the actual impacts of climate change factors on disease epidemiology. Epidemiologists should be encouraged to consider CO_2, O_3 and UV-B as factors in their work, especially with grain and forage crops.

Based on what we know about the individual effects of CO_2, O_3 and UV-B on plants, we can differentiate between them on the basis of growth promotion or restriction and reduction. In summary, we can propose two opposing scenarios for occurrence and incidence of plant diseases, with one based on growth stimulation by elevated CO_2 and the other on adverse effects of O_3 and UV-B.

Scenario one: Plant growth enhancement by CO_2 (Fig. 5)
Photosynthetic rates enhanced by increasing CO_2 will lead to a greater availability of assimilates. This will undoubtedly favor all sugar-dependent parasites like rusts, mildews, and also aphids vectors. These effects have been already demonstrated by data from Gassner and Straib (1930) and others. Enhanced crop growth will accumulate more biomass in the field. In the standing crop this may considerably change the microclimate and thus favor the epidemic spread of all diseases. Increasing amounts of crop residues after harvest will enlarge the inoculum of necrotrophs or bacteria, which remain in the field for the next season. On the whole, a CO_2 induced increase of plant productivity will probably in general promote the incidence and severity of plant diseases due to improved host qualities and plant cover.

Scenario two: Plant growth reduction and restriction by O_3 and UV-B (Fig. 6)
In case of factors reducing and restricting plant growth there are quite different reactions. Oxidative stress may induce resistance some specific fungal pathogens, e.g. UV-B induced synthesis of flavonoids or induction of other secondary metabolites may play an inhibitive role. Less available assimilates will certainly reduce powdery mildews and aphid vectors. Effects on microclimate and amount of crop residues will not be in the order of magnitude as expected for CO_2. Premature senescence, however, could play a significant role. Several studies have demonstrated that this may promote necrotrophs and reduce biotrophic pathogens like powdery mildews.

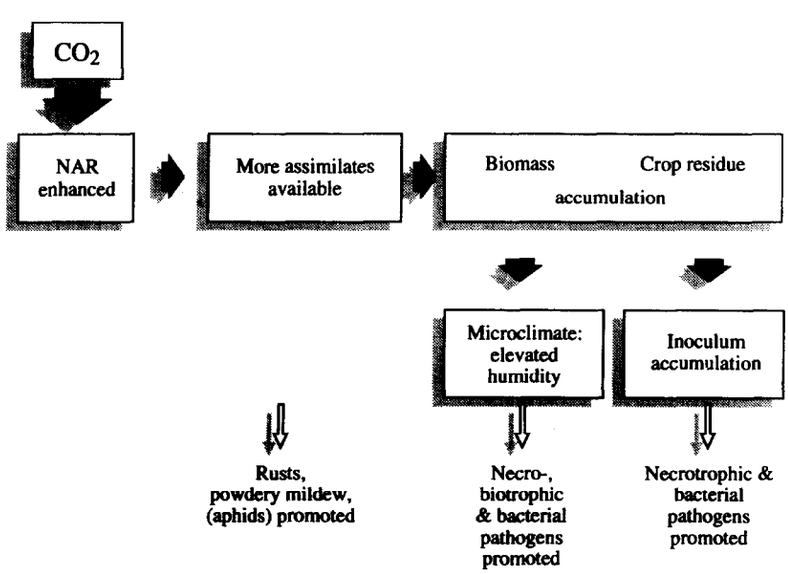

Fig. 5. Major effects of environmental changes on disease susceptibility: Plant growth enhancement.

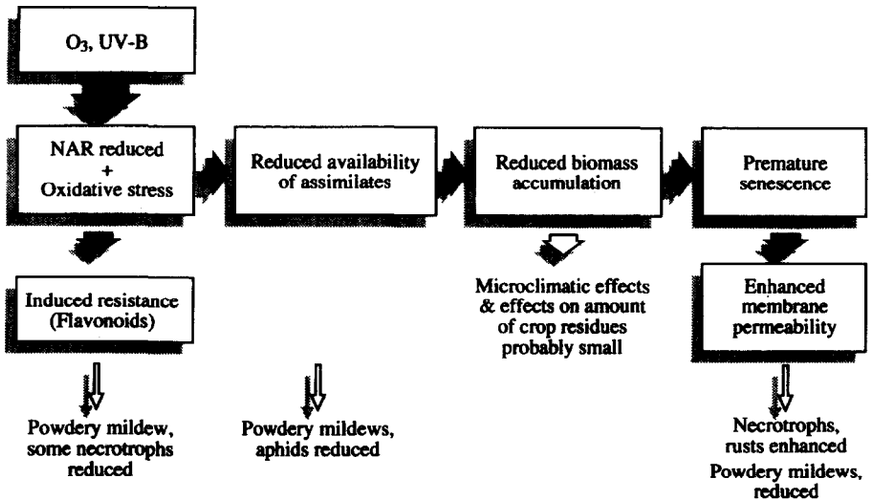

Fig. 6. Major effects of environmental changes on disease susceptibility: Plant growth restriction

A major factor hampering reliable prediction of the effects of increases of CO_2, O_3 and UV-B on plants and plant disease incidence is the lack of information on the interactive effects of these factors and the conditioning effects of present and possible future changes in temperature and soil and atmospheric moisture. Runeckles and Krupa (1994) have summarized what little is known about these interactions. The importance of interactions among CO_2, O_3 and UV-B in relation to plant responses and disease incidence are largely unknown (Allen, 1990).

Predictions have been made that the climate of the earth is changing and will continue to change. It is evident from the literature that CO_2, O_3 and UV-B alone can significantly affect the incidences and severity of plant diseases caused by biotic pathogens. Given the importance of plant diseases in world food and fiber production, it is essential to begin studying the interactive effects of increased CO_2, O_3 and UV-B (and other climate change factors) on plant diseases.

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