Genetic Mapping of Pathogenicity and Aggressiveness of *Gibberella zeae* (*Fusarium graminearum*) Toward Wheat

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ABSTRACT

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Gibberella zeae is the major fungal pathogen of *Fusarium* head blight of wheat and produces several mycotoxins that are harmful to humans and domesticated animals. We identified loci associated with pathogenicity and aggressiveness on an amplified fragment length polymorphism based genetic map of *G. zeae* in a cross between a lineage 6 nivalenol producer from Japan and a lineage 7 deoxynivalenol producer from Kansas. Ninety-nine progeny and the parents were tested in the greenhouse for 2 years. Progeny segregated qualitatively (61:38) for pathogenicity:non-pathogenicity, respectively. The trait maps to linkage group IV, which is adjacent to loci that affect colony pigmentation, peritheciium production, and trichothecene toxin amount. Among the 61 pathogenic progeny, the

amount of disease induced (aggressiveness) varied quantitatively. Two reproducible quantitative trait loci (QTL) for aggressiveness were detected on linkage group I using simple interval analysis. A QTL linked to the *TRI5* locus (trichodiene synthase in the trichothecene pathway gene cluster) explained 51% of the variation observed, and a second QTL that was 50 centimorgans away explained 29% of the phenotypic variation. *TRI5* is tightly linked to the locus controlling trichothecene toxin type. The two QTLs, however, were likely part of the same QTL using composite interval analysis. Progeny that produced deoxynivalenol were, on average, approximately twice as aggressive as those that produced nivalenol. No transgressive segregation for aggressiveness was detected. The rather simple inheritance of both traits in this interlineage cross suggests that relatively few loci for pathogenicity or aggressiveness differ between lineage 6 and 7.

Additional keywords: AFLP, cereal, deoxynivalenol, nivalenol, scab, trichothecenes, *Triticum aestivum*.

Gibberella zeae (Schwein.) Petch (anamorph *Fusarium graminearum* Schwabe) causes scab or head blight of wheat and other small grains. As one of the most important plant diseases in the United States and many other parts of the world (30), it incurs serious economic losses not only in terms of yield (53) but also by contamination of the grain with trichothecene mycotoxins. These compounds are potent inhibitors of eukaryotic protein synthesis and can cause serious mycotoxicoses in humans and domesticated animals (29,42). Trichothecenes also are toxic to plants (17), and these compounds are thought to play a role in pathogenicity, i.e., ability to cause disease, aggressiveness, or both, i.e., quantity of disease induced by a pathogenic isolate on a susceptible host (11,17,41).

Genotypic differences among isolates of *G. zeae* from collections and field populations have been extensively described (5,13,31,33,54,55). O'Donnell et al. (38) proposed that the species is divided into a series of seven phylogenetic lineages on the basis of DNA sequences of six genes. Interestingly, sequences of trichothecene pathway genes do not generate the same phylogenies (51). The lineages have different geographic distributions, differ in production of trichothecene mycotoxins, and may differ in their ability to cause disease on particular crops.

Deoxynivalenol (DON), its acetylated derivatives 3-acetyl deoxynivalenol (3-ADON), and nivalenol (NIV) are the most important trichothecenes found in cereals (29). DON, also known as vomitoxin, is the most common trichothecene in Europe and North America (40). NIV-producing strains of *G. zeae* have been reported in Europe (4), Africa (45), Asia (44), and South America (14) but not in North America (1). The occurrence of high levels of NIV-producing strains that produce little or no DON, and vice versa, is now well established (32), and a single gene (*TRI13*) responsible for the differential ability to produce DON or NIV has been identified (25). NIV is considered to be more toxic to animals than is DON (46).

The role of trichothecenes in plant disease is not clear. A positive correlation between aggressiveness and DON production by *G. zeae* and *F. culmorum* has been reported (15,19), but other results have shown no correlation or have been inconsistent (2,26,50). A trichothecene-deficient *G. zeae* mutant induced by disruption of *TRI5*, the gene encoding the first enzyme in the trichothecene pathway, was still pathogenic to wheat, rye (11,41), and maize (17) but was less aggressive than its wild-type progenitor. Recently, Bai et al. (3) reported that DON-nonproducing strains could cause initial infections on wheat spikes but could not spread beyond the initial infection, suggesting that DON is an aggressiveness, rather than a pathogenicity factor (17,41). There are several reports that DON-producing strains are more aggressive toward plants than are those that produce NIV (27,32,34); however, there is at least one contrary report (7).

We took an alternative approach to studies of pathogenicity and aggressiveness of *G. zeae* by utilizing the progeny of a cross used

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to generate a previously constructed genetic map of *G. zeae* (21). The mapping cross was made between a lineage 6 nivalenol producer and a lineage 7 deoxynivalenol producer. The existing genetic map makes it possible to perform quantitative trait locus (QTL) analysis on the progeny. In addition, because the progeny segregate for the amount and type of trichothecene toxin produced, we could test the hypothesis that these traits are related to the pathogenicity or the aggressiveness of the strain.

Our objectives in this study were to (i) estimate the number of QTLs responsible for pathogenicity and aggressiveness and to locate the QTLs on the existing map of *G. zeae*, and (ii) determine if pathogenicity or aggressiveness is associated with the type or amount of mycotoxin produced under laboratory conditions. A preliminary report of this study has been published (10).

MATERIALS AND METHODS

Mapping population. The mapping population analyzed is the same as that used by Jurgenson et al. (21) to create a genetic map for *G. zeae*. A DON-producing strain, Z-3639 (member of lineage 7 [38]), isolated from wheat in Kansas (5) and a NIV-producing strain, R-5470 (member of lineage 6 [38]), isolated from barley in Japan (provided by Paul E. Nelson, Department of Plant Pathology, Pennsylvania State University, University Park) served as the parents. The map is based on the segregation of 1,048 polymorphic amplified fragment length polymorphism (AFLP) markers to 468 loci in 99 haploid progeny. The total map length is approximately 1,300 centiMorgans (cM) with nine linkage groups and an average distance between loci of 2.8 cM.

Inoculum production. *G. zeae* was cultured on synthetic nutrient-poor agar (SNA) (37) in 90-mm-diameter petri dishes that were incubated at 24°C in the dark for the first 24 h and afterward exposed to two black light tubes (Philips TLO, 40 W/80; Royal Philips Electronics, Amsterdam) for 25/22°C (day/night) for 1 to 2 weeks until conidia formed. Not all strains produced sufficient conidia under these conditions. These strains grew as flat pionnotal colonies that conidiated when cultured on potato dextrose agar (PDA; Merck, Darmstadt, Germany). Conidia were washed from the plates with sterile tap water, and the concentration was adjusted to 5×10^4 spores per ml following a count in a hemacytometer.

Greenhouse tests and evaluation. Seeds of a susceptible German spring wheat genotype, Munk, were sown in plastic trays and cultivated in a greenhouse. After approximately 10 days, seedlings were transplanted in groups of six into plastic pots (13×13 cm²) containing compost soil and cultivated in a cool greenhouse with no temperature regulation. Plants were fertilized with nitrogen during the growing stage. Four plant heads per pot, with a total of five pots per strain were inoculated at mid-anthesis. Approximately 10 µl of a spore suspension was injected into the left and right floret of a central spikelet on both sides of each head (4×10 µl of injections per head) with a hypodermic needle (0.5-mm gauge) in a controlled plant growth chamber (21°C day/19°C night). Inoculated plants were covered with plastic sheets and incubated for 48 h in the dark (90 to 100% relative humidity), then uncovered and incubated for an additional 48 h (60 to 80% relative humidity) under artificial light. Because of limited space in the plant growth chamber, the progeny were chosen randomly and tested in 3 to 5 batches with both parental strains included in each batch. After the first 4 days of incubation, plants were transferred to a heated greenhouse with a mean temperature of 18 to 20°C and a day length (artificial light) of 16 h. The number of infected spikelets was counted 11, 14, 18, and 23 days after inoculation and calculated relative to the total number of spikelets per head. To minimize error, results from the four evaluation dates for each experiment were averaged (= raw values) and the means adjusted relative to the respective batch mean consisting of 10 to 24 pathogenic progeny per batch. This experiment was conducted

during 2001 and 2002. Toxin data were the same as used for producing the genetic map (21).

Statistical analyses. Data analyses were based on pot means, i.e., the mean of four heads. When the entire set of progeny was analyzed, the data were not normally distributed (Fig. 1). Therefore, analysis of variance (ANOVA) was calculated only for the pathogenic progeny. In the pathogenic progeny subpopulation, error variances were homogeneous across years according to Bartlett's test (43). The 2 years were treated as a series of random environments according to Cochran and Cox (9). Estimates of variance components were calculated as described by Snedecor and Cochran (43). Broad-sense heritabilities (H^2) were estimated on an entry-mean basis (12) as the proportion of genotypic to phenotypic variance. All analyses were performed with the computer package PLABSTAT (H. Friedrich Utz, University of Hohenheim, Stuttgart, Germany). The effects of genotypes, replicates, and years were assumed to be random variables. Data were analyzed for raw and adjusted disease severity.

Initial detection of QTLs for pathogenicity or aggressiveness was done with Map Manager QTX11 software (28) and MAP-MAKER for MacIntosh (24). Markers were subjected to simple interval analysis (16) using QGENE (35) to identify significant associations between AFLP markers and aggressiveness with a significance level at logarithm of odds (LOD) 3.0 (23). Relationships between QTLs were investigated by composite interval mapping using PLABQTL (47). Two separate analyses were run (all progeny, and pathogenic progeny only). QTL analyses were performed with adjusted disease severity values only.

RESULTS

Assessment of pathogenicity and aggressiveness. Disease severity increased with time (Table 1), with Z-3639 always the most aggressive (26% of the spikelets were infected 11 days postinoculation to 53% of the spikelets infected 23 days postinoculation). The mean values for the pathogenic progeny followed a similar pattern (18 to 36% infected spikelets over this 12-day period. R-5470 and the 38 nonpathogenic progeny were always the least aggressive, with the number of infected spikelets at 11 days postinoculation <0.3%. This value increased to only ~1.6% by 23 days postinoculation.

The frequency distribution of disease severity appeared bimodal (Fig. 1). The Kansas parental strain, Z-3639, was the most aggressive entry (average adjusted disease severity 215% across

Fig. 1. Frequency distribution of disease severity, percentage of infected spikelets adjusted to the batch mean for 99 progeny from the cross of *Gibberella zeae* strains Z-3639 (parental mean 215%) and R-5470 (4.3%) in the greenhouse in 2001 (■) and 2002 (□). The least aggressive group contains all 38 of the nonpathogenic progeny.

the years) and the Japanese strain, R-5470, was essentially non-pathogenic (average adjusted disease severity 4.3% across the years). Thirty-eight progeny were not pathogenic (adjusted disease severity <25%) with the members of this subpopulation reacting similarly to the Japanese parental strain. Sixty-one of the progeny were pathogenic in both years (adjusted mean disease severity $\geq 25\%$). The 61 pathogenic progeny varied in their disease severity, which was interpreted as differences in aggressiveness. Segregation was similar in both years, and no transgressive segregants were observed.

Significant ($P = 0.01$) differences in aggressiveness (Fig. 1) were confirmed in an ANOVA of the results from the pathogenic progeny (Table 2). Progeny $\times$ year interaction also was significant ($P < 0.01$). Estimates of the H^2 of aggressiveness across both years for raw and adjusted values were 0.82 and 0.81 (Table 2).

TABLE 1. Mean disease severity across 2 years (percentage of infected spikelets, raw values) in the greenhouse after inoculation of *Gibberella zeae* strains Z-3639 and R-5470, and the mean of 61 pathogenic and 38 nonpathogenic progeny of a cross between these two strains

Entry	Days after inoculation ^x			
	11	14	18	23
Z-3639	26 a	39 a	47 a	53 a
Pathogenic progeny	18 a	24 b	29 b	36 a
LSD _{5%} ^y	11	15	17	23
R-5470	0.3	0.4	0.7	1.6
Nonpathogenic progeny ^z	0.1	0.2	0.4	1.0

^x Numbers followed by different letters in the same column are significantly different at $P = 0.05$.

^y Least significant difference at $P = 0.05$.

^z Analysis of variance was not calculated because the frequency distribution deviates from normality.

TABLE 2. Variance component estimates and entry-mean heritabilities of disease severity of 61 pathogenic progeny of *Gibberella zeae* with raw values and following normalization to the respective batch means across 2 years

Parameter	DF	Raw values	Adjusted
Sources of variation			
Year (Y)	1	4.96	— ^x
Progeny (P)	60	127** ^y	1500**
P $\times$ Y	60	50**	600**
Pooled error	487	25	590
Heritability (H^2)		0.82	0.81
90% C.I. for H^2 ^z		0.70–0.89	0.69–0.89

^x Negative estimate.

^y ** = significant at $P = 0.01$ (F -test).

^z Confidence intervals (C.I.) on H^2 were calculated by the method of Knapp and Bridges (22).

TABLE 3. Marker and marker position, phenotypic and genetic effects, maximum logarithm of odds (LOD) scores, and proportions of phenotypic variance explained by markers (R^2) for percentage of infected spikelets in progeny of *Gibberella zeae* cross Z-3639 $\times$ R-5470 in two subsamples tested in the greenhouse for two experimental years

Subsample	Linkage group region	Nearest marker	Marker position ^x (cM)	Phenotypic difference of marker classes ^y		LOD	R^2 (%) ^z	Additive genetic effect
				Z-3639	R-5470			
All progeny	LG IV-1	<i>TOX1</i>	24	99	18	12	43	–76
		<i>PER1</i>	33	100	10	16	51	–84
		<i>PIG1</i>	40	100	2.3	20	60	–91
		<i>4B</i>	43	96	9.2	15	50	–84
		<i>4P</i>	100	98	7.3	18	56	–89
Pathogenic progeny only	LG IV-2							
	LG I-1	<i>TRI5</i>	111	130	66	9.6	51	–63
	LG I-2	<i>EAAMTG0655K</i>	156	118	65	4.6	29	–50

^x Based on existing genetic map of *G. zeae* (21).

^y All marker class differences were significant at $P < 0.001$. Adjusted mean disease severity expressed as percentage of infected spikelets following normalization to the respective batch means from two different years.

^z R^2 = percent phenotypic variance explained by each locus using single marker regression analysis.

The variances observed for the raw and the adjusted data were similar in proportion.

Chromosomal localization and gene action and interaction.

When the set of all progeny was analyzed by single marker regression, several markers on linkage group (LG) IV, including three phenotypic markers, *PER1* (perithecial production), *PIG1* (red pigment production), and *TOX1* (trichothecene toxin amount), had highly significant LODs for marker class means for disease severity (Table 3). Simple interval analysis revealed a large peak near *PIG1* and another similar peak around locus *4P* in both years. Composite interval analysis showed that the peak at *4P* decreased (2002) or disappeared (2001) when background effects at *PIG1* were controlled. This suggested that the two peaks are related. When pathogenicity was treated as a qualitative trait segregating 61:38 (new locus was designated *PATH1*), it mapped adjacent to *PIG1*, *PER1*, and *TOX1* (Fig. 2). The mapping program also could place *PATH1* near locus *4P* with slightly lower LOD. This again suggested that the two regions of LG IV are related. No other significant QTLs on other linkage groups were detected in the analysis of the full set of progeny.

When only the pathogenic progeny were analyzed by single marker regression, none of the markers on LG IV had a significant LOD. However, a large region on LG I, including markers *TRI5* and *EAAMTG0665K*, were associated with aggressiveness (Table 3). Differences in marker class means associated with these QTLs were smaller than the differences associated with the markers on LG IV, but still statistically significant ($P < 0.01$). Simple interval analysis revealed a QTL peak near *TRI5* and a smaller peak near *EAAMTG0655K* in both years (Fig. 3). Composite interval analysis showed that the peak at *EAAMTG0655K* disappeared in both years when background effects at *TRI5* were controlled. No reproducible QTLs were detected on any of the other seven linkage groups.

The two QTLs on LG I explain 51 and 29%, respectively, of the observed variation for aggressiveness in this cross (Table 3). The alleles for nonpathogenicity and lower aggressiveness all originated from the R-5470 parent, although some recombinants can be noted in each case (Table 4). The LG I-1 and LG I-2 QTLs for aggressiveness are not additive, since the mean disease severity (as adjusted percent infected spikelets), 62 to 68%, is the same for those with neither or either one of the two Z-3639 alleles, while the mean disease severity for progeny with both the LG I-1 and LG I-2 Z-3639 alleles is 131% (Table 4).

Relationship of toxin production to aggressiveness. Among the 99 progeny from this cross, 54 were previously classified as moderate- to high-level producers, and 45 were classified as very low-level producers of trichothecene mycotoxins in vitro (21). Toxin amount was strongly related to disease severity, with all of the nonpathogenic progeny classified as low-level producers (Fig. 4A). The adjusted mean disease severity for high-level pro-

ducers was 98% and the mean for low-level producers was 18%, which was significantly different ($P < 0.0001$). Fifty-four high level-producing progeny were classified for ability to produce nivalenol or deoxynivalenol (Fig. 4B). Twenty-six strains that produced nivalenol and twenty-eight strains that produced deoxynivalenol had adjusted mean disease severities of 131 and 67%, respectively ($P < 0.0001$).

DISCUSSION

This study is the first to assign specific chromosomal regions in *G. zeae* to differences in disease severity using QTL analysis. Most QTL analyses have been made in plant or animal systems, although there are a few studies with fungi, e.g., Hawthorne et al. (18) and Welz and Leonard (52). We exploited an existing relatively dense genetic map (21) based on 99 progeny from a wide cross of a lineage 6 nivalenol producer from Japan and a lineage 7 deoxynivalenol producer from Kansas.

On the basis of a 2-year greenhouse experiment, pathogenicity consistently segregated in a qualitative manner, 61:38. The pathogenicity locus in *G. zeae* was designated *PATH1* and mapped to LG IV (Fig. 2). Even under very favorable conditions for disease, nonpathogenic strains could only rarely spread beyond the inoculated spikelets. Although inheritance of pathogenicity as a single Mendelian gene has been reported in other fungi (e.g., *Cochliobolus carbonum* [36]), nonpathogenicity is an unusual character for field isolates of *G. zeae* (31,32). The segregation ratio for *PATH1* was significantly different from 1:1 ($P = 0.02$). Segregation ratios on LG IV are known to be distorted in this cross because of a putative chromosomal rearrangement and selection for a *nit* marker that was required to make the cross (21). The rearrangement could explain the observation of two related peaks on LG IV and the ambiguity of the linkage relationships for *PATH1*. Since the second peak was reduced or removed by composite

Fig. 2. Location of qualitative gene (*PATH1*) controlling pathogenicity on linkage group IV on the basis of segregation in 99 progeny in the greenhouse in 2001 and 2002.

interval analysis, it is likely that it is part of the same QTL as the first peak. If *PATH1* was at or near the breakpoints of the putative inversion on LG IV, there could appear to be two loci due to artifacts of the mapping process. Interestingly, when nonpathogenic progeny were removed from the analysis, no QTLs on LG IV could be detected. Therefore, the pathogenicity locus on LG IV was important for pathogenicity, but not for aggressiveness.

The parental strains differ in several additional phenotypic traits, including pigmentation (*PIG1*) and level of toxin produced

Fig. 3. Location of quantitative trait locus for aggressiveness on linkage group I (LG I) of *Gibberella zeae* based on segregation in 61 pathogenic progeny tested in the greenhouse in 2001 (dotted line) and 2002 (solid line). Bar indicates 20 cM. The logarithm of odds (LOD) significance likelihood of 3.0 is marked.

TABLE 4. Marker class means for adjusted mean percentage of infected spikelets at loci *TRI5* and *EAAMTG0655K* on linkage group I for 61 pathogenic progeny from *Gibberella zeae* cross Z-3639 × R-5470 tested in the greenhouse in two different years

<i>EAAMTG0655K</i>	<i>TRI5</i> ^y		Mean
	Z-3639	R-5470	
Z-3639	131 (31) ^z	68 (10)	100 (41)
R-5470	66 (1)	62 (19)	64 (20)
Mean	99 (32)	65 (29)	

^y Percentage of infected spikelets.
^z Numbers in parentheses indicate number of progeny represented in mean.

(*TOX1*), that map on linkage group IV (21). Pigmentation and pathogenicity are correlated in several plant pathogens including *Colletotrichum lagenarium* (39), *Nectria haematococca* (18), and *Magnaporthe grisea* (8), and *PIG1* was the marker most closely linked to *PATH1*. The level of toxin produced has been reported as a pathogenicity factor in the interaction between *G. zeae* and wheat and maize (3,11,17,41). The *TOX1* locus is associated with the amount of toxin produced in vitro, maps near *PATH1* on LG IV, and might play a role in determining pathogenicity. The large difference in disease severity between high-level toxin producers and low-level producers favors that hypothesis. The absence of progeny that produce high levels of deoxynivalenol or nivalenol, but that are nonpathogenic also supports the hypothesis. However, the existence of progeny (Fig. 4A) that produce low levels of toxin in vitro, but that are highly pathogenic argues against that hypothesis. It is possible that some progeny genetically capable of being high-level producers failed to produce high toxin levels in vitro. In that case, *TOX1* and *PATH1* could, in fact, be the same locus. It should be noted, however, that the type of toxin, but not the amount of toxin correlated between field and in vitro data (15).

Hou et al. (20) recently reported that a mitogen-activated protein kinase gene (*MGV1*) in *G. zeae* is involved in processes re-

lated to sexual reproduction and pathogenicity. The Japanese parental strain, R-5470, and the *MGV1* mutant (20) produce low levels of toxin, have reduced female fertility, and are nonpathogenic. Thus, R-5470 may carry a pleiotropic mutation similar to that in *MGV1*.

If the pathogenic progeny are evaluated as an independent subset, then the variation remaining for mean disease severity is still significant, and the effect of the locus on LG IV is masked. Under these conditions, two additional linked QTLs were identified on LG I that account for 51 and 29% of the variation associated with this trait in this cross (Table 3; Fig. 3). Since the smaller peak was removed in both years by composite interval analysis, it is likely part of the same QTL. We consider the differences affected by this QTL to be differences in aggressiveness sensu Vanderplank (48). Although quantitative differences in disease severity have been reported for field isolates of *G. zeae* (31–33), this study is the first to demonstrate quantitative differences in aggressiveness in a segregating population and to evaluate the genetic basis for these differences. QTL analysis clearly distinguished these loci for aggressiveness on LG I from those controlling pathogenicity on LG IV.

Interestingly, the *TRI5* gene, which encodes the enzyme trichodiene synthase (6) in the trichothecene biosynthetic pathway gene cluster in *G. zeae*, was closely linked to QTL LG I-1 (Table 3). *TRI5* presumably serves as a marker for the trichothecene cluster in which *TRI13*, the gene that determines whether nivalenol or deoxynivalenol will be produced (25), also resides. As far as we can tell, the genes in the trichothecene cluster other than *TRI13* are all functional in both strains. The *TRI13* allele from Z-3639 is presumably nonfunctional, which leads to the production of deoxynivalenol instead of nivalenol. On average, the deoxynivalenol-producing progeny from our cross were approximately twice as aggressive on wheat as the nivalenol-producing progeny (Fig. 4B). This result is consistent with correlations observed in field collections (27,32,34). Thus, our data are consistent with the hypothesis that the QTL for aggressiveness on LG I results from allelic differences at one or more of the loci in the trichothecene gene cluster, probably *TRI13*.

Previous studies (32,33) suggested that aggressiveness is a continuous character and therefore, probably the result of numerous quantitative genes. In contrast, this study suggests that only two reproducible loci affecting disease severity were segregating in this wide cross between lineage 6 and 7. We also found no evidence of transgressive segregation, which would support the hypothesis that many quantitative genes control aggressiveness. Still, some aggressiveness QTLs could have been missed in this study. First, it is possible that the parents were not polymorphic for some important QTLs. Second, the statistical power of the analysis is reduced by the relatively small number of pathogenic progeny and the segregation distortion on LGs II, IV, V, and VI. Third, some QTL may have been masked by environmental variables or might be more readily detected in field trials instead of greenhouse experiments. Additional mapping populations with more progeny might enable us to detect other QTLs that have lesser effects and perhaps map elsewhere in the genome. To assess accurately the effect of aggressiveness factors other than toxin type and remove toxin production from consideration as a pathogenicity factor, a cross between strains that produce similar levels of deoxynivalenol or nivalenol but differ in the level of disease severity should be analyzed.

Our results may have implications for the evolution of more aggressive *G. zeae* populations. We expected to see some transgressive segregation in the progeny. However, none of the progeny were more aggressive than the lineage 7, deoxynivalenol parent. Therefore, the risk of nivalenol-producing immigrants resulting in highly aggressive new strains into regions dominated by DON producers may be low. If highly resistant wheat genotypes are grown on a large scale, aggressiveness might increase in the

Fig. 4. Frequency distributions of infected spikelets (%) adjusted to their respective batch means in wheat cv. Munk inoculated with progeny from the cross of *Gibberella zeae* strains Z-3639 (high-level deoxynivalenol (DON) producer) and R-5470 (low-level nivalenol (NIV) producer) in the greenhouse across two experimental years. **A**, High (□) or low (■) levels of toxin production scored for all progeny (21). **B**, DON (□) or NIV (■) scored for high levels of toxin producers only (21).

pathogen population as a whole. Specific wheat cultivar $\times$ fungal strain interactions are unknown in this organism (49), but the lack of observed interactions may be because of, at least in part, to ignorance of the genetics of the pathogen. Now that a QTL for aggressiveness has been confirmed on LG I, it is possible to look for cultivar $\times$ strain interactions, e.g., by testing the aggressiveness of nivalenol and deoxynivalenol strains on highly resistant wheat varieties. Thus, QTL mapping of these traits in the *G. zeae* could identify genes involved in specific interactions between the host and the pathogen and provide basic information needed for the management of host and pathogen populations within the wheat agro-ecosystem.

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