NOTE

Fusarium Head Blight Symptoms and Mycotoxin Levels in Single Kernels of Infected Wheat Spikes

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Fusarium head blight (FHB) is a destructive fungal disease of wheat (*Triticum* sp.) and other cereal species. In North America, it is mainly caused by *Fusarium graminearum* Schwabe and its teleomorph *Giberella zeae* (Schw.) Petch and, to a lesser extent, by other *Fusarium* species (Parry et al 1995; Bottalico 1998; Bottalico and Perrone 2002). The infected cereal crops have reduced grain weight and yield, and the trichothecene mycotoxins, which accumulate in the infected grains, constitute a health risk to humans and animals (McMullen et al 1997). Various management practices are employed to reduce the yield losses and mycotoxin accumulation; however, these practices are meant to augment the use of resistant cultivars, which is the most effective and sustainable way to manage the impact of the disease (Pirgozliev et al 2003; Oliver et al 2005).

Host resistance and reaction of resistant wheat cultivars to FHB infection is quite complex (Mesterhazy 1995). Resistance mechanisms are often classified as passive or active. Passive mechanisms may include phenological and morphological traits such as plant height, the presence of awns, spikelet density, and time to flowering. Hilton et al (1999) noted a negative relationship between plant height and FHB resistance in wheat. Moreover, wheat and barley FHB resistance was significantly correlated with plant height and heading date (Zhu et al 1999; Klahr et al 2007). However, Jiang et al (2006) reported no noticeable genetic associations between FHB resistance and agronomic traits such as plant height, spike length, number of spikelets, and heading dates. This lack of consistency in trait correlations is not surprising given the variation in inoculum loads, sources of inoculum, weather conditions, and variable host stage during which the disease might develop, i.e., from anthesis to mid grain-filling.

Active mechanisms of FHB resistance have been classified as involving a number of resistance components: 1) resistance to invasion (type I); 2) resistance to spreading (type II); 3) resistance to mycotoxin accumulation (type III); 4) resistance to kernel infection (type IV); and 5) tolerance (type V) (Mesterhazy et al 1999). Spray inoculation followed by an evaluation of disease incidence is used to screen genotypes for type I resistance. Most often, this screening is carried out in field nurseries. Type II resistance is more routinely evaluated under controlled conditions by inoculating a middle floret of the spike and assessing disease spread. Resistance types III, IV and V are more difficult to assess and often are more expensive to screen. Consequently, breeding programs rarely evaluate for resistance types III, IV, and V (Gilbert and Tekauz 2000).

The assessed level of FHB infection and deoxynivalenol (DON) accumulation levels in grain are not always correlated (Birzele et al 2002). The lack of a correlation is likely due to the complex host resistance, differences in aggressiveness of the fungus, differences in DON production among *Fusarium* species and isolates, differences in growing climates, and differences in cropping rotations at the growing site (Walker et al 2001; Carter et al 2002).

Savard et al (2000) measured DON accumulation separately in floral parts and in the rachis and peduncle of artificially inoculated spikes of the susceptible spring wheat Roblin. High DON concentrations were measured in the inoculated florets at 4 days after anthesis. As the disease progressed, DON increased in spikelets below the inoculation point, while lower DON concentrations were detected in spikelets above the inoculation point.

Studying the progression of disease symptoms and mycotoxin accumulation among mature single kernels in wheat spikes relative to the point of infection may be helpful in understanding the reaction of a cultivar to FHB infection. The amount and distribution of mycotoxin among single kernels from artificially inoculated spikes may help determine whether a type II or type III resistance is expressed. The objective of this study was to use a single kernel analysis to assess the accumulation of DON and 15-*O*-acetyl-4-deoxynivalenol (15-ADON) in grain derived from artificially inoculated spikes of two wheat cultivars, one reported to be resistant and the other susceptible to FHB.

MATERIALS AND METHODS

Wheaton (FHB susceptible) (Mackintosh et al 2006) and PI 69251 (FHB moderately resistant) (Zhang and Jin 2003) were the genotypes compared for FHB symptoms and mycotoxin (DON and 15-ADON) accumulation. The plants were grown in 3-L pots with three plants per pot using Metro-Mix 200 potting medium (The Scotts Company, Marysville, OH) and maintained in a greenhouse under a 16 hr photoperiod, 22°C ± 4°C day temperature, and 18°C ± 2°C night temperature. The spikes were inoculated at anthesis by injecting a marked single central floret of a spikelet in the middle of the spike with 10 µL of macro conidia (1×10^5/mL) of *F. graminearum* isolate Z-3639 (NRRL accession 29169). After inoculation, plants were covered with plastic bags for 48 hr to maintain a humid microenvironment. Spikes were harvested when the kernels were mature. Kernels in each spikelet were manually removed beginning from the basal end upwards. The kernels in each spikelet were separately placed in cells of numbered pill boxes for further analysis. To identify the position of kernels in the spike in relation to the point of inoculation, the central spikelet with the inoculated floret was designated as spikelet 0; those above the central spikelet and progressing toward the distal end of the spike were assigned sequential positive integers; those below and progressing toward the basal end of the spike were assigned sequential negative integers.

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One kernel was randomly selected from each spikelet and examined microscopically for the presence (1) or absence (0) of visible *Fusarium* mycelia. Kernel weights were recorded and a visual assessment of the presence or absence of FHB damage was made. Kernels with a shrunken, discolored, pale, or whitish appearance were determined to exhibit damage due to FHB. The inoculated floret and those closest to it had very small, underdeveloped kernels. Those rudimentary kernels that weighed <3.0 mg were not used in this analysis. The levels of DON and 15-ADON were determined by GC-MS (Mirocha et al 1998; Jiang et al 2006). The composite DON values of the spikes were estimated using grain weight and DON content data.

Single kernel weight and mycotoxin data were subjected to a two sample *T*-test procedure to test whether the mean values of single kernel weight and mycotoxin levels between the two cultivars and between two different positions within the spikes were similar. The two different positions within the spike included 1) kernels above or below the inoculated middle spikelet and 2) kernels near or far from the middle inoculated spikelet. Kernels 1–5 above and below the inoculated spikelet were considered as the kernels near the inoculated spikelet while kernels 6–10 above and below the inoculated spikelet were considered as those far from the inoculated spikelet.

RESULTS AND DISCUSSION

The presence or absence of FHB damage and *Fusarium* mycelia, weight of kernels, and mycotoxin accumulation of single kernels randomly selected from spikelets above (+1 to +10), below (–1 to –10) and in the inoculated spikelet (0) of the three wheat spikes (S1–S3) are presented in Tables I and II for genotypes PI 69251 and Wheaton, respectively. Kernel weight, number of kernels with detectable levels of DON and 15-ADON, and the average mycotoxin levels in relation to the presence or absence of FHB damage in kernels (scab or *Fusarium* mycelia) are presented in Table III. Average weight and mycotoxin concentrations of single kernels between the two cultivars and in kernels extracted above, below, near or far from the middle inoculated spikelet of wheat spikes are given in Table IV.

Mycotoxin accumulation among the kernels along the spike was distinctly different for the two wheat lines. Mycotoxin accumulation in PI 69251 was mainly limited to the kernels below the point of inoculation, whereas the kernels above it, with or without visible FHB damage, had nondetectable or low mycotoxin accumulation (Table I). The levels of mycotoxins in kernels below the inoculated spikelet were significantly higher than the levels in kernels above in PI 69251; the average kernel weights were the same (Table IV). In contrast, higher mycotoxin concentrations were measured in kernels above and below the point of inoculation in spikes of Wheaton (Table II). In this cultivar, though the mycotoxin levels were not significantly different, kernels above the point of inoculation had significantly lower weights than the kernels below (Table IV).

Kernels close to the inoculated floret had the highest mycotoxin accumulation, and mycotoxin accumulation gradually decreased in kernels removed from spikelets located farther away from the point of inoculation. However, the weights and mycotoxins in kernels near and far from the central inoculated spikelet were not significantly different for cultivar PI 69251; quite the opposite was observed in susceptible cultivar Wheaton (Table IV). Higher mycotoxin concentrations and the presence of mycotoxins in a higher proportion of kernels resulted in spikes of Wheaton with higher composite mycotoxin levels (140.3–396.3 ppm of DON and 5.2–8.2 ppm of 15-ADON) (Table II). In contrast, comparatively lower mycotoxin levels and a higher proportion of kernels without mycotoxin above the point of inoculation resulted in spikes of PI 69251with lower mycotoxin levels (22.4–94.2 ppm of DON and 0.4–3.0 ppm of 15-ADON) in PI 69251 spikes (Table I). Overall, kernels of FHB-susceptible cultivar Wheaton had significantly higher mycotoxin levels than the kernels of moderately resistant PI 69251 (Table IV).

The low or nondetectable levels of mycotoxins in the spikelets above the point of inoculation in PI 69251 may be due to expression of a type II resistance mechanism. Kang and Buchenauer

TABLE I
***Fusarium* Head Blight Symptoms (scab, mycelia), Kernel Weight, and Mycotoxin Levels of Three PI 69251 Wheat Spikes (S1–S3)[a]**

	Kernel Property														
Kernel	FHB Damaged[b]			Mycelia[b]			Kernel Weight (mg)			DON (ppm)			15-ADON (ppm)		
Position	S1	S2	S3	S1	S2	S3	S1	S2	S3	S1	S2	S3	S1	S2	S3
10	1			1			18.8			nd[c]			nd		
9	0	0	0	0	0	0	22.0	18.9	18.8	nd	nd	nd	nd	nd	nd
8	1	0	0	1	0	0	19.8	10.1	18.4	nd	nd	nd	nd	nd	nd
7	1	0	0	0	0	0	23.3	24.9	18.4	nd	7.0	nd	nd	1.9	nd
6	1	0	1	1	0	0	24.8	19.3	16.0	nd	nd	nd	nd	nd	nd
5	1	0	1	0	0	0	24.7	24.9	18.0	nd	2.5	nd	nd	0.3	nd
4	1	0	1	1	0	0	22.1	27.7	16.6	2.6	4.1	nd	nd	0.5	nd
3	1	0	1	1	0	0	18.3	20.9	14.1	84.1	nd	nd	1.4	nd	nd
2		0	1		0	0		20.9	13.6		1.2	nd		nd	nd
1	1			1			18.9			75.6			1.3		
0	1			1			22.7			91.1			1.1		
–1	1			1			27.7			55.5			1.1		
–2	1			1			28.7			33.1			0.5		
–3	1	1	1	1	1	0	29.2	4.4	20.2	28.9	652.7	7.0	0.6	20.5	nd
–4	0	1	1	0	1	1	34.5	5.1	8.0	5.0	469.3	342.9	nd	11.0	5.1
–5	0	1	1	0	1	0	31.5	6.5	23.4	9.7	465.1	9.1	0.2	12.0	0.3
–6	0	1	1	0	1	1	32.2	9.0	14.2	1.6	318.5	82.2	nd	8.5	1.9
–7		1	1		1	0		8.2	20.5		394.1	29.6		9.0	1.7
–8		1	1		1	1		10.4	12.8		277.0	211.2		7.6	3.9
–9		1			1			11.2			258.2			10.7	
–10		1			1			12.6			126.1			5.0	
Mycotoxin[d]					22.4	94.2	32.5	0.4	3.0	0.7					

[a] S, spike number.
[b] Value of 1 represents presence of FHB damage or mycelia, value of 0 represents absence of FHB damage or mycelia on the grain.
[c] Nondetectable levels of DON.
[d] Estimated mycotoxin level of spike (composite value in ppm).

(1999) suggested that mycotoxins can be translocated up through the xylem vessels and phloem sieve tubes as well as down through the phloem sieve tubes of the rachis. The observed nondetectable or low mycotoxin levels of the kernels above the point of inoculation in PI 69251 spikes may be due to a xylem blockage during infection that restricts the upward translocation of mycotoxins. Savard et al (2000) suggested that *Fusarium* infection impedes flow through the xylem and phloem at the point of fungal entry into the rachis, preventing the delivery of water and nutrients to the upper spike. However, our measurement of mycotoxin accumulation in kernels above the inoculation point suggests that *Fusarium* does not block upward transport of mycotoxins. Histological studies focusing on the transport of water and nutrients through the xylem and phloem at the point of infection should help determine the physiological mechanism of resistance and whether resistance is genotype specific.

In both genotypes, some asymptomatic kernels had significant mycotoxin accumulation (1.6–45.7 ppm), while some FHB damaged kernels had nondetectable levels of mycotoxins. All Wheaton kernels but one had visual damage, and the single asymptomatic kernel had 45.7 ppm of DON and 2.2 ppm of 15-ADON (Table II). However, PI 69251had seven asymptomatic kernels in two spikes, and concentrations in these kernels were 1.2–9.7 ppm of DON. Only four out of these same kernels had detectable 15-ADON concentrations at 0.2–1.9 ppm. Asymptomatic kernels with mycotoxins did not exhibit *Fusarium* damage and none had visible mycelia growth (Table I). These results suggest that mycotoxins synthesized elsewhere are translocated in both directions from the point of inoculation within the spike. In addition, mycotoxins may be translocated to kernels that are not initially infected by the fungus, most likely later during grain fill. Four FHB damaged Wheaton kernels with or without visible mycelia did not have detectable DON or 15-ADON levels. In addition, most FHB damaged PI 69251 kernels from spikelets above the point of inoculation with or without mycelia had nondetectable levels of DON or 15-ADON. The presence of both symptomatic and asymptomatic kernels with nondetectable DON, may explain why FHB disease indices and bulk kernel DON content measurements are not always correlated (Birzele et al 2002). If one recognizes that asymptomatic kernels still can have high DON content, this may also be a reason that DON content in milled wheat can be high even if symptomatic kernels are removed as part of the milling process (Pate et al 2003).

FHB damaged kernels with visible mycelia had lower kernel weight and higher mycotoxin levels than damaged kernels without visible mycelia (Table III). The average concentration of mycotoxins in infected Wheaton kernels was approximately twice the amount present in PI 69251 kernels. A larger proportion of Wheaton kernels had DON, and all kernels that had DON also had 15-ADON. However, PI 69251 had a lower proportion of kernels with detectable DON. Some PI 69251 kernels that had DON did not have detectable levels of 15-ADON. Because the *Fusarium* strain and the environmental conditions under which the plants were maintained after inoculation were identical, the observed difference in mycotoxin concentrations in kernels of the two cultivars might be due to genotypic differences in resistance. Lemmens et al (2005) reported that FHB resistant wheat lines carrying *Fhb1* (Liu et al 2006) converted the externally applied DON to DON-3-*O*-glucoside, which is regarded as a detoxified compound. They also suggested that *Fhb1* (syn. *Qfhs.ndsu-3BS*) either encodes a DON-glucosyl-transferase or regulates the expression of such an enzyme. The low concentration of DON in kernels of PI 69251 may be the result of such a detoxification process. The DON concentration of kernels progressively farther away from the point of inoculation may provide information about the level of type II resistance of cultivar genotype. Therefore, the utility of using single kernel DON analyses is in enabling an assessment of both type II and type III resistance to FHB. The single kernel DON analysis by GC-MS is very expensive and the kernels must be destroyed in the process of analyzing them. Nondestructive near-infrared techniques have been employed to measure DON (Dowell et al 1999; Peiris et al 2010) and these may be of more practical use because they can help breeders evaluate more genotypes economically and rapidly, with the

TABLE II
***Fusarium* Head Blight Symptoms (FHB damage, mycelia), Kernel Weight, and Mycotoxin Levels of Three Wheaton Wheat Spikes (S1–S3)[a]**

Kernel Position	Kernel Property														
	FHB Damaged[b]			Mycelia[b]			Kernel Weight (mg)			DON (ppm)			15-ADON (ppm)		
	S1	S2	S3	S1	S2	S3	S1	S2	S3	S1	S2	S3	S1	S2	S3
10															
9															
8															
7	1			1			14.1			nd[c]			nd		
6	1			1			13.0			217.3			12.4		
5	1			1			9.2			342.3			11.7		
4	1	1	0	1	1	0	7.7	6.6	15.7	578.8	376.2	45.7	13.7	3.2	2.2
3	1	1	1	1	1	1	8.0	5.1	14.0	285.3	452.9	49.2	7.3	14.3	3.7
2	1	1	1	1	1	1	4.9	4.3	11.3	772.0	557.7	219.5	17.4	11.2	16.8
1			1			1			9.8			350.8			22.6
0															
–1	1	1	1	1	1	1	5.0	3.9	11.5	863.2	1008.4	573.5	13.9	14.9	17.6
–2	1	1	1	1	1	1	6.7	4.4	14.2	882.1	993.8	330.7	17.8	15.7	13.1
–3	1	1	1	1	1	1	7.6	4.8	15.9	846.7	626.0	240.9	14.2	11.1	6.8
–4	1	1	1	1	1	1	8.1	4.7	20.3	688.7	777.3	189.4	11.6	15.7	5.5
–5	1	1	1	1	1	1	9.8	7.4	23.1	579.4	557.8	116.3	10.9	11.9	1.9
–6	1	1	1	1	1	1	14.1	11.5	26.3	344.7	611.2	89.7	18.5	11.8	3.3
–7	1	1	1	1	1	1	27.0	15.6	26.5	nd	416.8	93.9	nd	11.1	1.5
–8	1	1	1	0	0	1	20.1	32.1	28.3	nd	nd	69.0	nd	nd	2.3
–9			1			1			27.7			53.8			1.4
–10			1			1			27.7			33.5			1.1
Mycotoxin[d]										317.3	396.3	140.3	8.2	7.9	5.2

[a] S, spike number.
[b] Value of 1 represents presence of FHB damage or mycelia, value of 0 represents absence of FHB damage or mycelia on the grain.
[c] Nondetectable levels of DON.
[d] Estimated mycotoxin level of spike (composite value in ppm).

TABLE III
Presence of FHB Damage (scab or mycelia) and Average Kernel Weight, Number of Kernels with Mycotoxins, and Average Mycotoxin Levels

Cultivar	Scab[a]	Mycelia[a]	Wt (mg)	Kernels	Kernels with DON	Avg DON (ppm)	Kernels with 15-ADON	Avg 15-ADON (ppm)
PI 69251	1	1	15.9	21	18	220.5	17	6.0
PI 69251	1	0	19.0	10	3	15.2	2	1.0
PI 69251	0	0	22.9	15	7	4.4	4	0.7
Wheaton	1	1	12.8	36	34	446.7	34	10.8
Wheaton	1	0	26.1	2	0	nd[b]	0	nd
Wheaton	0	0	15.7	1	1	45.7	1	2.2

[a] Value of 1 represents presence of scab or mycelia; value of 0 represents absence of scab or mycelia on the grain.
[b] Nondetectable levels of DON.

TABLE IV
Average Weight, DON, and 15-ADON Concentration in Single Kernels Between Two Cultivars and in Kernels Above, Below, Near or Far from the Inoculated Spikelet Within Each Cultivar[a]

Cultivar	Kernel Position	No.of Kernels	Weight (mg)	DON (ppm)	15-ADON (ppm)
PI 69251		45	18.8 ± 1.1	87.9 ± 23.8	2.3 ± 0.7
Wheaton		39	13.5 ± 1.3	390.6 ± 49.7	9.5 ± 1.0
Pr > |t|			0.0034	<0.0001	<0.0001
PI 69251	Above	25	19.8 ± 0.8	7.1 ± 4.4	0.2 ± 0.1
PI 69251	Below	20	17.5 ± 2.2	188.8 ± 44.1	5.0 ± 1.2
Pr > |t|			0.3608	0.0006	0.0012
Wheaton	Above	13	9.5 ± 1.1	326.7 ± 63.2	10.5 ± 1.9
Wheaton	Below	26	15.5 ± 1.8	422.6 ± 67.4	9.0 ± 1.3
Pr > |t|			0.0068	0.3071	0.5103
PI 69251	Near	23	20.1 ± 1.8	97.5 ± 39.2	2.3 ± 1.3
PI 69251	Far	22	17.5 ± 1.3	77.5 ± 27.0	2.3 ± 1.1
Pr > |t|			0.2551	0.6731	0.9392
Wheaton	Near	26	9.4 ± 1.0	511.7 ± 56.3	11.8 ± 1.1
Wheaton	Far	13	21.8 ± 2.0	148.4 ± 53.9	4.9 ± 1.7
Pr > |t|			<0.0001	<0.0001	0.0027

[a] Weight, DON, and 15-ADON values (± SE) are mean values of kernels from three spikes/cultivar. Two sample *T*-test probability values are given below for each pair of comparison.

added benefit of retaining the analyzed kernels for further analysis or generation advancement.

CONCLUSIONS

As a result of high mycotoxin concentrations in single kernels, the FHB susceptible wheat cultivar Wheaton exhibited significantly higher mycotoxin concentrations in kernels from inoculated spikes. The DON concentrations of kernels above and below the point of inoculation were high, but those farthest from the point of inoculation had significantly lower DON concentrations. In contrast, the moderately resistant accession PI 69251 had kernels with significantly lower mycotoxin levels and a lower proportion of kernels containing detectable mycotoxins, while kernels below the inoculated spikelet accumulated significantly higher levels of mycotoxins. The lower mycotoxin levels in PI 69251 may be due to an inhibition of the spread of the fungus within the spike as well as a detoxification of the DON produced by the fungus. Similar single kernel evaluations may provide valuable information about the relationship between visual FHB symptoms and DON accumulation in kernels. Analyses of bulked seed from a single spike for DON content will obscure how DON accumulates in the kernels as the spike develops and as fungal infection progresses in the spike. Consequently, analyzing single kernels for DON likely provides the best opportunity to distinguish between the various types of FHB resistance thought to function in a wheat plant.

LITERATURE CITED

Birzele, B., Meier, A., Hindorf, H., Kramer, J., and Dehne, H. W. 2002. Epidemiology of Fusarium infection and deoxynivalenol content in winter wheat in the Rhineland, Germany. Eur. J. Plant Pathol. 108:667-673.

Bottalico, A. 1998. Fusarium diseases of cereals: Species complex and related mycotoxins profiles in Europe. J. Plant Pathol. 80:85-103.

Bottalico, A., and Perrone, G. 2002. Toxigenic Fusarium species and mycotoxins associated with head blight in small-grain cereals in Europe. Eur. J. Plant Pathol. 108:611-624.

Carter, J. P., Rezanoor, H. N., Holden, D., Desjardins, A. E., Plattner, R. D., and Nicholson. P. 2002. Variation in pathogenicity associated with the genetic diversity of Fusarium graminearum. Eur. J. Plant Pathol. 108:573-583.

Dowell, F. E., Ram, M. S., and Seitz, L. M. 1999. Predicting scab, vomitoxin, and ergosterol in single wheat kernels using near-infrared spectroscopy. Cereal Chem. 76:573-576.

Gilbert, T., and Tekauz, A. 2000. Review: Recent developments in research on Fusarium head blight of wheat in Canada. Can. J. Plant Pathol. 22:1-8.

Hilton, A. J., Jenkinson, P., Hollins, T. W., and Parry, D. W. 1999. Relationship between cultivar height and severity of Fusarium ear blight in wheat. Plant Pathol. 48:202-2008.

Jiang, G., Dong, Y., Lewis, J. M., Siler, L., and Ward, R. W. 2006. Characterization of resistance to *Fusarium graminearum* in a recombinant inbred line population of wheat: Resistance to fungal spread, mycotoxin accumulation, and grain yield loss and trait relationships. Crop Sci. 46:2590-2597.

Kang, Z., and Buchenauer, H. 1999. Immunocytochemical localization of Fusarium toxins in infected wheat spikes by *Fusarium culmorum*. Physiol. Molec. Plant Pathol. 55:275-288.

Klahr, A., Zimmermann, G., Wenzel, G., and Mohler, V. 2007. Effects of environment, disease progress, plant height and heading date on the dtetction of QTLs for resistance to Fusarium head blight in an European winter wheat cross. Euphytica 157:17-28.

Lemmens, M., Scholz, U., Berthiller, F., Asta, C. D., Koutnik, A., Schuhmacher, R., Adam, G., Buestmayr, H., Mesterhazy, A., Krska, R., and Ruckenbauer, P. 2005. The ability to detoxify the mycotoxin deoxynivalenol colocalizes with a major quantitative trait locus for Fusarium head blight resistance in wheat. Molec. Plant Micro. Inter. 18:1318-1324.

Liu, S., Zhang, X., Pumphrey, M. O., Stack, R. W., Gill, B. S., and Anderson, J. A. 2006. Complex microcolinearity among wheat, rice and barley revealed by fine mapping of the genomic region harboring a major QTL for resistance to *Fusarium* head blight in wheat. Funct. Integrative Genom. 6:83-89.

Mackintosh, C. A., Garvin, D. F., Radmer, L. E., Heinen, S. J., and Muehlbauer, G. J. 2006. A model wheat cultivar for transformation to improve resistance to *Fusarium* head blight. Plant Cell Rep. 25:313-319.

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

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

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

Mirocha, C. J., Kolaczkowski, E., Xie, W., Yu, H., and Jelen, H. 1998. Analysis of deoxynivalenol and its derivatives (batch and single kernel) using gas chromatography/mass spectrometry. J. Agric. Food Chem. 46:1414-1418.

Oliver, R. E., Cai, X., Xu, S. S., Chen, X., and Stack, R. W. 2005. Wheat alien species derivatives: A novel source of resistance to Fusarium head blight in wheat. Crop Sci. 45:1353-1360.

Parry, D. W., Jenkinson, P., and McLeod, L. 1995. *Fusarium* ear blight (scab) in small cereals—A review. Plant Pathol. 44:207-238.

Pate, M. H., Sullins, R. D., and Faga, B. A. 2003. Impact of increased DON levels on the milling and baking industry. In: Proc. Nat. *Fusarium* Head Blight Forum. S. M. Canty, J. Lewis, L. Siler, and R. W. Ward, eds. Michigan State University: East Lansing, MI.

Peiris, K. H. S., Pumphrey, M. O., Dong, Y., Maghirang, E. B., Berzonsky, W., and Dowell, F. E. 2010. Near-Infrared spectroscopic method for identification of *Fusarium* head blight damage and prediction of deoxynivalenol in single wheat kernels. Cereal Chem. 87:511-517.

Pirgozliev, S. R., Edwards, S. G., Hare, M. C., and Jenkinson, P. 2003. Strategies for the control of Fusarium head blight in cereals. Eur. J. Plant Pathol. 109:731-742.

Savard, M. E., Sinha, R. C., Seamon, W. L., and Fedak, G. 2000. Sequential distribution of the mycotoxin deoxynivalenol in wheat spikes after inoculation with *Fusarium graminearum*. Can. J. Plant Pathol. 22:280-285.

Walker, S. L., Leath, S., Hagler, W. M., and Murphy, J. P. 2001. Variation among isolates of *Fusarium* graminearum associated with *Fusarium* Head Blight in North Carolina. Plant Dis. 85:404-410.

Zhang, X., and Jin, Y. 2003. Evaluation of spring wheat germplasm for *Fusarium* head blight resistance. In: Proc. Nat. *Fusarium* Head Blight Forum. S. M. Canty, J. Lewis, L. Siler, and R. W. Ward, eds. Michigan State University: East Lansing, MI.

Zhu, H., Gilchrist, L., Hayes, P., Kleinhofs, A., Kudrna, D., Liu, Z., Prom, L., Steffenson, B., Trojina, T., and Vivar, H. 1999. Does function follow form? Principal QTLs for *Fusarium* head blight (FHB) resistance are coincident with QTLs for inflorescence traits and plant height in a double haploid population of barley. Theor. Appl. Genet. 99:1221-1232.

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