RESISTANCE TO TAN SPOT IN A SYNTHETIC HEXAPLOID WHEAT COLLECTION MEASURED IN A SEEDLING GREENHOUSE ASSAY

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CHAPTER I

ABSTRACT

Tan spot caused by the fungal organism *Pyrenophora tritici-repentis* is an important foliar disease of wheat (*Triticum aestivum L.*) throughout the world. Tan spot becomes more noticeable in minimum tillage farming systems due to survival from one season to the next on remnant wheat stubble remaining on the surface of soil following harvest. In this study, 94 synthetic hexaploid wheat accessions developed at the International Corn and Wheat Improvement Center (CIMMYT) were evaluated for seedling resistance to tan spot in a greenhouse assay. The seedlings were inoculated with a chlorosis and necrosis producing isolate collected from Oklahoma and evaluated for percent leaf infection at the 3 to 5 leaf stage one week post-inoculation. Of the 94 synthetic hexaploid wheat accessions tested, 54% showed high resistance when compared to the resistant check, 'Red Chief'. These results show that synthetic hexaploid wheat may be a new source of resistance to tan spot. Rating of percentage leaf area infected determined visually was compared with ratings determined by using Assess (APS Press, 2002) software, which quantitatively measures percentage leaf area infected. The visual ratings were highly correlated with the digital ratings thereby validating the use of percent leaf area infection as a method for determining reaction of wheat to inoculation of tan spot.

CHAPTER II

INTRODUCTION

Pyrenophora tritici-repentis (Died.) (anamorph *Dreschlera tritici-repentis* (Died) Shoem.) is a homothallic (self-fertile) ascomycete that causes the foliar disease tan spot of wheat (*Triticum aestivum L*). In North Dakota, the disease has the potential to reduce yields by up to 50% with a mean of 12% loss per year (Hosford, 1982; Riede et al., 1996). Most often tan spot occurs within a complex of foliar spot diseases. This disease has great importance because of its effect on wheat, which is a principal food source for people worldwide.

Tan spot usually has a higher incidence in minimum tillage management systems where increased plant residue remains on the soil surface. This increase of residue leads to an increased level of inoculum, and is a growing problem in Oklahoma and the southern Great Plains due to the increased acreage of conservation tillage and limited use of rotation crops. Tan spot can be effectively controlled with fungicides, but applications may be cost-prohibitive. Therefore, using resistant cultivars is a cost-effective alternative.

Pyrenophora tritici-repentis (Ptr) survives from one season to the next as pseudothecia on wheat stubble. During the spring the pseudothecia release ascospores that are the primary inoculum source; then later in the season conidia are produced on conidiophores that serve as secondary inoculum. Conidia germinate directly on the leaf surface of both resistant and susceptible hosts and produce germ tubes from basal

and intercalary cells (Dushnicky et al., 1996). Conidia can produce up to four germ tubes that give rise to club-shaped or round appressoria (Dushnicky et al., 1996). In their experiment 35 to 40% of appressoria formed at stomata and the remaining formed above junctions of epidermal cell walls, epidermal cells, and trichomes. According to Dushnicky et al. (1998), the appressoria infect the epidermal cells and form vesicle-like intracellular structures that produce secondary hyphae that grow into intercellular spaces of the mesophyll in susceptible wheat hosts. They found that resistant hosts prevent the growth of secondary hyphae by confining the fungus with lignin or lignin-like material (Dushnicky et al. 1998).

Ptr produces a low molecular weight host-specific protein causing necrosis (Tox A), a low molecular weight protein causing chlorosis (Tox B) and a low molecular weight host-specific toxin that produces chlorosis (Tox C) (Tomás and Bockus, 1987; Lamari and Bernier, 1989b; Brown and Hunger, 1992; Ciufetti and Tuori, 1999). These toxins are secreted ahead of the secondary hyphae (Dushnicky et al., 1998). Tox A toxins cause brown flecks that grow into necrotic lesions. Tox C produces yellow halos that surround the lesions. Production of the toxin is highly correlated with virulence of the pathogen (Tomás and Bockus, 1987; Brown and Hunger, 1992; Balance and Lamari, 1998).

The most prevalent race of Ptr found in the Great Plains of the USA and Canada is race 1, which produces both Tox A and Tox C (Lamari and Bernier, 1989b). Multiple races have been identified and characterized based on the toxin(s) produced and the resulting symptoms on specific host genotypes. The most virulent races are numbered 1,

2, 3, and 5. Of these four races, race 1 is the only race that produces both toxins (Lamari and Bernier, 1989b).

A single recessive gene controls insensitivity and resistance to the necrosis toxin. In contrast, resistance to the chlorosis toxin (Tox C) is conferred by a single dominant gene (Lamari and Bernier, 1991). Singh and Hughes (2006) showed that Tox B caused necrosis in tetraploid wheat (*T. durum*), and mapped this segment to a genomic region on chromosome 3BL. Resistance to race 1 was reported to be controlled by a single recessive gene in cultivar Salamouni (Tadesse et al., 2006a). The gene was located on chromosome 3A using monosomic analysis and was designated *tsn4,* in which the *n* symbolizes necrosis (Appendix Table 1). Other resistance genes conferring resistance to race 1 have been found on the A and B genomes and are designated in the same manner (Tadesse et al., 2006b). On the other hand, sensitivity to necrosis was conditioned by a single dominant gene and located on chromosome 5BL (Lamari and Bernier, 1991; Faris et al., 1996; Anderson, 1999). This sensitivity to Tox A occurs when the toxin is internalized into the mesophyll cells of sensitive wheat cultivars through receptor mediated endocytosis (Manning and Ciuffetti, 2005).

Many studies show that tan spot reaction is qualitative, but other evidence supports quantitative differences in resistance. One such study (Faris and Friesen, 2005) showed that quantitative trait loci (QTLs) on chromosomes 1BS and 3BL were associated with resistance to the four main races of tan spot (Appendix Table 1). More recently another quantitative trait locus (QTL) on chromosome 3AS was shown to account for 23% of the phenotypic variation for disease reaction (Singh et al., 2008). Qualitative modes of inheritance have also been found in spring wheat cultivars, but some QTLs

were reported to be responsible for resistance to the chlorosis toxin produced by some isolates of Ptr. Resistance to chlorosis-inducing races 1 and 3 was controlled by a major QTL on chromosome 1AS designated *tsc1* (Faris et al., 1997; Effertz, 2001) (Appendix Table 1).

The correlation between insensitivity to Tox A and resistance to race 1 has not been demonstrated experimentally (Friesen et al., 2002). Seeds of the hard red spring wheat 'Kulm', which possesses a single dominant gene for toxin sensitivity, were soaked in the mutagen, ethyl methanesulfonate, and the M_2 plants were screened for reaction to Ptr Tox A (Friesen et al., 2002). Toxin insensitive mutant lines were susceptible to infection from inoculation of race 1 isolates. Thus Tox A may not be the only pathogenicity factor required to cause disease symptoms (Friesen et al., 2002). Riede et al. (1996) found similar results after comparing resistant bread wheat cultivars and synthetic hexaploid accessions for reaction to culture filtrate and conidial inoculation. Both studies supported the use of conidial inoculation instead of culture filtrate to identify genotypes with true resistance to the pathogen instead of insensitivity to the toxins produced by Ptr.

Although resistance genes are effective against many races of the pathogen, the number of genes available is small and few cultivars contain resistance. Therefore, the use of alternative genetic resources for incorporating resistance into breeding programs has focused on synthetic germplasm, which has already shown success for various diseases and insects (Riede et al, 1996). Synthetic hexaploid wheat is a colchicine-induced amphiploid from the hybrid between tetraploid wheat (usually *T. turgidum* or durum wheat) (BBAA) and diploid *Aegilops taushcii* (DD) (McFadden and Sears, 1944).

Synthetic wheat provides convenient access to desirable genes from *Aegilops tauschii* and *T. durum* for genetic improvement of common bread wheat. In a study by Tadesse et al, (2006b) out of 98 synthetic wheat lines that were screened for reaction to the most virulent isolate of Ptr, (race 1), 20 genotypes were found to be highly resistant. A high proportion of synthetic hexaploid wheat accessions showed resistance in an assay by Singh and Hughes (2006), pointing to the common parent *Aegilops tauschii* as a potential source of resistance (Singh and Hughes, 2006). Recently Tadesse et al. found a high level of resistance in synthetics, which was revealed in the D-genome monosomic lines of 'Chinese Spring' (Tadasse et al., 2006b). Recessive genes for resistance in synthetic wheat were located on chromosome 3D and designated *tsn3* and *tsn-syn1*; a dominant gene was designated *Tsn-syn2.* The durum parents showed differing reactions than the progeny resulting from crosses with *Aegilops taushcii,* which have high levels of resistance (Xu et al., 2004). These results indicate that resistance genes may be inherited from the *A. taushcii* parent rather than the *T. durum* parent.

The objectives of this study were to i) adopt, refine, and validate a greenhouse assay for reliable assessment of tan spot reaction, and ii) apply this assay to a stratified and diverse collection of synthetic hexaploid wheat accessions to identify new sources of resistance.

CHAPTER III

MATERIALS AND METHODS

Inoculum production

Wheat stubble exhibiting pseudothecia indicative of *Pyrenophora tritici-repentis* (Ptr) was collected from several locations in western Oklahoma in the summer of 2006. The pathogen was isolated by placing pieces of this wheat stubble onto water agar (2%) that induced the release of ascospores onto the surrounding agar. Ascospores were removed from the water agar and placed onto potato dextrose agar (PDA) made from real potatoes (rPDA) on which the cultures were maintained. The rPDA consisted of 200 g peeled potatoes in 500 ml water, 15 g agar, 20 g dextrose, and 500 ml water. This source of PDA was used instead of manufactured synthetic PDA because increased sporulation was observed on PDA made from potatoes.

To produce conidial inoculum, 5 mm plugs were transferred from the PDA onto V-8 agar (150 ml V-8 juice, 15 g agar, 3 g $CaCO_3$, and 850 ml water) (Brown and Hunger, 1987; Evans et al., 1993). The cultures were appressed with a sterile bent glass rod once growth reached 6 cm in diameter. The plates were incubated in light for 12 h at 24 C and then in dark for 12 h at 16 C to induce conidiophore and conidia production (Raymond et al., 1985).

After sporulation, plates were flooded with 10 ml distilled water, conidiophores and conidia were removed by light scraping with a rubber spatula. This mixture was

filtered through a layer of cheesecloth to remove excess agar (Lamari and Bernier, 1989a; Raymond et al., 1985). Spores were counted and adjusted to a concentration of 2,000 to 2,500 conidia mL^{-1}.

Infection and disease rating

At the 4- to 5-leaf stage, 35 ml of spore suspension was applied uniformly across all plants in the experiment to the point of incipient run-off using an atomizer (Devilbiss Co., Somerset, PA). Seedling plants were evaluated because previous reports showed that seedling ratings accurately predict yield loss (Rees et al., 1997). Seedlings were allowed to dry for 0.5 to 1.0 hour and then placed in a mist chamber maintained at 100 % relative humidity. After 48 hours, plants were moved to the greenhouse for 5 days, sub-irrigated, and rated for severity of tan spot seven days post-inoculation (Raymond et al., 1985). Experiments which had insufficient disease incidence were repeated. A solution of 20-20-20 fertilizer (20, 20, and 20 mg L^{-1} of N, P205, and K, respectively) was applied one week prior to inoculation.

Experiments

Experiment 1. A core collection of 94 out of 380 primary synthetic hexaploid wheat accessions from CIMMYT (International Corn and Wheat Improvement Center) was created by grouping the synthetics according to their common durum parentage, then selecting about one-third of the genotypes from each durum parent group (Table 1). Each synthetic hexaploid accession having a common durum parent was derived from a unique *Aegilops tauschii* accession.

Single seeds were planted into conetainers (Stuewe and Sons, Corvallis, OR) of 2.5 cm diameter by 61.5 cm in length filled with seedling soil media (Sun Gro, Bellevue,

WA). The 94 accessions were arbitrarily divided into two groups and tested in a randomized complete block design with ten runs per group and two plants per run (Appendix Figure 1). The blocking factor was represented by different runs. A maximum of only 40 seeds of each accession was available and an extra run was planted to account for insufficient germination. In addition to the accessions, three check cultivars were included in each run and provided a broad range of reaction types. The checks were 'Red Chief', a highly resistant line (Tadasse et al., 2006b); '2174', a moderately resistant line; and 'TAM 105', a universally susceptible line (W.W. Bockus, 2007, personal communication).

Experiment 2. An additional experiment was conducted to validate the visual scoring system using percentage infection data. A total of 27 lines from a collection of advanced breeding lines and advanced synthetic derivatives, from the Oklahoma State University wheat improvement program, were selected to represent the range of expected reaction to tan spot inoculation. Highly resistant lines were excluded because of insufficient symptom expression. Selected lines were planted in a completely randomized design and evaluated for percentage leaf infection according to the same procedures outlined in experiment 1. Leaves of each line were scanned using a (Epson Perfection 1650, Nagano, Japan) scanner with twain capabilities set at 300 dpi. The actual infected area of the leaf was determined using Assess (APS Press, 2002) software. The Assess software uses a Hue-Saturation-Intensity color model to allow for the separation of the leaf from the background and then the lesions from the leaf. Although the quantification of disease levels is calculated by the program, it is easily adjusted using threshold level sliders allowing for any plant disease to be measured including tan spot. For example a

study by Jackson et al. (2006) used Assess to measure the disease leaf area of oats infected with crown rust.

Statistical analysis

In experiment 1, the percentage leaf infection data was transformed using arcsin square root (Steele and Torrie, 1960). Accessions with a percentage of leaf infection within one protected least significant difference (LSD) value of the resistant check 'Red Chief' were considered resistant. Analysis of variance (ANOVA) was completed using the mixed procedure of SAS (SAS, 2002). The mixed procedure was also used to generate least squares means for both percentage leaf area and the arcsin square root data, and accession means were separated using the PDIFF option that generates an LSD. The reaction of two plants per run and accession was used to calculate the least squares mean, making each plant within a run, a subsampling unit. For experiment 2, regression analysis was conducted by correlating the visual ratings with digital ratings. The correlation procedure of SAS (SAS, 2002) was used in which the mean digital ratings of accessions served as the independent variable.

CHAPTER IV

RESULTS AND DISCUSSION

Experiment 1

Of the 94 synthetic hexaploid wheat accessions tested, 51 accessions (54%) produced a rating that was not significantly different than Red Chief, the resistant check, based on the least significant difference (LSD) value (transformed data) of 0.137 (Table 1). Crosses of *Aegilops tauschii* with common durum parents 'Altar 84', 'CETA', 'DVERD_2', 'GAN', 'GARZA/BOY', and 'SORA' had at least 60% of their synthetic progeny produce a resistant reaction (Table 1). The vast majority of accessions, with the exception of two, CIGM92.1643 and CIGM90.808, showed greater levels of resistance than the susceptible check, TAM 105. Several accessions showed resistance levels higher than that of the resistant check, and one accession, CIGM92.1643, showed greater susceptibility than the susceptible check.

Percentage leaf area affected varied from 15 to 65% before transformation and from 0.35 to 0.90 after arcsin square root transformation (Figure 1). A skewness test showed that the distribution for percentage leaf area infection was skewed toward values indicating greater resistance (Figure 1), a finding that would support further mining of resistant accessions in the larger collection of synthetics from which this core collection was sampled. The majority of accessions showed percentage leaf infection area of 35% or

less. Red Chief had a mean infection percentage of 20% and varied from 3 to 60% among runs. TAM 105 had a mean percentage leaf infection of 58% and varied from 30 to 87% among runs (Figure 1). Several reports have shown differing levels of resistance among synthetic hexaploid wheat accessions that were similar to the results reported in this study (Xu et al, 2004; Tadasse et al., 2006b; Singh et al., 2008).

The majority (90%) of synthetic accessions sampled for this study was previously not tested for reaction to tan spot. Those that were previously examined provided a unique opportunity to consider the consistency of reaction among evaluators. Of the ten accessions in common with a study by Xu et al., (2004) seven were confirmed resistant (*CIGM89.538-0Y, CIGM92.1723, CIGM89.559, CIGM88.1313a, CIGM87.2771-1B-0PR-0B,* CIGM86.950-1M-1Y-0B-0PR-0B, *CIGM86.953-1M-1Y-0B-0PR-0B*) and one was confirmed susceptible (CIGM89.561-0Y), while two of the common accessions showed conflicting results with the previous study (CIGM88.1313b and CIGM90.808) (Table 1).

The conflicting results could be related to the use of different types of evaluation procedures, virulence of isolates, differing environments, and number of replications. Evaluation procedures that score infection reaction by lesion size, percentage leaf infection area, and a combination of both have been used (Lamari and Bernier, 1989a; Riede et al., 1996; Raymond et al., 1985; respectively). Each procedure has the ability to assess response to inoculum infection as susceptible or resistant, but may not be easily compared to one another directly. A rating on the lesion type scale cannot be equated directly into a value for the percentage leaf infection. The only true comparison that can be made is whether a genotype is resistant or susceptible. The virulence of inoculum is

dependent on the type and concentration of infective propagules (conidia or conidiophores) used for inoculation. The virulence of each isolate is also dependent on the amount and type of toxins produced. In a comparison of the toxin production from different isolates, Brown and Hunger (1993), found that the amount of toxin produced varied between isolates. This toxin production has been found to be related to the virulence of those isolates (Ballance and Lamari, 1998). Evans et al. (1996) found that conidia were significantly more effective at causing infection than conidiophores. Increasing the concentration range from 2,000 to 2,500 conidia mL^{-1} in this study to 3,000 conidia mL^{-1} in the study by Xu et al. (2004) may explain the difference in lesion incidence. The amount and severity of infection also is highly dependent on the post-inoculation wet period and temperature (Hosford et al., 1986). As the temperature and duration of the post-inoculation wet period increase, the virulence of Ptr increases on wheat cultivar BH1146, until the temperature reaches 30 C. (Hosford et al., 1986). According to Xu et al. (2004) the post-inoculation wet period was 24 hours, and afterward the plants were held in a growth chamber. In this study, the post inoculation wet period was 48 hours and the plants were grown in a greenhouse. In the greenhouse, there is greater fluctuation of temperature and humidity, which may highly affect response to infection and thereby influence the overall reaction score compared with the more consistent temperatures of growth chambers.

In our assay, a mean of ratings from three leaves was taken because leaf position has a significant influence on tan spot severity (Raymond et al., 1985; Cox and Hosford, 1987). The older leaves tended to show a more susceptible reaction to infection while the youngest leaves showed more resistance. By using more than one leaf, the entire plant

reaction was better represented. Although spore concentration was consistently between 2000 to 2500 conidia mL^{-1}, the severity of infection varied naturally within runs, which could account for some of the variation across replications.

In the analysis of variance (ANOVA) the accession effect was highly significant, confirming that reaction to tan spot was dependent on the genotype of the synthetic hexaploid accession. The genetic variance component attributed to accessions was highly significant, as was the experimental error, showing that the environment had a large effect on tan spot reaction. The subsample variance component, which was attributed to variation among plants within an accession, represented the largest source of variation (Table 2). The experimental error variance component was the second largest source of variation (Table 2). Together, 70% of the total variation was attributed to the experimental and subsampling error. Part of the variation among plants could be attributed to a confounding genetic component relating to potential heterogeneity within accessions. Repeatability of the tan spot rating was estimated to be 0.81 on an accession-mean (basis) of the two subsamples and 10 runs (Campbell and Lipps, 1998) (Appendix Equation 1).

The variance component analysis implies that accuracy of the assay could be improved upon by increasing the number of plants of each accession within a replication, accounting for the variation contributed by the subsampling error. By allocating experimental resources using sufficient numbers of plants and replications, selection efficiency will greatly increase (Gauch and Zobel, 1996; Campbell and Lipps, 1998). Campbell and Lipps (1998) suggested that the greatest selection efficiency for selecting resistance to Fusarium head blight (caused by *Fusariam graminearum* Schwabe) came

from sampling at least 20 heads (subsamples) with six replications in at least three environments. The increase in selection efficiency is derived largely in part by limiting the probability of identifying an accession as more resistant than another when truly no difference exists (type I error) or by limiting the probability of identifying two accessions at the same level of resistance when they are truly different (type II error). Although optimum selection was achieved by increasing the number of sampled heads, replications, and environments, the costs were not necessarily optimized. The authors suggested eight heads (analogous to plants in this assay), and four replications as the most cost-effective sampling regime for identifying resistance to Fusarium head blight (Campbell and Lipps, 1998), given their error variance structure. Approaching the assay in such a manner could also allow for fewer replications, while accounting for more of the subsampling variation. In this study, by increasing the amount of plants or subsamples to five, the minimum number of replications needed to obtain a 20% difference in accession mean at the 0.25 probability level for type II error is seven (Table 3) (Campbell and Lipps, 1998). The replications needed only decreases to six when ten subsamples are used (Table 3) (Appendix Equation 2). As the number of subsamples increases and the chance of committing type II error increases and the number of replications decreases (Table 3). Therefore, the evaluator can limit the number of replications by increasing subsamples. If there is limited seed available the number of subsamples and replications must be balanced to facilitate optimization of resources.

The example of Fusariam head blight may have some application to our assay for tan spot, but their data were taken from the field. Using greenhouse evaluations presents one additional concern. In a study by Evans et al. (1999) the area under disease progress

curve (AUDPC) was measured for an experiment conducted in the field and compared to lesion lengths of a greenhouse trial. The authors found that the greenhouse lesion length and AUDPC values did not predict differences in grain yields of the tested wheat lines and cultivars (Evans et al., 1999). Therefore, a greenhouse assay may accurately identify a susceptible genotype, but may not necessarily identify genotypes with true resistance in a field environment. This knowledge makes eliminating both types of statistical errors essential to make a greenhouse assay beneficial. This also supports the use of field evaluation for identifying reaction to tan spot. Once the susceptible genotypes are eliminated, resources may be then focused on the enriched sample of genotypes to identify material that is most resistant (and with consistency) to tan spot.

Experiment 2

A high correlation was found between digital estimates of percentage leaf area infected and visual estimates of infected leaf area ($r^2 = 0.83$ $P < 0.01$). Those accessions that were classified as resistant or susceptible by visual ratings were separated into the two classifications similarly by the digital ratings. As the percentage of infection increased the visual estimates were lower than the digital scores, while the visual estimates were higher than the digital scores as the percentage of infection decreased (Figure 2). The scores were most closely associated around the 30 to 40% infection level (Figure 2). Analysis using Assess software validated the use of visual estimates of percentage leaf area for separating susceptible genotypes from resistant genotypes.

CHAPTER V

CONCLUSION

The evaluation method reported in this paper was proven to be a useful and efficient method of quantifying reaction to inoculation of tan spot in a greenhouse environment. This method has the capability to separate a large amount of genetic material for resistance or susceptibility to Ptr. By identifying and eliminating the susceptible genotypes the amount of resistant breeding material will be increased. This in turn leads to a greater chance of producing resistant cultivars. The assay may also be applied as a method of indirect selection for resistance in the field, allowing for the evaluation of large populations.

The synthetic hexaploid wheat accessions evaluated in this study showed great potential as a genetic resource to introduce potential novel genes for tan spot resistance. This discovery warrants further exploration of the genetic potential of synthetic hexaploid wheat. Using this material shall increase the genetic diversity of the wheat germplasm pool and help provide more avenues of resistance to tan spot and many other diseases.

Table 1. Identification of 94 synthetic hexaploid wheat accessions and their reaction to inoculation of *Pyrenophora tritici-repentis*.

		Percentage leaf infection			
		Transformed data †		Original data	
Pedigree	Selection number	Least-squares mean	SE	Least-squares mean	Lesion type rating‡
68.111/RGB-U//WARD RESEL/3/STIL/4/AE.SQ. (332)	*CIGM88.1297-0B*	0.44	0.031	18	
68.111/RGB-U//WARD RESEL/3/STIL/4/AE.SQ. (783)	*CIGM89.538-0Y*	0.52	0.036	24	1.67
68.111/RGB-U//WARD/3/AE.SQ. (454)	*CIGM92.1723*	0.50	0.032	23	1.33
68.111/RGB-U//WARD/3/FGO/4/RABI/5/AE.SQ. (629)	*CIGM90.590*	0.45	0.045	19	
68.111/RGB-U//WARD/3/FGO/4/RABI/5/AE.SQ. (809)	CIGM89.543-4B	0.70***	0.035	42	
68.111/RGB-U//WARD/3/FGO/4/RABI/5/AE.SQ. (809)	CIGM89.543-3B	0.69***	0.030	40	
68.111/RGB-U//WARD/3/FGO/4/RABI/5/AE.SQ. (809)	CIGM89.543-3B	0.65***	0.041	37	
68.111/RGB-U//WARD/3/FGO/4/RABI/5/AE.SQ. (878)	*CIGM89.559*	0.49	0.037	22	1.00
68.111/RGB-U//WARD/3/FGO/4/RABI/5/AE.SQ. (878)	CIGM89.559-4B	0.76***	0.040	48	
68.111/RGB-U//WARD/3/FGO/4/RABI/5/AE.SQ. (878)	CIGM89.559-1B	0.75***	0.041	46	
68.111/RGB-U//WARD/3/FGO/4/RABI/5/AE.SQ. (882)	CIGM89.561-0Y	0.68***	0.030	40	2.33
68.111/RGB-U//WARD/3/FGO/4/RABI/5/AE.SQ. (905)	*CIGM89.571-0Y*	0.39	0.028	15	
68112/WARD//AE.SQ. (369)	*CIGM88.1313a*	0.52	0.042	25	1.17
68112/WARD//AE.SQ. (369)	CIGM88.1313b	0.59**	0.037	31	1.67
68112/WARD//AE.SQ. (369)	CIGM88.1313-3B	0.56*	0.040	28	
ACO89/AE.SQ. (178)	*CIGM90.527*	0.55	0.036	27	
ACO89/AE.SQ. (521)	*CIGM89.473-0Y*	0.56	0.042	28	
AJAIA/AE.SQ. (330)	*CIGM92.1675*	0.50	0.034	23	
ALG86/4/FGO/PALES//MEXI_1/3/RUFF/FGO/5/ENTE/6/AE.SQ. (254)	CIGM89.393-0Y	0.57*	0.033	29	
ALG86/4/FGO/PALES//MEXI_1/3/RUFF/FGO/5/ENTE/6/AE.SQ. (518)	*CIGM90.545*	0.47	0.042	21	
ALTAR 84/AE.SQ. (211)	*CIGM87.2771-1B-0PR-0B*	0.45	0.050	19	1.00
ALTAR 84/AE.SQ. (223)	*CIGM87.2762-1B-0PR-0B*	0.49	0.029	22	
ALTAR 84/AE.SQ.(Y86-87 S401)	*CIGM87.2779-1B-0PR-0B*	0.50	0.033	23	
AOS/AE.SQ. (269)	CIGM88.1249-0B	0.57*	0.031	29	
ARLIN/AE.SQ. (295)	*CIGM92.1657*	0.50	0.030	23	
ARLIN/AE.SQ. (665)	CIGM90.888	0.57***	0.032	29	
CETA/AE.SQ. (174)	*CIGM93.183*	0.45	0.028	19	
CETA/AE.SQ. (540)	*CIGM93.399*	0.42	0.025	17	
CETA/AE.SQ. (895)	*CIGM89.567-1B*	0.49	0.043	22	
CETA/AE.SQ. (895)	CIGM89.567-3B	0.57*	0.037	29	
CETA/AE.SQ. (895)	*CIGM89.567*	0.53	0.031	26	
CHEN_7/AE.SQ. (429)	CIGM89.438-0Y	0.56*	0.042	28	
CPI/GEDIZ/3/GOO//JO/CRA/4/AE.SQ. (358)	CIGM90.817	0.69***	0.043	40	
CPI/GEDIZ/3/GOO//JO69/CRA/4/AE.SQ. (205)	CIGM88.1192-0B	0.62**	0.042	33	
CPI/GEDIZ/3/GOO//JO69/CRA/4/AE.SQ. (629)	CIGM90.534	0.59*	0.044	31	
CPI/GEDIZ/3/GOO//JO69/CRA/4/AE.SQ. (633)	CIGM89.501	0.57*	0.036	30	
CPI/GEDIZ/3/GOO//JO69/CRA/4/AE.SQ. (633)	CIGM89.501	0.69***	0.033	41	
CROC_1/AE.SQ. (168)	CIGM87.2755-1B-0PR-0B	0.75***	0.039	47	
CROC_1/AE.SQ. (177)	CIGM93.185	0.57*	0.038	29	
CROC_1/AE.SQ. (224)	CIGM86.950-1M-1Y-0B-0PR-0B	0.45	0.039	19	1.00
CROC_1/AE.SQ. (275)	CIGM93.218	0.64***	0.039	35	
CROC_1/AE.SQ. (444)	*CIGM93.244*	0.54	0.025	27	
CROC_1/AE.SQ. (662)	CIGM89.510-0Y	0.62**	0.270	34	
CROC_1/AE.SQ. (826)	*CIGM89.546-0Y*	0.52	0.039	25	
D67.2/P66.270//AE.SQ. (257)	CIGM90.808	0.76***	0.042	48	1.50
DOY1/AE.SQ. (1024)	CIGM93.298	0.68***	0.041	39	
DOY1/AE.SQ. (177)	CIGM93.187	0.60**	0.037	32	
DOY1/AE.SQ. (188)	*CIGM88.1175-0B*	0.51	0.040	23	
DOY1/AE.SQ. (264)	*CIGM93.211*	0.56	0.038	28	
DOY1/AE.SQ. (318)	CIGM93.223	0.61**	0.038	33	
DOY1/AE.SQ. (372)	*CIGM93.229*	0.46	0.032	20	
DOY1/AE.SQ. (415)	*CIGM92.1708*	0.52	0.033	25	
DOY1/AE.SQ. (418)	*CIGM93.239*	0.51	0.033	23	
DOY1/AE.SQ. (428)	CIGM92.1713	0.58*	0.021	30	
DOY1/AE.SQ. (446)	CIGM88.1343-0B	0.57*	0.033	30	
DOY1/AE.SQ. (446)	*CIGM88.1343-0B*	0.53	0.033	25	
DOY1/AE.SQ. (488)	CIGM88.1353-0B	0.63***	0.046	35	
DOY1/AE.SQ. (511)	CIGM88.1363-0B	0.65***	0.040	37	
DOY1/AE.SQ. (517)	*CIGM93.267*	0.53	0.032	26	
DOY1/AE.SQ. (532)	CIGM92.1754	0.60**	0.029	32	
DVERD_2/AE.SQ. (221)	*CIGM86.953-1M-1Y-0B-0PR-0B*	0.52	0.042	25	1.00
DVERD_2/AE.SQ. (247)	*CIGM88.1237-0B*	0.54	0.032	26	
DVERD_2/AE.SQ. (247)	*CIGM88.1237-0B*	0.52	0.028	25	

Table 1. cont'd

Pedigree	Selection number	Percentage leaf infection: Transformed data † Least-squares mean	SE	Original data Least-squares mean	Lesion type rating‡
FALCIN/AE.SQ. (389)	CIGM92.1702	0.62**	0.037	33	
GAN/AE.SQ. (163)	*CIGM93.177*	0.53	0.036	26	
GAN/AE.SQ. (257)	*CIGM90.807*	0.50	0.025	23	
GAN/AE.SQ. (267)	CIGM93.214	0.61**	0.035	33	
GAN/AE.SQ. (437)	CIGM90.583	0.57*	0.029	30	
GAN/AE.SQ. (890)	*CIGM90.909*	0.54	0.039	26	
GARZA/BOY//AE.SQ. (271)	*CIGM88.1250-0B*	0.48	0.046	22	
GARZA/BOY//AE.SQ. (280)	CIGM92.1643	0.90***	0.037	61	
GARZA/BOY//AE.SQ. (467)	*CIGM92.1733*	0.53	0.034	25	
LARU/AE.SQ. (333)	CIGM92.1678	0.72***	0.035	44	
LCK59.61/AE.SQ. (308)	CIGM90.810	0.58*	0.032	30	
LCK59.61/AE.SQ. (324)	*CIGM90.815*	0.52	0.034	25	
LCK59.61/AE.SQ. (689)	CIGM90.892	0.58**	0.045	30	
LCK59.61/AE.SQ. (783)	*CIGM90.900*	0.55	0.036	28	
RABI//GS/CRA/3/AE.SQ. (891)	*CIGM90.602*	0.43	0.029	17	
RABI//GS/CRA/3/AE.SQ. (895)	CIGM90.603	0.76***	0.030	47	
RABI//GS/CRA/3/AE.SQ. (895)	CIGM90.603	0.62**	0.035	34	
RABI//GS/CRA/3/AE.SQ. (914)	*CIGM90.606*	0.45	0.031	19	
RASCON/AE.SQ. (385)	*CIGM92.1701*	0.40	0.031	16	
ROK/KML//AE.SQ. (507)	CIGM92.1750	0.64***	0.026	36	
SCA/AE.SQ. (272)	CIGM93.216	0.68***	0.027	40	
SCOOP_1/AE.SQ. (434)	*CIGM88.1335-0B*	0.49	0.055	22	
SNIPE/YAV79//DACK/TEAL/3/AE.SQ. (633)	*CIGM90.872*	0.41	0.036	16	
SORA/AE.SQ. (192)	*CIGM90.540*	0.55	0.030	27	
SORA/AE.SQ. (208)	CIGM88.1195-0B	0.61**	0.033	33	
SORA/AE.SQ. (211)	*CIGM90.541*	0.54	0.041	26	
YAR/AE.SQ. (493)	CIGM89.463-0Y	0.61**	0.038	33	
YAR/AE.SQ. (524)	*CIGM89.474-0Y*	0.47	0.030	21	
YAV_2/TEZ//AE.SQ. (457)	*CIGM90.833*	0.45	0.032	19	
YAV79//DACK/RABI/3/SNIPE/4/AE.SQ. (460)	*CIGM88.1348-0B*	0.54	0.043	26	
YUK/AE.SQ. (434)	*CIGM88.1334-0B*	0.54	0.032	26	
Resistant Check 'Red Chief'		0.46	0.023	20	
moderate check 2174		0.74***	0.025	45	
susceptible check 'Tam 105'		0.86***	0.024	58	

*, **, *** Significantly different from the mean of the resistant check at the 0.05, 0.01 and 0.001 probability levels respectively

† Original percentage data transformed by arcsine square root.

‡ Lesion type ratings from Xu et al. (2004), using a scale of 1 (resistant) to 5 (susceptible)

Table 2. Selected mean squares and variance component estimates from the analysis of variance of percentage leaf infection after inoculation of 94 synthetic hexaploid wheat accessions with *Pyrenophora-tritici repentis*.

Source of Variation	df	Mean Square	Variance Component Estimate	SE
Run	9	0.279*	.0013	.0007
Accession	93	0.171***	.0070	.0013
Experimental Error	837	0.033***	.0081	.0009
Sub sampling Error	926	0.017	.0169	.0008

*, *** Significant at the 0.05 and 0.001 probability levels, respectively.

Table 3. Minimum number of replications to determine a 20% difference in accession mean (transformed units) for percentage leaf infection of wheat after tan spot inoculation†.

		Number of plants (subsamples)		
Type I alpha‡	Type 2 beta	2	5	10
.05	.05	15	11	10
.05	.10	13	10	8
.05	.25	10	7	6

† Calculated from Appendix equation 2.

‡ Based on a two-sided *t*-test.

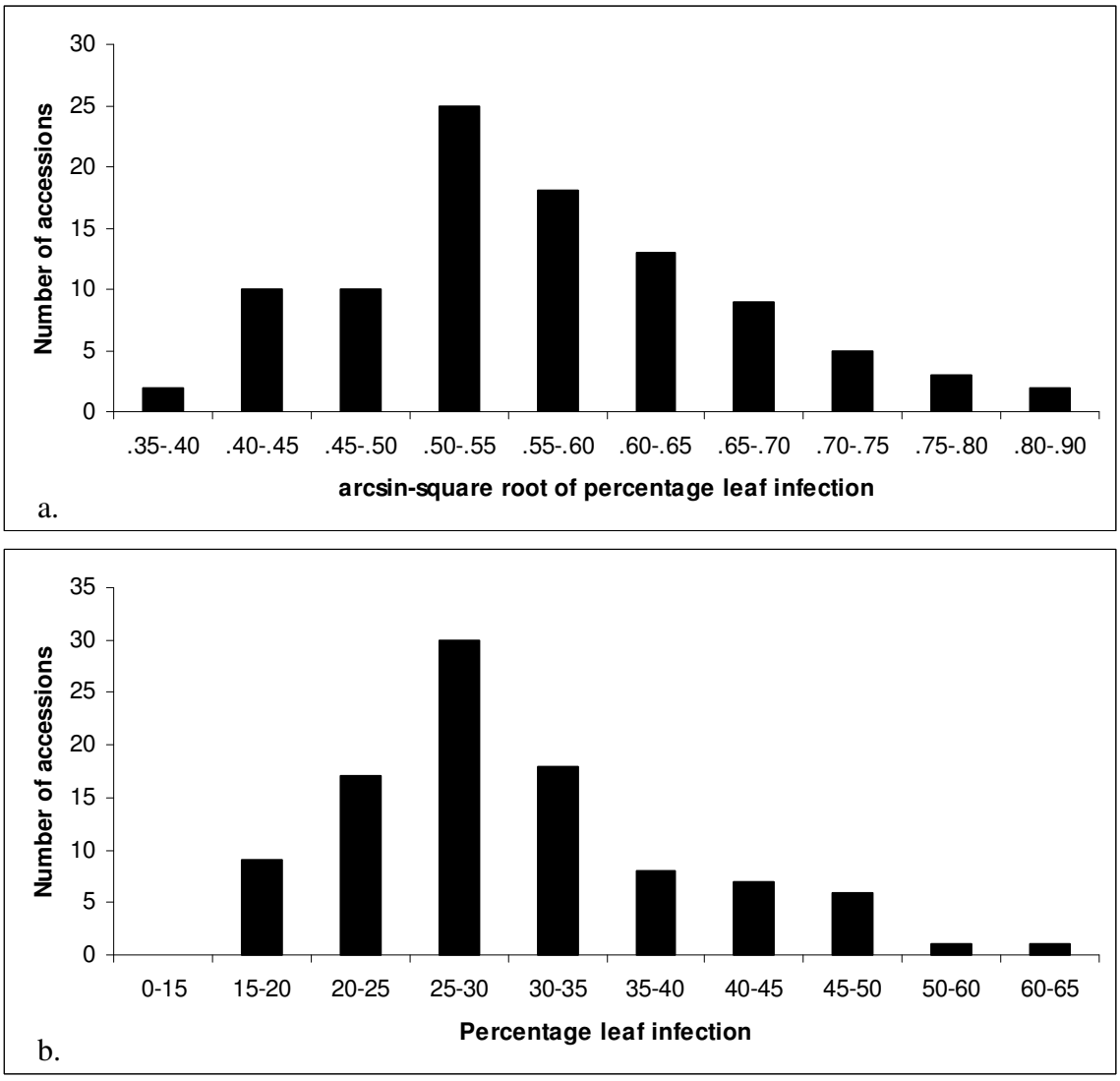

Figure 1. Frequency distribution for percentage leaf infection after tan spot inoculation of 94 synthetic hexaploid wheat accessions and check cultivars, expressed in non-transformed (%) (a) and transformed units (b).

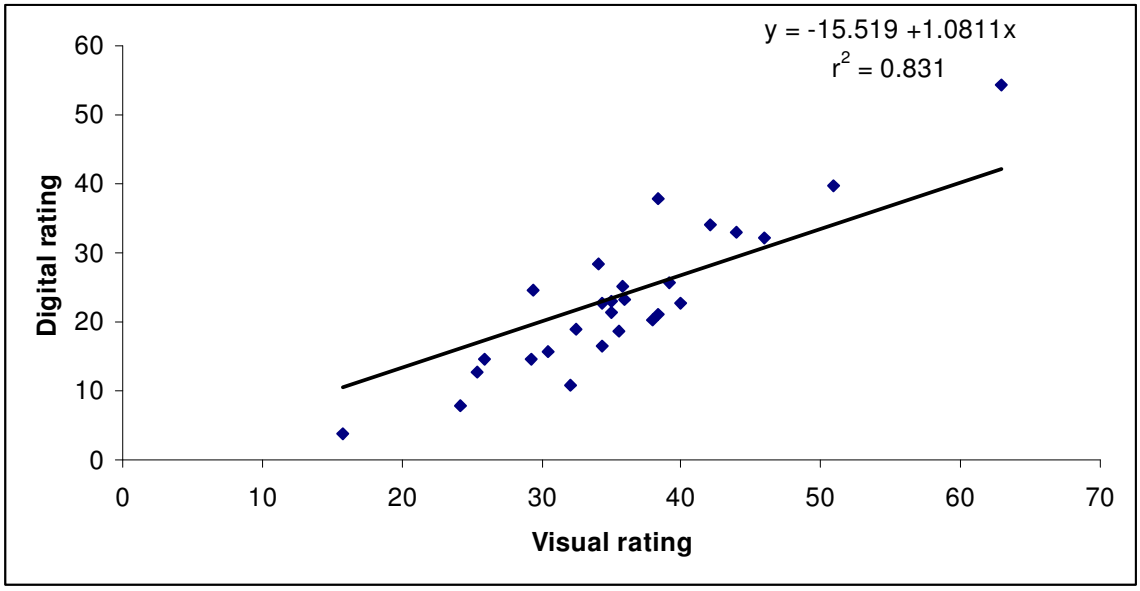

Figure 2. Correlation of visual scores and digital scores of percentage leaf infection after tan spot inoculation of 27 advanced breeding lines and check cultivars.

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APPENDIX

Table 1. Qualitative and quantitative resistance and sensitivity genes of wheat to the necrosis and chlorosis producing toxins of tan spot.

	Resistance to Necrosis	Resistance to Chlorosis	Sensitivity to Necrosis
Qualitative	Single Recessive Gene 3A tsn4 tsn1 on 5BL tsc2 on 3BL in tetraploids	Single Dominant Gene 2BS tsc2 Single recessive gene 1AS tsc1	Single Dominant Gene on 5BL
Quantitative	QTL 1BS QTL 3BL QTL 3AS QTL 5BL	QTL 1BS QTL 3BL QTL 1AS QTL 3AS QTL 5BL	

Equation 1. Repeatability

$$R^2 = \sigma^2_G / \{\sigma^2_G + [(1/r)(\sigma^2_{GxR})] + [(1/rp)(\sigma^2_{P|G|R})]\}$$

σ^2_G = Genetic variance
σ^2_{GxR} = Environmental variance
$\sigma^2_{P|G|R}$ = Subsampling variance
r = number of runs
p = number of plants (subsampling units)

Equation 2. Minimum number of replications

$$R > (2[T_{\alpha/2}+T_{\beta}]^2 \times [\sigma^2_{GxR} + (1/p(\sigma^2_{P|G|R})])/\delta^2$$

$T_{\alpha/2}+T_{\beta}$ = Value of T for 93 degrees of freedom
δ^2 = The value of the difference between two accession means

	1	2	3	4	5	6	7	8	9	10
1	54	83	97	93	77	93	49	81	97	65
2	88	64	59	70	79	70	85	55	56	66
3	52	96	76	86	85	77	59	83	50	61
4	69	60	95	82	56	88	95	86	90	89
5	91	75	71	87	84	57	53	76	91	92
6	57	48	72	73	53	58	79	51	72	87
7	68	63	74	62	78	48	74	82	69	80
8	66	80	90	67	50	94	60	52	71	62
9	51	55	94	58	92	67	54	63	78	96
10	81	89	65	61	49	68	64	84	73	75

Figure 1. Example of the randomized complete block design of experiment 1, group 2 run 3.

Table 2. Mean digital and visual reactions ratings to Ptr inoculation of advanced breeding wheat lines and check cultivars.

	Mean rating	
Synthetic derivative	Digital	Visual
1	28.27	34.00
2	25.15	35.83
3	14.59	25.91
4	25.73	39.09
5	32.18	46.00
6	54.45	62.92
7	33.97	42.14
8	22.69	34.38
9	24.49	29.37
10	16.5	34.25
11	7.98	24.25
12	39.69	50.90
13	21.34	35.00
14	33.01	44.00
15	22.82	40.0
16	14.72	29.18
17	20.25	38.00
18	23.13	35.91
Advanced breeding line		
Bullet	18.7	35.5
Doans	37.86	38.33
Duster	18.90	32.50
Fuller	23.10	35.00
OK02522W	15.62	30.50
TAM 112	7.90	32.0
Resistant check ‘Red Chief’	3.85	15.73
Moderate check 2174	12.61	25.38
Susceptible check ‘TAM 105’	21.17	38.33
LSD[†]	21.21	16.25

[†]Protected least significant difference value.

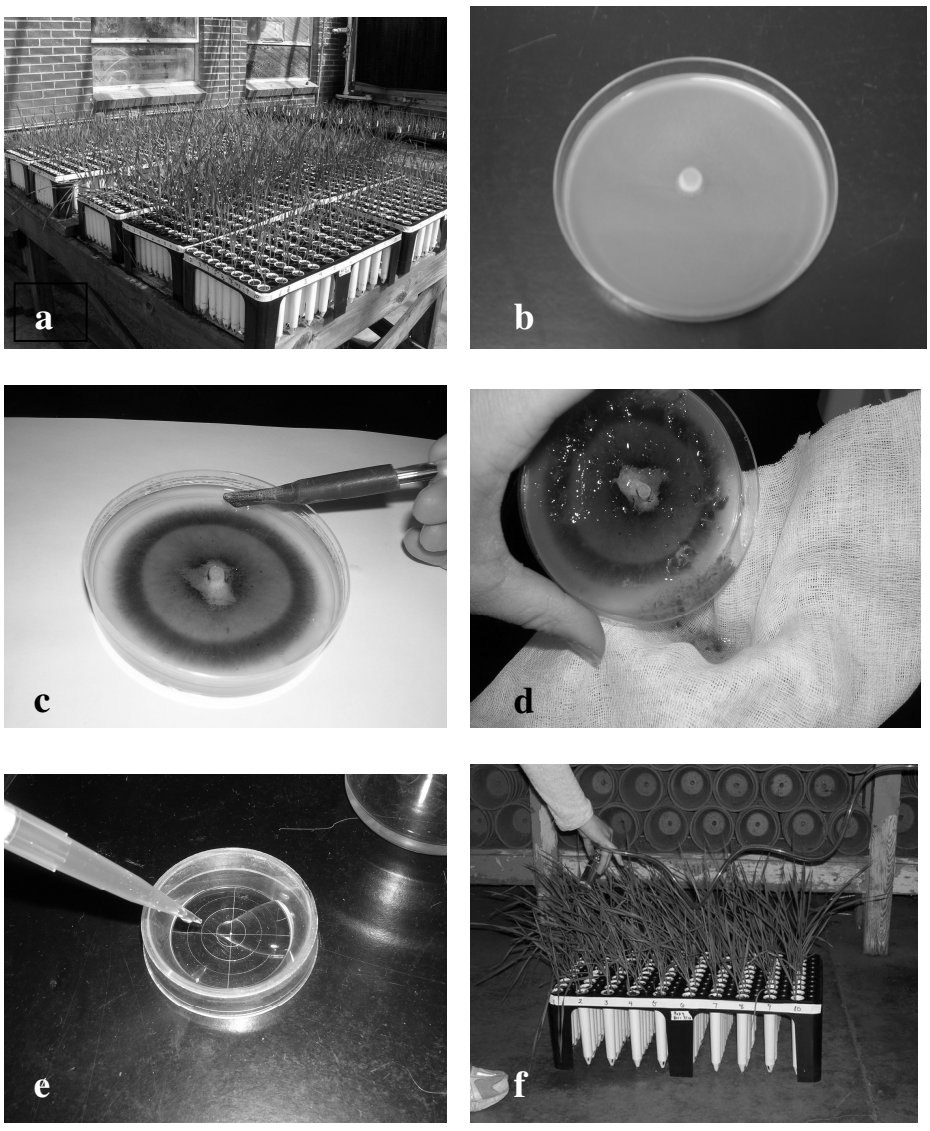

Figure 2. Photograph of steps of assay for reaction of wheat to inoculation of *Pyrenophora tritici-repentis*. (a) Accessions at 1 leaf to 5 leaf stages. (b) Ptr isolate on PDA. (c) Removal of conidia from a sporulating isolate maintained on vegetable agar. (d) Filtration through cheesecloth and creating a spore suspension. (e) Pipetting 1 mL spore suspension for counting. (f) Inoculating a run of accessions.

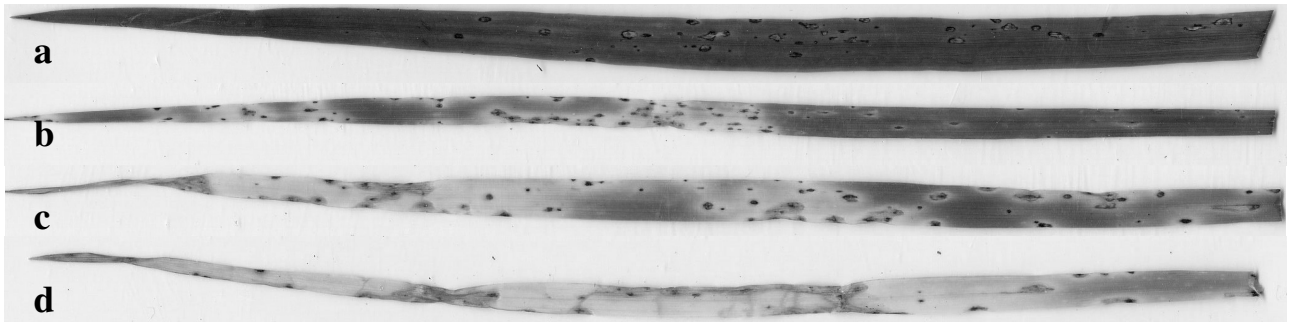

Figure 3. Photograph of wheat leaves after inoculation with tan spot. (a) 6% infection (b) 36% infection (c) 60% infection (d) 86% infection

VITA

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Candidate for the Degree of

Master of Science

Thesis: JANA FAYE MORRIS

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Major Field: Plant and Soil Science

Scope and Method of Study: A collection synthetic hexaploid wheat accessions developed at the International Corn and Wheat Improvement Center (CIMMYT) was evaluated for seedling resistance to tan spot in a greenhouse assay. The causal organism of tan spot *Pyrenophora trtitici-repentis* is an ascomycete that over-seasons on wheat stubble and can cause up to 50% loss in yield in favorable environments. Seedlings planted in a randomized complete block design were inoculated with a chlorosis and necrosis producing isolate collected from Oklahoma and evaluated for percent leaf infection at the 3 to 5 leaf stage one week post-inoculation. Of the 94 synthetic hexaploid wheat accessions tested, 54% showed high resistance when compared to the resistant check, 'Red Chief'.. The visual percentage leaf area infection ratings were compared with digital ratings calculated with Assess software (APS Press, 2002). The visual ratings were highly correlated with the digital ratings validating the use of visual estimates of percentage leaf area infection for tan spot reaction.

Findings and Conclusions: Synthetic hexaploid wheat is a potential source of resistance to tan spot. Resistance and susceptibility can be assessed using percentage leaf infection in a greenhouse assay.

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