Effect of Interplot Interference on the Assessment of Partial Resistance to Stem Rust in Durum Wheat

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ABSTRACT

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Two experiments were carried out in which different plot designs were compared to assess the level of interplot interference of stem rust (*Puccinia graminis* f. sp. *tritici*) on durum wheat (*Triticum turgidum* var. *durum*). The resistance levels of the most resistant genotypes were on average a factor of 17.3 (experiment 1) to 31.6 (experiment 2) lower in adjacent plots than in isolated plots, which were assumed to represent the true level of resistance of a genotype (no interplot interference). According to Spearman's rank correlation coefficients, ranging from 0.88 to 0.99, the ranking of genotypes was not affected by the presence or absence of interplot interference. Thus, selection for partial resistance in adjacent small plots is not affected by interplot interference. Using standard control cultivars with known levels of partial resistance will correct the underestimation of true levels of partial resistance and will help breeders determine the levels of disease that are still acceptable for selecting durum wheat genotypes for partial resistance to stem rust.

Disease resistance in breeders' fields is commonly evaluated in relatively small, adjacent plots in an environment with high disease. This way of evaluating genotypes may lead to an underestimation of disease resistance since genotypes with incomplete or partial resistance may be heavily affected by alloinfection from susceptible neighbors (17). This "representational error" (16) is caused by interplot interference, which can be negative or positive (3,4). In negative interplot interference, a net loss of spores from a small field plot causes slower disease development, and hence resistance is overestimated in comparison with a farmer's field. In the latter case, a net influx of spores causes faster disease increase, leading to underestimation of resistance.

The level of interplot interference depends on several factors including plot size, plot shape, distance between plots, and steepness of the disease gradient (10,16). All rust diseases of wheat are caused by airborne pathogens, but steepness of disease gradients and therefore levels of interplot interference may differ.

Numerous studies have been conducted to quantify interplot interference (1–7,9,12) but few studies have been conducted to quantify the effect of interplot interference on the assessment of incomplete or partial resistance. For leaf rust (*Puccinia hordei*) of barley (*Hordeum vulgare*) the level of partial resistance was greatly underestimated in small adjacent plots (breeders' plots) compared with isolated microfields representing farmers' fields (8,9). Genotypic differences were 130 times smaller in adjacent plots than in isolated plots. In powdery mildew of barley (*Erysiphe graminis* f. sp. *hordei*), interplot interference reduced genotypic difference by up to one-third (6) and changes in genotype ranking were observed. In yellow rust (*Puccinia striiformis* f. sp. *tritici*) on wheat (*Triticum aestivum*), interplot interference did not affect rankings and differences in disease severity between susceptible and partially resistant genotypes (2). Since the effect of interplot interference cannot be generalized, the present study was conducted to determine if and to what extend interplot interference affects the assessment of stem rust (*Puccinia graminis* Pers.:Pers. f. sp. *tritici* Eriks. & E. Henn.) resistance in durum wheat (*Triticum turgidum* L. var. *durum* (Desf.) MK).

MATERIALS AND METHODS

Two experiments were carried out to measure the effect of interplot interference on the assessment of stem rust resistance in durum wheat. Experiment 1 included four sub-experiments, each with a different plot design (Table 1), and ten durum wheat genotypes with different levels of partial resistance (Table 2). Bansi 168 was the susceptible check.

Each sub-experiment was planted as randomized complete block designs with three replications (15). Randomization was the same for all sub-experiments.

Design 1 consisted of microfields of six rows planted on three beds 2 m long. Beds were spaced at 90 cm and rows on a bed at 20 cm. The effective area for a microfield was 5.4 m^2. Neighboring plots were separated by 10 m of a resistant durum variety (Altar). For design 2, the same plot dimensions were used as in design 1, but plots were not separated. For design 3, plot size was reduced to one-third the size in design 1. Adjacent plots were planted as two rows on one bed, 2 m long , giving an effective area of 1.8 m^2 (this plot design is commonly used by breeders at CIMMYT). Plot size was further reduced in design 4 and measured only 0.45 m^2. Adjacent plots were planted as two rows on a bed, 0.5 m long. In the case of the adjacent plot designs, plots were planted as groups of five adjacent plots. Groups were separated by 1 m of path. Sub-experiments for designs 2, 3, and 4 were separated from each other by 10 m of resistant durum wheat and from design 1 by 25 m of resistant durum variety to minimize the interference between plot designs.

Experiment 2 consisted of three sub-experiments using designs 1–3 from experiment 1 (Table 1). The genotypes used included five durum wheat genotypes from experiment 1 and bread wheat cv. Morocco as susceptible check (Table 3). The designs were as described for experiment 1 with a few modifications. The distance between beds was reduced to 75 cm, which resulted in

a reduction of the effective plot size for designs 1 and 2 to 4.5 m^2 and for design 3 to 1.5 m^2. Moreover, resistant triticale was used instead of durum wheat to separate the three designs and neighboring plots within design 1. Finally, the distance between design 2 and 3 was increased to 25 m instead of 10 m. In the sub-experiments with design 2 and 3 blocks were separated by 1 m of path.

As in experiment 1, the three sub-experiments were each planted as a randomized complete block design with three replications and randomization was the same for all three sub-experiments.

Plots were planted on 25 November 1991 (experiment 1) and 26 November 1993 (experiment 2) at the CIANO (Centro de Investigación Agrícola del Noroeste) experimental station in Obregon, state of Sonora, Mexico. Plots were inoculated on 20 January in 1992 (experiment 1) and 1994 (experiment 2) with race GFC of stem rust (14). In each plot, 1 tiller per 0.5 m of bed was injected with a suspension of urediospores in water (13). This assured equal levels of initial inoculum per unit area in each plot.

Data were collected on 7 March, 13 March, and 19 March 1992 in experiment 1 and on 14 March and 24 March 1994 in experiment 2. Fifteen tillers per plot were sampled in experiment 1 and ten tillers in experiment 2. Two methods were used to assess the amount of rust on the upper two leaf sheaths of the

TABLE 1. Characteristics of plot designs used to assess interplot interference of stem rust on durum wheat

		No. of genotypes tested		Plot size (m^2)		Isolation of neighboring plots: Surrounding resistant crop		Isolation of neighboring plots: Distance (m) between plots	
Sub-experiment	Plot design	Exp. 1	Exp. 2	Exp. 1	Exp. 2	Exp. 1	Exp. 2	Exp. 1	Exp. 2
1	Large isolated	10	6	5.4	4.5	Durum	Triticale	10	10
2	Large adjacent	10	6	5.4	4.5	...	...	...	...
3	Medium adjacent	10	6	1.8	1.5	...	...	...	...
4	Small adjacent	10	6	0.45	...	...	...	...	...

TABLE 2. Resistance index (RI) and disease severity (DS) of ten durum wheat genotypes in isolated and three adjacent plot designs of different sizes at three assessment dates (experiment 1)[w]

	Isolated plots[x]		Adjacent plots[x]					
	5.4 m^2		5.4 m^2		1.8 m^2		0.45 m^2	
Genotype	RI	DS	RI	DS	RI	DS	RI	DS
Date 1								
Bansi 168	1.0	48.4 a[y]	1.0	57.1 a	1.0	61.1 a	1.0	38.8 a
D115	2.2	22.2 ab	1.6	35.7 ab	1.8	33.9 ab	1.9	20.4 ab
Marizo Sadovo	2.0	24.2 ab	1.5	38.1 ab	2.4	25.5 bc	1.5	25.8 ab
Red Kathia	2.4	20.2 ab	2.3	24.8 b	2.0	30.6 b	2.0	19.4 ab
Burhampul Local	5.6	8.6 bc	1.9	30.1 b	2.1	29.1 b	3.0	12.9 bc
Vavilov NI17403	8.4	5.8 cd	3.0	19.0 b	4.6	13.3 c	6.5	6.0 c
21563-AA'S'	21.5	2.3 de	14.4	3.9 c	4.9	12.5 c	6.1	6.4 c
Amal 72	340.3	0.14 ef	58.5	0.98 d	31.5	1.9 d	13.2	2.9 d
Giza 26	833.2	0.06 f	85.2	0.67 d	49.6	1.2 d	130.9	0.3 e
Jiloca	1,930.1	0.03 f	115.5	0.49 d	103.2	0.6 d	121.4	0.3 e
Mean	314.7	13.2 A[z]	28.5	21.1 C	20.3	20.3 C	28.7	13.3 B
Date 2								
Bansi 168	1.0 a	61.7 a	1.0	73.0 a	1.0	55.6 a	1.0	60.4 a
D115	1.7 a	36.3 a	1.3	56.2 b	1.2	46.3 a	1.8	33.6 ab
Marizo Sadovo	1.5	41.1 a	1.9	38.4 b	1.4	39.7 ab	1.6	37.8 ab
Red Kathia	1.4 ab	44.1 ab	1.5	48.7 b	1.0	54.3 a	1.3	46.5 ab
Burhampul Local	2.3	26.8 ab	1.6	45.6 b	1.3	42.8 ab	2.5	24.2 bc
Vavilov NI17403	3.9	6.9 bc	1.9	38.4 b	1.7	32.7 bc	2.4	25.2 bc
21563-AA'S'	13.6	4.5 c	8.0	9.1 c	3.1	17.9 bcd	4.1	14.7 c
Amal 72	535.3	0.12 d	33.7	2.2 d	12.2	4.6 cde	11.4	5.3 d
Giza 26	1,063.0	0.06 d	56.3	1.3 d	34.2	1.6 ef	65.0	0.9 e
Jiloca	1,063.0	0.06 d	138.4	0.5 e	70.0	0.8 f	116.7	0.5 e
Mean	215.6	22.2 A	24.6	31.3 B	12.7	29.6 B	20.8	24.9 B
Date 3								
Bansi 168	1.0	100.0 a	1.0	100.0 a	1.0	100.0 a	1.0	100.0 a
D115	1.8	55.6 bc	1.6	62.5 b	1.4	70.5 bc	2.1	47.6 bc
Marizo Sadovo	1.8	54.5 bc	1.6	62.5 b	1.8	55.3 c	1.9	52.6 bc
Red Kathia	1.3	76.9 b	1.3	75.2 b	1.3	75.5 b	1.6	62.5 b
Burhampul Local	2.0	50.0 bc	1.5	66.7 b	1.5	66.7 bc	2.0	50.0 bc
Vavilov NI17403	3.3	30.3 c	1.4	71.4 b	1.7	58.8 c	1.8	55.6 bc
21563-AA'S'	10.6	9.4 d	3.3	29.9 c	2.1	47.6 c	3.3	30.0 cd
Amal 72	101.4	1.0 e	10.2	9.8 cd	5.6	17.9 d	6.4	15.6 d
Giza 26	271.5	0.37 e	23.7	4.2 de	16.2	6.2 d	28.9	3.5 e
Jiloca	1,407.6	0.07 e	31.2	3.2 e	90.5	1.1 e	121.4	0.8 e
Mean	180.2	37.8 A	7.7	48.5 BC	12.3	49.9 C	17.0	41.8 B

[w] RI calculated as DS of BANSI 168 divided by DS of respective genotypes in same plot design.
[x] Neighboring isolated plots isolated by 10 m of resistant durum wheat assumed to have negligible interplot interference; sub-experiments with different adjacent plot sizes were separated from each other by 10 m of resistant durum wheat and from isolated plots by 25 m of resistant durum wheat.
[y] Values in column with same letter do not differ significantly. Analysis of variance based on logit-transformed DS of individual assessment dates.
[z] Values in row with same letter do not differ significantly. Analysis based on logit-transformed DS.

TABLE 3. Resistance index (RI) and disease severity (DS) of five durum wheat genotypes and one bread wheat genotype in isolated and two adjacent plot designs of different sizes at two assessment dates (experiment 2)[w]

	Isolated[x]		Adjacent[x]			
	4.5 m^2		4.5 m^2		1.5 m^2	1.5 m^2
	RI	DS	RI	DS	RI	DS
Date 1						
Morocco	1.0	75 a[y]	1.0	83 a	1.0	69 a
Vavilov NI17403	6.3	12 b	3.2	26 b	3.6	19 bc
21563-AA'S'	5.9	13 b	6.1	14 c	2.9	24 b
Amal 72	20.2	3.7 bc	24.3	3.4 d	10.4	6.6 c
Giza 26	41.4	1.8 c	22.9	4.4 d	12.7	5.5 d
Jiloca	1,492.0	0.05 d	137.7	0.6 e	32.6	2.1 d
Mean	261.1	18 A[z]	32.5	22 B	10.1	21 B
Date 2						
Morocco	1.0	100 a	1.0	100 a	1.0	100 a
Vavilov NI17403	1.7 b	60 b	1.0	97 a	1.1	93 a
21563-AA'S'	1.7 bc	59 bc	1.3	77 b	1.5	67 b
Amal 72	4.6 c	22 bc	2.3	43 c	2.1	48 c
Giza 26	6.7 c	15 c	3.3	30 c	4.1	24 d
Jiloca	275.3 d	0.4 d	11.6	8 d	6.0	17 d
Mean	58.2	43 A	3.6	58 B	2.5	59 B

[w] RI calculated as DS of Morocco divided by DS of respective genotypes in same plot design.
[x] Neighboring isolated plots isolated by 10 m of resistant durum wheat assumed to have negligible interplot interference; sub-experiments with different adjacent plot sizes were separated from each other by 10 m of resistant durum wheat and from isolated plots by 25 m of resistant durum wheat.
[y] Values in column with same letter do not differ significantly. Analysis of variance performed on individual experiment and assessment dates based on logit-transformed DS.
[z] Values in row with same letter do not differ significantly. Analysis based on logit-transformed DS.

TABLE 4. Analysis of variance for logit-transformed disease severity of stem rust for each assessment date within two experiments

	Experiment 1				Experiment 2		
		Mean squares[z]				Mean squares[z]	
Source	Degrees of freedom	Date 1	Date 2	Date 3	Degrees of freedom	Date 1	Date 2
Design (D)	3	19.71***	17.34***	21.37***	2	6.76***	15.00*
Replications within D	8	2.52	0.85	1.50	6	1.45	3.91
Genotype (G)	9	87.66***	97.53***	215.35***	5	55.93***	191.5**
G × D	27	1.43	1.97**	2.33**	10	3.20***	3.21
Error	72	0.91	1.06	1.00	30	0.62	3.89

[z] One, two, and three asterisks significant at $P = 0.05$, $P = 0.01$, and $P = 0.001$, respectively.

TABLE 5. Analysis of variance for logit-transformed disease severity of stem rust in two experiments combining assessment dates and plot designs

	Experiment 1		Experiment 2	
Source	Degrees of freedom	Mean squares[z]	Degrees of freedom	Mean squares[z]
Design (D)	3	57.80***	2	19.54***
Replications within D	8	3.69***	6	3.37
Genotype (G)	9	343.10***	5	220.04***
Assessment date (A)	2	303.33***	1	484.76***
G × A	18	19.87***	5	25.10***
G × D	27	4.56***	10	5.77**
A × D	6	0.35	2	1.81
G × A × D	54	0.41	10	0.50
Error	232	0.97	66	2.28

[z] One, two, and three asterisks significant at $P = 0.05$, $P = 0.01$, and $P = 0.001$, respectively.

tillers. In the first method, disease severity was estimated according to the Modified Cobb Scale (0–100%) (11). For the second method, the number of pustules per leaf sheath were counted. The lengths and widths of the leaf sheaths were measured to correct for differences in leaf sheath area.

At relatively high disease severity (>10%), only the Modified Cobb Scale was used. Pustule counts were not reliable because individual pustules tended to coalesce. At very low severity (<1%), only the number of pustules were counted. At intermediate severity (1–10%), numbers of pustules were counted and the disease severity was estimated. These data were used in regression analyses to determine the relationship between number of pustules per unit area of leaf sheath and disease severity for each genotype. Subsequently, the number of pustules was transformed into percent disease severity with the linear regression equations.

Since disease severity data were not normally distributed, they were converted to logits (17): Logit (DS) = Ln(DS/(100 − DS)) where DS is the disease severity in percentage. For DS = 0, logit(DS) was set at −9.21 and for DS = 100, logit (DS) was set at +9.21.

The mean logit of the upper two leaf sheaths was determined for each tiller and used to calculate plot means (mean of ten or fifteen tillers). Plot means were used as the statistical unit in analysis of variance. First, different sub-experiments and assessment dates were analyzed separately according to a randomized block design. In addition, sub-experiments (designs) were added as source of variance to the models to test the sub-experiment (= design) × genotype interaction for each assessment date (Table 4). Finally, assessment dates were added to the models (Table 5). All analyses were performed with version V609 of the statistical package SAS (SAS Institute, Inc., Cary, NC).

Plot means of logits were re-converted to disease severity in percentage. To correct for differences in disease level between plot designs, a resistance index (RI) was calculated for all genotypes in each plot design, relative to susceptible check (Bansi 168 for experiment 1 and Morocco for experiment 2) as follows: $RI_{ij} = DS_{is}/DS_{ij}$ where DS is the disease severity in plot design i of the susceptible check and DS is the disease severity in plot i of genotype j.

The RI-ratio (RIR) for a given genotype was calculated as $RIR_i = RI_{i(isol)}/RI_{j(adj)}$ where $RI_{i(isol)}$ is the resistance index for

TABLE 6. Resistance index ratio (RIR) calculated as resistance index (RI) in isolated plots divided by RI in adjacent plots of nine durum wheat genotypes in adjacent plots with different sizes (experiment 1)

	Assessment date									
	1			2			3			
Genotype	5.4 m^2	1.8 m^2	0.45 m^2	5.4 m^2	1.8 m^2	0.45 m^2	5.4 m^2	1.8 m^2	0.45 m^2	Mean
D115	1.4	1.2	1.2	1.3	1.4	0.9	1.1	1.3	0.9	1.2
Marizo Sadovo	1.3	0.8	1.3	0.8	1.1	0.9	1.1	1.0	0.9	1.0
Red Kathia	1.0	1.2	1.2	0.9	1.4	1.1	1.0	1.0	0.8	1.1
Burhampul Local	2.9	2.6	1.9	1.4	1.8	0.9	1.3	1.3	1.0	1.7
Vavilov NI17403	2.8	1.8	1.3	2.1	2.3	1.6	2.4	1.9	1.8	2.0
21563-AA'S'	1.5	4.4	3.5	1.7	4.4	3.3	3.2	5.0	3.2	3.3
Amal 72	5.8	10.8	25.8	15.9	43.9	47.0	9.9	18.1	15.8	21.4
Giza 26	9.8	16.8	6.4	18.9	31.1	16.4	11.4	16.7	9.4	15.2
Jiloca	16.7	18.7	15.9	7.7	15.1	9.1	45.1	15.6	11.6	17.3

TABLE 7. Resistance index ratio (RIR) calculated as resistance index (RI) in isolated plots divided by RI in adjacent plots of five durum wheat genotypes at two assessment dates in adjacent plots with different sizes (experiment 2)

	Date 1		Date 2		
Genotype	4.5 m^2	1.5 m^2	4.5 m^2	1.5 m^2	Mean
Vavilov NI17403	1.0	2.0	1.7	1.6	1.6
21563-AA'S'	2.0	1.8	1.3	1.2	1.6
Amal 72	0.8	1.9	2.0	2.2	1.7
Giza 26	1.8	4.0	2.0	1.6	2.4
Jiloca	10.8	45.8	23.7	45.9	31.6

genotype i in isolated large plots and $RI_{j(adj)}$ is the resistance index in one of adjacent plot designs. RIR indicates the level of over- or underestimation of resistance for a genotype in a given adjacent plot design, assuming that the true level of resistance was measured in isolated plots. A high RIR indicates a large underestimation of the resistance in the adjacent plots. For each assessment date, Spearman's rank correlation coefficients (15) between sub-experiments (plot designs) were calculated across genotypes to test the influence of plot design on genotype ranking.

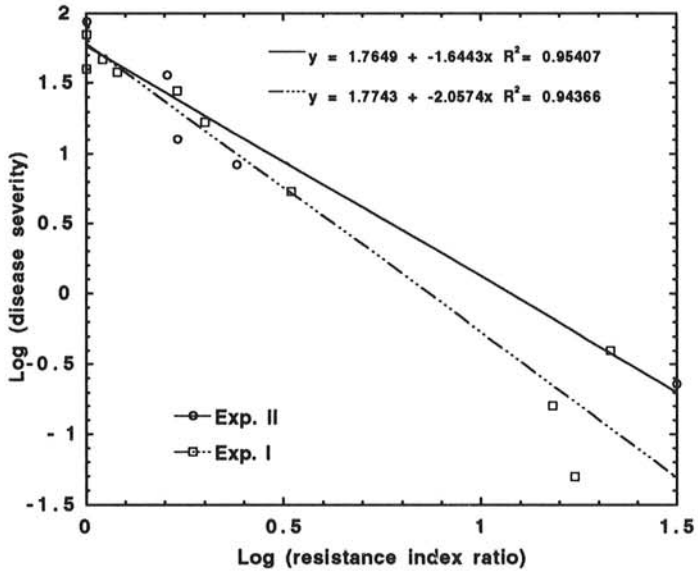

Fig. 1. Relationship between log-transformed mean disease severity in isolated plots and log-transformed mean resistance index ratio across adjacent plot designs and assessment dates in two experiments.

RESULTS

The stem rust epidemic progressed rapidly on the susceptible checks in both experiments. On the first assessment date in experiment 1 disease severity on Bansi 168 varied from 38.8 to 61.1% depending on the plot design (Table 2). Twelve days later (third assessment date), leaves of Bansi 168 were completely covered with stem rust in all plots. Disease severity on Morocco (experiment 2) varied from 69 to 75% on the first assessment date. Morocco was at 100% on the second assessment date (Table 3).

Significant genotypic differences for DS and thus for RI were detected in all plot designs and for all assessment dates in both experiments (Tables 4 and 5). The most susceptible genotypes were Bansi 168 in experiment 1 and Morocco in experiment 2. Jiloca was the most resistant genotype in both experiments (highest RI, Tables 2 and 3).

The interaction between genotype and plot design (G × D) was significant on assessment dates 2 and 3 of experiment 1 and on assessment date 1 of experiment 2 (Table 4). This interaction was also significant in the overall analysis in both experiments (Table 5). This means that, for a certain genotype, DS and thus RI varied with plot design. In isolated large plots, RIs were generally larger than in adjacent plots (Tables 2 and 3). Jiloca at the first assessment date of experiment 1 had an RI of 1,930.1; in adjacent large plots, the same genotype had an RI of 115.5. Hence, the resistance of Jiloca in adjacent large plots was underestimated by a factor 16.7 compared with the isolated large plots (Table 6). In experiment 2. at the first assessment date, the RI of Jiloca was 1,492 in isolated plots whereas in the large adjacent plots the RI was only 137.7 (Table 3), indicating an underestimation of resistance by a factor of 10.8 (Table 7). In general, both experiments gave similar RIR values. However, for some genotypes (e.g., 21563-AA'S') the RIR was much lower in experiment 2 than in experiment 1. This was related to a higher disease level (lower resistance level) in experiment 2 than in experiment 1 (Tables 2 and 3).

For both experiments, plants in isolated large plots had significantly lower average disease severity and higher average RI than the adjacent plot designs (Tables 2 and 3). The adjacent plot situations did not differ much in mean RI. This result shows that separation of plots has a major effect on epidemic development and genotypic differences. Differences in plot size in adjacent plot situations had only a small effect on epidemic development and genotypic differences.

RIR is a measure of the level of interplot interference in the different adjacent plot designs. It expresses the level of underestimation of resistance for a given genotype assuming that no interplot interference was present in the isolated plots. The level of underestimation depended on the level of resistance as measured in the isolated fields (Tables 6 and 7). In experiment 1, D115 (low level of resistance) varied in RIR from 0.9 to 1.4 with an average of 1.2, whereas the RIR of Jiloca (high level of resistance) varied from 7.7 to 45.1 with an average of 17.3. In experiment 2 similar results are found for Jiloca and 21563-AA'S'. For both experiments, the relationship between level of underestimation (RIR) and level of resistance expressed as disease level in isolated plots could be described by a linear equation of log-transformed DS in isolated plots and log-transformed RIR (mean across plot designs) (Fig. 1).

Spearman's rank correlation coefficients between different plot designs ranged from 0.88 to 0.99 in experiment 1 and from 0.88 to 0.98 in experiment 2. All correlation coefficients were highly significant ($P = 0.001$ for experiment 1 and $P = 0.05$ for experiment 2). Interplot interference did not significantly affect the ranking of genotypes in the two experiments.

DISCUSSION

Partial resistance is characterized by slow epidemic development despite a compatible interaction between host and pathogen (8). Selection for and evaluation of partial resistance in adjacent plots may be subjected to a representational error that underestimates resistance in adjacent fields (6,9,12,16). Our results indicate that expression of partial stem rust resistance in durum wheat was underestimated by interplot interference. The level of underestimation of resistance caused by interplot interference averaged a factor of 17.3 for the most resistant genotype in experiment 1. In experiment 2, the underestimation for the most resistant genotype was on average a factor of 31.6. Although annual variation exists due to environmental influences, the level of interplot interference observed in this study was much smaller than in barley leaf rust (8,9), in which estimates of resistance differed by a factor of 130. In contrast, barley powdery mildew showed lower levels of interplot interference, with genotypic differences in isolated plots only 3 times larger than in adjacent plots (6). In wheat yellow rust, genotypic differences in wheat yellow rust were not affected by interplot interference. Both isolated and adjacent plots gave similar genotype rankings and differences between genotypes (2).

Paysour and Fry (10) expected severe underestimation of treatment differences when dealing with pathogens like rusts and mildews that are expected to have flat disease gradients. Apparently, this generalization does not hold for the four diseases mentioned above. The four diseases suggest that a relationship exists between the level of systemic growth and the level of interplot interference. The more important systemic growth is for the epidemic development, the less important new infections will be. If new infections are relatively unimportant for disease development, the effect of inoculum exchange on the epidemic development will be smaller, and consequently the level interplot interference will be smaller.

The RIR, the ratio of RI measured in isolated plots and in adjacent plots, indicated that the level of interplot interference increased with increasing level of resistance in a nonlinear way. This can be explained in a simple model. Suppose genotype 1 is very susceptible and produces a large number of spores per unit time, say 100. Genotype 2 is moderately resistant and produces 50, and genotype 3, which is resistant, produces only 10 spores. Assume that isolated and adjacent plots of the same size lose the same proportion of inoculum, that the epidemic in isolated plots is determined by autoinfection only, and that the proportion of spores that participate in the epidemic reflects the genotypic differences in spore production. In adjacent plots, the epidemic also is determined by alloinfection due to spore import. Given these and other assumptions (10), inoculum increased 1.1 times for the susceptible genotype, 2 times for the intermediately resistant genotype, and 6 times for the resistant genotype (6 times increase). The relative importance of spore import increases in a nonlinear way with increasing levels of resistance resulting in higher levels of interplot interference, confirming our results.

The level of interplot interference is not determined by the genotype but by the resistance level of that genotype. Since the level of resistance of a genotype may vary with the environment, the level of interplot interference will also be affected. This is the case for Giza 26 and Amal 72. Their resistance levels were much lower in experiment 2 than in experiment 1 and as a consequence their level of interplot interference was much lower.

Paysour and Fry (10) defined an acceptable level of interplot interference as "one at which the epidemic behavior (e.g., area under disease progress curve) differs by no more than 10% from that which would be found in the absence of interplot interference." According to this definition, the level of interplot interference we observed would not be acceptable. Van der Plank (16), however, stated that the objective of an experiment determines whether or not one can accept the representational error regardless of the size of the error. In a breeding program large numbers of lines can only be tested in adjacent small plots. For breeders it is important to efficiently differentiate between susceptible and resistant genotypes (13). Our results showed that this was the case for all adjacent plot designs. Moreover, the genotype ranking in breeders' plots should be representative of the relative rankings in farmers' field. Spearman's rank correlation showed that the ranking of genotypes with different levels of partial resistance does not depend on the plot design. Using standard control varieties with different, known levels of partial resistance, the breeder can determine the level of disease severity acceptable for selection of partial resistance in small plot nurseries.

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