Transgressive Segregation, Heritability, and Number of Genes Controlling Durable Resistance to Stripe Rust in One Chinese and Two Italian Wheat Cultivars

Z. J. Zhang, G. H. Yang, G. H. Li, S. L. Jin, and X. B. Yang

First, second, and third authors: China Agricultural University, Beijing 100094, P. R. China; fourth author: Gansu Academy of Agricultural Science, Lanzhou 730070, P. R. China; and fifth author: Iowa State University, Ames 50011.

Accepted for publication 6 April 2001.

ABSTRACT

Zhang, Z. J., Yang, G. H., Li, G. H., Jin, S. L., and Yang, X. B. 2001. Transgressive segregation, heritability, and number of genes controlling durable resistance to stripe rust in one Chinese and two Italian wheat cultivars. *Phytopathology* 91:680-686.

Wheat (*Triticum aestivum*) cvs. Libellula (LB), San Pastore (SP), and Xian Nong 4 (XN4) possess durable resistance to stripe rust, caused by *Puccinia striiformis* f. sp. *tritici*, and cv. Ming Xian 169 (MX169) is highly susceptible to the rust. Inheritance of stripe rust resistance was studied by crossing the four cultivars and evaluating the resistance of parental, F₁, F₂, backcross, and F₃ plants in the fields. Transgressive segregation for resistance was observed in the resistant by resistant crosses of LB × XN4 and XN4 × SP, but not in cross LB × SP. These results indicate that (i) the resistance genes in XN4 are different from those in LB and SP, and (ii) LB and SP share common resistance genes.

The number of genes segregating for the resistance was estimated by quantitative methods from the data of F₂, backcross, and F₃ populations. LB and XN4 appear to have two to three resistance genes, and SP appears to have two to four resistance genes when crossed with MX169. The resistance gene number in resistant by resistant cross LB × XN4 was four to five, approximately equal to the sum of the genes in LB and XN4. Similarly, the resistance gene number in cross XN4 × SP was approximately equal to the sum of the genes in XN4 and SP. Broad-sense heritability was high in all crosses except LB × SP. Compared with the three MX169-involved crosses, narrow-sense heritability was higher in LB × MX169 and SP × MX169 crosses than in the XN4 × MX169 cross. The LB × XN4 and XN4 × SP crosses showed moderate narrow-sense heritability.

Additional keywords: quantitative genetics, slow rusting, yellow rust.

Stripe rust, caused by *Puccinia striiformis* Westend. f. sp. *tritici*, is an important disease of wheat (*Triticum aestivum* L.) in many regions of the world (12). In China, epidemics of this disease occur periodically and in some years cause destructive yield losses in the major wheat production region between the Great Wall and the Yangtze River (12,26). Use of host resistance is an effective, economical, and environmentally safe means for controlling this disease. Genes conferring race-specific resistance have been used in many instances, but this type of resistance is vulnerable to genetic changes in pathogen virulence. Extensive use of race-specific resistance in China resulted in rapid build-up of new stripe rust fungus races and, subsequently, host susceptibility (22,23,25). A striking example occurred in China in 1985 to 1990. The gene *Yr9* controlling race-specific resistance to stripe rust, after being widely used in the early 1980s, was rendered ineffective by new stripe rust fungus races (25), resulting in destructive wheat yield losses. Another example came from the Pacific Northwest of the United States where severe stripe rust epidemics occurred mainly because of extensive planting of wheat cultivars with race-specific resistance during the late 1950s (13). A more durable type of resistance to stripe rust is greatly needed worldwide. Johnson (8–10) introduced the term “durable resistance” meaning “resistance remains effective in a cultivar that is widely grown for a long period of time in an environment favorable to the disease.” Durable resistance to wheat stripe rust has been identi-

fied in Britain (8,9), China (30), and the United States (13). The durable resistance to stripe rust was observed in the form of high-temperature, adult-plant (HTAP) resistance (13) or slow-rusting resistance (10,30). The inheritance of the resistance is controlled by a few genes (2,15) or polygenes (8). Transgressive segregation has been observed in wheat for HTAP resistance (15) and slow-rusting resistance (21) to stripe rust.

Two wheat cultivars, Xian Nong 4 (XN4) and Libellula (LB), possess durable resistance to stripe rust in China (30). XN4 and LB have been commercially used cultivars since 1975 and 1980 (6), respectively, in Northwestern China where the environment is conducive to stripe rust, and new stripe rust fungus races occur frequently (22,23,25). XN4 was planted on 27,000 and 42,000 ha in 1981 and 1994, respectively, and LB was planted on 38,000 and 32,000 ha in 1986 and 1994, respectively (30). The acreage for both cultivars has increased in recent years (4). From 1975 to 1996, 11 new stripe rust fungus races were detected in Northwestern China, and many wheat cultivars with race-specific resistance were defeated by these races (22,23,25). However, resistance to stripe rust in cvs. XN4 and LB remains effective. Another wheat cultivar, San Pastore (SP), showed slow-rusting resistance to stripe rust when it was commercially grown in Northwestern China in the 1960s (6). Replaced by cultivars possessing better agronomic and locally adaptive traits from commercial fields, cv. SP still showed slow-rusting resistance in experimental plots in 1977 to 1993 (14,16,28). Despite the practical value of the three cultivars, inheritance of their resistance to stripe rust is unknown.

The objectives of this study were to estimate the number of genes controlling the resistance to stripe rust in wheat cvs. LB, SP, and XN4, to estimate heritability of the resistance, and to determine if the resistance genes in the cultivars are different.

Corresponding author: Z. J. Zhang; E-mail address: Zhang60@yahoo.com

Publication no. P-2001-0514-01R

© 2001 The American Phytopathological Society

MATERIALS AND METHODS

Wheat parents and crosses. Four winter wheat cultivars, LB, XN4, SP, and Ming Xian 169 (MX169), were used in crossing. LB is deposited as accession no. PI 295620 in the U.S. Department of Agriculture GRIN database with the pedigree Tevere/Giuliani/San Pastore, and SP is deposited as accession no. PI 259889 with the pedigree Balilla/Villa Glori. XN4 and MX169 are deposited, respectively, at the Food Crop Institute of Gansu Academy of Agricultural Science, Lanzhou, and at the Plant Protection Institute of Chinese Academy of Agricultural Science, Beijing, China. Both XN4 and MX169 are Chinese local cultivars with unknown pedigree. LB, XN4, and SP carry durable resistance to stripe rust in the form of slow-rusting resistance that retards infection, growth, and reproduction of stripe rust fungus on wheat plants. The slow-rusting resistance in the three cultivars was determined by measuring the latent period, pustule development, and urediospore production of stripe rust fungus on adult plants in experimental plots (27,28). MX169 is highly susceptible to all known stripe rust fungus races detected in China (22,23,25).

The experiments were carried out in the fields in Beijing where the winter wheat is sown in October and harvested the following June, and where it is unlikely for stripe rust to occur naturally (no natural occurrence of stripe rust was observed during 1995 to 2000 when this study was conducted). During the 1995-96 growing season, the four wheat cultivars were crossed in all 12 possible combinations, and F_1 seeds of all crosses were harvested. During the 1996-97 growing season, parental and F_1 seeds were planted, and F_2 and additional F_1 seeds were produced. Reciprocal F_1 plants were crossed with their respective female parents, with F_1 plants as pollen donors to produce backcross (BC) generations including BC to the more resistant parent (BC_r) and BC to the more susceptible parent (BC_s). In addition, 38 to 52 F_1 plants from each reciprocal cross were evaluated for resistance to stripe rust to test for reciprocal differences. No significant reciprocal differences were found (data not shown), and hence, reciprocal populations of each cross were combined. In later crop seasons, six crosses were tested including the three resistant by susceptible crosses, LB $\times$ MX169, SP $\times$ MX169, and XN4 $\times$ MX169, and the three resistant by resistant crosses, LB $\times$ SP, LB $\times$ XN4, and XN4 $\times$ SP.

Experiment for parental, F_1 , F_2 , and backcross populations. During the 1997-98 growing season, parental, F_1 , F_2 , BC_r , and BC_s populations were evaluated for resistance. On 3 and 4 October 1997, seeds were sown 20 cm apart in rows 2 m long with 10 seeds per row and 25-cm row space. Population size was 60 seeds per parent or per F_1 , 360 seeds per F_2 for resistant by susceptible crosses, 480 seeds per F_2 for resistant by resistant crosses, 120 seeds per BC for resistant by susceptible crosses, and 180 seeds per BC for resistant by resistant crosses. The population size was larger for resistant by resistant crosses than for resistant by susceptible crosses because we assumed that the former might involve more resistance genes than the latter. There were 4,920 seeds in the experiment and they were sown in 492 rows in six blocks. In each

block, there were 82 test rows including one row each of the four parents, 11 rows of progenies (i.e., F_1 , F_2 , BC_r , and BC_s) from each of the three resistant by susceptible crosses, and 15 rows of progenies from each of the three resistant by resistant crosses. Within each block, progeny rows from the same cross were grouped together, and the parental rows were randomly arranged between the cross groups. Within each cross group, F_1 , F_2 , BC_r , and BC_s populations were randomly intermixed. One row of cv. MX169 was sown every 2 rows of the 82 test rows in each block as inoculum spreader to enhance reinfection. Such layout was designed to condition a favorable and uniform environment for rust development. Blocks were used to facilitate inoculation and data collection, but were not used as replicates because each F_2 or backcross plant was a unique genetic or experimental unit.

On 9 and 10 April 1998, when the wheat was at tillering stages (from 21 to 29 in Zadoks et al.'s code; 29), the parental, F_1 , F_2 , BC_r , BC_s , and MX169 spreader plants in six blocks were inoculated with urediospores of Chinese race 31 of *P. striiformis* f. sp. *tritici*. This race is virulent on 14 of the 17 differential wheat varieties and has been a predominant race in China since 1994 (22). The original urediospores were provided by the Chinese National Cooperation Group for Surveying Wheat Stripe Rust Fungus Races and were increased on MX169 in greenhouses. In late afternoons, right before inoculation, fresh urediospores were mixed with talc (1:1,000, wt/wt), experimental field was irrigated thoroughly, plants were first sprayed with water containing 0.02% Tween 20 (Fisher Scientific China, Panyu), and spore/talc mixtures were uniformly dusted onto plants with a cyclone dusting machine (Plant Protection Instruments, Beijing, China) in a density of 200 g of spore/talc mixture per 100 m² of experimental field. After inoculation, each block was covered with a plastic sheet overnight to keep moisture. To condition a favorable and uniform environment for disease development, the experimental field was irrigated in late afternoons at 2- to 7-day intervals, depending on rainfall, until disease assessment began.

On 3, 10, 17, and 24 May 1998, when the wheat was at the growth stages from booting to milk (from 41 to 75 in Zadoks et al.'s code; 29), rust severity (percentage of leaf area with symptoms) of the top three leaves was recorded for each plant according to the 0 to 100% Peterson scale (18). Data collection was completed within 12 h on each recording date. Area under the disease progress curve (AUDPC) was calculated for individual plants using the original rust severity data with the formula

$$AUDPC = \sum [(x_i + x_{i+1})/2] t_i \quad (1)$$

in which x_i and x_{i+1} are severity (percentage of leaf area with symptoms) on date i and date $i + 1$, respectively, and t_i = days between date i and date $i + 1$. The mean AUDPC values of F_1 and segregating populations over all the plants in six blocks were calculated for each cross and were used to characterize the inheritance of resistance. The mean AUDPC value of each parent was obtained by averaging the AUDPC values of all plants in six blocks (Table 1).

TABLE 1. Means and standard deviations of area under disease progress curve (AUDPC) for parent wheat cvs. Libellula (LB), Ming Xian 169 (MX169), San Pastore (SP), and Xian Nong 4 (XN4), and their F_1 , F_2 , backcross to resistant parent (BC_r), and F_3 populations, tested with *Puccinia striiformis* f. sp. *tritici* in fields during 1997-1998 and 1999-2000 crop seasons

Means and standard deviations of AUDPC for each cross ($P_1 \times P_2$)							
Year	Generation	LB $\times$ MX169	SP $\times$ MX169	XN4 $\times$ MX169	LB $\times$ SP	LB $\times$ XN4	XN4 $\times$ SP
1997 to 1998	P_1	162 $\pm$ 42	467 $\pm$ 83	350 $\pm$ 91	162 $\pm$ 42	162 $\pm$ 42	350 $\pm$ 91
	P_2	1,365 $\pm$ 77	1,365 $\pm$ 77	1,365 $\pm$ 77	467 $\pm$ 83	350 $\pm$ 91	467 $\pm$ 83
	F_1	694 $\pm$ 79	898 $\pm$ 81	763 $\pm$ 86	298 $\pm$ 64	245 $\pm$ 88	382 $\pm$ 64
	F_2	660 $\pm$ 311	673 $\pm$ 320	767 $\pm$ 321	260 $\pm$ 130	211 $\pm$ 258	474 $\pm$ 272
	BC_r	226 $\pm$ 175	316 $\pm$ 237	509 $\pm$ 278	182 $\pm$ 146	160 $\pm$ 215	314 $\pm$ 203
1999 to 2000	P_1	148 $\pm$ 50	482 $\pm$ 93	347 $\pm$ 94	161 $\pm$ 41	152 $\pm$ 49	354 $\pm$ 98
	P_2	1,325 $\pm$ 91	1,356 $\pm$ 102	1,351 $\pm$ 105	436 $\pm$ 84	311 $\pm$ 96	452 $\pm$ 101
	F_3	578 $\pm$ 274	603 $\pm$ 335	791 $\pm$ 307	297 $\pm$ 139	242 $\pm$ 291	461 $\pm$ 327

Experiment for parental and F_3 populations. During the 1999-2000 growing season, 100 randomly sampled F_3 lines from each resistant by susceptible cross and 150 randomly sampled F_3 lines from each resistant by resistant cross were evaluated for resistance. On 3 to 6 October 1999, 80 seeds of each F_3 line were sown in a randomized block design of four replicates with 20 seeds per replicate, in 2-m rows with 10 cm between seeds and 25 cm between rows. One row of each parent was sown every 25 rows of F_3 . For a resistant by resistant cross, each replicate had 150 rows of F_3 and six rows of each parent. For a resistant by susceptible cross, each replicate had 100 rows of F_3 and four rows of each parent. Two rows of MX169 were sown perpendicular to the test rows on both sides as inoculum spreaders to enhance reinfection. On 10 and 11 April 2000, when the wheat was at tillering stages

(from 21 to 29 in Zadoks et al.'s code; 29), the parental, F_3 , and MX169 spreader plants were inoculated with the same race and methods used for the 1998 experiment. The experimental field was irrigated at 2- to 7-day intervals until disease assessment began. On 3, 10, 17, and 24 May 2000, when the wheat was at the growth stages from booting to milk (from 41 to 75 in Zadoks et al.'s code; 29), rust severity was recorded for each row by the same methods used for the 1998 experiment. Data collection was completed within 15 h on each recording date. AUDPC was calculated for individual rows by the same methods and formula used for the 1998 experiment. The mean AUDPC value of each F_3 population was calculated using the F_3 line means over four replicates, and the mean AUDPC values of parents over four replicates were calculated for each cross (Table 1).

Fig. 1. Frequency distributions of F_2 plants, F_3 lines, and parental plants or rows over 14 area under disease progress curve (AUDPC) value classes that are in increments of 100 AUDPC units per class, tested with Chinese race 31 of *Puccinia striiformis* f. sp. *tritici*. **A**, **B**, and **C** indicate the crosses of susceptible wheat cv. Ming Xian 169 (MX169) with resistant cvs. Libellula (LB), San Pastore (SP), and Xian Nong 4 (XN4), respectively.

Estimation of gene number and heritability. Number of genes segregating for resistance was estimated according to Wright's formulas (24) and a modified version for F_3 generation by Bjarko and Line (1).

$$F_2: n = GR^2 \times [1.5 - 2h(1 - h)] / \{8[V_{F_2} - (V_{P_s} + V_{P_r} + 2V_{F_1})/4]\} \quad (2)$$

$$BC_r: n = GR^2 / \{4[V_{BCr} - (V_{P_r} + V_{F_1})/2]\} \quad (3)$$

$$F_3: n = GR^2 / \{5.33[V_{F_3} - (V_{P_r} + V_{P_s})/2]\} \quad (4)$$

in which n = number of segregating genes, GR = genotypic range, V_{P_r} = variance of resistant parent, V_{P_s} = variance of susceptible

parent, V_{F_1} = variance of F_1 generation, V_{F_2} = variance of F_2 generation, V_{F_3} = variance of F_3 generation, V_{BCr} = variance of the backcross generation to the more resistant parent, and $h = (M_{F_1} - M_{P_r}) / (M_{P_s} - M_{P_r})$, in which M_{F_1} = mean of F_1 population, M_{P_r} = mean of resistant parent, and M_{P_s} = mean of susceptible parent. In each formula, the gene number was estimated by dividing the square of genotypic range by genotypic variance. In this study, genotypic range was estimated by using the phenotypic range of segregating population, which does not assume that segregating genes come from a single parent; thus, it can be applied to resistant by resistant crosses as well as to resistant by susceptible crosses. Genotypic variance was estimated by subtracting environmental variance from phenotypic variance of segregating populations.

Fig. 2. Frequency distributions of F_2 plants, F_3 lines, and parental plants or rows over 14 area under disease progress curve (AUDPC) value classes that are in increments of 100 AUDPC units per class, tested with Chinese race 31 of *Puccinia striiformis* f. sp. *tritici*. **A, B, and C** indicate the resistant by resistant crosses of cvs. Libellula (LB) $\times$ San Pastore (SP), LB $\times$ Xian Nong 4 (XN4), and XN4 $\times$ SP, respectively.

Heritability was estimated by the methods of variance components (17), F_3/F_2 regression (19), and standard units (3). The heritability estimated by variance components represents all genetic variance, and the heritability estimated by F_3/F_2 regression and standard units represents additive genetic variance plus a quarter of dominance variance, subject to some assumptions (3,17). For convenience, the former is called broad-sense heritability (BSH), and the latter called narrow-sense heritability (NSH).

RESULTS

Mean rust severity (percentage of leaf area with rust symptoms) was approximately 90% for susceptible cv. MX169 at the final recordings in both 1998 and 2000, whereas it varied from 8 to 46% for resistant cvs. LB, SP, and XN4, depending on cultivar and crop seasons. These results suggest that the disease pressure was sufficiently high to reveal genetic difference between resistance and susceptibility. The means and standard deviations of rust intensity expressed in AUDPC value for each generation are summarized in Table 1 and were used to estimate gene number and heritability.

F_2 plants, F_3 lines, and parental plants or rows were grouped into 14 AUDPC value classes with an increment of 100 AUDPC units per class. Distributions of F_2 plants, F_3 lines, and parental plants or rows over AUDPC classes are shown for each cross in Figures 1 and 2. In the resistant by susceptible cross, SP $\times$ MX169 (Fig. 1B), 12.3% of F_2 plants and 22% of F_3 lines had AUDPC values lower than the resistant parent, SP, suggesting that transgressive segregation for resistance occurred in this cross. In the other two resistant by susceptible crosses, LB $\times$ MX169 (Fig. 1A) and XN4 $\times$ MX169 (Fig. 1C), less than 5% of F_2 plants or F_3 lines had AUDPC values lower than their respective resistant parents. In the resistant by resistant cross, LB $\times$ XN4 (Fig. 2B), 45.1% of F_2 plants and 52% of F_3 lines had AUDPC values lower than the more resistant parent, LB, and 10.8% of F_2 plants and 13.9% of F_3 lines had AUDPC values higher than the more susceptible parent, XN4. These indicate that transgressive segregation for both resistance and susceptibility occurred in this cross. Similarly, transgressive segregation for both resistance and susceptibility was also observed in the resistant by resistant cross XN4 $\times$ SP (Fig. 2C). In the other cross, LB $\times$ SP (Fig. 2A), 3% of F_2 plants had AUDPC values lower than the more resistant parent, LB.

TABLE 2. Number of genes segregating for resistance to *Puccinia striiformis* f. sp. *tritici* in the crosses between wheat cvs. Libellula (LB), Ming Xian 169 (MX169), San Pastore (SP), and Xian Nong 4 (XN4), estimated by using area under disease progress curve (AUDPC) values of F_2 , F_3 , and backcross to resistant parent (BC_r) populations, with phenotypic range as the estimation of genotypic range (GR)

More resistant parent (P_r)	More susceptible parent (P_s)	Estimated gene number (n) based on the different segregating populations ^a		
		F_2	F_3	BC_r
LB	MX169	2.4	3.3	3.4
SP	MX169	2.3	3.2	4.1
XN4	MX169	2.2	2.4	3.4
LB	SP	1.6	2.5	2.2
LB	XN4	4.1	4.7	4.9
XN4	SP	5.6	3.6	6.2

^a For F_2 , $n = \{GR^2 \times [1.5 - 2h(1 - h)]\} / \{8[V_{F2} - (V_{Pr} + V_{Ps} + 2V_{F1})/4]\}$ in which n = number of segregating genes, GR = genotypic range estimated by phenotypic range, V_{F2} = variance of F_2 generation, V_{Pr} = variance of resistant parent, V_{Ps} = variance of susceptible parent, V_{F1} = variance of F_1 generation, and $h = (M_{F1} - M_{Pr}) / (M_{Ps} - M_{Pr})$ in which M_{F1} = mean of F_1 generation, M_{Pr} = mean of resistant parent, and M_{Ps} = mean of susceptible parent (24). For F_3 , $n = GR^2 / \{5.33[V_{F3} - (V_{Pr} + V_{Ps})/2]\}$ in which V_{F3} = variance of F_3 generation (1,24). For BC_r , $n = GR^2 / \{4[V_{BCr} - (V_{Pr} + V_{F1})/2]\}$ in which V_{BCr} = variance of the backcross generation to the more resistant parent (24).

The number of genes segregating for stripe rust resistance was estimated using the data in Table 1 and were summarized in Table 2. F_2 , F_3 , and BC_r provided similar estimates of gene number for each cross. With MX169 as the susceptible parent, two to three resistance genes appeared to be segregating in LB or XN4, and two to four in SP. For resistant by resistant crosses, one to two segregating genes were estimated in LB $\times$ SP, four to five in LB $\times$ XN4, and four to six in XN4 $\times$ SP. The estimated gene number in the resistant by resistant cross LB $\times$ XN4 (4.1 to 4.9) was generally more than that in the resistant by susceptible cross LB $\times$ MX169 (2.4 to 3.4) or XN4 $\times$ MX169 (2.2 to 3.4). A similar relationship existed in crosses XN4 $\times$ SP, XN4 $\times$ MX169, and SP $\times$ MX169, but did not in crosses LB $\times$ SP, LB $\times$ MX169, and SP $\times$ MX169.

Heritability for each cross was estimated by different methods (Table 3). BSH was generally higher than NSH. For each cross, the BSH estimated by F_2 variance components was nearly equal to that estimated by F_3 variance components, and the NSH estimated by F_2/F_3 regression was nearly equal to that estimated by standard units. BSH was equal to or higher than 0.9 in all crosses except in LB $\times$ SP. NSH varied with cross. For the three MX169-involved crosses, the NSH in LB $\times$ MX169 (0.79 or 0.83) was not substantially different from that in SP $\times$ MX169 (0.85 or 0.81), but obviously different from that in XN4 $\times$ MX169 (0.46 or 0.51). NSH in cross LB $\times$ XN4 (0.58 or 0.64) was not substantially different from that in cross XN4 $\times$ SP (0.62 or 0.60). The lowest NSH (0.39 or 0.44) was observed in cross LB $\times$ SP.

DISCUSSION

Of the three crosses involving highly susceptible wheat cv. MX169, crosses LB $\times$ MX169 and XN4 $\times$ MX169 did not show transgressive segregation (Fig. 1A and C). The small portion (less than 5%) of F_2 plants or F_3 lines with lower AUDPC values than their respective resistant parents might have resulted from chance. These results indicate that MX169 did not contribute any detectable resistance gene. This is consistent with the results reported by other researchers that MX169 was highly susceptible to all races of *P. striiformis* f. sp. *tritici* detected in China (22,23,25). However, in the other MX169-involved cross, SP $\times$ MX169, 12.3% of F_2 plants and 22% of F_3 lines had AUDPC values lower than resistant parent SP, suggesting that cross SP $\times$ MX169 apparently showed transgressive segregation for resistance (Fig. 1B). We could not, thus far, find factual basis to explain the transgressive segregation in cross SP $\times$ MX169, although a speculation is that this cross might involve gene interaction. One can assume that SP has a gene that may suppress the expression of a resistance gene when they coexist in the parent. In segregating generations, the resistance gene might separate from suppressor gene and, consequently, higher resistance can be expressed in some individuals.

Of the three resistant by resistant crosses, LB $\times$ SP did not show transgressive segregation (Fig. 2A) but LB $\times$ XN4 and XN4 $\times$ SP did for both resistance and susceptibility (Fig. 2B and C). These results indicate that SP and LB share common resistance

TABLE 3. Heritability of resistance to *Puccinia striiformis* f. sp. *tritici* in the crosses between wheat cvs. Libellula (LB), Ming Xian 169 (MX169), San Pastore (SP), and Xian Nong 4 (XN4), estimated by the methods of F_2 or F_3 variance components, F_3/F_2 regression, and standard units

Cross	Broad-sense heritability		Narrow-sense heritability	
	F_2 variance components	F_3 variance components	F_3/F_2 regression	Standard units
LB $\times$ MX169	0.95	0.93	0.79	0.83
SP $\times$ MX169	0.94	0.92	0.85	0.81
XN4 $\times$ MX169	0.93	0.90	0.46	0.51
LB $\times$ SP	0.75	0.77	0.39	0.44
LB $\times$ XN4	0.91	0.93	0.58	0.64
XN4 $\times$ SP	0.91	0.92	0.62	0.60

genes, and that the resistance genes in LB and SP are different from those in XN4. These observations agree with the genealogical records that SP is in the pedigree of LB (6,7). SP and LB were introduced from Italy to China, respectively, in the 1950s and 1960s (7), before which they were geographically separated from the Chinese local cv. XN4 and, hence, had little chance to share common resistance genes with XN4.

To further understand the inheritance of durable resistance to stripe rust in the three wheat cultivars, we estimated the number of genes controlling the resistance. In previous reports, both qualitative and quantitative methods were used to estimate gene number for durable resistance, adult plant resistance, or slow disease development resistance (1,2,5,11,15,20). In this study, F_2 plants and F_3 lines showed continuous distribution over AUDPC value classes (Figs. 1 and 2), and no discrete segregation was observed. We, therefore, used the quantitative method to estimate gene number. It appears that two to three genes in LB or XN4, and two to four genes in SP, segregated for resistance when they were crossed with susceptible wheat cv. MX169. Three to six genes segregated for resistance when XN4 was crossed with LB or SP, approximately equal to the sum of the genes in XN4 plus the genes in LB or in SP. These results further suggest that the resistance genes in XN4 are different from those in LB and SP. One to two genes segregated in the LB $\times$ SP cross, suggesting that the two cultivars may be different in some resistance genes, although they share common genes.

Our estimate is reasonable on the one hand that the estimated gene number in the resistant by resistant cross is more than that in the resistant by susceptible cross. For instance, gene number in resistant by resistant cross LB $\times$ XN4 is four to five, whereas gene number is two to three in resistant by susceptible cross LB $\times$ MX169 or XN4 $\times$ MX169. On the other hand, regarding the accuracy of the estimated gene number, the following considerations should be taken. First, the estimates were based on the following assumptions (24): no linkage exists between the genes involved; all resistance genes have equal effects; there are no dominance, no epistasis, and no genotype-environment interactions. Presence of linkage, dominance, or unequal effects at different loci can cause an underestimation of the number of resistance genes. The actual resistance gene number in LB, SP, and XN4, therefore, may be more than the estimated number. Presence of epistasis can cause either an overestimation or underestimation of the actual gene number. Epistasis may exist in cross SP $\times$ MX169. Second, we used the phenotypic range of segregating population as the estimate of genotypic range after Bjarko and Line (1). The advantage of this method is that the number of segregating genes in resistant by resistant crosses as well as in resistant by susceptible crosses can be estimated. This method does not assume that all resistance genes are in one parent, such as the method of using the difference between parental means as estimate of genotypic range, which is not suitable for estimating gene number in resistant by resistant crosses. However, phenotypic range is more vulnerable to environmental influence than the difference between parental means. To reduce the environmental influence, replicates were applied in the experiment for F_3 generation. Therefore, the estimates based on F_3 data are theoretically more accurate and reliable than those based on F_2 and BC_r data. Other measures were taken to reduce environmental influences by increasing the degree of homogeneity of the experiments. For instance, the experimental fields were frequently irrigated from inoculation to disease assessment, resulting in more uniform dew on the plant surface for infection than with naturally occurring dew (data not shown), and one row of highly susceptible wheat cv. MX169 was intermixed with every two rows of the test plants of parent, F_1 , F_2 , or BC to increase the uniformity of inoculum during reinfection.

The estimates of BSH, based on different generations, were very similar for each cross, and high in all crosses except LB $\times$ SP, suggesting that selection in early generations can be effective for

the stripe rust resistance in these crosses. This is in agreement with the results for HTAP resistance of wheat to stripe rust reported by Chen and Line (2). NSH estimates obtained by the two methods of F_3/F_2 regression and standard units were similar for each cross, suggesting that environmental influences did not affect the phenotypic variances in parents and progenies to a great extent. This suggestion can also be supported by the results that the mean AUDPC values in 1998 and 2000 were nearly equal for each parent and that the standard deviations in the 2 years were nearly equal for each parent (Table 1). The lowest NSH was observed in the cross of LB $\times$ SP. This can be explained if LB and SP share some common resistance genes, and consequently, genetic variance in this cross is small. Compared with the three MX169-involved crosses, the NSH in XN4 $\times$ MX169 is lower than that in LB $\times$ MX169 or SP $\times$ MX169, suggesting that the resistance in XN4 may not be transmitted as easily as that in LB or SP. Crossed with either MX169 or XN4, LB and SP showed a high level of similarity in NSH, suggesting that LB and SP share common inheritance manner of the resistance. The fact that NSH was not low in crosses LB $\times$ XN4 and XN4 $\times$ SP suggests that selection for enhanced resistance would not be difficult in these crosses.

In summary, LB and XN4 appear to have two to three genes controlling the resistance to stripe rust, and SP appears to have two to four. Italian cvs. LB and SP share common resistance genes with each other but not with Chinese cv. XN4. Transgressive segregation for resistance in the cross of XN4 with LB or SP indicates that a higher level of resistance can be expected from these crosses. The estimations of heritability indicate that selection in early generations can be effective for the stripe rust resistance in some crosses, the resistance in XN4 may not be transmitted as easily as that in LB or SP when crossed with susceptible cv. MX169, and selection for enhanced resistance would not be difficult in the cross of XN4 with LB or SP. These results are valuable for understanding the inheritance of durable resistance to stripe rust, as well as to breeding cultivars with better stripe rust resistance by using LB, SP, and XN4 as resistance resources.

ACKNOWLEDGMENTS

This study was supported by the International Foundation for Science in Sweden and the National Natural Science Foundation of China. We thank X. M. Chen for critically reviewing the manuscript before submission, and the two anonymous reviewers for valuable suggestions.

LITERATURE CITED

1. Bjarko, M. E., and Line, R. F. 1988. Heritability and number of genes controlling leaf rust resistance in four cultivars of wheat. *Phytopathology* 78:457-461.
2. Chen, X., and Line, R. F. 1995. Gene number and heritability of wheat cultivars with durable, high-temperature, adult-plant (HTAP) resistance and interaction of HTAP and race-specific seedling resistance to *Puccinia striiformis*. *Phytopathology* 85:573-578.
3. Frey, K. J., and Horner, T. 1957. Heritability in standard units. *Agron. J.* 49:59-62.
4. Gansu Seeds Station. 1998. Reports on the survey of the cultivation and distribution of wheat cultivars during the period 1980-1998. Gansu Seeds Stn., Lanzhou, P. R. China.
5. Griffey, C. A., and Das, M. K. 1994. Inheritance of adult-plant resistance to powdery mildew in Knox 62 and Massey winter wheats. *Crop Sci.* 34:641-646.
6. Hu, Y., and Wang, Y. 1989. Records of wheat varieties of Gansu province. *Gansu Acad. Agric. Sci.*, Lanzhou.
7. Jin, S. 1983. Genealogies of Wheat Varieties of China. Agricultural Press of China, Beijing.
8. Johnson, R. 1978. Practical breeding for durable resistance to rust diseases in self-pollinating cereals. *Euphytica* 27:529-540.
9. Johnson, R. 1981. Durable resistance: Definition, genetic control, and attainment in plant breeding. *Phytopathology* 71:567-568.
10. Johnson, R. 1992. Reflections of a plant pathologist on breeding for disease resistance, with emphasis on yellow rust and eyespot of wheat. *Plant Pathol.* 41:239-254.

11. Jones, I. T. 1986. Inheritance of adult-plant resistance to mildew in oats. *Ann. Appl. Biol.* 109:187-192.
12. Li, Z., and Shang, H. 1989. Wheat Stripe Rust Disease and Control. Science and Technology Press of Shang Hai, Shang Hai.
13. Line, R. F., Chen, X. M., and Qayoum, A. 1992. Races of *Puccinia striiformis* in North America, identification of resistance genes, and durability of resistance. Pages 280-282 in: *Proc. Eur. Mediterr. Cereal Rusts Conf.*, 8th.
14. Lu, D., Yuan, W., Li, J., Li, D., and Yu, X. 1980. Studies on stripe rust resistance of the cultivar resources of wheat. *Sci. Agric. Sin.* 13:15-22.
15. Milus, E. A., and Line, R. F. 1986. Number of genes controlling high-temperature, adult-plant resistance to stripe rust in wheat. *Phytopathology* 76:93-96.
16. National Cooperative Research Group on Horizontal Resistance to Yellow Rust of Wheat. 1983. Preliminary report on the screening of wheat cultivars with horizontal resistance to yellow rust (*Puccinia striiformis*) by regional trials. *Sci. Agric. Sin.* 16:58-67.
17. Nyquist, W. E. 1991. Estimation of heritability and prediction of selection response in plant populations. *Crit. Rev. Plant Sci.* 10:235-322.
18. Peterson, R. F., Campbell, A. B., and Hannah, A. E. 1948. A diagrammatic scale for estimating rust intensity on leaves and stems of cereals. *Can. J. Res. Sect. C.* 26:496-500.
19. Smith, H. C., and Kinman, M. L. 1965. The use of parent-offspring regression as estimator of heritability. *Crop Sci.* 5:595-596.
20. Wagoire, W. W., Stølen, O., Hill, J., and Ortiz, R. 1998. Inheritance of adult field resistance to yellow rust disease among broad-based hexaploid spring wheat germplasm. *Theor. Appl. Genet.* 97:502-506.
21. Wallwork, H., and Johnson, R. 1984. Transgressive segregation for resistance to yellow rust in wheat. *Euphytica* 33:123-132.
22. Wan, A., Wu, L., and Yuan, W. 1999. Physiological specialization of stripe rust of wheat in China during 1991-1996. *Chinese J. Plant Pathol.* 29:15-21.
23. Wang, K., Wu, L., and Meng, Q. 1986. Physiological specialization of stripe rust of wheat in China during 1975-1984. *Chinese J. Plant Pathol.* 16:79-85.
24. Wright, S. 1968. The genetics of quantitative variability. Pages 351-436 in: *Evolution and Genetics of Populations. Vol. 1. Genetic and Biometric Foundations.* University of Chicago Press, Chicago.
25. Wu, L., and Yang, H. 1993. Physiological specialization of stripe rust of wheat in China during 1985-1990. *Chinese J. Plant Pathol.* 23:269-274.
26. Yang, X. B., and Zeng, S. M. 1992. Detecting patterns of wheat stripe rust pandemics in time and space. *Phytopathology* 82:571-576.
27. Yuan, W., and Li, G. 1996. Identification of wheat cultivars with slow-rusting resistance to stripe rust. *Gansu Agric. Sci. Tech.* 5:28-30.
28. Yuan, W., Zhang, Z. J., Feng, F., and Zeng, S. M. 1995. Identification of wheat cultivars with slow-rust resistance to *Puccinia striiformis*. *Sci. Agric. Sin.* 28:35-40.
29. Zadoks, J. C., Chang, T. T., and Konzak, C. F. 1974. A decimal code for the growth stages of cereals. *Weed Res.* 14:415-421.
30. Zhang, Z. J. 1995. Evidence of durable resistance in nine Chinese land races and one Italian cultivar of *Triticum aestivum* to *Puccinia striiformis*. *Eur. J. Plant Pathol.* 101:405-409.