PATHOGEN IMPACT ON GRASSLAND PRODUCTIVITY IN A CHANGING ENVIRONMENT

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ABSTRACT
Many of the world's grasslands are in a state of flux brought on by increasing population and the consequences of overgrazing, desertification and salinisation. Declining range land has fueled the move to greater production of hay to supplement inadequate pastures and high animal numbers. Hectarage of monoculture forage crops is increasing and reliance on qualitative resistance for controlling plant diseases is high. Over all looms the reality of increasing atmospheric CO_2 and higher levels of UV-B radiation. This paper considers the consequences of management and changing environmental factors on grassland pathology and speculates on the response of disease resistance to future changes. Plant diseases measurably reduce native grassland yield only when more acute stresses such as overgrazing, water deficits, rodent damage and salinity are absent. Plant diseases are causing serious losses in managed grasslands throughout the world and plant breeders and pathologists are challenged to keep forage legumes and grasses one resistance gene ahead of the rapidly evolving pathogens. Maintenance of qualitative resistance requires intensive efforts because it often puts heavy selection pressure for greater virulence on pathogen populations. Frequently resistant cultivars last only 3 to 5 years before being devastated by new races of the pathogen. Quantitative (polygenic) resistance exerts little or no selection pressure on pathogen populations and consequently persists for decades. The mechanisms involved in quantitative resistance are not clear, but the environmental sensitivity of quantitative resistance may be due to its reliance on products of the phenylpropanoid pathway. Quantitative resistance is well suited for controlling grassland diseases because of its longevity and it should be deployed more frequently in the future.

KEYWORDS
Quantitative resistance, qualitative resistance, polygenic, race-specific, global climate change, UV-B, salinisation, pathology

INTRODUCTION
"Observe always that everything is the result of a change, and get used to thinking that there is nothing Nature loves so well", Marcus Aurelius Antoninus, 121-180 A. D.

Desertification, salinisation, and reduced grazing hectarage are changing the management of grasslands in many areas of the world. Grasslands, which currently cover approximately 20% of the global land area, are being diverted to crop land as population pressures increase. The conversion of native grasslands to crop lands frequently results in overgrazing and desertification of the remaining ranges (Hadley, 1993; Riveros, 1993). Grassland productivity in China declined 30-50% during the last 40 years, primarily due to overgrazing and desertification (Hu et al., 1992). The imbalance between animals and available grazing in China prompted a significant increase in production of alfalfa (*Medicago sativa*) hay under irrigation to supplement winter pastures (Riveros, 1993). Forage legumes are being utilized in short leys with cereals in West Asia and North Africa to meet increasing demands for livestock (Linke et al., 1993). New tropical legumes are being incorporated into pastures in the tropics and sub-tropics to reduce the need for expensive, petroleum-based, nitrogen fertilizers and to improve animal productivity (Humphreys, 1995; Lenné and Sonoda, 1990). Will the increased reliance on monocultures rather than diverse native grasslands affect the severity of plant diseases? Atmospheric CO_2 levels are expected to double by 2060 AD and UV-B irradiation is increasing as stratospheric ozone erodes (Atkinson, 1993; Tevini, 1994). Will these global climate changes alter plant response to pathogens? A myriad of abiotic and biotic factors affect grassland productivity and triage is needed to rank the factors constraining productivity. In general, plant diseases have the potential to seriously threaten grassland productivity, but their impact is apparent only after acute problems such as overgrazing, salinity, rodent damage and water deficits are resolved.

CURRENT DISEASE PROBLEMS
Plant diseases are causing serious losses in managed grasslands throughout the world. North America has seen an increase in the incidence and severity of crown and stem rot caused by *Sclerotinia trifoliorum* in fall-seeded, forage legumes, possibly a consequence of the increased popularity of conservation tillage (Hancock, 1996; Leath, 1989). *Phytophthora medicaginis* causes root rot of alfalfa seedlings and is responsible for establishment failures and stand decline (Gray et al., 1988). White clover (*Trifolium repans*) is severely affected by viruses that decrease seed yield and diminish persistence (Barnett and Diachun, 1985; McLaughlin et al., 1996). Red clover vein mosaic virus was found in 50% of the white clover assayed in Northeastern U. S. pastures (Sherwood, 1996). The etiology of root and crown rot of forage legumes is not completely understood, but root-rot complex limits the stand life of red clover (*Trifolium pratense*), birdsfoot trefoil (*Lotus corniculatus*) and white clover (Hancock, 1996; Leath, 1989). The impact of nematodes on forage production is not well characterized but nematodes prefer alfalfa over grasses when it is grown in mixed stands (Petersen et al., 1991).

Disease problems are not restricted to North America, in Columbia and Brazil, the forage legumes *Desmodium ovalifolium* and *Stylosanthes* sp. are severely affected by wart disease (*Synchytrium desmodii*) and anthracnose (*Colletotrichum gloeosporioides*) respectively (Humphreys, 1995). *Stylosanthes* was decimated by anthracnose within a few years of its introduction into Australian pastures and *C. gloeosporioides* continues to be a threat (Chakraborty et al., 1996). Subterranean clover (*Trifolium subterraneum*) in Australia is threatened by *Phytophthora clandestina*, a new *Phytophthora* species apparently confined to this host. *Phytophthora clandestina* was associated with the total loss of irrigated pastures in Victoria, Australia (Murray and Davis, 1996).

New Zealand has experienced a 36% decline in hectarage planted to alfalfa since the early 1980s, apparently due to multiple exotic diseases and pests and a subsequent erosion of grower confidence in the crop (Skipp and Hampton, 1996). Viruses are severe in New Zealand forage legumes. The nematodes, *Heterodera trifolii*, *Ditylenchus dipsaci* and *Meloidogyne hapla* were found to attack red clover when it was overdrilled into mixed pastures containing white clover (Mercer and Campbell, 1986). Crown rust (*Puccinia coronata*) causes severe epidemics on ryegrass in mixed ryegrass/clover pastures and no resistant varieties are available (Skipp and Hampton, 1996).

Forage and food legumes are important rotational crops in the well watered regions of North Africa and West Asia, but the parasitic flowering plant *Orobanche crenata* causes major economic losses to those crops (Linke et al., 1993). Snow mold caused by *Typhula*

ishikariensis is the primary forage disease in Norway where it attacks timothy (*Phleum pratense*), the major forage grass species (A.-M. Tronsmo, Norwegian Crop Research Institute, Ås, Norway, personal communication).

PATHOGEN RESPONSE TO AGRONOMIC MANAGEMENT CHANGES

Grassland management is changing in response to new technologies and the need for higher animal productivity. In some areas, herbicide weed control has replaced companion crops and monocultures have gained in popularity for hay production. Such management changes can significantly alter disease incidence and severity.

HERBICIDES

Hectarage in birdsfoot trefoil seed production in the Champlain Valley of New York declined 94% between 1958 and 1985 due to wilt caused by *Fusarium oxysporum*. Establishment of birdsfoot trefoil is somewhat difficult and generally requires the use of companion crops to suppress weeds. The introduction of 2,4-DB chemical weed control corresponded with the demise of the birdsfoot trefoil seed production in the Champlain Valley. Subsequent greenhouse experiments confirmed an additive increase in fusarium wilt in the presence of the herbicide, although field tests were inconclusive (Murphy et al., 1985). Pesticides may have subtle metabolic effects on non-target plants, which could alter their response to pathogens.

GRAZING

The availability of grazing tolerant alfalfa varieties has increased interest both in the U. S. and in Argentina in grazing rather than confinement management of dairy cows. Grazing was shown to have a beneficial effect on the incidence of foliar disease of *Stylosanthes* in Australia. The greater the grazing pressure, the lower the incidence of disease because frequent grazing removed inoculum from the pasture (Lenné and Sonoda, 1990). Although a beneficial effect from grazing is likely on foliar diseases of alfalfa, the effect of hoof damage on alfalfa crowns and the incidence of crown and root rot is of concern. However, Navarro and Hijano (1997), concluded that there was no difference in the level of crown and root disease in grazed alfalfa as compared to alfalfa managed for hay production.

MONOCULTURE

The need for increasing amounts of stored forage to supplement inadequate pastures, the introduction of new forage legumes into tropical grasslands, the use of forage species in short leys, and reliance on qualitative disease resistance have increased the vulnerability of grasslands to disease by increasing the hectarage in monoculture. Monocultures increase yield, but lack the buffering provided by the biodiversity of native grasslands and mixed stands (Pottinger et al., 1993). Viruses spread more slowly through mixed species stands because the non-host species are barriers to rapid vector spread (Higgins et al., 1996), and mixtures of hosts and non-hosts prevent a single pathogen from completely devastating a stand.

The introduction of *Stylosanthes* into Australia during the 1970s is a sobering example of the disease risk inherent in monocultures. The 'Townsville' cultivar (*S. humilis*) was planted on nearly 2 Mha of Australian pastures in the early 1970s. In 1973 *Colletotrichum gloeosporioides* appeared and within a few years anthracnose had destroyed most of the very susceptible 'Townsville' cultivar (Chakraborty et al., 1996). *Stylosanthes* is the premier tropical legume, but its susceptibility to anthracnose requires intensive breeding efforts since *C. gloeosporioides* can overcome qualitative resistance in as little as 6 years and new *Stylosanthes* accessions were found to have new virulent races of the pathogen within 3 years of introduction (Chakraborty et al., 1990).

'Grasslands Matua' (*Bromus willdenowii*) is a perennial prairie grass cultivar that is well adapted to the dryland and hill county of New Zealand. It is also of considerable interest in Australia, the United Kingdom and the U.S. Unfortunately, 'Grasslands Matua' is quite susceptible to head smut (*Ustilago bullata*), which reduces its seed yield, stand establishment, growth and survival. When infections were severe, seed production was reduced to 1.1 Mg ha^{-1} as compared to the 5.1 Mg ha^{-1} produced by unsmutted plots. Infected plants produced fewer tillers and approximately 99% died in the first year after planting (Fallon et al., 1988).

RESISTANCE

Host resistance is the only effective way to control plant diseases in forages, pastures and other grasslands. There are two major types of resistance, qualitative (race-specific) resistance and quantitative (polygenic) resistance. Both types are amenable to recurrent phenotypic selection, the most common method of breeding for disease resistance in forages (Casler and Pederson, 1996). Recurrent phenotypic selection involves screening the population of plants with the pathogen then selecting 100 or more resistant plants for intercrossing to develop a new population. The new population is then screened and the cycle is repeated as often as necessary to develop the desired level of resistance (Elgin, et al., 1988). Screening for disease resistance often requires working under controlled conditions in greenhouses or growth chambers, to ensure uniform pathogen pressure. Prolonged selection under these conditions without selection pressure for agronomic factors can result in the loss of persistence and/or yield despite an increase in disease resistance, indication that factors other than disease resistance are critical for stand longevity (Casler and Pederson, 1996). The reverse situation does not appear to be true, two cycles of seed increase without selection pressure for disease resistance did not diminish quantitative resistance to purple leaf spot (*Stagonospora arenaria)* in orchard grass (*Dactylis glomerata*) or to brown leaf spot (*Pyrenophora bromi*) in smooth bromegrass (*Bromus inermis*) (Berg and Sherwood, 1994).

Selection for agronomic characteristics without regard for disease resistance can produce cultivars with valuable agronomic traits that quickly succumb to pathogen pressure in the field. Winter hardiness was a limiting factor in the use of berseem clover *(T. alexandrinum)* and development of the more winter hardy 'Bigbee' cultivar expanded its range northward approximately 10^{o} in latitude. Unfortunately, 'Bigbee' berseem clover is extremely susceptible to the root-knot nematodes *Meloidogyne arenaria, M. incognita* and *M. javanica*, which effectively limits its usefulness (Baltensperger er al., 1985). In a similar situation, low-tannin *Lespedeza cuneata*, a perennial legume adapted to the southeastern U. S., was developed because the high- tannin cultivars, although very disease resistant, were not palatable to animals. The palatable, low-tannin lines were extremely susceptible to Rhizoctonia web-blight and a negative correlation was found between tannin content and disease resistance (Donnelly, 1983).

QUALITATIVE RESISTANCE

Qualitative resistance provides a high level of resistance to plant disease, however its use carries the risk of total crop failure and frequently requires continuous, intensive breeding efforts. Qualitative resistance exerts selection pressure on many pathogen populations for greater virulence because it prevents sporulation of all but the most virulent members of the population. The consequence of such selection pressure is the rapid appearance of new pathogen races.

Cultivars of *Stylosanthes* with qualitative resistance to *C. gloeosporioides* are rapidly defeated by the pathogen in Australia. The *Stylosanthes* cultivar 'Fitzroy' was discarded within 5 years of its release due to severe yield reductions from anthracnose (Chakraborty et al., 1990, 1996). Frequently, qualitative resistance can be maintained only through the continuous introduction of new resistance genes. Pathologists and plant breeders in Australia are approaching that problem from two directions. They have developed 'Siran', a synthetic cultivar of *Stylosanthes* that is a composite of cultivars with qualitative resistance to several different races of *C. gloeosporioides*. 'Siran' was released in 1991 and is persisting and performing well so far. The second approach is the development of cultivars such as 'Amiga' that have quantitative resistance to anthracnose. 'Amiga' was released in 1988 and continues to perform well (Chrakraborty et al., 1996).

QUANTITATIVE RESISTANCE

Quantitative (polygenic) resistance lacks the gene-for-gene relationship found in qualitative resistance and is controlled by numerous genes with small, additive effects (Christ et al., 1987). Confirming evidence for the presence of quantitative resistance requires the demonstration of additivity and a continuous distribution of response (Simmonds, 1991). Quantitative resistance supports coexistence rather than annihilation of either the pathogen, due to total plant immunity, or the host as a consequence of complete virulence. The genes involved in quantitative resistance frequently have additional non-defense related roles in the host (Bennetzen and Jones, 1992; Heath, 1991). Quantitative resistance does not exert strong selection pressure on pathogen populations for more virulence and thus is a viable alternative to the "boom and bust" cycle of qualitative resistance (Bennetzen and Jones, 1992; Simmonds, 1991).

Quantitative resistance is successful against Verticillium wilt (*V. albo-atrum*) in alfalfa. Susceptible alfalfa varieties suffered yield declines beginning in the third production year under irrigated conditions in Alberta, Canada, whereas resistant varieties yielded well for the full 7 years of the field trial (Huang et al., 1994). The quantitatively resistant *Stylosanthes* cultivar 'Amiga' has successfully withstood pressure from *C. gleosporioidies* for 8 years.

Although attractive, quantitative resistance is not widely used, primarily because it is not well characterized and is difficult for breeders to manipulate. Efforts are underway to elucidate the physiological mechanisms regulating the expression of quantitative resistance. Environmental sensitivity is a characteristic of quantitative resistance and recent work indicates that at least a portion of that sensitivity may be due to its need for products of the phenylpropanoid pathway, all of which are secondary metabolites and require energy for their production (Pennypacker et al., 1993). Shading experiments demonstrated that quantitative resistance to Verticillium wilt in alfalfa was lost when infected plants were grown under 40% of ambient photosynthetic photon flux density (PPFD). Photosynthesis was reduced significantly by the low light treatment, suggesting that carbon assimilation is critical for the expression of quantitative resistance (Pennypacker et al., 1994, 1995).

All plants require light for photosynthesis, so an additional experiment was conducted to determine whether the requirement for carbon assimilation was unique to quantitative resistance or whether it was shared by qualitative resistance. Alfalfa is an open-pollinated, autotetraploid therefore definitive physiological research requires the use of clones. To eliminate the problem of genetic heterogeneity between clones, quantitative or qualitative resistance was invoked within a single clone. *Verticillium albo-atrum* was used to activate quantitative resistance and *Fusarium oxysporum* f. sp. *medicaginis* to activate qualitative resistance. A complete factorial experiment was conducted using 4 alfalfa clones, 2 with high levels of quantitative resistance, one with moderate quantitative resistance, and one with no resistance to *V. albo-atrum*. All 4 clones had qualitative resistance to *F. oxysporum* f. sp. *medicaginis*. Under moderate PPFD (~500 µmols $m^{-2}s^{-1}$), all clones expressed resistance to Fusarium wilt, whereas a spectrum of response was seen to Verticillium wilt (Fig. 1). Qualitative resistance to Fusarium wilt was maintained under low light (~100 µmols $m^{-2}s^{-1}$) but quantitative resistance to Verticillium wilt was lost (Fig. 1), again supporting the hypothesis that quantitative resistance requires energy for its expression (Pennypacker and Risius, 1996).

Increased knowledge of the physiological mechanisms involved in quantitative resistance will aid plant breeders in their efforts to manipulate this desirable form of resistance. Quantitative resistance is well suited for grassland species because once it has been incorporated through recurrent phenotypic selection, it does not require continuing, high-cost breeding efforts.

PATHOGEN DIVERSITY

Qualitative resistance requires the constant search for new resistance genes and quantitative resistance benefits from new genetic sources as well. Frequently wild germplasm is used as a source of resistance genes, often under the assumption that wild germplasm has been exposed to a wide range of virulent pathogen races. (Lenné and Wood, 1991). In some cases, the centers of germplasm diversity do not parallel centers of diversity of the pathogen (Lenné and Wood, 1991). Rhodes (Ohio State University, personal communication) tested 700 *Medicago* plant introductions from diverse geographic regions for resistance to *Sclerotinia trifoliorum* and found that the most of the resistant introductions came from the cooler regions of the world rather than from the centers of diversity of *Medicago* (Fig. 2). The center of diversity for soybean (*Glycine max*) is eastern Asia however, high levels of resistance to rust (*Phakopsora pachyrhizi*) were found in Australia (Lenné and Wood, 1991).

BIOTECHNOLOGY AND PLANT BREEDING

Biotechnology has great potential for improving disease resistance, but although several disease resistance genes have been cloned (Lindow, 1995), as yet, none have been expressed in foreign germplasm. Forage legumes are inviting targets for biotechnological manipulation and partial genetic maps have been constructed for alfalfa and *Stylosanthes*. Care must be taken, however, when manipulating the phenolic metabolism of forage legumes to avoid negative effects on nodulation, palatability and antiquality factors (Higgins et al., 1996).

ENVIRONMENTAL CHANGE AND DISEASE RESISTANCE

Change, a dynamic process driven by human and natural forces, continually sculpts the world and its inhabitants. As Antoninus so aptly observed in the second century A.D., "there is nothing Nature loves so well". Plants have successfully adapted to innumerable climatic changes over the millennia. Will plant disease resistance, a product of human intervention, fare as well in a climate that is changing more rapidly that in past ages?

SALINISATION

Secondary or human induced salinisation, a problem as old as irrigation, remains a serious and growing problem. Salinisation caused the shift from wheat cultivation to more salt tolerant barley in the Tigris and Euphrates river valleys during the 3rd millenium

BC. By 1700 BC wheat was no longer grown in those valleys primarily due to salinisation (Ghassemi et al., 1995). Secondary salinisation is primarily a consequence of irrigation without adequate subsurface drainage. Water applied to a field in excess of the crop's requirements flows through the soil profile and raises the water table. When the water table approaches the soil surface, evaporation precipitates suspended salts and salinisation occurs (Ghassemi et al., 1995). Salinization can also be caused by land clearing and the replacement of trees with shallow rooted crops. Countries affected by secondary salinisation are predominantly in the arid and semi-arid regions of the world and include Argentina, Australia, Brazil, China, Chile, Russia, Central Asia, Egypt, India, Iran, Iraq, Pakistan, Peru, Spain, Syria, Thailand, Turkey, and the U. S.

Plants respond to salinity in a variety of complex ways, including excretion of salt through specialized glands, storage of salt in vacuoles, and maintenance of turgor through osmoregulation. Osmoregulation in response to salt stress involves the production of increased amounts of sucrose to provide the osmoticum for osmotic adjustment (Cushman et al., 1990). Carbon that is used for osmoregulation is not available for constructive metabolism and thus is an energy drain on the plant. Salt storage in vacuoles involves the active, ATP-requiring transport of Na^+ across the vacuole membrane, an additional energy drain on the plant.

The energy requirements of salt tolerance are evident in the increased respiration and reduced growth of salt stressed plants (Cushman et al., 1990). The metabolic cost of salt tolerance may divert resources necessary for the expression of quantitative disease resistance, which could explain the diminished resistance to Verticillium wilt in alfalfa grown under salinity stress reported by Howell et al. (1994). Quantitative resistance may be less effective in plants growing under salinity stress.

ATMOSPHERIC CO_2

The burning of fossil fuels, although a minor portion of the total global carbon budget, plays a major role in destabilizing the atmospheric CO_2 compensation point, the point were CO_2 released is balanced by CO_2 fixed through photosynthesis or absorbed by the ocean. As a result, CO_2 is increasing at a rate of 0.5% per year (Bowes, 1993). Atmospheric CO_2 has increased from 280 ppm in 1750 AD to 350 ppm in 1993 and is predicted to reach 600 ppm by 2060 (Atkinson, 1993). Doubling the CO_2 concentration of the atmosphere will have a major effect on plant growth. Currently, plants using the C_3 carbon-fixation pathway lose 25% of the carbon they fix because of photorespiration (Bowes, 1993). Greater availability of CO_2 would reduce competition between O_2 and CO_2 for the active site on the carboxylase enzyme Rubisco in C_3 plants, thus reducing photorespiration and increasing their net carbon gain. Increased growth rates and higher photosynthetic rates were recorded for C_3 plants when the CO_2 levels were doubled experimentally (Bowes, 1993). The increased photosynthetic rate reported for C_3 plants under experimentally elevated CO_2 also reduced the negative effects of salinity (Bowes, 1993). Salinity might not have a negative effect on quantitative resistance under higher atmospheric CO_2. C_4 plants are not expected to show a response to increasing CO_2 because their carboxylase enzyme is protected from O_2 and thus is already operating at optimum levels.

Based on the sensitivity of quantitatve resistance to light levels and by extrapolation, carbon assimilation (Pennypacker et al., 1994, 1995), quantitative resistance should function at a higher level in C_3 plants when atmospheric CO_2 doubles. Another factor that might influence plant disease resistance is a change in the chemical composition of plants under elevated CO_2. The roots of perennial ryegrass (*Lolium perenne*) grown under 700 ppm CO_2 had a significantly slower decomposition rate compared to roots grown under 350 ppm CO_2 (Gorissen, et al., 1995). Although the mechanism was unclear, Gorissen et al. speculated that either the measured increase in the C:N ratio under 700 ppm CO_2 or an increase in root lignin content might be responsible. Increased lignin content under elevated CO_2 might make roots more resistant to penetration by plant pathogens. However, balanced against that factor is the increase in the root:shoot ratio that many plants show under high CO_2 (Bowes, 1993). An increase in the number of roots would increase the exposure of the plant to soil borne pathogens and increase the potential for infection.

UV-B

Evidence that the stratospheric ozone layer is being eroded is undeniable and industrial chlorofluorocarbons are partially responsible (Bornman and Sundby-Emanuelsson, 1995). One consequence of the depletion of stratospheric ozone is increased levels of incoming UV-B radiation (290-320 nm). UV-B is biologically active and detrimental to living organisms. Sensitive plants exposed to UV-B frequently show reduced photosynthesis, a consequence of decreased Rubisco activity (Tevini, 1994). Reductions in yield and biomass often result from UV- B exposure, although there is genetic variation in sensitivity and half of the 300 species tested were not sensitive (Tevini, 1994). In addition to causing detrimental effects on photosynthesis, UV-B causes an increase in the activity of enzymes in the phenylpropanoid pathway that results in production of increased amounts of flavonoids, ferulic acid, and anthocyanines (Liu et al., 1995). The phenolic compounds accumulate in the leaf epidermis and mesophyll and are thought to shield the photosynthetic machinery from the UV-B radiation. The multiple and conflicting effects of UV-B on plant metabolism make predictions about its effect on plant disease resistance difficult. Reduced photosynthesis might reduce the effectiveness of quantitative resistance but increased activity in the phenylpropanoid pathway, which is involved in the expression of quantitative resistance may offset the negative effect of reduced photosynthesis. When the theorized beneficial effects of elevated CO_2 on quantitative resistance are considered against the potential negative effects of increased UV-B radiation, the fate of quantitative disease resistance is impossible to predict.

CONCLUSION

The effect of plant diseases will only be apparent when more acute constraints to grassland productivity are eliminated. The greater biodiversity of native grasslands buffers them against the detrimental effects of pathogens. In more highly managed systems, in particular monocultures that rely on qualitative disease resistance, pathogens have the potential to severely limit yield. Qualitative resistance carries the threat of total crop failure and requires intensive breeding efforts to remain one gene ahead of the pathogen. New pathogen races with greater virulence will continue to arise as long as there is strong selection pressure from qualitative resistance. Financial support for grassland and forage pathology is rapidly declining, therefore, it would be prudent for plant breeders to increase efforts to exploit quantitative resistance. Reduced reliance on monocultures and greater biodiversity in both forage and grassland systems should buffer forage production against the ravages of endemic and exotic pathogens, which are an unfortunate consequence of shortened travel time and increased global interactions. Quantitative resistance should become even more effective as the global climate changes.

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Figure 1
Response of 4 alfalfa clones to *Fusarium oxysporum* f. sp. *medicaginis* and *Verticillium albo-atrum* under two photosynthetic photon flux levels. The clones showed the spectrum of response to *V. albo-atrum* under high PPFD (~ 500 µmol $m^{-2}s^{-1}$), demonstrating that clones a2 and a18 had quantitative resistance to that pathogen. All of the clones had qualitative resistance to *F. oxysporum* f. sp. *medicaginis*. Note that qualitative resistance was not affected by low PPFD (~ 100 µmol m^{-2} s^{-1}) whereas, quantitative resistance was lost. Asterisk indicates significant differences within clones within light levels at $P = 0.001$.

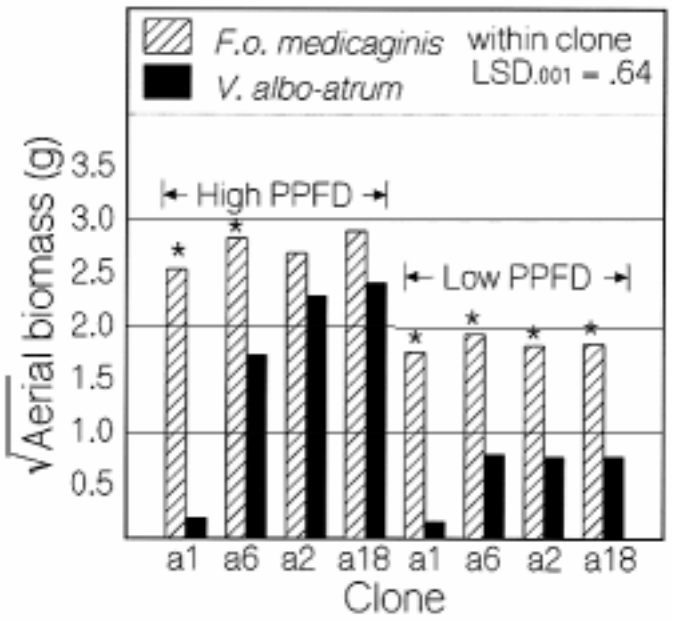

Figure 2
Geographic distribution of alfalfa introductions from the U. S. National Germplasm collection that had greater than 20% of the plants showing resistance to *Sclerotinia trifoliorum*. Note that the greatest number of plant introductions meeting that criteria were from the cooler regions of the world rather than from the center of diversity of alfalfa. Data was provided by Dr. Landon Rhodes, Dept. of Plant Pathology, Ohio State University, Columbus, OH.