Commentary

Climate change and plant pathosystems – future disease prevention starts here

The concentration of carbon dioxide (CO_2) in the atmosphere is increasing (Keeling *et al.*, 1989) and may double during this century (Bolin, 1986). The opportunities available for those interested in the study of plant diseases within the emerging field of global change science were noted over a decade ago (Bruck & Shafer, 1991). However, the manner in which increasing levels of atmospheric CO_2 will affect crop diseases remains virtually unstudied. One group which has taken up this challenge and has begun to address the effects of climate change and elevated atmospheric CO_2 on plant diseases is Sukumar Chakraborty and colleagues in Queensland, Australia. In this issue of *New Phytologist* (pp. 733–742), Chakraborty & Datta present results from an indepth investigation of the effects of elevated atmospheric CO_2 on a crop, *Stylosanthes scabra*, to one of its major diseases (anthracnose, caused by the fungus *Colletotrichum gloeosporioides*).

'Climate change will directly impact crops, as well as their interactions with microbial pests'

A glance at the past

Humans have been plagued by the effects of plant diseases since they first adopted an agrarian lifestyle, with the first causes ascertained and proven by early scientists such as Tillet, Prevost, and de Bary (Ainsworth, 1981; Agrios, 1988). We now know that numerous types of microorganisms (primarily fungi, bacteria, nematodes, and viruses) are the causal agents of diseases of both humans and plants. And we have seen how plant disease has drastically altered history (e.g. emigration from Ireland in the mid 1800s resulting from the potato famine, due to the fungus *Phytophthora infestans*, the causal agent of late blight of potato). We have also seen disease alter the ecological landscape of a country (e.g. chestnut blight, caused by the fungus *Cryphonectria* (*Endothia*) *parasitica*). History tells us that we can expect plant disease to have devastating effects in the future.

Plant diseases continue to destroy crops and reduce agronomic productivity – each year billions of dollars in yield are lost to diseases and millions more are spent managing these pests (Agrios, 1988). However, we now understand many of the underlying principles surrounding plant diseases, their epidemiology, and management (Fry, 1982). We know that differing disease-causing organisms require differing environmental conditions for pathogenesis. We have mapped the minimum, maximum, and optimum temperatures and moisture conditions for many pathogenic microbes. We have also devised a variety of means for managing these organisms to reduce their impacts, including integration of cultural, chemical and biological control strategies. We have observed for many years that some plants remain disease-free, while their neighbors become infected or die. These early observations have led to strategies for breeding crops which are resistant to infection and, with new advances in biotechnological methodologies (Bent, 2003), host plant genetics remains a primary weapon in our arsenal against plant disease.

Changing climate, changing research priorities

We cannot continue to rely on what we know now, as our current global environment is changing. Increases in atmospheric concentrations of greenhouse gases has brought about concern for rising temperatures, altered precipitation patterns, as well as numerous other potential changes in our global climate (Norby *et al.*, 2001; Paul, 2001). Climate change will directly impact crops, as well as their interactions with microbial pests (Rosenzweig *et al.*, 2000). While we can use current knowledge to predict how climatic changes might affect crop productivity and interactions of crop plants with disease-causing organisms, few data are available to validate such speculations. This fact remains as true for the known increase in atmospheric CO_2 concentration as it does for potential increases in temperature or altered precipitation patterns.

It is well established that elevated CO_2 increases growth and yield of most plant species (Kimball, 1983) and that this increase is generally caused by increased rates of photosynthesis (Amthor, 1995) and/or increased water use efficiency (Rogers & Dahlman, 1993). CO_2-induced changes in plant morphology, physiology, and biochemistry have the potential to effect the major diseases of the world's food and fiber crops. Further, as with aspects of climate change, it has been

suggested that generalities regarding effects of CO_2 on host–pathogen interactions can be theorized using knowledge of plant responses to elevated CO_2 and of ecophysiological differences among pathosystems (Runion *et al.*, 1994). However, the manner in which increasing levels of atmospheric CO_2 will affect crop diseases is only just beginning to be investigated.

What effects can elevated CO_2 have on pathosystems?

In this issue, Chakraborty & Datta present results from a study on the regionally important pasture legume *Stylosanthes scabra*. They investigated the effects of ambient and twice-ambient levels of atmospheric CO_2 on changes in aggressiveness, fecundity, and genotype of the fungal pathogen *Colletotrichum gloeosporioides* when grown for 25 successive infection cycles on host plant cultivars varying in genetic resistance to the disease. The findings are relevant not only to the genetics of an important host–pathogen interaction, but also to the epidemiology of this pathosystem. First, they demonstrate (using pathogen isolates collected from the field over the past 22 years) that, under field conditions, aggressiveness has increased towards the resistant, but not the susceptible, cultivar. It is interesting to note that the authors' use of the term aggressiveness – which they define as a property of the fungus reflecting the relative amount of damage caused to the host without regard to resistance genes – is synonymous with disease severity. Nonetheless, it seems logical that aggressiveness would not be altered on the susceptible cultivar as it can be readily infected, placing little or no pressure on the pathogen for adaptation to survive and reproduce. However, disease severity (and presumably inoculum production) was substantially lower on the resistant cultivar, 'forcing' the fungus to adapt by selecting for races which can infect this resistant host. The proportion of the overall population comprised by these races likely increased over time and resulted in increased severity.

The authors noted that aggressiveness increased over the course of the infection cycles on both cultivars when grown under ambient CO_2. They also note that this study is the first to document a change in pathogen aggressiveness when inoculated onto host plants and grown under elevated CO_2 – overall aggressiveness of both isolates was reduced on both resistant and susceptible cultivars. This suggests that host plants may benefit from future, higher atmospheric CO_2 concentrations through a reduction in damage from this pathogen. However, the overall reduction in pathogen aggressiveness resulted from an initial lag phase of 10 infection cycles, after which aggressiveness increased on both cultivars. Presumably, during this initial period the pathogen was adapting to whatever CO_2-induced changes in the host led to the initial decrease in aggressiveness, after which aggressiveness increased in a manner similar to that which occurred on plants grown under ambient CO_2. It has been suggested that an increase in production of defensive compounds and/or other changes in host physiology, morphology, or anatomy under elevated CO_2 could lead to reductions in incidence or severity, at least for some pathosystems (Runion *et al.*, 1994; Hibberd *et al.*, 1996; Chakraborty *et al.*, 2000; Hartley *et al.*, 2000). By carrying this pathosystem through numerous infection cycles, the authors correctly note that enhanced resistance at elevated CO_2 may not result in reduced host damage in the long term.

Perhaps one of the most important observations reported in this study, nevertheless following an earlier, similar finding using the same pathosystem (Chakraborty *et al.*, 2000), was an increase in fecundity (spores produced/lesion area) under elevated CO_2. This increase was noted for both isolates but was more consistent and pronounced for the more aggressive of the two. Spore production has critical implications for the epidemiology of any disease – an increase in spore numbers implies increased inoculum pressure for subsequent infection cycles and, generally, an increase in the spread and severity of disease. Although, through the inoculation methods used, the authors ignored the effects of fecundity on aggressiveness and suggest the increased fecundity was a result of a better canopy microclimate from larger plants under high CO_2, they nonetheless note that the high reproductive fitness of the more aggressive isolate is an important component of its high level of aggressiveness. They further note that increased fecundity under elevated CO_2 could have important implications in the functional duration of resistance in crop plants.

Interestingly, while genotypic alterations occurred in both *C. gloeosporioides* isolates on the susceptible cultivar at twice-ambient CO_2, they were not related to increased aggressiveness of the fungus. The authors duly note that: there are known mechanisms of genetic variation in this pathogen (i.e. hyper-variable chromosomes and retrotransposons, in addition to mutation and parasexual recombination); that aggressiveness groups can arise from differing genetic lineages, can be influenced by the physical environment, and may arise more frequently under weather conditions favorable for pathogen growth; and therefore aggressiveness should not necessarily be related to genetic alterations in the fungus. Still, it is curious that growth in elevated CO_2 resulted in genetic alterations in both pathogen isolates only on the susceptible cultivar, while this occurred only in ambient CO_2 for the more aggressive isolate on the resistant cultivar. While the authors note that growth in elevated CO_2 can result in numerous changes in host morphology, anatomy, and physiology, speculation on possible factors driving the more frequently noted alterations in genotype under high CO_2 would have been of interest. It is possible that an increase in host photosynthate production, providing a better substrate for fungal growth, resulted in an increase in spore production under elevated CO_2 (i.e. the increase in fecundity noted), which resulted in a more variable genetic composition of the fungus. However, as the fecundity of the

more aggressive isolate increased on the more resistant cultivar under twice-ambient CO_2 (which did not exhibit any genetic alterations), this explanation appears not to fit the results obtained in this study.

A look into the future

Will disease incidence or severity increase, decrease, or remain essentially unchanged under future projected climate and atmospheric composition conditions? This is a question of utmost importance to the future stability and security of food and fiber production. Undoubtedly, rising temperatures, altered precipitation patterns, and increases in atmospheric CO_2 concentration will elicit complex changes in plant pathosystems – these changes will vary depending on host responses, the pathosystem of interest, and with the specific environmental conditions in which they are grown. Although we can theorize that host–pathogen interactions might respond in somewhat predictable ways, our current lack of knowledge precludes having any real confidence in these predictions. Further, research on interacting effects of climatic variables with CO_2 have generally indicated that plant responses are complex, highly variable, and rarely follow predictable patterns.

Nonetheless, we must begin somewhere as the CO_2 concentration in the atmosphere rises, will probably continue to do so into the foreseeable future, and will likely elicit changes in the global climate. No definitive answers are forthcoming from the current study, nor should they have been expected – the question is too broad and variable to address in a single study. However, the study does provide evidence suggesting that: elevated atmospheric CO_2 can impact important crop pathosystems; pathogen aggressiveness/disease severity might be decreased under rising CO_2, which suggests there may be plant responses which can be taken advantage of within breeding programs; this decrease in pathogen aggressiveness may not hold in the long term; fecundity may increase, which implies farmers will likely need to alter disease management strategies; and pathogen evolution might be accelerated under a high CO_2 environment. There are many more questions than there are answers.

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