Inheritance of resistance to common bunt in spelt and common wheat[1]

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He, C. and Hughes, G. R. 2003. **Inheritance of resistance to common bunt in spelt and common wheat.** Can. J. Plant Sci. **83**: 47–56. Common bunt causes yield loss and reduces grain quality in both common and durum wheats in western Canada. Since the most cost-effective method of control is the use of host resistance, this study was conducted to provide information on the genetic control of bunt resistance in three potentially new sources: common wheat cultivar Triple Dirk and spelt wheat cultivars RL5407 and SK0263. The segregating populations from the three crosses Laura/Triple Dirk, Laura/RL5407 and Genesis/SK0263 were evaluated for common bunt resistance in the field for races T1, T13 and L7 and in the greenhouse for race T1. Genetic analysis indicated that Triple Dirk may carry a major gene controlling resistance to each of the bunt races T1, T13 and L7. The spelt wheat RL5407 may carry a major gene for resistance to both races T13 and L7 or genes conditioning resistance to T13 and L7 that are closely linked, and an additional major gene for resistance to race T1. The two major genes carried by RL5407 are believed to be different. SK0263 possibly carried two major genes for resistance to race T1. The disease data in F_1 and F_2 generations did not show any dominance for bunt resistance to race T1 in any of the three crosses. From crosses involving Triple Dirk, RL5407 and SK0263, selection of breeding lines highly resistant to common bunt can be effective in the progenies due to the nature of non-dominance and one- or two-gene controlled resistance.

Kew words: Wheat, common bunt, *Triticum* sp., *Tilletia* sp.

He, C. et Hughes, G. R. 2003. **Hérédité de la résistance à la carie chez l'épeautre et le blé**. Can. J. Plant Sci. **83**: 47–56. Dans l'Ouest canadien, la carie du blé entraîne des pertes de rendement et réduit la qualité du grain des variétés ordinaires et dures de blé. La méthode de lutte la plus efficace consiste à rendre l'hôte résistant à la maladie. La présente étude devait nous renseigner sur trois nouvelles sources potentielles de résistance à la carie : le cultivar de blé commun Triple Dirk et les cultivars d'épeautre RL5407 et SK0263. Les auteurs ont évalué les populations distinctes issues des trois croisements Laura/Triple Dirk, Laura/RL5407 et Genesis/SK0263 pour voir si elles résistaient aux races T1, T13 et L7 de la carie au champ et à la race T1 en serre. L'analyse génétique révèle que Triple Dirk pourrait porter un gène important codant la résistance aux races T1, T13 et L7. La variété d'épeautre RL5407, quant à elle, possèderait un gène majeur de résistance aux races T13 et L7 ou des gènes étroitement liés, conditionnant cette résistance, ainsi qu'un gène important de résistance à la race T1. On croit que les deux grands gènes de résistance de RL5407 sont différents. Par ailleurs, il se pourrait que SK0263 porte deux gènes majeurs de résistance à la race T1. Selon les données recueillies sur la F_1 et la F_2, aucun des trois croisements ne présente de gène dominant codant la résistance à la race T1. La sélection de lignées généalogiques très résistantes à la carie du blé parmi les hybrides de Triple Dirk, de RL5407 et de SK0263 pourrait s'avérer efficace puisque la résistance n'est pas codée par un gène dominant ou résulte de l'action d'un seul ou de deux gènes.

Mots clés: Blé, carie du blé, *Triticum* sp., *Tilletia* sp.

Common bunt, caused by *Tilletia tritici* (Bjerk.) G. Wint. *in* Rabenh. [syn. *T. caries* (DC.) Tul. & C. Tul.) and *T. laevis* Kuhn *in* Rabenh. (syn. *T. foetida* (Wallr.) Liro.], has occurred in all wheat-growing countries of the world (Munjal 1966; Bahadur and Singh 1987). It causes yield loss in common and durum wheats (*Triticum aestivum* L. and *T. turgidum* L.) (Holton 1947; Goel and Singh 1975) and reduces grain quality through the production of dark fungal spores, which release a fishy odour (Flor et al. 1932). Control of common bunt by chemical treatment of the seed is possible, but not always effective, nor is it the control method of choice because of cost and the potentially adverse effects on the environment and human health. The most economic and effective means of controlling common bunt of wheat is through the use of cultivars with bunt resistance (Smeltzer 1952; Gaudet and Puchalski 1989b; Goates 1996).

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Abbreviation: **SHD**, single head derived; **SSD**, single seed derived; **BC_1F_1**, backcross F_1, i.e., $P_1 \times (P_1 \times P_2)$; **$BC_1F_2$**, BC_1F_1 single plant derived lines; **RCBD**, randomized complete block design

Table 1. Crosses and generations used for genetic studies of bunt resistance genes from 1995 to 1997

	Inoculated with T1				Inoculated with L7 and T13
Cross	Summer 1995	Summer 1996	Winter 1996-1997	Summer 1997	Summer 1996
Laura/Triple Dirk	F_1, F_2, $F_{2:3}$	F_2, $F_{2:4}$, $F_{2:5}$, BC_1F_2	$F_{5:6}$ (SHD[z])	$F_{4:5}$, $F_{5:6}$[y] (SHD)	$F_{2:5}$
Laura/RL5407	F_1, F_2, $F_{2:3}$	F_2, $F_{2:4}$, $F_{2:5}$, BC_1F_2	$F_{4:5}$ (SHD)	$F_{4:6}$ (SSD)	$F_{2:5}$
Genesis/SK0263	F_1, $F_{2:3}$	F_2, $F_{2:4}$	–	$F_{4:5}$ (SHD)	–

[z]SHD = single head derived, SSD = single seed derived.
[y]$F_{5:6}$ for 1997 is a different set of SHD from that for 1996–1997 winter as two heads from each line were randomly harvested in F_5 generation in Summer 1996.

Spelt wheat is considered a valuable genetic source of desirable genes (Schmid and Winzeler 1990), such as disease resistance genes. Knowledge of the nature and number of genes in resistant sources facilitates disease resistance breeding by allowing better estimates of population size and the most effective generation to start selection. Past genetic studies on the inheritance of common bunt resistance, in various crosses, showed that its genetic control was due to a single gene (Smeltzer 1952; Metzger and Silbaugh 1971; Singh and Chopra 1986; Goates 1996) or two genes (Schaller et al. 1960; Metzger et al. 1979). The study by Knox et al. (1998) on common bunt resistance reported a major gene in both doubled haploid and random inbred populations. The gene action of the common bunt resistance can be dominant (Waud and Metzger 1970; Metzger and Silbaugh 1971; Metzger et al. 1979; Singh and Chopra 1986) or intermediate (Smeltzer 1952; Knox et al. 1998).

Fifteen common bunt (*Bt*) resistance genes have been identified in wheat (Goates 1996) and 10 of them (*Bt*1–10) were used in a study for identifying races present in western Canada (Gaudet and Puchalski 1989a). However, 7 (*Bt*1-4,6,7,9) of the 10 genes can be overcome by the combined virulence from races of *T. tritici* and *T. laevis* (Gaudet and Puchalski 1989a). Thus, to battle against this disease, identification of new resistance sources and information on the genetic control of common bunt resistance are needed for utilizing available resistance genes.

Therefore, to facilitate incorporating the bunt resistance into our wheat breeding program, this study was conducted to determine the genetic control for bunt resistance in a common wheat and two spelt wheat cultivars.

MATERIALS AND METHODS

The common wheat cultivar Triple Dirk and spelt wheat cultivars RL5407 and SK0263, all resistant to common bunt in preliminary testing, were crossed with susceptible cultivars Laura and Genesis. The crosses Laura/Triple Dirk, Laura/RL5407 and Genesis/SK0263 were used to produce 80–84 F_2 plants and to further generate 80–84 F_2-derived $F_{2:3}$ lines and 29–50 $F_{2:4}$ and $F_{2:5}$ lines (Table 1). The $F_{4:5}$ and $F_{5:6}$ lines for crosses Laura/Triple Dirk and Laura/RL5407 were single-head derived lines (SHD), which were produced from randomly chosen $F_{2:4}$ and $F_{2:5}$ lines, respectively, by randomly sampling a single head in each line. The BC_1-derived F_2 (BC_1F_2) lines were also created from individual plants of the BC_1F_1 generation for the back crosses Laura × (Laura × Triple Dirk) and Laura × (Laura × RL5407) (Table 1).

Three bunt races were used in this study including T1 and T13 of *T. tritici* and L7 of *T. laevis*. Race T1 was used as inoculum in all crosses from 1995 to 1997 since it had been found to be the least virulent race in preliminary study (Hoffmann and Metzger 1976) and thus was expected to identify the greatest range of resistance in the resistant sources used in this study. Races T13 and L7 were each used separately in 1996 to test the parental and $F_{2:5}$ generations of the crosses Laura/Triple Dirk and Laura/RL5407 to determine the genetic control of resistance to these two races and to allow comparison with the results obtained when race T1 was used.

In 1995, the parents, F_1, F_2 and $F_{2:3}$ lines of each of the crosses Laura/Triple Dirk, Laura/RL5407 and Genesis/SK0263 were tested in the field in separate experiments using a randomized complete block design (RCBD) with two replications. For each cross, 80 to 84 $F_{2:3}$ lines and eight plots of each of the F_1, F_2, and parental generations were grown. Except for the $F_{2:3}$ generation, the plots were hill-planted on 46-cm centers. Each plot contained 20 seeds of each parent, four to six seeds of the F_1 generation or 40 seeds of the F_2 generation. For the purpose of separating individual plants, each of the $F_{2:3}$ lines was seeded on a square surrounding the 46-cm center with 15 seeds on each of the four corners, totaling 60 seeds for each line. The experiments were carried out at the North Seed Farm, University of Saskatchewan, Saskatoon, SK, in a field where bunt tests had never been conducted. Seeds were planted 5–8 cm deep and the day/night soil temperatures during the planting period were within the optimum range of 5 to 15°C for bunt infection.

In 1996, the experiments involved the parents, F_2, 40 and 50 $F_{2:4}$ lines, 30 and 29 $F_{2:5}$ lines, and 11 and 35 BC_1F_2 progenies of the crosses Laura/Triple Dirk and Laura/RL5407, respectively. The parental, F_2 and $F_{2:4}$ (50 lines) generations of the cross Genesis/SK0263 were also tested. As in 1995, each experiment included eight plots of the parental and F_2 generations. All plots were hill-planted on 46-cm centers and one hill represented one plot. About 40 seeds were seeded in each plot of the F_2, $F_{2:4}$, $F_{2:5}$ and BC_1F_2 generations and 20 seeds for each plot of the parents. The experimental design was a RCBD with two replications.

Seeds of each treatment were dusted with about 0.8 g of teliospores of the appropriate bunt race and excess inoculum was removed by shaking the seeds on a fine mesh sieve. After inoculation with each race, the mesh sieve, the sampling spoon and the working counter were all completely cleaned and sterilized using about 1.0% sodium hypochloride solution. Seed samples were planted within 24 h of

Table 2. Breeding scheme, genetic expectation and suggested segregation ratios for one- and two-gene models based on the grouping of (resistant + heterozygous) : susceptible

Generation	Breeding scheme	Genetic expectation	Segregation ratio
		One-gene model	
$P_1 \times P_2$	Crossing	rr × RR	NA[z]
F_1	Bulked hybrids	Rr	NA
F_2	Bulked from F_1 plants	1/4RR + 1/2Rr + 1/4rr	3(R+H) : 1S
$F_{2:3}$	F_2 single-plant derived	1/4RR:1/2(1/4RR, 1/2Rr, 1/4rr):1/4rr	3(R+H) : 1S
$F_{2:4}$	Bulked from $F_{2:3}$ line	1/4RR:1/2(3/8RR, 1/4Rr, 3/8rr):1/4rr	3(R+H) : 1S
$F_{2:5}$	Bulked from $F_{2:4}$ line	1/4RR:1/2(7/16RR, 1/8Rr, 7/16rr):1/4rr	3(R+H) : 1S
$F_{4:5}$ (SHD)	Single-head derived from $F_{2:4}$ line	7/16RR:1/8(1/4RR, 1/2Rr, 1/4rr):7/16rr	9(R+H) : 7S
$F_{5:6}$ (SHD)	Single-head derived from $F_{2:5}$ line	15/32RR:1/16(RR, Rr, rr):15/32rr	17(R+H) : 15S
$F_{4:6}$ (SSD)	Single-seed descent from $F_{2:4}$ line	15/32RR:1/16(1/4RR, 1/2Rr, 1/4rr):15/32rr	17(R+H) : 15S
BC_1F_2	Single-plant derived from BC_1F_1	1/2Rr : 1/2rr	1(R+H) : 1S
		Two-gene model	
$P_1 \times P_2$	Crossing	$r_1r_1r_2r_2 \times R_1R_1R_2R_2$	NA
F_1	Bulked hybrids	$R_1r_1R_2r_2$	NA
F_2	Bulked from F_1 plants	$9/16R_1\_R2\_ + 3/16R_{1\_}\, r_2r_2 + 3/16r_1r_1R_{2\_} + 1/16r_1r_1r_2r_2$	15(R+H) : 1S
$F_{2:3}$	F_2 single-plant derived	$15/16\ (R_{1\_}R_{2\_}, R_{1\_}\, r_2r_2, r_1r_1R_{2\_}, r_1r_1r_2r_2)$[y] : $1/16\ r_1r_1r_2r_2$	15(R+H) : 1S
$F_{2:4}$	Bulked from $F_{2:3}$ line	$15/16\ (R_{1\_}R_{2\_}, R_{1\_}\, r_2r_2, r_1r_1R_{2\_}, r_1r_1r_2r_2) : 1/16\ r_1r_1r_2r_2$	15(R+H) : 1S
$F_{4:5}$ (SHD)	Single-head derived from $F_{2:4}$ line	$207/256(R_{1\_}R_{2\_}, R_{1\_}\, r_2r_2, r_1r_1R_{2\_}, r_1r_1r_2r_2) : 49/256\ r_1r_1r_2r_2$	207(R+H) : 49S

[z]NA = not applicable.
[y]Indicating all possible plant genotypes within a plot.

inoculation. At maturity, individual plants within each plot were pulled out and separated, and the spikes of each plant were smashed to check for bunt infection. Plants with at least one bunted kernel were rated as susceptible. Disease incidence of a plot was calculated as the percentage of susceptible plants to the total number of plants in that plot.

During the winter 1996/1997, seed of the parents and 27 $F_{2:5}$ single head-derived (SHD) $F_{5:6}$ lines of cross Laura/Triple Dirk was inoculated with race T1 and planted for disease evaluation in a RCBD with two replications in a growth cabinet. One pot of each $F_{5:6}$ line, with 12 to 15 seeds per pot, plus one pot of each parent were seeded in each block. Plants were grown in six-inch pots filled with Redi-Earth potting mix (W.R. Grace & Co. of Canada Ltd., Ajax, ON) and each pot was fertilized with about 5 g of the controlled release fertilizer OSMOCOTE PLUS™ 16-8-12 (Scotts-Sierra, Horticultural Products Company, Maryville, OH) after seeding. The plants were grown with an 18-h photoperiod and a temperature regime modified from that of Gaudet and Puchalski (1989b): 8°C/6°C day/night temperature for the first 3 wk, then 14°C/12°C from the 4th week until heading, and 23°C from heading until mature. In a second experiment, 60 $F_{4:5}$ SHD lines and two parents of cross Laura/RL5407 were planted in a growth cabinet for disease testing under the same growth conditions. Each $F_{4:5}$ line was seeded at 12 to 15 seeds per pot. Disease rating was based on individual spikes in each pot instead of individual plants because of the difficulty of separating individual plants due to the compact roots in the pot. The proportion of the number of diseased spikes to the total number of spikes in a pot was recorded as the disease incidence for that treatment.

Since most of the disease incidence ratings were continuously distributed, no discrete segregation patterns could be observed. To establish phenotypic groups for Mendelian analysis, the lowest value of the susceptible parent was used as the dividing point between susceptible and the other classes. The dividing point is based on the disease infection of the susceptible parent as this gives greater certainty of correspondence between phenotype and genotype, since disease escapes are more clearly identified than if the resistant parent data were used (Briggs 1940; Kornegay et al. 1993; Singh et al. 1995). However, unusual observations can occur in the susceptible parent distribution, due, for example, to disease escape. Using the lowest value of the susceptible parent biases the dividing towards resistance and inflates the range used to classify progenies as susceptible. Thus, the observations for the susceptible parent in each experiment were tested for extreme values or outliers before phenotypic grouping for Mendelian analysis. The Dixon's test was used for detecting the extreme values or outlying observations (Dixon 1953; Grubbs 1969). The criterion

$r = (x_2 - x_1)/(x_{n-1} - x_1)$ $(x_1<x_2<\ldots<x_{n-1}<x_n$, where x_1, x_2, …, x_n were observations, i.e. bunted %, for the susceptible parent)

is used to see if the smallest value is suspect. The ratio r was calculated and compared with the critical table values corresponding to different levels of P values for tests of significance. For instance, to test for extreme values among eight parental observations, the calculated criterion r was compared with the table values of 0.554 and 0.683 at the probability levels of 0.05 and 0.01, respectively. We tested for outliers in all experiments and used the lowest value of the homogeneous susceptible parent as the dividing point. In order to verify if this method of classification was correct, a P value was obtained using the following F test to ascertain whether the lines in a specific group were homogeneous or heterogeneous as expected. The value

$F = MS_{line}/MS_{error}$ (MS = mean square)

Table 3. Distribution of mean plot disease incidence in the experiments on bunt resistance to race T1 in the two crosses Laura/Triple Dirk and Laura/RL5407 conducted in the field in 1995 and 1996

	Midpoint value of bunt incidence (%)																
Generation	0	5	10	15	20	25	30	35	40	45	50	55	60	65	70	M ± SE	Total
Laura/Triple Dirk (1995–1996)																	
1995																	
P_r[z]	7	1														0.31 ± 0.88	8
F_1		1	2	1	2	0	1	0	1							19.30 ± 11.74	8
F_2			1	1	0	2	2	1	0	1						27.09 ± 10.66	8
$F_{2:3}$	7	11	11	7	8	4	10	11	5	3	4	1	0	1	1	22.89 ± 16.59	84
P_s							1	2	2	2	1					39.51 ± 6.96	8
1996																	
P_r		5	2	1												9.16 ± 4.84	8
F_2						2	1	2	1	0	1	1				36.74 ± 10.55	8
$F_{2:4}$		2	1	3	6	3	4	3	6	5	2	3	2			33.13 ± 14.99	40
$F_{2:5}$	1	1	2	1	3	3	3	3	4	3	2	4				32.86 ± 15.17	30
BC_1F_2						2	1	1	0	0	3	0	2	1	1	46.04 ± 11.77	11
P_s									1	2	2	2	1			47.32 ± 10.01	8
Laura/RL5407 (1995–1996)																	
1995																	
P_r	8															0.00 ± 0.00	8
F_1	4	0	2	1	0	1										6.98 ± 11.82	8
F_2	1	0	3	1	2	0	0	1								14.56 ± 9.31	8
$F_{2:3}$	15	24	9	11	6	3	2	5	1	0	0	3	0	0	1	13.37 ± 14.67	80
P_s					1	3	1	1	0	1	1					32.62 ± 12.19	8
1996																	
P_r		1	2	4	1											13.55 ± 4.72	8
F_2					1	1	0	3	2	0	1					34.65 ± 8.32	8
$F_{2:4}$	2	4	3	8	1	2	4	7	2	5	3	3	4	2		32.20 ± 19.37	50
$F_{2:5}$	1	1	1	2	1	3	1	2	5	3	2	2	3	2		37.99 ± 17.34	29
BC_1F_2			1	2	2	4	3	4	3	7	4	3	2			38.08 ± 13.37	35
P_s									1	2	1	2	1	1		52.83 ± 8.60	8

[z]P_r = Triple Dirk or RL5407, P_s = Laura.

with the degrees of freedom of (Line – 1) and (Rep – 1) (Line – 1), respectively.

For genetic analysis, the breeding schemes and the genetic expectation for the suggested segregation ratios for one- and two-gene models are listed in Table 2. For the goodness-of-fit tests, the grouped data in each of the segregating populations were used to test against the expected Mendelian ratios (Table 2). Since these tests involved only two classes, susceptible (S) versus the rest [i.e. resistant (R) + heterozygous (H)], Yate's correction for continuity was used to calculate adjusted χ^2 values (Steel and Torrie 1997).

In 1996, in order to test the independence of resistance to different bunt races, the seeds of $F_{2:5}$ lines for the two crosses, Laura/Triple Dirk (30 lines) and Laura/RL5407 (29 lines), were inoculated separately with each of the three races, i.e., T1 and T13 of *T. tritici* and L7 of *T. laevis*. Each of the cross × race combinations was arranged in a separate test with the same experimental design, i.e., RCBD with two replications. Eight parental plots were included for each of the two parents in each block. After the disease test, each of the same individual lines in different tests was classified into either (R + H) or S group based on its bunt reaction to each of the three bunt races. Then, a χ^2 test was used to test the independence of bunt reaction to two races in a 2 × 2 contingency table in order to determine if the gene resistant to one race was the same as the one resistant to another race of common bunt (Table 8).

RESULTS AND DISCUSSION

The frequency distributions of disease incidence on a per-plot basis were continuous for all experiments (Tables 3–5), except for the $F_{5:6}$ SHD lines of the cross Laura/Triple Dirk, which was discontinuous (Fig. 1). Past studies indicate that common bunt reaction could easily be affected by environmental conditions, such as soil temperature and moisture, soil type and seeding depth, etc., and by the amount of inoculum load on seeds (Reed 1928; Gaudet and Puchalski 1989b; He 1999), which made it difficult to obtain discrete segregation especially when tested in the field. In addition, all the parents except Genesis in three populations had a higher disease incidence in 1996 than in 1995 (Tables 3 and 4). The higher bunt infection in 1996 more likely resulted from more favorable environmental condition, i.e., lower temperature and higher moisture. In order to minimize the error of misclassification due to disease escape (Metzger et al. 1979), the phenotypic classification in this study was based on the susceptible parent (Tables 3–5). This is because a line with as high a disease rating as the susceptible parent is most likely a susceptible line, but a line with a low disease rating could be either a genetically resistant line or a line from the non-resistant groups resulted from disease escape. However, other researchers used the parental means (m) plus or minus the standard deviations (σ) to cut the continuous distributions of the segregating progenies into resistant or susceptible

Table 4. Distribution of mean plot disease incidence in the experiments on bunt resistance to race T1 in the cross Genesis/SK0263 conducted in the field in 1995 and 1996

	Midpoint value of bunt incidence (%)																						
Generation	0	5	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95	100	M ± SE	Total
Genesis/SK0263 (1995–1996)																							
1995																							
P_r [z]	6	1	1																			1.41 ± 2.97	8
F_1				1	1	0	0	3	0	2	0	1										35.83 ± 12.84	8
$F_{2:3}$	1	0	4	5	10	5	6	4	10	12	8	3	4	1	4	1	1	1				38.41 ± 18.43	80
P_s														1	2	1	0	1	2	0	1	80.18 ± 13.10	8
1996																							
P_r		2	3	2	0	1																12.46 ± 6.28	8
F_2									2	1	0	3	2									51.24 ± 7.50	8
$F_{2:4}$	1	1	0	0	3	2	1	6	4	12	3	9	4	4								43.56 ± 14.71	50
P_s										1	1	2	2	1	1							56.78 ± 9.59	8

[z] P_r = SK0263, P_s = Genesis.

Table 5. Frequency distribution of mean plot disease incidence in the experiments on bunt resistance to race T13 and L7 in the two crosses Laura/Triple Dirk and Laura/RL5407 conducted in the field in 1996

	Midpoint value of bunt incidence (%)																					
Generation	5	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95	100	M ± SE	Total
Laura/Triple Dirk																						
With T13																						
P_r [z]	1	3	1	2	0	0	1														12.46 ± 6.70	8
$F_{2:5}$					2	2	0	2	2	0	4	8	3	3	1	2	0	1			56.77 ± 15.66	30
P_s														2	1	0	2	2	1		83.51 ± 7.21	8
With L7																						
P_r	1	1	2	1	2	0	1														19.15 ± 8.03	8
$F_{2:5}$	1	1	0	0	0	1	2	3	3	1	3	4	2	3	2	3	1				54.48 ± 19.39	30
P_s												1	2	2	2	1					69.52 ± 6.73	8
Laura/RL5407																						
With T13																						
P_r	1	0	4	0	1	0	1	0	1												21.07 ± 13.41	8
$F_{2:5}$			1	0	0	3	1	0	1	4	1	3	6	3	1	5					58.87 ± 17.88	29
P_s															1	0	2	2	1	2	90.28 ± 6.87	8
With L7																						
P_r			2	1	2	1	2														23.90 ± 6.28	8
$F_{2:5}$	1	0	1	1	1	2	2	1	2	2	1	5	2	0	1	3	3	0	1		54.26 ± 23.48	29
P_s													3	1	0	2	2				75.13 ± 8.71	8

[z] P_r = Triple Dirk or RL5407, P_s = Laura.

(Griffey and Das 1994). Based on the computer simulation that we did, this method of classification can be affected, to a great extent, by the value of σ (He 1994, unpublished data). Especially, for common bunt, the disease reaction can vary substantially, resulting in a large σ, under different environmental conditions (Reed 1928; Gaudet and Puchalski 1989b; He 1999). Accordingly, we classified the segregating progenies using the actual distribution of the susceptible parent and tested the extreme observations, prior to the phenotypic grouping, within the susceptible parent distribution to ascertain the correctness of the classification for all the experiments of disease evaluation for resistance to three bunt races.

Resistance to Race T1

Laura/Triple Dirk

Since the distribution of bunt incidence to race T1 in the cross Laura/Triple Dirk was continuous (Table 3), the lowest bunt incidence of the susceptible parent was used as the cut-off point for phenotypic grouping. No extreme observation was found ($P > 0.30$, data not included), which suggests that there were no significant outlying data points. Thus, the lowest infection rating of the susceptible parent was used to divide lines of the continuously distributed segregating generations into two groups, i.e. susceptible (S) and resistant + heterozygous (R + H) (Tables 3–5), for genetic analysis (Tables 6 and 7).

Assuming that there was one major gene controlling bunt resistance to race T1 in Triple Dirk, the expected segregation would be that the F_2 single-plant derived lines fit a 3 (resistant + heterozygous):1 (susceptible) ratio (Table 2). After the χ^2 tests, the segregation ratios for the F_2-derived lines in the F_3, F_4 and F_5 generations of the cross Laura/Triple Dirk, all fit a 3 (R + H) : 1 (S) ratio (Table 6). These data indicate that a single major gene likely con-

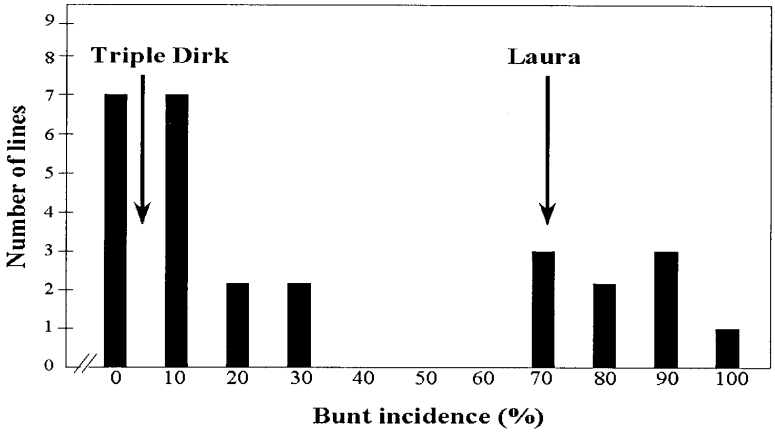

Fig. 1. Distribution of common bunt incidence for $F_{2:5}$ single-head derived $F_{5:6}$ lines of the cross Laura/Triple Dirk tested in the greenhouse to race T1 in 1997. (Left-most and right-most arrows indicate the resistant and susceptible parents, respectively.)

Table 6. Segregation for resistance to bunt races T1, T13 and L7 in the crosses Laura/Triple Dirk, Laura/RL5407 and Genesis/SK0263 in field tests conducted in 1995 and 1996

Cross	Generation	Observed no. R + H[z]	Observed no. S	Observed no. Total	Ratio tested	χ^2	*P* value
				Race T1			
Laura/Triple Dirk	$F_{2:3}$ (1995)	58 (0.00[y])	26 (0.37)	84	3 : 1	1.29	0.26
	$F_{2:4}$ (1996)	26 (0.05)	14 (0.85)	40	3 : 1	1.63	0.20
	$F_{2:5}$ (1996)	18 (0.04)	12 (0.95)	30	3 : 1	2.84	0.09
	BC_1F_2 (1996)	4 (0.48)(H)	7 (0.94)	11	1 : 1	0.36	0.55
Laura/RL5407	$F_{2:3}$ (1995)	59 (0.05)	21 (0.14)	80	3 : 1	0.02	0.89
	$F_{2:4}$ (1996)	33 (0.01)	17 (0.71)	50	3 : 1	1.71	0.19
	$F_{2:5}$ (1996)	17 (0.00)	12 (0.74)	29	3 : 1	3.32	0.07
	BC_1F_2 (1996)	19 (0.79)(H)	16 (0.99)	35	1 : 1	0.11	0.74
Genesis/SK0263	$F_{2:3}$ (1995)	72 (0.04)	8 (0.76)	80	15 : 1	1.33	0.25
	$F_{2:4}$ (1996)	26 (0.05)	24 (0.92)	50	15 : 1	141.48	<0.01
				Race T13			
Laura/Triple Dirk	$F_{2:5}$ (1996)	26 (0.01)	4 (0.13)	30	3 : 1	1.60	0.21
Laura/RL5407	$F_{2:5}$ (1996)	24 (0.02)	5 (1.00)	29	3 : 1	0.56	0.45
				Race L7			
Laura/Triple Dirk	$F_{2:5}$ (1996)	18 (0.09)	12 (0.68)	30	3 : 1	2.84	0.09
Laura/RL5407	$F_{2:5}$ (1996)	19 (0.00)	10 (0.38)	29	3 : 1	0.93	0.34

[z]Progenies with bunt incidence within the susceptible parent range were classified as susceptible (S); the rest were classified as resistant + heterozygous (R + H).
[y]The *P* value used to test for homogeneity of progenies within each phenotypic class (R + H or S).

trolled resistance in Triple Dirk. In addition, all *P* values ($P \geq 0.05$) for the homogeneity tests for the susceptible group showed non-significance, suggesting that the individual lines in the susceptible group were homogeneous; in the mean time, the *P* values for the R + H groups were small and significant for the F_2-derived lines ($F_{2:3}$, $F_{2:4}$, $F_{2:5}$) for resistance to race T1 ($P \leq 0.05$), indicating heterogeneity. This is consistent with the individuals within this group being genetically different. Therefore, this method of grouping based on the susceptible parent was appropriate for genetic analysis in this study (Table 6). In addition, the nature of heterogeneity for the R + H group also provides evidence that complete dominance of resistance to bunt was absent for resistance to race T1. In the meantime, lack of dominance can also be seen from the mean of F_1 infection rate of 19.30% that was between the resistant (0.31%) and the susceptible (39.51%) parents in the cross Laura × Triple Dirk (Table 3). This also agrees with the results of Knox et al. (1998) in which F_1 plants demonstrated intermediate resistance between the parents. The lack of dominance found for the resistance in the materials of this study can facilitate breeding for bunt resistance by simply selecting the highly resistant individuals as they will breed true and will not segregate in the progenies.

In addition, if the one-gene-control hypothesis stands, the backcross progenies should segregate in 1 (heterozygous) : 1 (susceptible) ratio. The test result showed that this one-gene hypothesis was confirmed by the fit of the 1:1 segregation ratio in BC_1-derived F_2 lines ($P = 0.55$). The two groups, i.e., the heterozygous and homozygous susceptible, in the BC_1F_2 generation should also show homogeneity. After analysis of variance of these two groups, the homogeneity test, indicated

Table 7. Segregation for resistance to bunt race T1 in single head/seed derived lines of three crosses tested in the field and in the greenhouse

Generation	Observed number R + H[z]	Observed number S	Observed number Total	Ratio tested	χ^2	*P* value
1997 Field Tests						
				Laura/Triple Dirk		
$F_{4:5}$ (SHD)	41 (0.05[y])	29 (0.98)	70	9 : 7	0.07	0.79
$F_{5:6}$ (SHD)	17 (0.57)	9 (0.12)	26	17 : 15	1.12	0.29
				Laura/RL5407		
$F_{4:6}$ (SSD)	35 (0.21)	24 (0.12)	59	17 : 15	0.68	0.41
				Genesis/SK0263		
$F_{4:5}$ (SHD)	35 (0.99)	45 (0.96)	80	9 : 7	4.59	0.03
				207 : 49	68.80	<0.01
1996–1997 Greenhouse Tests						
				Laura/Triple Dirk		
$F_{5:6}$ (SHD)	18 (0.30)	9 (0.14)	27	17 : 15	1.48	0.22
				Laura/RL5407		
$F_{4:5}$ (SHD)	41[x]	19	60	9 : 7	3.09	0.08

[z]Progenies with bunt incidence within the susceptible parent range were classified as susceptible (S); the rest were classified as resistant + heterozygous (R + H).
[y]*P* value used to test homogeneity of progenies within each phenotypic class (R + H or S).
[x]Test for homogeneity was not possible due to lack of replication.

by the large *P* values, showed homogeneity. The non-significant heterogeneity in the heterozygous group was expected for one-gene control of resistance in the BC_1F_2, since all progenies in that group would have the same genotype (*Rr*).

Moreover, for the hypothesis of a single-gene model for bunt resistance, segregation of the progenies should also fit a 9 (resistant + heterozygous) : 7 (susceptible) ratio for the $F_{4:5}$ SHD lines and a 17 (resistant + heterozygous) : 15 (susceptible) ratio for the $F_{5:6}$ SHD lines (Table 2). The χ^2 analysis indicated that segregation of the $F_{4:5}$ SHD lines, tested in the field, fit a 9 (resistant + heterozygous) : 7 (susceptible) ratio ($P = 0.79$) expected for one-gene model (Table 7). This result was consistent with that for the F_2-derived lines discussed previously (Table 6). Additional support for one-gene control of resistance in Triple Dirk was obtained both from the growth chamber test and from the field test in 1997 of the $F_{5:6}$ SHD lines where the segregation ratio consistently fit the expected 17:15 ratio (Table 7). The distribution of the disease incidence data for resistance to race T1 was discontinuous (Fig. 1), as would be expected for single gene segregation in the $F_{5:6}$ lines. The reason for the discontinuous segregation was because, first, the frequency of the two homozygous genotypes is high (94%); and second, only a small number of lines were tested in the growth chamber, resulting in that the expected very few heterozygotes were not sampled for disease testing. In addition, a big gap of separation for the $F_{5:6}$ SHD lines in the cross Laura/Triple Dirk as shown in Fig. 1 also resulted from the fact that plants tested in the growth chamber for bunt reaction may express maximum phenotypic differential, due to a more uniform and controlled environment than when tested in the field. Similarly, Knox et al. (1998) reported better separation of lines segregating for a single gene when grown in the growth chamber compared to the field. Furthermore, unlike the F_2 derived lines (Table 6), most of the *P* values of the homogeneity test for the (R + H) groups in the F_4- and F_5-derived (SHD or SSD) lines were unexpectedly large (Table 7). This could be caused (1) by sampling error where the heterozygous plants were not randomly picked for disease test due to their smaller proportion (6%) than that (50%) in the F_2-derived lines and small sample sizes of 26–80 lines, (2) by disease escape in bunt reaction for the heterozygous lines and/or (3) by other unknown reasons.

Table 8. Test of gene independence for resistance to races T1, T13 and L7 of common bunt of wheat in $F_{2:5}$ lines of the crosses Laura/Triple Dirk and Laura/RL5407

	Laura/Triple Dirk		Laura/RL5407	
	T13	L7	T13	L7
T1	0.01 (0.92)[z]	1.41 (0.24)	0.16 (0.69)	0.06 (0.81)
T13		0.04 (0.84)		8.24 (<0.01)

[z]χ^2 (*P* value).

Laura/RL5407

The χ^2 analysis showed that the F_2 single plant derived progenies including $F_{2:3}$, $F_{2:4}$ and $F_{2:5}$ lines of the cross Laura/RL5407 all segregated in a 3:1 ratio and the BC_1F_2 lines segregated in a 1:1 ratio, indicating that resistance was controlled by a single major gene (Tables 2 and 6). This hypothesis was supported by segregation of F_4 single seed derived (SSD) $F_{4:6}$ lines, which fit a 17:15 ratio (Table 7). The distribution of disease incidence for resistance to bunt race T1 for the $F_{4:5}$ SHD lines was bimodal (Fig. 2), which is expected for the segregation of progenies with monogenic inheritance, and its segregation fit a 9 (resistant + heterozygous) : 7 (susceptible) ratio (Table 7). However, this distribution of bunt incidence to race T1 for the cross Laura/RL5407 in Fig. 2 did not show discontinuity as Fig. 1 for the cross Laura/Triple Dirk. This is because the latter had a higher proportion of homozygotes for the $F_{5:6}$ lines in

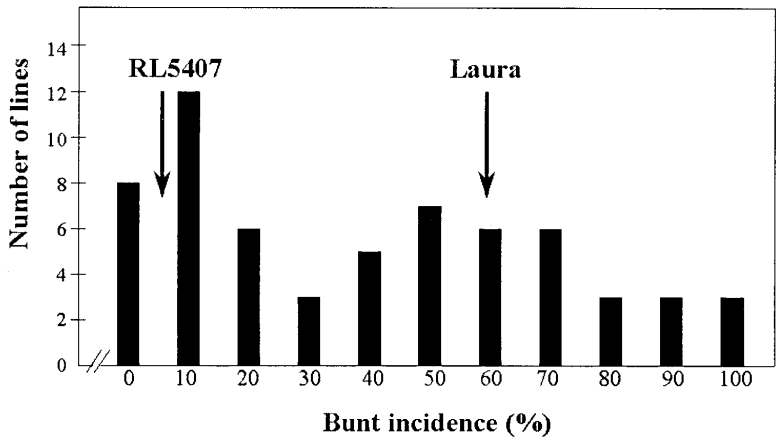

Fig. 2. Distribution of common bunt incidence for $F_{2:4}$ single-head derived $F_{4:5}$ lines of the cross Laura/RL5407 tested in the greenhouse to race T1 in 1997. (Left-most and right-most arrows indicate the resistant and susceptible parents, respectively.)

Laura/Triple Dirk than the former for the $F_{4:5}$ lines in Laura/RL5407. Second, the number of lines tested for bunt incidence to race T1 was greater for the $F_{4:5}$ SHD lines (60) in the cross Laura/RL5407 (Fig. 2) than for the $F_{5:6}$ lines (27) in the cross Laura/Triple Dirk (Fig. 1). These results are consistent with a number of previous studies, in which different individual bunt races were used as inoculum, which reported single gene resistance to common bunt in common wheat (Smeltzer 1952; Waud and Metzger 1970; Metzger and Silbaugh 1971; Singh and Chopra 1986; Goates 1996). In disease resistance breeding, single gene resistance such as the one from spelt wheat can be easily incorporated into adapted wheat cultivars that have desirable agronomic traits but are susceptible to the disease.

Although F_1 bunt incidence (6.98%) implied possible partial dominance for the resistance to bunt, the F_2 data in both 1995 and 1996 suggested no dominance (Table 3). However, the low bunt incidence in F_1 could result from the sampling error since sample size in F_1 was smaller (about five plants/plot) than that of F_2 (40 plants/plot). Therefore, selection of lines highly resistant to common bunt is possible in the population by taking advantage of non-dominant attribute for bunt resistance.

Genesis/SK0263

If the genetic control for bunt resistance in SK0263 was due to two major genes, then the F_2-derived progenies, i.e., $F_{2:3}$ and $F_{2:4}$ lines, should segregate in a ratio of 15(R + H): 1(S) (Table 2). Table 6 showed that the segregation ratio for $F_{2:3}$ lines of cross Genesis/SK0263 fit a two-gene model, for a ratio of 15:1. However, the segregation of $F_{2:4}$ lines did not fit either a one-gene or a two-gene model because of an excess of susceptible lines (Table 6). In addition, segregation of $F_{4:5}$ SHD lines of Genesis/SK0263 did not fit either a one-gene (9:7) or a two-gene (207:49) model ($P < 0.05$) (Table 7). This poor fit for the cross Genesis/SK0263 might be due to unusual disease development (Parker and Hooker 1993). The susceptible parent Genesis showed lower bunt incidence in 1997 (55.8%) and 1996 (56.8%) than in 1995 (80.2%), whereas bunt incidence of the resistant parent SK0263 was much higher in 1997 (26.5%) and 1996 (12.5%) than in 1995 (1.41%). The cause of these inconsistencies in bunt incidence between the parental generations is not clear. However, genotype × environment interaction could change the relative degree of disease infection among the different genotypes (Reed 1928; Gaudet and Puchalski 1989b) or differences in inoculum load could affect the chances of exposure of seeds to disease infection. The inconsistent results in different generations could also result from misclassification of genotypes (Clarke et al. 1994). But misclassification due to disease escape in the cross Genesis/SK0263 was unlikely since an excess number of progenies were rated as susceptible in both the $F_{2:4}$ (Table 6) and the $F_{4:5}$ (SHD) generations (Table 7). This higher-than-expected frequency of susceptible lines could also be caused by sampling error and/or natural selection against the resistance locus because of a detrimental pleiotropic effect or because of close linkage to an unfavorable locus.

Although using the lower end of bunt incidence from the susceptible parent as the cut-off point could minimize misclassification, inconsistent bunt reaction in the susceptible parent could affect the classification of the segregating progenies. Therefore, since the segregation of $F_{2:4}$ and $F_{4:5}$ (SHD) lines for the cross Genesis/SK0263 did not fit expected ratios due to excess of susceptible lines (Tables 6 and 7), the good fit of the $F_{2:3}$ lines to a 15:1 ratio does suggest that SK0263 likely possesses two genes controlling bunt resistance to race T1.

Resistance to Race T13

All of the plant materials including the parents and progenies had higher bunt incidence to race T13 (Table 5) than race T1 tested in 1996 (Table 3 and 4). None of the $F_{2:5}$ lines showed zero infection to race T13 for the two crosses Laura/Triple Dirk and Laura/RL5407. The midpoint values of bunt incidence for the $F_{2:5}$ lines ranged from 25 to 90% for the cross Laura/Triple Dirk and from 15 to 80% for the cross Laura/RL5407 (Table 5), which were higher than

those for the tests inoculated with race T1 (Tables 3 and 4). This result of different infection rate indicated that race T13 is more virulent than race T1 as race T1 was shown to be the least virulent race among the six bunt races, including T1, T6, T13, T19, L7 and L16, tested in the three experiments for the race-specificity resistance in 1994 (He 1999).

Segregation of the $F_{2:5}$ lines of crosses Laura/Triple Dirk and Laura/RL5407 fit a 3 (resistant + heterozygous) : 1 (susceptible) segregation ratio, suggesting monogenic control of resistance to race T13 in Triple Dirk and RL5407 (Table 6). In addition, the segregation of the $F_{2:5}$ lines for resistance to race T13 in the cross Laura/Triple Dirk fit not only a 15:1 ratio but also a 3:1 ratio. For a test of a 3:1 against 15:1 ratios, the minimum number of lines required is about 40 at the α level of 0.05 (Mather 1938; Hanson 1959), therefore, the number of lines (29–30) tested is on the small side. Nevertheless, the better fit to a 3:1 ratio ($P = 0.21$) suggests that Triple Dirk may possess one gene for resistance to race T13. Furthermore, for both crosses Laura/Triple Dirk and Laura/RL5407, the lines were homogeneous in the susceptible group but heterogeneous for the resistant + heterozygous group, confirming that these classifications were appropriate, even for the limited number of lines.

Resistance to Race L7

Similar to the tests for race T13, the two parents and the $F_{2:5}$ lines all had higher bunt incidence to race L7 (Table 5) than to race T1 tested in 1996 (Tables 3 and 4). Both resistant parents Triple Dirk and RL5407 showed slightly higher bunt incidence to race L7 than to race T13; however, the susceptible parent Laura had lower bunt incidence to race L7 than to race T13. This is consistent with the results of the test for race-specificity resistance involved the *Bt* genes (*Bt*1 – *Bt*10) and Laura, where Laura had a higher average bunt incidence to race T13 (90.1%) than to race L7 (86.6%) (He 1999).

In genetic analysis, the progenies of the $F_{2:5}$ generation in the cross Laura/Triple Dirk segregated in a 3 (R + H) : 1 (S) ratio for resistance to race L7 (Table 6). But the *P* value (0.09) for the homogeneity test in the (R + H) group was higher than the expected value of ≤0.05 as progenies of the cross Laura/Triple Dirk in the (R + H) group were expected to be heterogeneous for the resistance to race L7. This might be the result of a sampling error when only a limited number of $F_{2:5}$ lines were randomly sampled for disease evaluation; in other words, the segregating lines were not sampled due to the small number available. Likewise, the segregation of $F_{2:5}$ lines in the cross Laura/RL5407 also fit a 3:1 ratio, demonstrating that bunt resistance to race L7 was due to a single major gene.

In addition, although the three resistant parents, i.e., Triple Dirk, RL5407 and SK0263, showed different bunt incidence to three races (Tables 3–5), the study on race specificity of common bunt resistance in a split-plot design with four replications demonstrated that all of the three cultivars were uniformly resistant to the six races (T1, T6, T13, T19, L7 and L16) (He 1999). Triple Dirk, RL5407 and SK0263 showed an average incidence of 17, 10 and 15.4%, respectively, while the susceptible cultivar Laura had an average incidence of 70.5, 78.5 and 74.9%, respectively, in three different tests. Moreover, these six bunt races were used to form a mixture of inoculum for evaluating breeding lines in the cooperative tests of wheat cultivars in western Canada (Gaudet and Puchalski 1989b). Thus, identification of bunt resistance genes in the three cultivars employed in this study is practically useful for incorporating the genes into breeding lines to fight the bunt races prevalent in western Canada.

Gene Independence Tests

The resistance genes identified in RL5407 and Triple Dirk were tested for independence by determining if the disease reactions of the progenies to one race were independent of the reactions to the other races. Lack of independence would indicate the same gene conditioned resistance to two different races. For each race, the progenies were classified into two groups, i.e., susceptible and heterozygous + resistant, in a 2×2 contingency table constructed for each paired-race combination and the data were tested for independence using the χ^2 test.

From the χ^2 test, it was found that bunt reactions of the $F_{2:5}$ progenies of Laura/Triple Dirk were independent in all paired-race combinations (Table 8). Thus, Triple Dirk is hypothesized to carry different genes conferring resistance to each of the races T1, T13 and L7. However, the $F_{2:5}$ progeny reactions for the cross Laura/RL5407 were not independent for resistance to races T13 and L7 ($P < 0.01$, Table 8), suggesting that the genes identified in RL5407 for resistance to these two races were either the same gene or were closely linked. In previous genetic studies, different resistance genes, such as *Bt*8 (Waud and Metzger 1970) and *Bt*10 (Metzger and Silbaugh 1971), were found each to be resistant to many different bunt races. For the purpose of breeding for resistance, either a single gene or a cluster of linked genes conferring resistance to two or more races, is a valuable genetic resource. The gene carried by RL5407 that controls resistance to races T13 and L7 was not linked with the gene conferring resistance to race T1 (Table 8). However, the relationship of these genes to the named *Bt* genes (Schaller et al. 1960; Waud and Metzger 1970; Metzger and Silbaugh 1971; Metzger et al. 1979; Goates 1996) is still unknown.

In conclusion, we investigated the genetic control of common bunt resistance by testing different populations including the F_2 single plant derived, backcross F_1 derived, F_4/F_5 derived (SHD or SSD) populations, and found that the cultivar Triple Dirk appeared to carry three major genes for common bunt resistance; each of them conferred resistance to each of the three races T1, T13 and L7. The spelt wheat RL5407 possibly carried a single major gene or closely linked genes for resistance to both races T13 and L7 and another major gene for resistance to race T1. In addition, bunt incidence in the F_1 and F_2 generations also suggested that the resistance to race T1 was due to additive gene action in these two crosses. Therefore, selection of lines highly resistant to common bunt by incorporating the resistance from Triple Dirk and RL5407 should be effective in the wheat breeding program due to lack of dominance and the one- or two-gene controlled resistance.

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