Bread wheat cultivar PBW 343 carries residual additive resistance against virulent stripe rust pathotype

Article *in* Journal of Crop Improvement · February 2017

DOI: 10.1080/15427528.2016.1263262

CITATION
1

READS
444

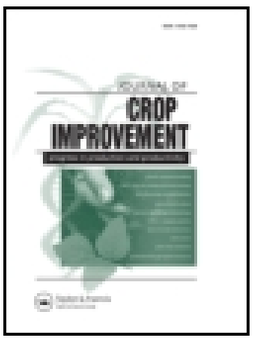
4 authors:

Rupinder Pal Singh
Punjab Agricultural University Seed Farm, Naraingarh
36 PUBLICATIONS **25** CITATIONS
SEE PROFILE

Puja Srivastava
Punjab Agricultural University
44 PUBLICATIONS **37** CITATIONS
SEE PROFILE

Achla Sharma
Punjab Agricultural University
62 PUBLICATIONS **166** CITATIONS
SEE PROFILE

Navtej Singh Bains
Punjab Agricultural University
73 PUBLICATIONS **410** CITATIONS
SEE PROFILE



Journal of Crop Improvement

ISSN: 1542-7528 (Print) 1542-7536 (Online) Journal homepage: http://www.tandfonline.com/loi/wcim20

Bread wheat cultivar PBW 343 carries residual additive resistance against virulent stripe rust pathotype

Rupinder Pal Singh, Puja Srivastava, Achla Sharma & N S Bains

To cite this article: Rupinder Pal Singh, Puja Srivastava, Achla Sharma & N S Bains (2017): Bread wheat cultivar PBW 343 carries residual additive resistance against virulent stripe rust pathotype, Journal of Crop Improvement, DOI: 10.1080/15427528.2016.1263262

To link to this article: http://dx.doi.org/10.1080/15427528.2016.1263262

Published online: 17 Feb 2017.

JOURNAL OF CROP IMPROVEMENT
http://dx.doi.org/10.1080/15427528.2016.1263262

Taylor & Francis
Taylor & Francis Group

Bread wheat cultivar PBW 343 carries residual additive resistance against virulent stripe rust pathotype

Rupinder Pal Singh, Puja Srivastava, Achla Sharma, and N S Bains

Department of Plant Breeding & Genetics, Punjab Agricultural University, Ludhiana, Punjab 141004, India

ABSTRACT

With continuous outbreaks of stripe rust (*Puccinia striiformis* f. sp. *tritici*) epidemics and rapid breakdown of deployed resistance in bread wheat (*Triticum aestivum* L.) cultivars in North West Plains Zone (NWPZ) in India warrant knowledge and deployment of new and durable sources of resistance to stripe rust. Bread wheat cultivar PBW 343, until recently the most widely cultivated wheat variety in India, is now highly susceptible to stripe rust (score 9 on a 1–9 scale), whereas PBW 621 (score 5.05–5.65) and HD 2967 (score 5.40–6.20) show low levels of resistance. We conducted an experiment, spanning three crop seasons (2013–2014 to 2015–2016), in which parental lines, F_1 and F_2 populations, F_3 and F_4 families from two bread wheat crosses, PBW 621/PBW 343 and HD 2967/PBW 343 were generated and evaluated for stripe rust resistance against a virulent pathotype. While the F_1 revealed partial dominance, the segregation pattern for stripe rust resistance in F_2 and F_3 showed transgressive segregation for resistance in both crosses. Chi-square analysis indicated that resistant segregants possessed two genes, one contributed by PBW 621 or HD 2967 (depending on the cross) and the other, unexpectedly but obviously, came from the most susceptible cultivar, PBW 343. Possible genetic mechanisms for this residual resistance and implications for breeding programs are discussed.

ARTICLE HISTORY
Received 23 August 2016
Accepted 17 November 2016

KEYWORDS
adult plant resistance; disease resistance; genetic analysis; *Puccinia striiformis* f. sp. *tritici* and *Triticum aestivum*

Introduction

Stripe rust, caused by *Puccinia striiformis* f. sp. *tritici* (*Pst*), is presently considered one of the most important diseases of wheat throughout the world (Chen et al. 2014). The principal outcome of stripe rust epidemics is reduced grain yield and quality. The extent of loss primarily depends upon the resistance level of the cultivars (Beard et al. 2007). Despite increased dependence on fungicides in recent years, resistance breeding is the most preferred disease-management strategy (McDonald and Linde 2002). More than 70 stripe rust- resistance genes, in addition to many uncatalogued major and minor genes in wheat breeding programs, have been identified in wheat (McIntosh et al. 2014). The pathogen continues to evolve with respect to

CONTACT Rupinder Pal Singh rupinder03.pau@gmail.com PAU Regional Research Station, Kapurthala, Punjab 144601, India.

virulence and aggressiveness, often rendering resistant varieties susceptible. Consequently, emphasis has been placed on the identification and deployment of new sources of resistance. Various strategies aimed at enhancing durability of resistance have been explored (Chen 2005). Resistance attributable to minor genes, in the absence of major effective genes, has been shown to be a good source of durable resistance, provided a sufficient number of such genes can be accumulated to confer a high level of resistance (Singh, Huerta-Espino, and Rajaram 2000).

Wheat cultivar PBW 343, an Attila sib (ND/VG9144//KAL/BB/3/YACO/4/VEE#5), is a selection made at Punjab Agricultural University, Ludhiana, Punjab, India, from a set of lines called "Veery wheat derivatives" developed at CIMMYT, Mexico, based on an initial round of spring wheat × winter wheat hybridization. After its release in the North West Plain Zone (NWPZ) of India in 1995, PBW 343 emerged as a mega cultivar. By 2002–03, PBW 343 occupied more than 90% of the wheat-growing area in Punjab and about 7 million hectares across the Indo-Gangetic Plains of India (http://libcatalog.cimmyt.org/download/cim/94397.pdf). Apart from carrying multiple disease resistance, the 1B/1 R wheat-rye translocation harbored by PBW 343 also possibly enhanced its adaptation zone and productivity (Rajaram et al. 1983). By virtue of stripe rust-resistance gene *Yr27*, probably derived from Selkirk parentage, PBW 343 had withstood the spread of *Yr9* virulence (pathogenicity of virulent race of stripe rust pathogen resulting in breakdown of *Yr9* resistance), to which many other Veery derivatives succumbed (McDonald et al. 2004; McIntosh et al. 2003). The spread and increasing adaptation of *Yr27* virulence (it refers to development of virulence in pathogen against *Yr27* gene in wheat) to local conditions became evident in the form of stripe rust assuming damaging proportions in 2007 across an extensive wheat belt adjoining the Himalayan foothills (Prashar et al. 2007). The emergence of this well-adapted *Yr27* virulence by the pathogen was followed by a step-wise escalation in aggressiveness of the pathotype, as evidenced by the rapid decline in resistance in a set of post-*Yr27* gene wheat releases that did not possess major resistance genes (e.g., DBW 17, PBW 550, PBW 621, and HD 2967).

In the backdrop of these evolutionary changes in the pathogen, newer wheat cultivars, viz., PBW 621 (KAUZ//ALTAR84/AOS/3/MILAN/KAUZ/4/HUITES) and HD 2967 (ALD/COC//URES/3/HD2160M/HD2278), which were released in 2011 for timely sowing under irrigated conditions of NWPZ of India, showed progressive loss of resistance to stripe rust. The segregants from the crosses of PBW 343 with PBW 621 and HD 2967 were found to possess a significant level of resistance. Though they were, initially, perceived to possess significantly high levels of stripe rust resistance but showed progressive loss of resistance over time. In this study, we analysed the segregation pattern of the progenies of these two crosses and discuss the implications for breeding for resistance to stripe rust.

Materials and methods

The experimental materials comprised F_1 and F_2 populations and F_3 and F_4 families from the followsing two crosses: PBW 621 × PBW 343 and HD 2967 × PBW 343. The F_1 and F_2 populations were screened in the 2013–2014 crop season, F_3 families in 2014–2015 and resistant and susceptible F_4 bulks in 2015–2016. A pathotype strain representing aggressive *Yr27* gene virulence was collected in the 2013–2014 season from a farmer's field in the foothill area of Anandpur Sahib (31.23°N, 76.51°E) in Punjab, India, and multiplied on PBW343 for future inoculations. The pathotype (hereafter referred to as *Pst* pt. AS) showed a virulence on PBW 343 isogenic materials carrying *Yr5, Yr10*, and *Yr15* genes; moderate virulence on *Yr17, Yr40*, and *Yr70* genes; and complete virulence against materials carrying *Yr9* and *Yr27* genes. Pathotype *Pst* pt. AS was used to test parental lines at the seedling stage. Fully expanded primary leaves (7–9 days old) were inoculated with urediniospores suspended in a mixture of 30:70 mineral oil and petroleum ether. Plants were then transferred to a dew chamber set at 10°C, with 16 h light and 8 h dark photoperiod for 48 hours, following which they were transferred to the greenhouse set at 14–16°C temperature. Stripe rust reaction assessment at the seedling stage was done 14 days after inoculation as per Nayar, Prashar, and Bhardwaj (1997). Infection types (ITs) for seedling response were recorded on a 0–4 scale, where 0 score represents an immune response and a score of 4 represents a highly susceptible response (Roelfs 1984).

For adult-plant reaction, parental lines were space-planted during the 2013–2014 crop season in rows that were two meters long and spaced 30 cm apart. Also, during this season, single plants of F_2 populations were space-planted; four rows of parental checks were planted after every 25 rows. In the following season (2014–2015), F_3 families (each family represented by 40–50 plants) were screened. The parental controls were planted after every 25 entries. Single-plant progenies from homozygous resistant and susceptible F_3 lines, along with parents, were screened in the 2015–2016 crop season. For field inoculations, urediniospores of pathotype *Pst* pt. AS, suspended in 10 L water with two drops of Tween 20, were sprayed during end December to early January using an ultra-low volume applicator on clear evenings with good expectation of dew to ensure good disease infection.

Field assessment of adult-plant stripe-rust reaction was based on a continuous 1–9 scale (Bariana et al. 2007). Three successive disease observations were recorded on parental lines at 10-day intervals, starting in the first week of February. The disease score on other entries was recorded when the susceptible check (PBW 343) had reached a score of 8 on a 1–9 scale, which corresponded to 80–90% leaf area affected (as observations were recorded on successive leaves, there was a possibility of increase as well as decrease in the level of disease).

Statistical analysis

Chi-square tests were used to determine the goodness of fit of the observed numbers of plants or lines to the predicted segregation ratios of F_2 and F_3 populations to establish the number of stripe rust-resistance genes and mode of inheritance. The Chi-square value was calculated using the following expression:

$$X^2 = \sum \frac{(\text{observed} - \text{expected})^2}{\text{expected}}$$

Results

When inoculated with pathotype *Pst* pt. AS, the seedling IT of both PBW 621 and HD2967 was 3, whereas it was 4 for PBW 343. Thus, all three parental lines showed seedling susceptibility. In adult-plant tests in the field, PBW 343 had a final average score of 9, whereas PBW 621 and HD 2967 had final scores varying from 5.05 to 5.65 and 5.40 to 6.20, respectively (Table 1). During the 2013–2014 season, 76 F_1 plants of PBW 621 × PBW 343 and 82 F_1 plants of HD 2967 × PBW 343 were moderately resistant, with an average score of 5.46 and 5.53, respectively. The segregation pattern for stripe rust resistance in the F_2, F_3, and F_4 generations from the two crosses (Tables 2 and 3) did not show qualitative distinct classes, which suggested the presence of multiple genetic factors. F_2 plants showed higher resistance (scores of 1 to 4) than the resistant parent in both the crosses. These were combined into one transgressive category. The susceptible parent (PBW 343) displayed the highest score of 9 on the 1–9 scale. Demarcating extreme categories and combining intermediate disease categories, the F_2 in both crosses tended to have a symmetric distribution. The number of susceptible F_3 families was higher than the number of susceptible F_2 plants. Presence of a genetic framework at the base of the observed classes was evident from the largely true-breeding nature of both resistant and susceptible progenies. Twenty F_4 families from the PBW 621 × PBW 343 cross derived from resistant F_3 families were homozygous resistant and two showed segregation, whereas all F_4 families derived from susceptible F_3 families were homozygous susceptible. Twenty-two F_4 families from the HD 2967 × PBW 343 cross were resistant and one showed segregation, whereas 29 F_4 families from susceptible F_3 families were susceptible and one showed segregation (F_4 family was considered segregating if at least one of the five single plant progenies from an F_3 line showed segregation).

The obtained quasi-quantitative segregation was not amenable to a simple Mendelian genetic analysis. Nevertheless, the relative proportion of the resistant categories in both crosses followed a simple and similar pattern. In the case of

Table 1. Average disease reaction of parental lines and their F_1 at the adult plant stage during three crop seasons.

	2013–14 Season				2014–15 Season				2015–16 Season			
Parental lines and hybrids	Score I†	Score II‡	Final score§	Average score	Score I	Score II	Final score	Average score	Score I	Score II	Final score	Average score
PBW 621	3.75	4.20	5.05	4.95	4.15	6.05	5.55	5.45	4.45	6.10	5.65	5.60
HD 2967	3.70	4.55	5.40	5.30	4.55	5.65	6.15	5.60	4.80	5.75	6.20	5.90
PBW 343	8.60	8.90	9.00	8.90	8.80	9.00	9.00	9.00	8.80	9.00	9.00	9.00
PBW 621 × PBW 343	4.07	4.84	5.46	5.32	-	-	-	-	-	-	-	-
HD 2967 × PBW 343	4.08	4.89	5.53	4.96	-	-	-	-	-	-	-	-

†Score I was taken when disease severity on PBW 343 reached 70%.
‡Score II was taken when disease severity on PBW 343 reached 80%.
§Final Score was taken when PBW 343 lost its foliage because of 100% disease.

Table 2. Segregation of F_2, F_3 and F_4 generations of PBW 621 × PBW 343 cross.

				F_4 families	
Phenotypic classes	Disease score	F_2 plants	F_3 families	Homozygous	Segregating
Resistant	1–4	28	22	20	2
Intermediate or Segregating	5–7	332	331	-	-
Susceptible	8–9	24	31	31	0

Table 3. Segregation of F_2, F_3 and F_4 generations of HD 2967 × PBW 343 cross.

				F_4 families	
Phenotypic classes	Disease score	F_2 plants	F_3 families	Homozygous	Segregating
Resistant	1–4	27	23	22	1
Intermediate or Segregating	5–7	351	344	-	-
Susceptible	8–9	20	31	29	2

PBW 621 × PBW 343 cross, 20 F_2 plants out of 384 were authenticated (at F_3 and F_4 stages) to be homozygous resistant. For HD 2967 × PBW 343 cross, 22 F_2 plants out of 398 were confirmed to be homozygous resistant. As the number of plants in this category fit the 1:15 expected ratio for PBW 621 × PBW 343 (Chi-square value of 0.710, probability 0.399; df = 1) and for HD 2967 × PBW 343 (Chi-square value of 0.352, probability of 0.552; df = 1), the plants with higher levels of resistance than the resistant parents were hypothesized to carry two genes, one from the resistant parent (PBW 621 or HD 2967, depending on the cross) and one from susceptible parent PBW 343, each.

Discussion

The results indicated that probably minor genes with additive effects conferred stripe rust resistance in highly resistant lines, as widely reported additivity of minor gene conferred stripe rust resistance (Parlevliet 2002) as well as through susceptible parents contributing to resistance in an additive manner (Dutta et al. 2008). The post-PBW 343 stripe rust-resistance breakdown scenario in the NWPZ of India was characterized by a phase of run-away evolution (Chandler, Ofria, and Dworkin 2013), as a result of whose aggressiveness, even resistant cultivars such as PBW 621 and HD 2967 had succumbed to the disease. Interestingly, these lines and the more comprehensively defeated PBW 343 harbored resistance genes. The completely ineffective resistance gene (the resistance conferred by the gene earlier was broken down due to virulence of the pathotype) present in PBW 343 is able to contribute significantly when it is combined with genes from PBW 621 as well as HD 2967 (additive-epistatic behavior of minor genes when together). We hypothesized that even when an aggressive race overcomes resistance in a variety and makes it almost completely susceptible, it seems to breakdown only a subset of diverse quantitatively determined resistance mechanisms. As a result, such

susceptible lines have the potential of contributing resistance when recombination provides the opportunity for a new assemblage of resistance components. Are the surviving resistance components more durable than the ones that were overcome by the pathogen? Another question is whether this resistance is governed by the defeated major genes, the so-called ghost resistance hypothesis. Nass et al. (1981) used near-isogenic winter wheat lines to attempt to determine the effect of individual genes for resistance to *Erysiphe graminis* (powdery mildew pathogen). Compared to the susceptible cultivar "Chancellor," near- isogenic lines (NILs) of Chancellor carrying mildew resistance genes *Pm3c, Pm4*, or *MA* had fewer sporulating colonies and lower spore production per lesion when inoculated with *E. graminis* isolate 144, even though this isolate was considered virulent against these genes. They concluded that these effects on isolate 144 were residual effects of *Pm* 3c, *Pm4*, and *MA*.

The genes that continued to operate in current study were more effective when combined with other genes (from the other parent) in the segregating material. In the encountered resistance, "defeated" or "ghost" resistance finds a mention in the literature. For example, Li et al. (1999) demonstrated that a defeated rice resistance gene acted as a quantitative trait locus (QTL) against a virulent strain of *Xanthomonas oryzae* pv. *oryzae*. There are several compelling lines of evidence which indicate that allelic variants at major resistance genes account for a proportion of quantitative disease resistance in plants (Young 1996). Co-localization of residual minor genes and major resistance genes has been noted in several species, including rice (Xiao et al. 2007), maize (Wang et al. 1994), and potato (Gebhardt and Valkonen 2001) and reported to be based on multiple genes with additive effects. Thus, typical behavior of operative resistance genes in PBW 621/PBW 343 and HD 2967/PBW 343 crosses was clearly visible, albeit with only two such genes in each cross. However, it remains to be seen if these genes provide durable resistance by exposing the gene pyramided lines to the evolving pathotypes. The classical example of "Cappelle-Desprez" as a cultivar with durable resistance to stripe rust was recently revisited in the context of South African *Pst* races (Agenbag et al. 2012). Several QTL for resistance from cultivar "Cappelle-Desprez" were evident in crosses with the susceptible cultivar Palmiet. In parallel to the findings of this study, susceptible cultivar Palmiet was shown to carry a resistance QTL (like PBW 343 here).

Conclusion

The resistant segregants from each cross among wheat varieties (PBW 621/PBW 343 and HD 2967/PBW 343) were hypothesized to carry two genes of resistance against stripe rust disease of wheat, one each from the resistant and susceptible parent. The results indicated that probably these minor genes conferred stripe rust resistance in the highly resistant lines obtained, as indicative of the additivity of minor genes.

Author Contribution Statement

Rupinder Pal Singh: He has conducted the entire study during the period of his PhD research curriculum and has himself done all the work listed in this article and has written the manuscript as submitted to your esteemed journal.

Puja Srivastava: She, being an integral team member has contributed towards guidance in genetic analysis and writing the manuscript.

Achla Sharma: Being a geneticist, she has played a key and instrumental role in rust epidemiology characterization and field inoculations and genetic analysis of the reactions so obtained during the whole research program.

N. S. Bains: Being the major guide in the research, right from the gestation of the concept and throughout conduct of the research, he has played a pivotal role in directing the entire study towards its goal and also has been instrumental in giving valuable inputs for manuscript writing.

Conflict of Interest

The authors declare that they have no conflict of interest.

Funding

The senior author acknowledges the grant received from the Department of Science and Technology, Govt. of India, New Delhi, through INSPIRE Ph.D. FELLOWSHIP PROGRAMME to pursue the research project.

References

Agenbag G. M., C. M. Bender, Z. A. Pretorius, R. Prins, and L. A. Boyd. 2012. Identification of adult plant resistance to stripe rust in wheat cultivar Cappelle-Desprez. *Theor Appl Genet* 125:109–20.

Bariana, H. S., H. Miah, G. N. Brown, N. Willey, and A. Lehmensiek. 2007. Molecular mapping of durable rust resistance in wheat and its implication in breeding. In Proceedings of the 7th International Wheat Conference, Mar del Plata, Argentina, 29 November–2 December, 2005, 723–28.

Beard, C., K. Jayasena, G. Thomas, and R. Loughman. 2007. Managing stripe rust and leaf rust of wheat. Farmnote. 43/2005. Deptt. Agric. Govt. West Aust.

Chandler, C. H., C. Ofria, and I. Dworkin. 2013. Runaway sexual selection leads to good genes. *Evolution* 67:110–19. doi:10.1111/evo.2013.67.issue-1.

Chen, W., C. Wellings, X. Chen, Z. Kang, and T. Liu. 2014. Wheat stripe (yellow) rust caused by *Puccinia striiformis* f.sp. *tritici*. *Molecular Plant Pathology* 15:433–46. doi:10.1111/mpp.2014.15.issue-5.

Chen, X. M. 2005. Epidemiology and control of stripe rust on wheat. *Canadian Journal of Plant Pathology* 27:314–37. doi:10.1080/07060660509507230.

Dutta, D., S. K. Nayar, S. C. Bhardwaj, M. Prashar, and K. Subodh. 2008. Detection and inheritance of leaf rust resistance in common wheat lines Agra Local and IWP94. *Euphytica* 159:343–51. doi:10.1007/s10681-007-9522-3.

Gebhardt, C., and J. P. T. Valkonen. 2001. Organization of genes controlling disease resistance in the potato genome. *Annual Reviews of Phytopathology* 39:79–102. http://libcatalog.cimmyt.org/download/cim/94397.pdf (accessed January 22, 2016).

Li Z. K., L. J. Luo, H. W. Mei, A. H. Paterson, X. H. Zhao, D. B. Zhong, Y. P. Wang, X. Q. Yu, L. Zhu, R. Tabien, J. W. Stansel, and C. S. Ying. 1999. A "defeated" rice resistance gene acts as a QTL against a virulent strain of Xanthomonas oryzae pv. oryzae. *Mol Genet* 261:58–63.

McDonald, B. A., and C. Linde. 2002. The population genetics of plant pathogens and breeding strategies for durable resistance. *Euphytica* 124:163–80. doi:10.1023/A:1015678432355.

McDonald, D. B., R. A. McIntosh, C. R. Wellings, R. P. Singh, and C. J. Nelson. 2004. Cytogenetical studies in wheat XIX. Location and linkage studies on gene *Yr27* for resistance to stripe (yellow) rust. *Euphytica* 136:239–48. doi:10.1023/B:EUPH.0000032709.59324.45.

McIntosh, R. A., K. M. Devos, J. Dubcovsky, C. F. Morris, and W. J. Rogers. 2014. Catalogue of gene symbols for wheat: 2014 supplement. In *Proceedings of 12th International Wheat Genetics Symposium*, Yokohama, Japan, September 8–13, 2013.

McIntosh, R. A., Y. Yamazaki, K. M. Devos, J. Dubcovsky, W. J. Rogers, and R. Appels. 2003. Catalogue of gene symbols for wheat. In *Proc of 10th International Wheat Genetics Symposium*, ed. N. E. Pogna, N. Romano, E. A. Pogna, and G. Galterio, vol. 4, 8. Rome, Italy: Instituto Sperimentale per la Cerealcoltura.

Nass, H. A., W. L. Pedersen, D. R. McKenzie, and R. R. Nelson. 1981. The residual effects of some "defeated" powdery mildew resistance genes in isolines of winter wheat. *Phytopathology* 71:1315–18.

Nayar, S. K., M. Prashar, and S. C. Bhardwaj. 1997. *Manual of current techniques in wheat rusts*. Flowerdale, Shimla 171002, India: Research Bulletin No. 2, Directorate of Wheat Research, Regional Station.

Parlevliet, J. E. 2002. Durability of resistance against fungal, bacterial and viral pathogens; present situation. *Euphytica* 124:147–56. doi:10.1023/A:1015601731446.

Prashar, M., S. C. Bhardwaj, S. K. Jain, and D. Datta. 2007. Pathotypic evolution in *Puccinia striiformis* in India during 1995–2004. *Australian Journal of Agricultural Research* 58:602–04. doi:10.1071/AR07126.

Rajaram, S., C. H. E. Mann, G. Qrtiz-Ferrara, and A. Mujeeb-Kazi. 1983. Adaptation, stability and high yield potential of certain 1B/1R CIMMYT wheats. In *Proc. 6 Int. Wheat. Genet. Symp*, ed. S. Sakamoto, 613–21. Kyoto, Japan: Kyoto University.

Roelfs, A. P. 1984. Race specificity and methods of study. In *The cereal rusts vol. I; Origins, specificity, structure and physiology*, eds. A. P. Roelfs, and W. R. Bushnell, 131–64. CIMMYT, Mexico. Orlando: Academic Press.

Singh, R. P., J. Huerta-Espino, and S. Rajaram. 2000. Achieving near-immunity to leaf and stripe rusts in wheat combining slow rust resistance genes. *Acta Phytopathalogica Et Entomologica Hungarica* 35:133–39.

Wang, G. L., D. J. Mackill, J. M. Bonman, S. R. McCouch, M. C. Champoux, and R. J. Nelson. 1994. RFLP mapping of genes conferring complete and partial resistance to blast in a durably resistant rice cultivar. *Genetics* 136:1421–34.

Xiao, W., J. Zhao, S. Fan, L. Li, J. Dai, and M. Xu. 2007. Mapping of genome-wide resistance gene analogs (RGAs) in maize (Zea mays). *Theoretical and Applied Genetics* 115:501–08. doi:10.1007/s00122-007-0583-4.

Young, N. D. 1996. QTL mapping and quantitative disease resistance in plants. *Annual Review of Phytopathology* 34:479–501. doi:10.1146/annurev.phyto.34.1.479.