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Genetic Variation for Resistance to Russian Wheat Aphid in F_2-Derived Families of Wheat (*Triticum aestivum* L.)

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ABSTRACT

The Russian wheat aphid (RWA), *Diuraphis noxia* (Mordvilko), is a significant insect pest of wheat worldwide. The objective of this study was to assess the genetic variation within and between F2-derived families for reaction to RWA using F3 and F4 families originating from individual F2 plants of a cross between the susceptible line (synthetic hexaploid-11) and the resistance cultivar ('Halt'). The RWA damage of individual plants within each family was measured using different procedures. Their reaction types were combined into a single data for each individual family (derived from an individual F2 plants) and subjected to statistical analysis. Results indicated that the genetic variation between F2-derived families is greater than within F2-derived families for RWA resistance. Broad-sense heritability of RWA resistance, calculated by partitioning phenotypic variation into genetic and environmental components, was 73.2%. A narrow-sense heritability estimate of 30% was obtained for the RWA resistance in the 'Halt' × synthetic hexaploid-11 cross using parent-offspring (F3: F4) regression procedure.

Keywords: Genetic variation, Heritability, Insect resistance, Russian wheat aphid, Wheat

INTRODUCTION

The Russian wheat aphid (RWA), *Diuraphis noxia* (Mordvilko) (Homoptera: Aphididae), is one of the most destructive pests in wheat (*Triticum aestivum* L.) and other small-grain cereals in several areas of the world (Archer and Bynum, 1992). Since its detection in Texas in 1986, RWA has become a major economic pest of wheat and barley (*Hordeum vulgare* L.) in the western United States (Nkongolo *et al*., 1991). The pest causes leaf rolling and streaking, head trapping, and even death in heavily infested plants (Quick *et al*., 1991). Direct economic losses in small grains in the United States during 1985 to 1995 that incurred as a result of reduced yield and increased production costs were estimated to be about $500 million (Webster *et al*., 2000).

Developing resistant cultivars is the most economic and environmentally safe method of eliminating the use of insecticides and reducing crop losses. Sources of resistance have been reported in wheat and other related species (Du Toit, 1987; Nkongolo *et al*., 1991; Quick *et al*., 1991; Porter *et al*., 1993). Several dominant genes conferring resistance to RWA have been identified in wheat, namely *Dn1*, *Dn2* (Du Toit, 1987), *Dn4* (Nkongolo *et al*., 1991), *Dn5* (Liu *et al*., 2002), *Dn6* and *Dn7* (Marais and Du Toit, 1993), *Dn8*, *Dn9* and *Dnx* (Liu *et al*., 2002).

Host resistance to the RWA is based on antixenosis, antibiosis and tolerance. Castro *et*

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al. (2001) identified the wheat chromosomes involved in antibiosis, antixenosis and tolerance resistance to RWA, in 'Hope' cultivar and a 'synthetic hexaploid' wheat, using intervarietal chromosome substitution lines. The *Dn4* gene was located on chromosome 1DS and originated from a Russian bread wheat accession PI 372129 (Nkongolo *et al*., 1991). The microsatellite (SSR) markers and a morphological marker linked to the RWA resistance gene (Dn4) were identified by Arzani *et al*. (2004). 'Halt', the first RWA-resistant U.S. wheat cultivar expresses *Dn4* resistance that is effective in controlling RWA (Quick *et al*., 1996). In the United States, only one biotype existed following its first detection in 1986 until the spring 2003, when a new biotype appeared in Colorado (Haley *et al*., 2004). The new biotype (biotype B) was virulent to *Dn4* and eight other known resistance genes in wheat. However, the old biotype is still widespread, and was observed to occur on the same plants as the new biotype. Other U.S. cultivars containing *Dn1* or *Dn2* are also being bred in Idaho (Souza *et al*., 1997). Hence, durable resistance to RWA deploying minor genes is also inevitable. It has been reported that resistance in '94M370' (possessing *Dn7*) is based on antixenosis, whereas resistance to 'Halt' (possessing *Dn4*) and PI262660 (possessing *Dn2*) is based on tolerance (Smith *et al*., 1992; Anderson *et al*., 2003). Lage *et al*. (2004) examined the antixenosis reaction of several synthetic hexaploid wheats and demonstrated that the synthetic hexaploid wheats attracted significantly fewer aphids than Seri M82, and pubescent leaves may contribute to the antixenosis properties in these synthetic hexaploids.

Knowledge about the inheritance of this resistance would be beneficial in planning breeding programs. The amount of genetic variation within and between families of lines has a significant bearing on when and how selection should be practiced. There is no report of the extent of within- and between-family variation for RWA resistance. The objective of this research was to assess the genetic variation within and between F_2-derived F_3 and F_4 families (originating from a cross between the resistant cultivar 'Halt' and the susceptible line, synthetic hexaploid-11) for reaction to RWA.

MATERIALS AND METHODS

Plant Materials

One hundred and ten F_2–derived F_3 ($F_{2:3}$) families originating from a cross between the susceptible line (synthetic hexaploid-11) and the resistant cultivar ('Halt') were used in this study. Resistance in PI 372129 (*Dn4*) has been reported as being controlled by a single dominant gene. Halt has the RWA resistant gene, *Dn4*, which derived from PI 372129. Halt was selected from the crosses Sumner/CO8200, F1//PI 372129, F1/3/' TAM 107' and released to Colorado seed producers in 1994 (Quick *et al*., 1996). F_2-derived F_4 families were evaluated for RWA reaction during February-March 2002 and the data were only used as progeny for estimating the narrow-sense heritability in a parent-offspring (F_3: F_4) regression analysis. The synthetic hexaploid-11 was obtained from CIMMYT, Mexico (*T. turgidum*-D67.2/P66.270//*Aegilops tauschii*).

F_4 seeds harvested from F_3 plants which were used for RWA assessments were employed as $F_{2:4}$ families. These self-pollinated progenies of each individual F_2 ($F_{2:3}$ and $F_{2:4}$ families) were planted in a greenhouse infested with RWA (insectarium) at the Soil and Crop Science Department, Colorado State University and used for RWA phenotypic assessments.

RWA Resistance Evaluation

Seedling reaction to the RWA was studied during two seasons (November –December 2000 and 2001) using $F_{2:3}$ families. The $F_{2:3}$ families had two replicates within each year and each replicate had 15 plants. Parents, F_3 families and check cultivars ('Carson', 'TAM 107' (susceptible) and 'Halt' (resis-

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tance)) were planted in a 3:2:1:1 (v/v/v/v) soil mix consisting of soil, peat moss, vermiculite, and perlite in a 25 by 50 cm flat trays (12 rows). Each row contained 15 plants with a row spacing of 4-cm. Row number six of each tray (in the middle) was divided into three parts and allocated to three check cultivars. A randomized complete block design with four replications (two replicates per year) was used. The trays were uniformly watered and fertilized throughout the experiment. The seedling were grown in the greenhouse under a 16 hour photoperiod at 25±2°C.

The original RWA biotype (biotype A) was used for screening. Seedlings at the one-leaf stage were infested with five RWAs per plant. The aphids were obtained from a laboratory colony reared on a mixture of 'Carson', 'TAM107' and 'Oslo' wheat cultivars and initiated from aphids collected from volunteer common wheat near Briggsdale, Colorado. Late instars were separated from early instars using sieves with different mesh sizes. Noninfested leaves were cut into ≅5 mm sections. These leaf sections were mixed with collected late instars. A section containing about five RWAs was placed next to the leaf base of each seedling. As the leaf sections desiccated, the aphids moved to the growing portions of the plants. Plants were checked daily to ensure that the insects were evenly distributed over all plants in the flats.

RWA damage (leaf rolling and leaf chlorosis) was scored according to Nkongolo *et al*. (1991). Seedling damage was measured on a 1 to 9 scale, 1 denoting healthy plants and 9 denoting dying or dead plants. Symptom expression in susceptible and resistant seedlings was recorded when the susceptible check showed severe leaf rolling and chlorotic streaking (scores of 8-9 in 21-28 d). Seedlings with chlorotic spots caused by aphid feeding and without leaf rolling (scores 1-4), and seedlings with chlorotic streaking and leaf rolling (scores 5-9) were recorded as resistant and susceptible, respectively. A chi-square test for goodness of fit to phenotypic segregation ration of 3:1 (resistant: susceptible) were used to determine the mode of inheritance of the RWA resistance gene. The damage to individual plants within a family was also quantified using percentage of infection and reaction status of highly resistance (VR), resistance (R), moderately resistance (MR), moderately susceptible (MS), susceptible (S), and highly susceptible (VS).

Genetic Variation and Heritabilities

The percentage of plants showing leaf chlorosis and leaf rolling (PCR %) was also recorded. Plant growth rates and the damage (PGD) caused to the leaves, including the presence of tightly rolled leaves, white streaking and chlorotic lesions were recorded (Smith *et al*., 1991). The PGD values of 0.0, 0.2, 0.4, 0.6, 0.8 and 1.0 (correspond to 0 to 5 scores of Smith *et al*., 1991) corresponding to the reaction types of VR, R, MR, MS, S and VS were used, respectively.

These reaction types were combined according to Smith *et al*. (1991) [RT% = (LD × PCR% × PGD)/9] into a single quantitative data set for each family and subjected to statistical analysis. An arcsin $\sqrt{x}$ transformation was applied to the data with binomial distribution where the percentages covered a wide range of values (Steel and Torrie, 1980). The homogeneity of the variances between years (2000 vs. 2001) was tested and indicated to be homogenous (Steel and Torrie, 1980). Analyses of variances between and within families were conducted using PROC GLM (SAS, 1997). Broad-sense heritability of RWA resistance was calculated by partitioning phenotypic variation into genetic and environmental components (Allard, 1999). Genetic variance was estimated by $[(MS_G - MS_E)/r]$ where MS_G = family's mean square, MS_E = experimental error's mean square and r = replication number. Narrow-sense heritability was estimated using a parent-offspring (F_3: F_4) regression procedure (Allard, 1999).

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Table 1. Means, ranges, mean squares (MS), phenotypic and genotypic variances of RWA reactions [RT%= (LD × PCR% × PGD)/9] for F_2-derived families in the 'Halt'×'synthetic hexaploid-11' cross.

Source of variation	Mean of RT (%)	Range of RT (%)	MS	Phenotypic variance	Genotypic variance
Within F3 families	0-100	0-100	0.065	0.046	0.019
Within F4 families	0-80	0-100	0.027	-[a]	-[a]
Between F3 families	41.68	0-100	2.235***	1.12	0.82
Between F4 families	36.21	0-100	0.441***	-[a]	-[a]

Mean squares (MS), phenotypic and genotypic variances are based on √x transformed data;
*** Significant at P<0.001;
[a] not calculated.

RESULTS AND DISCUSSION

Variance components within and between F_2-derived families for RWA reaction indicated that none of the within-family variances was significant (Table 1). The between-family variances of F_3 families were highly significant and much greater in magnitude than the within-family component. The F_3 between-family variances were 34 times greater than the within-family components. Although the variances within families for RWA resistance were not significant, there was variation within families due to segregation.

Broad-sense heritability (H_b) of RWA resistance, calculated by partitioning phenotypic variation of F_3 families into genetic and environmental components, was 73.2%. A regression analysis between F_3 and F_4 families for RWA resistance was established. The calculated regression coefficient (b = 0.30) was given as an estimate of narrow-sense heritability for the RWA resistance in the Halt×synthetic hexaploid-11 cross.

The reactions of the families were also qualitatively assessed by classifying the family responses into three groups of: homozygous resistant, heterozygous resistant and susceptible. Among the $F_{2:3}$ families tested from the cross 'Halt' (R) × synthetic hexaploid-11(S), 14 families were homozygous resistant, 66 heterozygous, and 27 homozygous susceptible. Results of chi-square analysis revealed that the original F_2 progenitors of these families genotypically did not segregate in a ratio of 1RR: 2Rr: 1rr (χ^2 = 9.0, P < 0.01) while phenotypically they segregated in a ratio of 3R-: 1rr (χ^2 = 0.547, P = 0.73).

Genetic variation between F_2 derived families would be expected to be greater than within families in self-fertilizing crops (Jensen, 1988). Our results indicated that significant genetic variation for genes of minor effect are associated with the major RWA resistance locus (*Dn4*). This might be a reason why the calculated R^2 (0.21) was not as high as anticipated in this study.

The low value of narrow-sense heritability (30%) when compared with the high broad-sense heritability (73.2%) for RWA resistance in the Halt × Synthetic hexaploid-11 cross suggests that the resistance is also governed by non-additive genetic effects in this cross. Since dominance effects disappear as homozygosity is approached with inbreeding, variation between families is thus expected to be greater than within families for RWA resistance after F_3 generation. Although the results of this study indicate that between-family selection for RWA resistance is more effective than within-family selection, due to a relatively low narrow-sense heritability (30%), the resistance may not readily be improved by selection for the modifier genes controlling RWA resistance in this cross.

The segregation ratio for RWA resistance in the cross of synthetic hexaploid-11 (susceptible parent) × 'Halt' (resistant parent containing *Dn4*) was consistent with the ex-

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pected Mendelian segregation ratio of 3R-: 1rr (χ^2 = 0.0031, P = 0.95). This result is in agreement with that of Nkongolo (1991) who used three crosses between the PI 372129 parent and 'Lamar', 'Carson' and 'TAM107' wheat cultivars. On the other hand, Liu *et al*. (2002) reported a significant deviation from the expected ratio of 3: 1 in one of the two crosses (Thunderbird (S) × PI 372129).

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تنوع ژنتیکی مقاومت به شته روسی گندم در فامیل‌های حاصل از F2 در گندم

(*Triticum aestivum* L.)

ا. ارزانی و ن. لپیتان

چکیده

شته روسی گندم (RWA)، *Diuraphis noxia* (Mordvilko) حشره آفت مهم گندم در سطح دنیاست. مقاومت میزبان به RWA بر اساس آنتی زنوز، آنتی بیوز و تحمل استوار می باشد. هدف از این مطالعه ارزیابی تنوع ژنتیکی داخل و بین فامیل‌های حاصل از F_2 برای واکنش به RWA با استفاده از فامیل های F_3 و F_4 (از تک بوته های F_2 تلاقی بین لاین حساس synthetic hexaploid-11 و رقم مقاوم Halt) بود. خسارت RWA به تک بوته‌ها داخل هر فامیل با استفاده از روش‌های مختلف اندازه‌گیری شد. این نوع واکنش ها به یک داده برای هر فامیل (حاصل از تک بوته های F_2) تبدیل شده و سپس مورد تجزیه و تحلیل آماری قرار گرفت. نتایج نشان داد که تنوع ژنتیکی بین فامیل ها بیشتر از داخل فامیل‌های حاصل از F_2 برای مقاومت به RWA می‌باشد. وراثت‌پذیری کل مقاومت به RWA که از طریق تجزیه واریانس فنوتیپی به اجزای ژنتیکی و محیطی برآورد شد برابر با ٪۷۳/۲ بود. وراثت‌پذیری خصوصی با برآوردی مساوی ٪۳۰ برای مقاومت به شته روسی در تلاق Halt × synthetic hexaploid-1 با استفاده از روش رگرسیون والد–نتاج (F_3: F_4) بدست آمد.

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