CANADIAN JOURNAL OF PLANT PATHOLOGY 16:163–169, 1994

Genetic relationships between reaction to common root rot and yield in the progeny of a barley cross

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The heritability of resistance to common root rot, caused by *Cochliobolus sativus*, in 125 progeny (F6-F8) of a cross between barley cultivars Argyle (resistant) and Melvin (tolerant) was 56.7% ± 4.1 and the minimum number of genes by which the two parents differed for the expression of disease severity was three. The heritability of yield for the same population was 35.3 % ± 3.8, and the number of loci estimated was at least three. There was a greater range in yield and disease expression in the Peace region of Alberta than at Saskatoon, Saskatchewan. At sites in the Peace from 1988 to 1990, the means of some progeny exceeded those of the parents; this did not occur at Sasktoon. Cluster analysis grouped the progeny into three classes; 45% of the lines were resistant (similar to Argyle), 45% tolerant (similar to Melvin), and 10% were susceptible. The rank of lines for disease reaction was generally similar for most resistant and tolerant progeny at both locations. The regression of grain yield on disease severity with all progeny was negative ($y = -4.9\ X + 1625$ at $P = 0.01$) but the correlation was weak ($R = -0.24$). This relationship was stronger when the tolerant group of lines was removed from the regression ($R = -0.53$, $P < 0.001$). The relationship between yield and disease among the resistant and tolerant lines was positive ($y = 5.7\ X + 1136$, $R = 0.28$, $P < 0.01$), yet a within-class correlation between yield and disease in the tolerant group indicated a negative association ($R = -0.34$, $P < 0.01$). This study suggests that a breeding strategy for developing lines resistant to common root rot, while maintaining high yield, should involve screening to eliminate the highly susceptible lines from crosses and then selecting the best yielding lines with the lowest disease.

Bailey, K.L., and R.I. Wolfe. 1994. Genetic relationships between reaction to common root rot and yield in the progeny of a barley cross. Can. J. Plant Pathol. 16: 163–169.

L'héritabilité de la résistance à la pourriture sèche, causée par le *Cochliobolus sativus*, d'une descendance de 125 lignées (F6-F8) issue d'un croisement entre les cultivars d'orge Argyle (résistant) et Melvin (tolérant) était de 56,7 % ± 4,1 et le nombre de gènes minimal permettant de différencier les deux parents dans leur manifestation de la gravité de la maladie était de trois. L'héritabilité du rendement de cette même population était de 35,3 % ± 3,8, et le nombre estimé de loci était d'au moins trois. Il y avait des écarts plus grands de rendement et de gravité de la maladie dans la région de Peace en Alberta qu'à Saskatoon en Saskatchewan. Les moyennes de quelques lignées excédaient celles des parents dans certains sites de Peace de 1988 à 1990; ceci ne s'était pas manifesté à Saskatoon. L'analyse de grappe a permis de grouper la descendance en trois classes; 45 % des lignées étaient résistantes (semblables à Argyle), 45 % tolérantes (semblables à Melvin), et 10 % étaient sensibles. L'échelle de gravité de la maladie de la descendance était généralement semblable pour la plupart des lignées résistantes et tolérantes aux deux localités. Il y avait un coefficient de régression négatif du rendement en grain par rapport à la gravité de la maladie dans toute la descendance ($y = -4,9\ X + 1625$ à $P = 0,01$), mais une faible corrélation ($R = -0,24$). Ce rapport était plus fort quand le groupe de lignées tolérantes a été retiré de la régression ($R = -0,53$, $P < 0,001$). Le rapport rendement et maladie des lignées résistantes et tolérantes a été positif ($y = 5,7\ X + 1136$, $R = 0,28$, $P < 0,01$), pourtant une corrélation à l'intérieur des classes rendement et maladie du groupe des tolérants a démontré une association négative ($R = -0,34$, $P < 0,01$). Cette étude semble indiquer qu'une stratégie d'amélioration génétique des lignées résistantes à la pourriture sèche, tout en maintenant un rendement élevé, devrait inclure un tamisage pour éliminer des lignés très sensibles provenant de croisements, et par la suite, une sélection des lignées ayant les plus hauts rendements tout en ayant le plus bas niveau de maladie.

The relationship between disease and yield is complex, often confounded by genetic and environmental factors, and their interactions. Traditionally, yield losses resulting from disease are measured by comparing yields from infected plants to yields from nondiseased plants (James 1974, Main 1977). Estimation of loss is difficult in host-pathogen systems such as common root rot on barley, where the symptoms are not easily visible, plants are not killed outright, and there is no completely effective chemical control to provide "healthy" checks.

Losses in grain yield attributed to common root rot caused by *Cochliobolus sativus* (Ito & Kurib.) Drechsl. ex Dastur [anamorph *Bipolaris sorokiniana*

Table 1. Analysis of variance, expected mean squares, and linear function of genetic and environmental variance components used to calculate heritability of common root rot severity and yield at two locations, 1988–1990

Source of variation	df	Mean squares	Expected mean squares	Linear functions of genetic and environmental variance components
Analysis for nonsegregating generations (parents)*				
Replicate	r-1	MS1	$\sigma^2 + g\,\sigma_r^2$	
Genotype	g-1	MS2	$\sigma^2 + r\,\sigma_g^2$	
Error	(r-1)(g-1)	MS3	σ^2	$(Ve_w + Vg_w) + Ve_b$
Analysis for segregating lines (progeny)				
Replicate	r-1	MS4	$\sigma^2 + g\,\sigma_r^2$	
Genotype	g-1	MS5	$\sigma^2 + r\,\sigma_r^2$	$[(Ve_w + Vg_w) + Ve_b] + rVg_b$
Error	(r-1)(g-1)	MS6	σ^2	$(Ve_w + Vg_w) + Ve_b$

*Parental cultivars are assumed to be homozygous, so $Vg_w = 0$.

(Sacc. in Sorok.) Shoem., syn. *Helminthosporium sativum* Pamm., King and Bakke] average 10% in barley but may increase to 42%, depending on the cultivar (Piening 1973, Piening et al. 1976, Tinline & Ledingham 1978). These estimates were obtained by comparing yields of individual infected plants to yields of plants with no root rot. The single plant method may not reflect what happens in a field population where healthy plants may compensate for the diseased ones (Grey & Mathre 1984).

Piening (1973) suggested that barley cultivars vary in their ability to tolerate root rot since cultivars with similar levels of disease differed in grain yield. Tinline and Ledingham (1978) found a frequent lack of correlation between grain yield and disease in barley. This was related primarily to the fact that cultivars with intermediate disease reactions often had the least reduction in yield. Duczek (1984) indicated an inconsistent correlation between disease rating and grain yield loss, although increased disease intensity was generally associated with yield loss. The objective of this study was to determine the genetic relationship between common root rot severity and grain yield in single seed descent progeny of a cross between the barley cultivars Argyle and Melvin, resistant and tolerant respectively to common root rot.

Methods and materials

Single seed descent was used to advance a random selection of 125 F2 progeny of the cross Argyle × Melvin to the F4. Subsequently, each progeny line was increased as a bulk to the F6, when there was sufficient seed to begin yield and disease testing. These cultivars were chosen because they have a lot of common parentage and thus show many similarities, such as in yield and maturity, but differ widely for common root rot reaction. Data from the 1978 Western Co-operative Barley Test (Minutes of the 34th Meeting of the Expert Committee on Grain Breeding, Saskatoon, Saskatchewan, 1979, pp.

70–101) supports this statement. The mean % common root rot from three tests in disease nurseries was significantly lower for Argyle (29%) than for Melvin (74%). Agronomic tests conducted at 17 sites in Alberta, Saskatchewan, and Manitoba, showed that Argyle and Melvin were similar in yield, averaging 4630 kg ha[-1] and 4610 kg ha[-1], respectively. Agronomic tests at three sites in Ontario and Quebec also showed no significant differences in yield for Argyle (3560 kg ha[-1]) and Melvin (3680 kg ha[-1]).

The progeny and the parents were arranged in a randomized complete block design with 2–4 replications and grown in disease nurseries at Saskatoon, Saskatchewan, and in the Peace River region, Alberta, from 1988 to 1990. The sites in the Peace region were Fairview in 1988, Beaverlodge in 1989, and Wembly in 1990.

Seeds were sown 6 cm deep at a rate of 60 seeds m[-1] in 4-row (1 m wide), 6-m length plots. Disease severity was expressed as the percentage of 30 plants with discoloration on the subcrown internode greater than 50%. At Saskatoon, plants from the outer row were rated at soft dough to maturity, while the plants from the Peace region were randomly sampled from each plot after grain harvest. All yield estimates were expressed as kg ha[-1].

Analyses of variance were used to calculate the variance components for common root rot severity and grain yield (Table 1). Heritabilities were estimated using the following equation:

$$\text{Heritability} = Vg_b/(Vg_b + ((Ve_b + Vg_w + Ve_w)/r)),$$

where Vg_b and Vg_w are the genotypic variances among lines and within lines: Ve_b and Ve_w are the environmental variances among lines and within lines, respectively; and r is the number of replicates (Bailey et al. 1988). Each variance component was estimated by solving the equations relating mean squares to linear functions of genetic and environmental variance components. These were calculated

from Table 1 as V_{g_b} = (MS5 − MS6)/r, V_{g_w} = MS6 − MS3, and (V_{e_w} + V_{e_b}) = MS3.

The Castle-Wright method of moments, modified by Mulitze and Baker (1985), was used to estimate the minimum number of loci controlling the expression of common root rot severity and yield that differed between the two parents. The equation used was:

$$\text{Number of loci} = R^2/(8\ C_{ggg}/(2 - 1/2^g),\ \text{where}$$

R = the difference between the largest and smallest family mean in generation g multiplied by the estimate of heritability of family means for the same generation, C_{ggg} = genetic variance among families in generation g, and g = the number of selfing generations after the F2, for which g = 0.

Statistical assessment included analysis of variance, regressions of yield on disease and maturity, and cluster and discriminant analysis (SAS 1985). Prior to cluster analysis, the data were standardized to a mean of zero and a variance of one, since yield and disease were not measured in the same units. Similar genotypes were grouped into clusters using the SAS Fastclus procedure, based on disease and yield means over years and locations. The procedure used disjoint clustering, so that each progeny line could be placed in only one cluster. Several models were tested by changing the criteria for maximum number of clusters formed (i.e. 10, 5, 4, 3, and 2). The model with three clusters formed discrete groups with the least overlap in the radius of each group. The clusters were used to classify each line as resistant, tolerant, or susceptible, based on the similarity of the cluster to Argyle, Melvin, or neither parent, respectively. After

defining the clusters, SAS Stepdisc discriminant procedure was used to calculate the correlation of yield and disease both within classes and among classes.

Result

Significant differences ($P < 0.05$) in disease and yield were observed among the progeny and parents of the cross Argyle × Melvin in most years at both locations; there were no differences in yield at Beaverlodge in 1989 and at Saskatoon in 1990. Based on three-year averages, the severity of common root rot was marginally higher, and yields lower, at Saskatoon than in the Peace region (Table 2). Argyle had lower disease severity than Melvin, but Melvin yielded more than Argyle. Therefore, Argyle was classified as resistant and Melvin as tolerant.

The distributions of disease rating and of yield of progenies were restricted to fewer classes at Saskatoon than in the Peace region (Table 2). Most of the progenies tested at Saskatoon were intermediate to the parents in disease severity (103 of the 125 progenies or 83%) and similar to Argyle in yield (84 of the 125 progenies or 67%). In the Peace region, most of the progenies (57 of 125 progenies or 46%) were intermediate to the parent's disease severity but 10% (13/125) of the lines were more severely diseased than Melvin. The yields of 54% (67/125) of the progenies equalled or exceeded that of Melvin.

The average heritability of common root rot reaction was 56.7% (S.E. 4.1) and the average minimum number of loci which differed between the two parents was 2.6 (S.E. 0.1) genes. Estimates of average heritability for yield were considerably lower at

Table 2. Distribution of mean common root rot severities (CRR, %) and mean yields (kg ha^{-1}) of Argyle, Melvin, and 125 progeny lines at Saskatoon, and in the Peace region, 1988–1990

Location and line	Trait	Mean	S.E.	A	B	C	D	E	F	G	H	I	J
Saskatoon													
Argyle	CRR	40	14				1						
Melvin	CRR	66	8							1			
Progeny	CRR	49	12				8	43	60	14			
Argyle	Yield	1980	348		1								
Melvin	Yield	2240	322				1						
Progeny	Yield	2020	278	16	84	24	1						
Peace region													
Argyle	CRR	33	12				1						
Melvin	CRR	55	7						1				
Progeny	CRR	40	10			1	26	57	28	2	3	5	3
Argyle	Yield	3224	432						1				
Melvin	Yield	3726	460								1		
Progeny	Yield	3390	394		3	3	4	6	15	27	32	25	10

[†]Disease severity (CRR) classes : A = 0–10, B = 11–20, C = 21–30, D = 31–40, E = 41–50, F = 51–60, G = 61–70, H = 71–80, I = 81–90, J = 91–100.
Yield classes: A = 1050–1800, B = 1801–2100. C = 2101–2400, D = 2401–2700, E = 2701–3000, F = 3001–3300, G = 3301–3600, H = 3601–3900, I =3 901–4200, J = 4201–4700.

Table 3. Estimates of genetic (Vg_w, Vg_b,) and environmental ($Ve_w + Ve_b$) variance components, heritability (h^2) in the F6 through F8 generations, differences between the largest and smallest family means (R), and the minimum number of loci controlling common root rot severity and yield at Saskatoon and in the Peace region, 1988–1990

| Genetic parameters | Common root rot severity | | | | | | Yield | | | | | |
| | Saskatoon | | | Peace region | | | Saskatoon | | | Peace region | | |
	1988	1989	1990	1988	1989	1990	1988	1989	1990	1988	1989	1990
Vg_b	2.7	83.2	102.1	29.2	83.2	81.1	1178	4492	9040	53167	12588	3507
Vg_w	25.7	72.0	0.0	42.7	75.8	32.3	12550	56669	20156	31567	161167	0
$Ve_w + Ve_b$	1.9	27.3	356.9	103.1	27.3	83.3	345	11659	131326	14102	33695	24364
R	4.9	31.4	30.5	15.5	30.4	33.0	79	212	187	1066	356	191
h^2	22.9	77.0	61.5	28.6	76.3	73.7	22	21	19	70	21	59
No. of loci	2	3	2	2	3	3	3	3	1	53	3	3
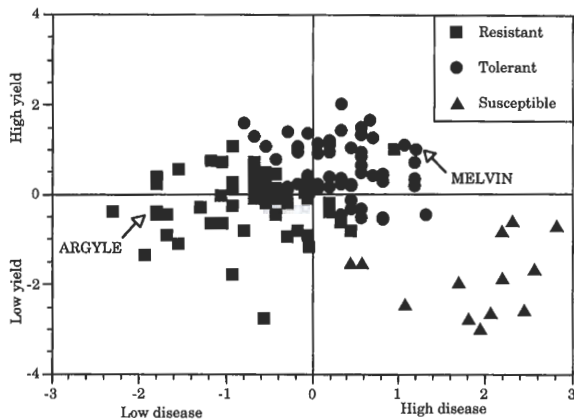
Figure 1. Clustering of progeny genotypes from the barley cross Argyle × Melvin with similar disease reactions and yields from Saskatoon and the Peace region over three years.

35.3% (S.E. 3.8) and the average number of loci that differed between the two parents was 10.5 (S.E. 3.5) genes. Estimates of heritability and number of loci varied over year and location (Table 3). The number

of genes for yield estimated from the Peace region was highly skewed by the 1988 data. The average number of loci for yield would decrease to 2.6 (S.E. 0.2) if the number of genes calculated from the Peace region in 1988 (53 genes) was excluded.

Cluster analysis indicated that the means of the progeny could be grouped into three classes, based on their disease severity and yield (Fig. 1). There were 56 resistant (i.e. similar to Argyle), 56 tolerant (i.e. similar to Melvin), and 13 susceptible (i.e. different from Argyle and Melvin) progeny from the cross of Argyle × Melvin. All lines in the susceptible group had high disease and low yield. Most of the lines clustered in the resistant group had low disease, with either low yield (30 lines) or high yield (21 lines). The tolerant group had most lines combining either high yield and low disease (18 lines) or high yield and high disease (31 lines). The boundaries between the resistant and tolerant groups overlapped slightly. The total number of progeny lines in each quadrant of Figure 1 were 30 with low disease and low yield, 25 with high disease and low yield, 39 with low disease and high yield, and 31 with high disease and high yield.

Table 4. Regression of mean yield on mean disease severity (n = 127) among barley lines classed as resistant, susceptible, and tolerant from a cross between Argyle × Melvin at Saskatoon and the Peace region, 1988–1990

| Sample	Combinations of disease groups analyzed	Regression equation (y = mx + b)	Correlation coefficient (R)	Level of significance (P)
Saskatoon	Resistant and susceptible	y = -1.7x + 1029	- 0.11	0.3783
	Tolerant and susceptible	y = 3.3x + 813	+0.19	0.1082
	Resistant and tolerant	y = 0.9x + 939	+0.06	0.4944
	Resistant, tolerant, susceptible	y = 1.8x + 883	+0.12	0.1672
Peace region	Resistant and susceptible	y = -8.8x + 2086	- 0.60	0.0001
	Tolerant and susceptible	y = -16.4x + 2735	- 0.72	0.0001
	Resistant and tolerant	y = 9.7x + 1401	+0.34	0.0002
	Resistant, tolerant, susceptible	y = -8.1x + 2180	- 0.41	0.0001
Average	Resistant and susceptible	y = - 8.5x + 1706	- 0.53	0.0001
	Tolerant and susceptible	y = -19.9x + 2499	- 0.69	0.0001
	Resistant and tolerant	y = 5.7x + 1136	+0.28	0.0026
	Resistant, tolerant, susceptible	y = -4.9x + 1625	- 0.24	0.0065
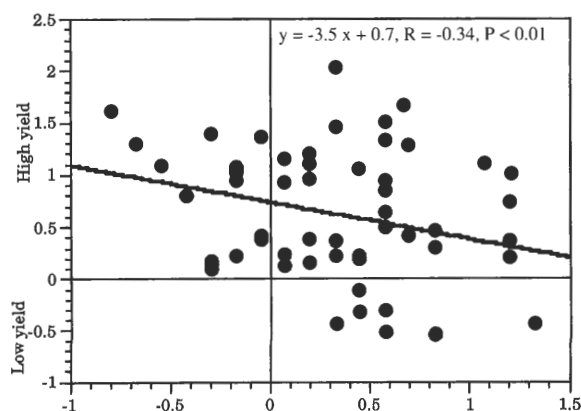
Figure 2. Clustering of the tolerant genotypes from the cross Argyle × Melvin and the regression of mean yield on mean disease from Saskatoon and the Peace region over three years.

A significant negative regression between grain yield and disease severity was found for the 125 progenies and the two parents averaged over the locations (Table 4). The correlation co-efficient was negative but weak (R = –0.24 at $P < 0.01$). There were no significant within-class correlations between yield and disease in the resistant or susceptible classes. There was a negative within-class correlation between yield and disease for lines classed as tolerant (R = –0.34, $P < 0.01$). Regression analysis of the 56 lines in the tolerant class showed that yields decreased as disease severity increased (Fig. 2).

The rank of progeny lines for disease severity at Saskatoon differed from that in the Peace region, with a correlation coefficient of R = 0.12 at $P = 0.16$. Within-class correlations indicated that the rank of progeny was similar at both locations for those classed as resistant (R = 0.57, $P < 0.001$) and for those considered tolerant (R = 0.45, $P < 0.01$). There was no relationship between the two regions for lines classed as susceptible (R = 0.06, $P > 0.10$). The rank of progeny for grain yield was weakly correlated between Saskatoon and the Peace region (R = 0.33, $P < 0.001$).

Examination of regressions and correlations of mean disease severity versus yield of the progeny and parents at each location showed no significant relationship at Saskatoon and a negative relationship in the Peace region (Table 4). Relationships between disease and yield in combinations of disease classes were determined by regressing yield on disease using only individuals in those classes of resistant, susceptible, or tolerant progeny being compared. In Saskatoon, there were no significant relationships. In the Peace region, the relationship between yield and disease was strongly negative for the combination of resistant and susceptible proge-

nies, and also for the combination of tolerant and susceptible progenies. The relationship between yield and disease when data for resistant and tolerant progenies were combined was weaker and positive.

Discussion

In barley the expression of common root rot severity was more heritable than that of yield, (Table 3), but environment had a large influence on the expression of both traits. The minimum number of loci controlling the expression of disease and yield by which the two parents differed was approximately 3 genes for disease resistance and 3 to 11 genes for grain yield. These results are similar to estimates of heritability of common root rot resistance and the number of loci differentiating parents of two wheat crosses (Bailey et al. 1988). Others have reported that disease reaction to common root rot in barley is controlled by as few as two genes in some parents (Loiselle 1965) and polygenically in other lines (Cohen et al. 1969). It is possible that some genes have linkages or pleiotropic effects, influencing both disease and yield, while other genes act independently. It is not possible to determine precisely the number of loci involved in the expression of a trait and the numbers may be an artifact of an experiment. The true number of genes involved in the expression of a trait may be higher than that calculated because the procedure can only estimate those genes that are different between the two parents and not the total number of genes that the parents carry. The calculations still have a useful purpose in appraising the breeding value of a trait to determine whether relatively few genes or many genes differentiate the parents of crosses.

All possible combinations of the traits occurred within the collection of progeny, i.e. low disease-low yield, low disease-high yield, high disease-low yield, high disease-high yield (Fig. 1), which suggests that disease resistance, disease tolerance, and yield are genetically independent. Therefore, a breeder should be able to select for any combination of the traits in a segregating population. There is no evidence to indicate that low disease-high yield combinations occur more or less frequently than high disease-high yield combinations.

Location had a significant influence on the expression of both disease and yield (Table 2). The range of disease severity and yield of the progeny lines generally lay within the range of the parents at Saskatoon and exceeded the range between the parents in the Peace region. The rank of progeny for disease severity was similar at both locations, with the exception of some susceptible genotypes that appeared to be resistant at Saskatoon. Duczek (1984) indicated that the rank of barley cultivars for disease

reaction to common root rot was similar when tested at Saskatoon and Beaverlodge, despite differences in the overall level of disease severity. The weak correlation with yield observed at both locations in this study was expected, since the heritability of yield is low. In this study, selection for potential breeding lines would have been most effective in the Peace region, where the maximum expression of the traits was achieved.

The concept of tolerance in relation to plant disease, as described by Mussell (1980), is still a topic of much discussion. It is clear that some cultivars with high levels of disease yield more grain than do other cultivars, which have lower disease levels (Piening 1973, Tinline & Ledingham 1978, Duczek 1984). In the current study, the word tolerance was applied in this sense to Melvin as compared to Argyle. Cluster analysis indicated that, from a cross between a resistant and a tolerant cultivar, the progeny may be resistant, tolerant, or susceptible (Fig. 1). The relationship between grain yield and disease severity for a population of 125 progeny lines from a single cross was very small, but significant and negative (Table 4). Therefore, in a large, genetically related population, many of the most severely diseased lines will have the lowest yields and the less severely diseased lines will have higher yields. Negative relationships between disease and grain yield or yield components have been observed previously for barley and wheat cultivars (Machacek 1943, Piening 1973, Verma et al. 1976, Tinline & Ledingham 1978, Kidambi et al. 1985, Duczek 1984, Nutter et al. 1985, Wildermuth et al. 1992).

The negative relationship between yield and disease was moderate to strong for the disease group combinations of resistant or tolerant lines compared to the susceptible ones. Highly susceptible lines had a negative effect on yield. On the other hand, when the susceptible lines were not considered in the disease group combination, the relationship between disease severity and yield for the resistant and tolerant groups changed to a weak but significant positive correlation (Table 4). Tinline and Ledingham (1978) suggested that a simple correlation between disease ratings and grain yield was unlikely when resistance differs from tolerance. This study supports that conclusion.

Selection strategies in a breeding program may operate at two levels in order to improve common root rot resistance while trying to maximize yield. The selection of cultivars representing the lower and upper limits of acceptable disease severity may be critical to the classification of resistant, tolerant, and susceptible lines. For example, if selection criteria are based on similarities to Argyle, then lines with tolerance will be discarded. The first step in the program would be to eliminate the most severely diseased lines, which will carry genes for susceptibility with genes for tolerance. These are lines that exceed the maximum limit of acceptable disease severity. Unfortunately, this step is difficult in segregating generations due to the heritability of disease expression.

The second step in the program should place more emphasis on estimating yield and disease together in order to select those remaining individuals with lower disease and higher yield. The correlation between disease and yield for the disease group combination of resistant and tolerant lines was positive, which suggests a genetic linkage between high yield and susceptibility in Melvin and lower yields and resistance in Argyle. But there was also a negative relationship between disease and yield for lines within the tolerant group (Fig. 2). Therefore, even among tolerant individuals, a breeder could select those lines with the lower disease and higher yields. Strong selection for yield combined with common root rot resistance might break some genetic linkages, if they exist.

This strategy for selection is not completely new as it has been accomplished intuitively for many years by selecting mainly for high yield and sometimes for low disease. This study has demonstrated that the strategy was correct. In recent years, most breeders have eliminated highly susceptible lines from their programs. Much of the current breeding material qualifies for the second phase of selection. As pointed out, it is important to maintain the screening process to minimize the number of highly susceptible lines being used as breeding material, and to identify the best yielding lines with the lowest disease.

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