Assessment and Reaction of *Triticum aestivum* Genotypes to *Fusarium graminearum* and Effects on Traits Related to Grain Yield and Seed Quality

By

Matthew Randolph Chappell

Thesis submitted to the Faculty of Virginia Polytechnic Institute and State University in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

In

Crop and Soil Environmental Sciences

APPROVED:

_____	_____
Dr. Carl Griffey, Chair	Dr. Erik Stromberg
_____	_____
Dr. Glen Buss	Dr. Marcus Alley

Keywords: *Fusarium graminearum*, *Triticum aestivum*, yield loss, scab, wheat

December 14, 2001
Blacksburg, Virginia, U.S.A.

Assessment and Reaction of *Triticum aestivum* Genotypes to *Fusarium graminearum* and Effects on Traits Related to Grain Yield and Seed Quality

By
Matthew Randolph Chappell

Dr. Carl A. Griffey - Major Professor
Crop and Soil Environmental Science

(Abstract)

Fusarium graminearum (Schwabe), causal organism of fusarium head blight (FHB), has become a major pathogen of wheat (*Triticum aestivum* L.) throughout North America. Since its discovery in the United States, the disease has spread south and east until at present it is an annual threat for growers of winter wheat in the Mid-Atlantic region. Yield losses for soft red winter (SRW) wheat averaged 908 kg ha^{-1} in the FHB outbreak of 1998 (Griffey et al., 1999). The economic loss from this single FHB epidemic was an estimated 8.5 million dollars.

Environmental conditions favorable for FHB development, including above average rainfall and temperatures during anthesis, have become more common in the Upper-Midwestern wheat-growing region over the past decade, leading to substantial losses in wheat and barley crops. This, coupled with low prices being paid for wheat, has prompted research toward solving the problem of FHB across the nation. The majority of labor and financial resources devoted to FHB research are dedicated to incorporating FHB resistance into adapted wheat lines. While this is a prudent method of combating this disease, this process will take many years to complete.

We have examined all FHB assessment parameters, which include FHB incidence, FHB severity, FHB index, percentage fusarium damaged kernels (percentage FDK), and 15-acetyl deoxynivalenol toxin (DON toxin) accumulation, to ascertain which assessment parameters best quantify FHB resistance levels in addition to grain yield and grain volume weight (GVW) losses. FHB index provides the most reliable in-field assessment of a genotype's resistance level, whereas percentage FDK provides a reliable measure of a genotype's resistance level post-harvest. FHB index and percentage FDK are also the most predictive assessment parameters with regard to grain yield and GVW

loss. A wide range in both level and type of resistance was observed among genotypes examined in this study. The cultivars Agripro Patton, Ernie, INW9824, Roane, and the experimental line NY87048W-7388 consistently had lower scores for FHB assessment parameters and lower losses of grain yield and GVW.

Acknowledgements

I would not have been able to complete this research without the guidance and paychecks of Dr. Carl Griffey, and I thank him for both in addition to the vast knowledge of plant breeding which he has bestowed upon me. Jianli Chen has also been a wonderful mentor, teaching me the ways of scab, for which I am extremely grateful. Tom Pridgen has been irreplaceable as a friend and mentor, and without his assistance in virtually everything relating to the nursery this research would have been a nightmare (especially irrigation!). I would also like to thank my committee members for their support and suggestions along the way, which has given me a much clearer understanding of proper research techniques, technical writing, and presentation techniques. The members of the small grains group have also been outstanding to work with. From my first days, Wendy Rohrer, Wynse Brooks, Jianli Chen, Mark Vaughn, Bob Paris, John Zwonitzer, and Amanda Bullard welcomed me with open arms and allowed me to take part in all aspects of the program.

My family has been extremely supportive of everything that I have set out to do in life, and I thank them for the encouragement, laughs, and putting up with rather infrequent visits over the last 3.5 years. I would like to especially thank 'Mother Mary' for her understanding and being there every time I needed to vent and 'Pa' who always had a good baseball game on and good conversation waiting.

Last but not least I would like to thank Debby Kubisiak, who has stood strong through the trying times during the last six months, supported my work, and pushed me in every aspect of my life. Without her smiles, laughs, and questions I would not have been able to make it this far. She has also supported my decision to continue my education at the University of Georgia in a field somewhat separated from that which I am in now, and I appreciate her support in what was a soul-searching decision. I would also like to thank her for reading and editing, a task which itself is about as exciting as watching paint dry.

Table of Contents

List of Tables

List of Appendices

CHAPTER 1

EXPANDED LITERATURE REVIEW

Historical Impacts of Fusarium Head Blight

The first description of fusarium-like fungi attacking *Zea* spp. and *Triticum* spp. was reported in the United Kingdom in 1884 by W.G. Smith in a book entitled 'Diseases of Field Crops, Chiefly such as are Caused by Fungi' (Parry et al., 1984). F.D. Chester provided the first detailed description of fusarium head blight (FHB) in a research bulletin of the Delaware Agriculture Experiment Station (Chester, 1890). Ten years later (1900), *Fusarium graminearum* was identified in cereal crops for the first time in the United States in Minnesota (Canadian Grain Commission, 2000).

By the year 1917, thirty-one of forty states surveyed by the USDA reported FHB with estimated losses of 288,700 metric tons of wheat and barley combined, although the hardest hit areas consistently were Ohio, Indiana, and Illinois. In 1919, the largest outbreak of wheat scab to date was recorded, with losses of 2.18 million metric tons of wheat over the entire United States. Subsequently, there was a marked drop in losses due to FHB as more intensive management practices were adopted in the last half of the 20th century that aided in residue decomposition.

During this period, FHB epidemics were threatening cereal crops throughout the world. Losses in China, where the disease is endemic, have been reported nearly every year of recorded history and have been as high as 1.03 million metric tons in a single year. Korea suffered from near famine conditions in 1963, when FHB was deemed responsible for loss of nearly the entire barley crop. Epidemics have also been reported, less frequently, in Canada, Argentina, and Japan.

A surge in the use of conservation tillage, by U.S. producers, in the 1980's resulted in a dramatic increase in FHB incidence and greater losses. As conservation practices spread throughout the United States, losses due to FHB spread geographically as well. This situation became a national concern in 1993, when FHB reached epidemic proportions over the tri-state area of Minnesota, North Dakota and South Dakota as well as a large portion of Canada. Losses over this area in 1993 alone reached an estimated $1 billion, which is one of the greatest crop disasters recorded in U.S. history (McMullen et al., 1997). In Minnesota, 18.5 percent of the planted crop was so severely infected that it was deemed unharvestable (Jones and Mirocha, 1999). FHB epidemics occurred again in

1994 and 1996 over the same region. In 1996, epidemic conditions spread to the states of Iowa, Arkansas, Louisiana, Indiana, Illinois, Wisconsin, Michigan, and Ohio. Losses in Michigan and Ohio were estimated at 156 million USD (McMullen et al., 1997).

Total losses from FHB over the entire U.S. since 1991 have been estimated at 13.62 million metric tons, at a value as high as $2.6 billion dollars (USW&BSI Newsletter, 1999). Each epidemic of the last decade has coincided with above average rainfall during flowering and milk stages of crop development. This is a daunting statistic considering the predicted climatic changes occurring, with increasing spring and summer rainfall reported over the wheat producing areas of the plains states over the last decade (McMullen et al., 1997).

In Virginia, losses due to FHB were noticeable yet relatively insignificant until 1998, when epidemic levels of scab were widespread throughout the state. The average SRW wheat yield statewide was 3027 kg ha^{-1}, which was 1547 kg ha^{-1} less than the previous year and 908 kg ha^{-1} less than the six year average. This amounted to a 155,233 metric ton drop from 1997 and a 92,595 metric ton drop from the previous six year average. Fiscal losses were estimated to be 14.4 million dollars compared to 1997 and 8.5 million dollars compared to the six year average (Griffey et al., 1999).

Fusarium graminearum Identification and Life Cycle

Gibberella zeae (Schwein) Petch (anamorph: *Fusarium graminearum* Schwabe) is an ascomycete fungus known to cause stalk and ear rot in *Zea* spp., crown rot in *Dianthus* spp., and head scab in *Triticum* spp. and *Hordeum* spp. (Bowden and Leslie, 1999). In Virginia, as well as other mild continental climates (SE United States, Southern China, and Southern Europe) *Gibberella zeae* in its perfect stage or *F. graminearum* in its imperfect stage is the predominant causal organism of FHB. In colder continental climates, such as the plains of Canada, North China, and Eurasia, the predominant species is *F. poae*. In cool maritime climates such as the Pacific Northwest of the United States, Scandinavia, and Eastern Canada, *F. avenaceum* and *F. culmorum* are the predominant causal organisms of FHB. However, year to year variation has been noted with regard to species makeup in the aforementioned physiographic regions (Martens et al., 1994).

The disease cycle of FHB commences with formation of perithecia (perfect stage of the fungi) on senescing plant tissue previously colonized by *F. graminearum*. The perithecia (*Gibberella zeae*) over-winter on undecomposed plant residues of the previous crop. As temperatures increase in spring months of the subsequent year, infection of cereals initiates as *Gibberella zeae* forcibly discharge ascospores (aerial spores) produced within the perithecia. Once discharged, the ascospores are wind dispersed and infect cereals via germination of ascospores and penetration of the flowering structure or glume. Immediately following infection, mycelia and/or hyphae will spread through the spike and colonize both systemically and saprophytically. Mycelia may also act as a secondary source of inoculum, splash or wind dispersed onto later tillers. Infection may also occur via conidia, which form on undecomposed crop residue and are splash dispersed upward to the spike. However, conidia only account for a small portion of infections, due to their lack of being transported by wind (Francl et al., 1999; Martens et al., 1994).

Australian researchers have divided *F. graminearum* into two groups based on ecological adaptability and sexual cycles. Group I, found in arid regions of the world (South Africa, North America, and Australia), causes crown rot of cereals and grasses. Members of this group are heterothallic, infertile, or both. Group II is the causal organism of FHB in cereals within the Eastern United States, Canada, Europe, and the Far East and is primarily an airborne pathogen. This group is homothallic and produces ample quantities of perithecia, which are the main source of over-wintering inoculum. The relationship between Groups I and II is unknown to researchers, however it is postulated that perithecia (sexual bodies) produced by Group II are essential for survival in colder continental climates and hence they are a product of evolutionary divergence. This sexual cycle may also be a means for outcrossing within Group II and increase the pathogen's ability to adapt to differing climates, host resistance, and fungicides. Occurring in milder climates, Group I has no need for over-wintering structures, and therefore this group has evolved with a life cycle lacking a sexual stage (Bowden and Leslie, 1999; Aoki and O'Donnell, 1999).

Two classes have been described within Group II of *F. graminearum*. Type A is pathogenic to both *Triticum* spp. and *Zea* spp., reproduces rapidly, forms colonies composed of reddish mycelia, and produces low amounts of mycotoxins. Type B is non-

pathogenic to *Zea* spp., reproduces more slowly, forms light brown lesions (as opposed to red colonies), and is responsible for accumulation of high levels of mycotoxins in cereals (Cullen et al., 1982). Type A comprises 95 percent of all *F. graminearum* strains collected. To date, no relationship based on adaptability, life cycles, or morphology between the two types has been established (Bowden and Leslie, 1999).

Fusarium graminearum Inoculum Sources

It has been demonstrated that *F. graminearum* survives between wheat rotations as well as between wheat and maize or wheat and barley rotations, both on living and senescent plant parts (Bai and Shaner, 1994). *Fusarium* spp. have been recovered from a number of grass species and is well known for its ability to successfully colonize maize stalks and ears (Stack, 1999). Although ascospores, conidia, chlamydospores, and hyphal fragments can all serve as sources of inoculum, ascospores and macroconidia are crucial in early infections due to their ability to be air or water-splash dispersed.

Crop residue is of greatest concern in the distribution of inoculum at the onset of an epidemic. Crop debris which harbors *F. graminearum* when left on the soil surface includes maize, wheat, barley, wild oats, sorghum, soybean, and rice. This plant material is often colonized by mycelia, which may over-winter in mild years and serve as a source for producing airborne inoculum the following growing season. Additionally, a number of wild grasses are suspected to harbor the pathogen, although research has not been completed on this subject (Bai and Shaner, 1994; Miller et al., 1998). Even if crop debris is free of the pathogen at time of harvest, it has been reported that later colonization can occur and lead to future infections (Miller et al. 1998). This is especially important in areas such as eastern Virginia where wheat, barley, soybean, and maize are grown over large areas in close proximity or in crop rotations.

Inoculum dispersal on a local (100 m – 50 km) and mesoscale (< 50 km) level is not fully understood at the present time due to a lack of experimental protocol needed for accurate measurement of spore dispersal and travel. However, Francl et al. (1999) surveyed multiple locations, both within and isolated from known inoculum sources, to gain a better understanding of the number of spores that successfully travel from inoculum sources to adjacent wheat fields. They found ascospores on wheat spikes at

sites distant from known inoculum sources. The number of spores recovered at distant (<10 Km) sites was often sufficient to induce disease, given proper environmental conditions. The limiting factor in this research is that the source of inoculum found on spikes collected at sites removed from known sources was unknown to the researchers, therefore precise distance of spore travel could not be adequately assessed.

In a separate study, Fernando et al. (1997) studied the localized spread of FHB from an artificially inoculated point using two forms of inoculum. Ascospores, released from perithecia on *Gibberella zeae* colonized maize kernels, were used as an inoculum source. FHB determinations were made within plots to determine spread of the pathogen. Conidia were also applied, sprayed onto spikes in the center of the test plot, and FHB determinations were recorded to measure spread of the inoculum. In ascospore and conidial inoculated plots, it was found that prevailing winds were a significant factor in FHB spread, although ascospores utilized wind to more efficiently spread through the plot. Ascospores induced FHB over a greater area and with greater severity than conidia, with a range of 5 to 22 meters (spread was significantly greater downwind) and 5 meters, respectively. Spread of the pathogen in the case of both inoculum sources dropped to 10 percent beyond the aforementioned distances, which resulted in minimal disease.

Infection and Disease Development

Infection of wheat spikes by *Fusarium* spp. may occur at any time from spike emergence -- Zadoks' growth stage 50 (Zadoks' 50) -- through late milk (Zadoks' 77) (McMullen et al., 1997). However, most severe infections occur at anthesis (Zadoks' 60-69). Infection occurs primarily when ascospores or conidia are deposited on or within the flowering structure of the spike. There are contradictory reports concerning the chemical stimulus for spore germination and initial growth. However, with the exception of a single study (Nkongolo et al. 1993), it is widely accepted that the high levels of choline and betaine contained in anthers serve as stimulants for infection and growth of the fungus, after which the fungus grows downward through the developing caryopsis. At this point, colonization may cease or may continue to spread into the adjacent florets via the rachis (Bai and Shaner, 1994). Studies examining host tissue degradation have noted that as colonization progresses throughout the spike, cellulose, xylan, and pectin are

reduced in the cell walls of colonized ovaries, lemma, and rachis (Kang and Buchenauer, 2000). This cell wall degradation, which occurs when infection takes place at anthesis, is often responsible for discontinued kernel development or severely discolored and shriveled seed (Stack, 1999).

Preceding or subsequent to flowering, infection may also occur via ascospores, mycelia, conidia, chlamydospores, or hyphal fragments deposited directly into the glume, rachis, or palea (Bai and Shaner, 1994). Infections of this type often lead to tombstone kernels, reduced size in mature kernels, or visually symptom free kernels that can only be distinguished by toxicological screening (Stack, 1999).

After infection and during favorable conditions for colonization, *Fusarium* spp. will develop and spread through the rachis and adjacent florets, growing systemically or saprophytically. In the case of saprophytic growth, the fungus may spread to adjacent heads via physical contact (conidia or mycelia), splash dispersal (conidia), or wind dispersal (ascospores). Mycelia are the primary means of saprophytic spread, and appears as orange-red lesions on the rachis or glume. The spores produced are easily spread to adjacent florets via wind or water.

Systemic growth of the pathogen often appears as senescence of the spike from the infection point to the top of the spike and/or downward to the culm. This senescence is a result of clogging of the vascular tissues within the rachis by mycelia (Bai and Shaner, 1994) or degradation of cell wall materials (Kang and Buchenauer, 2000). As systemic colonization progresses, the senescent tissue will take on a pink coloration. In the case of both saprophytic and systemic growth, perithecia of *Gibberella zeae*, the perfect stage of the fungus, may appear as purple-black lesions throughout the colonized areas of the floret. These perithecia will appear late in the season, serving as over-wintering structures to initiate infection in subsequent growing seasons. In addition, mycelia and hyphal fragments will colonize crop residues not colonized at the time of harvest, which will further perpetuate the pathogen (Bai and Shaner, 1994).

Environmental Conditions Favorable for Spore Release, Infection, and Disease Development

To date, Anderson (1948) has provided the most comprehensive account of optimal growth conditions for *F. graminearum*. He reported that the optimum temperature for both infection and development is 25°C. Infection and development are suppressed at 15°C and 32°C. Moisture is the limiting factor in infection and disease development. Moisture in the form of rainfall, fog, or heavy dew for 36-72 hours is required for optimal infection and development. However, shorter durations of wetness at anthesis may trigger infection, after which longer moist periods may lead to severe colonization. It has been observed that at optimal temperatures, as little as 16 hours of moist conditions will lead to substantial infection and colonization (Bai and Shaner, 1994).

Environmental conditions sufficient for ascospore discharge have been found to differ somewhat from conditions favorable for disease development. While sufficient moisture in the form of rain, dew, fog, etc. has been proven necessary for development and dispersal of perithecia and mycelia on debris, it has been found that the desiccation of mature perithecia prompts release of ascospores. Ascospores are released typically from 1600 to 2400 hrs, as a sharp rise in humidity or lowering of barometric pressure may serve as the stimulus for release. Excessive moisture ceases all ascospore release, however splash dispersal of mycelia and conidia will predominate as the primary inoculum source during these periods (Paulitz, 1996).

Effect of Tillage Practices on *Fusarium* spp. Survival

Conservation tillage is the single most important management decision a producer of small grains must make with respect to FHB. Until the middle 1970's, producers were instructed that a clean field at planting increased yields of all field crops. Several events spawned the inception of conservation tillage and no-tillage, which are defined as systems that leave no less than 30 or 60 percent, respectively, of crop residues on the soil surface (VFBN, 2000; Bockus and Shroyer, 1998). The initial stimulus for conservation tillage was promoted as researchers discovered that massive amounts of topsoil were being lost, and crop productivity was declining as a direct result of this

phenomenon. A Kansas study typified this occurrence by reporting that every 2.5 cm of topsoil loss resulted in a 77 kg ha^{-1} loss per year in wheat production (Bockus and Shroyer, 1998). Steady increases in fuel prices over the last twenty years also persuaded producers to accept conservation tillage as an essential management practice.

Government mandated tillage practices for farmers nationwide have also contributed to increased FHB outbreaks. In the Midwestern FHB outbreak of 1993, surveys were collected from producers in order to correlate tillage practices with yield losses. It was found that those producers using conservation tillage suffered greater losses than those who used chisel or moldboard plowing prior to planting. For nearly every epidemic since, surveys have yielded similar results and have prompted research into the area of management decisions that reduce the severity of FHB (McMullen et al., 1997).

Research conducted at the University of Minnesota has provided the most comprehensive study on tillage practices and their effect on FHB. This research has addressed two questions; 1) What is the decomposition rate of crop residues under various tillage schemes?, and 2) How long does *F. graminearum* survive in crop residues? It was found that large discrepancies exist concerning the amount of cereal crop residue available for colonization by *F. graminearum* in various tillage schemes. Chisel plowing in the Minnesota test plots lead to 43 percent of original crop residues being retained after one year of fallow conditions. With 7.5 to 10 cm chisel plowing, the amount of residue remaining dropped to 21 percent. The results of 15 to 21 cm chisel and moldboard plowing yielded similar results.

Inoculum survival within this Minnesota study among various tillage schemes was based on the number of observed nodes colonized by *F. graminearum*. Large discrepancies were found in the percentage of inoculum survival for these various tillage schemes. Surface chisel plowing allowed the highest survival rate of all plowing schemes, with 67 percent of observed nodes colonized after one year of fallow conditions. The percentage of nodes colonized for 7.5 to 10 cm chisel plowing was 63 percent, and 54 percent for 15 to 20 cm chisel plowing. Moldboard plowing at 15 to 20 cm offered the best control of *F. graminearum* with only 50 percent survival over a one-year fallow period (Pereyra et al., 1999). In a separate report, it was determined that sporulation of perithecia is observed for three or more years from a single source of

inoculum (Francl et al., 1999). This would suggest that as a buildup of debris occurs in a minimum tillage scheme, inoculum potential rises exponentially.

The Minnesota data implies that tillage schemes can reduce, but not completely control *Fusarium* spp. survival. The reduction of crop residues with moldboard plowing leads to less debris for *Fusarium* spp. to colonize and therefore reductions in inoculum over fallow periods (Pereyra et al., 1999).

Virginia has been noted as a leader in conservation tillage, with 46.2 percent or 44,873 hectares of winter wheat grown under conservation tillage schemes in 1999-00 (VFBN, 2000). While this is a positive attribute environmentally, it will only add to the potential problem of FHB epidemics.

Chemical Control of Fusarium Head Blight

Research on the use of fungicides to control *Fusarium* spp. has been underway since 1977. However, studies have been largely discredited due to variable disease levels achieved by researchers and treatment effects that are consistently measured but using different disease assessment parameters. Only recently have studies conclusively determined the feasibility of fungicide use in controlling FHB. To date the most conclusive study, provided by Jones (2000), has found that benomyl and tebuconazol are the most effective fungicides available for reducing disease incidence, severity, and number of fusarium damaged kernels.

Several questions remain that must be addressed prior to accepted use of these chemicals for control of FHB. The first pertains to chemical residues in food, feed, and beverage products. This is of utmost concern, due to the oncogenic properties of the two above-mentioned chemicals coupled with their late application (Zadoks' 60). Returns on the investment of chemical application must also be considered prior to recommending these chemicals for use. Jones (2000) established that chemical control is cost effective in susceptible and moderately susceptible genotypes, and less cost effective in resistant and moderately resistant genotypes. Most susceptible genotypes are declining in popularity, which shifts the focus of research on the cost effectiveness of chemicals on moderately resistant genotypes. With the current low prices being paid for wheat and the low return

from treating resistant and moderately resistant genotypes, it may not be feasible for producers to utilize these options without first formulating a disease forecasting system.

To date, solutions to these questions remain unclear and therefore the use of fungicides to control FHB seems a distant prospect. However, research is underway on new application technology and new chemical formulations specific to *Fusarium* spp. (McMullen et. al, 1997; Jones, 2000).

Resistance and Quantifying Resistance in Wheat Genotypes

Host resistance to FHB has been classified into five categories or "types", each of which is believed to be independent in assessing a genotype's resistance, tolerance, or susceptibility. Type I resistance is defined as complete resistance to infection, and was first described by Schroder and Christensen (1963). Type II resistance, also described by Schroder and Christensen (1963), refers to the host's ability to block pathogen spread within the spike once infection has occurred. Type I and II resistances have been commonly measured using spike and floret inoculation, respectively, under greenhouse conditions. Under such conditions, genotype x environment effects can be minimized and type I and II resistance can be easily quantified using a five-point scale developed by Wang et al. (1982) and modified by Xu and Fan (1985).

Miller et al. (1985) described type III resistance as the host's ability to block the accumulation of mycotoxins within infected kernels. Measurement of type III resistance is performed by chemical analysis of grain samples. Mesterhazy et al. (1999) have reported wide variation in type III resistance within genotypes between test years. Test year differences are the result of genotype x isolate x environment interactions, which are complex and not well understood. Multiple test year data are required to discern type III resistance and minimize environmental and isolate effects.

Type IV resistance, described by Mesterhazy (1995), refers to the host's ability to maintain sound kernels in colonized florets, thus reducing GVW loss. Measurement of type IV resistance has been accomplished by visually assessing the percentage fusarium damaged kernels within a sample or calculation of a GVW loss in inoculated versus non-inoculated field trial. Mesterhazy first described type V resistance in 1995 as the host's ability to maintain grain yield with colonization, also termed tolerance. Measuring this

resistance type has been accomplished by comparison of grain yield produced in inoculated versus non-inoculated plots. The latter method has been criticized due to varying effects within and between plots; however, results obtained using this method have correlated well with results using test weight or 1000 kernel weight (Mesterhazy et al., 1999).

Active versus Passive Resistance Mechanisms

Active (physiological or biochemical) resistance includes all resistance types discussed above. It can include inhibition of infection, restriction of colonization after infection, metabolic degradation of chemicals produced by the pathogen, and ultimately restriction of GVW and grain yield losses. Currently, breeding efforts throughout the world are focused primarily on type II resistance. Reports have indicated that under epidemic conditions type I resistance is easily overcome, and thereafter type II resistance becomes the most promising line of defense (Mesterhazy, 1995). Less attention has been given to breeding for types III, IV, and V resistances due to the difficulty in quantifying these types of resistance in wheat genotypes.

Mechanical resistance and escape mechanisms include morphological characteristics such as plant height, presence of awns, floret density on a spike, flowering time and duration, waxy glumes, and degree to which florets open (Mesterhazy, 1995; Bai et al., 1994). Mesterhazy (1995) described the ideal genotype as one with a height of greater than 100 cm. Data has shown that plants shorter than 100 cm are closer to debris (soil surface) and therefore inoculum is in closer proximity to the spike leading to increased disease. Awns have also been linked to increased disease as awns hold moisture, keeping the spike wet for longer periods and increased surface area to capture spores (Mesterhazy, 1995). However, more recent reports have contradicted this statement, as spore numbers on awned vs. awnless genotypes were not significantly different (Francl et al., 1999).

Flowering time and duration also provide an escape mechanism for genotypes. Genotypes that flower concurrently with favorable environmental conditions for spore dispersal and infection are more likely to develop FHB. In addition, disease is decreased in genotypes with a shorter flowering period and in those genotypes that release anthers

quickly (Mesterhazy, 1995). Less is known about other mechanical resistance mechanisms, but theories suggest that waxy glumes serve as a barrier to infection and help to extrude moisture and tight glumes serve to restrict access of airborne inoculum to flowering structures.

Current Sources of Resistance/Tolerance found in *Triticum* spp.

Plant pathologists and breeders first reported differences in wheat genotypes for resistance to FHB in the 1920's. These genotype differences were classified both broadly and in many cases incorrectly due to a lack of knowledge regarding genotype x environment effects (Stack, 1999). It is widely accepted that the best solution for controlling the complex problem of FHB in Virginia, and throughout the world, is the introgression of resistance into elite adapted genotypes. Substantial progress has been made in breeding for FHB resistance within the United States, especially during the last decade. However, to date sources with complete resistance have not been found (Ban, 1997). The best-known resistance sources have come from Chinese, Japanese, and Brazilian spring wheats.

China suffers from nearly annual FHB epidemics throughout the nation, and it is postulated that natural selection has favored those genotypes exhibiting moderate resistance. This is especially true in the Yangtze Valley, where FHB outbreaks are often severe. Breeding efforts in China have resulted in the release of FHB resistant type II cultivars Sumai 3, Ning 7840, Ning 8026, W14, Shaan 85, Fan 1, Futai 8944, and Futai 9002 (Wang and Wang, 1991; Bai and Shaner, 1994). Japanese wheat lines Nobeokabozu Komugi, YFGZ, and Saikai 165 are also reported to carry good type II resistance (Yu, 1991; Snijders, 1990a; Ban and Suenaga, 2000). These spring wheat lines, in addition to Frontana (type I), are not adapted within the U.S. (Singh et al., 1995). While breeding efforts are underway to introgress resistance from these lines into elite genotypes, only a few genotypes have been released with resistance from these parents. This is due to poor combining ability of these genotypes with respect to yield and quality. Progeny derived from crosses with these resistant genotypes are also tall, later maturing, and have fewer florets per head, all of which are unfavorable characteristics (Bai and Shaner, 1994).

Sumai 3 possesses the best known combining ability for FHB in association with yield related traits and is being widely used as a parental source in FHB breeding programs in the United States. Ning 7840 and Ning 8026, both derived from Sumai 3 and postulated to carry some of the same resistance genes, are also widely used in breeding programs. This has raised questions regarding reliance on resistance from Sumai 3 and its progeny as the primary genetic source and eventual erosion of resistance (Bai and Shaner, 1994). However, it has been determined using diallel analysis that Ning 7840 (progeny of Sumai 3) and Frontana contain different resistance genes. This will allow breeders to pyramid resistance genes from diverse genetic backgrounds (Van Ginkel et al., 1996).

Unlike resistance, which is defined as the ability to directly resist infection and spread of the pathogen, tolerance is defined as the ability of a diseased genotype to restrict damage and retain kernels once infected. Resistance is commonly measured in greenhouse screenings, which are uniform and repeatable. Tolerance is much more difficult to measure due to inconsistent disease levels from year to year in field trials, which provide the only accurate measurement of tolerance. However, with improved inoculation methods, Mesterhazy et al. (1999) have proven that there are significant genotypic differences in tolerance levels to FHB. The findings of Mesterhazy et al. (1999) are interesting, however there are significant limitations in use of tolerant genotypes for breeding and cultivation. The inherent problem in the use of tolerant genotypes for breeding will be determining the level of tolerance and determining the heritability and genetic control of this reaction.

Mesterhazy et al. (1999) concluded that genotypes exhibiting tolerance occur much more frequently than those exhibiting resistance. Interestingly, it was also observed that genotypes derived from the same parentage were likely to show similar levels of tolerance to FHB. This would suggest an additive-dominance model of inheritance with respect to tolerance. Little screening has been performed to assess tolerance levels, possibly due to the need for field trials over multiple years to adequately discern tolerance from environmental effects. It may be more feasible to backcross type II resistance into genotypes which exhibit tolerance to FHB, which includes reduced grain yield and GVW loss. Backcrossing type II resistance into tolerant genotypes would

circumvent the need to identify genes responsible for type IV and V resistance, which to date are unidentified.

Resistance Found in Wild Relatives

Extensive studies have been published on the search for *Triticeae* species possessing resistance to FHB. Mielke (1988) examined species of *Triticum* and *Aegilops* genera, and identified no accessions in either genus with FHB resistance or tolerance levels equal or superior to that found in current genotypes of *Triticum aestivum*. Ban (1997) found that accessions AG.91-35 and AG.91-24 of *Elymus humidus* exhibited better type II resistance than Sumai 3. In the most promising survey of wild relatives to date, Wan et al. (1997) identified 13 species of *Roegneria*, *Hystrix duthiei*, and *Psathyrostachys juncea* possessing better type II resistance than Sumai 3. Of these, *Roegneria tsukushiensis* var. *transiens* and *R. ciliaris* expressed complete resistance under artificial inoculation and natural epidemic conditions. These findings are promising in that such species may serve as future sources of resistance genes to be introgressed into adapted genotypes.

Heritability of Resistance

Buerstmayr et al. (2000) recently published the most comprehensive study on heritability of FHB resistance in *Triticum* species. This research focused on estimating broad-sense heritability in two *Triticum* species populations. The wheat cultivar Capo (moderately susceptible) was crossed with UNG-226 (resistant) and SVP-72017 (resistant) to form these two recombinant F_4–derived populations. In order to minimize and comprehend genotype x environmental interaction, inoculation of the two populations was carried out under glasshouse conditions in addition to field conditions. Results of this research were positive in several aspects. Foremost, broad-sense heritabilities (H) of 0.75 and 0.77 were measured under glasshouse conditions in the two populations, which implies that selection of resistant genotypes is possible. With these findings in mind, area under the disease progress curve (AUDPC) was measured using a visual symptom rating system. Resistance was determined from AUDPC to be quantitative in the two populations with a stepwise distribution including transgressive

segregates in both populations. The continuous distribution of classes for AUDPC also implies that FHB resistance is controlled by a polygenic system. In addition, additive genetic variance was greater than additive x additive epistasis. This also favors selection of resistant genotypes in breeding programs.

In a separate study, Waldron et al. (1999) measured heritability of resistance in 112 recombinant inbred lines (RILs) from a single cross of Sumai 3/Stoa. Inoculation of 9-10 heads per RIL was carried out under greenhouse conditions to reduce genotype x environment interaction. Heritability on an entry-mean basis was measured at 0.78, whereas on a plot basis heritability was measured at 0.49. In addition, FHB severity was found to have a continuous distribution in the 112 RILs, which suggests polygenic inheritance. In a similar study, Bai et al. (1999) measured mean broad-sense heritability in 133 RILs derived from a cross of Ning 7840/Clark at 0.86 (ranged from 0.79-0.91). Saur and Trottet (1992), studying heritability in 56 S_1 families and their S_2 progeny measured broad-sense heritability within generations ranging from 0.80 to 0.90. From these studies, it can be deduced that early-generation selection for FHB resistance would be more effective in recurrent selection schemes. Additionally, between generation heritabilities were much lower and varied (0.23 to 0.58), which suggests that siblings of tested plants should be used to determine heritability in recurrent selection breeding rather than offspring of tested genotypes.

Several other studies have reported similar findings, but with less reproducibility. Snijders (1990b), using ten F_2 populations derived from a half-diallel cross, measured FHB symptoms by inoculating individual plants. He reported heritability in the range of 0.50 to 0.89 in the ten populations. However, in the F_3 generation, Snijders (1990c) was unable to reproduce the results as heritabilities ranged from 0.00 to 0.96 with no correlation among populations or environments. Singh et al. (1995) reported heritability ranging from 0.66 to 0.93 in F_6 derived populations of crosses of Frontana with 'Inia 66', 'Opata 85', and 'Pavon 76' using single floret inoculation. These results were well correlated over replications and significant within populations, however were based upon tests only carried out in one environment.

Durability of Resistance

The effectiveness and success of resistant genotypes released in the future will greatly depend on the durability and stability of resistance. Durability of resistance is dependent on pathogen variation, host-pathogen interaction, mechanism of resistance, and cultural practices. Variation in *F. graminearum* has proven to be immense. However, gene-for-gene interaction between the pathogen and host appear to be non-existent. This is due to a low degree of pathogenic specialization and incomplete host resistance, which is quantitatively inherited. Because little selection pressure has been exerted on *F. graminearum* populations in the past, genotype x pathogen interactions have not evolved and, therefore, the pathogen has evolved as a non-genotype specific pathogen. Resistance, therefore, is thought to be horizontal as defined by Van der Plank and potentially quite durable despite large pathogen variation (Miedaner, 1997; Snijders and Eeuwijk, 1991).

Mesterhazy et al. (1999) addressed the question of changing population structure within *Fusarium* spp. and possible effects on loss of cultivar-specific resistance. A current resistant source was tested over sixteen environments for four years, and no erosion of resistance was observed. In a separate study, Eeuwijk et al. (1995) tested twenty-five genotypes in six locations over a three-year period with the same results. Both studies concluded that erosion of resistance would likely be extremely slow and stepwise, if it occurred at all. However, the true test of durability has not been examined, and only can be ascertained after resistant genotypes are grown widely by producers over many years. Selection pressure under such circumstance will determine whether isolates capable of overcoming specific resistance genes can evolve (Miedaner, 1997).

Inheritance of Resistance to Fusarium Head Blight

Christensen et al. (1929) first described that FHB resistance is an inherited characteristic and noted transgressive segregation in populations derived from a resistant by susceptible cross. Bai et al. (1989), using a 3x3 half-diallel cross of three resistant and three susceptible genotypes, concluded that variation among the six parents was conferred by three gene-loci and several minor modifying genes. In addition, it was determined that inheritance of FHB resistance is a partially or fully dominant trait. Wang

et al. (1991), studying six crosses between one susceptible and six resistant genotypes concluded that four scab resistance genes were present in the resistant genotypes (Fan 60069, Sumai 3, Ning 7840, Fanshan, Long 96b-1165, and Ke 80F3-119) in varying numbers. The presence of varying numbers of resistant genes in the progeny of each cross supported an additive gene effect model, which was important in determining the level of resistance of the progeny. Lin et al. (1992), also using half-diallel crosses, found that the inheritance of scab resistance is governed by dominant genes, which act in an additive manner. The resistance genes found in Wang-shui-bai, Sumai 3, and Xin-zhong-chang are dominant and these three genotypes possess good general combining ability (GCA) for traits such as GVW and plant height.

These findings led to further studies on inheritance of FHB resistance by means of a recurrent selection program. With early studies agreeing that inheritance fits an additive-dominance model, recurrent selection should produce genotypes with high levels of resistance. Jiang et al. (1992) studied inheritance of resistance in a recurrent selection population using the male-sterile gene Ms^2 over two years (1989-1990). Results showed that phenotypic recurrent selection reduced incidence of FHB in populations up to 19.17 percent. Jiang and Wu (1993), reporting on the same recurrent selection program with an additional year's data (1989-1991), noted that recurrent selection had continued to produce progeny with resistance greater or equal to the most resistant parent. The greatest gains were realized the first two years of selection in this scheme. It was noted that FHB resistance, in addition to characteristics such as plant height, grain weight, and 1000 grain weight can be simultaneously selected for. These results are supported by research of Yang et al. (2000), who used a recurrent selection program to achieve a 25 percent increase in the frequency of individuals with greater resistance than Sumai 3 by the C_4F_1 generation. Selection for plant height, GVW, and 1000 KW was also successful and two genotypes were released from this program.

Using 10x10 full-diallel analysis, Snijders (1990a; 1990c) studied the GCA versus specific combining ability (SCA) of FHB resistance sources. GCA includes additive and additive x additive variance. In calculating GCA and SCA it was determined that GCA effects are most important in FHB resistance. The SCA effects indicated that there were no combinations, which had resistance higher or lower than expected from the GCA

effect or resistance level of the parent. This implies that the most resistant progeny can be produced by crossing two parents with the greatest negative GCA (differing resistance genes). The level of FHB resistance of the parental line should be a good estimate of the resistance level of the progeny. It should be noted that crossing of resistant lines with identical resistance genes would not result in improved resistance in progeny. It was also determined that there were dominance effects in the direction of resistance, which agrees with earlier studies.

Genes Contributing to Resistance and Current Marker Research

Only recently has research on gene number, gene location, and molecular marker discovery been undertaken with any confidence. Many different approaches have been undertaken to locate resistance genes in a variety of genotypes, with the greatest amount of research being focused upon finding genes and gene location in the Chinese cultivar Sumai 3 and its progeny.

The first study on gene number and location of resistance genes in Sumai 3 was carried out by Yu (1982). It was deduced, using monosomic analysis, in a Sumai 3/ Chinese Spring cross that five genes contributed to Type II resistance on chromosomes 1B, 2A, 5A, 6D, and 7D of Sumai 3. However, resistance genes from Chinese Spring may have confounded results in this study. In a later study, Yu (1991) found genes for resistance in PHJZM (progeny of Sumai 3) on chromosomes 3B, 5B, 6B, 6D, and 7A. Also identified was a single resistance gene in YGFZ on chromosomes 3A and a single resistance gene on chromosome 4D of WN2. Ban and Suenaga (1997), using linkage analysis, located one resistance gene from Sumai 3 on the long arm of 5A or 6B. However, precise location was undetermined due to the linkage of the FHB resistance gene with a suppressor gene for awnedness, which has alleles located on both chromosomes.

Buerstmayr et al. (1997), studying two progeny of Sumai 3, found only chromosomes 3B and 6B in common for proposed resistance genes in both progeny, whereas chromosomes 1B, 3A, 4B, 4D, 5A, and 6D carried resistance genes in one of two progeny. In a later backcross reciprocal monosomic analysis study, Buerstmayr et al. (1999), identified chromosomes 1B, 4B, 5A, 6B, and 6D as contributing to the resistance

of U136.1. Interestingly, U136.1 is thought to carry resistance genes from both Nobeokabozu and Sumai 3. However, results from these two studies are typical of results from many researchers who use monosomic analysis in determining gene number and location in which there has rarely been agreement, even when the genotype Sumai 3 and its progeny are utilized. Yet it is of importance to note that chromosomes 4D, 5A, 6B, 6D, and 7A are frequently cited in monosomic analysis studies using progeny of Sumai 3.

In an effort to more quickly and precisely map FHB resistance genes, Bai et al. (1999) performed amplified fragment length polymorphism (AFLP) analysis on 133 RILs derived using single seed decent of the cross Ning 7840/Clark. Using 300 combinations of AFLP primers, the 133 RILs were screened for polymorphisms using bulked segregate analysis. Eleven markers within one linkage group (covering 39.4 cM) were identified which were responsible for 60 percent of the genetic variation of FHB resistance in Ning 7840 (a derivative of Sumai 3). In addition, r^2 values for these markers are high (10-53 percent in single marker regression analysis), implying their location is relatively close to a quantitative trait loci (QTL), which was initially thought to be on chromosome 7B. To explore the possibility of using these markers in marker assisted selection, AUDPC was calculated for one of the eleven markers. AUDPC indicted that as an indirect selection tool, the marker would yield 32 percent highly resistant, 54 percent moderately resistant, 14 percent moderately susceptible, and no highly susceptible plants.

Bi et al. (2000) converted the 11 AFLP markers found by Bai et al. (1999) into sequence tagged sites (STS) and/or single nucleotide primers (SNP) markers. Nine of the 11 AFLP markers linked to FHB resistance were isolated from polyacrylamide gel, with seven linked to one major QTL and the remaining two markers were linked to two separate minor QTLs. Southern blot analysis showed that six of the nine markers, linked to a major QTL, were low copy clones. Two types of allele specific primers were designed based upon AFLP marker sequence. The first was internal to the AFLP selective primers (*EcoR1* and *Mse1*), and these gave no allele-specific amplification. The second type of primer was designed to include one of the two AFLP selective primers, and five of six constructed primers identified as STSs, reproduced the polymorphisms seen using the original AFLP markers. These five primers revealed a single base change at position 91 between Ning 7840 and Clark. This point mutation was used to design a SNP termed

SNPmaj (4.8 cM from AFLP marker), and later interval analysis showed SNPmaj and STSmaj (2.9 cM from AFLP marker) mapped to the same linkage group (LG1), which spans 81.2 cM. Additionally, another allele-specific marker belonging to the linkage group LG2 (covering 73.6 cM) was found and termed STSmin.

Using an alternative approach to produce PCR-based markers, Zhou et al. (2000) screened 93 SSR markers for polymorphisms in the mapping population created by Bai et al. (1999). It was found that 34 of the single sequence repeats (SSRs) were polymorphic between the two genotypes, with the SSR *Xgwm 389* (chromosome 3BS) being linked with the AFLP markers associated with the major QTL identified by Bai et al. (1999). This prompted the analysis of all SSRs associated with chromosome 3B. Two of the SSRs on chromosome 3BS (*Xgwm533* and *Xgwm493*) and one SSR on 3BL (*Xgwm340*) showed polymorphisms between Ning 7840 and Clark. The three SSRs on 3BS were determined to be members of the same linkage group. The discovery that *Xgwm533* on 3BS was linked with the major QTL discovered by Bai et al. (1999) agrees with the findings of Anderson et al. (1999), who associated this SSR with a QTL for resistance in Sumai 3.

Based upon LOD (logarithm of the ratio of the odds that two loci are linked) scores, the order of the three SSRs on 3BS is as follows: *Xgwm389* – 5.3cM - *Xgwm533* – 4.8cM - *Xgwm493*. *Xgwm533* explained 44 percent of the phenotypic variation of FHB resistance and is more closely linked with the FHB resistance QTL than *Xgwm389* (36 percent variation explained) or *Xgwm493* (34 percent variation explained), the two of which are believed to flank the QTL. Therefore, if *Xgwm389* and *Xgwm493* are utilized in marker- assisted selection (MAS), the probability of missing the major QTL by selecting both markers is 0.25 percent.

Using the above mentioned mapping population, Bai et al. (2000) constructed a molecular map using 568 AFLP markers with the goal to map the QTL for low DON toxin accumulation. Using data from two greenhouse replications, a single major QTL was located which explained 23-26 percent of total phenotypic variance. Ironically, this QTL was located in the same region as the QTL found in the 1999 study (postulated to be on 3B). However, the variance explained by this QTL was low, which implies that there may be other loci involved in reduced DON toxin levels or that extremely high disease

levels may have masked the expression of the QTL. Ongoing work is being done to answer these two possibilities.

In a separate study, Waldron et al. (1999) used RFLP marker analysis to find markers associated with FHB resistance. One hundred and twelve RILs were developed from the cross Sumai 3/Stoa and evaluated in greenhouse trials for resistance. Five genomic regions were found to be associated with FHB resistance, with three regions being derived from Sumai 3 and two from Stoa. Using interval mapping analysis, chromosome 2AL from Stoa was determined to carry a major resistance gene, designated *QFhs.ndsu-2A* (marker designation *XkuH16*). Stoa was also found to contribute a minor gene on chromosome 4BL. More importantly, chromosome 3BS was determined to carry major resistance gene from Sumai 3, designated *QFhs.ndsu-3B*. The marker associated with this gene (*Xcdo981*) explained 15.4 percent of the phenotypic variation seen in greenhouse screenings and using the three markers (*Xcdo98, Xbcd331,* and *Xcdo524*) associated with Sumai 3, 29.5 percent of phenotypic variation was explained. These findings agree with the work by Zhou et al. (2000), working with the mapping population from Bai et al. (1999). This work also agrees with Grausgruber et al. (1999) who used a set of substitution lines of single chromosomes from cultivars Cheyenne, Hope, and Lutescens 62 into Chinese Spring. Their work also identified chromosomes 3B and 5A as contributing to the FHB resistance of Chinese Spring.

Several studies have indicated the possible presence of FHB resistance genes linked with known QTLs. The first such observation was made by Snijders (1990a), who observed increased resistance in awned genotypes derived from the same cross. Presence of awns is based on absence of a single dominant gene *B1* on chromosome 4B in the parent, which would suggest that the resistance gene is either linked to the gene for presence of awns or linked in repulsion to the suppressor gene for presence of awns. Ban and Suenaga (2000) observed a similar segregation among progeny of the crosses Sumai 3/Gamenya and Sumai 3/Emblem. They determined recombination values of 15.1±3.3 percent and 21.4±4.3 percent between one FHB resistance gene and *B1*, which was located on chromosome 5A.

In a separate study, Ittu et al. (2000) examined 108 RILs from the cross of F1054W/Sinceron for resistance to FHB. Three years of AUDPC data indicated that

genes conferring resistance to FHB are associated with the *GliR1* allele on chromosome T1BL.1RS of Sinceron. In addition, a smaller yet significant association was observed with the *GliD1b* allele on chromosome 1D. Selection for the *GliR* gene would reduce bread making quality of progeny, yet it is suggested that the FHB resistance gene may not be on the translocated rye chromosome arm 1RS and therefore linked loosely with the *GliR* allele. In this situation, the linkage could be broken or parents could be identified with the resistance gene minus the 1RS arm.

Effects of Fusarium Head Blight on Grain Volume Weight and Grain Yield

GVW is an important parameter used in assessing resistance or tolerance of a genotype to FHB. Negative correlations between FHB and GVW have been reported by Saur (1991), Saur and Trottet (1992), and Saur and Benacef (1993). Studies have repeatedly shown that FHB reduces GVW in two ways. Kernels affected by infection and colonization of florets appear as pink, chalky white, or pale gray shriveled seed with little or no endosperm. These seeds will significantly reduce GVW of a sample, even when they only account for a small percentage of the sample. In highly susceptible genotypes, up to 80 percent of florets can show these symptoms, leading to dramatic reductions in grain-volume weight (Jones and Mirocha, 1999). The second cause of grain-volume weight reduction occurs without direct kernel colonization. When the rachis becomes blocked by mycelia, senescence of the florets above and below the infection point may lead to kernels that show no visible symptoms. However, the seeds are less dense due to reduced nutrient uptake as a result of vascular blocking (Bai and Shaner, 1994).

Yield is also affected by FHB in two ways. Kernels located in florets that have been colonized by *F. graminearum* are often expelled with chaff during harvest. In highly susceptible genotypes this can amount to 30-70 percent of kernels (Bai and Shaner, 1994; Miedaner, 1997). Kernels that have not been colonized by *F. graminearum,* yet suffer from reduced size and density as a result of vascular blocking, also decrease yield. These kernels are often retained by the combine at harvest but reduce overall grain-volume weight and hence yield (Bai and Shaner, 1994).

Relationship Between Fusarium Head Blight and Mycotoxins

In Mid-Atlantic and Northeastern states, the primary mycotoxin produced by *F. graminearum* in the heads of wheat is DON toxin (Miedaner, 1997). This toxin has been recovered from all parts of the spike and was observed to be most abundant in the rachis, followed by the glume, and least concentrated in the kernels (Sinha and Savard, 1997). These findings support the hypothesis that DON toxin is essential for the systemic spread of the pathogen through the spike. The level of DON toxin produced by various isolates also differs drastically, and correlates with an isolate's pathogenicity (Miedaner, 1997).

Sinha and Savard (1997) measured DON toxin levels in various wheat kernels from colonized spikes, including normal looking kernels, kernels of normal color but reduced in size, and kernels that showed visible symptoms such as pink or chalky appearance. Their results were surprising, in that 50 percent of normal or reduced-size kernels had detectable DON toxin concentrations, although typically at 5 ppm or less. DON toxin was detected in all kernels with visible colonization, but the range of DON toxin concentration varied greatly from 1 ppm to 600 ppm. These results typify studies conducted by Mesterhazy et al. (1999), Jones and Mirocha (1999), and Miedaner (1997).

Literature Cited

Aoki, T. and K. O'Donnell. 1999. Morphological and molecular characterization of *Fusarium pseudograminearum* sp. nov., formerly recognized as the group 1 population of *F. graminearum*. Mycologia 91:597-609.

Anderson. 1948. The development of *Gibberella zeae* headblight of wheat. Phytopathology 38:595-611.

Anderson, J.A., B.L. Waldron, B. Moreno-Sevilla, R.W. Stack, and R.C. Frohberg. 1999. Detection of *Fusarium* head blight resistance QTL in wheat using AFLPs and RFLPs. v.1.p.135-137. *In* Proceedings of the 4th International Wheat Genetics Symposium.

Anderson, J.A., S. Liu, M.O. Pumphrey, J.L. Gonzalez-Hernandez, and E.J. Wennerlind. 2000. A protocol for marker-assisted selection of a Fusarium head blight resistance gene derived from Sumai 3. p.239-243. *In* Proceedings of the 2000 National Fusarium Head Blight Forum.

Bai, Guihua, Q. Xiao, and J. Mei. 1989. Studies on the inheritance of scab resistance in six wheat varieties. Acta Agriculturae Shanghai 5/4:17-23.

Bai, G.-H., F.L. Kolb, G. Shaner, and L.L. Domier. 1999. Amplified fragment length polymorphism markers linked to a major quantitative trait locus controlling scab resistance in wheat. Phytopathology 89:343-348.

Bai, G.-H., R. Plattner, G. Shaner, and F. Kolb. 2000. Molecular mapping of a QTL for deoxynivalenol tolerance in wheat. p.13-16. *In* Proceedings of the 2000 National Fusarium Head Blight Forum.

Bai, G.-H. and G. Shaner. 1994. Scab of wheat: prospects for control. Plant Disease 78:760-765.

Ban, Tomohiro. 1997. Evaluation of resistance to *Fusarium* head blight in indigenous Japanese species of *Agropyron* (*Elymus*). Euphytica 97:39-44.

Ban, T. and K. Suenaga. 1997. Inheritance of resistance to Fusarium head blight caused by *Fusarium graminearum* in wheat. Cereal Research Communications 25:727-728.

Ban, T. and K. Suenaga. 2000. Genetic analysis of resistance to Fusarium head blight caused by *Fusarium graminearum* in Chinese wheat cultivar Sumai 3 and the Japanese cultivar Saikai 165. Euphytica 113:87-99.

Bi, I.V., F.L. Kolb, L.K. Boze, G. Bai, and L.L. Domier. 2000. Development of STSS and SNPS linked to Fusarium head blight resistance of wheat using AFLPs and antifungal gene analogs. p.55-58. *In* Proceedings of the 2000 National Fusarium Head Blight Forum 55-58.

Bockus, W.W. and J.P. Shroyer. 1998. The impact of reduced tillage on soilborne plant pathogens. Annual Review of Phytopathology 36:485-500.

Bowden, R. 1995. Effects of reduced tillage on wheat diseases. KSU Extension Bulletin no. PP01-7/95. http://www.ksu.edu/plantpath/extension/facts/wheat15.html

Bowden, R and J.F. Leslie. 1999. Sexual recombination in *Gibberella zeae*. Phytopathology 89:182-188.

Buerstmayr, H., B. Steiner, M. Lemmens, and P. Ruckenbauer. 1997. Chromosomal location of *Fusarium* head blight resistance genes in wheat. Cereal Research Communications 25:731-732.

Buerstmayr, H., M. Lemmens, G. Fedak, and P. Ruckenbauer. 1999. Back-cross reciprocal monosomic analysis of Fusarium head blight resistance in wheat (*Triticum aestivum* L.). Theoretical and Applied Genetics 98:76-85.

Buerstmayr, H., B. Steiner, M. Lemmens, and P. Ruckenbauer. 2000. Resistance to Fusarium head blight in winter wheat: heritability and trait associations. Crop Science 40:1012-1018.

Canadian Grain Commission. 2000. Brief history of Fusarium head blight. http://wpg216.cgc.ca/Pubs/fusarium/fusarium-e2.htm#macinnes23

Chester, F.D. 1890. The scab of wheat. Delaware Agricultural Experiment Station Report 3:89-90.

Christensen, J.J., E.C. Stakman, and F.R. Immer. 1929. Susceptibility of wheat varieties and hybrids to fusarial head blight in Minnesota. Minnesota Agricultural Experiment Stations Bulletin 59.

Cullen, D., R.W. Caldwell, and E.B. Smalley. 1982. Cultural characteristics, pathogenicity, and zearalenone production by strains of *Gibberella zeae* isolated from corn. Phytopathology 72:1415-1418.

Eeuwijk, van F.A., A. Mesterhazy, Ch.I. Kling, P. Ruckenbauer, L. Saur, H. Burstmayr, M. Lemmens, L.C.P. Keizer, N. Maurin, and C.H.A. Snijders. 1995. Assessing non-specificity of resistance in wheat to head blight caused by inoculation with European strains of *Fusarium culmorum, F. gramenarium,* and *F. nivale* using a multiplicative model for interaction. Theoretical and Applied Genetics 90:221-228.

Fernando, W.G.D., T.C. Paulitz, W.L. Seaman, P. Dutilleul, and J.D.Miller. 1997. Head blight gradients caused by Gibberella zeae from area sources of inoculum in wheat field plots. Phytopathology 87:414-421.

Francl, L., G. Shaner, G. Bergstrom, J. Gilbert, W. Pedersen, R. Dill-Macky, L. Sweets, B. Corwin, Y. Jin, D. Gallenberg, and J. Wiersma. 1999. Daily inoculum levels of Gibberella zeae on wheat spikes. Plant Disease 83:662-666.

Griffey, C.A., J. Chen, T. Pridgen, M. Chappell, and E. Stromberg. 1999. Research on Fusarium Head Blight in the Virginia Tech small grains program. p.95-98. *In* 1999 Joint Research Conference of the Eastern Wheat and Southern Small Grain Workers.

Grausgruber, H., M. Lemmens, H. Burstmayr, and P. Ruckenbauer. 1997. Genetic analysis of *Fusarium* head blight resistance and toxin tolerance in wheat, using inter-varietal chromosome substitution lines. Cereal Research Communications 25:743-744.

Hyde, Norm. 2000. Wheat harvest shrinks. Virginia Farm Bureau Newsletter. http://www.vafb.com/news.

Ittu, M., N.N. Saulsscu, I. Hagima, G. Ittu, and P. Mustatea. 2000. Association of Fusarium head blight resistance with gliadin loci in a winter wheat cross. Crop Science 40:62-67.

Jiang, G., Z. Wu, Z. Chen, D. Huang, Q. Ziao, H. Chen, H. Zhu, and Y. Fang. 1992. Studies on the development of scab-resistance gene pool in wheat—A preliminary report on effects of population improvement in the scab-resistance resource gene pool. Scientia Agricultura Sinica 25/6:30-37.

Jiang, G. and Z. Wu. 1993. Studies on the development of scab-resistance gene pool in wheat III—Exploration of the establishment of Base populations and their recurrent selection. Acta Agronomica Sinica 19:441-452.

Jones, R.K. and C.J. Mirocha. 1999. Quality parameters in small grains from Minnesota affected by Fusarium head blight. Plant Disease 83:506-511.

Jones, R.K.. 2000. Assessments of Fusarium head blight of wheat and barley in response to fungicide treatment. Plant Disease 84:1021-1030.

Kang, Z. and H. Buchenauer. 2000. Ultrastructural and cytochemical studies on cellulose, xylan, and Pectin degradation in wheat spikes infected by *Fusarium culmorum*. Journal of Phytopathology 148:263-275.

Lin, Y, Z. Yang, and Z. Wu. 1992. Genetic analysis of resistance to scab (*Gibberella zeae*) in wheat varieties from different regions. Acta Agriculturae Shanghai 8:31-36.

Martens, J.W., W.L. Seaman, and T.G. Atkinson. 1988. Diseases of Field Crops in Canada. The Canadian Phytopathological Society; Ontario, Canada. p 37.

McMullen, M., R. Jones, and D. Gallenberg. 1997. Scab of whet and barley: a re-emerging disease of devastating impact. Plant Disease 81:1340-1348.

Mesterhazy, A. 1995. Types and components of resistance to Fusarium head blight of wheat. Plant Breeding 114:377-386.

Mesterhazy, A., T. Bartok, C.G. Mirocha, and R. Komoroczy. 1999. Nature of wheat resistance to Fusarium head blight and the role of deoxynivalenol for breeding. Plant Breeding 118:97-110.

Miedaner, L. 1997. Breeding wheat and rye for resistance to Fusarium diseases. Plant Breeding 116:201-220.

Mielke, H., 1988. Untersuchungen uber *Fusarium culmorum* (W.G.Sm) Sacc. Als Fuss- and Ahrenkrankheitserreger beim Weizen. Mitteilungen aus der Biologischen Bundesanstalt fur Land- und Forstwirtschaft Berlin-Dahlem 238:101pp.

Miller, J.D., J.C. Young, and D.R. Sampson. 1985. Deoxynivalenol and FHB resistance in spring cereals. Phytopathology 113:359-367.

Miller, D.J., J. Culley, K. Fraser, S. Hubbard, F. Meloche, T. Ouellet, W.L. Seaman, K. Turkington, and H. Voldeng. 1998. Effect of tillage practice on Fusarium head blight of wheat. Canadian Journal of Plant Pathology 20:95-103.

Nkongolo, K.K., D. Dostaler, and L. Couture. 1993. Effet de la betaine, de la choline et d'extraits d'antheres du ble sur la croissance du *Fusarium graminearum*. Revue Canadienne De Phytopathologie 15:81-84.

Parry, D.W., T.R. Pettitt, P. Jenkinson, and A.K. Lees. 1984. The cereal Fusarium complex. p. 301-320. *In* Blakeman, J.P. and B. Williamson (eds.). Ecology of Plant Pathogens. CAB International, Wallingford OX, UK.

Paulitz, T. 1996. Diurnal release of ascospores by Gibberella zeae in inoculated wheat plots. Plant Disease 80:674-678.

Pereyra, S.A., R. Dill-Macky, and A.L. Sims. 1999. Survival and inoculum potential of Fusarium graminearum in wheat residues. p.96-99. *In* Proceedings of the U.S. Wheat and Barley Scab Initiative Annual Forum. p. 96-99.

Saur, L. 1991. Recherche de geniteurs de resistance a la fusariose de l'epi causee par *Fusarium culmorum* chez le ble et les especes voisines. Agronomie 11:535-541.

Saur, L. and M. Trottet. 1992. Heritabilite de la resistance a la fusariose de l'epi et selection recurrente dans une population de ble tendre. Agronomie 12:297-302.
Saur, L. and N. Benacef. 1993. Relation entre les symptomes de fusariose de l'epi et la perte de rendement chez le ble tendre. Agronomie 13:829-833.
Schroeder, H.W. and J.J. Christensen. 1963. Factors affecting resistance of wheat to scab caused by *Gibberella zeae*. Phytopathology 53:831-838.
Singh, R.P., H. Ma, and S. Rajaram. 1995. Genetic analysis of resistance to scab in spring wheat cultivar Frontana. Plant Disease 79:238-240.
Sinha, R.C. and M.E. Savard. 1997. Concentration of deoxynivalenol in single kernels and various tissues of wheat heads. Canadian Journal of Plant Pathology 19:8-12.
Snijders, C.H.A. 1990a. Diallel analysis of resistance to head blight caused by *Fusarium culmorum* in winter wheat. Euphytica 50:1-9.
Snijders, C.H.A. 1990b. The inheritance of resistance to head blight caused by *Fusarium culmorum* in winter wheat. Euphytica 50: 11-18.
Snijders, C.H.A. 1990c. Response to selection in F^2 generations of winter wheat for resistance to head blight caused by *Fuusarium culmorum*. Euphytica 50:163-169.
Snijders, C.H.A. and F.A. Van Eeuwijk. 1991. Genotype x strain interactions for resistance to *Fusarium* head blight caused by *Fusarium culmorum* in winter wheat. Theoretical and Applied Genetics 81:239-244.
Stack, R.W. 1999. Return of an old problem: Fusarium head blight of small grains. APSnet. http://www.scisoc.org.
U.S. Wheat and Barley Scab Initiative. 1999. Scab research initiative underway with Glickman announcement of $3 million funding boost. v.2.p.1. *In* Scab News.
Van Ginkel, Maarten, W. Van Der Schaar, Y. Zhuping, and S. Rajaram. 1996. Inheritance of resistance to scab in two wheat cultivars from brazil and china. Plant Disease 80:863-867.
Virginia Farm Bureau Newsletter. 1999. Virginia farmers ahead of nation in conservation tillage. http://www.vafb.com/news/1999/112999_3.htm.
Waldron, B.L, B. Moreno-Sevilla, J.A. Anderson, R.W. Stack, and R.C. Frohberg. 1999. RFLP mapping of QTL for Fusarium head blight resistance in wheat. Crop Science 39:805-811.
Wan, Y-F., C. Yen, and J-L. Yang. 1997. The diversity of head-scab resistance in Triticeae and their relation to ecological conditions. Euphytica 97:277-281.
Wang, Y.Z., X.N. Yong, and Q.P. Xiao. 1982. The improvement of identification technique of scab resistance of wheat and the development of resistant sources. Scientia Agricultura Sinica. 5:67-77.
Wang, Y and J. Wang. 1991. Genetical studies on the resistance to scab spread in wheat varieties. Jilin Academy of Agricultural Sciences 1:21-28.
Xu, Y.G., and Z.D. Fan. 1985. Evaluation and differentiation of pathogenicity of *Fusarium graminearum* on wheat cultivars. Acta Phytopathology Sinica 12(4):53-57.
Yang, Z.P., X.Y. Yang, and D.C. Huang. 2000. Improvement of resistance to *Fusarium* head blight by recurrent selection in an intermating breeding spring wheat population using the dominant male sterile gene ms_2. Euphytica 112:79-88.
Yu, Yu-Jun. 1982. Monosomic analysis for scab resistance and yield components in the wheat cultivar Soo-mo 3. Cereal Research Communications 10:185-189.

Yu, Yu-Jun. 1991. Genetic analysis for scab resistance in five wheat varieties, PHJZM, HHDTB, CYHM, YGFZ, and WN2. Acta Agronomica Sinica 17:248-254.

Zhou, W.C., F.L. Kolb, G.H. Bai, G. Shaner, and L.L. Domier. 2000. SSR mapping and sub-physical location of a major scab resistance QTL in wheat. p.69-73. *In* Proceedings of the 2000 National Fusarium Head Blight Forum.

CHAPTER II

Assessment and Reaction of *Triticum aestivum* Genotypes to *Fusarium graminearum* and Effects on Traits Related to Grain Yield and Seed Quality

ABSTRACT

Fusarium graminearum (Schwabe), causal organism of fusarium head blight (FHB), has become a major pathogen of wheat (*Triticum aestivum* L.) throughout North America. Since its discovery in the United States, the disease has spread south and east until at present it is an annual threat to growers of winter wheat in Virginia. Yield losses of soft red winter (SRW) wheat in Virginia averaged 908 kg ha^{-1} in the FHB outbreak of 1998 (Griffey et al., 1999). The economic loss from this single FHB epidemic was an estimated 8.5 million dollars. Total losses from FHB over the entire U.S. since 1991 have been estimated at 13.62 million metric tons, at a value as high as $2.6 billion dollars (USW&BSI Newsletter, 1999).

This study was conducted to examine all disease assessment parameters currently used to assess FHB disease levels to identify the parameters most useful in discerning resistance and predicting losses in grain yield and grain volume weight (GVW). Currently FHB incidence, FHB severity, FHB index, percentage fusarium damaged kernels (percentage FDK), GVW, and 15-acetyl deoxynivalenol toxin (DON toxin) analysis are used to quantify resistance. The second objective was to evaluate wheat genotypes commonly grown in Virginia and several others reported to have resistance or tolerance to FHB to expeditiously determine which, if any, genotypes express resistance to FHB under epidemic conditions.

Twenty (1997-98) and thirty (1998-99 and 1999-00) wheat genotypes were grown in Montgomery Co., VA and Westmoreland Co., VA (1999-00 only). Field design was a split-block with six replications evenly divided into an inoculated block and non-inoculated control block. Tests in Montgomery Co., VA were inoculated by spraying conidial suspension to genotypes according to growth stage, whereas the test in Westmoreland Co., VA was inoculated by spreading *Gibberella zeae*-colonized maize kernels. Mist-irrigation was applied post-inoculation to foster disease development. FHB field ratings were conducted 21 days post-anthesis. Grain yield, GVW, percentage FDK, and DON toxin accumulation were determined post harvest.

FHB index provided the most reliable in-field assessment of a genotype's resistance and estimate of losses in grain yield and GVW ($p \leq 0.05$ all environments) under FHB epidemic conditions. Likewise, percentage FDK provided a reliable post-

harvest measure of the aforementioned variables ($p \leq 0.0001$) in all environments. Additionally, percentage FDK was a reliable predictor of the level of DON toxin accumulated in wheat kernels ($p \leq 0.05$) in all environments. DON toxin accumulation was also highly correlated with grain yield and GVW ($p \leq 0.05$) in all environments, but its use may be restricted by cost and timely availability of data. In inoculated and irrigated trials, FHB severity alone was a dependable predictor of grain yield and GVW ($p \leq 0.05$) all environments, yet sole use of severity to assess FHB is not generally recommended, especially in non-inoculated or in non-irrigated studies.

A wide range in both level and type of resistance was observed among genotypes, which followed a continuous distribution in this study. The cultivars Agripro Patton, Ernie, INW9824, Roane, and the experimental line NY87048W-7388 consistently had low scores for FHB assessment parameters according to least significant difference (LSD) analysis and consistently had lower grain yield and GVW losses according to Duncan's Multiple Range test. Conversely, Coker 9835 and Gore had high scores in all environments for all FHB assessment parameters and sustained significant losses in grain yield and GVW.

INTRODUCTION

Gibberella zeae (Schwein) Petch (anamorph: *Fusarium graminearum* Schwabe) is an ascomycete fungus known to cause stalk and ear rot in *Zea* spp., crown rot in *Dianthus* spp., and head scab in *Triticum* spp. and *Hordeum* spp. (Bowden and Leslie, 1999). In mild continental climates, *F. graminearum* is the predominant causal organism of FHB. FHB has become a major pathogen of wheat (*Triticum aestivum* L.) throughout North America and FHB epidemics during the 1990's were devastating to producers. In Virginia, yield losses due to FHB in SRW wheat averaged 908 kg ha^{-1} in 1998 (Griffey et al., 1999). The economic loss from this single FHB epidemic was an estimated 8.5 million dollars. Total losses from FHB over the entire U.S. since 1991 have been estimated at 13.6 million metric tons, at a value of $2.6 billion dollars (USW&BSI Newsletter, 1999).

Mycelia and conidia of *F. graminearum* survive on wheat and barley residues from one season to the next under continuous cereal cultivation as well as on living and senescent maize tissues (Bai and Shaner, 1994; Stack, 1999). Although ascospores, conidia, mycelia, and hyphal fragments can all serve as sources of inoculum, ascospores and conidia are crucial in early infections due to their ability to be air or splash dispersed (Bai and Shaner, 1994; Miller et al., 1998). Fernando et al. (1997) determined that ascospores are the most efficient form of inoculum, and can be wind dispersed from 5-22 meters (spread significantly greater downwind) from a single inoculum source. However, conidia also are mobile and can incite significant disease within 5 meters of the inoculation point as well, which demonstrates that the use of conidia as inoculum in field trials is an efficient means of inducing artificial infection.

Infection of wheat spikes by *Fusarium* spp. may occur from spike emergence, Zadoks' growth stage 50 (Zadoks' 50) (Chang et al., 1974), through late milk (Zadoks' 77). However, infection occurs most frequently during anthesis (Zadoks' 60-69). Infection occurs primarily when ascospores or conidia are deposited on or within the flowering structure of the spike (McMullen et al., 1997). Prior or subsequent to flowering, infection may also occur via ascospores, mycelia, conidia, chlamydospores, or hyphal fragments deposited directly on or into the glume, rachis, or palea (Bai and

Shaner, 1994). Early infections can lead to aborted kernel development, tombstone kernels, or reduced size in mature kernels, whereas later infections often lead to symptomless kernels that can only be distinguished by toxicological screening (Stack, 1999).

Under favorable environmental conditions following infection, *F. graminearum* will colonize through the rachis and adjacent florets, growing systemically or saprophytically (Anderson, 1948; Bai and Shaner, 1994). In the case of saprophytic colonization, the fungus may spread to adjacent heads via physical contact (conidia or mycelia), splash dispersal (conidia), or wind dispersal (ascospores). Mycelia are the primary means of saprophytic growth, and appear as orange-red lesions on the rachis or glume. Systemic colonization often appears as premature senescence of the spike from the infection point to the top of the spike and downward to the culm. This senescence is a result of clogging of the vascular tissues within the rachis by mycelia (Bai and Shaner, 1994) or degradation of cell wall materials by toxins produced by the fungus (Kang and Buchenauer, 2000).

Conservation tillage, defined as a system in which more than 30 percent of crop residues are left on the soil surface, is said to be the single largest contributing factor to increased FHB levels in North America. Government mandated tillage practices for producers, increasing fuel prices, and the need to reduce topsoil loss have been the primary motives for increased conservation tillage (Bockus and Shroyer, 1998). In the Midwestern FHB outbreak of 1993, losses were estimated at $1 billion in the upper Midwest of the U.S. and Canadian plains. Surveys were collected from producers in order to correlate tillage practices with grain yield losses. It was found that producers using conservation tillage suffered significantly greater losses than those who used chisel or moldboard plowing prior to planting. For nearly every epidemic since, surveys have yielded similar results and have prompted research in the area of residue management that reduces severity of FHB (McMullen et al., 1997). Pereyra et al. (1999) determined that tillage schemes reduce yet do not completely control *F. graminearum* survival from year to year. Moldboard plowing leads to significant reductions in crop residue for *F. graminearum* to colonize and, therefore, reductions in inoculum over fallow periods. Pereyra et al. (1999) also determined that ascospores are released from perithecia for up

to three years, which implies that as buildup of debris occurs due to conservation tillage, spore release rises exponentially. This would explain the increasing frequency and severity of FHB outbreaks in areas with increased conservation tillage over the last decade.

Active host resistance to FHB has been classified into five categories or "types", each of which is believed to be independent in assessing a genotype's resistance or susceptibility. Type I resistance is described as withstanding infection, and was first described by Schroder and Christensen (1963). Type II resistance, also described by Schroder and Christensen (1963), refers to the host's ability to restrict pathogen spread within the spike once infection has occurred. Type I and II resistance has generally been measured using spike and floret inoculation, respectively, under greenhouse conditions. Under greenhouse conditions, genotype x environment effects can be minimized and resistance can be quantified. Miller et al. (1985) described type III resistance as the host's ability to block the accumulation of mycotoxins within infected kernels. Measurement of type III resistance is performed by chemical analysis of grain samples. However, Mesterhazy et al. (1999) reported wide variation in type III resistance in genotypes between environments, as a result of genotype x isolate x environment interactions, which are complex and not well understood. Type IV resistance, described by Mesterhazy (1995), refers to the host's ability to maintain sound kernels in colonized florets, thus reducing GVW loss. Measurement of type IV resistance has been accomplished by visually assessing the percentage FDK within a sample or comparison of GVWs from inoculated and non-inoculated plots. Mesterhazy first described type V resistance in 1995 as the host's ability to maintain grain yield under infection. Measuring this resistance type has been accomplished by comparison of grain yields from inoculated versus non-inoculated plots (Mesterhazy et al., 1999).

Active (physiological or biochemical) resistance includes all resistance types discussed above and is the focus of this research. Breeding efforts throughout the world are focused primarily on type II resistance. Reports have indicated that under epidemic conditions type I resistance is overcome, and thereafter type II resistance becomes the most promising line of defense (Mesterhazy, 1995). Less attention has been given to

resistance types III, IV, and V due to the ability to quickly quantify type II resistance in greenhouse studies.

It is widely accepted that the best solution for controlling the complex problem of FHB throughout the world is the introgression of resistance into elite adapted genotypes. Substantial progress has been made in breeding for FHB resistance, especially during the last decade (Bai and Shaner, 1994; McMullen et al., 1997). However, to date no source with complete resistance has been found (Ban, 1997). The best-known resistance sources have come from Chinese, Japanese, and Brazilian spring wheats (Bai and Shaner, 1994; Ban and Suenaga, 2000; Singh et al., 1995; Snijders, 1990a; Wang and Wang, 1991; Yu, 1991). China suffers from annual FHB epidemics throughout the nation, and it is postulated that natural selection has favored those genotypes exhibiting resistance. This is especially true in the Yangtze Valley, where FHB outbreaks are often severe. Breeding efforts in China have resulted in the release of FHB resistant type II cultivars Sumai 3, Ning 7840, Ning 8026, W14, Shaan 85, Fan 1, Futai 8944, and Futai 9002 (Wang and Wang, 1991; Bai and Shaner, 1994). Additionally, Japanese wheat lines Nobeokabozu Komugi, YFGZ, and Saikai 165 are reported to carry type II resistance (Yu, 1991; Snijders, 1990a; Ban and Suenaga, 2000). These type II resistance sources and Frontana, a type I resistance source, are not adapted in the U.S. Efforts are underway to introgress resistance from these lines into elite genotypes, yet only a few genotypes have been released with resistance from these parents. This is due to poor combining ability of these genotypes with respect to yield and quality. Progeny derived from crosses with these resistant genotypes are also tall, later maturing, and have fewer florets per head, which are unfavorable characteristics (Bai and Shaner, 1994). Sumai 3 and its progeny possess the best available combining ability for FHB resistance in association with yield and quality related traits and are being widely used as parental sources of FHB resistance. This has raised questions regarding reliance on resistance from Sumai 3 and its progeny as the primary genetic source and possible erosion of resistance (Bai and Shaner, 1994).

Type IV and V resistance have been defined as the ability of a diseased genotype to retain sound kernels and yield, respectively. Type IV and V resistances are difficult to measure due to inconsistent disease levels from year to year in field trials, which provide

the only accurate measurement. However, with improved inoculation methods, Mesterhazy et al. (1999) have proven that there are significant cultivar differences in type IV and V resistance levels to FHB. An inherent problem in incorporating type IV and V resistance into a breeding program is the inability to accurately assess such resistance, identify genotypes with stable resistance, and identify the genes conferring this resistance. Mesterhazy et al. (1999) concluded that genotypes exhibiting type IV and V resistance occur much more frequently than those exhibiting type I and II resistance and that inheritance of type IV and V resistance conformed to an additive-dominance model.

GVW is an important parameter used in assessing resistance or tolerance of a genotype to FHB. Negative correlations between FHB and GVW have been reported by Saur (1991), Saur and Trottet (1992), and Saur and Benacef (1993). Studies have repeatedly shown that FHB reduces GVW in two ways. Kernels within infected and colonized florets appear as pink, chalky white, or pale gray shriveled seed with little or no endosperm. These seeds will significantly reduce GVW of a sample, even when they only account for a small percentage of the sample (Jones and Mirocha, 1999). The second cause of grain-volume weight reduction occurs without direct kernel colonization. When the rachis becomes blocked by mycelia, premature senescence of the florets located above and below the point of colonization results in kernels showing no symptoms. However, these seeds are less dense due to reduced nutrient uptake as a result of vascular blocking (Bai and Shaner, 1994).

Yield is also affected by FHB in two ways. Kernels located in florets that have been infected and colonized by *F. graminearum* are often expelled with chaff during harvest. In highly susceptible genotypes this can amount to 30 to 70 percent of kernels (Bai and Shaner, 1994; Miedaner, 1997). Kernels that have not been colonized by *F. graminearum*, yet suffer from reduced size and density as a result of vascular blocking, also decrease yield by reducing overall grain-volume weight (Bai and Shaner, 1994).

The primary mycotoxin produced by *F. graminearum* in the heads of wheat is DON toxin (Miedaner, 1997). This toxin is produced in all parts of the spike and was observed to be most abundant in the rachis, followed by the glume, and least concentrated in the kernels (Sinha and Savard, 1997). Sinha and Savard (1997) measured DON toxin levels in various wheat kernels from colonized spikes, including normal looking kernels,

kernels of normal color but reduced in size, and kernels that showed visible symptoms such as pink or chalky appearance. Their results were surprising, in that 50 percent of apparently normal or reduced-size kernels had detectable DON toxin concentrations, although typically at 5 ppm or less. DON toxin was detected in all kernels with visible infection, but the range of DON toxin concentration varied greatly from 1 ppm to 600 ppm. These results concur with those obtained by Mesterhazy et al. (1999), Jones and Mirocha (1999), and Miedaner (1997).

The current study had two research objectives. The first objective was to examine all disease assessment parameters currently used to assess FHB and identify the parameters most useful in discerning resistance and predicting losses in yield and quality. Identification of the most precise and time-efficient in-field assessment parameter will be useful to producers in scouting for FHB disease levels and quantifying losses prior to harvest. For breeders, identifying both in-field and post-harvest assessment parameters which best quantify resistance and losses in yield and quality will provide a means of saving valuable time when assessing large numbers of experimental lines. Currently FHB severity, FHB incidence, FHB index, percentage FDK, GVW, 1000 kernel weight (KW), and toxin analysis are used to quantify resistance.

The second objective was to examine wheat genotypes commonly grown in Virginia to expeditiously determine which, if any, genotypes expressed resistance or tolerance to FHB under epidemic conditions. In addition, several genotypes not adapted to Virginia, yet reported to have resistance or tolerance to FHB, were examined to ascertain performance under epidemic conditions in Virginia.

MATERIALS AND METHODS

Winter Wheat Genotypes and Experimental Lines. Genotypes evaluated in this study can be grouped into two classes. The first class was comprised of adapted SRW wheat experimental lines and cultivars commonly grown by producers in the Mid-Atlantic region (VA, MD, NC, SC, TN, and KY). These genotypes included the cultivars: Agripro Foster, Agripro Mason, Agripro Patton, Coker 9803, Coker 9835, FFR 555W, Gore, Jackson, Madison, Pioneer 2552, Pioneer 2580, Pioneer 2643, Pioneer 2684, Pocahontas, Quantum 706, Roane, SS 550, Sisson, Wakefield, and experimental lines VA96W-326, VA96W-329, and VA96W-348. The second class was comprised of genotypes with known or suspected resistance to FHB and included cultivars Cayuga, Ernie, Freedom, and INW 9824, and experimental lines IL94-1909, NY8704W-7388, and OH 552. In total twenty-nine genotypes were evaluated for reaction to FHB in tests conducted during the 1997-98 through 1999-2000 seasons.

Test Sites and Cultural Practices. Field trials were conducted during the 1997-98 through 1999-00 seasons in Montgomery County, VA and in the 1999-00 season in Westmoreland, County, VA. Soils at the Montgomery County location are classified as a Hayter fine loam (fine loamy, mixed mesic ultic, and hapludalf). The location is within the ridge and valley region of the state and located in USDA hardiness zone 6a. Westmoreland County, Virginia was selected as an additional test site due to its physiographic location, in one of the major wheat production areas, which is within the coastal plain of Virginia. Soil at this location is classified as a State fine sandy loam (fine loamy, mixed, semiactive thermic, and typic hapludult). This location is within USDA hardiness zone 7b.

Field trials in each environment were comprised of six replications of each genotype, divided into two treatments containing three replications each (Appendix A). Each plot measured 30.48 m^2 (100 ft^2) and consisted of seven rows, each separated by 18 cm (7 in) in Montgomery, Co., VA and six rows, each separated by 15.3 cm (6 in) in Westmoreland Co., VA. A split-block design was used in all tests and consisted of genotypes as the main block factor and treatments as the sub-block factor. One block, comprised of three replications of each genotype, was inoculated with *F. graminearum*,

while the other block, comprised of three replications of each genotype, was not inoculated and served as the control. The treated and non-treated blocks were separated by a 8.84 m triticale border, which served as a buffer to limit spread of spores from inoculated to non-inoculated blocks.

Planting density was based upon 1000 kernel weight with a target density of 79 seeds/m drill row (24 seeds/ft drill row). Seed of genotypes used for planting each test originated from a single seed source. Prior to planting seed was treated with triadimenol (Baytan 30F, 12.75 g ai/45.36 Kg), imidacloprid (Gaucho 480F, 25.50 g ai/45.36 Kg), and phthalimide (Captan 50WP, 35.41 g ai/45.36 Kg). Triadimenol was applied to control powdery mildew [caused by *Blumeria graminis* f. sp. *Tritici* (DC.) E.O. Speer], imidacloprid to control aphids and therefore Barley Yellow Dwarf Virus, and phthalimide to control seedborne fungi.

Pre-plant fertilizer applications included 25N-60P-90K (1997-98), 25N-100P-100K (1998-99), 25N-50P-100K (1999-00, Montgomery Co., VA), and 30N-40P-60K (1999-00, Westmoreland Co., VA). Plots were planted using a Hege seven-row planter (15.3 cm between rows) in Montgomery Co, VA and an Almaco six-row planter (17.8 cm between rows) in Westmoreland Co., VA. Spring nitrogen was applied at Zadoks' 30 in Montgomery Co., VA at a rate of 67.24 Kg ha^{-1} (1997-98; 1998-99) and 84.06 Kg ha^{-1} (1999-00) with an application of Harmony Extra herbicide (35.1 ml ha^{-1}) in all tests. Spring nitrogen was split into two applications in the Westmoreland Co., VA test, the first application of 44.9 Kg ha^{-1} was conducted at Zadoks' 25 with an application of Harmony Extra herbicide (35.1 ml ha^{-1}) and the second application of 73.0 Kg ha^{-1} at Zadoks' 30. Presence of other diseases in addition to fusarium head blight in the 1998-99 test year necessitated the monitoring of leaf rust (caused by *Puccinia triticina* f. sp. *tritici* Eriks.), glume blotch [caused by *Staganospora nodorum* (Berk.) Castellani & E.G. Germano], take-all (caused by *Gaeumannomyces graminis* var. *tritici*), and powdery mildew. In an effort to control all fungal diseases except for FHB, triadimefon (Bayleton 50DF, 214 g ai ha^{-1}) was applied to tests conducted in the 1999-00 season from seven days post anthesis (Zadoks' 71) through the end of the milk stage (Zadoks' 77). This treatment was repeated at two-week intervals as needed to control diseases other than FHB.

Plots were harvested using a small plot combine during the first week of July in Montgomery Co., VA and the last week of June in Westmoreland Co., VA. To retain fusarium damaged kernels, blower levels of the combine were adjusted so that diseased kernels were not expelled with chaff. At harvest, grain from all plots was individually bagged and retained for further analyses, which included grain yield, GVW, percentage FDK, and DON toxin accumulation.

Inoculum Production and Inoculation Procedures. *F. graminearum* was isolated from colonized wheat kernels obtained from several locations throughout Virginia and cultured to produce inoculum. The most aggressive isolates were selected by growing cultures on half-strength potato dextrose agar (PDA) and measuring growth of the colony. The isolates with the most rapid growth on agar plates were selected and used to produce inoculum in all years. Conidia were produced by plating mycelia or conidia on half-strength PDA (pH 4.0-4.5) plates that were incubated at 20-25° C under a 12-hour photoperiod provided by full-spectrum growth lights. Conidia were harvested by washing them from the plates using sterile water. Tween 20 was added to the spore suspension at a rate of 1:100. These suspensions were stored at 2-4° C. At the time of inoculation, spore suspensions were diluted to a concentration of 5×10^4 spores ml^{-1} and applied directly to plots by spraying wheat spikes in the Montgomery Co., VA tests. Plots of each genotype were inoculated at the Montgomery Co., VA location at heading (Zadoks' 59) and again at fifty-percent anthesis (Zadoks' 65) with a 0.0108 L m^{-2} conidial suspension at 50,000 spores ml^{-1}. To reduce drift and facilitate proper timing of application, all conidial suspensions were applied using a CO_2 pressurized backpack sprayer with 80110 TeeJet Flatfan tips at 45 psi. Additionally, all applications were performed from 0800 hours to 1030 hours to reduce drift.

Test plots in Westmoreland Co., VA (1999-00) were inoculated by spreading *Gibberella zeae* colonized maize kernels, to negate the need for continuous monitoring of each genotype's growth stage and daily inoculation of plots based upon growth stage. Production of this inoculum was carried out by placing 500g of autoclaved maize kernels in a No. 2 US quart Mason jar with 100-200 ml conidial suspension. Jars were covered using antibacterial and antifungal filter paper to allow proper airflow and grown at room

temperature for 14 days. Jars were shaken twice per week to loosen maize kernels and ensure adequate aeration. At the end of this period, colonized maize kernels were air dried and weighed into 454-gram samples for application within plots. Colonized maize seed was spread into inoculated plots at a rate of 39.1 g m^{-2} at Zadoks' 37-41.

Each location received overhead mist irrigation following inoculation, to promote disease development. Irrigation, in the form of non-chlorinated surface water, was applied from 0800 hours to 0930 hours and again from 1800 hours to 1930 hours unless environmental conditions deemed irrigation unnecessary (rain, dense fog, or heavy dew). Irrigation was supplied using Wade-Rain 7.62cm aluminum main line pipe with 165.1cm risers placed on 3.05m centers. Spinner-type heads were installed onto risers to provide evenly distributed coverage with minimal drift at 35psi and 4.09 liters per minute.

Disease Assessment and Post-Harvest Analyses. Field assessments conducted over all years included heading date, flowering date, head type, and plant height at maturity. Disease ratings of barley yellow dwarf virus, leaf rust, glume blotch, take-all, and powdery mildew were assessed as warranted by significant disease incidence and severity as in 1989-99 when triadimefon was not applied to test plots. Disease data were used to determine possible correlation and interactions with FHB, grain yield, and GVW loss. Post-harvest measurements and calculations included seed moisture, plot weight (grams), GVW, and grain yield. A Dickey-John grain-analysis machine (DICKEY-john Corp., Auburn, IL) was used to determine seed moisture and GVW. Plot weight was obtained by weighing the entire seed sample harvested from each plot. Yield was calculated on the basis of plot weight adjusted to 13.5 percent moisture and the standard (60 lb bu^{-1}; 67.19 kg hl^{-1}) GVW for SRW wheat.

Field assessments of *F. graminearum* included FHB incidence, FHB severity, and FHB index recorded for each plot at twenty-one days post inoculation. Within each plot, two arbitrary samples of fifty spikes were isolated and loosely taped together to form two bundles of spikes that were evaluated for FHB incidence and severity. FHB incidence is a measurement of the percentage of diseased heads within the plot. The number of heads having one or more florets colonized with *F. graminearum* were counted in each bundle. The same two bundles in each plot were rated at 14 and 21 days. FHB severity is a

measurement of the percentage of colonized florets per spike. Within each plot, an average number of florets per spike were determined by counting floret numbers from three to five spikes and averaging this number. Then, utilizing the same 100 spikes bundled to calculate FHB incidence, the number of infected florets per spike was counted. FHB severity was calculated using ten randomly selected spikes per bundle, due to time required to conduct FHB severity ratings. The number of diseased florets per head was then divided by mean number of florets per head of a given cultivar to procure an average number of diseased florets. The resulting value is FHB severity. FHB index is obtained for each plot by multiplying FHB incidence and FHB severity and multiplication of the product times 100. It is a single value used by researchers to assess FHB and combines FHB incidence and FHB severity.

Post harvest assessment of FHB included determination of percentage fusarium damaged kernels (FDK), DON toxin concentration, and calculation of GVW and grain yield loss. Percentage FDK was determined by randomly counting 200 seeds from a sample of each plot. Among these 200 seeds, the number of seeds showing FHB symptoms, which include pink or white discoloration, reduced or shriveled appearance, or both was determined and percentage of diseased versus non-diseased kernels was calculated as [(diseased seed/total seed)*100]. GVW loss and yield loss, expressed as a percentage, were calculated as [(GVW or yield of inoculated plot/GVW or yield of non-inoculated plot)*100]. In calculating losses for each genotype, one plot from inoculated and non-inoculated blocks were paired, repetitions 1 and 4, 2 and 5, and 3 and 6. Thus, three values for yield loss and grain volume-weight loss were obtained per genotype per environment. DON toxin is produced by *F. graminearum* in infected plant tissue, including the kernels. Michigan State University, using enzyme-linked immunosorbent assay (ELISA), analyzed a 500g seed sample from each plot for DON toxin concentration (ppm).

Data Analysis. All tests were analyzed as a split block design with 20 entries in 1997-98 and 30 entries in 1998-99 and 1999-00. Each test included an inoculated and non-inoculated treatment block, each containing three replications. Data sets from each treatment (block) included three values per genotype for yield, GVW, FHB incidence,

FHB severity, FHB index, percentage FDK, DON toxin accumulation, plant height, heading date, and flowering date.

Genotypes evaluated in less than three environments were not included in the statistical analysis over environments. Effects and interactions of years, treatments, replications, and entries were calculated using general linear model (GLM) procedures, due to differing number of genotypes in each environment and differing genotypes between environments. Interactions and mean squares within environments for treatments and entries were determined using the ANOVA procedure, due to uniform data sets in each environment. To assess the performance of each genotype, in each environment, and within the treated block, LSD values were obtained to group genotypes at $P = 0.05$ with regard to FHB disease assessment parameters.

Three values for percent yield loss and percent GVW loss were calculated for each environment. Grain yields and GVWs were paired by entry using the fixed model: replication 1 versus 4, 2 versus 5, and 3 versus 6 in order to derive three percent losses for each entry in each environment. Statistical analysis of the percent losses required transformation of data as illustrated by Gomez (1984). Data sets in which all values fell exclusively between 30.0-70.0 percent required no transformation. Data sets which included values between 0.0-30.0 percent or 70.0-100.0 percent were transformed by taking the square root of the percentage. Data sets with values spanning two or more of the aforementioned ranges were transformed by taking the arc sin of the percentage. Transformed data sets were analyzed using ANOVA procedures and means were separated into groups using Duncan's Multiple Range test at $P = 0.05$.

Correlation analysis was performed separately for each environment to procure relationships between disease assessment parameters, grain yield, and GVW. Correlation analysis was also used to determine if associations exist between other diseases and lodging with grain yield and GVW.

RESULTS

Year, Treatment, Entry, and Interactions Between and Within Years. Year, treatment, entry, and entry within treatment differences were all significant ($P < 0.0001$) for all FHB assessment parameters, grain yield, and GVW over all environments combined (data not shown). Thus, significant environmental effects and interactions as well as a lack of complete homogeneity of variances restricted data from being pooled over environments. Hence, statistical analyses were performed for each environment separately. Significant variation among environments is common in tests involving plant diseases due to pathogen variation and genotype x environment interactions.

Entry and treatment mean squares were significant ($P \leq 0.05$) for all variables in each environment (Table 1). The 1997-98 Montgomery Co., VA and 1999-00 Westmoreland Co., VA environments were the only ones in which significant differences were not observed for all variables among entries within treatments. In order to elucidate whether entries in the 1997-98 Montgomery Co., VA test also performed similarly within both inoculated and non-inoculated blocks with respect to GVW and FHB incidence, treatments were analyzed separately. In this analysis, entry differences were significant ($P \leq 0.01$) for grain yield, GVW, and all FHB assessment parameters in the inoculated block (data not shown). In the 1999-00 Westmoreland Co., VA test, significant yet variable disease levels were observed in the non-inoculated block and is a probable explanation for the lack of overall entry within treatment differences for grain yield, FHB severity, percentage FDK, and DON toxin accumulation. When the two blocks were analyzed separately, entry differences were significant ($P \leq 0.001$) for grain yield, GVW, and all FHB assessment parameters in the inoculated block (data not shown). Variability in disease levels within the non-inoculated block was high, with disease levels higher in the plots adjacent to the inoculated block. Lack of significant difference among entries in the non-inoculated block was observed for grain yield, GVW, and FHB assessment parameters. Unlike the Montgomery Co., VA tests where inoculations were conducted by spraying conidia, the test in Westmoreland Co., VA was inoculated by spreading *Gibberella zeae*-colonized maize kernels that released ascospores. Ascospores are a more mobile form of spore than conidia and also are continuously released from the colonized maize kernels. This resulted in longer periods of exposure of spikes to spores than from

direct spraying of conidia, in which spikes are exposed only once per application. Considering the volume of maize kernels applied, it is likely that ascospores traveled via wind from the inoculated plots to the non-inoculated control plots in high enough volume to create significant disease. This phenomenon would explain the level of disease declining as distance from the source increased.

Effects of *Staganospora nodorum* and Lodging on FHB Assessment, Grain Yield, and Grain Volume Weight. In the 1998-99 Montgomery Co., VA test, *Staganospora nodorum* and lodging had confounding effects on the assessment and impact of *F. graminearum* on grain yield and GVW (Tables 2,3). Lower correlation values in the 1998-99 test likely resulted from significant and differential grain yield and GVW losses caused by *S. nodorum* and lodging. *S. nodorum* had a confounding effect on both FHB assessment and grain yield, while lodging confounded grain yield and GVW. A significant ($p \leq 0.0001$) amount of variation in grain yield ($r^2 = 0.23$) was due to *S. nodorum*. Lodging accounted for significant ($p \leq 0.0001$) variation in grain yield ($r^2 = 0.25$) and GVW ($r^2 = 0.17$). *S. nodorum* also was associated with FHB incidence ($p \leq 0.05$) and percentage FDK ($p \leq .01$) in this environment. Although r^2 values were below 0.10 for both of the aforementioned associations (Table 3), any variation in FHB assessment, which can be attributed to *S. nodorum*, undoubtedly had an effect on FHB disease assessment results. Simultaneous occurrence of both spike diseases likely decreased the precision of FHB field assessments. *S. nodorum* may also confound the identification of percentage FDK in grain samples due to kernel shriveling caused by both diseases. The significant ($p \leq .01$) association between *S. nodorum* and percentage FDK ($r^2 = 0.08$) in the current study implies that DON toxin analysis may provide a better assessment of FHB levels than percentage FDK in years when high levels of both *S. nodorum* and *F. graminearum* are observed.

Performance of entries, with regard to FHB assessment parameters, grain yield, and GVW in the 1998-99 Montgomery Co., VA test often differed from that observed in the other environments. This variation in entry performance can be attributed largely to confounding effects due to *S. nodorum* and lodging (Table 3). The variation in grain yield ($r^2 = 0.02 – 0.04$) and GVW ($r^2 = 0.06 – 0.16$) attributed to preharvest FHB assessment

parameters (Table 2) were consistently lower in this environment compared to other environments and indicates that assessments of FHB were confounded by *S. nodorum*. Additionally, little variation was observed among genotypes for mean grain yield loss and GVW loss on the basis of Duncan's Multiple Range test (data not presented). This can be attributed to the confounding effects of both lodging and *S. nodorum* (Table 2). For these reasons, data from the 1998-99 Montgomery Co., VA test is not included in subsequent evaluations of FHB assessment parameters or genotype performance. The aforementioned data is presented to confirm that *S. nodorum* and lodging greatly impact and confound results obtained in FHB trials. In subsequent field trials, triadimefon was applied at two-week intervals post-anthesis as recommended by Mesterhazy et al. (1999), to control *S. nodorum* without affecting FHB. Additionally, ethephon was applied at Zadoks' 47 to reduce lodging effects.

Association of FHB Assessment Parameters and Phenotypic Traits to Grain Yield and Grain Volume Weight. Plant height, heading date, and flowering date were not correlated with FHB incidence, FHB severity, FHB index, percentage FDK, DON toxin accumulation, grain yield, or GVW in any environment (data not shown). It can be assumed that precise timing of conidial suspension applications to each genotype based upon growth stage resulted in uniform disease and minimized escape or mechanical resistance in the Montgomery Co. tests. The lack of correlation between the aforementioned variables in the Westmoreland Co. test implies that use of *Gibberella zeae*-colonized maize, at a high rate, provided for a sufficient level of disease over the duration of the flowering period of all genotypes and, therefore, minimized escape and mechanical resistance.

Correlation analysis of data from each environment, excluding the 1998-99 Montgomery Co., VA test, was conducted for grain yield and GVW with disease assessment parameters, which included FHB incidence, FHB severity, FHB index, percentage FDK, and DON toxin accumulation (Table 2). FHB severity and FHB index were comparable in their reliability and accuracy in predicting losses. FHB severity accounted for 24 to 70 percent of the variation in yields and 34 to 74 percent of the variation in GVWs ($p \leq 0.001$). FHB index accounted for 35 to 74 percent of the

variation in grain yields and 44 to 75 percent of the variation in GVWs. FHB incidence was significantly ($p \leq 0.001$) and negatively correlated with grain yield and GVW, however r^2 values were generally lower than those for FHB severity and FHB index. FHB incidence accounted for 27 to 55 percent of variation in grain yields and 29 to 58 percent of the variation in GVWs.

Percentage FDK was significantly ($p \leq .0001$) and negatively correlated with grain yield and GVW, and accounted for 25 to 52 percent of variation observed in grain yields and 44 to 69 percent of variation in GVWs (Table 2). DON toxin accumulation was also correlated ($p \leq .01$) with grain yield and GVW in all three environments. DON toxin accumulation explained 12 to 56 percent of the variation in grain yields and 7 to 61 percent of the variation in GVWs. The lower r^2 values for DON toxin accumulation versus percentage FDK imply that percentage FDK may have been a more reliable post-harvest measurement of FHB disease levels in cases where *S. nodorum* was not present.

DON toxin accumulation was negatively correlated ($p \leq .01$) with both grain yield and GVW over all environments, yet its reliability in explaining variation in these FHB-dependent variables varied greatly (Table 2). This, coupled with cost of sample analysis and delayed availability of data, may restrict its use in early generation selection. Although r^2 values generally were low to moderate (0.07 – 0.61), their significance implied that genotypes which are prone to higher grain yield and GVW losses are also more likely to accumulate DON toxin.

Association of Genotypes and FHB Assessment Parameters. Means for FHB assessment parameters from inoculated blocks were calculated for each entry. Genotypes were separated into groups on the basis of least significant differences between means ($p = 0.05$), in order to delineate the most resistant and most susceptible genotypes on the basis of each FHB assessment parameter (Table 4). Coefficient of variance (CV) for FHB index was moderately high (53.44) in the 1997-98 Montgomery Co., VA test. However, this elevated CV value can be attributed to the variation within FHB incidence and FHB severity, which are used to calculate FHB index.

FHB index and percentage FDK were the two most consistent and reliable disease assessment parameters with regard to explaining variation in grain yield and GVW under

FHB epidemic conditions. Those genotypes with a consistently low percentage FDK and FHB index over multiple environments likely possess the best and most stable overall resistance. FHB severity was also a reliable means of ascertaining resistance levels and grain yield and GVW losses in inoculated and irrigated yield trials.

1997-98 Montgomery Co., VA Test. FHB index means ranged from 5.2 to 48.0 with an overall mean FHB index value of 17.8 (Table 4). Ernie, INW 9824, Roane, Coker 9803, Freedom, Wakefield, and Pioneer 2580 had distinctly low FHB index values (5.2 – 13.7), whereas Gore had a distinctly high FHB index value (48.0). FHB severity means ranged from 0.16 to 0.59 with an overall mean of 0.27. Ernie, INW 9824, Roane, Coker 9803, and Freedom had the lowest FHB severity values (0.16 – 0.22), while Gore had a distinctly high FHB severity value (0.59). FHB incidence means ranged from 28.3 to 80.0 with an overall mean of 54.4. Ernie was the only genotype having a distinctly low FHB incidence (28.3), whereas Coker 9835 had a distinctly high FHB incidence value (80.0). Percentage FDK means ranged from 8.5 to 32.5 with an overall mean of 18.9. Ernie, INW 9824, Roane, and Agripro Foster had distinctly low percentage FDK values (8.5 – 12.7), while Coker 9835 and Gore had distinctly high percentage FDK values (31.3 – 32.5). DON toxin means ranged from 4.62 ppm to 12.92 ppm with an overall mean of 7.80 ppm. Ernie, INW 9824, Roane, Coker 9803, Freedom, and Agripro Foster had distinctly low DON toxin values (4.62 – 6.10), whereas Coker 9835 and Gore had distinctly high DON toxin values (11.28 – 12.92). Considering all FHB assessment parameters, Ernie was the most resistant genotype in this environment, followed by INW 9824 and Roane, while Gore and Coker 9835 were the most susceptible genotypes.

1999-00 Montgomery Co., VA Test. FHB index means ranged from 9.1 to 68.7 with an overall mean of 30.9 (Table 4). Ernie, Agripro Patton, NY 87048W-7388, INW 9824, Roane, OH 552, and Pioneer 2552 had distinctly low FHB index values (9.1 – 22.6), whereas Gore had a distinctly high FHB index value (68.7). FHB severity means ranged from 0.16 to 0.69 with an overall mean of 0.36. Ernie, Agripro Patton, NY87048W-7388, INW 9824, IL94-1909, Roane, Coker 9803, OH 552, and Pioneer 2552 had distinctly low FHB severity values (0.16 – 0.28), whereas Gore had a distinctly

high FHB severity value (0.69). FHB incidence values ranged from 52.7 to 99.0 with an overall mean of 83.6. Ernie, Agripro Patton, and INW 9824 had distinctly low FHB incidence values (52.7 – 64.7). Conversely, Gore, Coker 9835, Pocahontas, Pioneer 2643, FFR 555W, SS 550, Sisson, Jackson, Pioneer 2684, Pioneer 2580, VA96W-348, Agripro Foster, Freedom, Roane, IL 94-1909, and NY87048W-7388 had distinctly high FHB incidence values (85.0 – 99.0). Percentage FDK ranged from 30.0 to 89.7 with an overall mean of 57.8. NY87048W-7388, IL94-1909, Roane, and Quantum 706 had distinctly low percentage FDK values (30.0 – 36.7), whereas Gore, Coker 9835, Pioneer 2643, Madison, and Pioneer 2580 had distinctly high percentage FDK values (84.2 – 89.7). DON toxin means ranged from 0.48 ppm to 15.93 ppm with an overall average of 5.43 ppm. Agripro Patton, NY87048W-7388, OH 552, VA96W-326, and Agripro Mason had distinctly low DON toxin values (0.48 – 1.49), whereas Gore had a distinctly high DON toxin value (15.93). On the basis of all FHB assessment parameters, Agripro Patton and NY87048W-7388 were the most resistant genotypes, followed by Ernie, INW 9824, Roane, and OH 552. Gore was the most susceptible cultivar.

1999-00 Westmoreland Co., VA Test. FHB index means ranged from 7.5 to 80.7 with an overall mean of 36.4 (Table 4). NY87048W-7388, IL94-1909, Cayuga, and Freedom had distinctly low FHB index values (7.5 – 15.5), whereas Coker 9835 had a distinctly high FHB index value (80.7). FHB severity ranged from 0.16 to 0.81 with an overall mean of 0.39. Agripro Patton, NY87048W-7388, IL94-1909, Cayuga, Freedom, Agripro Foster, and Quantum 706 had distinctly low FHB severity values (0.16 – 0.25), whereas Coker 9835 had a distinctly high FHB severity value (0.81). FHB incidence ranged from 47.0 to 100.0 with an overall mean of 87.6. NY87048W-7388 and IL 94-1909 had distinctly low FHB incidence values (47.0, 56.3). Conversely, Gore, Coker 9835, Pocahontas, Pioneer 2643, FFR 555W, SS 550, Sisson, Jackson, Pioneer 2684, Madison, Pioneer 2580, VA96W-348, Agripro Mason, Pioneer 2552, OH 552, and Roane had distinctly high FHB incidence values (91.7 – 100.0). Percentage FDK ranged from 10.2 to 84.5 with an overall mean of 49.1. Agripro Patton, NY87048W-7388, INW 9824, IL94-1909, Roane, and Quantum 706 had distinctly low percentage FDK values (10.2 – 22.5), whereas Gore, Coker 9835, Pioneer 2643, FFR 555W, Madison, and Pioneer 2580

had distinctly high percentage FDK values (76.7 – 84.5). DON toxin means ranged from 0.30 ppm to 5.79 ppm with an overall mean of 2.16 ppm. Agripro Patton, NY87048W-7388, INW 9824, IL94-1909, Coker 9803, Cayuga, Freedom, and Agripro Foster had distinctly low DON toxin values (0.30 – 0.96), whereas Gore had a distinctly high toxin value (5.79). Overall, NY87048W-7388 and IL94-1909 were the most resistant genotypes, followed by Agripro Patton, Cayuga, and Freedom. Coker 9835 and Gore were the most susceptible genotypes.

Genotypes Expressing FHB Resistance Over Multiple Environments. Ernie was the most resistant genotype examined on the basis of FHB index, ranking first with mean FHB index values of 5.2 and 9.1 in the 1997-98 and 1999-00 Montgomery Co., VA environments, respectively. Roane, INW 9824, NY87048W-7388, and Freedom also had distinctly low mean FHB index values in two of three years examined (Table 4).

In two environments, Ernie also was the most resistant genotype on the basis of FHB severity, with mean FHB severity values of 0.16 and 0.17 in the 1997-98 and 1999-00 Montgomery Co., VA environments, respectively. Other genotypes which consistently had low FHB severity means in two environments were NY87048W-7388, INW 9824, Roane, Coker 9803, Freedom, and IL94-1909. These genotypes, which consistently had low FHB severity values, can be inferred as having type II resistance.

The only genotype having distinctly low mean FHB incidence in two environments was Ernie, which had mean FHB incidence values of 28.3 and 52.7 in the 1997-98 and 1999-00 Montgomery Co., VA environments, respectively. The low FHB incidence values for Ernie indicate that this genotype exhibits a level of type I resistance greater than that of other genotypes in this study. This finding was not expected in that Ernie has not been reported to exhibit type I resistance in greenhouse screenings.

Roane was the only genotype that exhibited a distinctly low percentage FDK in three environments, having mean values of 12.2, 34.2, and 22.5 percent. Agripro Patton, NY87048W-7388, Quantum 706, INW 9824, and IL94-1909 had distinctly low values for percentage FDK in two environments. It can be inferred that these genotypes possess type IV resistance, which is the ability of a genotype to retain sound kernels when infected and colonized. Coker 9803, Agripro Patton, and NY87048W-7388 had distinctly

low DON toxin values over two environments. It can be assumed that these genotypes possess a level of type III resistance, which restricts DON toxin accumulation in infected kernels.

The five most resistant genotypes examined based on mean values for FHB assessment parameters, grain yield loss, and grain volume weight loss over all environments (Table 5) were Ernie, NY 87048W-7388, Agripro Patton, INW 9824, and Freedom. Coker 9835 and Gore consistently had high overall mean values for the same parameters, which indicate that these genotypes lack any FHB resistance.

Grain Yield and Grain Volume Weight Losses in Tested Genotypes. A continuous distribution was observed among genotypes for grain yield and GVW in all environments. In order to standardize loss values, percentage loss was calculated for genotypes in each environment (Table 6). A significant difference in percent GVW loss was not observed ($p = 0.4613$) among genotypes in the 1997-98 Montgomery Co., VA test.

Grain yield losses ranged from 3.3 to 48 percent in the 1997-98 Montgomery Co., VA test with an overall mean loss of 24.0 percent (Table 6). In the 1999-00 Montgomery Co., VA test, grain yield losses ranged from 7.1 to 49.0 percent with an overall mean loss of 25.1 percent. In the 1999-00 Westmoreland Co., VA test grain yield losses ranged from –11.8 to 23.5 percent with a mean loss of 9.5 percent. The lower losses observed in the 1999-00 Westmoreland Co., VA environment were due to a significant incidence of FHB in the non-inoculated block. However, significant ($p \leq 0.05$) entry differences were observed for grain yield in both the inoculated and non-inoculated block. Ernie, INW 9824, Roane, and Freedom had low grain yield losses in all three environments in which they were tested. Likewise, Agripro Patton, NY87048W-7388, IL94-1909, and OH 552 had low grain yield losses in the two environments in which they were tested. The assumption can be made that these genotypes possess type V resistance, on the basis of their ability to retain grain yield when infected by *F. graminearum*. Conversely, Gore, Coker 9835, and FFR 555W had high grain yield losses in three environments, and it can be assumed that these genotypes possess little or no type V resistance to FHB.

GVW losses ranged from 1.8 to 22.5 percent in the 1999-00 Montgomery Co., VA environment with an overall mean of 9.3 percent (Table 6). In the 1999-00 Westmoreland Co., VA environment GVW losses ranged from –4.7 to 14.0 percent with an overall mean of 5.0 percent. The lower loss values observed in the 1999-00 Westmoreland Co., VA environment were once again due to a significant incidence of FHB in the non-inoculated block. Significant ($p \leq 0.05$) entry differences in GVW were observed in both inoculated and non-inoculated blocks. Genotypes having distinctly low GVW loss over two environments included Agripro Patton, NY87048W-7388, INW 9824, Roane, and OH 552. The assumption can be made that these genotypes possess type IV resistance, on the basis of their ability to retain GVW when infected by *F. graminearum*. Conversely, Gore had distinctly high GVW losses in two environments, and therefore, likely possesses little or no type IV resistance to *F. graminearum*.

DISCUSSION

Research conducted in the current study addressed many questions regarding the practical use of FHB assessment parameters and resistance levels of SRW wheat genotypes commonly cultivated in Virginia and other Mid-Atlantic states as well as other wheat genotypes possessing putative FHB resistance.

Producers and researchers need a preharvest method to assess FHB, which potentially includes FHB incidence, FHB severity, and FHB index. It was concluded that FHB index is the most reliable in-field method currently employed to assess genotypes for FHB resistance and provides a reliable prediction of grain yield and GVW losses (Table 2).

The precision of FHB severity and FHB index to assess disease levels and predict grain yield and GVW losses in this study were comparable. However, there is an inherent risk in only using FHB severity to assess overall disease levels. Factors such as resistance types and mechanisms, uniformity in inoculation, and disease development must me considered. If a genotype actively restricts pathogen spread (type II resistance) after infection, using only FHB severity to assess disease levels is an appropriate means of ascertaining potential grain yield and GVW losses. Such is the case in irrigated yield trials where infection and pathogen spread are promoted. In such trials, type I resistance can still reduce disease incidence, but disease in inoculated trials generally is uniform, thus reducing the importance of measuring FHB incidence. Hence, type II resistance, which FHB severity quantifies, will be of greatest importance. However, if type I resistance is exhibited by a genotype, FHB incidence could be low, yet FHB severity high, in which case disease would be restricted in area but not in severity. In this situation, only using FHB severity, to predict disease levels and quantify grain yield and GVW losses likely would fail in accuracy. However, use of FHB severity alone would significantly reduce time required for in-field disease assessment.

FHB index, which combines FHB incidence and FHB severity into a single value, is a more reliable FHB assessment parameter, particularly in non-epidemic years or in plots where disease incidence is not uniform. Using FHB index as an in-field assessment tool also enables researchers to ascertain a genotype's type I and type II resistance levels, as FHB incidence measures type I resistance and FHB severity measures type II

resistance. FHB incidence would not be a reliable or accurate predictor of FHB disease levels in either of the aforementioned circumstances, and should not be used solely to assess disease levels or to predict grain yield or GVW losses.

In this study percentage FDK and DON toxin accumulation were correlated ($p \leq 0.001$) in three of four environments (Table 3). Percentage FDK and DON toxin accumulation were therefore comparable with regard to assessing disease levels and predicting grain yield and GVW losses post-harvest. This contradicts the findings of Mesterhazy et al. (1999) in which a significant correlation ($p \leq 0.05$) was observed between percentage FDK and grain yield, yet correlation between DON toxin accumulation and grain yield was not significant. DON toxin data is essential to ascertain a genotype's level of type III resistance (Mesterhazy et al., 1999) whereas percentage FDK data predicts a genotype's type IV resistance level (Mesterhazy, 1995); these two resistance types are therein reportedly independent. Using percentage FDK as a post-harvest assessment parameter has the following two advantages: researchers lacking DON toxin testing facilities can eliminate cost of testing grain samples for toxin accumulation and conclusions regarding disease levels can be made rapidly. In summary, percentage FDK is recommended for assessing type III and IV resistance in screening and selection of early generations. Due to cost and time concerns, DON toxin testing can be postponed until screening of advanced lines as a means for verifying type III resistance.

A continuous distribution was observed among genotypes studied with respect to all FHB assessment parameters, grain yield loss, and GVW loss, indicating that all resistance types were quantitative in expression. This agrees with the findings of Buerstmayr et al. (2000), in which inheritance of resistance was reported as being quantitative in two *Triticum* populations and with the findings of Waldron et al. (1999), who reported that type II resistance in 112 recombinant inbred lines derived from a resistant/susceptible cross followed a continuous distribution. Results of the current study indicated that under field conditions, genotypes which exhibit type II resistance occur with the same frequency as genotypes exhibiting resistance types IV and V. This contradicts Mesterhazy et al. (1999), who proposed that genotypes exhibiting resistance

types I and II occur much less frequently than those genotypes exhibiting types IV or V resistance.

Type I resistance has been measured in greenhouse studies using FHB incidence, which also was used in the current field study to identify genotypes with type I resistance. To date, the cultivar Frontana is the only documented *Triticum* genotype possessing type I resistance (Van Ginkel et al., 1996; Singh et al., 1995). Ernie was the only cultivar in this study that had distinctly low FHB incidence values in two environments, implying that Ernie likely possesses type I resistance. This potential type I resistance observed in Ernie could be conferred by an unknown resistance gene or by a form of mechanical resistance mechanism. This cultivar therefore merits further study to delineate its level of resistance to FHB.

Type II resistance is typically assessed using FHB severity in greenhouse trials (Wang et al., 1982; Xu and Fan, 1985) and was used in the current field study in a comparable manner. Coker 9803, Ernie, IL94-1909, NY87048W-7388, and Roane had distinctly low FHB severity values over two or more environments and, therefore, these genotypes likely possess a form of type II resistance. Genotypes such as Gore and Coker 9835, which consistently had high FHB severity ratings, as great as 0.81, had significant GVW losses. This confirms the findings of Jones and Mirocha (1999), in that dramatic GVW reductions occur due to significant colonization of spikes. The findings of this study regarding type II resistance in the aforementioned genotypes are somewhat different than the results of greenhouse screening studies in which most of these genotypes express little resistance compared to Chinese sources (The Ohio State University, 1999; Griffey et al., 1998; Wang and Wang, 1991; Bai and Shaner, 1994). This discrepancy brings into question the validity of sole reliance upon greenhouse screening to identify type II resistance and the relationship between greenhouse and field expression of type II resistance.

DON toxin accumulation within grain samples was used to determine the level of type III resistance of genotypes examined in the present study. Agripro Patton, Coker 9803, NY87048W-7388, and VA96W-326 had distinctly low DON toxin accumulation in two or more environments, which implies that these genotypes likely possess some type III resistance. Genotypes examined in this study that possessed type III resistance also

possessed type V resistance. These findings contradict Mesterhazy (1999), who saw no correlation between DON toxin accumulation in grain and grain yield loss.

The negative effect of FHB on GVW in this study agrees with other studies (Saur, 1991; Saur and Trottet, 1992; Saur and Benacef, 1993) in which reduction of GVW due to FHB was observed. In this study, type IV resistance, defined as the ability of a genotype to restrict GVW loss despite *F. graminearum* infection and colonization, was measured in two ways. The first uses percentage FDK -- an indirect measurement based on visual assessment of kernels which show symptoms of FHB colonization, including shriveled and reduced-size kernels. Percentage FDK was negatively correlated ($p \leq 0.0001$) with GVW in all environments, suggesting that this assessment parameter provides a reliable means for extrapolating GVW loss, and therefore quantifying type IV resistance. IL94-1909, INW 9824, Quantum 706, and Roane had distinctly low percentage FDK over two or more environments, implying that these genotypes have some type IV resistance. Percent GVW loss, a direct measurement of type IV resistance, yielded similar results to those obtained using percentage FDK. Agripro Patton, INW 9824, NY87048W-7388, OH 552, and Roane had significantly low GVW losses over two environments. The cultivars IL94-1909 and OH 552 did not consistently have significantly low values for both percentage FDK and percent GVW loss.

Type V resistance, defined as the ability of a genotype to retain yield despite *F. graminearum* infection and colonization, was directly measured by obtaining percent grain yield loss. Agripro Patton, Freedom, Ernie, IL94-1909, INW 9824, NY87048W-7388, OH 552, and Roane had distinctly low percent grain yield losses in two or more environments. Hence, these genotypes likely have some type V resistance.

Although FHB index does not directly measure any of the aforementioned resistance types, it can be used to discern the presence of resistance types I and II in a tested genotype. Additionally, FHB index can be used to indirectly assess resistance types IV and V. Ernie and Roane had distinctly low FHB index values over two environment as well as low losses in grain yield and GVW. Therefore, mean FHB index also provides a reliable estimate of percent grain yield loss and percent GVW loss.

A trend is observed when genotypes are compared with regard to all FHB assessment parameters, grain yield loss, and GVW loss. Genotypes having the highest

levels of resistance and lowest losses in grain yield and GVW, generally expressed multiple types of resistance. NY87048W-7388 exhibited resistance types II, III, IV, and V. Roane exhibited resistance types II, IV, and V. Agripro Patton exhibited resistance types III, IV, and V. Coker 9803 exhibited resistance types II and III. Ernie exhibited type I, II, and IV resistance. OH 552 exhibited type IV and V resistance. The most susceptible cultivars, Coker 9835 and Gore, consistently had high scores for all FHB assessment parameters and suffered significant losses in grain yield and GVW over all environments.

Difficulties encountered and causal factors that should be addressed when conducting field research on FHB include the necessity of having proper inoculation techniques, accurate and reliable disease scoring methods, controlling other plant diseases (particularly spike diseases), and utilizing procedures for harvesting and cleaning seed that produce representative samples. In this study, as in all yield trial research involving plant diseases, these issues must be addressed to ensure accuracy and reliability of results.

In the current study, two forms of inoculum were used to establish *F. graminearum* in test plots. Application of conidial suspensions in the Montgomery Co., VA tests, while laborious, was a highly uniform and controlled inoculation method, which restricted *F. graminearum* to the inoculated block. Therefore, this technique is recommended for FHB yield loss trials and advanced pure-line trials where genotypes can be inoculated according to growth stage. In the 1999-00 Westmoreland Co., VA location, *Gibberella zeae*-colonized maize kernels were utilized as the inoculum source. This inoculation method greatly reduced labor requirements, yet was not well-suited for a yield loss study. The inherent difficulty in the using *Gibberella zeae*-colonized maize was that this type of inoculum produces ascospores, which unlike conidia are more mobile and spread greater distances by wind. In the Westmoreland Co., VA test, ascospores were wind-driven from the inoculated block into the non-inoculated block and caused significant disease despite the blocks being separated by an 8.8 m triticale boarder. Fernando et al. (1997) reported that significant disease can occur up to 22 m from a source of inoculum which releases ascospores, which agrees with the findings of this study. Unlike a timed and directed spray using a conidial suspension, *Gibberella zeae*-

colonized maize releases spores continually throughout the flowering period, which leads to a longer exposure period of the wheat spikes. While the use of *Gibberella zeae*-colonized maize may not be appropriate for all yield loss studies, it should be noted that its use in screening nurseries is recommended. Use of this method of inoculation in screening nurseries, particularly those comprised of heterogeneous breeding populations, where the goal is to assess resistance and not yield loss, would save a great deal of time in inoculation.

Accuracy of scoring *F. graminearum* in the field can be reduced by many factors, which must be mitigated or controlled. The presence of other diseases may directly or indirectly confound data collected on the disease of interest when scoring in the field. Spike diseases, such as those caused by *S. nodorum, Xanthomonas translucens* f. sp. *undulosa* (S. J. & R.), *Pseudomonas atrofaciens* (McCull) and *F. graminearum*, cause similar symptoms on colonized wheat spikes and proliferate under similar environmental conditions (Eyal et al., 1987; Shaner and Buechley, 1995). Therefore, promotion of FHB through irrigation also promotes the development of diseases such as glume blotch, black chaff, and basal glume rot. Triadimefon gives chemical control of *Staganospora nodorum* and other diseases without affecting *F. graminearum*, therefore its use is highly recommended in FHB field studies. Lodging is enhanced in irrigated tests, which affects and confounds yield and GVW. In such trials, lodging should be controlled with the use of a growth regulator such as ethephon.

In a yield loss study, the type of harvest and seed cleaning methods used are critical and greatly effect data reliability. In yield loss studies involving *F. graminearum*, where a major effect of the disease is reduced GVW, optimizing harvesting techniques becomes even more important. It is essential that the blower on the combine be adjusted to minimize airflow in order to retain as many of the colonized kernels as possible. The tradeoff will be an increase in the amount of trash in grain samples, thus necessitating further cleaning post-harvest. Post-harvest cleaning also must be done with great care, as to not dispel colonized and reduced weight kernels from the grain sample.

The techniques used in this study were refined over the three years in which field trials were conducted. An efficient system was developed for quantifying FHB resistance type II (pathogen spread after infection), type III (DON toxin accumulation), type IV

(GVW loss), and type V (grain yield loss). FHB screening studies will provide the most reliable and accurate data when appropriate and timely inoculation methods are employed, mist-irrigation is applied to plots following inoculation, other diseases and lodging are controlled, and grain is properly harvested and cleaned.

This research was initially undertaken to find the most efficient and reliable assessment parameter to quantify FHB resistance levels of SRW wheat in field studies. In inoculated, irrigated tests where uniform disease is obtained, the amount of labor required can be cut in half or more without losing accuracy when FHB severity or percentage FDK are used alone or in combination to decipher FHB resistance levels. For researchers or producers whose nurseries or fields are not inoculated or irrigated, FHB index is the most reliable method of quantifying resistance levels and losses in tested genotypes. FHB index is the method used by most researchers currently. This study confirms that FHB index provides a more accurate and reliable estimate of genotype response to FHB than FHB severity alone. FHB incidence varied considerably among environments and was the least effective and reliable FHB assessment parameter. Additionally, percentage FDK provided a precise and reliable assessment of resistance. Use of percentage FDK to initially screen genotypes for type III resistance to DON toxin accumulation could save substantial amounts of money from testing grain samples for DON toxin accumulation.

This study provides beneficial information to both wheat researchers and producers in confronting a pathogen with economically devastating implications, for which chemical control is not yet feasible. The types and levels of FHB resistance or lack thereof in several SRW wheat genotypes has been confirmed or proposed. A continuous distribution was observed among genotypes for FHB incidence, FHB severity, FHB index, DON toxin accumulation, percentage FDK, and losses in grain yield and GVW. These assessment methods were evaluated with respect to their predictive value and further analyzed regarding applicability in agricultural and research settings. The findings of this study indicated that all FHB resistance types follow a quantitative mode of inheritance. Additionally, the quantitative expression of FHB resistance observed in this study corroborates the complexity of FHB resistance, which is conferred by multiple mechanisms.

Of noteworthy importance is the fact that resistance to FHB was identified in genotypes in the current field trials, while few of these genotypes have been reported to possess type I or type II resistance in greenhouse tests. These results indicate that the resistance in such genotypes may be conditioned by specific environmental conditions that initiate or enhance the expression of resistance. Genes imparting physiological or mechanical resistance may not be expressed in greenhouse-grown plants due to the lack of appropriate stimuli. In greenhouse trials, plants are subjected less to biotic and abiotic stress, which naturally induce plants to accrue physiological and morphological defenses against pathogens. Hence, there may be genes, whose expression either alone or in tandem, confer or enhance all types of resistance to FHB yet are not expressed nor identified in greenhouse tests. This study confirms the presence and complexity of genes conferring FHB resistance in SRW wheat genotypes, and highlights the need for additional research to characterize such genes and the mechanisms conferring resistance under field conditions.

LITERATURE CITED

Anderson. 1948. The development of *Gibberella zeae* headblight of wheat. Phytopathology 38:595-611.

Bai, G.-H. and G. Shaner. 1994. Scab of wheat: prospects for control. Plant Disease 78:760-765.

Ban, Tomohiro. 1997. Evaluation of resistance to *Fusarium* head blight in indigenous Japanese species of *Agropyron* (*Elymus*). Euphytica 97:39-44.

Ban, T. and K. Suenaga. 1997. Inheritance of resistance to Fusarium head blight caused by *Fusarium graminearum* in wheat. Cereal Research Communications 25:727-728.

Bockus, W.W. and J.P. Shroyer. 1998. The impact of reduced tillage on soilborne plant pathogens. Annual Review of Phytopathology 36:485-500.

Bowden, R and J.F. Leslie. 1999. Sexual recombination in *Gibberella zeae*. Phytopathology 89:182-188.

Buerstmayr, H., B. Steiner, M. Lemmens, and P. Ruckenbauer. 2000. Resistance to Fusarium head blight in winter wheat: heritability and trait associations. Crop Science 40:1012-1018.

Chang, T.T., C.F. Konzak and J.C. Zadoks. 1974. A Decimal Code for the Growth Stages of Cereals. Weed Research 14:415-421.

Eyal, Z., A.L. Scharen, J.M. Prescott, and M. van Ginkel. 1987. The septoria diseases of wheat: Concepts and methods of disease management. CIMMYT, Mexico. D.F., Mexico.

Fernando, W.G.D., T.C. Paulitz, W.L. Seaman, P. Dutilleul, and J.D.Miller. 1997. Head blight gradients caused by Gibberella zeae from area sources of inoculum in wheat field plots. Phytopathology 87:414-421.

Gomez, A. 1984. Statistical Procedures for Agricultural Research, 2d edition. Wiley Publishing, New York.

Griffey, C.A., J. Chen, T. Pridgen, M. Chappell, and E. Stromberg. 1998. Assessment and selection for scab resistance in soft red winter wheat. p.73-75. *In* 1998 National Fusarium Head Blight Forum Proceedings.

Griffey, C.A., J. Chen, T. Pridgen, M. Chappell, and E. Stromberg. 1999. Research on Fusarium Head Blight in the Virginia Tech small grains program. p.95-98. *In* 1999 Joint Research Conference of the Eastern Wheat and Southern Small Grain Workers.

Jones, R.K. and C.J. Mirocha. 1999. Quality parameters in small grains from Minnesota affected by Fusarium head blight. Plant Disease 83:506-511.

Kang, Z. and H. Buchenauer. 2000. Ultrastructural and cytochemical studies on cellulose, xylan, and Pectin degradation in wheat spikes infected by *Fusarium culmorum*. Journal of Phytopathology 148:263-275.

McMullen, M., R. Jones, and D. Gallenberg. 1997. Scab of whet and barley: a re-emerging disease of devastating impact. Plant Disease 81:1340-1348.

Mesterhazy, A. 1995. Types and components of resistance to Fusarium head blight of wheat. Plant Breeding 114:377-386.

Mesterhazy, A., T. Bartok, C.G. Mirocha, and R. Komoroczy. 1999. Nature of wheat resistance to Fusarium head blight and the role of deoxynivalenol for breeding. Plant Breeding 118:97-110.

Miedaner, L. 1997. Breeding wheat and rye for resistance to Fusarium diseases. Plant Breeding 116:201-220.
Miller, J.D., J.C. Young, and D.R. Sampson. 1985. Deoxynivalenol and FHB resistance in spring cereals. Phytopathology 113:359-367.
Miller, D.J., J. Culley, K. Fraser, S. Hubbard, F. Meloche, T. Ouellet, W.L. Seaman, K. Turkington, and H. Voldeng. 1998. Effect of tillage practice on Fusarium head blight of wheat. Canadian Journal of Plant Pathology 20:95-103.
Pereyra, S.A., R. Dill-Macky, and A.L. Sims. 1999. Survival and inoculum potential of Fusarium graminearum in wheat residues. p.96-99. *In* Proceedings of the U.S. Wheat and Barley Scab Initiative Annual Forum. p. 96-99.
Saur, L. 1991. Recherche de geniteurs de resistance a la fusariose de l'epi causee par *Fusarium culmorum* chez le ble et les especes voisines. Agronomie 11:535-541.
Saur, L. and M. Trottet. 1992. Heritabilite de la resistance a la fusariose de l'epi et selection recurrente dans une population de ble tendre. Agronomie 12:297-302.
Saur, L. and N. Benacef. 1993. Relation entre les symptomes de fusariose de l'epi et la perte de rendement chez le ble tendre. Agronomie 13:829-833.
Schroeder, H.W. and J.J. Christensen. 1963. Factors affecting resistance of wheat to scab caused by *Gibberella zeae*. Phytopathology 53:831-838.
Shaner, G., and G. Buechley. 1995. Epidemiology of leaf blotch of soft red winter wheat caused by Septoria tritici and Stagonospora nodorum. Plant Dis. 79:928-938.
Singh, R.P., H. Ma, and S. Rajaram. 1995. Genetic analysis of resistance to scab in spring wheat cultivar Frontana. Plant Disease 79:238-240.
Sinha, R.C. and M.E. Savard. 1997. Concentration of deoxynivalenol in single kernels and various tissues of wheat heads. Canadian Journal of Plant Pathology 19:8-12.
Snijders, C.H.A. 1990a. Diallel analysis of resistance to head blight caused by *Fusarium culmorum* in winter wheat. Euphytica 50:1-9.
Stack, R.W. 1999. Return of an old problem: Fusarium head blight of small grains. APSnet. http://www.scisoc.org.
The Ohio State University: Horticulture and Crop Science Series 690. Uniform Winter Wheat Fusarium Head Blight Screening Nursery: 1999 Nursery Report. Wooster, OH: Ohio Agricultural Research and Development Center, 1999.
U.S. Wheat and Barley Scab Initiative. 1999. Scab research initiative underway with Glickman announcement of $3 million funding boost. v.2.p.1. *In* Scab News.
Van Ginkel, Maarten, W. Van Der Schaar, Y. Zhuping, and S. Rajaram. 1996. Inheritance of resistance to scab in two wheat cultivars from brazil and china. Plant Disease 80:863-867.
Waldron, B.L, B. Moreno-Sevilla, J.A. Anderson, R.W. Stack, and R.C. Frohberg. 1999. RFLP mapping of QTL for Fusarium head blight resistance in wheat. Crop Science 39:805-811.
Wang, Y.Z., X.N. Yong, and Q.P. Xiao. 1982. The improvement of identification technique of scab resistance of wheat and the development of resistant sources. Scientia Agricultura Sinica. 5:67-77.
Wang, Y and J. Wang. 1991. Genetical studies on the resistance to scab spread in wheat varieties. Jilin Academy of Agricultural Sciences 1:21-28.

Xu, Y.G., and Z.D. Fan. 1985. Evaluation and differentiation of pathogenicity of *Fusarium graminearum* on wheat cultivars. Acta Phytopathology Sinica 12(4):53-57.

Yu, Yu-Jun. 1991. Genetic analysis for scab resistance in five wheat varieties, PHJZM, HHDTB, CYHM, YGFZ, and WN2. Acta Agronomica Sinica 17:248-254.

Table 1. Analysis of variance and mean squares of FHB assessment parameters, grain yield, and grain volume weight for four environments analyzed independently.

	Mean Squares							
Source [1]	**Grain Yield**	**Grain Volume Weight**	**FHB Incidence**	**FHB Severity**	**FHB Index**	**FDK** [2]	**DON** [3]	**GB** [4]
1997-98 Montgomery Co.								
ENTRY	422.9 ***	70.5 ***	821.4 ***	545.0 ***	545.3 ***	264.1 ***	25.5 ***	
TREATMENT	9900.8 ***	110.2 ***	69024.0 ***	9205.0 ***	17593.4 ***	7552.5 ***	2801.4 ***	
ENT(TRT)	85.5 ***	3.3 *NS*	210.8 *NS*	179.8 ***	272.6 ***	49.2 ***	7.7 ***	
1998-99 Montgomery Co.								
ENTRY	609.5 ***	14.7 ***						3.2 ***
TREATMENT	11424 ***	151.6 ***						40.1 ***
ENT(TRT)	142.9 *	2.2 **						2.2 **
1999-00 Montgomery Co.								
ENTRY	194.5 ***	19 ***	250.5 ***	2.4 ***	223.7 ***	633.1 ***	23.9 *	
TREATMENT	22281 ***	1238.2 ***	249835.8 ***	39.1 ***	40435 ***	91125 ***	1066.5 ***	
ENT(TRT)	148.4 ***	8.4 ***	273.1 ***	1.8 ***	221.8 ***	371.6 ***	24.9 *	
1999-00 Westmoreland Co.								
ENTRY	394.6 ***	100.8 ***	2656.4 ***	13.9 ***	2249 ***	2848.9 ***	1193.3 ***	
TREATMENT	1271.1 ***	316.5 ***	15998.9 ***	5.57 **	554.2 **	3758.4 ***	9332.6 ***	
ENT(TRT)	28.5 NS	6.7 *	431.1 ***	0.9 NS	93.4 *	102.5 NS	97.8 NS	

Asterisks denote significance at P<0.05 (*), P<0.01 (**), and P<0.001 (***).
NS - not significant

1- Four tests were conducted, each having one inoculated treatment and one non-inoculated control.
2- Percentage fusarium damaged kernels (FDK)
3- DON toxin accumulation
4- Glume blotch (*Staganospora nodorum*)

Table 2. r^2 (coefficient of determination) values and associated p-values for FHB disease assessment parameters, lodging, and *Staganospora nodorum* (Glume Blotch) with yield and grain volume weight (GVW) in four environments analyzed separately.

	1997-98 Montgomery Co., VA				1998-99 Montgomery Co., VA				1999-00 Montgomery Co., VA				1999-00 Westmoreland Co., VA			
	Grain Yield		GVW		Grain Yield		GVW		Grain Yield		GVW		Grain Yield		GVW	
FHB Incidence	0.55	***	0.58	***	0.02		0.06	*	0.27	***	0.29	***	0.33	***	0.53	***
FHB Severity	0.70	***	0.74	***	0.04	*	0.15	***	0.24	***	0.34	***	0.51	***	0.72	***
FHB Index	0.74	***	0.75	***	0.05	*	0.16	***	0.35	***	0.44	***	0.53	***	0.75	***
Percentage FDK	0.51	***	0.57	***	0.14	***	0.37	***	0.25	***	0.44	***	0.52	***	0.69	***
Deoxynivalenol (DON)	0.56	***	0.61	***	0.09	**	0.12	***	0.12	***	0.07	*	0.27	***	0.54	***
S. Nodorum	NA		NA		0.23	***	0.02		NA		NA		NA		NA	
Lodging	NA		NA		0.25	***	0.17	***	NA		NA		NA		NA	

NA, absence of significant levels of *Staganospora nodorum* or lodging.
Asterisks denote significance at P<0.05 (*), P<0.01 (**), and P<0.001 (***).

Table 3. r^2 (coefficient of determination) values among FHB disease assessment parameters, in four environments, analyzed separately. r^2 values with *Staganospora nodorum* are also included for the 1998-99 Montgomery Co., VA location.

	FHB Incidence	FHB Severity	FHB Index	Percentage FDK	Deoxynivalenol (DON)
1997-98 Montgomery Co., VA					
FHB Incidence	1.00 ***				
FHB Severity	0.78 ***	1.00 ***			
FHB Index	0.89 ***	0.78 ***	1.00 ***		
Percentage FDK	0.81 ***	0.81 ***	0.79 ***	1.00 ***	
Deoxynivalenol (DON)	0.81 ***	0.89 ***	0.85 ***	0.90 ***	1.00 ***
1998-99 Montgomery Co., VA					
FHB Incidence	1.00 ***				
FHB Severity	0.48 ***	1.00 ***			
FHB Index	0.77 ***	0.91 ***	1.00 ***		
Percentage FDK	0.38 ***	0.40 ***	0.49 ***	1.00 ***	
Deoxynivalenol (DON)	0.45 ***	0.41 ***	0.43 ***	0.50 ***	1.00 ***
Staganospora nodorum	0.24 *	0.13 (NS)	0.20 *	0.28 **	0.28 **
1999-00 Montgomery Co., VA					
FHB Incidence	1.00 ***				
FHB Severity	0.54 ***	1.00 ***			
FHB Index	0.72 ***	0.96 ***	1.00 ***		
Percentage FDK	0.34 ***	0.38 ***	0.44 ***	1.00 ***	
Deoxynivalenol (DON)	0.20 *	0.13 (NS)	0.17 (NS)	0.21 *	1.00 ***
1999-00 Westmoreland Co., VA					
FHB Incidence	1.00 ***				
FHB Severity	0.73 ***	1.00 ***			
FHB Index	0.79 ***	0.99 ***	1.00 ***		
Percentage FDK	0.71 ***	0.80 ***	0.82 ***	1.00 ***	
Deoxynivalenol (DON)	0.72 ***	0.75 ***	0.84 ***	0.82 ***	1.00 ***

Asterisks denote significance at P<0.05 (*), P<0.01 (**), and P<.001 (***)
NS - not significant

Table 4. Mean FHB index (IND), FHB severity (SEV), FHB incidence (INC), percentage fusarium damaged kernels (FDK), and DON toxin accumulation (in ppm) by genotype in each environment. Bold values are significantly lower at p = 0.05.

	1997-98 Montgomery Co., VA					1999-00 Montgomery Co., VA					1999-00 Westmoreland Co., VA				
LINE *	**IND**	**SEV**	**INC**	**FDK**	**DON**	**IND**	**SEV**	**INC**	**FDK**	**DON**	**IND**	**SEV**	**INC**	**FDK**	**DON**
ERNIE	**5.2**	**0.16**	**28.3**	**12.7**	**5.88**	**9.1**	**0.17**	**52.7**	52.7	7.28	24.8	0.28	87.3	28.5	2.85
NY 87048W-7388						**19.9**	**0.22**	87.7	**36.7**	**0.99**	**7.5**	**0.16**	**47.0**	**11.2**	**0.30**
AGRIPRO PATTON						**10.0**	**0.16**	**61.0**	45.2	**0.48**	20.6	**0.24**	87.0	**21.2**	**0.63**
INW 9824	**8.2**	**0.18**	41.0	**8.5**	**4.62**	**19.6**	**0.27**	**64.7**	53.5	3.40	21.6	0.28	76.0	**21.5**	**0.86**
IL 94-1909						24.5	**0.28**	86.3	**30.3**	13.23	**9.6**	**0.16**	**56.3**	**10.2**	**0.88**
FREEDOM	**9.7**	**0.18**	48.7	14.7	**5.57**	32.1	0.34	95.0	64.7	1.78	**15.5**	**0.20**	75.7	34.5	**0.55**
ROANE	**10.2**	**0.19**	47.0	**12.2**	**6.10**	**22.6**	**0.26**	85.0	**34.2**	6.44	28.1	0.29	96.0	**22.5**	1.27
COKER 9803	**11.3**	**0.22**	44.0	15.0	**5.52**	23.5	**0.28**	83.0	43.0	5.12	26.9	0.32	85.0	38.3	**0.88**
CAYUGA						32.6	0.44	73.0	47.5	6.50	**9.7**	**0.17**	56.7	41.0	**0.59**
OH 552						**20.9**	**0.26**	75.7	65.0	**0.84**	25.1	0.27	91.7	46.3	1.42
PIONEER 2552	16.0	0.24	61.7	17.8	9.30	**22.3**	**0.28**	80.3	51.5	13.90	35.0	0.38	93.0	57.5	3.45
AGRIPRO FOSTER	15.5	0.25	52.3	**9.3**	**5.58**	38.4	0.41	94.0	48.5	5.94	19.6	**0.25**	75.0	24.0	**0.96**
QUANTUM 706						35.5	0.48	72.7	**30.0**	3.29	18.0	**0.25**	71.7	**21.3**	1.07
WAKEFIELD	**13.3**	0.28	45.7	16.0	7.55	35.1	0.46	73.3	58.3	3.86	35.3	0.40	89.3	43.8	1.72
VA 96W-329						28.1	0.33	83.0	70.0	5.01	28.0	0.32	88.0	40.7	1.77
VA 96W-326						30.8	0.38	78.3	47.2	**0.59**	28.7	0.35	80.3	42.0	1.10
JACKSON	14.5	0.24	49.3	27.3	8.40	34.9	0.37	92.7	69.8	2.61	45.3	0.46	98.0	63.5	2.11
AGRIPRO MASON	15.8	0.27	49.0	16.2	7.63	36.8	0.47	78.3	41.0	**1.49**	45.1	0.48	94.7	46.8	1.79
PIONEER 2580	**13.7**	0.25	52.7	25.7	8.72	36.5	0.41	87.3	86.3	4.58	47.8	0.48	99.3	76.7	3.77
MADISON	18.7	0.34	44.7	20.5	9.18	29.0	0.35	76.0	84.2	2.95	54.2	0.55	99.3	77.8	2.21
VA 96W-348						28.9	0.31	94.3	60.5	2.42	39.8	0.41	97.3	67.3	2.52
PIONEER 2684	21.7	0.30	65.7	23.2	8.85	33.8	0.35	95.7	70.3	9.46	48.7	0.51	94.7	61.0	2.79
SS 550						28.4	0.30	96.0	50.7	10.70	43.9	0.45	98.0	54.8	3.81
FFR 555 W	22.8	0.32	58.0	18.2	8.73	43.5	0.46	95.0	73.0	4.11	51.1	0.52	97.7	83.2	3.28
PIONEER 2643	22.7	0.31	66.7	16.7	7.42	40.7	0.42	97.0	86.2	8.56	57.3	0.58	99.0	77.7	3.43
SISSON						30.4	0.33	91.3	61.3	4.01	52.3	0.53	99.0	60.0	2.52
POCAHONTAS	22.7	0.32	62.3	23.7	8.98	52.8	0.54	97.3	73.8	6.16	61.1	0.61	100.0	71.0	3.36
COKER 9835	33.2	0.38	80.0	31.3	11.28	42.4	0.44	96.7	86.7	7.03	80.7	0.81	100.0	84.5	3.77
GORE	48.0	0.59	67.7	32.5	12.92	68.7	0.69	99.0	89.7	15.93	61.3	0.62	99.3	78.8	5.79
MEAN	17.8	0.27	54.4	18.9	7.80	30.9	0.36	83.6	57.8	5.43	36.4	0.39	87.6	49.1	2.16
LSD (p = 0.05)	9.12	0.08	11.44	5.10	1.97	14.24	0.12	15.30	14.07	0.93	9.86	0.09	9.47	13.34	0.76
C.V.	53.44	31.05	21.94	28.16	26.38	33.82	25.52	13.41	17.81	32.77	19.83	17.26	7.91	19.92	21.84

* Lines are ranked in ascending order based upon Mean FHB index values over all environments.

Table 5. Mean values, over all environments of FHB index (IND), FHB incidence (INC), FHB severity (SEV), percentage fusarium damaged kernels (FDK), DON toxin accumulation, percent grain yield loss, and percent grain volume weight loss with ranks for each mean.

LINE *	FHB IND	RANK	YIELD LOSS	RANK	GVW LOSS	RANK	FHB INC	RANK	FHB SEV	RANK	PERCENTAGE FDK	RANK	DON TOXIN	RANK
ERNIE	13.05	*1*	7.22	*7*	3.89	*10*	56.11	*1*	0.20	*3*	31.28	*7*	5.34	*19*
NY 87048W-7388	13.70	*2*	3.64	*1*	1.38	*2*	67.34	*4*	0.19	*1*	23.92	*3*	0.65	*2*
AGRIPRO PATTON	15.34	*3*	3.84	*2*	2.17	*5*	74.00	*12*	0.20	*2*	33.17	*9*	0.56	*1*
INW 9824	16.46	*4*	12.00	*12*	5.20	*15*	60.56	*2*	0.24	*6*	27.83	*6*	2.96	*8*
IL 94-1909	17.05	*5*	6.34	*5*	3.42	*8*	71.33	*7*	0.22	*4*	20.25	*1*	7.06	*25*
FREEDOM	19.07	*6*	6.86	*6*	0.70	*1*	73.11	*9*	0.24	*5*	37.95	*11*	2.63	*7*
ROANE	20.27	*7*	10.72	*10*	1.43	*4*	76.00	*14*	0.25	*7*	22.95	*2*	4.60	*17*
COKER 9803	20.58	*8*	19.82	*20*	4.97	*13*	70.67	*6*	0.27	*9*	32.12	*8*	3.84	*13*
CAYUGA	21.15	*9*	11.24	*11*	4.68	*12*	64.84	*3*	0.31	*12*	44.25	*14*	3.55	*11*
OH 552	23.03	*10*	5.32	*4*	2.32	*6*	83.67	*20*	0.27	*8*	55.67	*20*	1.13	*4*
PIONEER 2552	24.43	*11*	23.10	*21*	6.91	*21*	78.33	*15*	0.30	*10*	42.28	*13*	8.88	*28*
AGRIPRO FOSTER	24.48	*12*	17.35	*18*	5.53	*16*	73.78	*11*	0.30	*11*	27.28	*5*	4.16	*14*
QUANTUM 706	26.71	*13*	8.99	*8*	3.29	*7*	72.17	*8*	0.37	*16*	25.67	*4*	2.18	*5*
WAKEFIELD	27.91	*14*	18.93	*19*	6.54	*19*	69.44	*5*	0.38	*19*	39.39	*12*	4.38	*16*
VA 96W-329	28.03	*15*	4.92	*3*	1.41	*3*	85.50	*22*	0.33	*13*	55.34	*19*	3.39	*10*
VA 96W-326	29.74	*16*	10.22	*9*	4.04	*11*	79.33	*16*	0.37	*17*	44.59	*15*	0.85	*3*
JACKSON	31.55	*17*	25.93	*25*	10.22	*25*	80.00	*18*	0.36	*14*	53.55	*18*	4.37	*15*
AGRIPRO MASON	32.59	*18*	16.82	*17*	3.82	*9*	74.00	*13*	0.41	*22*	34.67	*10*	3.64	*12*
PIONEER 2580	32.67	*19*	23.54	*23*	8.26	*23*	79.78	*17*	0.38	*20*	62.89	*26*	5.69	*21*
MADISON	33.97	*20*	15.96	*15*	6.32	*18*	73.33	*10*	0.41	*23*	60.84	*25*	4.78	*18*
VA 96W-348	34.36	*21*	12.02	*13*	5.75	*17*	95.83	*28*	0.36	*15*	63.92	*27*	2.47	*6*
PIONEER 2684	34.73	*22*	23.21	*22*	8.70	*24*	85.34	*21*	0.39	*21*	51.47	*16*	7.03	*24*
SS 550	36.15	*23*	14.23	*14*	5.09	*14*	97.00	*29*	0.38	*18*	52.75	*17*	7.26	*26*
FFR 555 W	39.13	*24*	35.36	*27*	13.42	*27*	83.56	*19*	0.43	*25*	58.11	*22*	5.37	*20*
PIONEER 2643	40.21	*25*	26.96	*26*	11.64	*26*	87.56	*24*	0.44	*26*	60.17	*23*	6.47	*23*
SISSON	41.33	*26*	16.28	*16*	7.41	*22*	95.17	*27*	0.43	*24*	60.67	*24*	3.27	*9*
POCAHONTAS	45.52	*27*	23.99	*24*	6.66	*20*	86.55	*23*	0.49	*27*	56.17	*21*	6.17	*22*
COKER 9835	52.11	*28*	40.93	*29*	14.16	*28*	92.22	*26*	0.54	*28*	67.50	*29*	7.36	*27*
GORE	59.33	*29*	40.28	*28*	15.07	*29*	88.67	*25*	0.63	*29*	67.00	*28*	11.55	*29*

* Lines are ranked in ascending order based upon Mean FHB index values over all environments.

Table 6. Mean percentage losses of grain yield and grain volume weight of genotypes in four environments. Those genotypes with statistically similar losses in a single environment are grouped on the basis of Duncan's Multiple Range Test.

	1997-98 Montgomery Co., VA		1999-00 Montgomery Co., VA		1999-00 Westmoreland Co., VA	
LINE *	GRAIN YIELD	GVW	GRAIN YIELD	GVW	GRAIN YIELD	GVW
ERNIE	5.7 a	-0.3 ab	10.9 abc	6.5 bcd	5.0 abcd	5.5 abcdefgh
NY 87048W-7388			8.3 a	4.1 abc	2.6 abcd	0.0 abc
AGRIPRO PATTON			9.5 ab	4.4 abc	2.0 abc	2.1 abcde
INW 9824	16.6 ab	8.4 ab	14.8 abcdef	4.3 a	4.6 abcd	2.8 abcdef
IL 94-1909			21.1 abcdefgh	8.4 defg	-2.1 ab	1.9 abcd
FREEDOM	3.3 a	-3.4 a	17.6 abcdefg	6.9 cde	-0.3 ab	-1.4 ab
ROANE	15.3 ab	0.0 ab	7.1 abcd	1.8 a	9.8 abcd	2.4 abcdefgh
COKER 9803	21.6 b	0.3 ab	26.4 efghij	8.6 defgh	11.5 bcd	6.0 abcdefgh
CAYUGA			20.7 abc	7.9 def	13.1 bcd	6.1 abcdefgh
OH 552			15.5 abcdef	3.9 abc	0.4 ab	3.1 abcdef
PION 2552	20.9 b	6.6 ab	29.6 ghijkl	9.2 defgh	18.8 bcde	4.9 abcdefgh
AGRIPRO FOSTER	19.2 b	2.8 ab	24.6 defghi	8.6 defgh	8.2 abcd	5.1 abcdefgh
QUANTUM 706			17.7 abcdefg	7.2 cde	9.3 bcd	2.7 abcdef
WAKEFIELD	22.5 b	4.9 ab	27.7 fghijk	10.5 defghi	6.6 abcd	4.2 abcdefg
VA 96W-329			26.5 efghij	8.9 defgh	-11.8 a	-4.7 a
VA 96W-326			21.4 abcdefgh	10.9 defghi	9.2 bcd	1.3 abcd
JACKSON	29.5 bcd	10.7 ab	29.9 ghijkl	10.9 efghi	18.5 bcde	9.0 cdefg
AGRIPRO MASON	25.1 b	4.8 ab	17.5 abcdefg	6.6 bcd	7.8 abcd	0.0 abc
PION 2580	26.0 b	8.1 ab	31.6 hijklm	11.9 fghi	13.0 bcd	4.8 abcdefgh
MADISON	22.9 b	4.2 ab	22.0 bcdefgh	8.8 defgh	3.0 abcd	5.9 abcdefgh
VA 96W-348			24.1 cdefghi	9.4 defgh	11.9 bcd	7.8 abcdefgh
PION 2684	27.8 bc	7.7 ab	39.5 klmn	14.6 i	2.4 abc	3.8 abcdef
SS 550			36.3 ijklm	12.1 fghi	6.4 abcd	3.2 abcdef
FFR 555 W	40.9 de	15.0 b	42.9 mn	13.3 hi	22.3 cde	12.0 fgh
PION 2643	26.9 bc	6.0 ab	38.1 jklmn	15.6 i	15.8 bcd	13.3 gh
SISSON			30.7 ghijklm	10.8 defghi	18.1 bcde	11.4 efgh
POCAHONTAS	21.3 b	4.0 ab	35.9 ijklm	12.9 ghi	14.8 bcd	3.1 abcdef
COKER 9835	46.9 e	14.1 b	41.0 lmn	14.4 i	34.9 e	14.0 h
GORE	48.0 e	12.3 b	49.3 n	22.5 j	23.5 de	10.4 defgh
Pr > F	< 0.0001	0.4613	< 0.0001	< 0.0001	0.0033	0.0012
Grand Mean	24.0	5.6	25.1	9.3	9.5	5.0
C.V.	29.39	32.00	27.19	12.07	11.63	29.49

* Lines are ranked in ascending order based upon Mean FHB index values over all environments.

Appendix A. Field Design utilized at all locations. The diagram represents 30 genotypes which are replicated six times in 30.48m^2 plots.

INOCULATED BLOCK			<--25 ft.-->	NON-INOCULATED BLOCK		
Boarder	**Row**	**Boarder**		**Row**	**Boarder**	**Row**
Rep. #1	Rep. #2	Rep. #3		Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3	**Triticale**	Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3	**Boarder**	Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3	**Range**	Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3		Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3		Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3	**Triticale**	Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3	**Boarder**	Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3	**Range**	Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3		Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3		Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3	**Triticale**	Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3	**Boarder**	Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3	**Range**	Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3		Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3		Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3	**Triticale**	Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3	**Boarder**	Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3	**Range**	Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3		Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3		Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3	**Triticale**	Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3	**Boarder**	Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3	**Range**	Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3		Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3		Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3	**Triticale**	Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3	**Boarder**	Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3	**Range**	Rep. #4	Rep. #5	Rep. #6
Rep. #1	Rep. #2	Rep. #3		Rep. #4	Rep. #5	Rep. #6
Boarder	**Row**	**Boarder**		**Row**	**Boarder**	**Row**

<---------------------------------192.5 ft.--------------------------------->

Appendix B. Minimum and maximum temperatures for the 1997-98 Montgomery Co., VA test site by date. Dates which are highlighted are those in which anthesis occurred and inoculation was performed.

Date	Minimum Temp.(C)	Maximum Temp.(C)		Date	Minimum Temp.(C)	Maximum Temp.(C)
31-Mar	16.11	25.00		30-Apr	11.67	18.33
1-Apr	8.33	22.22		1-May	7.78	19.44
2-Apr	1.11	23.33		2-May	7.22	17.78
3-Apr	7.78	18.33		3-May	7.22	21.11
4-Apr	0.56	9.44		**4-May**	**5.56**	**18.89**
5-Apr	-2.78	14.44		**5-May**	**4.44**	**19.44**
6-Apr	-2.22	18.33		**6-May**	**8.89**	**23.89**
7-Apr	1.67	22.78		**7-May**	**11.67**	**18.89**
8-Apr	6.67	26.67		**8-May**	**12.78**	**20.56**
9-Apr	6.11	19.44		**9-May**	**10.00**	**21.67**
10-Apr	2.78	12.22		**10-May**	**12.22**	**18.89**
11-Apr	-2.78	15.56		**11-May**	**11.67**	**19.44**
12-Apr	-1.67	18.33		**12-May**	**11.67**	**21.11**
13-Apr	2.78	20.56		13-May	11.67	23.89
14-Apr	4.44	21.11		14-May	10.56	27.22
15-Apr	9.44	24.44		15-May	13.33	28.89
16-Apr	13.89	21.67		16-May	15.56	30.00
17-Apr	8.89	25.00		17-May	8.33	30.56
18-Apr	6.67	12.22		18-May	8.33	28.33
19-Apr	6.67	10.56		19-May	15.00	31.67
20-Apr	2.22	16.67		20-May	15.56	28.89
21-Apr	5.00	18.33		21-May	12.22	26.11
22-Apr	2.78	15.00		22-May	11.11	24.44
23-Apr	1.67	16.67		23-May	14.44	24.44
24-Apr	3.89	20.00		24-May	13.89	23.33
25-Apr	6.67	22.78		25-May	14.44	27.78
26-Apr	10.56	25.00		26-May	14.44	25.56
27-Apr	-0.56	14.44		27-May	14.44	23.33
28-Apr	0.56	19.44		28-May	16.67	28.33
29-Apr	5.00	22.78		29-May	14.44	30.00
				30-May	16.67	31.67

Appendix C. Minimum and maximum temperatures for the 1998-99 Montgomery Co., VA test site by date. Dates which are highlighted are those in which anthesis occurred and inoculation was performed.

Date	Minimum Temp.(C)	Maximum Temp.(C)		Date	Minimum Temp.(C)	Maximum Temp.(C)
1-Apr	7.74	16.78		1-May	-1.146	19.32
2-Apr	5.635	22.73		2-May	-1.858	21.01
3-Apr	4.705	24.45		3-May	-1.875	22.56
4-Apr	8.5	24.18		4-May	1.01	26.16
5-Apr	10.02	18.53		5-May	6.141	22.67
6-Apr	7.73	19.69		6-May	10.56	25.59
7-Apr	5.596	22.73		7-May	8.18	25.41
8-Apr	1.36	26.32		8-May	9.79	20.46
9-Apr	11.01	25.58		9-May	4.962	21.53
10-Apr	12.11	21.94		**10-May**	**5.239**	**25.7**
11-Apr	9.69	23.18		**11-May**	**4.784**	**25.44**
12-Apr	4.906	15.86		**12-May**	**6.939**	**26.13**
13-Apr	4.058	14.65		**13-May**	**8.98**	**23.38**
14-Apr	-0.372	18.35		**14-May**	**8.1**	**14.67**
15-Apr	6.747	9.33		**15-May**	**5.857**	**19.58**
16-Apr	6.681	12.99		**16-May**	**2.161**	**21.49**
17-Apr	3.636	11.05		**17-May**	**5.444**	**24.91**
18-Apr	4.617	8.92		**18-May**	**11.07**	**25.57**
19-Apr	-0.985	14.47		**19-May**	**13.19**	**19.74**
20-Apr	4.079	15.52		**20-May**	**4.667**	**22.68**
21-Apr	0.449	21.02		21-May	3.688	25.78
22-Apr	2.39	27.58		22-May	7.38	23.93
23-Apr	6.693	25.27		23-May	12.89	23.93
24-Apr	7.31	17.55		24-May	13.18	17.24
25-Apr	-1.724	20		25-May	5.845	19.42
26-Apr	3.445	15.26		26-May	5.944	20.71
27-Apr	11.79	18.67		27-May	5.151	20.72
28-Apr	6.111	11.39		28-May	3.558	24.76
29-Apr	5.394	8.54		29-May	5.053	26.92
30-Apr	4.197	16.08		30-May	5.868	27.32
				31-May	8.42	25.78

Appendix D. Minimum and maximum temperatures for the 1999-00 Montgomery Co., VA test site by date. Dates which are highlighted are those in which anthesis occurred and inoculation was performed.

Date	Minimum Temp.(C)	Maximum Temp.(C)		Date	Minimum Temp.(C)	Maximum Temp.(C)
1-Apr	7.64	15.29		1-May	4.679	21.47
2-Apr	9.87	20.96		2-May	2.058	23.92
3-Apr	2.081	14.89		3-May	7.42	24.88
4-Apr	1.049	12.8		4-May	8.71	26.55
5-Apr	-0.344	22.69		5-May	11.72	28.7
6-Apr	2.403	24.41		6-May	10.53	29.6
7-Apr	1.077	16.14		7-May	8.73	27.38
8-Apr	-0.813	10.83		**8-May**	**10.74**	**27.96**
9-Apr	3.866	18.64		**9-May**	**11.18**	**25.52**
10-Apr	4.627	19.58		**10-May**	**2.285**	**27.15**
11-Apr	2.323	11.83		**11-May**	**7.43**	**29.48**
12-Apr	0.288	6.326		**12-May**	**12.81**	**30.05**
13-Apr	3.058	8.48		**13-May**	**11.62**	**20.65**
14-Apr	7.9	15.6		**14-May**	**3.578**	**19.28**
15-Apr	9.99	23.47		**15-May**	**-0.209**	**21.16**
16-Apr	8.09	19.39		**16-May**	**8.64**	**24.58**
17-Apr	9.38	11.94		**17-May**	**9.7**	**28.84**
18-Apr	5.973	18.4		18-May	10.95	27.63
19-Apr	3.471	23.22		19-May	13.8	25.89
20-Apr	5.905	17.87		20-May	14.21	23.75
21-Apr	4.938	9.44		21-May	9.8	20.64
22-Apr	6.658	16.53		22-May	8.64	22.9
23-Apr	0.998	10.76		23-May	13.26	27.38
24-Apr	6.613	10.07		24-May	12.52	21.8
25-Apr	2.293	15.15		25-May	6.006	25.25
26-Apr	-0.98	17.15		26-May	11.2	25.42
27-Apr	0.443	12.92		27-May	14.34	24.47
28-Apr	2.835	16.69		28-May	11.79	15.71
29-Apr	0.89	20.45		29-May	5.475	19.75
30-Apr	-0.118	24.19		30-May	9.04	27.08

Appendix E. Minimum and maximum temperatures for the 1999-00 Westmoreland Co., VA test site by date. Dates which are highlighted are those in which anthesis occurred.

Date	Minimum Temp.(C)	Maximum Temp.(C)		Date	Minimum Temp.(C)	Maximum Temp.(C)
1-Apr	1.11	17.78		**1-May**	**7.78**	**26.67**
2-Apr	10.56	21.11		**2-May**	**17.22**	**25.56**
3-Apr	14.44	23.89		**3-May**	**9.44**	**23.89**
4-Apr	15.56	22.22		**4-May**	**9.44**	**26.67**
5-Apr	3.33	16.11		**5-May**	**15.56**	**31.11**
6-Apr	5.56	26.67		**6-May**	**17.22**	**32.22**
7-Apr	8.33	25.56		**7-May**	**21.67**	**33.89**
8-Apr	15.56	26.67		**8-May**	**18.33**	**33.33**
9-Apr	1.11	23.89		**9-May**	**20.00**	**32.22**
10-Apr	4.44	21.11		**10-May**	**21.11**	**31.67**
11-Apr	10.00	23.89		**11-May**	**13.33**	**28.89**
12-Apr	11.67	22.78		**12-May**	**18.89**	**32.22**
13-Apr	3.89	12.78		13-May	19.44	34.44
14-Apr	5.00	15.00		14-May	15.00	31.67
15-Apr	11.11	17.22		15-May	14.44	25.56
16-Apr	15.00	25.56		16-May	9.44	23.89
17-Apr	12.22	25.56		17-May	13.89	26.67
18-Apr	8.33	18.33		18-May	18.33	31.11
19-Apr	6.67	17.22		19-May	18.89	30.56
20-Apr	4.44	21.11		20-May	15.00	20.00
21-Apr	11.67	22.78		21-May	13.89	18.89
22-Apr	7.78	16.11		22-May	15.00	18.33
23-Apr	9.44	17.22		23-May	10.56	22.22
24-Apr	**6.11**	**19.44**		24-May	17.22	30.00
25-Apr	**7.78**	**17.22**		25-May	20.00	29.44
26-Apr	**3.89**	**15.00**		26-May	14.44	27.22
27-Apr	**6.67**	**13.33**		27-May	13.33	26.11
28-Apr	**6.67**	**17.78**		28-May	11.67	19.44
29-Apr	**5.00**	**19.44**		29-May	10.00	17.22
30-Apr	**10.00**	**22.22**		30-May	11.67	16.67
				31-May	11.67	23.89

Appendix F. Comparison of ranks for FHB index and percent grain yield loss for genotypes for each environment.

	1997-98 Montgomery Co., VA				1999-00 Montgomery Co., VA				1999-00 Westmoreland Co., VA			
LINE *	FHB INDEX	INDEX RANK	YIELD % LOSS	LOSS RANK	FHB INDEX	INDEX RANK	YIELD % LOSS	LOSS RANK	FHB INDEX	INDEX RANK	YIELD % LOSS	LOSS RANK
ERNIE	5.17	1	5.7	2	9.13	1	10.9	3	24.84	9	5.0	10
NY 87048W-7388					19.87	4	8.3	1	7.52	1	2.6	6
AGRIPRO PATTON					10.03	2	9.5	2	20.64	7	2.0	7
INW 9824	8.17	2	16.6	4	19.62	3	14.8	5	21.58	8	4.6	9
IL 94-1909					24.51	9	21.1	12	9.59	2	-2.1	2
FREEDOM	9.67	3	3.3	1	32.09	16	17.6	9	15.46	4	-0.3	3
ROANE	10.17	4	15.3	3	22.55	7	7.1	4	28.10	13	9.8	13
COKER 9803	11.33	5	21.6	8	23.49	8	26.4	17	26.92	11	11.5	18
CAYUGA					32.59	17	20.7	10	9.71	3	13.1	20
OH 552					20.94	5	15.5	6	25.11	10	0.4	4
PIONEER 2552	16.00	11	20.9	7	22.33	6	29.6	19	34.96	15	18.8	26
AGRIPRO FOSTER	15.50	9	19.2	5	38.35	24	24.6	15	19.60	6	8.2	14
QUANTUM 706					35.45	21	17.7	8	17.97	5	9.3	17
WAKEFIELD	13.33	6	22.5	10	35.12	20	27.7	18	35.29	16	6.6	11
VA 96W-329					28.05	10	26.5	16	28.00	12	-11.8	1
VA 96W-326					30.79	15	21.4	11	28.68	14	9.2	16
JACKSON	14.50	8	29.5	15	34.89	19	29.9	20	45.25	20	18.5	25
AGRIPRO MASON	15.83	10	25.1	11	36.82	23	17.5	7	45.11	19	7.8	15
PIONEER 2580	13.67	7	26.0	12	36.53	22	31.6	22	47.82	21	13.0	21
MADISON	18.67	12	22.9	9	28.99	13	22.0	13	54.24	25	3.0	8
VA 96W-348					28.93	12	24.1	14	39.79	17	11.9	19
PIONEER 2684	21.67	13	27.8	14	33.84	18	39.5	26	48.67	22	2.4	5
SS 550					30.35	14	30.7	24	52.30	24	18.1	12
FFR 555	22.83	16	40.9	16	43.52	27	42.9	28	51.05	23	22.3	27
PIONEER 2643	22.67	14.5	26.9	13	40.67	25	38.1	25	57.30	26	15.8	22
SISSON					28.42	11	36.3	21	43.87	18	6.4	24
POCAHONTAS	22.67	14.5	21.3	6	52.78	28	35.9	23	61.11	27	14.8	23
COKER 9835	33.17	17	46.9	17	42.44	26	41.0	27	80.71	29	34.9	29
GORE	48.00	18	48.0	18	68.68	29	49.3	29	61.31	28	23.5	28

* Lines are ranked in ascending order based upon Mean FHB index values over all environments.

Appendix G. Comparison of ranks for percentage FDK and percent grain yield loss for genotypes for each environment.

	1997-98 Montgomery Co., VA				1999-00 Montgomery Co., VA				1999-00 Westmoreland Co., VA			
LINE *	% FDK	FDK RANK	YIELD % LOSS	LOSS RANK	% FDK	FDK RANK	YIELD % LOSS	LOSS RANK	% FDK	FDK RANK	YIELD % LOSS	LOSS RANK
ERNIE	12.67	4	5.7	2	52.67	13	10.9	3	28.50	8	5.0	10
NY 87048W-7388					36.67	4	8.3	1	11.17	2	2.6	6
AGRIPRO PATTON					45.17	7	9.5	2	21.17	3	2.0	7
INW 9824	8.50	1	16.6	4	53.50	14	14.8	5	21.50	5	4.6	9
IL 94-1909					30.33	2	21.1	12	10.17	1	-2.1	2
FREEDOM	14.67	5	3.3	1	64.67	18	17.6	9	34.50	9	-0.3	3
ROANE	12.17	3	15.3	3	34.17	3	7.1	4	22.50	6	9.8	13
COKER 9803	15.03	6	21.6	8	43.00	6	26.4	17	38.33	10	11.5	18
CAYUGA					47.50	9	20.7	10	41.00	12	13.1	20
OH 552					65.00	19	15.5	6	46.33	15	0.4	4
PIONEER 2552	17.83	9	20.9	7	51.50	12	29.6	19	57.50	18	18.8	26
AGRIPRO FOSTER	9.33	2	19.2	5	48.50	10	24.6	15	24.00	7	8.2	14
QUANTUM 706					30.00	1	17.7	8	21.33	4	9.3	17
WAKEFIELD			22.5	10	58.33	15	27.7	18	43.83	14	6.6	11
VA 96W-329					70.00	21	26.5	16	40.67	11	-11.8	1
VA 96W-326					47.17	8	21.4	11	42.00	13	9.2	16
JACKSON	27.33	15	29.5	15	69.83	20	29.9	20	63.50	21	18.5	25
AGRIPRO MASON	16.17	7	25.1	11	41.00	5	17.5	7	46.83	16	7.8	15
PIONEER 2580	25.67	14	26.0	12	86.33	27	31.6	22	76.67	24	13.0	21
MADISON	20.50	11	22.9	9	84.19	25	22.0	13	77.83	26	3.0	8
VA 96W-348					60.50	16	24.1	14	67.33	22	11.9	19
PIONEER 2684	23.17	12	27.8	14	70.25	22	39.5	26	61.00	20	2.4	5
SS 550					50.67	11	30.7	24	54.83	17	18.1	12
FFR 555	18.17	10	40.9	16	73.00	23	42.9	28	83.17	28	22.3	27
PIONEER 2643	16.67	8.0	26.9	13	86.17	26	38.1	25	77.67	25	15.8	22
SISSON					61.33	17	36.3	21	60.00	19	6.4	24
POCAHONTAS	23.67	13.0	21.3	6	73.83	24	35.9	23	71.00	23	14.8	23
COKER 9835	31.33	16	46.9	17	86.67	28	41.0	27	84.50	29	34.9	29
GORE	32.50	17	48.0	18	89.67	29	49.3	29	78.83	27	23.5	28

* Lines are ranked in ascending order based upon Mean FHB index values over all environments.

Appendix H. Comparison of ranks for FHB index and percent grain volume weight loss for genotypes for each environment.

	1999-00 Montgomery Co., VA				1999-00 Westmoreland Co., VA			
LINE *	**FHB INDEX**	**INDEX RANK**	**GVW % LOSS**	**LOSS RANK**	**FHB INDEX**	**INDEX RANK**	**GVW % LOSS**	**LOSS RANK**
ERNIE	9.13	1	6.5	6	24.84	9	5.5	19
NY 87048W-7388	19.87	4	4.1	3	7.52	1	0.0	3.5
AGRIPRO PATTON	10.03	2	4.4	5	20.64	7	2.1	7
INW 9824	19.62	3	4.3	4	21.58	8	2.8	10
IL 94-1909	24.51	9	8.4	11	9.59	2	1.9	6
FREEDOM	32.09	16	6.9	8	15.46	4	-1.4	2
ROANE	22.55	7	1.8	1	28.10	13	2.4	8
COKER 9803	23.49	8	8.6	12.5	26.92	11	6.0	21
CAYUGA	32.59	17	7.9	10	9.71	3	6.1	22
OH 552	20.94	5	3.9	2	25.11	10	3.1	11.5
PIONEER 2552	22.33	6	9.2	16	34.96	15	4.9	17
AGRIPRO FOSTER	38.35	24	8.6	12.5	19.60	6	5.1	18
QUANTUM 706	35.45	21	7.2	9	17.97	5	2.7	9
WAKEFIELD	35.12	20	10.5	18	35.29	16	4.2	15
VA 96W-329	28.05	10	8.9	15	28.00	12	-4.7	1
VA 96W-326	30.79	15	10.9	21	28.68	14	1.3	5
JACKSON	34.89	19	10.9	21	45.25	20	9.0	24
AGRIPRO MASON	36.82	23	6.6	7	45.11	19	0.0	3.5
PIONEER 2580	36.53	22	11.9	21	47.82	21	4.8	16
MADISON	28.99	13	8.8	14	54.24	25	5.9	20
VA 96W-348	28.93	12	9.4	17	39.79	17	7.8	23
PIONEER 2684	33.84	18	14.6	27	48.67	22	3.8	14
SS 550	28.42	11	10.8	19	43.87	18	11.4	26
FFR 555	43.52	27	13.3	25	51.05	23	12.0	27
PIONEER 2643	40.67	25	15.6	28	57.30	26	13.3	28
SISSON	30.35	14	12.1	23	52.30	24	3.2	13
POCAHONTAS	52.78	28	12.9	24	61.11	27	3.1	11.5
COKER 9835	42.44	26	14.4	26	80.71	29	14.0	29
GORE	68.68	29	22.5	29	61.31	28	10.4	25

* Lines are ranked in ascending order based upon Mean FHB index values over all environments.

Appendix I. Comparison of ranks for percentage FDK and percent grain volume weight loss for genotypes for each environment.

	1999-00 Montgomery Co., VA				1999-00 Westmoreland Co., VA			
LINE *	**% FDK**	**FDK RANK**	**GVW % LOSS**	**% LOSS RANK**	**% FDK**	**FDK RANK**	**GVW % LOSS**	**% LOSS RANK**
ERNIE	52.67	13	6.5	6	28.50	8	5.5	19
NY 87048W-7388	36.67	4	4.1	3	11.17	2	0.0	3.5
AGRIPRO PATTON	45.17	7	4.4	5	21.17	3	2.1	7
INW 9824	53.50	14	4.3	4	21.50	5	2.8	10
IL 94-1909	30.33	2	8.4	11	10.17	1	1.9	6
FREEDOM	64.67	18	6.9	8	34.50	9	-1.4	2
ROANE	34.17	3	1.8	1	22.50	6	2.4	8
COKER 9803	43.00	6	8.6	12.5	38.33	10	6.0	21
CAYUGA	47.50	9	7.9	10	41.00	12	6.1	22
OH 552	65.00	19	3.9	2	46.33	15	3.1	11.5
PIONEER 2552	51.50	12	9.2	16	57.50	18	4.9	17
AGRIPRO FOSTER	48.50	10	8.6	12.5	24.00	7	5.1	18
QUANTUM 706	30.00	1	7.2	9	21.33	4	2.7	9
WAKEFIELD	58.33	15	10.5	18	43.83	14	4.2	15
VA 96W-329	70.00	21	8.9	15	40.67	11	-4.7	1
VA 96W-326	47.17	8	10.9	21	42.00	13	1.3	5
JACKSON	69.83	20	10.9	21	63.50	21	9.0	24
AGRIPRO MASON	41.00	5	6.6	7	46.83	16	0.0	3.5
PIONEER 2580	86.33	27	11.9	21	76.67	24	4.8	16
MADISON	84.19	25	8.8	14	77.83	26	5.9	20
VA 96W-348	60.50	16	9.4	17	67.33	22	7.8	23
PIONEER 2684	70.25	22	14.6	27	61.00	20	3.8	14
SS 550	50.67	11	10.8	19	54.83	17	11.4	26
FFR 555	73.00	23	13.3	25	83.17	28	12.0	27
PIONEER 2643	86.17	26	15.6	28	77.67	25	13.3	28
SISSON	61.33	17	12.1	23	60.00	19	3.2	13
POCAHONTAS	73.83	24	12.9	24	71.00	23	3.1	11.5
COKER 9835	86.67	28	14.4	26	84.50	29	14.0	29
GORE	89.67	29	22.5	29	78.83	27	10.4	25

* Lines are ranked in ascending order based upon Mean FHB index values over all environments.

Matthew Randolph Chappell

Vita

Matthew Randolph Chappell was born on October 7, 1976 in Richmond, VA. Mother Mary C. Williams and stepfather Gene F. Williams of Dinwiddie County, VA, reared him with support of grandfather George Chappell. His secondary education was obtained at Dinwiddie County High School in Dinwiddie, VA where he graduated in June of 1994. His undergraduate education was carried out at Virginia State University, Sam Houston State University, and Virginia Polytechnic Institute & State University. He graduated with a B.S. degree in Horticulture from Virginia Polytechnic Institute & State University in May of 1998. From May of 1998 until August of 2000 Matthew was enrolled as a Master's student and worked on research projects within the small grains breeding group of the Crop and Soil Environmental Sciences Department at Virginia Polytechnic Institute and State University. Matthew was awarded a William J. Fulbright Fellowship for study abroad which enabled him to complete a comprehensive survey of European *Triticum* genotypes for resistance to *F. culmorum* and *F. graminearum*. This research was conducted at the Swedish University for Agricultural Science in Alnarp, Sweden from September of 2000 until July of 2001. Upon return to the United States, Matthew resumed his studies and duties within the small grains breeding group at Virginia Polytechnic Institute & State University in addition to completing his Master's thesis.