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GENETIC VARIATION FOR FUSARIUM HEAD BLIGHT RESISTANCE IN SOFT RED WINTER WHEAT

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ABSTRACT OF THESIS

GENETIC VARIATION FOR FUSARIUM HEAD BLIGHT RESISTANCE IN SOFT RED WINTER WHEAT

Fusarium graminearum, the causative agent of Fusarium head blight, is an economically important pathogen of wheat (*Triticum aestivum*). Breeding Fusarium head blight (FHB) resistant wheat requires knowledge of the underlying genetic control of FHB resistance.

Two nine-parent diallel analyses were completed in greenhouse and field environments. Combining abilities, variance component ratios, and narrow sense heritabilities for FHB resistance and deoxynivalenol levels were calculated. Significant general and specific combining ability effects were observed. Resistance to FHB seems to be mostly controlled by additive genetic effects with some dominance noted in the field. Resistance noted in the greenhouse environment may not hold up in the field.

Genetic parameters for FHB resistance and four related traits were estimated in three populations. Moderate to high broad sense heritabilities for FHB severity and Fusarium damaged kernels (FDK) were observed. Incidence of FHB had low to moderate broad sense heritabilities. Correlations between FDK and severity and FDK and incidence were moderate and low, respectively, and do not support indirect selection for FHB severity or incidence based on FDK data alone. Substantial predicted gains from family selection were observed and therefore selection of FHB resistant wheat lines should be based on family means and not individual selection.

KEYWORDS: Fusarium head blight, deoxynivalenol, soft red winter wheat, heritability, resistance

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THESIS

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GENETIC VARIATION FOR FUSARIUM HEAD BLIGHT RESISTANCE IN SOFT RED WINTER WHEAT

THESIS

A thesis submitted in partial fulfillment of
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By

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Lexington, Kentucky

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Chapter 1

Introduction

Fusarium head blight (FHB), or head scab, caused by *Fusarium graminearum* is a historically devastating disease of wheat and barley throughout the world. The People s Republic of China, Canada, parts of southern Africa, eastern Europe, South America, and the United States all have recorded Fusarium head blight outbreaks and each country continues to struggle with this destructive disease.

Bai and Shaner (1994) reported that wheat scab can greatly reduce grain yield and quality. Diseased kernels are commonly classified by the term tombstones . These severely shriveled kernels are most commonly blown out of the combine due to their minute weight and not even harvested. Tombstones that manage to be harvested further harm profits by reducing test weight.

Bai and Shaner (1994) reported further losses from head scab caused by the production of mycotoxins. The mycotoxin deoxynivalenol (DON) can be produced by *F. graminearum* on wheat kernels and has been linked with livestock feed refusal.

Lower yields, reduced quality, and DON contamination have greatly affected the prices received for grain by wheat and barley producers. Windels (2000) reported that wheat and barley losses caused by scab epidemics in the US during the 1990 s are estimated at close to $3 billion. Farmers in the Red River Valley of North Dakota, USA, Minnesota, USA, and Manitoba, Canada have been ruined by successive years of head scab outbreaks that have forced some farmers to bankruptcy.

In Kentucky, almost the entire wheat crop is planted following corn, which is an alternate host for the perfect stage of *F. graminearum*. The prevalent cropping system of no till or minimal till wheat production may influence FHB levels by providing sufficient inoculum levels. Incorporating FHB resistance into soft red winter wheat is considered to be the most effective control strategy (Mesterhazy et al., 1999). Wheat breeding programs across the United States are feverishly searching for resistance in adapted wheat lines. To confound the problem of developing resistant wheat cultivars, most

resistant genotypes are susceptible to other common diseases, lower yielding, and do not meet end-use quality standards (Buerstmayr et al., 1996).

One step in the process of breeding FHB resistant winter wheat lines is to determine the nature of the genetic control of the trait. Fusarium head blight resistance is considered to be a quantitative trait controlled by many genes and affected by environmental conditions (Mesterhazy, 1995, Miedaner et al., 2001). Different sources of resistance and different types of resistance have been identified (Mesterhazy, 1995). Given the complexity of this disease, this study was conducted to better understand the underlying genetic control of FHB resistance.

Chapter 2

Literature Review

Economic Losses Associated with Fusarium Head Blight

Fusarium head blight (FHB), or head scab, caused by *Fusarium graminearum* is a historically devastating disease of wheat and barley around the world. In 1890 J.C. Arthur recorded an outbreak of head scab in Indiana (Arthur, 1891). The People s Republic of China, Canada, parts of southern Africa, eastern Europe, South America, and the United States all have recorded Fusarium head blight outbreaks and each country continues to struggle with this destructive disease.

Bai and Shaner (1994) reported that wheat scab can greatly reduce grain yield and quality. Diseased kernels are commonly classified by the term tombstones . These severely shriveled kernels are most commonly blown out of the combine due to their minute weight and thus not even harvested. Tombstones that manage to be harvested further reduce profits by lowering test weight.

Bai and Shaner (1994) reported further losses from head scab caused by the production of mycotoxins. The mycotoxin deoxynivalenol, DON, can be produced by *F. graminearum* on wheat kernels and has been linked with livestock feed refusal. In 1993 the US Food and Drug Administration refocused their attention from a 2 ppm level of concern for DON in raw grain and enforced a standard of 1 ppm for finished flour products (Windels, 2000). A 1999 study in Minnesota found that 90.4% of hard red spring wheat samples taken exceeded a DON concentration of 2 ppm (Jones, 1999).

Lower yields, reduced quality, and DON contamination have greatly affected the prices received for grain by wheat and barley producers. Windels (2000) reported that wheat and barley losses caused by scab epidemics in the US during the 1990 s are estimated at close to $3 billion. Farmers in the Red River Valley of North Dakota, USA, Minnesota, USA, and Manitoba, Canada have been forced into bankruptcy by successive years of head scab outbreaks. Other farmers have been forced to gain additional employment off the farm. Farm auctions, foreclosures, retail business

failures, and credit difficulties have all increased in rural communities in the Red River Valley that are highly dependent on wheat and barley production (Windels, 2000). Windels further stated that FHB has increased in severity in recent years due to the combination of wet, humid weather conditions when plants are most susceptible and the ever increasing practice of conservation tillage (Windels, 2000).

Pathology and Epidemiology of *Fusarium graminearum*

Fusarium graminearum is the pathogen that has caused most of the outbreaks of FHB in the United States, Canada, South America, China, and Japan (Stack, 1999). *F. graminearum* is a fungal pathogen that can cause crown rot or head blight. Two groups of the pathogen have been devised based on their preference of plant tissue and ability to form perithecia (Sutton, 1982). Group 1 isolates prefer crowns of host plants and do not readily form perithecia. Group 2 isolates are associated with head blighting and do form perithecia easily both in culture and in nature. Group 2 isolates are those that are associated with FHB.

Symptoms of FHB include premature blighting or bleaching of the wheat head before anthesis. Water soaked lesions may appear on the florets at the onset of the disease. Salmon colored light pink spore masses may become evident on the florets of infected heads under heavy disease pressure. Grains from infected florets may not form at all or may be characteristic tombstones . Tombstone grains are shriveled, much smaller than normal, and pale white, or pinkish in color.

Inoculum sources are mainly thought to include corn residue and other crop debris that give rise to spore producing perithecia. Ascospores and macroconidia are the infective fungal vehicles. Ascospores are mainly 3-septate, hyaline, and relatively uniform in size (Sutton, 1982). Macroconidia are more variable in size and can have 3 to 7 septations.

Inoculum production is favored by warm moist conditions in temperatures ranging from 20 to 30°C. Both wind and rain-splash dispersal techniques have been described for *F. graminearum* (Parry et al., 1995).

No specific pathogenic races of *F. graminearum* have been reported. Variability among isolates does exist however. Bai and Shaner (1996) reported variability among isolates in their ability to cause FHB, with Chinese isolates causing more disease severity. Mesterhazy (1988) also reported different disease severities depending on the isolate used. Isolates also vary in their ability to produce conidia, in the growth rate of mycelium, and in the production of deoxynivalenol. For these reasons, using a mixture of different isolates and isolates from different origins is recommended when conducting FHB disease screening tests.

In an experiment designed to determine interactions between eight *Fusarium* isolates and nine wheat genotypes, Bai and Shaner (1996) reported that the six isolates from the United States did not produce significantly ($p=0.05$) different mean disease severities in the set of genotypes. The two isolates from China did produce greater scab severity than the US isolates. More importantly, no genotype by isolate interaction was found. Each of the eight isolates repeatedly identified the most resistant and most susceptible genotypes with total agreement.

Mesterhazy et al. (1999) did report a significant ($p<0.001$) genotype by isolate interaction in one set of multiple year data yet in another set of multiple year data this interaction was not significant ($p>0.05$). Mesterhazy et al. continues to point out that in the experiment where he found a significant genotype by isolate interaction this interaction was only significant for the severity trait and not for kernel ratings or DON levels. Furthermore, the genotype by isolate interaction was very low when compared with the main effects of genotypes, isolates, and years. Of most significance was the year by isolate interaction leading to the conclusion that the environmental conditions surrounding the development of the disease have the most influence on the aggressiveness of the isolates.

Types and Sources of Resistance to *Fusarium* Head Blight in Wheat

Mesterhazy (1995) proposed five mechanisms for resistance to FHB. They were type I: resistance against initial infection, type II: resistance to pathogen spread, type III: resistance to kernel infection, type IV: yield tolerance, and type V: resistance to toxin

production. He later added two other types of resistance type VI: resistance to later blighting and type VII: resistance to blighting above the point of inoculation (Mesterhazy, personal communication, 2001). Mesterhazy also postulated that other agronomic factors such as plant height or dwarfness, presence of awns, high spikelet density, and late flowering date also contributed to FHB infections.

Resistance to FHB varies greatly among wheat genotypes. The Chinese cultivar Sumai 3 is the best known and most widely researched resistant cultivar. Sumai 3 s pedigree contains the two moderately susceptible cultivars Funo and Taiwanmai (Bai et al., 2000a). It has been rated as resistant to highly resistant in many screening experiments. Although Sumai 3 possesses very good type II FHB resistance, it is susceptible to other diseases and shatters easily.

In addition to the Chinese wheats such as Sumai 3 and its derivatives, two other sources of FHB resistance are wheats from eastern Europe and Brazil (Miedaner, 1997). From these areas come such cultivars as Arina and Frontana . Frontana has been the subject of considerable FHB research with inconsistent results. Singh et al. (1995) reported that the resistance of Frontana was controlled by the additive interaction of a minimum of three minor genes. Van Ginkel et al. (1996) reported that the resistance of Frontana and Ning , a derivative of Suami 3, was due to two different dominant genes in each cultivar with all four separate genes being different.

In a 1997 experiment in Hungary, 108 winter and spring wheat genotypes that ranged in susceptibility were screened for resistance to FHB (Lemmens et al., 1997). The team of researchers found highly significant differences between the genotypes for each FHB response variable. Buerstmayr et al. (1996) screened 96 winter wheat genotypes, 2 winter triticale genotypes, and 38 spring wheat genotypes in the field using a macroconidial spore suspension and reported that the most resistant spring wheat genotypes are more resistant than the most resistant winter wheat genotypes. Little work has been published which supports this assertion.

In a screening of 1076 accessions of the *Triticum* species, only 30 of the genotypes tested showed high resistance, in which only the inoculated floret was diseased (Yong-Fang et al., 1997). Interestingly these 30 genotypes were more resistant than the Sumai 3 genotype in this screening. Two of these 30 highly resistant

genotypes (PI36224 and NK+VI) were from the United States. The rest of the genotypes were of Chinese origin.

Ban reports that wild relatives of wheat such as *Agropyron (Elymus)* also contain resistance to FHB (Ban, 1997). One accession each of *Agropyron humidum* and *Agropyron ciliare* had higher resistance than Sumai 3 when sprayed with a conidial suspension in a growth chamber.

Resistance to FHB is considered to be a quantitative trait and is therefore likely governed by several genes (Buerstmayr et al., 1999). In a study in which six eastern European resistant lines and one susceptible line were intercrossed, heterosis for resistance was common, indicating that the parental genotypes possess different resistance genes (Buerstmayr et al., 1999). Bai and Shaner (1994) reported that because the genes for resistance in different cultivars appear to be on different chromosomes, crosses between these cultivars may yield transgressive segregates with greater resistance than any of the parents.

Type I and Type II Resistance Screening Methods

Screening for FHB resistance is carried out in many different ways and by many national and international breeding programs. Research is most often focused on type I and type II resistance which are: resistance to initial infection and resistance to spread throughout the spike, respectively. These two types of resistance can be screened for using distinct inoculation procedures. Grain spawn inoculum (infested corn (*Zea mays L.*) for example) or a macroconidial spray is most often used when screening for type I resistance. Type II resistance is measured through the use of a point inoculation technique that involves injecting a spore suspension directly into the wheat spikelet.

Type I resistance is often screened for in the field environment. Typically grain spawn inoculum is spread within the field during the time wheat is in the boot stage (Feekes scale 9) to allow the grain spawn enough time to produce perithecia. Grain spawn inoculum is usually prepared in the lab using either corn or wheat kernels. The grain spawn inoculation technique most closely simulates a natural epidemic (Rudd et al., 2001). Disease evaluations usually start around 21 days after anthesis. Many

different control genotypes with known resistance profiles are planted to gauge disease pressure and development. Early flowering resistant and susceptible genotypes and late flowering resistant and susceptible genotypes are planted throughout screening nurseries for this purpose. Disease scoring includes both the recording of incidence and severity. Incidence is usually recorded as a percentage of infected heads per total heads or area. Severity is recorded on an individual spike basis as the percent of spike blighted by FHB. Severity is commonly recorded for 10-25 spikes per plot. Incidence may be more related to type I resistance with severity being more related to type II resistance.

Macroconidial spore suspensions are also utilized for type I screenings instead of the grain spawn technique. Wheat plots are sprayed with the suspension at the time of anthesis. Disease ratings are similar to those reported for the grain spawn methods. This method can be utilized in both the field and greenhouse environments.

Type II screening methods are predominantly carried out in the greenhouse environment through the use of point inoculations in wheat spikes, although CIMMYT has outlined point inoculation techniques in the field (Gilchrist et al., 1996). Point inoculations are carried out by directly placing either *F. graminearum* ascospores or macroconidia into a single wheat floret. Hypodermic syringes, repeat dispensing pipets, small tufts of cotton soaked in spore suspensions, and colonized wheat and/or millet seeds have all been reported delivery devices (Rudd et al., 2001). When liquid spore suspensions are used for point inoculations, 2 μL to 10 mL of a spore suspension are injected into a single floret. Reported concentrations of spore suspensions can range from 50,000 to 100,000 spores/mL. Disease evaluations are usually made 21 days after inoculation although some programs record the progression of the disease over time by taking several weekly or daily ratings. In these cases area under the disease progress curves are calculated.

Regardless of the screening method used, timing of inoculum delivery is key. The delivery of *F. graminearum* spores to the wheat spike at the proper time is a main consideration in screening protocols. Most breeding programs and pathology experiments report that inoculations occur at anthesis (Feekes stage 10.5). This

developmental growth stage is reported as the most susceptible time for FHB to infect wheat spikes (Sutton, 1982).

Proper environmental conditions including high humidity are necessary for FHB to develop. This requirement for high humidity is most commonly satisfied in greenhouse experiments through the use of a humidity chamber where injected plants are placed for 12 to 72 hours depending on the protocol used (Rudd et al., 2001). Some researchers only place the injected spike into a small plastic bag to increase humidity (Teich and Michelutti, 1993). In field experiments elaborate sprinkler irrigation systems are utilized as well as other bagging techniques.

Although most researchers do provide additional humidity in some way, Evans and Dill-Macky (2001) have data to support that providing additional humidity may not be completely necessary. In a screening of four wheat genotypes under four irrigation volume treatments the researchers found that the four genotypes could be correctly differentiated for FHB incidence, severity, visually scabby kernels, and DON concentration most consistently under the no mist treatment.

Screening genotypes for FHB resistance is not a simple, quick, or cheap task. Other abiotic and biotic factors such as freeze damage and take-all (*Gaeumannomyces graminis var. tritici*) can mask classic FHB disease symptoms making disease evaluations difficult. Most often severity is recorded as the number of diseased spikelets over the total number of spikelets per spike. Counting the total number of spikelets on several individual spikes in replicated plots for many genotypes in more than one environment can be a daunting task even for a team of researchers. And finally, with intricate irrigation systems and the total number of person hours needed to score multiple genotypes, the cost of one FHB data point has been reported as six US dollars (Van Sanford et al., 2001).

Kernel Resistance

Mesterhazy (1995) reported the first evidence of kernel resistance to FHB in wheat. His research identified certain genotypes that were not significantly different in FHB severity ratings but were significantly different in their kernel infection ratings.

Genotypes that possess kernel resistance may be identified as susceptible when scored visually for FHB severity (i.e. based on spikelet symptoms), but when scored visually for seed infection they appear less susceptible. Mesterhazy et al. (1999) identified four genotypes that posses this type of resistance: Arina , Kr-Mon , Ni-Mon*Kr , and Mon-Ar .

Most often kernel resistance is characterized by the percentage of scabby or tombstone seeds. Most researchers report data on the amount of *Fusarium* damaged kernels or FDK.

Although Mesterhazy et al. (1999) outlines and discusses as many as five different types of resitance to FHB he concludes with the statement that breeding should focus on the visual assessment of symptoms as all of the other resistance factors (kernel resistance, DON accumulation, and yield tolerance) are more or less correlated with low FHB severity.

Deoxynivalenol Production and its Role in the Infection Process

Fusarium graminearum isolates differ in their ability to produce deoxynivalenol (DON) (Stack et al., 2000, Mesterhazy et al., 1999). It has been claimed by some researchers that DON is a virulence factor in FHB development. Stack et al. (2000) did not find a significant correlation between DON levels in the grain and isolate toxin potential. Twelve isolates that differed in virulence were used in this study. DON levels in the harvested grain were most highly correlated to the percent of tombstones and were not correlated to the isolate s toxin production.

Other researchers are not in agreement on this topic. Mesterhazy et al. (1999) did find a significant positive correlation ($r = 0.89$) between aggressiveness of isolates and their DON production. This was a larger study comparing eight isolates in 25 genotypes with three years of data.

Desjardins et al. (1996) produced several trichothecene-nonproducing mutant strains of *F. graminearum*. Deoxynivalenol is one toxin within the trichothecene toxin family. These trichothecene-nonproducing mutants colonized wheat heads, but produced significantly ($p<0.05$) less disease when tested in the field on both hard red

spring and soft red winter wheats. When tested on the soft red winter wheat Clark total yield per spike was reduced by 58% when compared to the wild-type strain inoculation. The trichothecene-nonproducing mutants were not significantly different from the non-inoculated control when total yield per spike was analyzed on the cultivar Clark. Desjardins et al. concludes by stating trichothecenes are virulence factors in wheat head scab .

F. graminearum macroconidia do not contain DON when tested in pure form outside a host. But when injected into barley, toxin was detected in the host as early as 36 hours post-inoculation (Evans et al., 2000). Snijders and Krechting (1992) report that DON is transported from the chaff to the young kernel and the pathogen then colonizes the kernel. When different floral parts of a wheat head are tested individually DON concentrations are highest in the rachis (Sinha and Savard, 1997).

Deoxynivalenol, as well as fungal spores, can be isolated from healthy looking seed. Out of 100 healthy looking normal seeds from a commercial field in Ontario, Sinha and Savard (1997) found detectable DON levels in 52 seeds. This does not support the selection of low toxin producing varieties based on visual seed evaluation alone.

Mesterhazy et al. (1999) postulated from their research that a cultivar has a significant influence on DON production in the infected tissue. Miller et al. (1985) also discussed this phenomenon in their research in which resistant genotypes prevented synthesis or promoted degradation of the toxin.

The role and significance of DON in FHB resistance remains unclear at the present time. What is clear is that the level of DON in a particular genotype is a result of the genotype itself, the fungal isolate, the environment and the interactions of all of these factors.

Genetic Parameters and Chromosomal Locations of Fusarium Head Blight Resistance Genes

Significant genetic variation for FHB resistance exists in wheat. Resistance is thought to be controlled by a few genes with major effects and other numerous genes with minor effects (Bai et al., 1999, Waldron et al., 1999, Snijders, 1990b). Many

quantitative trait loci (QTL) experiments have been undertaken with the goal of finding the genes or quantitative trait loci (QTL) responsible for FHB resistance in wheat. Table 2.1 provides a summary of some of these studies. Most frequently, resistance QTL have been mapped to chromosomes 3B, 5A and 6B.

Bai et al. (1999) identified eleven AFLP markers that showed a significant relationship with type II resistance in a spike. They also discovered one major QTL between the AFLP markers AAC/CGAC3 and GCTG/CGAC1 on linkage group 7 that explained up to 60% of the genetic variation recorded in their greenhouse injection experiment. Later research also identified this same QTL for lower DON levels (Bai et al., 2000b). They proposed that marker assisted selection (MAS) based on these markers would serve a good potential.

Waldron et al. (1999) discovered two major QTL: one located on 3BS and the other on 2AL. They also mapped RFLP markers to each of these QTL and reported markers Xcdo981 and XkuH16 would map to each of these QTL respectively. Using the three RFLP markers Xcdo981, XkuH16, and Xwg909, 29.5% of the phenotypic variation was explained in the cross Sumai 3 × Stoa. Marker assisted selection based on these markers has also been suggested.

Bai et al. (2000a) generated eleven different resistant × susceptible crosses and found that the additive-dominance effects model best fit 8 out of the 11 crosses. Other more complex models were also significant including some models containing dominance and epistatic effects. However, the authors found that when compared to the additive effects, the dominant and epistatic effects accounted for only a small portion of the genetic effects.

Snijders (1990b) also reported that additive effects of resistance are larger than dominance effects and suggested that epistasis may control some of the variation when he screened 45 crosses with *Fusarium culmorum*.

Waldron et al. (1999) report data that show susceptible cultivars (Stoa in their particular research) may contain resistance alleles not found in resistant cultivars (Sumai 3). These resistance alleles found in susceptible genotypes, when combined with other resistant alleles found in resistant genotypes, could result in increased levels of resistance.

Breeding for Fusarium Head Blight Resistance

Wheat breeders across the United States are feverishly searching for FHB resistance in adapted wheat lines and cultivars. The federal government appropriated $5 million dollars of funding in fiscal year 2001 through the US Wheat and Barley Scab Initiative to support efforts in finding cultural, chemical, and breeding methods to control FHB (http://www.scabusa.org; verified January 28, 2002). Both national and international meetings and forums for breeders and other researchers have been held to facilitate the exchange of information and the advancement toward the common goal of reducing FHB epidemics.

Marker assisted selection (MAS) for FHB resistance is receiving a great deal of attention as researchers search to find the genes responsible for resistance. Marker assisted selection for FHB resistance has been proposed based on the quantitative nature of the trait and the expensive cost of adequately rating genotypes through traditional screening nurseries.

Van Sanford et al. (2001) proposed the establishment of a National Genotyping Center in the Unites States to help breeding programs reach the goal of FHB resistant cultivars. The proposed center would rely heavily on marker assisted selection methods to help breeding programs release FHB resistance genotypes. Marker assisted selection is most appropriate when traits of interest are difficult and costly to measure (Yousef and Juvik, 2001) as is the case for FHB resistance.

Two SSR markers on 3BS (Xgwm389 and Xgwm493) were found to be associated with FHB resistance in a Ning7840/Clark population (Zhou et al., 2000). Zhou went on to propose that these flanking markers could directly be used for MAS for FHB resistance in wheat due to their high stability and repeatability.

Along with MAS, traditional breeding methods such as the pedigree method and single seed descent have proven useful in breeding for FHB resistance in wheat (Rudd et al., 2001). Yang et al. (2000) have also shown that recurrent selection is highly successful in producing FHB resistant selections that are also agronomically desirable.

Gains in breeding for FHB resistance in the United States have been reported without the use of the well-known Asian resistant sources (Rudd et al., 2001). Incidental sources of resistance have been found through the routine screening of elite materials in U.S. breeding programs. Several resistant varieties including 2375 , Ernie (McKendry et al., 1995), and Freedom (Gooding et al., 1997) have been identified through these routine screenings (Rudd et al., 2001). These lines are more favorable to breeders than Sumai 3 derived lines because they are well adapted, have acceptable agronomic characteristics, and meet end-use quality standards.

Breeding FHB resistant genotypes will most assuredly take time and diligent efforts of researchers given the complexity of FHB resistance, and the quantitative control of this trait effective resistance will most likely involve some use of MAS and multiple quantitative trait loci.

Table 2.1: Proposed chromosomal location of Fusarium head blight resistance quantitative trait loci in diverse wheat populations, 1982-2000.

Genotype Tested	Chromosome Locations of Reported Resistance QTL s	Method Used*	Author	Genotype Classification	Genotype Market Class
U-136.1	5A, 1B, 3B, 4B, 6B, 6D	Back-cross reciprocal monosomic analysis	Buerstmayr, Lemmens, and Ruckenbauer (1997)	Resistant	Hungarian winter wheat
U-226.1	3A, 3B, 6B, 4D	Back-cross reciprocal monosomic analysis	Buerstmayr, Lemmens, and Ruckenbauer (1997)	Susceptible	Hungarian winter wheat
RIL* of Sincron x F1054W	T1BL.1RS and 1D	Allele association	Ittu, Saulescu, Hagima, Ittu, and Mustatea (2000)	Resistant x Susceptible RIL	Non-Chinese winter wheat

*Recombinant inbred line (RIL), Restriction fragment length polymorphism (RFLP), Amplified fragment length polymorphism (AFLP), Single sequence repeat (SSR)

Table 2.1 (cont): Proposed chromosomal location of Fusarium head blight resistance quantitative trait loci in diverse wheat populations, 1982-2000.

Genotype Tested	Chromosome Locations of Reported Resistance QTL s	Method Used*	Author	Genotype Classification	Genotype Market Class
Sumai 3	2A, 5A, 1B, 6D, 7D	Monosomic Analysis	Yu (1982)	Resistant	Chinese Spring wheat
F_5 derived RIL of Sumai 3 x Stoa	3BS, 2AL, 4BL, 6BS,	RFLP	Waldron, Moreno-Sevilla, Anderson, Stack, and Frohberg (1999)	Resistant x Moderately Susceptible	Chinese Spring x American Spring wheat
RIL of Ning 7840 x Clark	7B	AFLP	Bai, Kolb, Shaner, Domier (1999)	Resistant x Susceptible	Chinese Spring x American winter wheat
$F_{2:3}$ Ning 7840 x Freedom	3BS, 3AL, 2AS, 7BS, 6BS, 5AL	SSR	Gupta, Lipps, and Campbell (2000)	Resistant x Susceptible	Chinese Spring x American winter wheat

*Recombinant inbred line (RIL), Restriction fragment length polymorphism (RFLP), Amplified fragment length polymorphism (AFLP), Single sequence repeat (SSR)

Chapter 3

Diallel Analysis of Resistance to Fusarium Head Blight in Soft Red Winter Wheat

Introduction

Among the many diseases that infect wheat, Fusarium head blight (FHB) is one that causes many negative effects not only to the plant itself but also to its harvested grain. FHB, caused by the fungus, *Fusarium graminearum*, reduces test weight and yield, and produces deoxynivalenol (DON), a harmful toxin in harvested grain (Bai and Shaner, 1994, Tuite et al. 1990). The release of genetically resistant cultivars is considered to be the most effective control against FHB. Resistance to FHB is quantitative in nature and found in several spring wheat cultivars including the Chinese Sumai 3 . However, resistance to FHB in adapted winter wheats is not as pronounced (Buerstmayr et al., 1996).

Objectives for this study included gaining a better understanding of (FHB) resistance in Kentucky-adapted soft red winter wheat (SRWW) lines, identifying promising combinations of parents for the selection of improved breeding lines, and studying the relationship between the greenhouse and field screening environments.

Literature Review

Plant breeders should be aware of the complexity of the resistance to *Fusarium* (Mesterhazy, 1995).

Significant genetic variation for FHB resistance exists in wheat. Mesterhazy (1995) proposed five mechanisms for resistance to FHB. They included: 1) resistance against initial infection, 2) resistance to pathogen spread, 3) resistance to kernel infection, 4) yield tolerance, and 5) resistance to toxin production. He later included the addition of two more types of resistance: 6) resistance to late blighting and 7) resistance to spread above the point of inoculation (Mesterhazy, personal communication, 2001).

Resistance to FHB is considered to be a quantitative trait and is therefore likely governed by several genes (Buerstmayr et al., 1999). Resistance is thought to be controlled by a few genes with major effects and other numerous genes with minor effects (Bai et al., 1999, Waldron et al., 1999, Snijders, 1990b). Bai et al. (2000a) suggests that additive genetic effects are the more important factor in controlling resistance to FHB although some dominant and epistatic effects were found. Depending on the genotypes used, many researchers have reported that resistance to FHB is controlled by one to three genes (Bai et al., 2000a) one to six genes (Snijders, 1990b) or two genes (Van Ginkle et al., 1996).

Fusarium graminearum is the pathogen that has caused most of the outbreaks of FHB in the United States, Canada, South America, China, and Japan (Stack, 1999). FHB can greatly reduce grain yield and quality and further losses from FHB are caused by the production of mycotoxins. The mycotoxin deoxynivalenol, DON, can be produced by *F. graminearum* on wheat kernels and has been linked to livestock feed refusal. Genotypes have been identified that produce minimal DON even when highly infected with FHB (Bai et al., 2001). Correlations between DON production and FHB severity have been reported as both high (Miedaner et al., 2001) and low (Liu et al., 1997). Testing thousands of lines in a breeding program each year may not be feasible for routine selection of low DON-producing lines. Therefore, selection based on other related characters such as FHB severity or percent scabby seed has been proposed (Bai et al, 2001, Mesterhazy, 1999).

Fusarium culmorum is the pathogen that induces FHB in most of Europe. Diallel analyses among winter wheat lines have been completed using *F. culmorum* isolates (Buerstmayr et al, 1999, Snijders, 1990a). Both Buerstmayr et al. (1999) and Sniders (1990a) found that the general combining ability effects were most important; and therefore, resistance would be uniformly passed on to all progeny. Buerstmayr et al. (1999) did report minor ($P<0.05$) reciprocal effects for *Fusarium* damaged kernels but not for FHB severity.

In spring wheats several FHB resistant cultivars have been developed through the use of the highly resistant Chinese spring line Sumai 3 or derivatives from it. McVey (Busch et al., 2001) and Alsen (http://www.ag.ndsu.nodak.edu/alsen.htm,

verified January 25, 2002) are two such spring wheat cultivars. The resistance within Sumai 3 has also been used to develop some winter wheats including the Pioneer cultivar 25R18 . The narrow use of resistance sources may become problematic in the future assuming that the pathogen contains genetic diversity within its natural population.

This study was completed to: 1) gain a better understanding of FHB resistance in Kentucky-adapted soft red winter wheat lines, 2) identify promising combinations of parents for the selection of improved breeding lines, and 3) study the relationship between the greenhouse and field screening environments.

Materials and Methods

F_1 Crossing Cycle

Two nine parent diallel crossing schemes were constructed. One diallel focused on FHB spikelet severity (diallel 1) and the other on deoxynivalenol production (diallel 2). Each of the diallels contained parents that ranged from susceptible to resistant genotypes (Table 3.1). All genotypes are adapted to the southeastern United States and have acceptable agronomic characters and end use qualities.

The parents of both diallels were artificially vernalized. Seeds were placed onto 2.5 in^2 blotter paper that had been soaked in a mixture of LSP (Thiabendazole) and Raxil (Thiram). The blotter paper and seeds were then put into small plastic bags. The seed packets were placed into a vernalization chamber set at 4°C on 12 November 1999 where they remained for eight weeks.

Seedlings were transplanted into greenhouse pots on 7 January 2000. The soil mixture used was two parts soil, two parts Pro-Mix and one part sand. Seedlings were grown under artificial lighting and were fertilized with a water soluble 20-20-20 fertilizer four times. During the vegetative stage of plant growth daylength was 12 hours; during flowering daylength was 16 hours. Approximate greenhouse temperatures were 70°F during the day and 50°F during the night. Emasculations were completed and pollination was via the approach method. Reciprocal F_1 crosses were also made when possible to investigate the maternal effects of FHB (*F. graminearum*) resistance.

Macroconidial spore suspension

Twelve cultures of *Fusarium graminearum* were obtained from scabby wheat seed by surface sterilization and plating onto acidified potato dextrose agar. Ten of these isolates were obtained from different geographical regions of Kentucky; one isolate was obtained from Indiana and one from Virginia. To induce sporulation, mycelium from the cultures was plated onto carnation leaf agar. Plating a single-spore onto APDA ensured culture purity. The cultures were then increased on PDA.

Macroconidial suspensions were prepared by placing two mycelial plugs from a culture of *F. graminearum* in 100 mL of carboxymethylcellulose (CMC) liquid media. Flasks were placed on a shaker (115 rpm) for 2 weeks at 24°C. Spore suspensions were prepared by filtering the cultures through a 3.0 mm Millipore filter system. Macroconidia were resuspended in sterile water and streaked onto mung bean agar plates. The plates were incubated for 7 days then washed with sterile water. The washed suspension from each of the twelve isolates was then combined and calibrated to 600,000 spores/mL with the aid of a hemocytometer. From this spore suspension a 3 μL aliquot containing approximately 1,000 spores was injected into wheat spikes.

Greenhouse Screening

The F_1 progeny, reciprocals and their parents were vernalized on 17 August 2000 and transplanted into the greenhouse on 12 October 2000. Vernalization procedures and greenhouse management was the same as previously outlined. Ten seeds of each F_1 cross and parent were planted in a completely random design. Injections were made using a macroconidial spore suspension at the concentration of 1,000 spores/3μL distilled water. As each wheat spike reached anthesis, a central floret was marked using a permanent marker. This marked floret was then injected by dispensing 3μL of the spore suspension from a digital microliter pipette. After plants had been injected they were moved into a mist humidity chamber for three consecutive nights. Plants were removed from the mist humidity chamber on the fourth day and returned to the greenhouse bench. Plants were scored for FHB spikelet severity

twenty-one days post inoculation. FHB spikelet severity was calculated as the number of FHB infected spikelets over the total number of spikelets.

Field Screening

The same F_1 progeny and their parents were planted in small 5-seed hill plots on 15 October 2000 in Lexington, KY. Reciprocals were combined to ensure that enough seed was available for all F_1 crosses. Unfortunately due to low F_1 seed numbers, two parents and all of their corresponding progeny had to be eliminated from diallel 1, and one parent and all of its corresponding progeny had to be eliminated from diallel 2. Plots were hand-planted in a randomized complete block design with three replications. Row spacing between the hill plots was 61 cm.

An overhead mist irrigation system on an automatic self timer was installed to provide adequate moisture and humidity for an FHB epidemic. During the hours between 6 and 8 AM the irrigation came on for a duration of 5 minutes in 15 minute intervals. The irrigation schedule also included a 10 minute misting every 20 minutes during the hours between 8 and 10 PM.

Ten spikes per plot were injected with a macroconidial spore suspension at anthesis as described earlier. One significant addition was the covering of the injected spikes with glassine bags to guard against natural infection from wind-blown ascospores. The glassine bags were placed on the spikes at the time of injection (anthesis) and secured with a staple. The bags remained on the spikes until 21 days post injection at which time they were removed to record spikelet severity. The total number of spikelets per spike and the total number of infected spikelets per spike were recorded and a new glassine bag was fastened over the spike where it remained until harvest. At harvest maturity individual injected spikes were harvested. Auxiliary spikes within the hill plots that were not injected were also harvested. These auxiliary spikes were infected with FHB via wind-blown ascospores from nearby grain spawn inoculum that was spread within a neighboring FHB field screening nursery. Four control spikes were not injected but were covered with the glassine bags to investigate the effectiveness of the glassine bags at keeping out wind-blown ascospores. The glassine

bags proved to keep out the wind-blown ascospores as no symptoms were noted on the non-injected covered heads.

Deoxynivalenol Analysis

After harvest, the ten injected spikes from each hill plot from the field experiment were hand threshed in bulk. The auxiliary spikes within each hill plot were also harvested and threshed with a stationary thresher with the fan set at the lowest setting as to not blow out tombstones. All harvested samples were hand cleaned. Both the injected spikes and auxiliary spikes were analyzed for DON concentration in diallel 2. Only the auxiliary spikes in diallel 1 were analyzed for DON concentration.

A five gram sample of grain from each F_1 and parent was analyzed for DON using the EZ-Quant Vomitoxin Test Kit from the Diagnostix Company. Each sample was ground in a coffee grinder for 15 seconds. The coffee grinder was vacuumed between samples to protect against any cross-contamination. Twenty-five mL of distilled water was added to each ground sample and the remainder of the test was completed following the protocols contained within the EZ-Quant Vomitoxin Test Kit. Two aliquots from each DON extraction were pulled to provide replication when analyzing the injected spikes that were threshed in bulk. Two field replications were sampled for the DON analysis on the auxiliary spikes. Only seed from the field experiment was subjected to the DON analysis.

Seed Quality Evaluation

The harvested grain from the auxiliary spikes was visually inspected and evaluated for percentage of Fusarium damaged kernels (FDK) or tombstones . This was done by visually estimating the percent of Fusarium infected kernels. This method has been well correlated (r = 0.92) with other methods where actual counts of Fusarium infected kernels are completed (Dill-Macky et al., 2001).

Statistical Analysis

The data from the individual spikes in both the greenhouse and field studies were averaged to give genotype means. These genotype means (Tables 3.2, 3.11, 3.16, 3.20, 3.27, 3.34, 3.43, 3.48, 3.52, and 3.59) were used in all diallel analyses. Analysis of variance was performed on all variables of interest using a completely random design in the greenhouse experiment and a randomized complete block design in the field experiment. Correlations of interest were estimated by using SAS procedure CORR (SAS, 1990).

General combining abilities (GCA) and specific combining abilities (SCA) were calculated for traits of interest using various methods as described in Griffing (1956). In the greenhouse experiment in which reciprocals were planted, Griffing s Methods 1, 3 and 4 were used. Method 1 includes all genotypes, (the F_1 s, reciprocals, and parents). Method 3 includes the F_1 s and reciprocals but the parents are excluded. With Method 4, only F_1 s are included in the analysis. Method 2 includes one set of F_1 s and the parents. In the field experiment Griffing s Methods 2 and 4 were used.

Not all F_1 reciprocal crosses were made; thus, the diallel data set was not complete. In those situations in which missing data was a problem two solutions presented themselves. The first solution simply was to repeat the reciprocal mean twice and lose one degree of freedom in the analysis. The other solution was to eliminate those parents which had missing data points from the diallel in order to construct a complete diallel data set. Both solutions were used and are presented (Tables 3.3, 3.7, 3.35, and 3.39).

The parents chosen in the diallel crosses can be best regarded as a fixed set of parents when the trait being analyzed is the sole trait for which they were selected. For example, the parents in diallel 1 were specifically chosen based on their range of severity for FHB resistance without any regard to their DON production. Thus when analyzing diallel 1 for severity the chosen parents best fit the fixed model, yet when analyzing for DON concentration the chosen set of parents can be regarded as a random set of parents. Therefore, both fixed and random models were used and thus both Baker s (1978) component of variance ratios $2\sigma_g^2/(2\sigma_g^2 + \sigma_s^2)$ and narrow sense heritabilities were calculated for each diallel. Narrow sense heritabilities were estimated according to Griffing (1956) with the use of the following formula:

$$h^2 = \frac{2\sigma_g{}^2}{2\sigma_g{}^2+\sigma_s{}^2+\sigma_e{}^2}$$

where $\sigma_g{}^2$= the variance of the general combining ability

$\sigma_s{}^2$= the variance of the specific combining ability

$\sigma_e{}^2$= the residual error.

Results and Discussion

Diallel 1 (Severity Diallel)

Greenhouse Experiment

Parent, F_1, and reciprocal FHB severity means from diallel 1 screened in the greenhouse are presented in Table 3.2. The parents chosen for this diallel represent varying levels of FHB resistance ranging from the most susceptible Clark (76.93%) to the resistant 25R18 (5.17%). All are adapted soft red winter wheats. The overall severity mean in the greenhouse experiment was 18.02% for diallel 1. Ranking the nine parents in order of most resistant to most susceptible based on the severity means from the greenhouse screening results in 25R18 < Freedom < Patton < Ernie < Patterson < Foster < CK 9663 < 2555 < and Clark. 25R18 is regarded as resistant, and Freedom and Ernie both have reported to be moderately resistant to FHB. Both 2555 and Clark are regarded as highly susceptible and both are used as susceptible checks in many FHB screenings.

Method 1 Nine Parent Analysis. The analysis of variance table presented in Table 3.3 provides the analysis based on Griffing s Method 1. In this set of crosses, two data points were missing (Freedom x Patterson; and Freedom x 25R18). For these two data points, the cross means were simply repeated and substituted for the missing reciprocal means and then two degrees of freedom were subtracted from the error term. Highly significant differences existed among the genotypes for FHB severity. Both GCA and SCA effects were also highly significant. The GCA mean square was nearly ten times the magnitude of the SCA mean square. Reciprocal effects for FHB severity were significant at P=0.02.

The GCA effects presented in Table 3.4 indicate that Freedom, Patton, and 25R18 have good general combining ability for resistance to FHB. In fact, a glance at Table 3.2 reveals that Freedom, Patton, and 25R18 produced superior hybrids that were more resistant than the parents themselves. The genotypes Clark, CK 9663, and 2555 produced inferior hybrids as noted by their positive GCA effects. One observation of interest from these GCA effects, is that the cultivar Ernie did not impart any resistance to its progeny. A GCA effect of 0.68 was calculated for Ernie. Ernie is reported to be tolerant to FHB (McKendry, 1995) yet it did not pass on this tolerance to its progeny.

In Table 3.5 the SCA effects of the F_1 crosses are presented. One hybrid (Ernie/Clark) produced a significant (exceeds its standard error) positive SCA effect. In this specific hybrid a FHB severity mean of 58.44% was observed, which was much higher than the rest of the Ernie hybrids. Two hybrids (Freedom/Clark and Clark/25R18) had FHB severity means of 5.70% and 4.91%, respectively and each produced a significant negative SCA effect. These hybrids were as resistant as the most resistant parent 25R18, which had a FHB severity mean of 5.17%. The Freedom/Clark cross demonstrates that FHB resistance in winter wheat can be attained without the use of Chinese resistance sources.

Reciprocal effects were significant in the overall analysis and a significant reciprocal effect was observed for the cross Patterson/2555 (Table 3.6). The cross Patterson/2555 was more resistant that the cross 2555/Patterson. When used in this cross as a female 2555 increased susceptibility; therefore, based on this observation, when making crosses between resistant and susceptible genotypes the resistant parent should be used as the female. However, when used as a female in other crosses, 2555 reduced susceptibility although the reciprocal effects for these crosses were not significant. For example, the cross Freedom/2555 had a mean FHB severity of 13.73% while the cross 2555/Freedom had a mean FHB severity of 9.85%. So, always using the resistant genotype as the female in crosses may not prove wise. Although statistically significant, reciprocal effects were much less significant than GCA effects. Some maternal effects for FHB resistance in some winter wheat genotypes may exist, yet progress towards breeding a FHB resistant genotype should primarily focus on the

choice of the two parents and not on the choice of which one should be the maternal parent.

Baker s (1978) component of variance ratio was calculated to be 0.72 (Table 3.67). This ratio is further support to the conclusion that the GCA effects (and thus additive effects) are the predominant factor in controlling resistance to FHB. If one relaxes the traditional fixed model requirement, a narrow sense heritability estimate was estimated as 0.59 (Table 3.67).

Method 1 Seven Parent Analysis. Deleting the two parents that had missing data points (Patterson and 25R18) from the analysis produces a diallel of seven parents with no missing data. This seven parent diallel ANOVA table (Table 3.7) gives similar results to the nine parent diallel analysis when using Griffing s Method 1. Highly significant differences among the genotypes were observed along with GCA and SCA effects. Because one of the parents taken out of the diallel was Patterson, which had a significant reciprocal effect in the previous nine parent analysis, the reciprocal effects for the seven parent diallel were not significant at the 5% level. As observed in the previous analysis, the GCA mean square was much larger than the SCA mean square. The large GCA mean square in relation to the SCA mean square suggests that resistance to FHB must be controlled by additive effects.

Deleting Patterson and 25R18 from the analysis did not change the GCA effects substantially. Significantly negative GCA effects were observed for Freedom and Patton with significantly positive GCA effects observed for Clark, CK 9663, and 2555 (Table 3.8).

SCA effects for the seven parent diallel are given in Table 3.9. The two hybrids Freedom/Clark and Patton/Clark were more resistant with Ernie/Clark once again being more susceptible.

Reciprocal effects were not significant in the overall analysis, and thus no specific hybrids produced significant reciprocal effects (Table 3.10).

Baker s (1978) component of variance ratio was calculated to be 0.70 (Table 3.67). This high ratio leads to the conclusion that GCA effects are the principal factor in determining FHB resistance. If one relaxes the traditional fixed model requirement, a narrow sense heritability estimate was estimated as 0.60 (Table 3.67).

Method 3 Seven Parent Analysis. The F_1 and reciprocal genotype means used in the seven parent Method 3 diallel analysis are presented in Table 3.11. The Method 3 analysis of variance showed highly significant differences among the crosses in the greenhouse (Table 3.12). General combining ability and specific combining ability effects were also highly significant. Reciprocal effects were significant at the 5% level (p=0.0358). Based on this analysis, there seems to be some slight reciprocal effects in FHB resistance. The GCA mean square was predominant leading to the conclusion that most of the variation in these parents was additive.

The GCA effects for each parent are presented in Table 3.13. General combining ability is the average effect of the parent on the mean of all of its F_1 progeny. From this data it is shown, that on average Freedom, Patton and Foster produced more resistant F_1 crosses while Clark and CK 9663 produced more susceptible crosses. Therefore, Freedom, Patton, and Foster would be considered superior parents to use for breeding FHB resistant wheat in general. Although these parameter estimates pertain only to this set of parents, the breeder may choose to extrapolate these results to other potential parents. In that case, Freedom, Patton, and Foster would be used to lower susceptibility; while Clark and CK 9663 would be avoided for this purpose. Those parents which did not have significant GCA effects (Ernie and 2555 in this case) are not superior parents to use freely for rapid FHB resistance breeding.

Continuing to use a Method 3 analysis the SCA for each F_1 was calculated (Table 3.14). Specific combining ability is the effect of a specific F_1 cross when compared to all other F_1 crosses containing the two parents in that particular cross. Due to the high standard error of these estimates (SE = 9.83) not many SCA effects were significant. However, three hybrid combinations did produce significant ($P<0.05$) SCA effects. The hybrids Freedom/Patton and Ernie/Clark had positive SCA effects. These specific hybrid combinations are of no use for a breeding program as their FHB response was toward greater susceptibility. One hybrid of great interest is Freedom/Clark in which the SCA effect is significantly negative. This hybrid is superior to all of the other Freedom and Clark hybrids, and superior to Freedom, the moderately resistant parent. So when crossed specifically with Freedom, Clark does have some merit as an acceptable parent to use in breeding for FHB resistance. This is similar to

the situation observed in the cross of Sumai 3 x Stoa in which the susceptible parent Stoa actually contributed some resistance alleles (Waldron et al., 1999). The resistant Chinese line Sumai 3 was itself the result of crossing two moderately susceptible cultivars (Bai and Shaner, 1994). The difficulty for the breeder is that this determination can not be made without some analysis based on crosses (like a diallel) or on molecular marker data as was the case in the work of Waldron et al. (1999). Furthermore, the fact that the cross Ernie/Clark produces a significant positive SCA effect causes some speculation that the resistance alleles contained within Ernie are different than those contained within Freedom.

Reciprocal effects of each F_1 are shown in Table 3.15. Although the reciprocal effects were significant in the overall analysis, no specific hybrid had a reciprocal effect that exceeded the standard error.

Baker s component of variance ratio (Baker, 1978) was calculated as 0.84 (Table 3.67). Thus, additive effects are the leading contributor to FHB resistance. If one relaxes the traditional requirements of a fixed model analysis and calculates a narrow sense heritability, the estimate is 0.68 (Table 3.67).

Method 4 Nine Parent Analysis. The data presented in Table 3.16 are the F_1 genotype means from the two reciprocal observations for the nine parent diallel. Where only one reciprocal mean was observed that single mean was reported. Combining the reciprocal means was considered valid as only one hybrid produced a reciprocal effect that exceeded the standard error in the original Method 1 nine parent analysis.

Using the Method 4 analysis, there existed highly significant differences among the crosses, GCA effects, and SCA effects (Table 3.17). The GCA mean square dwarfed the SCA mean square by a power of 10. Additive effects of the genes controls the variation observed in these genotypes in the greenhouse screening environment.

GCA effects from this analysis identified Freedom, Patton, and 25R18 as possible parents to use toward breeding more FHB resistant winter wheat (Table 3.18). A significantly positive GCA effect was observed for Clark. On average, Clark was susceptible as were most of its progeny.

There were no SCA effects which were significant (Table 3.19).

Baker s (1978) component of variance ratio was calculated to be 0.87 (Table 3.67). Resistance to FHB must therefore be controlled by additive gene effects. If one relaxes the traditional fixed model requirement, a narrow sense heritability estimate was estimated to be 0.74 (Table 3.67).

Field Experiment — Severity

In the field screening, reciprocals were bulked to provide enough F_1 seed for most of the crosses except the crosses involving 25R18 and Freedom. These crosses and parents were therefore not planted in the field screening experiment. Average FHB severity for the parents and crosses are presented in Table 3.20. The average severity mean in the field was 39.04%, much higher than the greenhouse severity mean of 18.02%. In general, the field environment provided much greater disease pressure than the greenhouse environment. White cloudy mycelium developing on the injected spikes in the field that were covered with the glassine bags could be seen with the naked eye (Figure 3.1). Most likely this was due to the increased humidity under the glassine bags. FHB infected wheat spikes within the same field under the same irrigation schedule but not covered with a glassine bag did not produce this mycelial growth nor was such growth noted in the greenhouse experiments. Spikes that were not injected but were covered with glassine bags also did not produce any mycelial growth or symptoms (Figure 3.2).

Method 2 Analysis. The ANOVA table for the randomized complete block experiment in the field shows a significant difference among the three replications in the field (Table 3.21). The three replications in the field were blocked according to the relation of field to the prevailing wind. The spatial relation within the field environment could have contributed to the significant variation seen among the three field replications; therefore, blocking was effective. In any event, significant variation among the crosses was also identified in the field (Table 3.21). GCA and SCA effects were also both significant with the GCA mean square predominating.

Table 3.22 shows the GCA effects for the seven parents in the field. With Freedom and 25R18 missing from the set of parents, the best parents to use among

those remaining are Patton and Ernie. Patterson and CK 9663 report significant positive GCA effects.

SCA effects are given in Table 3.23, but the standard error precluded any effects from being significant.

Baker s (1978) component of variance ratio was calculated to be 0.69 (Table 3.67). This ratio is lower than the ratios observed in the greenhouse. If one relaxes the traditional fixed model requirement, a narrow sense heritability estimate was estimated to be 0.63 (Table 3.67).

Method 4 Analysis. The ANOVA shown in Table 3.24 reveals highly significant differences among the replications and crosses. Both GCA and SCA effects were also highly significant. A comparison of the greenhouse and field mean square errors from a Method 4 analysis (311.49 (Table 3.17) vs. 372.63 (Table 3.24)) yields the conclusion that the two environments had the same amount of unexplained variation.

General combining abilities were calculated using Griffing s Method 4. Based on this analysis, Patton and Ernie were the best parents to use noting their significantly negative GCA effects (Table 3.25). Patterson increased susceptibility noting its significantly positive GCA effect.

Looking at Method 4 SCA effects from the field (Table 3.26), there is a high standard error. This causes all of the SCA effects to be non-significant.

Baker s (1978) component of variance ratio was calculated to be 0.67 (Table 3.67). If one relaxes the traditional fixed model requirement, a narrow sense heritability estimate was estimated to be 0.62 (Table 3.67).

Field Experiment — DON

The DON concentrations within each F_1 and parent are presented in Table 3.27. The data in this table is the DON concentration reported in ppm as sampled from the auxiliary spikes within two replications of the hill plots. The DON concentrations from the injected heads from this diallel were not tested due to the preliminary finding that there was no significant difference among the crosses or parents when analyzing the injected heads from diallel 2 (Appendix A).

Method 2 Analysis. Variation among the two replications tested for DON concentration was significant (Table 3.28). This could be expected noting the significant variation in FHB severity among replications found in Table 3.21. There was also significant variation among the genotypes for DON concentration (Table 3.28). General combining ability effects and SCA effects were also significant with the GCA mean square being twice that of the SCA mean square.

General combining ability effects were calculated and are presented in Table 3.29. In general terms, Patton, Clark, Patterson, and CK 9663 were superior parents to use in aims of decreasing the level of DON produced. Ernie and 2555 increased the level of DON production in their hybrids.

A lower standard error estimate resulted in many of the SCA effects being declared significant (Table 3.30). Five such effects were significantly negative and of interest here. Patton/Patterson, Patton/Foster, 2555/Foster, Clark/CK 9663, and Foster/CK 9663 all had significantly negative SCA effects for DON concentrations. These crosses may posses some type V resistance (resistance to DON accumulation) mechanisms as described by Mesterhazy (1995).

Baker s (1978) component of variance ratio was calculated to be 0.36 (Table 3.67). Due to the fact that the parents chosen in this diallel were selected based on their range in FHB severity they can be considered a random set of parents with regard to their DON levels. Therefore, using a random model, a narrow sense heritability estimate was estimated to be 0.28 (Table 3.67).

Method 4 Analysis. In Table 3.31 the ANOVA for a Method 4 approach still reveals the significant variation among the field replications and crosses for the DON levels. And the GCA and SCA effects continue to be significant and in the same order of magnitude as the previous analysis.

General combining ability effects revealed that Patton, Clark, and CK 9663 are the better parents to use in breeding for low DON producing hybrids (Table 3.32). Patterson was identified as a superior parent in the Method 2 analysis but not in the Method 4 analysis. Comparing the calculated GCA effects for the two traits (FHB severity (Table 3.25) and DON levels (Table 3.32)) uncovers a unique situation. From Table 3.25, Ernie was identified as a parent having a significantly negative GCA effect

and thus would significantly reduce FHB severity in its hybrids. However from Table 3.32, it is shown that Ernie would increase DON levels in hybrids based on its significantly positive GCA effect for DON levels. The genotype Ernie may posses type II resistance to FHB spread within the spike but may not contain much type V resistance to DON levels.

Other comparisons between the FHB severity GCA effects (Table 3.25) and the DON GCA effects (Table 3.32) are compelling. Clark, on average, did not show much promise in reducing FHB severity given the positive GCA effect in Table 3.25. However, Clark might be recommended to a breeder interested in producing wheat lines with lower DON levels given the significantly negative GCA effect for Clark in Table 3.32. Coker 9663 and Clark were similar in terms of both FHB severity and DON levels. Coker 9663 would not be a choice in breeding for type II resistance but would be a candidate for breeding for type V resistance. Significantly negative GCA effects were observed for both traits in Patton. Patton would therefore contain both type II and type V resistance.

Three of the five hybrids identified as having significantly negative SCA effects through the Method 2 analysis were again identified through the Method 4 analysis with the hybrids 2555/Foster and Foster/CK 9663 being the two exceptions that were not declared significant under the Method 4 analysis (Table 3.33). One additional cross (Ernie/Patterson) had a significantly negative SCA effect; this cross did not produce a significant SCA effect under the Method 2 approach, although the effect was negative.

Baker s (1978) component of variance ratio was calculated to be 0.37 (Table 3.67). Due to the fact that the parents chosen in this diallel were selected based on their range in FHB severity they can be considered a random set of parents with regard to their DON levels. Therefore, using a random model, a narrow sense heritability estimate was estimated to be 0.28 (Table 3.67).

Diallel 2 (DON Diallel)

Greenhouse Experiment

Table 3.34 shows the parent and cross severity means of the 10 injected spikes from the greenhouse screening for diallel 2. The overall greenhouse severity mean for

diallel 2 was 18.47% which was very similar to the greenhouse severity for diallel 1 which was 18.02%. Ranking the parental genotypes in order of FHB severity yields the following: Roane < Freedom < Kaskaskia < CK 9474 < Patton < KY86C-127-3 < 25R26 < CK 9663 < KY89C-804-14-2.

Method 1 Nine Parent Analysis. The ANOVA table for a Method 1 approach shows significant genotype variation (Table 3.35). General combining ability effects and SCA effects were also significant. Reciprocal effects were not significant for this set of diallel crosses in contrast to what was found in diallel 1. The magnitude of the GCA mean square to the SCA mean square (approximately a 8:1 ratio) supports the role of additive variation.

Although this set of diallel parents is best regarded as a random set of lines with regard to the trait of severity, GCA, SCA, and reciprocal effects were calculated. General combining ability effects are given in Table 3.36. Freedom was identified as an excellent parent to use toward reducing FHB severity. Kaskaskia also had a significantly negative GCA effect. Surprisingly, Roane had a significantly positive GCA effect. Roane is considered to be tolerant to FHB (Griffey et al., 2001) and could even be labeled resistant based on the mean FHB severity of only 7.20% as observed in the greenhouse environment (Table 3.34). However, Roane s progeny were not superior.

Specific combining ability effects are given in Table 3.37 for the Method 1 nine parent analysis. Two SCA effects exceeded the estimate of the standard error, one significantly positive the other significantly negative. The cross CK 9663/Roane had a significantly positive SCA effect. The cross KY86C-804-14-2/Kaskaskia had a significantly negative SCA effect.

Variation in the reciprocal effects was not declared significant, and thus no reciprocal effects were significant for any of the crosses (Table 3.38).

Baker s (1978) component of variance ratio was calculated to be 0.36 (Table 3.67). Due to the fact that the parents chosen in this diallel were selected based on their range in DON levels they can be considered a random set of parents with regard to their FHB severities. Therefore, using a random model, a narrow sense heritability estimate was estimated to be 0.28 (Table 3.67).

Method 1 Seven Parent Analysis. Deleting the two parents that had missing data removed 25R26 and Kaskaskia from the analysis. The resulting seven parent Method 1 analysis is given in Table 3.39. Significant genotypic variation remains along with significant GCA and SCA effects. Reciprocal effects remain non-significant. The GCA mean square continued to be much larger than the SCA mean square.

Removing Kaskaskia from the data set changed the GCA effects. Freedom was still identified as a superior parent, but now along with Patton and CK 9474 (Table 3.40). Patton was also identified as a superior parent when analyzed in diallel 1 (Table 3.8).

The cross CK 9663/Roane resulted in a significantly positive SCA effect (Table 3.41). The cross CK 9474/KY89C-804-14-2 had a significantly negative SCA effect. This cross contains the genotype KY89C-804-14-2, which had a significantly positive GCA effect (Table 3.40), and CK 9474 which had a significantly negative GCA effect (Table 3.40). This same situation was also observed in diallel 1 with the cross Freedom/Clark. From this it appears that FHB resistant hybrids may be produced by crossing resistant genotypes by susceptible genotypes. Other studies have reported that resistant varieties could be produced from resistant by susceptible crosses (Snijders, 1990c, Waldron et al., 1999).

Although non significant, the reciprocal effects are presented in Table 3.42.

Baker s (1978) component of variance ratio was calculated to be 0.44 (Table 3.67). Due to the fact that the parents chosen in this diallel were selected based on their range in DON levels they can be considered a random set of parents with regard to their FHB severities. Therefore, using a random model, a narrow sense heritability estimate was estimated to be 0.37 (Table 3.67).

Method 3 Seven Parent Analysis. The genotype means used for this Method 3 seven parent analysis are presented in Table 3.43. 25R26 and Kaskaskia were removed along with all of their corresponding hybrids.

The ANOVA table for this analysis (Table 3.44) substantiates the interpretations in the earlier two analyses. Significant variation among the crosses, GCA and SCA effects was observed along with the non-significance of reciprocal effects. The GCA mean square was nearly seven times the SCA mean square. Additive genetic effects seem to predominate.

Freedom and Patton had significantly negative GCA effects (Table 3.45). CK 9663 and Roane had significantly positive GCA effects.

Table 3.46 contains the SCA effects calculated for this Method. The cross CK 9663/Roane was the only significant effect, and this effect was significantly positive.

Variation among reciprocals was not significant and thus no reciprocal effects were significant (Table 3.47).

Baker s (1978) component of variance ratio was calculated to be 0.82 (Table 3.67). Due to the fact that the parents chosen in this diallel were selected based on their range in DON levels they can be considered a random set of parents with regard to their FHB severities. Therefore, using a random model, a narrow sense heritability estimate was estimated to be 0.63 (Table 3.67).

Method 4 Analysis. The reciprocal genotype means were combined and averaged to give the genotype means reported in Table 3.48. This was done in order to have a complete set of data for the nine parents. In the situation where only the F_1 was made and no reciprocal cross was present, the original F_1 genotype mean was used.

Highly significant differences among the crosses were observed (Table 3.49). General combining ability and SCA effects were both significant. The majority of variation among the crosses was due to the GCA effects and thus most of the variation can be attributed to additive effects.

General combining ability effects were calculated from Griffing s Method 4 analysis (Griffing, 1956). Roane and CK 9663 showed positive GCA effects for severity while Freedom showed a negative GCA effect (Table 3.50).

Specific combing ability effects were calculated and are shown in Table 3.51 where the cross CK 9663/Roane was identified as having a significantly positive SCA effect.

Baker s (1978) component of variance ratio was calculated to be 0.81 (Table 3.67). Due to the fact that the parents chosen in this diallel were selected based on their range in DON levels they can be considered a random set of parents with regard to their FHB severities. Therefore, using a random model, a narrow sense heritability estimate was estimated to be 0.67 (Table 3.67).

Field Experiment — Severity

In the field, Kaskaskia and its progeny were not planted due to insufficient F_1 seed. The parent and cross severity means in the field environment are presented in Table 3.52. The overall severity mean in the field environment was much higher than the overall severity mean observed in the greenhouse (67.66% vs 18.47%, respectively). Obviously the field environment produced greater disease pressure.

Method 2 Analysis. The ANOVA table for a Method 2 analysis shows highly significant differences among crosses for the severity data (Table 3.53). The GCA effects and SCA effects were also highly significant. However a major change occurred in the amount of variation attributed to SCA effects. The mean square associated with the SCA effects increased from that shown in the greenhouse. The GCA mean square is not vastly greater than the SCA mean square. From this ANOVA table it is apparent that the GCA mean square is in the same order of magnitude as the SCA mean square. This leads to the hypothesis that along with additive effects, dominance effects may also control some of the variation expressed in the field.

GCA effects were calculated using Griffing s Method 2 (Griffing, 1956) and they are presented in Table 3.54. In the field, Roane s GCA effect for severity was negative while in the greenhouse the GCA effect was positive. As mentioned previously, Roane is cited to be tolerant to FHB (Griffey et al., 2001) and one would expect a negative GCA based on this. In the field, a significantly negative GCA was observed. Roane itself appeared tolerant as observed in the field with the second lowest mean FHB severity (Table 3.52). However, in the field experiment Roane s progeny also appeared tolerant unlike the situation observed in the greenhouse.

Specific combining abilities for the crosses are shown in Table 3.55. One cross of great interest is the cross between the two Kentucky lines KY89C-804-14-2/KY86C-127-3. This cross had a significantly negative SCA effect for FHB severity as screened in the field environment. Both parents were susceptible (Table 3.52) and had positive GCA effects (Table 3.54). The pedigrees of these two parents do not contain any Chinese scab-resistant sources or other known sources of resistance. The two lines are elite breeding material within the University of Kentucky wheat breeding program

and are well adapted to the southeastern US wheat production area. Breeding for FHB resistance in adapted material maybe possible as evidenced by this cross.

Baker s (1978) component of variance ratio was calculated to be 0.05 (Table 3.67). Due to the fact that the parents chosen in this diallel were selected based on their range in DON levels they can be considered a random set of parents with regard to their FHB severities. Therefore, using a random model, a narrow sense heritability estimate was estimated to be 0.04 (Table 3.67). This is the lowest heritability estimate observed in this study and further supports the theory that dominance may equally control the variation as noted from the GCA and SCA mean squares in the ANOVA.

Method 4 Analysis. The ANOVA table for this Method 4 analysis also shows highly significant differences among crosses for the severity data (Table 3.56). The GCA effects and SCA effects were also highly significant. The mean square associated with the SCA effects increased and is now in the same order of magnitude as the GCA mean square. This was similarly found in the Method 2 analysis.

Again GCA effects were calculated using Griffing s Method 4 (Griffing, 1956) and they are presented in Table 3.57. Roane s GCA effect for severity in the field remains significantly negative as found through the Method 2 analysis.

Specific combining abilities for the crosses are shown in Table 3.58. The Kentucky cross KY89C-804-14-2/KY86C-127-3 had a significantly negative SCA effect for FHB severity as screened in the field environment.

The Baker (1978) component of variance ratio was calculated to be 0.08 (Table 3.67). Due to the fact that the parents chosen in this diallel were selected based on their range in DON levels they can be considered a random set of parents with regard to their FHB severities. Therefore, using a random model, a narrow sense heritability estimate was estimated to be 0.08 (Table 3.67). This low heritability estimate supports the role of dominance effects.

Field Experiment — DON

The mean DON concentrations for each parent and F_1 as sampled from two field replications are presented in Table 3.59. It should be noted that the overall DON average for this diallel (7.78 ppm) was much lower than the DON average for diallel 1

(46.54 ppm). The parents chosen for diallel 2 were selected based on the DON trait. Ranking the parents in order of their DON levels gives the following: Freedom < 25R26 < Roane < Patton < CK 9474 < CK 9663 < KY86C-127-3 < KY89C-804-14-2. The DON concentrations from the injected heads are given in Appendix A. There were no significant difference among the parents or crosses for this trait as sampled from the injected spikes. The bagged heads were considered to be under an extremely high pressure disease environment which made it impossible to perceive any differences in DON levels.

Method 2 Analysis. There was no significant DON variation among the two replications tested at P=0.05 (Table 3.60). Significant DON variation among the genotypes did exist. General combining ability and SCA effects were also significant. The GCA mean square was nearly 3 times that of the SCA mean square. Additive gene effects seem to control resistance to DON.

Possible parents to concentrate on toward breeding for low DON include 25R26 and Freedom based on their negative GCA effects (Table 3.61).

Specific combining abilities were calculated and are shown in Table 3.62. Three crosses resulted in a significantly negative SCA effect. These were the crosses 25R26/CK 9663, 25R26/Roane, and KY86C-127-3/Patton. One cross that met but did not exceed the standard error estimate was KY89C-804-14-2/KY86C-127-3. This is the same cross that resulted in a highly negative SCA effect for severity in the field environment (Table 3.58). This cross would therefore contain both type II (resistance to spread within the spike) and type V (resistance to DON production) resistance mechanisms.

Baker s (1978) component of variance ratio was calculated to be 0.39 (Table 3.67). If one relaxes the traditional fixed model requirement, a narrow sense heritability estimate was estimated to be 0.28 (Table 3.67).

Method 4 Analysis. The Method 4 analysis resulted in no significant variation detected among the replications for the DON trait (Table 3.63). Significant variation among the crosses was detected with GCA and SCA effects being significant.

General combining abilities were calculated and are shown in Table 3.64. 25R26 was identified as a superior parent for reducing DON levels. 25R26 may therefore

contain some type V resistance. The Kentucky line KY86C-127-3 was also identified as a superior parent for type V resistance. In this Method 4 analysis Freedom s GCA effect did not exceed the standard error estimate, as it did using the Method 2 approach (Table 3.61), although it was negative.

Specific combining abilities were calculated and are shown in Table 3.65. Along with the three crosses identified through the Method 2 approach two other crosses look promising based on their significantly negative SCA effects as shown in Table 3.65. These two crosses are KY86C-127-3/Freedom and Freedom/Patton.

The Baker (1978) component of variance ratio was calculated to be 0.48 (Table 3.67). If one relaxes the traditional fixed model requirement, a narrow sense heritability estimate was estimated to be 0.33 (Table 3.67).

Correlations Between FHB Severity, DON Level, and FDK

Correlation coefficients between field FHB severity, greenhouse FHB severity, DON level, and FDK for both of the diallels are given in Table 3.66. Most correlations were not significant. In diallel 1 the correlation between field severity and FDK was significant ($P<0.01$) but only moderate ($r = 0.48$). Bai et al. (2001) reported a higher correlation coefficient of 0.54 ($P<0.01$) between severity and FDK in a field screening environment. Moderate correlations between FHB related traits could be expected due to the many different types of resistance noted in wheat. Breeding a wheat cultivar that contains all seven types of resistance will therefore be difficult. In diallel 2 the correlation coefficient between DON level and field FHB severity was $r = 0.39$ ($P<0.05$). A similar correlation was found to exist between greenhouse FHB severity and DON ($r = 0.36$, $P<0.05$). These low correlations do not support the selection of FHB resistant lines based on DON data alone and do not agree with the literature. Bai et al. (2001) reported a higher correlation coefficient of 0.65 ($P<0.01$) between proportion of scabbed spikelets and DON in a greenhouse screening where only injected spikes were analyzed for DON.

Greenhouse Versus Field Screening Environment

Disease severity was highest in the field. The overall severity mean for the field experiment was very high at 67.05% compared to the greenhouse severity mean of 18.74%. The field environment was obviously more favorable for infection. The CV for the severity data was much lower in the field environment (28.07%) than that of the greenhouse (101.29%). With this drastic reduction in the CV it can be recommended that the field environment provided the better screening environment.

The correlation coefficient between the two environments for the severity data was very low at r = -0.087 and was not statistically significant. The greenhouse and field environments were not related and thus resistance that is noted in the greenhouse may not hold up in the field. This is an important point for breeders to remember when selection is practiced based on greenhouse data alone.

Summary

Breeding for resistance to FHB in soft red winter wheat is possible without the use of Chinese spring resistance sources. Possible soft red winter wheat parents to use toward breeding low FHB severity lines include Freedom, Patton, Ernie, and Roane. Possible soft red winter wheat parents to use toward breeding low DON producing lines include Patton, Clark, CK 9663, 25R26, and KY86C-127-3. Specific combinations of resistant by susceptible crosses may be more resistant than the most resistant parent as in the case of Freedom x Clark. Other combinations of resistant by resistant crosses may not be more resistant as in the case of Freedom x Patton. Significant reciprocal effects were noted in only one cross (Patterson/2555), but were much less significant than GCA effects. Reciprocal effects of FHB resistance are mostly non-existent. Resistance to FHB seems to be mostly controlled by additive gene effects with some dominance effects noted in the field environment. As observed in this study, the greenhouse and field environments were vastly different and thus resistance that is noted in the greenhouse may not hold up in the field.

Table 3.1 Traits of interest for the soft red winter wheat parents used in the diallel crossing schemes, Lexington, KY 2000.

Parent	Diallel	FHB Rating	DON Rating	Area of Adaptation	Year of Release	Height (cm)	Heading Date (Julian)	Sumai 3 alleles
Freedom	1	MR		Ohio	1991	89	129	None
Ernie	1	MR		Missouri	1994	84	125	None
Patton	1	MR		Northeastern US	1998	91	128	None
Foster	1	MS		Kentucky	1996	86	128	None
Clark	1	S		Indiana	1988	86	128	None
Patterson	1	MS		Indiana	1995	91	132	None
CK 9663	1	MS		Mid-South	1996	97	134	None
2555	1	S		SE Corn Belt	1986	86	128	None
25R18	1	R		US Corn Belt	1999	86	129	present
25R26	2		MS	US Corn Belt	1999	78	134	None
CK 9663	2		S	Mid-South	1996	97	134	None
CK 9474	2		R	SE Corn Belt	1998	89	132	None
Roane	2		MS	Virginia	1999	81	126	None
KY89C-804-14-2	2		MR	Kentucky	NR†	84	128	None
KY86C-127-3	2		MR	Kentucky	NR†	86	129	None
Freedom	2		MR	Ohio	1991	89	129	None
Kaskaskia	2		MR	Illinois	1998	94	131	None
Patton	2		MR	Northeastern US	1998	91	128	None

Data from the 1999 Kentucky Wheat Variety Trial, Lexington, KY.
† Not Released

Table 3.2: Mean Fusarium head blight severity (%) measured in the greenhouse in nine wheat genotypes , their diallel progeny, and reciprocals, Lexington, KY 2000.

	Freedom	Ernie	Patton	Foster	Clark	Patterson	CK 9663	2555	25R18
Freedom	7.97	7.01	11.30	8.38	5.70	†	21.30	13.73	†
Ernie	7.54	11.73	7.11	11.43	58.44	35.41	17.42	25.59	6.39
Patton	11.58	11.88	10.43	5.68	24.39	11.40	11.64	10.61	13.94
Foster	5.66	11.18	8.67	27.56	37.20	11.78	17.54	14.55	5.56
Clark	20.12	37.57	6.77	21.66	76.93	26.77	51.76	25.30	11.69
Patterson	8.41	14.47	7.36	23.15	41.15	12.38	21.88	9.20	11.97
CK 9663	11.19	25.57	13.35	18.72	29.35	14.04	43.61	24.92	6.33
2555	9.85	25.25	8.97	13.11	46.30	36.55	34.46	53.35	4.91
25R18	6.74	6.26	5.47	5.00	8.70	9.97	10.34	9.71	5.17

Chosen based on their range in severity
† Missing data

Table 3.3: Analysis of variance of Fusarium head blight severity measured in the greenhouse in nine wheat genotypes , their diallel progeny, and reciprocals, Lexington, KY 2000.

Source	df	SS	MS	p-value
Genotypes	80	139557.85	1744.47	0.0000
GCA	8	83169.51	10396.19	0.0000
SCA	36	38228.41	1061.90	0.0000
Recip	36	18159.93	504.44	0.0200
Error	595	191083.74	321.15	
Total	677	330641.59		

Chosen based on their range in severity

Table 3.4: General combining ability effects (g_i) of Fusarium head blight severity measured in the greenhouse in nine wheat genotypes , their diallel progeny, and reciprocals, Lexington, KY 2000.

Parent	GCA Effect
Freedom	-7.78*
Ernie	0.68
Patton	-7.15*
Foster	-2.52
Clark	15.95*
Patterson	-0.17
CK 9663	5.41*
2555	5.56*
25R18	-9.98*

SE (g_i) = 3.98

Chosen based on their range in severity

Table 3.5: Specific combining ability effects (s_{ij}) of Fusarium head blight severity measured in the greenhouse in nine wheat genotypes , their diallel progeny, and reciprocals, Lexington, KY 2000.

	Freedom	Ernie	Patton	Foster	Clark	Patterson	CK 9663	2555	25R18
Freedom		-3.39	8.61	-0.44	-13.02*	-1.40	0.86	-3.74	6.74
Ernie			-1.80	-4.62	13.62*	6.66	-2.36	1.42	-2.14
Patton				-0.92	-10.98	-1.06	-3.52	-6.38	9.07
Foster					-1.76	2.39	-2.52	-6.97	0.02
Clark						0.42	1.44	-3.46	-13.53*
Patterson							-5.04	-0.28	3.35
CK 9663								0.96	-4.85
2555									-6.03
25R18									

SE (s_{ij}) = 11.35
Chosen based on their range in severity

Table 3.6: Reciprocal effects (r_{ij}) of Fusarium head blight severity measured in the greenhouse in nine wheat genotypes , their diallel progeny, and reciprocals, Lexington, KY 2000.

	Freedom	Ernie	Patton	Foster	Clark	Patterson	CK 9663	2555	25R18
Freedom		-0.26	-0.14	1.36	-7.21	0.00	5.05	1.94	0.00
Ernie			-2.39	0.13	10.43	10.47	-4.08	0.17	0.06
Patton				-1.49	8.81	2.02	-0.85	0.82	4.23
Foster					7.77	-5.69	-0.59	0.72	0.28
Clark						-7.19	11.20	-10.50	1.49
Patterson							3.92	-13.67*	1.00
CK 9663								-4.77	-2.01
2555									-2.40
25R18									

SE (r_{ij}) = 12.67
Chosen based on their range in severity

Table 3.7: Analysis of variance of Fusarium head blight severity measured in the greenhouse in seven wheat genotypes , their diallel progeny, and reciprocals, Lexington, KY 2000.

Source	df	SS	MS	p-value
Genotypes	48	107358.73	2236.64	0.0001
GCA	6	67133.63	11188.94	0.0000
SCA	21	29235.62	1392.17	0.0000
Recip	21	10989.48	523.31	0.0670
Error	373	128690.44	345.02	
Total	421	236049.17		

Chosen based on their range in severity

Table 3.8: General combining ability effects (g_i) of Fusarium head blight severity measured in the greenhouse in seven wheat genotypes , their diallel progeny, and reciprocals, Lexington, KY 2000.

Parent	GCA Effect
Freedom	-10.18*
Ernie	-1.60
Patton	-9.93*
Foster	-4.49
Clark	16.19*
CK 9663	5.19*
2555	4.82*

SE (g_i) = 4.60

Chosen based on their range in severity

Table 3.9: Specific combining ability effects (s_{ij}) of Fusarium head blight severity measured in the greenhouse in seven wheat genotypes , their diallel progeny, and reciprocals, Lexington, KY 2000.

	Freedom	Ernie	Patton	Foster	Clark	CK 9663	2555
Freedom		-1.80	10.70	0.85	-13.94*	0.40	-3.70
Ernie			0.18	-3.45	12.57*	-2.94	1.35
Patton				0.75	-11.52*	-3.61	-5.95
Foster					-3.11	-3.40	-7.34
Clark						-1.66	-6.05
CK 9663							-1.17
2555							

SE (s_{ij}) = 11.41
Chosen based on their range in severity

Table 3.10: Reciprocal effects (r_{ij}) of Fusarium head blight severity measured in the greenhouse in seven wheat genotypes , their diallel progeny, and reciprocals, Lexington, KY 2000.

	Freedom	Ernie	Patton	Foster	Clark	CK 9663	2555
Freedom		-0.26	-0.14	1.36	-7.21	5.05	1.94
Ernie			-2.39	0.13	10.43	-4.08	0.17
Patton				-1.49	8.81	-0.85	0.82
Foster					7.77	-0.59	0.72
Clark						11.20	-10.50
CK 9663							-4.77
2555							

SE (r_{ij}) = 13.13
Chosen based on their range in severity

Table 3.11: Mean Fusarium head blight severity (%) measured in the greenhouse in the diallel progeny and reciprocals of seven wheat genotypes , Lexington, KY 2000.

	Freedom	Ernie	Patton	Foster	Clark	CK 9663	2555
Freedom		7.01	11.30	8.38	5.70	21.30	13.73
Ernie	7.54		7.11	11.43	58.44	17.42	25.59
Patton	11.58	11.88		5.68	24.39	11.64	10.61
Foster	5.66	11.18	8.67		37.20	17.54	14.55
Clark	20.12	37.57	6.77	21.66		51.76	25.30
CK 9663	11.20	25.57	13.35	18.72	29.35		24.92
2555	9.85	25.25	8.97	13.11	46.30	34.46	

Chosen based on their range in severity

Table 3.12: Analysis of variance of Fusarium head blight severity measured in the greenhouse in the diallel progeny and reciprocals of seven wheat genotypes , Lexington, KY 2000.

Source	df	SS	MS	p-value
Crosses	41	55363.23	1350.32	0.0000
GCA	6	35455.94	5909.32	0.0000
SCA	14	9777.89	698.42	0.0032
Recip	21	10129.33	482.35	0.0358
Error	316	91552.07	289.72	
Total	357			

Chosen based on their range in severity

Table 3.13: General combining ability effects (g_i) of Fusarium head blight severity measured in the greenhouse in the diallel progeny and reciprocals of seven wheat genotypes , Lexington, KY 2000.

Parent	GCA Effect
Freedom	-9.23*
Ernie	2.03
Patton	-9.37*
Foster	-5.19*
Clark	13.89*
9663	5.16*
2555	2.70

SE (g_i) = 4.98

Chosen based on their range in severity

Table 3.14: Specific combining ability effects of Fusarium (s_{ij}) head blight severity measured in the greenhouse in the diallel progeny and reciprocals of seven wheat genotypes , Lexington, KY 2000.

	Freedom	Ernie	Patton	Foster	Clark	9663	2555
Freedom		-4.34	11.23*	2.63	-10.56*	1.51	-0.48
Ernie			-1.97	-4.35	13.28*	-4.50	1.88
Patton				2.93	-7.75	-2.10	-2.34
Foster					1.92	-0.64	-2.49
Clark						2.70	0.41
9663							3.03
2555							

SE (s_{ij}) = 9.83
Chosen based on their range in severity

Table 3.15: Reciprocal effects (r_{ij}) of Fusarium head blight severity measured in the greenhouse in the diallel progeny and reciprocals of seven wheat genotypes , Lexington, KY 2000.

	Freedom	Ernie	Patton	Foster	Clark	9663	2555
Freedom		-0.26	-0.14	1.36	-7.21	5.05	1.94
Ernie			-2.39	0.13	10.43	-4.08	0.17
Patton				-1.49	8.81	-0.85	0.82
Foster					7.77	-0.59	0.72
Clark						11.20	-10.50
9663							-4.77
2555							

SE (r_{ij}) = 12.04
Chosen based on their range in severity

Table 3.16: Mean Fusarium head blight severity (%) measured in the greenhouse in the diallel progeny of nine wheat genotypes , Lexington, KY 2000.

	Freedom	Ernie	Patton	Foster	Clark	Patterson	CK 9663	2555	25R18
Freedom	°	7.15	11.44	7.02	14.71	8.41	14.08	11.79	6.74
Ernie	°	°	9.08	11.31	48.01	23.89	21.50	25.45	6.34
Patton	°	°	°	7.01	16.04	9.26	12.45	9.30	9.48
Foster	°	°	°	°	29.43	17.76	18.17	13.96	5.48
Clark	°	°	°	°	°	33.96	40.56	35.25	10.27
Patterson	°	°	°	°	°	°	17.96	23.59	11.17
CK 9663	°	°	°	°	°	°	°	31.05	8.59
2555	°	°	°	°	°	°	°	°	8.60
25R18	°	°	°	°	°	°	°	°	°

Chosen based on their range in severity

Table 3.17: Analysis of variance of Fusarium head blight severity measured in the greenhouse in the diallel progeny of nine wheat genotypes , Lexington, KY 2000.

Source		df	SS	MS	p-value
Crosses		35	62079.97	1773.71**	0.0000
	GCA	8	47353.12	5919.14**	0.0000
	SCA	27	14726.85	545.44**	0.0120
Error		550	171318.76	311.49	
Total		585	233398.73		

Chosen based on their range in severity

Table 3.18: General combining ability effects (g_i) of Fusarium head blight severity measured in the greenhouse in the diallel progeny of nine wheat genotypes , Lexington, KY 2000.

Parent	GCA Effect
Freedom	-7.31*
Ernie	2.89
Patton	-6.92*
Foster	-3.19
Clark	13.68*
Patterson	1.93
CK 9663	4.55
2555	3.78
25R18	-9.41*

SE (g_i) = 6.29
Chosen based on their range in severity

Table 3.19: Specific combining ability effects (s_{ij}) of Fusarium head blight severity measured in the greenhouse in the diallel progeny of nine wheat genotypes , Lexington, KY 2000.

	Freedom	Ernie	Patton	Foster	Clark	Patterson	CK 9663	2555	25R18
Freedom		-4.99	9.11	0.96	-8.22	-2.77	0.28	-1.25	6.89
Ernie			-3.46	-4.95	14.88	2.51	-2.50	2.21	-3.70
Patton				0.56	-7.27	-2.31	-1.74	-4.13	9.24
Foster					2.39	2.47	0.25	-3.19	1.52
Clark						1.79	5.77	1.23	-10.56
Patton							-5.08	1.32	2.08
CK 9663								6.15	-3.12
2555									-2.34
25R18									

SE (s_{ij}) = 15.28
Chosen based on their range in severity

Table 3.20: Mean Fusarium head blight severity (%) measured in the field in seven wheat genotypes and their diallel progeny, Lexington, KY 2001.

	Patton	Ernie	2555	Clark	Patterson	Foster	CK 9663
Patton	34.08	17.99	46.79	42.24	36.74	16.36	29.13
Ernie		27.99	31.73	24.77	35.33	37.27	35.52
2555			53.48	44.35	47.98	37.80	60.63
Clark				43.04	60.47	28.52	43.57
Patterson					60.43	46.62	54.00
Foster						33.08	41.98
CK 9663							57.46

Chosen based on their range in severity

Table 3.21: Analysis of variance of Fusarium head blight severity measured in the field in seven wheat genotypes and their diallel progeny, Lexington, KY 2001.

Source	df	SS	MS	p value
Rep	2	14954.65	7477.33	0.0001
Genotypes	27	114103.21	4226.05	0.0001
GCA	6	81282.27	13547.04	0.0000
SCA	21	32820.94	1562.90	0.0000
Error	780	298483.07	382.67	
Total	809	427540.94		

Chosen based on their range in severity

Table 3.22: General combining ability effects (g_i) of Fusarium head blight severity measured in the field in seven wheat genotypes and their diallel progeny, Lexington, KY 2001.

Parent	GCA Effect
Patton	-6.96*
Ernie	-9.37*
2555	4.53
Clark	1.10
Patterson	9.10*
Foster	-5.04
CK 9663	6.63*

SE (g_i) = 6.04
Chosen based on their range in severity

Table 3.23: Specific combining ability effects (s_{ij}) of Fusarium head blight severity measured in the field in seven wheat genotypes and their diallel progeny, Lexington, KY 2001.

	Patton	Ernie	2555	Clark	Patterson	Foster	CK 9663
Patton		-5.68	9.21	8.09	-5.41	-11.65	-10.55
Ernie			-3.44	-6.97	-4.41	11.67	-1.75
2555				-1.29	-5.66	-1.69	9.46
Clark					10.25	-7.55	-4.18
Patterson						2.55	-1.74
Foster							0.38
CK 9663							

SE (s_{ij}) = 17.56
Chosen based on their range in severity

Table 3.24: Analysis of variance of Fusarium head blight severity measured in the field in the diallel progeny of seven wheat genotypes , Lexington, KY 2001.

Source°	df	SS	MS	p value
Rep	2	9694.15	4847.08	0.0001
Crosses	20	78037.48	3901.87	0.0001
GCA	6	53262.70	8877.12	0.0000
SCA	14	24774.78	1769.63	0.0000
Error	580	216127.00	372.63	
Total	602	303859.01		

Chosen based on their range in severity

Table 3.25: General combining ability effects (g_i) of Fusarium head blight severity measured in the field in the diallel progeny of seven wheat genotypes , Lexington, KY 2001.

Parent	GCA Effect
Patton	-8.99*
Ernie	-10.32*
2555	7.01
Clark	1.94
Patterson	9.38*
Foster	-5.14
CK 9663	6.12

SE (g_i) = 7.99

Chosen based on their range in severity

Table 3.26: Specific combining ability effects (s_{ij}) of Fusarium head blight severity measured in the field in the diallel progeny of seven wheat genotypes , Lexington, KY 2001.

	Patton	Ernie	2555	Clark	Patterson	Foster	CK 9663
Patton		-1.72	9.73	10.26	-2.68	-8.55	-7.03
Ernie			-3.99	-5.88	-2.77	13.69	0.68
2555				-3.64	-7.45	-3.11	8.46
Clark					10.11	-7.32	-3.53
Patterson						3.33	-0.54
Foster							1.95
CK 9663							

SE (s_{ij}) = 15.76
Chosen based on their range in severity

Table 3.27: Mean deoxynivalenol levels (ppm) measured in the field in seven wheat genotypes and their diallel progeny, Lexington, KY 2001.

	Patton	Ernie	2555	Clark	Patterson	Foster	CK 9663
Patton	45.82	57.36	60.43	37.06	25.85	31.65	40.01
Ernie		45.46	64.50	50.65	42.58	56.67	49.04
2555			65.72	48.06	52.40	51.35	59.86
Clark				29.09	61.02	35.63	28.00
Patterson					26.02	65.73	32.89
Foster						56.66	35.66
CK 9663	°	°	°	°	°	°	48.02

Chosen based on their range in severity

Table 3.28: Analysis of variance of deoxynivalenol levels measured in the field in seven wheat genotypes and their diallel progeny, Lexington, KY 2001.

Source	df	SS	MS	p value
Rep	1	1420.37	1420.37	0.0001
Genotypes	27	8677.47	321.39	0.0001
GCA	6	3836.53	639.42	0.0000
SCA	21	4840.94	230.52	0.0020
Error	27	1924.53	71.28	
Total	55	12022.38		

Chosen based on their range in severity

Table 3.29: General combining ability effects (g_i) of deoxynivalenol levels measured in the field in seven wheat genotypes and their diallel progeny, Lexington, KY 2001.

Parent	GCA Effect
Patton	-3.15*
Ernie	4.37*
2555	10.63*
Clark	-5.97*
Patterson	-4.43*
Foster	1.96
CK 9663	-3.42*

SE (g_i) = 2.61

Chosen based on their range in severity

Table 3.30: Specific combining ability effects (s_{ij}) of deoxynivalenol levels measured in the field in seven wheat genotypes and their diallel progeny, Lexington, KY 2001.

	Patton	Ernie	2555	Clark	Patterson	Foster	CK 9663
Patton		9.59*	6.40	-0.36	-13.12*	-13.70*	0.04
Ernie			2.94	5.70	-3.91	3.79	1.55
2555				-3.14	-0.35	-7.79*	6.11
Clark					24.88*	-6.91	-9.14*
Patterson						21.65*	-5.80
Foster							-9.42*
CK 9663							

SE (s_{ij}) = 7.57
Chosen based on their range in severity

Table 3.31: Analysis of variance of deoxynivalenol levels measured in the field in the diallel progeny of seven wheat genotypes , Lexington, KY 2001.

Source°	df	SS	MS	p value
Rep	1	1457.66	4847.08	0.0004
Crosses	20	6269.68	313.48	0.0018
GCA	6	2878.12	479.69	0.0010
SCA	14	3391.56	242.25	0.0120
Error	20	1594.99	79.75	
Total	41	9322.32		

Chosen based on their range in severity

Table 3.32: General combining ability effects (g_i) of deoxynivalenol levels measured in the field in the diallel progeny of seven wheat genotypes , Lexington, KY 2001.

Parent	GCA Effect
Patton	-5.89*
Ernie	7.79*
2555	10.95*
Clark	-4.28*
Patterson	-0.27
Foster	-1.03
CK 9663	-7.27*

SE (g_i) = 3.70
Chosen based on their range in severity

Table 3.33: Specific combining ability effects (s_{ij}) of deoxynivalenol levels measured in the field in the diallel progeny of seven wheat genotypes , Lexington, KY 2001.

	Patton	Ernie	2555	Clark	Patterson	Foster	CK 9663
Patton		8.49*	8.39*	0.26	-14.96*	-8.40*	6.21
Ernie			-1.22	0.16	-11.92*	2.93	1.55
2555				-5.58	-5.25	-5.54	9.21*
Clark					18.60*	-6.03	-7.42*
Patterson						20.06*	-6.54
Foster							-3.01
CK 9663							

SE (s_{ij}) = 7.29
Chosen based on their range in severity

Table 3.34: Mean Fusarium head blight severity (%) measured in the greenhouse in nine wheat genotypes , their diallel progeny, and reciprocals, Lexington, KY 2000.

	25R26	CK 9663	CK 9474	Roane	KY89C-804-14-2	KY86C-127-3	Freedom	Kaskaskia	Patton
25R26	45.15	17.81	15.72	20.97	5.42	9.86	5.34	5.98	10.54
CK 9663	†	69.53	16.59	58.03	28.97	21.00	9.62	9.70	26.45
CK 9474	†	28.20	20.40	22.61	6.78	29.54	14.16	14.35	9.52
Roane	24.63	49.36	29.81	7.20	32.21	40.24	19.90	†	12.78
KY89C-804-14-2	18.93	31.70	18.65	42.79	84.26	25.45	17.63	†	14.47
KY86C-127-3	18.00	23.70	15.67	25.80	18.55	33.47	12.90	†	14.13
Freedom	9.69	17.42	6.23	5.26	19.51	11.86	7.40	12.63	5.76
Kaskaskia	16.09	10.74	5.21	22.12	5.03	13.08	4.20	19.99	13.26
Patton	7.95	19.31	8.03	18.67	28.83	16.47	5.53	16.25	22.48

Chosen based on their range in deoxynivalenol levels.
† Missing data

Table 3.35: Analysis of variance of Fusarium head blight severity measured in the greenhouse in nine wheat genotypes , their diallel progeny, and reciprocals, Lexington, KY 2000.

Source	df	SS	MS	p-value
Genotypes	80	146256.44	1828.21	0.0000
GCA	8	57434.41	7179.30	0.0000
SCA	36	78858.76	2190.52	0.0000
Recip	36	9963.26	276.76	0.9380
Error	640	269784.15	421.54	
Total	725	416040.59		

Chosen based on their range in deoxynivalenol levels.

Table 3.36: General combining ability effects (g_i) of Fusarium head blight severity measured in the greenhouse in nine wheat genotypes , their diallel progeny, and reciprocals, Lexington, KY 2000.

Parent	GCA Effect
25R26	-2.21
CK 9663	9.72*
CK 9474	-2.94
Roane	6.18*
KY89C-804-14-2	7.66*
KY86C-127-3	1.43
Freedom	-8.78*
Kaskaskia	-6.76*
Patton	-4.31

SE (g_i) = 4.56

Chosen based on their range in deoxynivalenol levels.

Table 3.37: Specific combining ability effects (s_{ij}) of Fusarium head blight severity measured in the greenhouse in nine wheat genotypes , their diallel progeny, and reciprocals, Lexington, KY 2000.

	25R26	CK 9663	CK 9474	Roane	KY89C-804-14-2	KY86C-127-3	Freedom	Kaskaskia	Patton
25R26		-9.18	1.40	-0.64	-12.76	-4.76	-0.97	0.53	-3.71
CK 9663			-3.86	18.33*	-6.52	-8.27	-6.89	-12.22	-2.00
CK 9474				3.50	-11.48	4.64	2.44	0.01	-3.45
Roane					4.19	5.94	-4.29	3.23	-5.61
KY89C-804-14-2						-6.57	0.21	-15.35	-1.18
KY86C-127-3							0.26	-1.06	-1.29
Freedom								4.48	-0.74
Kaskaskia									6.35
Patton									

SE (s_{ij}) = 13.01

Chosen based on their range in deoxynivalenol levels.

Table 3.38: Reciprocal effects (r_{ij}) of Fusarium head blight severity measured in the greenhouse in nine wheat genotypes , their diallel progeny, and reciprocals, Lexington, KY 2000.

	25R26	CK 9663	CK 9474	Roane	KY89C-804-14-2	KY86C-127-3	Freedom	Kaskaskia	Patton
25R26		0.00	0.00	-1.83	-6.76	-4.07	-2.18	-5.05	1.30
CK 9663			-5.80	4.34	-1.37	-1.35	-3.90	-0.52	3.57
CK 9474				-3.60	-5.94	6.93	3.96	4.57	0.75
Roane					-5.29	7.22	7.32	0.00	-2.94
KY89C-804-14-2						3.45	-0.94	0.00	-7.18
KY86C-127-3							0.52	0.00	-1.17
Freedom								4.21	0.12
Kaskaskia									-1.49
Patton									

SE (r_{ij}) = 14.52
Chosen based on their range in deoxynivalenol levels.

Table 3.39: Analysis of variance of Fusarium head blight severity measured in the greenhouse in seven wheat genotypes , their diallel progeny, and reciprocals, Lexington, KY 2000.

Source	df	SS	MS	p-value
Genotypes	48	119260.37	2484.59	0.0001
GCA	6	56843.57	9473.93	0.0000
SCA	21	54631.56	2601.50	0.0000
Recip	21	7785.24	370.73	0.6700
Error	425	185956.99	437.55	
Total	473			

Chosen based on their range in deoxynivalenol levels.

Table 3.40: General combining ability effects (g_i) of Fusarium head blight severity measured in the greenhouse in seven wheat genotypes , their diallel progeny, and reciprocals, Lexington, KY 2000.

Parent	GCA Effect
CK 9663	10.57*
CK 9474	-5.34*
Roane	3.61
KY89C-804-14-2	9.48*
KY86C-127-3	0.06
Freedom	-11.49*
Patton	-6.89*

SE (g_i) = 5.18

Chosen based on their range in deoxynivalenol levels.

Table 3.41: Specific combining ability effects (s_{ij}) of Fusarium head blight severity measured in the greenhouse in seven wheat genotypes , their diallel progeny, and reciprocals, Lexington, KY 2000.

	CK 9663	CK 9474	Roane	KY89C-804-14-2	KY86C-127-3	Freedom	Patton
CK 9663	°	-5.79	16.56*	-12.67	-11.24	-8.52	-3.76
CK 9474	°		4.99	-14.37*	4.93	4.07	-1.95
Roane	°			1.46	6.39	-2.49	-3.94
KY89C-804-14-2	°				-10.49	-2.38	-3.90
KY86C-127-3	°					0.85	-0.83
Freedom	°						1.06
Patton	°	°	°	°	°	°	°

SE (s_{ij}) = 12.85

Chosen based on their range in deoxynivalenol levels.

Table 3.42: Reciprocal effects (r_{ij}) of Fusarium head blight severity measured in the greenhouse in seven wheat genotypes , their diallel progeny, and reciprocals, Lexington, KY 2000.

	CK 9663	CK 9474	Roane	KY89C-804-14-2	KY86C-127-3	Freedom	Patton
CK 9663		-5.80	4.34	-1.37	-1.35	-3.90	3.57
CK 9474			-3.60	-5.94	6.93	3.96	0.75
Roane				-5.29	7.22	7.32	-2.94
KY89C-804-14-2					3.45	-0.94	-7.18
KY86C-127-3						0.52	-1.17
Freedom							0.12
Patton							

SE (r_{ij}) = 14.79

Chosen based on their range in deoxynivalenol levels.

Table 3.43: Mean Fusarium head blight severity (%) measured in the greenhouse in the diallel progeny and reciprocals of seven wheat genotypes , Lexington, KY 2000.

	CK 9663	CK 9474	Roane	KY89C-804-14-2	KY86C-127-3	Freedom	Patton
CK 9663		16.59	58.03	28.97	21.00	9.62	26.45
CK 9474	28.20		22.61	6.78	29.54	14.16	9.52
Roane	49.36	29.81		32.21	40.24	19.90	12.78
KY89C-804-14-2	31.70	18.65	42.79		25.45	17.63	14.47
KY86C-127-3	23.70	15.67	25.80	18.55		12.90	14.13
Freedom	17.42	6.23	5.26	19.51	11.86		5.76
Patton	19.31	8.03	18.67	28.83	16.47	5.53	

Chosen based on their range in deoxynivalenol levels.

Table 3.44: Analysis of variance of Fusarium head blight severity measured in the greenhouse in the diallel progeny and reciprocals of seven wheat genotypes , Lexington, KY 2000.

Source	df	SS	MS	p-value
Crosses	41	54537.02	1330.17	0.0000
GCA	6	35354.47	5892.41	0.0000
SCA	14	11442.58	817.33	0.0100
Recip	21	7739.96	368.57	0.5130
Error	363	139703.73	384.86	
Total	404	194240.75		

Chosen based on their range in deoxynivalenol levels.

Table 3.45: General combining ability effects (g_i) of Fusarium head blight severity measured in the greenhouse in the diallel progeny and reciprocals of seven wheat genotypes , Lexington, KY 2000.

Parent	GCA Effect
CK 9663	7.89*
CK 9474	-4.57
Roane	10.60*
KY89C-804-14-2	3.41
KY86C-127-3	0.39
Freedom	-10.57*
Patton	-7.15*

SE (g_i) = 5.74

Chosen based on their range in deoxynivalenol levels.

Table 3.46: Specific combining ability effects (s_{ij}) of Fusarium head blight severity measured in the greenhouse in the diallel progeny and reciprocals of seven wheat genotypes , Lexington, KY 2000.

	CK 9663	CK 9474	Roane	KY89C-804-14-2	KY86C-127-3	Freedom	Patton
CK 9663	°	-1.88	14.25*	-1.92	-6.88	-4.76	1.19
CK 9474	°		-0.78	-7.08	5.83	4.37	-0.46
Roane	°			2.54	1.08	-8.41	-8.68
KY89C-804-14-2	°				0.88	4.77	4.44
KY86C-127-3	°					1.61	1.11
Freedom	°						2.41
Patton	°						

SE (s_{ij}) = 11.33

Chosen based on their range in deoxynivalenol levels.

Table 3.47: Reciprocal effects (r_{ij}) of Fusarium head blight severity measured in the greenhouse in the diallel progeny and reciprocals of seven wheat genotypes , Lexington, KY 2000.

	CK 9663	CK 9474	Roane	KY89C-804-14-2	KY86C-127-3	Freedom	Patton
CK 9663		-5.80	4.34	-1.37	-1.35	-3.90	3.57
CK 9474			-3.60	-5.94	6.93	3.96	0.75
Roane				-5.29	7.22	7.32	-2.94
KY89C-804-14-2					3.45	-0.94	-7.18
KY86C-127-3						0.52	-1.17
Freedom							0.12
Patton							

SE (r_{ij}) = 13.87
Chosen based on their range in deoxynivalenol levels.

Table 3.48: Mean Fusarium head blight severity (%) measured in the greenhouse in the diallel progeny of nine wheat genotypes , Lexington, KY 2000.

	25R26	CK 9663	CK 9474	Roane	KY89C-804-14-2	KY86C-127-3	Freedom	Kaskaskia	Patton
25R26	°	17.81	15.72	22.80	13.37	13.72	7.52	12.30	9.25
CK 9663	°		22.70	53.47	30.33	22.35	13.52	10.27	22.88
CK 9474	°			26.21	13.03	22.97	9.98	7.50	8.77
Roane	°				37.23	33.02	12.58	22.12	15.88
KY89C-804-14-2	°					22.18	18.67	5.03	21.65
KY86C-127-3	°						12.38	13.08	15.09
Freedom	°							11.10	5.65
Kaskaskia	°								14.18
Patton	°								

Chosen based on their range in deoxynivalenol levels.

Table 3.49: Analysis of variance of Fusarium head blight severity measured in the greenhouse in the diallel progeny of nine wheat genotypes , Lexington, KY 2000.

Source	df	SS	MS	p-value
Crosses	35	58749.91	1678.57**	0.0000
GCA	8	41278.32	5159.79**	0.0000
SCA	27	17471.59	647.09**	0.0060
Error	553	197005.33	349.92	
Total	598	255755.25		

Chosen based on their range in deoxynivalenol levels.

Table 3.50: General combining ability effects (g_i) of Fusarium head blight severity measured in the greenhouse in the diallel progeny of nine wheat genotypes , Lexington, KY 2000.

Parent	GCA Effect
25R26	-4.13
CK 9663	7.42*
CK 9474	-2.07
Roane	11.70*
KY89C-804-14-2	2.87
KY86C-127-3	1.91
Freedom	-7.14*
Kaskaskia	-6.55
Patton	-4.01

SE (g_i) = 6.66

Chosen based on their range in deoxynivalenol levels.

Table 3.51: Specific combining ability effects (s_{ij}) of Fusarium head blight severity measured in the greenhouse in the diallel progeny of nine wheat genotypes , Lexington, KY 2000.

	25R26	CK 9663	CK 9474	Roane	KY89C-804-14-2	KY86C-127-3	Freedom	Kaskaskia	Patton
25R26		-3.15	4.25	-2.44	-3.05	-1.74	1.12	5.30	-0.29
CK 9663			-0.32	16.67*	2.37	-4.66	-4.43	-8.27	1.79
CK 9474				-1.09	-5.44	5.45	1.53	-1.56	-2.82
Roane					4.98	1.73	-9.65	-0.71	-9.49
KY89C-804-14-2						-0.27	5.27	-8.97	5.11
KY86C-127-3							-0.07	0.04	-0.49
Freedom								7.11	-0.88
Kaskaskia									7.06
Patton									

SE (s_{ij}) = 16.2
Chosen based on their range in deoxynivalenol levels.

Table 3.52: Mean Fusarium head blight severity (%) measured in the field in eight wheat genotypes and their diallel progeny, Lexington, KY 2001.

	25R26	CK 9663	CK 9474	Roane	KY89C-804-14-2	KY86C-127-3	Freedom	Patton
25R26	66.20	66.22	56.55	46.09	66.12	75.87	54.16	68.75
CK 9663		71.59	71.20	70.28	78.82	69.96	81.37	67.44
CK 9474			73.78	54.40	86.72	66.52	81.59	69.99
Roane				56.78	66.96	50.28	66.26	65.63
KY 89C 804 14-2					78.09	47.07	67.61	59.43
KY 86C 127-3						81.67	77.24	84.75
Freedom							54.62	60.15
Patton								75.64

Chosen based on their range in deoxynivalenol levels

Table 3.53: Analysis of variance of Fusarium head blight severity measured in the field in eight wheat genotypes and their diallel progeny, Lexington, KY 2001.

Source	df	SS	MS	p value
Rep	2	15544.82	7772.41	0.0001
Genotypes	35	115237.46	3292.5	0.0001
GCA	7	33598.82	4799.83	0.0000
SCA	21	81638.64	3887.55	0.0000
Error	1025	376871.2	367.68	
Total	1062			

Chosen based on their range in deoxynivalenol levels

Table 3.54: General combining ability effects (g_i) of Fusarium head blight severity measured in the field in eight wheat genotypes and their diallel progeny, Lexington, KY 2001.

Parent	GCA Effect
25R26	-4.28
CK 9663	3.95
CK 9474	2.56
Roane	-7.55*
KY89C-804-14-2	1.99
KY86C-127-3	2.61
Freedom	-1.13
Patton	1.85

SE (g_i) = 5.67

Chosen based on their range in deoxynivalenol levels

Table 3.55: Specific combining ability effects (s_{ij}) of Fusarium head blight severity measured in the field in eight wheat genotypes and their diallel progeny, Lexington, KY 2001.

	25R26	CK 9663	CK 9474	Roane	KY89C-804-14-2	KY86C-127-3	Freedom	Patton
25R26		-1.12	-9.39	-9.74	0.74	9.88	-8.08	3.52
CK 9663			-2.97	6.22	5.21	-4.26	10.89	-6.03
CK 9474				-8.27	14.50	-6.30	12.50	-2.08
Roane					4.85	-12.44	7.28	3.67
KY89C-804-14-2						-25.19*	-0.91	-12.07
KY86C-127-3							8.10	12.64
Freedom								-8.23
Patton								

SE (s_{ij}) = 17.39

Chosen based on their range in deoxynivalenol levels

Table 3.56: Analysis of variance of Fusarium head blight severity measured in the field in the diallel progeny of eight wheat genotypes , Lexington, KY 2001.

Source	df	SS	MS	p-value
Rep	2	9088.99	4544.50	0.0001
Crosses	27	93533.36	3464.20	0.0001
GCA	7	28388.29	4055.47	0.0000
SCA	20	65145.07	3257.25	0.0000
Error	791	280102.18	354.11	
Total	820			

Chosen based on their range in deoxynivalenol levels

Table 3.57: General combining ability effects (g_i) of Fusarium head blight severity measured in the field in the diallel progeny of eight wheat genotypes , Lexington, KY 2001.

Parent	GCA Effect
25R26	-5.93
CK 9663	5.99
CK 9474	2.93
Roane	-8.24*
KY89C-804-14-2	0.56
KY86C-127-3	0.39
Freedom	3.17
Patton	1.13

SE (g_i) = 7.19

Chosen based on their range in deoxynivalenol levels

Table 3.58: Specific combining ability effects (s_{ij}) of Fusarium head blight severity measured in the field in the diallel progeny of eight wheat genotypes , Lexington, KY 2001.

	25R26	CK 9663	CK 9474	Roane	KY89C-804-14-2	KY86C-127-3	Freedom	Patton
25R26		-0.88	-7.51	-6.78	4.44	14.36	-10.12	6.50
CK 9663			-4.77	5.48	5.22	-3.46	5.16	-6.73
CK 9474				-7.34	16.17*	-3.85	8.43	-1.13
Roane					7.59	-8.92	4.28	5.69
KY89C-804-14-2						-20.93*	-3.17	-9.31
KYC86-127-3							6.63	16.18*
Freedom								-11.20
Patton								

SE (s_{ij}) = 15.90
Chosen based on their range in deoxynivalenol levels

Table 3.59: Mean deoxynivalenol levels (ppm) measured in the field in eight wheat genotypes and their diallel progeny, Lexington, KY 2001.

	25R26	CK 9663	CK 9474	Roane	KY89C-804-14-2	KY86C-127-3	Freedom	Patton
25R26	5.8	4.9	5.7	3.0	6.2	7.9	7.5	6.3
CK 9663		9.5	10.9	8.9	10.6	8.6	12.0	12.2
CK 9474			7.3	7.2	10.0	8.1	6.1	8.4
Roane				6.0	9.4	8.3	9.7	10.8
KY89C-804-14-2					11.7	7.4	6.4	8.3
KY86C-127-3						11.0	3.7	5.6
Freedom							2.8	5.4
Patton								6.5

Chosen based on their range in deoxynivalenol levels

Table 3.60: Analysis of variance of deoxynivalenol levels (ppm) measured in the field in eight wheat genotypes and their diallel progeny, Lexington, KY 2001.

Source	df	SS	MS	p value
Rep	1	10.87	10.87	0.0888
Genotypes	35	422.27	12.06	0.0002
GCA	7	178.12	25.45	0.0000
SCA	28	244.15	8.72	0.0006
Error	35	124.21	3.55	
Total	71			

Chosen based on their range in deoxynivalenol levels

Table 3.61: General combining ability effects (g_i) of deoxynivalenol levels measured in the field in eight wheat genotypes and their diallel progeny, Lexington, KY 2001.

Parent	GCA Effect
25R26	-1.69*
CK 9663	1.69*
CK 9474	0.10
Roane	-0.07
KY89C-804-14-2	1.16*
KY86C-127-3	0.16
Freedom	-1.35*
Patton	0.00

SE (g_i) = 0.56

Chosen based on their range in deoxynivalenol levels

Table 3.62: Specific combining ability effects (s_{ij}) of deoxynivalenol levels measured in the field in eight wheat genotypes and their diallel progeny, Lexington, KY 2001.

	25R26	CK 9663	CK 9474	Roane	KY89C-804-14-2	KY86C-127-3	Freedom	Patton
25R26 °		-2.88*	-0.51	-2.99*	-1.04	1.68	2.79*	0.23
CK 9663 °			1.30	-0.54	-0.07	-1.05	3.86*	2.69*
CK 9474 °				-0.65	0.92	0.08	-0.39	0.56
Roane °					0.48	0.47	3.36*	3.09*
KY89C-804-14-2 °						-1.71	-1.18	-0.63
KY86C-127-3 °							-2.85*	-2.32*
Freedom °								-1.01
Patton °								

SE (s_{ij}) = 1.71

° Chosen based on their range in deoxynivalenol levels

Table 3.63: Analysis of variance of deoxynivalenol levels measured in the field in the diallel progeny of eight wheat genotypes , Lexington, KY 2001.

Source°	df	SS	MS	p-value
Rep	1	3.48	3.48	0.3532
Crosses	27	297.37	11.01	0.0044
GCA	7	137.41	19.63	0.0010
SCA	20	159.96	8.00	0.0400
Error	27	105.15	3.89	
Total	55	405.99		

Chosen based on their range in deoxynivalenol levels

Table 3.64: General combining ability effects (g_i) of deoxynivalenol levels measured in the field in the diallel progeny of eight wheat genotypes , Lexington, KY 2001.

Parent	GCA Effect
25R26	-2.21*
CK 9663	2.17*
CK 9474	0.25
Roane	0.39
KY89C-804-14-2	0.55
KY86C-127-3	-0.86*
Freedom	-0.66
Patton	0.36

SE (g_i) = 0.75
Chosen based on their range in deoxynivalenol levels

Table 3.65: Specific combining ability effects (s_{ij}) of deoxynivalenol levels measured in the field in the diallel progeny of eight wheat genotypes , Lexington, KY 2001.

	25R26	CK 9663	CK 9474	Roane	KY89C-804-14-2	KY86C-127-3	Freedom	Patton
25R26 ˚		-2.90*	-0.20	-2.99*	0.03	3.16*	2.56*	0.33
CK 9663 ˚			0.61	-1.55	0.00	-0.57	2.62*	1.79*
CK 9474 ˚				-1.32	1.32	0.89	-1.29	-0.01
Roane ˚					0.56	0.96	2.14*	2.20*
KY89C-804-14-2 ˚						-0.14	-1.33	-0.45
KY86C-127-3 ˚							-2.58*	-1.73
Freedom ˚								-2.13*
Patton ˚								

SE (s_{ij}) = 1.67

Chosen based on their range in deoxynivalenol levels

Table 3.66: Correlation coefficients between related traits in a diallel focused on Fusarium head blight severity (top number within the cell) and a diallel focused on deoxynivalenol levels (bottom number within the cell), Lexington, KY 2001.

	Greenhouse FHB Severity	DON Level	FDK
Field FHB Severity	0.19 0.10	0.11 0.39*	0.48** 0.16
Greenhouse FHB Severity		-0.32 0.36*	0.19 -0.02
DON level			0.32 0.02

**P<0.01 *P<0.05

FHB=Fusarium head blight, DON=deoxynivalenol, FDK=Fusarium damaged kernels

Table 3.67: Baker s (1978) component of variance ratios and narrow sense heritabilities of Fusarium head blight severity (%) as measured in the greenhouse and field and deoxynivalenol (DON) (ppm) as measured in the field in two diallels of soft red winter wheat, Lexington, KY 2000-2001.

Environment	Genotypes Tested	Trait	Diallel 1		Diallel 2	
			Baker Ratio	h^2	Baker Ratio	h^2
Greenhouse	9 Parents, progeny and reciprocals	Severity	0.72	0.59	0.36	0.28
	7 Parents, progeny and reciprocals	Severity	0.70	0.60	0.44	0.37
	Progeny and reciprocals of 7 parent diallel	Severity	0.84	0.68	0.82	0.63
	Progeny only of 9 parent diallel	Severity	0.87	0.74	0.81	0.67
Field	Parents and progeny	Severity	0.69	0.63	0.05	0.04
	Progeny only	Severity	0.67	0.62	0.08	0.08
	Parents and progeny	DON	0.36	0.28	0.39	0.28
	Progeny only	DON	0.37	0.28	0.48	0.33

Parents in diallel 1 were chosen based on their range in severity. Parents in diallel 2 were chosen based on their range in deoxynivalenol levels.

Figure 3.1: Example of wheat spike injected with *Fusarium graminearum* that had been covered with a glassine bag from anthesis to harvest maturity. Notice the white cloudy mycelial growth on the spikelets.

Figure 3.2: Example of control wheat spikes (at harvest maturity) that were not injected with *Fusarium graminearum* but were covered with glassine bags. No symptoms are present.

Chapter 4

Identifying Resistance and the Relationship Between Spikelet Symptoms and Kernel Infection in *Fusarium graminearum* Infected Soft Red Winter Wheat

Introduction

Fusarium head blight (FHB), also known as head scab, caused by *Fusarium spp.,* has been a historically devastating disease of wheat and barley around the world. In Kentucky, the prevalent cropping system of no till or minimal till wheat production may influence head scab levels by providing sufficient inoculum levels. Incorporating FHB resistance into soft red winter wheat is considered to be the most effective control strategy.

Mesterhazy et al. (1999) report five different modes of resistance to FHB. Type II (resistance to spread within the spike) is commonly measured in greenhouse inoculation experiments. Type IV (resistance to kernel infection) is not well understood but has been researched. Mesterhazy reported in 1997 that there are genotypes that have less kernel infection than anticipated, based on FHB values. This experiment was conducted to better understand the interaction of spikelet infection and kernel infection due to FHB and thus provide information on the most effective breeding and selection methods.

Materials and Methods

In the fall of 1999, 29 soft red winter wheat lines and 21 F_1 hybrids were evaluated in the greenhouse for Type II resistance to *Fusarium graminearum*. The fifty wheat genotypes were planted in the greenhouse on 11 October 1999 in a completely randomized design with 10 replications per genotype. The soil mixture used was two parts soil, two parts Pro-Mix and one part sand. Seedlings were grown under artificial lighting and were fertilized with a water soluble 20-20-20 fertilizer four times. During the vegetative stage of plant growth daylength was 12 hours; during flowering daylength

was 16 hours. Approximate greenhouse temperatures were 70°F during the day and 50°F during the night.

Type II Screening

Macroconidial suspensions were prepared in the lab from a mixture of eleven different *F. graminearum* isolates. At the time of anthesis a central floret of each spike was marked with a permanent non-toxic pen and inoculated by pipetting 3 μL of the spore suspension containing approximately 1,000 spores. After inoculation, plants were placed in a humidity chamber for three consecutive nights. Plants were moved out of the chamber on the fourth day and scored for disease development on the 21st day post-inoculation. The number of diseased spikelets and the total number of spikelets were recorded for each inoculated spike. The spikelet infection rate was calculated as the percentage of diseased spikelets per total spikelets.

Kernel Assessment

Each inoculated spike was harvested and the kernels from each spike were plated onto acidified potato dextrose broth agar to quantify the presence of *F. graminearum* in the kernels. Seeds from each spike were plated onto the agar according to the spatial arrangement of the spikelets from which they came. The number and position of blank spikelets containing no seed were recorded. Plates were incubated for 7 days at 20ßC. After incubation, those kernels that showed the presence of *F. graminearum* were recorded. Kernel infection rating is reported as the percentage of seed showing positive *F. graminearum* colonization.

Statistical Analysis

Data was analyzed using a completely random design with the following model:

$$Y = \mu + G_i + \mathrm{E}_{ij}$$

where Y = the observation

μ = the overall mean

G_i = the genotype

E_{ij} = the residual error.

Correlations of interest were carried out using SAS procedure CORR (SAS, 1990). A t-test was calculated backwards to find the appropriate number of replications using the following formula:

$$t = \frac{x_1 - x_2}{\sqrt{\frac{MSE}{n}}}$$

Results and Discussion

The 50 genotypes differed in their response to FHB (Table 4.1 and 4.2). There was significant variation in the severity of infection among genotypes as well as significant variation in the kernel infection among genotypes. The 50 genotype means are shown in Table 4.3. The number of replicates per genotype varied due to low numbers of F_1 seed and the loss of some seedlings due to de-vernalization.

Relationship Between Spikelet Infection and Kernel Infection

From Table 4.3 we see that some genotypes did have a lower kernel infection rate than expected from their spikelet infection rate. These genotypes would possess Type IV resistance based on Mesterhazy's explanation. For example, Glory, which had a spikelet infection rate of 45.6%, had a kernel infection rate of only 30.5% (Table 4.3). The correlation coefficient between these two variables was r = 0.52 (P<0.0001). The relationship between kernel infection and spikelet infection is moderate and agrees with other correlation coefficients found in the literature (Mesterhazy et al., 1999). Figure 4.1 graphically displays the relationship between the two traits in this study.

Effect on Selection

If we set a hypothetical selection criteria of 10% and keep only those genotypes showing less than 10% spikelet infection, 28 genotypes would have been selected. Of those 28 genotypes, 7 actually were above the 10% infection level based on kernel

infection data and 4 genotypes would not have been selected based on spikelet infection but should have been selected based on kernel infection data.

A one tailed t test was completed to compare the two overall means. The result from this test revealed that the difference between overall kernel infection mean and overall spikelet infection mean was not significant at the 5 or 10% level. Although the overall means are not different, differences in spikelet infection and kernel infection are noted on an individual genotype mean basis.

Injection Inconsistencies

Table 4.4 provides a breakdown of all observations made in this study. In the first scenario where neither plant symptoms were observed or fungus presence was recorded, the necessary conditions for FHB development did not occur. This could be attributed to nonviable spores, improper environmental conditions, or ill-timed injections. Most likely these escapes are due to injections made prior to or post-anthesis, the stage at which the plant is most susceptible to infection. The next scenario describing visual symptoms but no actual fungus present in the kernels could be accounted for by early senescence fooling the human eye. A white head symptom has also been described in the literature where the wheat spike is not actually infected with the fungus but assimilate is shut off to the spike thus causing the white head appearance (Snijders and Krechting, 1992). This scenario could also be explained by true kernel resistance. These situations where the plant looked infected yet the kernels did not contain the fungus would be the highest form of kernel resistance. The line 90C-383-18 may contain kernel resistance as spikelet symptoms without fungus presence in the kernels was observed on seven out of nine replicates. The final situation that warrants some attention is most troubling. No visual symptoms were noted in the plants but indeed they were infected and the fungus was present in the kernels. It is not uncommon to isolate *F. graminearum* from sound-looking kernels, yet not only did these kernels look sound but the spikelets looked sound as well. This scenario occurred only 6% of the time (Table 4.4) and could possibly be attributed to the improper human judgment of symptoms.

What should n be?

Based on the error mean square contained in this study, 14 replicates would reduce error variance sufficiently to detect a difference of 10% in spikelet infection. Fifty-six replicates are needed to increase the detection level to 5%. To detect a difference of 10% in kernel infection, 16 replicates are sufficient. Eighty-one replicates are sufficient at the 5% detection level. Noting that 56 and 81 replicates are economically non-feasible for most university breeding programs, a recommendation that 15 replicates be used in greenhouse experiments with similar levels of experimental variation is made. Of course the inherent variation within an experiment greatly influences the number of replicates needed. As the variation decreases the number of necessary replicates also decreases.

Spread Through the Spike

Plating the kernels in order, according to their arrangement on the spike, allows one to follow the spread of the fungus through the spike. Based on the results from the plating data one could reconstruct the presence of the fungus within each spike. From this enormous amount of data it appears that the fungus spirals both up and down the spike infecting florets.

Summary

Type IV resistance to FHB does exist in soft red winter wheat as observed in this study. Genotypes possessing Type IV resistance have fewer Fusarium-infected kernels than one would predict based on spikelet ratings alone. Genotypes possessing good Type I resistance may not contain any Type IV resistance. This is an important point for breeders practicing selection based on Type I data alone as the selections made from such an effort may have poor kernel quality.

Table 4.1: Analysis of variance for kernel infection by *F. graminearum* in 50 soft red winter wheat genotypes, Lexington, KY 1999.

Source	df	SS	MS	F value
Genotype	49	52034.47	1061.93	2.11***
Error	312	156813.99	502.61	
Total	361	208848.47		

***P<0.0001

Table 4.2: Analysis of variance for spikelet infection by *F. graminearum* in 50 soft red winter wheat genotypes, Lexington, KY 1999.

Source	df	SS	MS	F value
Genotype	49	4.98	0.10	2.17***
Error	291	13.61	0.05	
Total	340	18.59		

*** P<0.0001

Table 4.3: Comparison of kernel and spikelet infection (%) by *Fusarium graminearum* in several soft red winter wheat genotypes and F_1's.

Pedigree	Spikelet Infection	n	Kernel Infection	n
CK 9663	15.33	6	20.32	6
Ernie	8.25	4	1.42	10
Patterson	6.17	6	2.50	5
Clark	42.43	7	30.02	10
CK 9474	8.63	8	8.01	10
Patton	6.63	8	4.31	10
Foster	13.67	6	3.72	7
FFR 566	7.00	9	1.95	8
Glory	45.57	7	30.47	7
91C-092-1	8.89	9	25.18	10
90C-383-18	6.56	9	1.85	9
90C-042-37	16.44	9	3.42	9
91C-22-1-15	14.22	9	16.89	10
92C-486-100	16.67	3	8.89	5
92C-45-50	7.75	4	3.40	7
91C-060-37	6.50	8	8.35	9
91C-22-19	7.00	2	0.00	3
92C-433-64	8.00	10	19.33	10
92C-433-17	25.71	7	30.25	9
91C-215-7	24.90	10	31.43	6
91C-260-6	51.00	6	34.44	6
91C-261-28	6.50	4	8.54	6
91C-221-21	8.38	8	23.80	10
91C-088-11	8.70	10	7.41	9
89C-804-14	35.20	5	29.19	8
91C-117-27	31.63	8	41.35	9
90C-049-31	12.00	9	18.73	10
90C-054-6	57.25	4	23.29	5
91C-117-32	13.25	8	22.51	8
Foster/91C-117-32	5.50	6	2.21	4

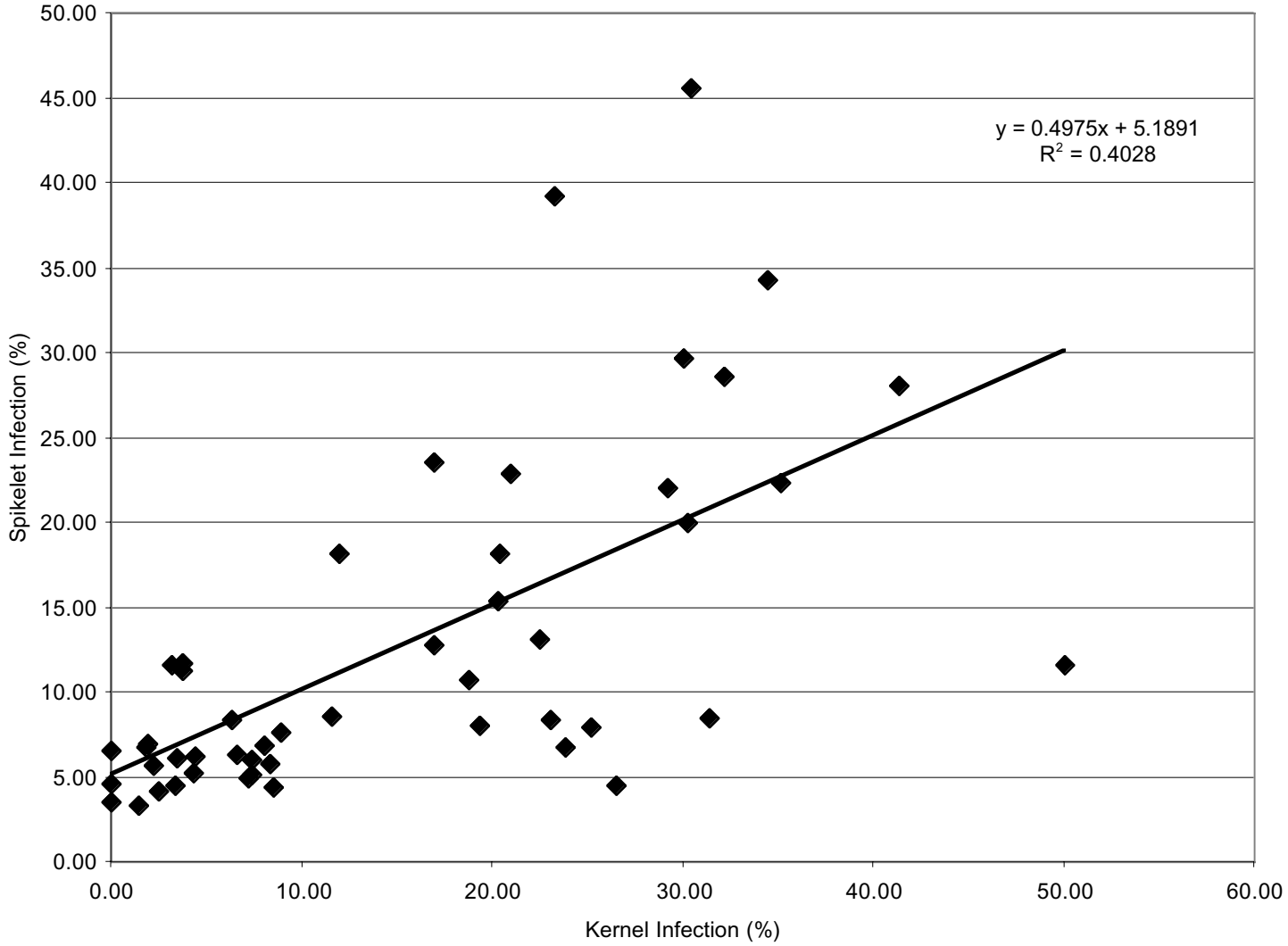
Table 4.3 (cont): Comparison of kernel and spikelet infection (%) by *Fusarium graminearum* in several soft red winter wheat genotypes and F_1's.

Pedigree	Spikelet Infection	n	Kernel Infection	n
Foster/Patton	9.57	7	50.00	3
Coker 9663/Foster	21.00	7	16.92	6
Coker 9663/91C-117-27	16.75	4	23.02	8
Coker 9663/Patton	14.83	6	3.76	7
KY 89C-804-14-1/Patton	32.13	8	32.11	9
KY 89C-804-14-1/91C-117-32	8.17	6	7.36	6
KY 89C-804-14-1/Patton	7.75	8	11.56	5
Patton/ Foster	5.71	7	26.52	6
Patton/Coker 9663	18.14	7	20.38	7
Patton/ Glory	24.71	7	3.76	7
Glory/Coker 9663	34.43	7	21.00	7
Glory/91C-117-32	8.38	8	6.34	8
91C-117-32/90C-054-6	25.17	6	11.97	8
90C-054-6/91C-117-32	6.13	8	4.43	7
Patton/Foster	10.83	6	0.00	2
Patton/Glory	8.67	6	0.00	5
Coker 9663/91C-117-32	11.63	8	3.19	8
Coker 9663/Patton	29.83	6	35.08	8
Foster/Glory	26.00	4	7.14	2
KY 89C-804-14-1/Patton	8.67	6	6.59	8
Mean	16.53		15.56	
CV	130.83		144.09	

Table 4.4: Comparison of Spikelet Infection Levels to Kernel Infection Levels

	Plates showing NO fungus present	Plates showing fungus present	Total Observations
Spikelets showing NO symptoms	38 (10.5%)	22 (6.07%)	60 (16.57%)
Spikelets showing symptoms	126 (34.8%)	176 (48.62%)	302 (83.42%)
Total Observations	164 (45.30%)	198 (54.69%)	362

Figure 4.1 Relationship between Fusarium head blight spikelet infection and kernel infection in 50 soft red winter wheat genotypes, Lexington, KY 1999.



Chapter 5

Genetic Parameter Estimates of Fusarium Head Blight Resistance and Related Traits in Three $F_{2:3}$ Soft Red Winter Wheat Populations

Introduction

Fusarium head blight (FHB), caused by *Fusarium graminearum*, is an economically important disease of wheat. Nationwide, FHB has caused wheat producers losses totaling some $2.5 billion (Windels, 2000). Fusarium head blight reduces yield, reduces quality, and produces a mycotoxin named deoxynivalenol in the grain which can limit the grain s marketability. Deoxynivalenol is a trichothecene toxin that can cause depression of the immune system, nausea, and vomiting in humans (Peraica et al., 1999) as well as feed refusal and poor weight gain in animals (Meronuck and Xie, 2000).

Genetic variation in FHB resistance is present in wheat (Buerstmayr et al., 1996, Bai et al, 2001, Mesterhazy et al., 1999). The Chinese cultivar Sumai 3 is the best known and most widely researched resistant cultivar. In addition to the Chinese wheats such as Sumai 3 and its derivates, two other sources of FHB resistance are wheats from eastern Europe and Brazil (Miedaner, 1997). From these areas come such FHB resistant cultivars as Arina and Frontana . Resistance to FHB is quantitative and thought to be controlled by a few genes with major effects and other numerous genes with minor effects (Bai et al., 1999, Waldron et al., 1999, Snijders, 1990b).

Fusarium head blight, or head scab, has received a large amount of attention in the US. The US Wheat and Barley Scab Initiative was created in 1997 and millions of dollars have been appropriated toward the goal of solving FHB. Breeding FHB resistant cultivars is viewed as the best control mechanism (Mesterhazy, 1997). Many US breeding programs screen for FHB resistance and several FHB resistant spring wheat cultivars have been released including Alsen (http://www.ag.ndsu.nodak.edu/alsen.htm, verified January 25, 2002), McVey (Busch et al., 2001), and BacUp (Busch et al., 1998). The winter wheat cultivar 25R18 has resistance to FHB from Chinese spring wheat sources (D. Van Sanford, personal communication, 2001). Other winter wheats

such as Ernie (McKendry et al., 1995) Freedom (Gooding et al., 1997) and Roane (Griffey et al., 2001) are viewed as tolerant to FHB, but do not contain resistance genes from any Chinese spring wheat sources.

Breeding a soft red winter wheat cultivar that is resistant to FHB, well adapted to the southeastern US, and has other good agronomic characters such as short plant height and high yield potential is a goal of the University of Kentucky soft red winter wheat breeding program. This study was completed with this objective in mind.

Materials and Methods

Three $F_{2:3}$ soft red winter wheat populations were planted in hill plots on 16 October 2000 with 10 seeds per replication. Row spacing between the hill plots was 61 cm. Pedigrees of the three populations are given in Table 5.1. Each population contained within its pedigree a derivative of the resistant Chinese cultivar Sumai 3. Plots were hand planted in a randomized complete block design with three replications. Forty $F_{2:3}$ lines per population were evaluated. The previous crop was corn (*Zea mays L.*) and the seedbed had been chisel plowed and disked. An overhead mist irrigation system on an automatic self timer was installed to provide adequate moisture and humidity for an FHB epidemic. The irrigation schedule included two periods of water delivery: one during the hours between 6 and 8 AM and the other during the hours between 8 and 10 PM. The morning irrigation schedule delivered water every 15 minutes in 5 minute periods. The evening irrigation schedule delivered water every 20 minutes in 10 minute periods.

Macroconidial spore suspension

Twelve cultures of *Fusarium graminearum* were obtained from scabby wheat seed by surface sterilization and plating onto acidified potato dextrose agar. Ten of these isolates were obtained from different geographical regions of Kentucky; one isolate was obtained from Indiana and one from Virginia. To induce sporulation, mycelium from the cultures was plated onto carnation leaf agar. Plating a single-spore

onto acidified potato dextrose agar ensured culture purity. The cultures were then increased on potato dextrose agar.

Macroconidial suspensions were prepared by placing two mycelial plugs from a culture of *F. graminearum* in 100 mL of carboxymethylcellulose (CMC) liquid media. Flasks were placed on a shaker (115 rpm) for 2 weeks at 24°C. Spore suspensions were prepared by filtering the cultures through a 3.0 mm Millipore filter system. Macroconidia were resuspended in sterile water and streaked onto mung bean agar plates. The plates were incubated for 7 days then washed with sterile water. The washed suspension from each of the twelve isolates was then combined and calibrated with the aid of a hemocytometer.

Grain Spawn Inoculum

The field inoculation protocol was modeled after the method of Fauzi and Paulitz (1994) with some modification. Mason jars were used to contain grain spawn inoculum. Each jar contained 500 g of corn (*Zea mays L.)* and 376 mL of water, which was added to provide adequate moisture for the pathogen to grow. The corn was allowed to imbibe the water overnight and then the jars were autoclaved. Twelve *F. graminearum* isolates were used to inoculate the jars, with each jar receiving only one particular isolate. Inoculations were made through potato dextrose agar plugs. The jars were shaken daily to disperse the inoculum within the jars of corn. The jars remained on a lab bench at room temperature for three weeks. After three weeks, when the corn was adequately colonized by the fungus, the grain spawn was thoroughly mixed to incorporate the twelve isolates into one mixture. On 10 April 2001 wheat plots were inoculated prior to heading (GS 7, Feekes Scale) by spreading 3.31g/ft^2 of the inoculated corn mixture within the plots. Plots were mist irrigated three times daily for fifteen minutes to keep the inoculum moist until 30 April 2001 when the irrigation system was set to regular programmed irrigation schedule. Plots were also sprayed with a macroconidial spore suspension (110,000 sp/mL) when 50% of the plot reached the flowering stage (GS 10.5, Feekes Scale). Spraying the plots with the spore suspension ensured that all plots would receive disease pressure regardless of their flowering time.

Field Disease Evaluations

Anthesis notes were taken daily. Those plots that reached anthesis first were scored first. Plots were scored approximately 21 days post anthesis. Disease incidence was calculated as the total number of diseased spikes per plot divided by the total number of spikes per plot. Disease severity was calculated as the number of diseased spikelets divided by the total number of spikelets for thirteen infected spikes per plot. Average plant height (cm) of each hill plot was also measured.

Fusarium Damaged Kernel Evaluation

Plots were hand harvested and threshed with a stationary thresher with the fan set at the lowest speed as not to blow out severely diseased (tombstone) kernels. Fusarium damaged kernels (FDK) from each hill plot were then evaluated as follows. A 200 seed sample was randomly taken and was sorted into two classes (healthy and non-healthy) based on the visual appearance of the seed. The primary constituent of the non-healthy seed class were tombstone kernels. The number of seeds in each class was counted and the percentage of scabby (non-healthy) seed was used as the estimate of FDK.

Statistical Analysis

Data was analyzed using the following model for all five traits (height, severity, incidence, anthesis date, and FDK) :

$$Y_{ij} = \mu + \beta_i + G_j + E_{ij},$$

where Y_{ij}=the observation on the ith block and jth genotype

μ = the overall mean

β_i=the ith block

G_j=the jth genotype

E_{ij}=the residual error.

Severity data taken on the thirteen individual heads was averaged together to give a mean severity for each hill plot. Other traits were measured on a plot basis. These hill

plot means were used in all analyses. Broad sense heritabilities were calculated for the five traits using the method of Knapp et al. (1985). Correlations of interest were estimated using SAS procedure CORR (SAS, 1990).

Results and Discussion

Table 5.2 gives the mean, coefficient of variation, genetic variance, and residual error variance for each of the five traits in the three populations studied. In all three populations some lines were very late in flowering and thus were not scored for disease traits, but height notes were taken on these lines. Thus, the number of lines varies from 40 to 37 depending on the trait analyzed.

Population 1 had the lowest mean FHB severity (33.47%) of the three populations. There was no significant variation (P=0.05) among the replications of Population 1 for height, severity, or incidence (Tables 5.3, 5.4, and 5.5). There was significant variation (P<0.05) among the replications of Population 1 for date of anthesis and FDK (Tables 5.6 and 5.7). Significant variation among the $F_{2:3}$ lines existed for all five traits (Tables 5.3, 5.4, 5.5, 5.6, and 5.7) in this population. Height, severity, and date of anthesis in Population 1 were highly heritable with estimates of $h_b^2 = 0.77$, 0.83, and 0.79, respectively (Table 5.18). Heritability of incidence (0.56) and FDK (0.53) were moderate. Given these heritability estimates, the selection of resistant lines from within this population would be possible.

Population 2 was similar to Population 1 in all traits with only a slightly higher mean FHB severity of 48.17% (Table 5.2). Significant variation (P=0.05) among the replications in Population 2 was not observed in any of the five traits (Tables 5.8, 5.9, 5.10, 5.11, and 5.12). Significant variation was observed in the lines of Population 2 for each of the five traits. Date of anthesis was the most heritable trait in Population 2 with an estimate of $h_b^2 = 0.85$ (Table 5.18). Height was also a highly heritable trait in this population ($h_b^2 = 0.71$, Table 5.18). The heritabilities of severity and FDK were moderately high, and the heritability of incidence was the lowest of the traits. The confidence interval for the heritability of incidence estimate ranged from 0.17 to 0.68 (Table 5.18). The mean square error (MSE) associated with incidence in Population 2

is the largest MSE of the five traits (Tables 5.8, 5.9, 5.10, 5.11, and 5.12). All of these findings support the conclusion that incidence is not highly heritable and the environment likely plays the more important role in determining this trait. Microenvironmental variation within the field surely existed and most likely led to varying levels of disease throughout the field. Wind movement across the field may have caused the amount of disease incidence to differ. However, these environmental variations can be downplayed in this experiment because each hill plot was treated similarly at the time of anthesis with the application of a macroconidial spore suspension of known concentration and the variation among the replications for incidence was not significant. Therefore, the amount of disease inoculum can be viewed as constant, yet incidence was still controlled by other environmental conditions.

Population 3 was similar to the other two populations in height, severity, incidence, and date of anthesis. The mean FDK for Population 3 was the highest among the three populations at 60.46% (Table 5.2). Significant variation (P=0.05) among the replications was not observed for height, severity, incidence, or date of anthesis (Tables 5.13, 5.14, 5.15, and 5.16). There was significant variation among the replications in FDK (Table 5.17). Variation among lines in this population was significant for each trait (Tables 5.13, 5.14, 5.15, 5.16, and 5.17). Heritabilities as calculated from Knapp et al. (1985) are given in Table 5.18. Height was a highly heritable trait in this population ($h_b^2 = 0.72$). A moderate heritability was calculated for severity, with the confidence interval ranging from 0.45 to 0.79. Both date of anthesis and FDK were moderately heritable with estimates of $h_b^2 = 0.63$. Heritability of incidence ranged from very low (0.05) to moderate (0.60, Table 5.18). This finding reinforces the notion that environmental variation must play a critical role in controlling variation in incidence.

The high (0.83, Population 1) to moderate (0.63, Population 2) heritabilities observed for severity across all three populations are somewhat surprising but hopeful to a breeder. These estimates are surprising due to the pre-conceived notion it will be very difficult to breed for resistance to this complex disease. Nevertheless, high heritabilities of FHB severity were observed in this study. The heritabilities reported in this study do agree with other published estimates. Bai et al. (2000a) reported heritabilities ranging from 0.91 to 0.80 on F_5 and F_6 recombinant inbred lines from the

cross Ning 7840/Clark. Buerstmayr et al. (2000) reported broad sense heritabilities of FHB severity on F_4 derived lines screened with *Fusarium culumorum* macroconidial sprays in repeated field experiments of 0.80 and 0.73.

Correlations between the five traits are given in Table 5.19. The correlation between severity and incidence was significant ($P<0.0001$) although the association between the two traits was moderate ($r = 0.3439$). This correlation suggests that the two traits are not closely related, thus there may have been lines within these populations with a high level of incidence (>75%) but a low level of severity (<25%). Lines displaying this type of reaction may contain some type II resistance to FHB (resistance to spread within the spike). One such soft red winter wheat cultivar 25R18 displays this same type of reaction: every spike within a plot may have some FHB symptoms but the infection is contained to only one spikelet on every infected spike (D. Van Sanford and B. Kennedy, personal communication, 2001). This same resistance reaction was observed on two lines in Population 1, lines 3 and 35. From Population 1, line 3 had a mean severity of 23.68% and a mean incidence of 81.43%. Also in Population 1, line 35 had a mean severity of 22.09% and a mean incidence of 76.48%. Thus, these two lines as observed in this field screening environment may contain some type II resistance.

Type I resistance (resistance to initial infection) is considered to be measurable by incidence. However, type I resistance is easily confused with disease escape mechanisms. In fact, investigators at CIMMYT question the existence of type I resistance (L. Gilchrist, personal communication, 2000). Eleven lines in this study had incidence levels less than 50%. For example, an incidence of 44.76% and a severity of 34.79% was observed on line 17 from Population 2. This line was of average height (94 cm) and had a mean anthesis date of 135 days (1 day later than the population average). Thus this line probably did not escape infection due to height or maturity. Other lines with lower incidence values were observed but these lines tended to be later maturing than the population averages. There is some evidence of type I resistance in this population but it is not overwhelming.

The correlation between anthesis date and severity was significant ($P<0.001$) along with the correlation between anthesis date and incidence. These two correlations

involving anthesis date were significant but low (r = -0.2210 and r = -0.2148), respectively. Interestingly, the correlations were negative. This means that earlier maturing lines were more susceptible and later maturing lines were more resistant. This same phenomenon has been reported in the literature. Bai and Shaner (1994) state that most of the resistant cultivars in China are later maturing. However, biologically these low correlations do not hold much meaning or value to a breeder interested in breeding for FHB resistance. Moreover, the critical time of infection for this disease is anthesis; so it is not surprising that the date of anthesis has an effect on the degree of disease. Depending on the date of anthesis the line could escape FHB disease pressure all together.

Correlations between the other traits and FDK were all significant to some degree. Those correlations of most biological interest are the correlations between severity and FDK and incidence and FDK. The correlation between severity and FDK was moderate (r = 0.3461, P<0.001). The correlation between incidence and FDK was low at r = 0.1146 and was only significant at the 10% level. These correlations do not agree with Bai et al., (2001) who reported a much higher correlation between percent scabby spikelets and percent scabby seed of r = 0.54 as measured in a similarly inoculated field environment. The correlations of FDK to disease levels (as measured by severity and incidence) are of particular interest to breeders for two reasons: one economic and the other genetic. Screening genotypes for their FHB response based on severity and incidence involves a substantial effort of reading multiple individual spikes within replicated plots before, during, and after anthesis when other breeding decisions and screenings are also on the plant breeder s to do list. If a strong correlation between FDK and FHB disease levels (such as incidence and severity) existed the plant breeder would save a considerable amount of time by not having to read all of those genotypes for their FHB disease response in the field and simply screen the genotypes after harvest by taking FDK data. However, the correlation reported in this study does not support this. This low correlation between FDK and disease levels also supports the role of type IV (resistance to kernel infection) in certain wheat genotypes as noted by Mesterhazy (1995). Some genotypes may have had a low severity but a high FDK and vice versa. Resistant looking genotypes as noted by field symptoms may not

continue to be resistant when the harvested grain is evaluated. Conversely, genotypes appearing as susceptible in the field may look great when their harvested grain is evaluated. These possibilities underscore the plight of a plant breeder searching for a FHB resistant genotype.

Hilton et al. (1999) and Mesterhazy (1995) report a significant negative correlation between plant height and severity. Both researchers suggest that taller plants are more resistant and that shorter plants are more susceptible, theoretically based on the distance of the spike from the inoculum on the ground. The correlation between plant height and severity in this experiment was not significant. This non-significant correlation between height and severity was most notably due to the fact that the plants in this study were sprayed with a spore suspension at the time of anthesis therefore reducing any effect of plant height on disease severity.

Narrow sense heritability of FHB severity was estimated for each of the three populations (Table 5.20). To attain these narrow sense heritabilities a second analysis of the data was completed under the following model:

$$Y_{ij} = \mu + F_i + E_{ij}$$

where Y_{ij}= the observation

μ =the overall mean

F_i=the i^{th} family

E_{ij}=the residual error.

The one way ANOVA partitions variance among the $F_{2:3}$ families and within the $F_{2:3}$ families. The within family variation contained both the within family genetic variation and the within family environmental variation.

To proceed further, an estimate of the within family environmental variation observed in FHB severity was needed. Two soft red winter wheat cultivars 2555 and Ernie were grown adjacent to the populations and were treated with the same disease pressure. Seven replications of these two cultivars were scored for FHB severity on 13 individual spikes per replication. The individual spike to spike analysis of these cultivars yielded two estimates of the within family environmental variation (117.95 for Ernie and 835.70 for 2555). The mean of the two estimates (476.83) was used for Population 1 noting that the FHB severity mean of Population 1 (33.47%) was lower than the FHB

severity observed in 2555 which was 55.56%. These estimates of the within family environmental variation were subtracted from the within family variation resulting in an estimate of the within family genetic variation.

The among family genetic variance contains all the additive variance and 1/4 of the dominance variance. The within family genetic variance contains 1/2 the additive variance and 1/2 the dominance variance.

$$\sigma_G^2 = \sigma_A^2 + \frac{1}{4}\sigma_D^2$$

$$\sigma_{WG}^2 = \frac{1}{2}\sigma_A^2 + \frac{1}{2}\sigma_D^2$$

Solving these two equations simultaneously gives estimates of the additive genetic variance and the dominance genetic variance. These estimates are given in Table 5.20. In Population 3 the estimate for dominance variance results in a negative number. Therefore, it was assumed to be equal to zero.

Heritabilities for FHB severity based on individual selection were estimated by the following equation.

$$h_{ind}^2 = \frac{\sigma_A^2}{\sigma_A^2 + \sigma_D^2 + \sigma_{WE}^2}$$

The estimates for the three populations are given in Table 5.20 and all were very low. Individual spike to spike variation in FHB severity was observed in the field environment. Of this spike to spike variation, approximately 85% is mostly attributed to within family environmental variation and 15% to within family genetic variation. Breeding FHB resistant lines should therefore not be focused on individual spike selection.

Narrow sense heritabilities among F_3 family means for FHB severity were calculated using the following formula:

$$h^2_{(NS)\,F_3\bar{x}} = \frac{\sigma_A^2}{\sigma_A^2 + \frac{1}{4}\sigma_D^2 + \frac{\sigma_e^2}{r}}.$$

The narrow sense heritabilities among F_3 family means are higher than the narrow sense heritabilities based on individuals (Table 5.20). The heritabilities among F_3 family means support the conclusion that selection for FHB resistance should be focused on family or plot means rather than on individuals.

The genetic gain from selection based on F_3 family means was predicted using the formula:

$$\Delta G = h^2 i \sigma_p$$

where h^2 = the narrow sense heritability based on F_3 family means

i = the selection intensity

σ_p = phenotypic standard deviation

A 10% selection intensity was assumed. Population 3 resulted in the highest predicted genetic gain of 15.89% (Table 5.20). This gain from one cycle of selection would lower the mean severity of Population 3 from 44.61% to 28.72% which is a reduction of 35%. Yang et al. (2000) observed a realized gain from one cycle of selection on the number of infected spikelets of 12.5%. This realized gain of 12.5% agrees with the predicted gain of 15.89% reported in this study.

Summary

High broad sense heritabilities were observed for height in all three soft red winter wheat populations tested. Moderately high to high broad sense heritabilities were observed for date of anthesis. Moderate to high broad sense heritabilities were observed for FHB severity and FDK. Incidence of FHB had low to moderate broad sense heritabilities. Microenvironmental conditions seem to be more important at controlling the variation observed in the incidence of FHB in this study. Type II resistance to FHB was observed on two lines in Population 1. Type I resistance was observed on one line in Population 2. The correlations between FDK and severity and FDK and incidence were moderate and low, respectively, and do not support indirect selection of FHB severity or incidence based on FDK alone. The heritabilities for severity based on individual spike selection were very low. As observed in this study, the environmental spike to spike variation is important and therefore will not allow much breeding progress when selection is practiced on individual spikes. Heritabilities based on family means are higher than heritabilities based on individual selection. Selection of FHB resistant lines should therefore be focused on family or plot means. Substantial predicted genetic gains from selection based on F_3 family means were observed and are hopeful to breeders.

Table 5.1: Pedigree information of three $F_{2:3}$ soft red winter wheat populations

	Pedigree
Population 1	Ning 7840/2691//2684/3/Elkhart
Population 2	Purdue 5/Foster//Foster
Population 3	Ning 7840/2691//2684/3/25R57

Table 5.2: Mean ($\bar{x}$), Coefficient of variation (CV), Among family variance (σ_g^2), and Error variance (σ_e^2) of height, severity, incidence, anthesis date, and Fusarium damaged kernels in (FDK) three $F_{2:3}$ soft red winter wheat populations, Lexington, KY 2001.

	Population 1 Ning 7840/2691 //2684/3/Elkhart	Population 2 Purdue 5 /Foster//Foster	Population 3 Ning 7840/2691 //2684/3/25R57
		Height (in)	
$\bar{x}$	33.49	37.79	33.84
CV	9.89	5.03	7.08
σ_g^2	12.08	2.92	4.98
σ_e^2	10.98	3.62	5.74
		Severity (%)	
$\bar{x}$	33.47	48.17	44.61
CV	22.38	25.94	27.79
σ_g^2	89.21	88.93	100.47
σ_e^2	56.14	156.12	153.64
		Incidence (%)	
$\bar{x}$	65.08	69.42	64.32
CV	26.45	21.97	21.77
σ_g^2	125.02	73.83	36.22
σ_e^2	296.31	232.50	196.15
		Anthesis Date (Julian)	
$\bar{x}$	136.13	134.42	134.54
CV	1.27	1.45	1.43
σ_g^2	3.79	7.19	2.12
σ_e^2	3.03	3.82	3.71
		FDK (%)	
$\bar{x}$	40.13	44.63	60.46
CV	37.40	32.29	21.07
σ_g^2	84.69	111.97	91.69
σ_e^2	225.27	207.62	162.35

Table 5.3: Analysis of variance for height in soft red winter wheat Population 1 (Ning 7840/2691//2684/3/Elkhart), Lexington, KY, 2001.

Source	df	SS	MS	EMS
Rep	2	58.79	29.39	
Entry	39	1841.35	47.21**	$\sigma_e^2 + r\sigma_g^2$
Error	77	845.59	10.98	σ_e^2
Total	118	2745.73		

**P<0.001

Table 5.4: Analysis of variance for Fusarium head blight severity in soft red winter wheat Population 1 (Ning 7840/2691//2684/3/Elkhart), Lexington, KY, 2001.

Source	df	SS	MS	EMS
Rep	2	335.04	167.52	
Entry	36	11656.08	323.78**	$\sigma_e^2 + r\sigma_g^2$
Error	69	3873.91	56.14	σ_e^2
Total	107	15865.04		

**P<0.001

Table 5.5: Analysis of variance for Fusarium head blight incidence in soft red winter wheat Population 1 (Ning 7840/2691//2684/3/Elkhart), Lexington, KY, 2001.

Source	df	SS	MS	EMS
Rep	2	176.54	88.27	
Entry	36	24169.39	671.37**	$\sigma_e^2 + r\sigma_g^2$
Error	69	20445.17	296.31	σ_e^2
Total	107	44791.10		

**P<0.001

Table 5.6: Analysis of variance for date of anthesis in soft red winter wheat Population 1 (Ning 7840/2691//2684/3/Elkhart), Lexington, KY, 2001.

Source	df	SS	MS	EMS
Rep	2	20.48	10.24^	
Entry	36	518.52	14.40**	$\sigma_e^2 + r\sigma_g^2$
Error	69	209.18	3.03	σ_e^2
Total	107	748.18		

^P<0.05 **P<0.001

Table 5.7: Analysis of variance for Fusarium damaged kernels in soft red winter wheat Population 1 (Ning 7840/2691//2684/3/Elkhart), Lexington, KY, 2001.

Source	df	SS	MS	EMS
Rep	2	4512.64	2256.32**	
Entry	39	18693.70	479.33**	$\sigma_e^2 + r\sigma_g^2$
Error	74	16669.72	225.27	σ_e^2
Total	115	39876.06		

**P<0.001

Table 5.8: Analysis of variance for height in soft red winter wheat Population 2 (Purdue 5/Foster//Foster), Lexington, KY, 2001.

Source	df	SS	MS	EMS
Rep	2	4.36	2.16	
Entry	39	483.13	12.39**	$\sigma_e^2 + r\sigma_g^2$
Error	78	282.35	3.62	σ_e^2
Total	119	769.79		

**P<0.001

Table 5.9: Analysis of variance for Fusarium head blight severity in soft red winter wheat Population 2 (Purdue 5/Foster//Foster), Lexington, KY, 2001.

Source	df	SS	MS	EMS
Rep	2	247.81	123.91	
Entry	39	16493.89	422.92**	$\sigma_e^2 + r\sigma_g^2$
Error	77	12020.98	156.12	σ_e^2
Total	118	28762.68		

**P<0.001

Table 5.10: Analysis of variance for Fusarium head blight incidence in soft red winter wheat Population 2 (Purdue 5/Foster//Foster), Lexington, KY, 2001.

Source	df	SS	MS	EMS
Rep	2	51.66	25.83	
Entry	39	17705.07	453.98*	$\sigma_e^2 + r\sigma_g^2$
Error	77	17902.34	232.50	σ_e^2
Total	118	35659.07		

*P<0.01

Table 5.11: Analysis of variance for date of anthesis in soft red winter wheat Population 2 (Purdue 5/Foster//Foster), Lexington, KY, 2001.

Source	df	SS	MS	EMS
Rep	2	8.25	4.12	
Entry	39	990.56	25.39**	$\sigma_e^2 + r\sigma_g^2$
Error	77	294.18	3.82	σ_e^2
Total	118	1292.99		

**P<0.001

Table 5.12: Analysis of variance for Fusarium damaged kernels in soft red winter wheat Population 2 (Purdue 5/Foster//Foster), Lexington, KY, 2001.

Source	df	SS	MS	EMS
Rep	2	340.79	170.39	
Entry	39	21197.25	543.52**	$\sigma_e^2 + r\sigma_g^2$
Error	65	13495.62	207.62	σ_e^2
Total	106	35033.67		

**P<0.001

Table 5.13: Analysis of variance for height in soft red winter wheat Population 3 (Ning 7840/2691//2684/3/25R57), Lexington, KY, 2001.

Source	df	SS	MS	EMS
Rep	2	31.11	15.56	
Entry	39	806.63	20.68**	$\sigma_e^2 + r\sigma_g^2$
Error	72	413.41	5.74	σ_e^2
Total	113	1251.16		

**P<0.001

Table 5.14: Analysis of variance for Fusarium head blight severity in soft red winter wheat Population 3 (Ning 7840/2691//2684/3/25R57), Lexington, KY, 2001.

Source	df	SS	MS	EMS
Rep	2	595.37	297.68	
Entry	39	17747.12	455.05**	$\sigma_e^2 + r\sigma_g^2$
Error	71	10908.44	153.64	σ_e^2
Total	112	29250.92		

**P<0.001

Table 5.15: Analysis of variance for Fusarium head blight incidence in soft red winter wheat Population 3 (Ning 7840/2691//2684/3/25R57), Lexington, KY, 2001.

Source	df	SS	MS	EMS
Rep	2	208.22	104.11	
Entry	39	11887.05	304.80*	$\sigma_e^2 + r\sigma_g^2$
Error	71	13926.95	196.15	σ_e^2
Total	112	26022.23		

*P<0.05

Table 5.16: Analysis of variance for date of anthesis in soft red winter wheat Population 3 (Ning 7840/2691//2684/3/25R57), Lexington, KY, 2001.

Source	df	SS	MS	EMS
Rep	2	19.11	9.55	
Entry	39	392.28	10.06**	$\sigma_e^2 + r\sigma_g^2$
Error	72	266.89	3.71	σ_e^2
Total	113	678.28		

**P<0.001

Table 5.17: Analysis of variance for Fusarium damaged kernels in soft red winter wheat Population 3 (Ning 7840/2691//2684/3/25R57), Lexington, KY, 2001.

Source	df	SS	MS	EMS
Rep	2	11858.55	5929.28**	
Entry	38	16622.09	437.42*	$\sigma_e^2 + r\sigma_g^2$
Error	32	5195.12	162.35	σ_e^2
Total	72	33675.76		

*P<0.01 **P<0.001

Table 5.18: Broad sense heritability estimates and 90% confidence intervals for three $F_{2:3}$ soft red winter wheat populations, Lexington, KY 2001.

Trait	Population 1	Population 2	Population 3
Height	0.77	0.71	0.72
	$0.62<h^2>0.86$	$0.53<h^2>0.82$	$0.55<h^2>0.83$
Severity	0.83	0.63	0.66
	$0.71<h^2>0.89$	$0.40<h^2>0.77$	$0.45<h^2>0.79$
Incidence	0.56	0.49	0.36
	$0.27<h^2>0.73$	$0.17<h^2>0.68$	$0.05<h^2>0.60$
Anthesis Date	0.79	0.85	0.63
	$0.65<h^2>0.87$	$0.76<h^2>0.91$	$0.40<h^2>0.77$
Fusarium Damaged Kernels	0.53	0.62	0.63
	$0.24<h^2>0.71$	$0.38<h^2>0.77$	$0.35<h^2>0.79$

Table 5.19: Correlation coefficients for height, severity, incidence, and Fusarium damaged kernels (FDK) as sampled in three $F_{2:3}$ soft red winter wheat populations, Lexington, KY, 2001.

	Height	Severity	Incidence	Anthesis Date	FDK
Height		0.0719	0.0315	-0.1206*	-0.2229***
Severity			0.3439***	-0.2210***	0.3461***
Incidence				-0.2148***	0.1146^
Anthesis Date					-0.1742**
FDK					

^P<0.10 *P<0.05 **P<0.01 ***P<0.001

Table 5.20 Genetic parameters and heritabilities of Fusarium head blight severity (%) in three $F_{2:3}$ soft red winter wheat populations as measured in the field, Lexington, KY, 2001.

	Population 1 Ning 7840/2691 //2684/3/Elkhart	Population 2 Purdue 5/Foster //Foster	Population 3 Ning 7840/2691 //2684/3/25R57
σ_G^2	96.13	113.53	118.17
σ_W^2	558.43	965.72	887.18
σ_{WE}^2	476.83	835.70	835.70
σ_{WG}^2	81.60	130.02	51.48
σ_A^2	73.77	64.69	118.17
σ_D^2	89.43	195.35	0.00
h_{ind}^2	0.12	0.06	0.12
$h^2_{(NS)_{F_3\bar{x}}}$	0.64	0.39	0.69
ΔG	12.14	8.88	15.89
h_{BS}^2	0.83	0.63	0.66

σ_G^2= among family genetic variation
σ_W^2= within family genetic variation
σ_{WE}^2= within family environmental variation
σ_{WG}^2= within family genetic variation
σ_A^2= additive genetic variation
σ_D^2= dominance genetic variation
h_{ind}^2= narrow sense heritability based on individual selection
$h^2_{(NS)_{F_3\bar{x}}}$= narrow sense heritability based on F_3 family mean selection
ΔG= predicted genetic gain (%) from one cycle of selection based on F_3 family means
h_{BS}^2= broad sense heritability

Appendix A

Mean deoxynivalenol levels (ppm) from injected heads covered with glassine bags measured in the field in eight wheat genotypes and their diallel progeny, Lexington, KY 2001

	25R26	CK 9663	CK 9474	Roane	KY89C-804-14-2	KY86C-127-3	Freedom	Patton
25R26	31.60	38.46	30.40	22.32	20.38	44.95	30.29	31.59
CK 9663		30.71	29.89	28.37	36.55	30.42	25.58	29.06
CK 9474			32.70	24.28	38.28	28.82	34.89	36.14
Roane				21.17	42.07	22.89	30.69	31.64
KY89C-804-14-2					34.41	32.08	24.99	29.15
KY86C-127-3						22.54	31.46	38.01
Freedom							23.06	33.03
Patton								37.29

Chosen based on their range in deoxynivalenol levels

Analysis of variance of deoxynivalenol levels from injected heads covered with glassine bags measured in the field in eight wheat genotypes and their diallel progeny, Lexington, KY 2001

Source	df	SS	MS	p value
Rep	1	85.61	85.61	0.1578
Genotypes	35	2449.05	69.97	0.0601
Parents	7	518.86	74.12	0.2726
Crosses	27	1873.89	69.40	0.0729
Error	35	1438.51	41.10	
Total	71	3973.17		

Chosen based on their range in deoxynivalenol levels

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