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E. C. K. Pang · G. M. Halloran

The genetics of blackleg [*Leptosphaeria maculans* (Desm.) Ces. et De Not.] resistance in rapeseed (*Brassica napus* L.).

I. Adult-plant resistance in F_2 and first-backcross populations

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Abstract The genetic control of adult-plant blackleg [*Leptosphaeria maculans* (Desm.) Ces. et De Not.] resistance in rapeseed (*Brassica napus* L.) was studied in the F_2 and first-backcross populations of the cross "Maluka" (blackleg-resistant) × "Niklas" (highly susceptible). A *L. maculans* isolate possessing high levels of host specificity (MB2) was used in all inoculations. Resistance/susceptibility was evaluated using three separate measures of crown-canker size, i.e. the percentage of crown girdled (%G), external lesion length (E) and internal lesion area (%II). Disease severity scores for the F_2 and first-backcross populations based on E and %II gave discontinuous distributions, indicating major-gene control for these measures of resistance; but those for %G were continuous, indicating quantitative genetic control for this measure. Chi-square tests performed on the (poorly-defined) resistance classes, based on E, in the F_2 and first-backcross populations indicated the likelihood for resistance being governed by a single, incompletely dominant major gene. Although the distributions of the F_2 and first-backcross populations, based on%II, were clearly discontinuous, the observed segregation ratios for resistance and susceptibility did not fit any of the numerous Mendelian ratios which were considered. Differences in inheritance of resistance according to the assessment method and blackleg isolate used, were discussed.

Key words Genetics · Adult-plant · Blackleg resistance · *Brassica napus* · *Leptosphaeria maculans* · Australian cultivar

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E. C. K. Pang (✉) · G. M. Halloran
Joint Centre for Crop Improvement, Faculty of Agriculture, Forestry and Horticulture, The University of Melbourne, Parkville, Victoria 3052, Australia

Introduction

Most of the available information on the inheritance of blackleg (*Leptosphaeria maculans*) resistance in rapeseed (*Brassica napus*) was reviewed recently by Rimmer and van den Berg (1992). These authors noted that although blackleg is the most important disease of rapeseed worldwide, and that much effort is being expended on breeding for resistance, there is a paucity of published data concerning the inheritance of blackleg resistance. The existence of significant host pathogen interactions, resulting from the inoculation of a number of *B. napus* cultivars with ascospores and pycnidiospores of *L. maculans* (Thurling and Venn 1977; Cargeeg and Thurling 1979, 1980; Cargeeg 1980) indicated that resistance could be determined by the combined effects of major and minor genes. Studies by Delwiche (1980) indicated that resistance to cotyledon-lesion development was possibly under the control of two dominant genes. Sawatsky (1989) found that adult-plant resistance in two spring rapeseed lines (R83-14 and R83-17) to a Manitoba *L. maculans* isolate was governed by two dominant, complementary, genes. RFLP mapping of adult-plant blackleg resistance in doubled-haploid (DH) lines (grown in a number of environments) from a cross between the resistant line "Cresor" and the susceptible cultivar "Westar" indicated that 72% of the variation was accounted for by a single, major, QTL (Dion et al. 1995). A second QTL, detected in only one environment, accounted for a further 8% of the variation.

The aim of the present study was to analyse the F_2 and first-backcross populations of crosses between blackleg-resistant and -susceptible parents for possible monogenic or oligogenic (Mendelian) control of blackleg resistance. A subsequent study (Pang and Halloran 1996) will attempt to analyse blackleg resistance as a metric (quantitative) trait.

Materials and methods

Fungal cultures

A single-ascospore isolate of *L. maculans*, designated MB2, was used in all inoculations. A previous study on virulence specificity in *L. maculans* (Pang and Halloran 1995) showed that the virulence specificity of MB2 was high. The consequences, in terms of the effects of isolate virulence specificity on the inheritance study, will be discussed later.

B. napus genotypes, experimental populations and cultural conditions

A set of reciprocal crosses were made between the Australian blackleg-resistant cultivar "Maluka" and the susceptible Swedish cultivar, "Niklas". The parental plants had been grown under natural daylength in a glasshouse maintained at 25°C (day) and 15°C (night), at the School of Agriculture, Forestry and Horticulture, the University of Melbourne, Parkville. A total of 20 F_1 populations were produced. Selfed seeds from the parents of these F_1 populations were pre-germinated in Petri dishes on filter paper wetted with a 0.05% w/v Benlate (benomyl, Dupont) suspension and subsequently transferred to 17-cm-diameter pots containing a soil-peat-sand mix. Seedlings were thinned to four per pot 2 weeks after transfer. The parental lines were infected via the wound-inoculation technique described previously (Pang and Halloran 1995), at growth stage 2 3–2.4 of the Harper and Berkenkamp scale (1975). The parental lines were assessed for blackleg resistance/susceptibility 5 weeks after inoculation. External lesion length (E) and the percentage of stem girdled (%G) were used as indicators of disease severity.

As the result of these assessments, a single F_1 population (with "Maluka" as the female parent) was selected from the initial crosses, based on the wide separation between the resistance/susceptibility scores of its parents. Thirty to forty plants of the selected F_1 population were selfed to produce the F_2, and were additionally backcrossed to both the resistant and susceptible parents to produce the BCP_1 (to "Maluka") and BCP_2 (to "Niklas") populations.

Plants from the parental, F_1, F_2 and first-backcross populations were grown in a glasshouse under a regime of 15°C (night) and 25°C (day), with a 11–12 h photoperiod. Seedlings were thinned to four per pot 10 days after germination. A week prior to inoculation, the pots were randomly allocated among the benches in the glasshouse. To investigate the possibility of maternal inheritance of blackleg resistance/susceptibility, 16 plants from each of four other F_1 populations, in which "Maluka" was alternately used as the male or female parent, were grown concurrently with plants from the six populations above and were inoculated and assessed for blackleg resistance. However, only one disease severity measure, external lesion length (E), was used to gauge crown-canker development in these F_1 populations.

Infection procedure and disease severity assessment

The preparation of inoculum, the application of pycnidiospores and the wound-inoculation procedure were as described previously (Pang and Halloran 1995). Plants from the six populations were inoculated at growth stage 2.4–2.5 on the scale devised by Harper and Berkenkamp (1975). To encourage infection, transparent polythene hoods were placed over benches containing inoculated plants for 3 days to maintain high humidity.

Assessment of stem-canker development was conducted at 5 weeks after inoculation. The measurements made were: external lesion length (E), the percentage of stem girdled (%G) and the percentage of internal infection (%I). No transformations were performed on the data, as the coefficients of variation and the standard errors (of the means) for the six populations indicated that the variances were uncorrelated with their respective means.

Genetic analysis

The additive, dominance, and environmental components of variation, together with the broad- and narrow-sense heritability values for blackleg resistance were estimated by the following equations, from Mather and Jinks (1971):

$$D = 4V_{F2} - 2(V_{BCP1} + V_{BCP2})$$

$$H = 4(V_{BCP1} + V_{BCP2} - V_{F2} - E_w)$$

$$E_w = \frac{1}{4}(V_{P1} + V_{P2} - V_{F1})$$

$$h^2_{ns} = \frac{\frac{1}{2}D}{\frac{1}{2}D + \frac{1}{4}H + E_w}$$

$$h^2_{bs} = \frac{\frac{1}{2}D + \frac{1}{4}H}{\frac{1}{2}D + \frac{1}{4}H + E_w}$$

where,

D =additive component
H =dominance component
E_w=environmental component
h^2_{ns}=narrow-sense heritability
h^2_{bs}=broad-sense heritability.

The limitations of these equations for estimating the genetic and environmental components of variation from the variances of experimental populations were discussed by Mather and Jinks (1971). With only four equations to estimate D, H, and E_w, there was no scope for calculating the standard deviations of the estimates of the three components; nor was it possible to test for the goodness-of-fit of the additive-dominance model.

Chi-square tests were used to gauge the goodness-of-fit of observed segregation ratios for blackleg resistance in the F_2 and first-backcross populations to various Mendelian ratios, based on the hypotheses of a one-, two- or three-gene control of resistance, with varying combinations of interaction, epistasis and dominance.

Results

The mean external lesion lengths (E) for the two pairs of F_1 populations differed significantly between, but not within, the pairs (Table 1). This indicated the likelihood that the inheritance of blackleg resistance, as measured by E, was not affected by cytoplasmic factors. Further, the disparity between the two sets of F_1 means indicated that the parental cultivars used may not have been homozygous for blackleg resistance/susceptibility.

Table 1 Means and standard errors for blackleg resistance, based on external lesion length (E), for two pairs of F_1 populations, derived from reciprocal crosses between "Maluka" (P_1) and "Niklas" (P_2). L.S.D. $_{(a=0.05)}$=3.68, with 63 *df*

F_1 population	"Maluka" parent[a]	External lesion length (E) (mm)
M3F	F	10.9±0.69
M3M	M	9.5±1.43
M4F	F	20.4±2.82
M4M	M	17.4±2.96

[a] F="Maluka" used as female parent; M="Maluka" used as male parent

Crown-canker development gauged by the percentage of crown girdled (%G)

The frequency distributions of blackleg disease severity for %G, showed that both parents were highly variable for this trait (Fig. 1). This high variability, which led to a significant overlap between the two distributions, may indicate either genetic heterogeneity for blackleg resistance, or the sensitivity of this trait to the environment. The mean disease severity for the susceptible parent, "Niklas", was only twice as large as that of the resistant parent, "Maluka". The mean disease severity of the F_1 population was 26% less than the mid-parent value (34.8%), indicating the likelihood of dominance for blackleg resistance (%G) (Fig. 1).

There was a high degree of overlap between the distributions of the two first-backcross populations for%G. The mean of the backcross to the resistant parent, "Maluka" (BCP_1), was slightly larger than the F_1 mean (Fig. 1). This result was inconsistent with the indication of strong dominance at loci controlling blackleg resistance observed in F_1 plants. This inconsistency was most likely due to bias in the small sample of F_1 plants used, but a much larger number of plants was used to estimate the BCP_1 mean. The mean of the backcross to the susceptible parent, "Niklas" (BCP_2), was almost equal to that of its recurrent parent, indicating the absence, or very low levels, of dominance for blackleg resistance, which did not accord with the F_1 observations.

There were no clear discontinuities in the F_2 distribution (Fig. 1), indicating that blackleg resistance, as measured by %G, may be inherited as a quantitative character. Despite the absence of marked discontinuities, which may have signified major-gene segregation, the distribution of the F_2 population was not strictly unimodal. At least two possible (poorly defined) resistance classes were present, their modes occurring in the 30 and 40% classes (Fig. 1). On the premise that these two possible resistance classes represented actual sub-populations of resistant and susceptible plants within the F_2, and that the distributions of these populations were, respectively, highly negatively and positively skewed, the number of plants in the "resistant" subpopulation would then comprise 117 individuals with 83 individuals in the "susceptible" population, which fits a ratio of 9 resistant: 7 susceptible (χ^2=0.41; P=0.50–0.75) This would indicate the presence of two unlinked complementary genes with blackleg resistance expressed only when the dominant alleles of both genes were present at each locus. Despite the close fit of the 9 resistant: 7 susceptible ratio to the F_2 data, a comparison of the expected segregation ratios based on the two-gene hypothesis (1:0 for BCP_1; 1:3 for BCP_2) with observed ratios in the BCP_1 and BCP_2 indicated that the segregation patterns in these two populations were not adequately explained by the two-gene hypothesis (χ^2=5.43 ; P=0.010–0.025, for the BCP_2 comparison). The failure of the first-backcross data to corroborate the two-gene hypothesis may indicate that the observed phenotypic classes in the F_2 were merely artifacts of the scale used. Further, even if the observed classes represented actual sub-populations of resistant and susceptible plants within the F_2, the possible heterozygosity of both parents for blackleg resistance may render it difficult to accurately assign Mendelian ratios to the segregation patterns in this population.

Table 2 Genetic variances and heritabilities for blackleg resistance derived from the analysis of parental, F_1, F_2 and first-backcross progeny of the cross between "Maluka" (resistant) and "Niklas" (susceptible)

Genetic parameters	Disease severity measures		
	% G	E	% II
D	254.88	882.47	324.00
H	117.56	2264.70	2606.31
E_w		167.02	312.03
V_A	127.44	441.24	162.00
V_D	29.39	566.17	651.58
V_E	230.08	167.02	312.03
V_P	386.91	1174.43	1125.61
h^2_{gs} (%)	32.9	37.6	14.4
h^2_{bs} (%)	40.5	85.8	72 3
$\sqrt{H/D}$ [a]	0.68	2.57	2.84

[a] $\sqrt{H/D}$ = dominance ratio

With the assumption that the additive-dominance model adequately describes the genetics of blackleg resistance (for %G), the standard analysis outlined by Mather and Jinks (1971) revealed that the major portion of the heritable variation for resistance was due to additive genetic effects (Table 2). This indicated that there is a high potential for improving the level of blackleg resistance, as most of the genetic variation is fixable by selection. The moderately high narrow- (32.9%) and broad- (40.5%) sense heritabilities, coupled with the observation of possible phenotypic classes within the F_2, indicated the likelihood of oligogenic control of resistance.

Crown-canker development gauged by the external lesion length, E

Using E, the parental populations were found to be less variable for blackleg resistance, than when using the %G in gauging crown-canker development (Fig. 2). The mean disease severity for the susceptible parent, "Niklas" was over three-times greater than that of the resistant parent. There was little overlap between the two parental distributions. The P_1 ("Maluka") distribution, however, contained a few outliers in the 70-mm class. The mean disease severity of the F_1 population was 42% less than the mid-parent value (32.05 mm), indicating strong dominance for blackleg resistance (Fig. 2). Further, the distribution of the F_1 population strongly resembled that of the resistant parent, indicating the likelihood of dominance for resistance.

There was a substantial overlap between the distributions of the resistance scores of the first-backcross popu-

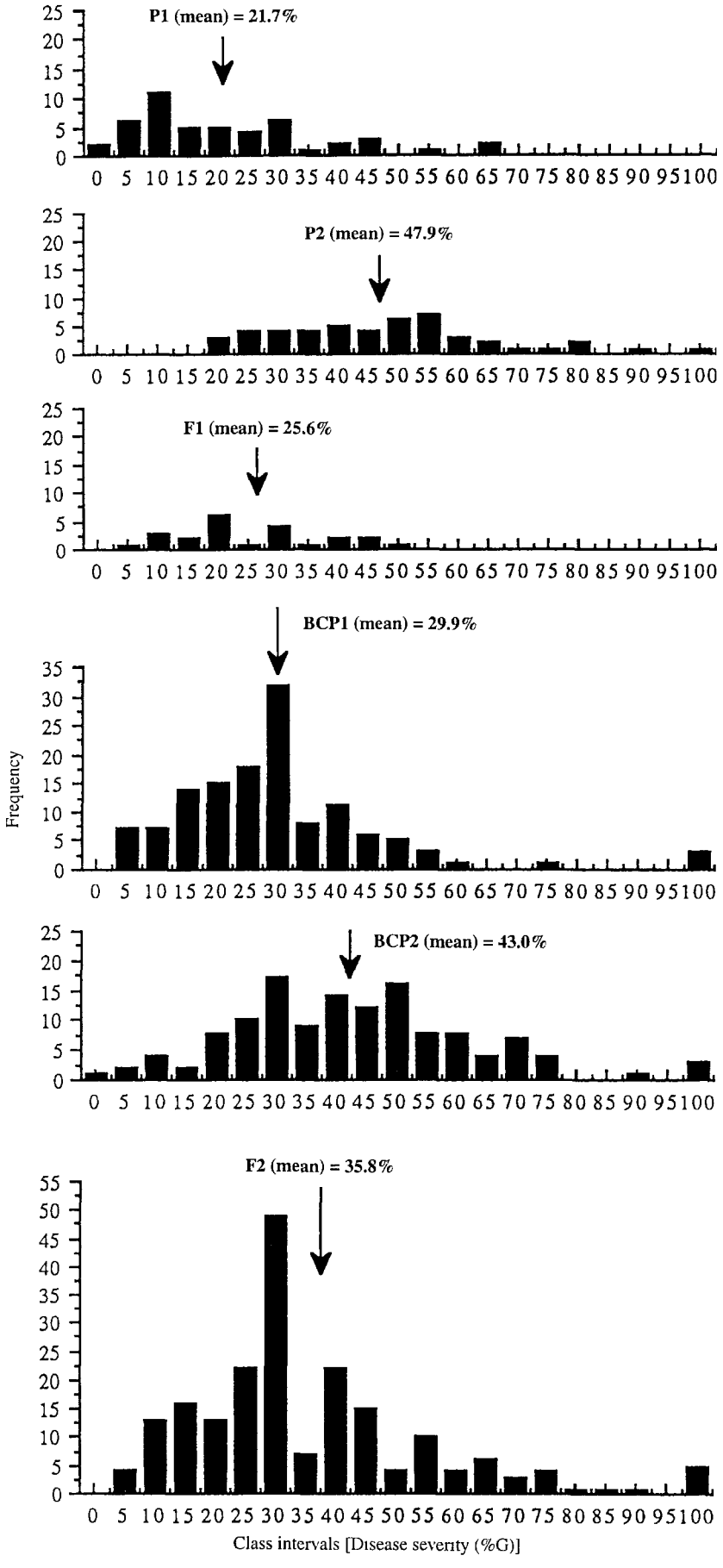

Fig. 1 Frequency distributions of blackleg resistance (based on %G) for parental, F_1, F_2 and first-backcross populations of "Maluka" (P_1)×"Niklas" (P_2)

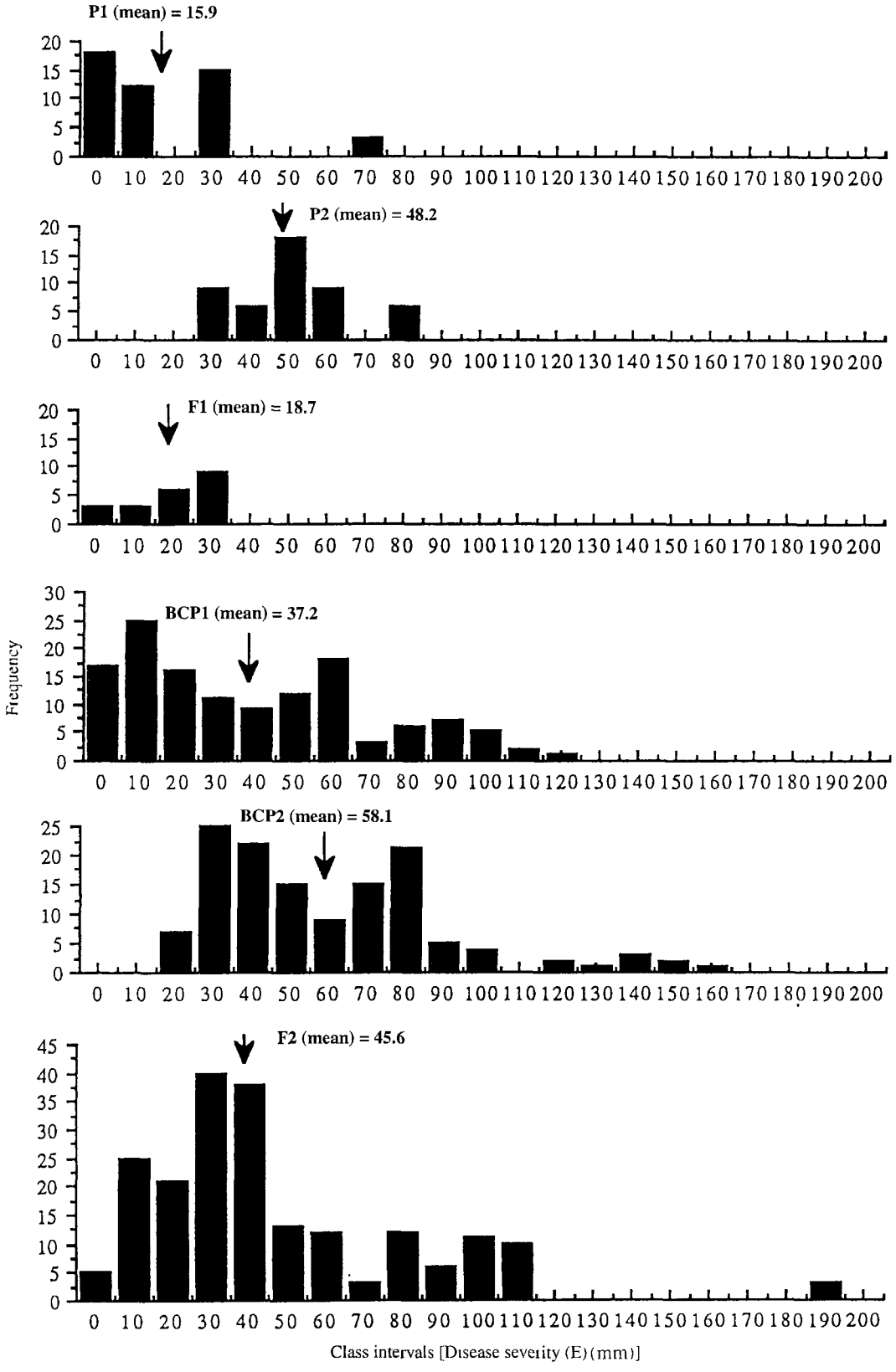

Fig. 2 Frequency distributions of blackleg resistance (based on E) for parental, F_1, F_2 and first-backcross populations of "Maluka" (P_1)×"Niklas" (P_2)

lations. The modes of the BCP_1 distribution were 10 mm and 60 mm, while those of the BCP_2 were 30 mm and 80 mm. There was a marked absence of individuals in the 0.0-mm and 10-mm classes in the BCP_2 population. The bimodality in both the BCP_1 and BCP_2 distributions, cou-

Table 3 Chi-square test of a one-gene hypothesis for segregation for blackleg resistance (using external lesion length, E) in the F_2 and first-backcross populations of "Maluka" (P_1)×"Niklas" (P_2)

Phenotype	Genotype[a]	F_2 ratio		BCP_1 ratio		BCP_2 ratio	
		Observed	Expected	Observed	Expected	Observed	Expected
Resistant	*AA*	51	49.8	58	64.5	0	0
Intermediate	*Aa*	103	99.5	50	64 5	69	66
Susceptible	*aa*	45	49.8	21[b]	0.0	63	66
	Total	199		129		132	
	χ^2	0.615 (P=0.50–0.75)		3.914 (P=0.025–0.05)		0.279 (P=0.50–0.75)	

[a] *AA*=Lesion lengths from 0–25.0 mm, *Aa*=25.5–65.0 mm, *aa*=65.5–200 mm
[b] "Susceptible" outliers

pled with indications from F_1 data of strong dominance at loci controlling blackleg resistance, indicated major-gene control of this trait.

Although the segregation pattern for the F_2 population indicated the likelihood for three resistance classes, there were no marked discontinuities in the distribution for blackleg resistance. The three modes of the F_2 distribution were at 10, 30 and 80 (or 100) mm, resembling those found previously in the BCP_1 and BCP_2 distributions. Based on these modes, the F_2 may be subdivided into three phenotypic classes – (1) "Resistant", (2) "Intermediate" and (3) "Susceptible". The intervals used to delineate these classes were: 0.0–25.0 mm, 25.5–65.0 mm and 65.5–200 mm. The resultant division of the F_2 into 51 "Resistant": 103 "Intermediate": 45 "Susceptible" conformed to a 1R:2I:1S ratio (Table 3). A chi-square test of this hypothesis (Table 3) indicated no significant differences (α=0.05) between the observed and expected ratios in the F_2 and BCP_2 populations. However, the observed segregation ratios in the BCP_1, based on the selected group intervals, were significantly different from expected. This disparity was due to the presence of a number of "susceptible" outliers (Fig. 2; Table 3). The tentative conclusion is that the expression of external lesion length, induced by the *L. maculans* isolate MB2, is controlled by a single, incompletely dominant major gene. However, the absence of clear discontinuities in the F_2 and first-backcross distributions suggested that the effects of this gene may be modified by that of other genes.

Crown-canker development gauged by internal infection, %II

The frequency distributions of blackleg disease severity, using%II, showed that both parental populations were highly variable for blackleg resistance (Fig. 3). The mean disease severity for the F_1 population was slightly less than that for the resistant parent, indicating strong dominance at loci controlling blackleg resistance.

The distributions of the BCP_1 and BCP_2 populations overlapped completely (Fig. 3), with strong indications of modality within these distributions. In BCP_1 they were at 5, 35 and 70%, with a large proportion of plants also falling into the 100% class. The high peaks in the 100% class, for the BCP_1, BCP_2 and F_2 distributions were probably due to difficulties in accurately measuring internal lesion sizes in excess of 80% of the crown transverse area. Hence, most of the internal lesion sizes in excess of 80% were assigned to the 100% class. Modes in the BCP_2 were at 5, 35 and 75%, in close correspondence with those of the BCP_1. The F_2 distribution was clearly discontinuous (Fig 3), with the observed modes in close correspondence with those of the first-backcross populations. The observed segregation ratios in the F_2 and first-backcross populations, however, did not fit any of the Mendelian ratios which were considered.

The apparent discontinuities in the distributions of the P_1, P_2, BCP_1, BCP_2 and F_2 indicated that blackleg resistance, as measured by %II, may be oligogenically controlled. This indication was supported by the high broad-sense heritability for this trait (Table 2). The narrow-sense heritability was disproportionately smaller than the broad-sense value, which reflected strong dominance/epistasis at loci for this trait.

Discussion

The findings of the present study indicated that resistance to canker formation, following wound inoculation with pycnidiospores of the virulent *L. maculans* isolate, MB2, may be oligogenically controlled. Cargeeg (1980) and Cargeeg and Thurling (1980) proposed, from observations of significant host×pathogen interactions in the *L. maculans* – *B. napus* pathosystem, that resistance to blackleg may be determined by the combined effects of major and minor genes. This type of genetic control of resistance has been reported for the maize (*Zea mays*) – northern leaf blight (*Cochlibolus carbonum*) pathosystem by various workers (Ullstrup and Brunson 1947; Leonard 1974; Hamid et al. 1982). In the family Brassicaceae, the resistance of radish (*Raphanus sativus*) to white rust (*Albugo candida*) is controlled by a single, dominant major gene, the action of which is modified by environmentally sensitive minor genes (Williams and Pound 1963). RFLP mapping and concurrent Mendelian inheritance studies using doubled-hap-

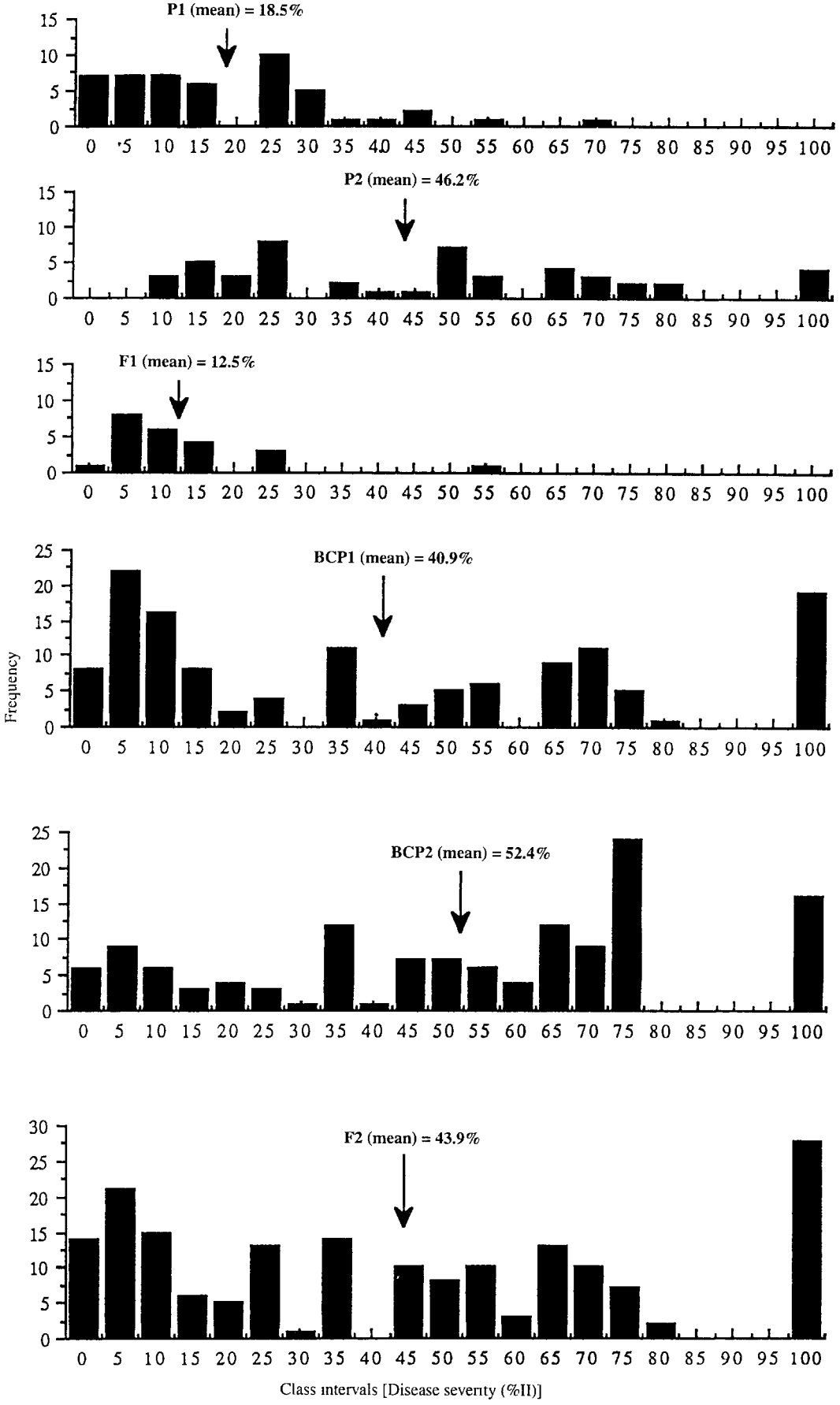

Fig. 3 Frequency distributions of blackleg resistance (based on%II) for parental, F_1, F_2 and first-backcross populations of "Maluka" (P_1)×"Niklas" (P_2)

loid lines, conducted by Dion et al. (1995), indicated that adult-plant blackleg resistance in the cultivar "Cresor" may be controlled by a single, dominant major gene. However, the results are inconclusive in that the segregation ratios for resistance:susceptibility did not fit the model for single-gene inheritance (39R:59S, χ^2=11.44).

The apparent oligogenic control of crown-canker development may have depended on the particular isolate used. The isolate MB2 was found in a previous study (Pang and Halloran 1995) to possess high levels of specific virulence. In particular, the host specificity of this isolate, as with a few other isolates, was with the clone-line "NCII", which was derived from a single plant randomly selected from the F_2 population of "Maluka"×"Niklas", the latter being used in the present study. According to Flor's (1971) gene-for-gene hypothesis, alleles conditioning resistance/susceptibility in a host may be detected only when the corresponding alleles for virulence/avirulence are present in the isolate(s) of the pathogen used for infection. Under this hypothesis, the use of MB2 may have allowed the detection of major genes for resistance within the populations derived from "Maluka"×"Niklas". A previous study (Pang and Halloran 1995) indicated that two-thirds of the *L. maculans* isolates examined possessed non-specific, rather than specific, virulence. If such non-specific virulence is governed polygenically, the results of the present study may have indicated a quantitative mode of inheritance for resistance to crown-canker development, had an isolate with high levels of non-specific virulence been used instead of MB2. The duality of the genetic basis of host resistance to different strains/races of the pathogen within a single pathosystem is not uncommon. In the maize – northern leaf blight pathosystem, resistance to races 1 of *C. carbonum* is conditioned by a single dominant gene (Ullstrup and Brunsun 1947), whereas resistance to races 2 and 3 may be under polygenic control (Leonard 1974; Hamid et al. 1982). In the *B. napus* – *L. maculans* pathosystem, the genetic control of blackleg resistance in cultivars such as "Maluka" may thus possess both a qualitative (major-gene) and quantitative component, as proposed earlier by Cargeeg (1980) and Cargeeg and Thurling (1980).

In the present study, blackleg resistance was gauged by three traits (measures) representing various dimensions of the canker. This use of multiple measures of a single trait (crown-canker development) clearly indicated that the outcome of a genetic study is highly dependent on the choice of measurements used. The expression of the first trait, %G, was found to be mostly under additive genetic control. The occurrence of (poorly defined) resistance classes in the F_2 and first-backcross distributions indicated that this trait may be under oligogenic control. Possible oligogenic control of resistance, together with moderate broad- and narrow-sense heritabilities, indicated that selection should be effective in improving resistance to crown-canker, as measured by %G. The expression of the second trait, external lesion length (E), was found to be possibly, under the control of a single, incompletely-dominant, major gene. The results obtained were equivocal, due to the significant departure of the BCP_1 ratios from expected. The confirmation of the one-gene hypothesis would have been best conducted by analysing the segregation patterns of the F_3 and subsequent generations. This is currently underway. The expression of the third trait, percentage of internal infection (%II), was found to possess a large dominance/epistasis component. The segregation patterns in the F_2 and first-backcross generations for this trait indicated the likelihood of oligogenic control of resistance. This could not be confirmed from F_2 data due to possible heterozygosity of the parents for this trait.

Taken together, the results of the present study indicated that the expression of crown-canker development in the F_2 and backcross progeny of "Maluka"×"Niklas" possessed both a major- and minor- (quantitative) gene component. This result is by no means conclusive, and the possibility that blackleg resistance is governed purely by genes, each of small effect (minor genes or polygenes), cannot be precluded. A further paper (Pang and Halloran 1996) will examine blackleg resistance as a quantitative trait.

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References

Cargeeg LA (1980) Host-pathogen relationships of the blackleg disease of rape (*Brassica napus* and *Brassica campestris*) caused by *Leptosphaeria maculans*. PhD Thesis, University of Western Australia

Cargeeg LA, Thurling N (1979) Seedling and adult plant resistance to blackleg [*Leptosphaeria maculans* (Desm.) Ces. et de Not.] in spring rape (*Brassica napus* L.) Aust J Agric Res 30:37–46

Cargeeg LA, Thurling N (1980) Contribution of host- pathogen interactions to the expression of the blackleg disease of spring rape (*Brassica napus* L.) caused by *Leptosphaeria maculans* (Desm.) Ces. et de Not. Euphytica 29:465–476

Delwiche PA (1980) Genetic aspects of blackleg (*Leptosphaeria maculans*) resistance in rapeseed (*Brassica napus*). PhD thesis, University of Wisconsin-Madison

Dion Y, Gugel RK, Rakow GFW, Séguin-Swartz G, Landry BS (1995). RFLP mapping of resistance to the blackleg disease [causal agent, *Leptosphaeria maculans* (Desm.) Ces. et de Not.] in canola (*Brassica napus* L.). Theor Appl Genet 91:1190–1194

Flor HH (1971) Current status of the gene-for-gene concept. Annu Rev Phytopathol 9:275–296

Hamid AH, Ayers JE, Hill RR Jr (1982) Host isolate interactions in corn inbreds inoculated with *Cochliobolus carbonum* race 3. Phytopathology 72:1169–1173

Harper FR, Berkenkamp B (1975) Revised growth stage key for *Brassica campestris* and *B. napus*. Can J Plant Sci 55:657–658

Leonard KJ (1974) Genetic isolation of races 2 and 3 of *Cochliobolus carbonum* in North Carolina (abstract) Proc Am Phytopathal Soc 1:94

Mather K, Jinks JL (1971) Biometrical genetics: the study of continuous variation. 2nd edn. Chapman and Hall, Great Britain

Pang ECK, Halloran GM (1995) Adaptability and virulence specificity in Australian strains of blackleg [*Leptosphaeria maculans* (Desm.) Ces. et De Not.] on different host genotypes of rapeseed (*Brassica napus* L.). Aust J Agric Res 46:971–984

Pang ECK, Halloran GM (1996) The genetics of blackleg [*Leptosphaeria maculans* (Desm.) Ces. et De Not.] resistance in rapeseed (*Brassica napus* L.). II. Seedling and adult-plant blackleg resistance as quantitative traits. Theor Appl Genet 93:941–949

Rimmer SR, van den Berg CGJ (1992) Resistance of oilseed *Brassica* spp. to blackleg caused by *Leptosphaeria maculans*. Can J Plant Pathol 14:56–66

Sawatsky WM (1989) Evaluation of screening techniques for resistance to *Leptosphaeria maculans* and genetics of resistance in *Brassica napus*. MSc thesis, University of Manitoba, Winnipeg, Manitoba

Thurling N, Venn LA (1977) Variation in the responses of rapeseed (*Brassica napus and Brassica campestris*) cultivars to blackleg (*Leptosphaeria maculans*) infection. Aust J Exp Agric Anim Husb 17:445–451

Ullstrup AJ, Brunson AM (1947). Linkage relationships of a gene in corn determining susceptibility to a *Helminthosporium* leaf spot. J Am Soc Agron 39:606–609

Williams PH, Pound GS (1963). Nature and inheritance of resistance to *Albugo candida* in radish. Phytopathology 53:1150–1154