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EVALUATION OF SOME FLAX PLANT INTRODUCTIONS FOR POWDERY MILDEW RESISTANCE UNDER NATURAL INFECTION CONDITIONS

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ABSTRACT

A two-year outdoor pot experiment was conducted at Giza Agricultural Research Station to (1) estimate heritability of powdery mildew resistance when disease incidence (DI), and disease severity (DS), were used as criteria to evaluate resistance, (2) assess resistance to powdery mildew of 43 flax plant introductions. Genotype components of variance of both DI and DS were very highly significant ($p \leq 0.000$) each year indicating that extensive genetic variation for DI and DS was present within the tested genotypes. Heritability for DI ranged from 71.86 to 95.93%, while heritability for DS ranged from 91.59 to 94.35%. Genetic advance expected from selection based on DI ratings ranged from 29.21 to 50.78, while that based on DS ranged from 39.62 to 40.45. DI ratings ranged from 30 to 100% in 2001, and from 10.67 to 96.67% in 2002. DS ratings ranged from 15.85 to 100% in 2001, and from 15.13 to 92.47% in 2002. Selection of the introductions for powdery mildew resistance was based on DS and not DI because DS was environmentally more stable. Of the 43 flax plant introductions valuated in the present study, introductions nos. 1, 3, and 7 were the only introductions, which showed satisfactory levels of resistance in both years. Some of the other introductions were resistant to the disease in only one year, which indicates that their performance lacked stability. A part from introductions nos. 1, 3 and 7, most of the other genotypes did not have satisfactory levels of resistance in both years. The introductions 1, 3 and 7 could be used as new sources of resistance to enhance resistance to powdery mildew in the local breeding materials, which would reduce reliance on chemical fungicides for controlling the disease.

INTRODUCTION

Powdery mildew (PM) of flax (*Linum usitatissimum* L.) is currently the most common, conspicuous, widespread, and easily recognized foliar disease of flax. In Egypt, flax is grown for both seeds and fibers in the Nile Delta, in particular the northern governorates. This area is characterized by the prevalence of warm weather during the late period of flax growing season. Such weather favors epiphytotic spread of the disease when virulent isolates of the causal agent occur.

The fungus responsible for this disease has been recorded as a species of *Erysiphe* (Nyvall, 1981) but owing to the fact that the perfect stage of the fungus, which must be found and identified before the fungus can be referred to as a species of *Erysiphe*, has not been recorded in Egypt, the name of the fungus will be referred to in the present work as that of its imperfect (conidial) stage – *Oidium lini* Škoric (Muskett and Colhoun, 1947) – the only form in which it has been found in Egypt. The perfect stage of the pathogen has been

referred to in other countries as *Erysiphe polygoni* DC.ex Merat (Nyvall, 1981) and *E. cichoracearum* DC., (Agrios, 1988).

Under Egyptian conditions, owing to the absence of the perfect stage, it is not known for certain how the fungus survives from growing season to season. It may over winter on volunteer flax or other hosts (Nyvall, 1981).

Currently, resistance is not available in commercially grown flax cultivars in Egypt. Therefore, in years when environmental conditions favor the development of the disease, foliar applications of fungicides has become the only commercially available management practice for the disease (Mansour, 1998). Complete dependence on fungicides for the disease control carries risks for the producers, in that accurate coverage and distribution of fungicides may not be achieved and there are potential problems with correct timing of applications. Furthermore, increasing concern of the environment will likely mean greater regulation of fungicide usage (Pearce *et al.*, 1996).

One alternative strategy for control of PM of flax that has yet to be fully utilized is the identification and incorporation of host resistance genes into commercial cultivars. However, one obvious problem faced by flax breeders attempting to breed for resistance to PM is the lack of satisfactory levels of resistance in the local breeding materials (Aly *et al.*, 2001).

The first objective of this study was to estimate heritability of PM resistance when disease incidence (DI) or disease severity (DS) was used as criterion for evaluating resistance. A second objective was to screen 43 introduced flax genotypes for relative resistance to PM infection. If found, highly resistant genotypes could subsequently be incorporated into ongoing disease resistance breeding work.

MATERIALS AND METHODS

Flax plant introductions

Seeds of flax plant introductions were obtained from Institute fur Pflanzengenetik und Kulturpflanzenforschung (Plant Genetics and pLant Research Institute), Gatersleben, Germany.

Outdoor experiment

Seeds of flax genotypes were planted on 15 November 2000 and on 20 November 2001 in a natural soil dispensed in 25-cm diameter clay pots (20 seeds/pot). The pots were distributed outdoors in a randomized complete block design of three replications. PM was allowed to develop naturally. DI and DS (Nutter *et al.*, 1991) were rated visually on 25 April 2001 and on 19 April 2002. DI was measured as percentage of infected plants/pot. DS was measured as percentage of infected leaves/plant in a random sample of 10 plants/pot.

Genetic parameters:

1. Heritability in the broad sense (h^2) was calculated according to the following formula:

$$\frac{\text{Genotypic variance } (\sigma^2 g)}{\text{Phenotypic variance } (\sigma^2 ph)} \times 100 \text{ (Miller } et\ al.\text{, 1958)}$$

Where $\sigma^2g = [(\sigma^2e + r\,\sigma^2g) - \sigma^2e]/r$

$\sigma^2ph = [(\sigma^2e + r\,\sigma^2g)/r$

2. Genetic advance expected from selection (GA) was calculated according to the following formula: $(\sigma^2g / \sigma^2ph)\ K \times \sqrt{\sigma^2ph}$,

where K = 2.06 at 5% selection intensity (Miller *et al.*, 1958).

Statistical analysis of the data

Analysis of Variance (ANOVA) was performed on disease intensity variables (DI and DS) to determine genotype effects. Mean comparisons for variables were made among genotypes by using Duncan's multiple range test. ANOVA of the data was performed with the MSTAT-C Statistical Package (A Microcomputer Program for the Design, Management and Analysis of Agronomic Research Experiments, Michigan State Univ., USA).

RESULTS AND DISCUSSION

The present study was conducted in 2000/2001 and 2001/2002 growing seasons (hereafter referred to as years 2001 and 2002, respectively) to evaluate 43 introduced flax genotypes for relative resistance to PM infection. Of the 43 genotypes, 3 were of unknown origin, while the remaining 40 genotypes originated from 20 countries (Table 1). Bulgaria, Italy, and Russia were represented by 11 (25.6%), 4 (9.3%), and 3 (10%) genotypes, respectively, while each of the other countries was represented by 1 (2.3%) or 2 (4.7%) genotypes.

Genotype components of variance of both DI and DS were very highly significant ($P < 0.000$) each year indicating that extensive genetic variation for DI and DS was present within the tested genotypes (Tables 2 and 3). In addition, high heritability values for DI and DS was obtained each year demonstrating the genetic bases for DI and DS (Table 4). The high estimate of DI and DS heritabilities each year clearly demonstrated that considerable progress in breeding for PM resistance could be expected in current breeding programs if DI or DS is used as criterion for evaluating resistance. Thus, GA based on DI ratings ranged from 29.21 to 50.78, while that based on DS ratings ranged from 39.62 to 40.45. DI heritability showed considerable variation from year to year, while DS heritability changed slightly from one year to another (Table 4). Therefore, it seems reasonable to conclude that DS, in comparison with DI, was less affected by the changes in environmental conditions from year to year.

All the genotypes under consideration were symptomatic, which indicated that natural conditions and levels of inoculum in 2001 and 2002 were favorable for the disease development. DI ratings ranged from 30 to 100% in 2001 and from 10.67 to 96.67 in 2002. DS ratings ranged from 15.85 to 100% in 2001, and from 15.13 to 92.47% in 2002 (Tables 5 and 6). It is noteworthy that, in 2001, the general means of DI and DS ratings of the 43 genotypes were 83.41 and 85.88%, respectively; however, in 2002, the general means of DI and DS ratings decreased to 64.41 and 59.31%, respectively. These reductions in disease intensity ratings in 2002 could be due to less favorable

environmental conditions and/or less virulent physiological races of the pathogen (Leath *et al.*, 1991).

Table 1.Geographic origins of flax genotypes used in tne present study.

No.	Genotype	Geographic origin
1	Line 187/78	Argentina
2	Line 367/78	Hungary
3	Line 370/78	Ukraine
4	Line 376/78	Portugal
5	Line 387/78	Italy
6	Line 407/87	China
7	Line 493/79	Bulgaria
8	Line 929/79	Bulgaria
9	Line 931/81	Bulgaria
10	Line 935/95	Bulgaria
11	Line 937/80	Bulgaria
12	Line 938/79	Bulgaria
13	Line 939/79	Bulgaria
14	Line 940/79	Bulgaria
15	Line 943/79	Bulgaria
16	Line 944/99	Bulgaria
17	Line 950/75	Bulgaria
18	Line 951/79	Latvia
19	Line 1038/81	China
20	Line 1070/83	Lithuania
21	Line 1117/81	Spain
22	Line 1150/80	Turkey
23	Line 1169/80	Chile
24	Line 1173/80	Brazil
25	Line 1196/83	India
26	Line 1236/81	Turkey
27	Line 1676/84	Russia
28	Line 1679/85	Russia
29	Line 1680/85	Russia
30	Line 1744/90	Italy
31	Line 1766/90	Korea
32	Line 1778/90	Korea
33	Line 1792/90	Italy
34	Line 1796/92	Bulgaria
35	Line 1868/95	Georgia
36	Line 1870/96	Ukraine
37	Line 1195/83	Spain
38	Line 212/78	Ethiopia
39	Line 947/96	Unknown
40	Line 978/78	Unknown
41	Line 979/78	Unknown
42	Line 1767/90	Italy
43	Line 612/95	Hungary

Table 2.Form and expected mean squares for analysis of variance of powdery mildew incidence data from 43 flax genotypes screended for relative resistance in an outdoor pot experiment in Giza.

Source of variation	D.F.	2000/2001			2001/2002			Expected mean square[a]
		M.S.	F. value	P > F	M.S.	F. value	P > F	
Replication	2	481.171	1.4637	0.2372	5316.147	65.9574	0.0000	$\sigma^2e + g\sigma^2r$
Genotype	42	1168.315	3.5540	0.0000	1981.204	24.5808	0.0000	$\sigma^2e + r\sigma^2g$
Error	84	328.734			80.600			σ^2e

[a] σ^2e, σ^2r and σ^2g are variances due to experimental error, replications, and genotypes, respectively; g and r, respectively, are no. of genotypes and no. of replications.

Table 3.Form and expected mean squares for analysis of variance of powdery mildew severity data from 43 flax genotypes screended for relative resistance in an outdoor pot experiment in Giza.

Source of variation	D.F.	2000/2001			2001/2002			Expected mean square[a]
		M.S.	F. value	P > F	M.S.	F. value	P > F	
Replication	2	393.292	5.5850	0.0053	10948.987	94.4544	0.0000	$\sigma^2e + g\sigma^2r$
Genotype	42	1246.528	17.7016	0.0000	1378.996	11.8963	0.0000	$\sigma^2e + r\sigma^2g$
Error	84	70.419			115.918			σ^2e

[a] σ^2e, σ^2r and σ^2g are variances due to experimental error, replications, and genotypes, respectively; g and r, respectively, are no. of genotypes and no. of replications.

Table 4.Heritability in the broad sense (h^2) and genetic advance expected from selection (GA) for powdery mildew intensity variables for 43 flax genotypes screended for relative resistance in an outdoor pot experiment in Giza.

Year	Powdery mildew incidence		Powdery mildew severity	
	h^2	GA	h^2	GA
2000/2001	71.86	29.21	94.35	39.62
2001/2002	95.93	50.78	91.59	40.45

At this point, the question that may arise is which criterion should be used in screening genotypes for PM resistance, DI or DS? From a practical point of view, DI is more appropriate than DS for screening breeding materials because it is more easily acquired (Rouse *et al.*, 1981), which would greatly facilitate the selection process. However, from a genetic point of view, DS is more appropriate than DI for screening breeding materials because it was environmentally more stable, which implies that selection of the genotypes with low DS over a number of generations would greatly improve the resistance to PM. Therefore, in the present study, selection of flax genotypes for PM resistance was based on DS and not DI.

Table 5.Powdery mildew incidence[a] for 43 flax genotypes screened for relative resistance in an outdoor pot experiment in Giza.

No.	Genotype	2000/2001	2001/2002
1	Line 187/78	30.00 F [b]	16.00 PQ
2	Line 367/78	63.33 A-F	10.67 Q
3	Line 370/78	55.67 B-F	22.67 O-Q
4	Line 376/78	75.67 A-D	28.00 M-P
5	Line 387/78	87.33 A-G	32.00 M-P
6	Line 407/87	87.33 A-G	21.33 O-Q
7	Line 493/79	47.67 D-F	28.00 M-P
8	Line 929/79	53.33 C-F	29.33 M-P
9	Line 931/81	100.00 A	25.33 N-Q
10	Line 935/95	87.67 A-C	61.33 G-J
11	Line 937/80	64.67 A-E	64.00 F-J
12	Line 938/79	65.00 A-E	64.00 F.J
13	Line 939/79	76.67 A-D	58.67 H-J
14	Line 940/79	38.00 EF	37.33 L-O
15	Line 943/79	97.33 A	40.00 K-N
16	Line 944/99	70.00 A-E	42.67 K-M
17	Line 950/75	57.00 B-F	50.67 J-L
18	Line 951/79	90.67 AB	77.67 B-G
19	Line 1038/81	93.00 A	54.67 I-K
20	Line 1070/83	100.00 A	85.00 A-D
21	Line 1117/81	97.00 A	83.33 A-D
22	Line 1150/80	94.00 A	79.67 A-F
23	Line 1169/80	96.33 A	83.67 A-D
24	Line 1173/80	97.33 A	84.00 A-D
25	Line 1196/83	97.67 A	85.00 A-D
26	Line 1236/81	57.33 B-F	80.33 a-f
27	Line 1676/84	94.00 A	74.33 C-H
28	Line 1679/85	94.00 A	83.67 A-D
29	Line 1680/85	95.33 A	87.33 A-C
30	Line 1744/90	100.00 A	76.33 B-G
31	Line 1766/90	100.00 A	86.33 A-C
32	Line 1778/90	100.00 A	91.00 A-C
33	Line 1792/90	100.00 A	90.00 A-C
34	Line 1796/92	99.33 A	91.67 A-C
35	Line 1868/95	94.67 A	66.00 E-J
36	Line 1870/96	99.67 A	67.67 D-I
37	Line 1195/83	99.67 A	83.00 A-E
38	Line 212/78	97.67 A	86.67 A-C
39	Line 947/96	100.00 A	93.67 AB
40	Line 978/78	66.67 A-E	96.67 A
41	Line 979/78	66.67 A-E	90.33 A-C
42	Line 1767/90	100.00 A	73.67 C-H
43	Line 612/95	100.00 A	86.00 A-C
	Mean	83.41	64.41

[a] Disease icnidence is the percentage of infected plants/pot.
[b] Means followed by the same letter(s) are not signifcantly different ($P \leq 0.05$) according to Duncan's multiple range test.

Table 6.Powdery mildew severity[a] for 43 flax genotypes screended for relative resistance in an outdoor pot experiment in Giza.

No.		2000/2001		2001/2002	
		%	Ranking	%	Ranking
1	Line 187/78	15.85 H[b]	1	15.13 NO	2
2	Line 367/78	57.70 F	5	6.73 O	1
3	Line 370/78	34.01 G	2	24.17 M-O	4
4	Line 376/78	61.22 EF	7	31.35 L-N	7
5	Line 387/78	86.94 A-C	12	25.95 M-O	5
6	Line 407/87	90.30 AB	14	15.21 NO	3
7	Line 493/79	38.43 G	3	27.13 MN	6
8	Line 929/79	73.78 C-E	10	48.66 H-L	11
9	Line 931/81	94.18 AB	19	36.90 J-M	9
10	Line 935/95	95.66 A	23	74.30 A-F	32
11	Line 937/80	95.85 A	24	61.19 D-I	19
12	Line 938/79	99.53 A	38	79.08 A-E	39
13	Line 939/79	91.06 AB	15	60.92 D-I	18
14	Line 940/79	57.95 F	6	35.48 K-M	8
15	Line 943/79	92.56 AB	17	43.33 I-M	10
16	Line 944/99	68.21 D-F	8	52.49 G-K	13
17	Line 950/75	94.36 AB	21	48.82 H-L	12
18	Line 951/79	97.75 A	29	68.10 B-H	24
19	Line 1038/81	99.19 A	33	65.10 D-H	20
20	Line 1070/83	95.64 A	22	71.94 A-G	31
21	Line 1117/81	94.35 AB	20	78.51A-E	37
22	Line 1150/80	98.37 A	30	56.07 F-J	15
23	Line 1169/80	93.07 AB	18	69.84 B-H	25
24	Line 1173/80	89.22 AB	13	89.37 AB	42
25	Line 1196/83	95.95 A	26	74.42 A-F	33
26	Line 1236/81	57.28 F	4	70.67 B-G	27
27	Line 1676/84	69.89 D-F	9	70.15 B-G	26
28	Line 1679/85	78.31 B-D	11	66.42 C-H	21
29	Line 1680/85	99.28 A	35	78.28 A-E	36
30	Line 1744/90	99.28 A	36	66.91 C-H	22
31	Line 1766/90	99.93 A	42	53.43 F-K	14
32	Line 1778/90	99.90 A	40	67.17 C-H	23
33	Line 1792/90	99.78 A	39	75.00 A-F	35
34	Line 1796/92	99.91 A	41	74.92 A-F	34
35	Line 1868/95	99.44 A	37	58.37 E-I	17
36	Line 1870/96	97.29 A	28	56.45 F-J	16
37	Line 1195/83	100.00 A	43	71.81 A-G	30
38	Line 212/78	99.24 A	34	80.21 A-D	40
39	Line 947/96	98.72 A	32	87.05 A-C	41
40	Line 978/78	92.14 AB	16	92.47 A	43
41	Line 979/78	98.54 A	31	78.59 A-E	38
42	Line 1767/90	95.94 A	25	71.02 B-G	29
43	Line 612/95	96.74 A	27	70.80 B-G	28
	Mean	85.88		59.31	

[a] **Disease severity is the percentage of infected leaves/plant in a random sample of 10 plants/pot.**

[b] **Means followed by the same letter(s) are not signifcantly different ($P \leq 0.05$) according to Duncan's multiple range test.**

Disease resistance genotype should meet two requirements: First, it should have a satisfactory level of resistance. Second, it should have a stable performance when it is grown under different environments. Of the 43

genotypes evaluated in the present study, genotypes Nos. 1, 3 and 7 were the only genotypes, which met these requirements because they showed low DS in both years (Table 6). The rankings of the 3 genotypes changed very slightly from one year to another, which indicates that they had a stable performance in resisting the disease. Some of the other genotypes, like genotypes Nos. 2, 4 and 5, were resistant to the disease; however, their resistance was restricted to one year – that is, their performance lacked stability. Apart from genotypes 1, 3 and 7, most of the other genotypes did not have satisfactory levels of resistance in both years. The genotypes 1, 3, and 7 may be used as new sources of resistance to enhance PM resistance in the local breeding materials, which would reduce reliance on chemical fungicides for controlling the disease.

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تقييم بعض مستوردات الكتان من حيث المقاومة لمرض البياض الدقيقى تحت ظروف العدوى الطبيعية

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أجريت الدراسة الحالية لمدة عامين خارج الصوبة فى محطة البحوث الزراعية بالجيزة، وكانت أهداف الدراسة هى على النحو التالى: (١) تقدير معامل التوريث لصفة مقاومة الكتان لمرض البياض الدقيقى عند استعمال حدوث المرض أو شدة المرض كمعيار لتقييم صفة المقاومة. (٢) تقييم ٤٣ تركيب وراثى مستورد للكتان من حيث المقاومة لمرض البياض الدقيقى. أظهرت الدراسة أن التراكيب الوراثية المختبرة كانت مصدراً عالى المعنوية للتباين فى حدوث المرض وشدته فى كل عام من عامى الدراسة. تراوحت قيمة معامل التوريث لصفة حدوث المرض من ٧١,٨٦ إلى ٩٥,٩٣%، فى حين تراوحت قيمة معامل التوريث لصفة شدة المرض من ٩١,٥٩ إلى ٩٤,٣٥%. تراوح مدى التحسين الوراثى المتوقع من الانتخاب لصفة حدوث المرض من ٢٩,٢١ إلى ٥٠,٧٨، فى حين تراوح مدى التحسين الوراثى المتوقع من الإنتخاب لصفة شدة المرض من ٣٩,٩٢ إلى ٤٠,٤٥. تراوح حدوث المرض للتراكيب الوراثية المختبرة من ٣٠ إلى ١٠٠% فى عام ٢٠٠١، ومن ١٠,٦٧ إلى ٩٦,٦٧% فى عام ٢٠٠٢، أما شدة المرض فقد تراوحت من ١٥,٨٥ إلى ١٠٠% فى عام ٢٠٠١، ومن ١٥,١٣ إلى ٩٢,٤٧% فى عام ٢٠٠٢. استعملت شدة المرض – وليس حدوث المرض – كمعيار لانتخاب التراكيب الوراثية المقاومة للمرض لأن شدة المرض كانت اقل تأثراً بالتغيرات فى الظروف البيئية من حدوث المرض. التراكيب الوراثية ١ و ٣و ٧ هى الوحيدة التى أظهرت مستويات مقبولة من المقاومة للمرض فى كل عام من عامى الدراسة. على الرغم من أن بعض التراكيب الوراثية الأخرى أظهرت مستويات مقبولة من المقاومة للمرض، إلا أن هذه المقاومة كانت مقصورة على عام واحد دون الآخر، مما يدل على أن أداء هذه التراكيب الوراثية فى مقاومة المرض كان يفتقر إلى صفة الثبات. أغلب التراكيب الوراثية – فيما عدا التراكيب ١ و ٣ و ٧ – كانت تفتقر إلى مستويات مقبولة من المقاومة للمرض فى كل عام من عامى الدراسة. تدل نتائج الدراسة الحالية على أنه من الممكن استعمال التراكيب الوراثية ١ و ٣ و ٧ لرفع درجة مقاومة مواد التربية المحلية لمرض البياض الدقيقى، مما سيقلل من درجة الاعتماد على المبيدات الفطرية الكيماوية فى مقاومة هذا المرض.