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Further contributions to the development of a differential set of pea cultivars (*Pisum sativum*) to investigate the virulence of isolates of *Aphanomyces euteiches*

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Abstract

Common root rot (*Aphanomyces euteiches* Drechs.) has become a very destructive disease in the French pea crops since 1993. For an accurate investigation of the virulence variability among French *A. euteiches* populations and between French and foreign populations, a new set of differential pea genotypes was developed. Thirty-three American and European pea lines, displaying different levels of resistance, were screened in a growth chamber against two French isolates. Symptoms (disease severity from 0 to 5, evaluating symptom surface on roots and epicotyl) and percentage of top fresh weight (inoculated/uninoculated top fresh weight ratio) were measured. From this screening 12 relatively resistant lines, from various genetic backgrounds, were identified along with a highly susceptible control. This set of 13 genotypes was inoculated under controlled conditions with 14 isolates from France, Sweden, USA, Canada and New Zealand, to investigate genotype–isolate interactions. Root symptoms were rated (disease severity), and a susceptibility/resistance threshold was established at disease severity = 1. Significant quantitative interactions were observed, and five 'resistance patterns' were identified, leading to a set of six pea genotypes: Baccara (susceptible), Capella, MN313, 902131, 552 and PI180693. Fields trials of this set in 1999 and 2000 gave the same resistance rankings than in growth chamber conditions. This set will allow more accurate assessments of the variability in virulence/aggressiveness of *A. euteiches* isolates from France and foreign countries, and further investigations of the epidemiological and genetic basis of pea–*A. euteiches* interactions.

Introduction

Aphanomyces euteiches Drechs., a soilborne oomycete pathogen, was first described in Wisconsin (Jones and Drechsler, 1925) as the causal agent of the common root rot of pea. It has since been reported in North America, Europe, Japan, Australia and New Zealand (Wade, 1955; Yokosawa et al., 1974; Manning and Menzies, 1980; Hagedorn, 1984; Persson et al., 1997), becoming the major factor reducing pea production throughout the world. In the absence of efficient control methods (Papavizas and Ayers, 1974), the most promising way of controlling the disease has been through the development of resistant varieties. The efficiency and durability of a resistance breeding strategy relies on both the availability of a germplasm with a good level of resistance, and the knowledge of the variability of the pathogen.

A great breeding effort has been done in the USA since the 1960s (Lockwood, 1960; Lockwood and Ballard, 1960), and many lines, displaying partial resistance, have been released by the public breeders (Davis et al., 1976; 1995; King et al., 1981; Kraft, 1981; 1989; 1992; Gritton, 1992; 1995). Moreover, an extensive evaluation programme has been conducted within the Pullman Plant Introduction collection to identify new sources of resistance (Malvick and Percich, 1996; 1999). In Sweden, some resistant/tolerant lines were also reported (Engqvist, 1992), including Capella which is the only tolerant commercial variety known (registered by Svalov AB in 1990).

In France, pea crops have considerably increased since the beginning of the 1980s. However, further extension of this crop has been limited in the last few years by the development of Aphanomyces root rot, which has caused major crop losses, mainly in the most productive regions of the Parisian Basin (Didelot and Chaillet, 1995). The evaluation under French field conditions of some of the best American resistant pea lines gave mixed results. This contrast may be due to the use of different resistance evaluation methodologies, but is most likely related to differences of aggressiveness or virulence that may exist between French and American populations of the pathogen.

The pathogenic variability of *A. euteiches* has been studied in the United States (Beute and Lockwood, 1967; Malvick and Percich, 1998a), in Sweden (Sundheim, 1972) and New Zealand (Manning and Menzies, 1984), using sets of differential pea lines as descriptors. However, the genetic basis of the resistance of these differential lines was not assessed, and no global study was made to evaluate and characterise the overall variability of the pathogen, on an international scale. No data exist on the pathogenic variability of the pathogen in France, and we lacked useful tools, particularly differential series, to describe it. Preliminary resistance tests showed that the lines of the most recent differential set (Malvick and Percich, 1998a) could not distinguish groups within the French strains.

The main objective of this study was to develop a set of differential pea lines that could assess the pathogenic variability among French isolates, and between French and foreign isolates. A three-step procedure was followed. Screening of many wild accessions as well as breeders' lines identified material from different genetic backgrounds and with a good level of resistance to the French strains. From this screening test, a group of 13 lines, called the pre-set, was identified, with the aim of finding differential genotype–isolate interactions, although the genetic determinism of the resistance remains unknown. The pre-set was then confronted with two groups of *A. euteiches* isolates, one from France and the other from different foreign countries. From this confrontation, different resistance patterns, as well as interaction patterns, could be found. These patterns allowed the identification of the most accurate lines to constitute an adequate pea differential set. The second objective was to compare the seedling resistance displayed by this set in controlled conditions to its resistance/tolerance behaviour at a more developed stage in natural field conditions.

Materials and methods

Pisum sativum *genotypes*

Thirty-three pea lines/varieties were evaluated (Table 1). The pea breeding lines were chosen according to their geographical and breeding origin, and their degree of resistance or tolerance (Davis et al., 1995; Gritton, 1995; Kraft, 1989; 1992; Kraft et al., 1998). Some were dry pea varieties registered in France and Sweden, and others, known to have a degree of resistance, were released by US public breeders: J.M. Kraft (USDA-ARS Prosser), E.T. Gritton (University of Wisconsin – Madison), and D.W. Davis (University of Minnesota – Twin Cities). Some of these breeder's lines (especially those of Gritton and Kraft) were already sorted as partially resistant from preliminary field trials in France in 1995 and 1997. Among the Plant Introductions (PI) from the USDA pea germplasm collection of Pullman, some were chosen from the best accessions according to the studies of Malvick and Percich (1996; 1999). More details on these accessions are available in the USDA Germplasm Resources Information Network (http://www.ars-grin.gov/cgi-bin/npgs/html/desc_form.pl?177).

Aphanomyces euteiches *isolates*

The 14 isolates of *A. euteiches* tested in this study were isolated from France and foreign countries (Table 2), and were all pathogenic on pea. The French isolates were sampled from soil by baiting with pea seedlings (*Pisum sativum* cv. Baccara) and isolated from diseased root tips according to Wicker et al. (2001), except Ae169 which was isolated on diseased lentil (*Lens culinaris*) from a naturally infested field. The foreign

Table 1. *Pisum sativum* accessions evaluated for resistance and tolerance against *Aphanomyces euteiches*

Name/code	Status	Origin	Reference or collection
BACCARA	Spring dry pea variety	France	Ets DESPREZ
SOLARA	Spring dry pea variety	Netherlands	Ets CEBECO
MISTRAL	Winter dry pea variety	France	Ets SERASEM
CAPELLA	Spring dry pea variety	Sweden	Svalöf Weibull AB
AGAT	Pea line	Belarus	Vavilov Plant Introduction Station
EFFEKT	Pea line	Belarus	Vavilov Plant Introduction Station
WI8904	Canning pea breeding line	USA	E.T. Gritton, 1992
WI9416	Canning pea breeding line	USA	E.T. Gritton, 1995
200	Canning pea breeding line	USA	RRRS cycle #8 – E.T. Gritton, 1995
552	Canning pea breeding line	USA	Cycle #8 RRRS – resistant to powdery mildew – E.T. Gritton, 1995
560	Canning pea breeding line	USA	Cycle #8 RRRS – resistant to powdery mildew – E.T. Gritton, 1995
90-2079	Canning pea breeding line	USA	J.M. Kraft, 1992
90-2131	Canning pea breeding line	USA	J.M. Kraft, 1992
90-2322	Canning pea breeding line	USA	J.M. Kraft, 1992
MN313	Canning pea breeding line	USA	D.W. Davis et al., 1995
MN314	Canning pea breeding line	USA	D.W. Davis et al., 1995
MELROSE	Pea line	USA	USDA Plant Introduction Station, Pullman
PI169600	Pea line	Turkey	USDA Plant Introduction Station, Pullman
PI180693	Pea line	Germany	USDA Plant Introduction Station, Pullman
PI210641	Pea line	USA	USDA Plant Introduction Station, Pullman
PI210642	Pea line	USA	USDA Plant Introduction Station, Pullman
PI244158	Pea line	Netherlands	USDA Plant Introduction Station, Pullman
PI244162	Pea line	Netherlands	USDA Plant Introduction Station, Pullman
PI257593	Pea line	Ethiopia	USDA Plant Introduction Station, Pullman
PI269802	Pea line	UK	USDA Plant Introduction Station, Pullman
PI272212	Pea line	Ethiopia	USDA Plant Introduction Station, Pullman
PI306592	Pea line	Hungary	USDA Plant Introduction Station, Pullman
PI343984	Pea line	Turkey	USDA Plant Introduction Station, Pullman
PI343986	Pea line	Turkey	USDA Plant Introduction Station, Pullman
PI393487	Pea line	Czech Republic	USDA Plant Introduction Station, Pullman
PI413686	Pea line	Hungary	USDA Plant Introduction Station, Pullman
PI413696	Pea line	Hungary	USDA Plant Introduction Station, Pullman
PI471166	Pea line	India	USDA Plant Introduction Station, Pullman

isolates, kindly provided by researchers, were all baited on pea except Ae173, which was baited on green bean. These bulk-isolates were characterised as *A. euteiches* according to the key of Scott (1961), maintained on corn meal agar (CMA) (Difco, Detroit, MI) slants at 10 °C, and transferred every six months on fresh CMA slants.

Screening of pea germplasm

The 33 pea lines were inoculated with two French isolates of *A. euteiches*: Ae5 and Ae44. Zoospores of *A. euteiches* were produced (Wicker et al., 2001), counted with a Malassez hemacytometer, and the inoculum concentration adjusted to 200 zoospores ml^{-1}. Each line was tested: inoculated with either of the two isolates or uninoculated. Each treatment consisted of a tray with five randomized plastic pots containing 250 ml of vermiculite (VERMEX M, Bretagne Matériaux, 35570 L'Hermitage, France). These five pots were considered as replicates. Four pea seeds were sown per pot, and the vermiculite was dampened with tap water. Plants were grown in a growth chamber with a 16 h light : 8 h dark regime, and with a temperature regime of 25 and 23 °C, respectively.

Seven-day-old seedlings were inoculated by dispensing 20 ml of a zoospore suspension on the vermiculite over the entire area of the pot, providing a total of 10^3 zoospores per plant. The same quantity of water was dispensed on the uninoculated control. Vermiculite was saturated with water to optimise infestation, and was watered as required over the following days to keep the substrate moist. Seven days

Table 2. Aphanomyces euteiches isolates inoculated on pea genotypes for the development of a set of differential pea genotypes

Code	Name	Date of isolation	Geographical origin		Host specificity[2]	
			Country	Region[1]	VP	Pathotype
Ae5		1995	France	EPB	PVAF	B
Ae6		1995	France	EPB	PVA	B
Ae33		1996	France	WPB	PVA	B
Ae40		1996	France	South-West	PVAF	B
Ae44		1996	France	EPB	PVAF	B
Ae78		1996	France	EPB	PVAF	A
Ae83		1997	France	Brittany	PVA	B
Ae84		1997	France	South-West	PVAF	A
Ae169		1998	France	EPB	PVAFB	
Ae147	LP83	1994	Sweden		PVA	
Ae139	ICMP12294	1994	New Zealand		PVAF	
Ae163	SRSF-0671	Unknown	Canada		PVA	
Ae164	SRSF-0672	Unknown	Canada			
Ae172	P54	Unknown	USA		PVA	
Ae173	B16	Unknown	USA		PVAF	

[1]Regions of origin of the isolates comprise the following departments: Eastern Parisian Basin (EPB): Seine-et-Marne, Aisne, Marne, Val de Marne. Western Parisian Basin (WPB): Eure, Eure-et-Loir, Loir-et-Cher, Seine-Maritime, Yvelines. Brittany: Finistère and Morbihan. South-West: Charente-Maritime and Pyrénées-Atlantiques.

[2]The host specificity was described by 'virulence phenotypes' (VP) groups and 'pathotypes' (Wicker et al., 2001). (P, V, A, F, B = pathogenic on pea, vetch, alfalfa, broad bean, green bean, respectively) – the pathotype A was at least moderatly aggressive on all five species; the pathotype B was aggressive on pea, mildly aggressive on vetch and alfalfa, poorly aggressive on broad bean.

after inoculation, all plants were removed from the vermiculite and disease severity (DS) was assessed visually according to a 0–5 scale: (0) no symptoms; (1) discoloured traces on rootlets; (2) discoloured to honey-brown zones on rootlets and part of the tap root, covering at least one-fourth of the root system; (3) honey-brown, soft zones covering at least half of the root system, epicotyl in some cases discoloured and water-soaked, but still firm; (4) most of the root system soft and honey-brown to dark brown, epicotyl soft and brown, shrivelled, first leaves yellowing; (5) plant dead. In addition, plants were cut just above the cotyledonary insertion zone, and the fresh weight of the plant aerial parts was recorded. The plants of each pot were counted and bulked before being weighed. The fresh weight of a pot was considered as the fresh weight of four plants.

Percentage of Fresh Weight (PFW) of a line was calculated as: (Fresh weight of the inoculated treatment $*$ (Fresh weight of the uninoculated treatment)$^{-1}$) $*$ 100, based on equal numbers of inoculated and uninoculated plants.

Reaction of the pre-set of P. sativum *lines to French and foreign isolates of* A. euteiches

From the 33 pea lines tested, 13 were selected to constitute a 'pre-set' of differential pea genotypes, which was inoculated with two groups of isolates of *A. euteiches*. The first group constituted seven isolates, displaying a lot of variability according to geographical origins: Europe (Ae5 and Ae147), Canada (Ae163 and Ae164), the USA (Ae172 and Ae173) and New Zealand (Ae139). The second group constituted eight French isolates (Ae5, Ae6, Ae33, Ae40, Ae78, Ae83, Ae84, Ae169), chosen according to their degree of host specificity on five legume species (Wicker et al., 2001) and to their diverse geographical origin (Table 2). Each group was tested in a separate experiment, with Ae5 being tested in both dates as a reference. The experimental design, method and dose of inoculation, and incubation conditions were as described above, except that there was no uninoculated control for each pea genotype.

At this stage, the level of disease was described by the DS. The genotype, isolate and genotype $\times$ isolate

interaction effects on DS were analysed for variance (ANOVA). The DS difference of each genotype related to the susceptible control Baccara was evaluated by ΔDS. ΔDS was calculated for each genotype : isolate combination according to the formula: $\Delta DS_{genotypeX:isolateY} = DS_{Baccara:isolateY} - DS_{genotypeX:isolateY}$. A pathogenicity threshold was used to establish 'patterns of resistance to the infection' within this pre-set. According to Malvick and Percich (1998b), a plant should be considered susceptible to *A. euteiches* if the pathogen can be reisolated from the roots after inoculation, and/or if oospores are observed within the roots. Preliminary experiments (Wicker et al., 2001) showed us that pathogen isolation and oospore observations were possible if the root symptoms resulted in a DS of 1 or higher. Therefore, the pathogenicity threshold was determined at DS = 1: if the mean DS is equal or superior to 1, the plant is considered susceptible; if it is below 1, the plant is considered resistant to the infection.

Comparison between the seedling and field reaction of the set of six pea genotypes

The correlation was examined between the resistance of young seedlings as assessed in a growth chamber, and the resistance/tolerance of grown up plants as assessed in the field under natural conditions. For that purpose, the genotype rankings of the range under controlled and natural conditions were compared, and correlation analyses were made between the resistance variables in growth chamber and in the field.

The six genotypes of the final set were inoculated nine times with Ae5, incubated and rated (disease severity) under the conditions described above. The genotype, experiment effects on DS were analysed for variance (ANOVA) to assess the reproductibility among experiments. The DS data considered for correlation analysis were the mean results of the six lines over the nine experiments.

The six pea genotypes of the final range were evaluated for resistance in a naturally infested field at Courtacon (France). Peas were sown on 1st April in 1999 and 23rd March in 2000 on a two-row plot size of 2.50 m in a complete block design with three replicates. In both years, the DS was evaluated by two indices. A root disease index (RDI) was rated on 12 plants per plot at the 5–6 leaf stage according to the 0–5 scale described above. Ratings were made 41 days after sowing in 1999, and 49 days after sowing in 2000. An aerial index (AI) evaluating the stunting and yellowing symptoms of the plant aerial parts was recorded for each plot as a whole, on a 1–9 scale, with 1 = healthy plants and 9 = dead plants (Duparque and Devaux-Boitel, 2001). In 1999, AI was measured at the beginning of flowering (AI1, 56 days after sowing) and during the seed set period (AI2, 69 days after sowing). In 2000, ratings for AI1 were made at the beginning of the seed set period (75 days after sowing) and for AI2 at the end of the seed set period (84 days after sowing).

Data analysis

Genotype effects on DS and PFW values, genotype, isolate and genotype × isolate interaction effects on DS in the test of the preset, were analysed for variance (ANOVA) and means were compared with the Newman–Keuls test ($p = 0.05$), using the GLM procedure of the software SAS (SAS Institute Inc., Cary, NC).

Genotype and experiment effects on mean DS from growth chamber tests, and on RDS and AI1 and AI2 results from field trials, were analysed for variance (ANOVA) and means were compared as described above.

Pearson correlation coefficients were calculated between the mean DS results incited by Ae5 on the pea set in growth chamber, and the RDS and aerial indices at the first (AI1) and second date (AI2) observed in the field in 1999 and 2000, using the PROC CORR of the SAS software (SAS Inc., Cary, NC, USA).

Results

Screening of pea germplasm

Seven days after inoculation, the genotype effect on disease ratings was significant ($p = 0.05$), but no completely resistant genotypes were found among the 33 genotypes tested (Table 3). The variation appeared to be continuous, from moderate to highly susceptibility. Ae5 was clearly more aggressive than Ae44, causing a higher DS and a lower PFW. The rankings of pea lines from susceptible to resistant were correlated between isolates, for DS ($r = 0.64$, $p < 0.01$) and for PFW ($r = 0.62$, $p < 0.01$).

Table 3. Mean disease severities on roots and epicotyl and per cents of top fresh weight on 33 pea genotypes, 7 days after inoculation with two *Aphanomyces euteiches* isolates

Disease severity[1]				Per cent of the top fresh weight[2]			
Genotype	Origin[3]	Ae5[4]	Ae44	Genotype	Origin	Ae5	Ae44
SOLARA	FR	4.53 *a*	3.55 *b*	PI272212	P	42.01 *a*	53.88 *ab*
BACCARA	**FR**	**4.20 *b***	**4.40 *a***	SOLARA	FR	43.96 *a*	62.03 *abc*
EFFEKT	B	4.00 *bc*	3.20 *bcd*	**BACCARA**	**FR**	**50.52 *ab***	**49.69 *a***
MISTRAL	FR	3.75 *cd*	3.00 *bcde*	EFFEKT	B	52.28 *abc*	81.86 *cdefg*
AGAT	B	3.70 *cde*	3.42 *bc*	PI343986	P	57.73 *abcd*	77.32 *cde*
PI244158	P	3.65 *cdef*	3.00 *bcde*	PI269802	P	58.11 *abcde*	72.94 *bcd*
PI257593	P	3.52 *defg*	2.80 *bcde*	560	ETG	58.48 *abcde*	90.14 *defg*
PI413696	**P**	**3.50 *defgh***	**2.75 *cde***	**CAPELLA**	**Sva**	**60.44 *abcde***	**73.19 *bcd***
CAPELLA	**Sva**	**3.47 *defgh***	**3.32 *bcd***	PI244158	P	60.97 *abcdef*	86.81 *defg*
90-2079	JMK	3.47 *defgh*	3.15 *bcd*	90-2079	JMK	62.51 *abcdefg*	72.10 *bcd*
PI272212	P	3.45 *defgh*	3.05 *bcde*	PI169600	P	67.14 *bcdefgh*	73.81 *bcde*
MN314	**DWD**	**3.45 *defgh***	**2.55 *def***	PI257593	P	67.68 *bcdefgh*	89.34 *defg*
PI269802	P	3.42 *defgh*	2.75 *cde*	**PI413696**	**P**	**70.95 *bcdefghi***	**80.81 *cdefg***
PI210641	**P**	**3.40 *defgh***	**3.20 *bcd***	**PI393487**	**P**	**71.18 *bcdefghi***	**103.08 *fg***
MN313	**DWD**	**3.40 *defgh***	**2.68 *cdef***	**MN314**	**DWD**	**71.86 *bcdefghi***	**74.48 *bcde***
PI169600	P	3.37 *defgh*	3.07 *bcde*	**90-2131**	**JMK**	**72.01 *bcdefghi***	**78.48 *cdef***
PI306592	P	3.35 *defgh*	3.00 *bcde*	AGAT	B	72.90 *bcdefghi*	72.70 *bcd*
WI9416	ETG	3.32 *defgh*	3.05 *bcde*	MISTRAL	FR	74.49 *cdefghi*	102.96 *fg*
WI8904	ETG	3.30 *defgh*	3.25 *bcd*	**PI244162**	**P**	**74.69 *cdefghi***	**89.26 *defg***
90-2322	JMK	3.30 *defgh*	2.85 *bcde*	PI413686	P	75.15 *cdefghi*	98.81 *efg*
PI393487	**P**	**3.25 *efghi***	**2.10 *fg***	200	ETG	75.76 *cdefghi*	72.66 *bcd*
PI210642	**P**	**3.22 *fghi***	**3.05 *bcde***	**552**	**ETG**	**75.78 *cdefghi***	**92.09 *defg***
PI413686	P	3.20 *ghi*	2.60 *def*	PI471166	P	76.83 *defghi*	92.48 *defg*
90-2131	**JMK**	**3.15 *ghi***	**3.00 *bcde***	PI343984	P	77.38 *defghi*	77.14 *cde*
200	ETG	3.10 *ghi*	3.05 *bcde*	PI306592	P	78.27 *defghi*	84.53 *cdefg*
PI471166	P	3.10 *ghi*	2.90 *bcde*	**PI210642**	**P**	**80.00 *defghi***	**90.02 *defg***
MELROSE	**P**	**3.10 *ghi***	**2.67 *cdef***	WI8904	ETG	80.40 *defghi*	79.57 *cdef*
560	ETG	3.05 *ghi*	3.05 *bcde*	**PI210641**	**P**	**80.77 *efghi***	**94.15 *defg***
PI343986	P	3.00 *ghi*	2.95 *bcde*	90-2322	JMK	82.24 *fghi*	78.85 *cdef*
552	**ETG**	**2.95 *hi***	2.62 *cdef*	WI9416	ETG	84.99 *ghi*	83.64 *cdefg*
PI244162	**P**	**2.95 *hi***	**2.37 *efg***	**MN313**	**DWD**	**85.26 *hi***	**97.94 *efg***
PI343984	P	2.75 *i*	3.00 *bcde*	**PI180693**	**P**	**88.31 *i***	**105.07 *g***
PI180693	**P**	**2.75 *i***	**2.00 *g***	**MELROSE**	**P**	**93.23 *i***	**99.80 *efg***
Mean[5]		**3.27 *a***	**2.96 *b***	**Mean**[5]		**71.6 *b***	**82.78 *a***

[1]Disease severity was assessed according to a 0–5 scale – See the text for details.
[2]Per cent of top fresh weight was calculated as the ratio: (aerial fresh weight of the inoculated line/aerial fresh weight of the uninoculated control) × 100.
[3]Country of origin, or germplasm collection, FR: variety registered in France, B: lines from Belarus, Sva: variety from Svalöf Weibull, ETG: from E.T. Gritton, JMK: from J.M. Kraft, DWD: from D.W. Davis, P: Pullman germplasm bank. The genotypes typed in bold were selected for the constitution of the preset.
[4]Means followed by the same letter in a column are not significantly different ($p = 0.05$) according to Newman–Keuls test.
[5]Means followed by the same letter in a row are not significantly different ($p = 0.05$) according to Newman–Keuls test.

Resistance behaviour considering the disease severity

The DS values ranged from 4.53 to 2.75 with Ae5, and from 4.4 to 2.0 with Ae44. With Ae5, the most susceptible lines were the European varieties (Baccara, Solara and Effekt); the breeding lines were intermediate; three lines from ET Gritton (200, 560 and 552) and one from JM Kraft (902131), as well as some Pullman lines displayed the lowest DS. With Ae44 also, the European varieties (particularly Baccara, Solara and Agat) were the most affected. Most of the lines, including the Kraft material, displayed intermediate resistance; the most resistant lines were PI180693, PI393487, PI244162, as well as 552 and MN314.

No line was highly resistant to one isolate and highly susceptible to the other. However, some quantitative interactions were observed between the pea genotypes and the two isolates. Most of the genotypes were equally diseased with both isolates, at all levels of diseasement. As an example, Baccara was susceptible to Ae5 et Ae44, PI210641 and Capella were moderately susceptible; PI210642, 552, PI244162 and Melrose were moderately resistant (Table 3).

Some genotypes were less diseased with Ae44 than with Ae5. Solara was susceptible to Ae5 but moderately susceptible to Ae44; MN313, MN314 and PI413696 were moderately susceptible to Ae5 and moderately resistant to Ae44. PI393487 was much less diseased with Ae44 than with Ae5 (Table 3). For PI343984, the disease level was higher with Ae44 than with Ae5. However, this difference was not statistically significant.

Resistance/tolerance behaviour considering the per cent of top fresh weight

The PFW values were ranked from 42.01% to 93.23% with Ae5, 49.29% to 105.07% with Ae44. With Ae5, the most susceptible lines were Baccara, Solara and PI272212. Among the best lines, were Melrose, PI180693, MN313, WI9416 and 902322. With Ae44, Baccara, Solara and PI272212 were also the most susceptible. The most resistant lines were PI180693, PI393487, Mistral and Melrose. So Melrose and PI180693 were the most resistant lines to both isolates. Mistral and PI393487, as well as 560, were resistant to Ae44 but intermediate with Ae5. No line was completely resistant to one isolate and susceptible to the other.

The two resistance criteria were significantly correlated, for Ae5 ($r = -0.589$, $p < 0.01$) as well as for Ae44 ($r = -0.697$, $p < 0.01$). However some lines, although attacked by the pathogen and having an intermediate DS, could stand the disease better than others, keeping a top biomass close to that of the uninoculated control. For example, MN314, showing with Ae5 the same DS as PI272212, had a significantly higher PFW than this line.

Constitution of the pre-set

To constitute the pre-set, we chose the most resistant lines, taking care to keep the genetic background variability, as well as those displaying quantitative interactions with the isolates. Baccara was kept as a susceptible control. The best line from the independent breeding programmes was selected: 902131 from J.M. Kraft, 552 from E.T. Gritton, MN313, MN314 from D.W. Davis (which were moderately resistant to Ae44 and moderately susceptible to Ae5) and Capella from Svalof (although its behaviour was intermediate). PI180693 was the best line according to DS and PFW with both isolates, and Melrose was among the best lines for DS and the second best for PFW. PI244162 was among the best lines for DS to both isolates. PI210641 and PI210642, despite moderately resistant regarding DS, were among the best lines for PFW. For both criteria, PI413696 and PI393487 displayed quantitative interactions: they were moderately susceptible with Ae5, but among the best lines with Ae44. These 13 lines constituted the pre-set.

Reaction of the pre-set of P. sativum *lines to French and foreign* A. euteiches *isolates*

As the two separate experiments gave similar DS data for Ae5, results from both experiments were pooled. Genotype, isolate and genotype × isolate interaction effects on the DS were highly significant ($p < 0.01$). The contribution to the global variance of the interaction (represented by the mean square) was much lower than the contributions of the genotype and isolate effects (Table 4). Thus, differences between isolates and between genotypes were predominant.

Globally, Baccara was the most susceptible cultivar, followed by Capella; 552, Melrose and PI180693 were the most resistant cultivars (Table 5). The application of the threshold DS = 1 distinguished six distinct patterns among the 13 genotypes of the pre-set. Besides, some cultivar : isolate combinations displayed statistically significant quantitative interactions (Table 5) within these patterns. MN313 and MN314 were highly resistant to Ae173 and Ae139, but susceptible to Ae163

Table 4. Analysis of variance for disease index data on 13 pea genotypes, 7 days after inoculation with isolates of *Aphanomyces euteiches* from different geographical origins

Source of variation	Degrees of freedom	Mean square (MS)
Genotype	12	9.52*
Isolate	13	55.82*
Isolate × genotype	156	0.80*
Error	788	0.08

*$p < 0.01$.

Table 5. Mean disease severity and pathogenicity pattern incited by isolates of *Aphanomyces euteiches* on the preset of 13 pea cultivars

Genotype	*A. euteiches* isolates														Mean
	Ae5[1] (FRA)	Ae169 (FRA)	Ae33 (FRA)	Ae84 (FRA)	Ae40 (FRA)	Ae83 (FRA)	Ae147 (SUE)	Ae172 (USA)	Ae6 (FRA)	Ae78 (FRA)	Ae173 (USA)	Ae139 (NZe)	Ae163 (CAN)	Ae164 (CAN)	
BACCARA[2]	***4.48 a***[3]+[4]	***4.33 b***+	***4.68 a*** +	***3.55 ab***+	***3.40 a***+	***3.25 a***+	***3.38 a***+	***3.00 a***+	***2.65 a***+	***3.20 a***+	***2.90 a***+	***2.30 a***+	***1.95 a***+	***1.30 a***+	***3.17 a***
CAPELLA	***3.26 b***+	***4.88 a***+	***3.78 b***+	***3.73 a***+	***3.27 ab***+	***3.20 a***+	***3.05 bc***+	***2.25 abcd***+	***1.93 bc***+	***2.55 abc***+	***2.60 ab***+	***2.45 a***+	***1.42 bcd***+	***0.93 b***−	***2.81 b***
PI210641	3.31 *b*+	3.50 *cd*+	3.00 *e*+	3.00 *c*+	2.75 *b*+	2.85 *ab*+	3.05 *bc*+	2.55 *abc*+	1.20 *de*+	1.55 *d*+	1.85 *cd*+	2.15 *ab*+	1.30 *cd*+	0.40 *def*−	2.32 *ef*
PI210642	3.03 *bc*+	3.40 *cdef*+	3.42 *cd*+	3.30 *bc*+	3.20 *ab*+	2.90 *ab*+	3.00 *bc*+	2.75 *ab*+	2.30 *ab*+	1.78 *cd*+	1.10 *e*+	2.71 *a*+	1.05 *d*+	0.10 *f* −	2.43 *cd*
PI413696	3.20 *bc*+	3.45 *cde*+	3.25 *cde*+	3.00 *c*+	2.95 *ab*+	3.00 *ab*+	2.90 *bc*+	1.80 *cde*+	2.05 *bc*+	2.65 *ab*+	2.30 *bc*+	2.25 *a*+	1.25 *cd*+	0.60 *bcde*−	2.48 *c*
90-2131	***3.13 bc***+	***3.35 cdef***+	***3.10 de***+	***2.95 c***+	***3.05 ab***+	***2.00 d***+	***3.05 bc***+	***1.80 cde***+	***1.60 cd***+	***1.50 d***+	***2.00 cd***+	***2.10 ab***+	***0.95 d***−	***0.35 ef***−	***2.21 fg***
PI244162	3.00 *bc*+	3.38 *cdef*+	3.30 *cde*+	3.10 *c*+	2.80 *b*+	2.90 *ab*+	2.90 *c*+	2.45 *abcd*+	2.15 *abc*+	1.90 *bcd*+	2.05 *cd*+	2.35 *a*+	0.95 *d* −	0.78 *bcd*−	2.43 *cd*
PI393487	3.00 *bc*+	3.05 *ef*+	3.00 *e*+	3.00 *c*+	3.00 *ab*+	3.00 *ab*+	2.95 *bc*+	1.75 *cde*+	1.60 *cd*+	2.20 *bcd*+	2.55 *ab*+	2.05 *ab*+	0.95 *d* −	0.85 *bc*−	2.35 *de*
MELROSE	2.88 *c*+	3.17 *def*+	3.20 *cde*+	3.05 *c*+	3.05 *ab*+	2.85 *ab*+	2.90 *c*+	1.70 *cde*+	1.25 *de*+	1.70 *d*+	1.55 *d*+	1.32 *cd*+	0.35 *e*−	0.10 *f* −	2.08 *h*
MN314	3.20 *bc*+	3.70 *c*+	3.53 *bc*+	2.90 *c*+	3.00 *ab*+	3.05 *ab*+	3.27 *ab*+	2.05 *bcde*+	1.55 *cd*+	1.60 *d*+	0.10 *f* −	0.55 *e*−	1.45 *bcd*+	0.25 *ef*−	2.16 *gh*
MN313	***3.23 bc***+	***3.40 cdef***+	***3.05 de***+	***2.80 c***+	***2.72 b***+	***3.32 a***+	***3.00 bc***+	***2.05 bcde***+	***1.05 de***+	***1.90 bcd***+	***0.10 f***−	***0.95 de***−	***1.78 ab***+	***0.50 cdef***−	***2.13 gh***
552	***2.90 c***+	***3.15 def***+	***3.05 de***+	***2.80 c***+	***2.70 b***+	***2.55 bc***+	***3.00 bc***+	***1.60 de***+	***1.10 de***+	***1.45 d***+	***1.60 d***+	***0.81 de***−	***1.65 abc***+	***0.80 bcd***−	***2.80 h***
PI180693	***3.00 bc***+	***3.00 f***+	***2.30 f***+	***1.92 d***+	***1.61 c***+	***2.30 cd***+	***2.95 bc***+	***1.25 e***+	***0.73 e***−	***0.60 e***−	***2.00 cd***+	***1.60 bc***+	***0.35 e***−	***0.15 f***−	***1.70 i***

[1]Mean of two experiments.
[2]The genotypes marked in bold were selected to compose the final set of differentials.
[3]Means followed by the same letter in a column are not significantly different ($P = 0.05$) according to the Newman–Keuls test.
[4]The isolates are defined as virulent (+) or avirulent (−) according to the threshold DS = 1 – See the text for details.

and Ae83. 902131 was moderately resistant to Ae83 and Ae78, but intermediate with all the other isolates. PI210642 was moderately resistant to Ae173 but susceptible to Ae139 and Ae6. PI180693 was significantly the most resistant line with Ae33, Ae84, Ae40, Ae78, Ae163, Ae164. Capella was significantly less diseased than Baccara with Ae5, Ae33, Ae6, Ae147, Ae164, equally susceptible with Ae84, Ae40, Ae83, Ae172, Ae78, Ae173, Ae139, and more diseased than Baccara with Ae169. 552 was resistant to Ae139, Ae6, Ae172, but was moderately susceptible with Ae40 and Ae147.

MN313 and MN314 incited the most important ΔDS (2.80) when inoculated with Ae173. Then followed PI180693 with Ae78 (ΔDS = 2.60) and Ae33 (ΔDS = 2.38), then PI210642 with Ae173 (ΔDS = 1.80), 552 with Ae78 (ΔDS = 1.75), 902131 with Ae78 (ΔDS = 1.70), Melrose with Ae163 and Ae5 (ΔDS = 1.60).

The final pea set was constituted from the genotypes displaying the most significant quantitative interactions, the most important ΔDS (i.e. the most important gain in quantitative resistance regardless of the isolate aggressiveness), within each of the patterns inferred from the pathogenicity threshold. Baccara was chosen as the susceptible control, and MN313, PI180693, 552, 902131 and Capella were the differential lines.

Reproductibility among experiments, on the reference Ae5

DS values incited by Ae5 on the six pea lines of the final set were not significantly different over experiments for all the lines except the susceptible control Baccara (Table 6).

Comparison between the seedling and field reaction of the set of six pea genotypes

The six genotypes displayed similar resistance rankings in controlled conditions as in the field (Table 7): Baccara and Capella were the most susceptible lines, whereas PI180693 and 552 were the most resistant ones. The lines displayed less RDS variability (from 2.56 to 3.63) than in growth chamber (2.75–4.15). Regarding the AI at both dates (AI1 and AI2), Baccara and Capella also appeared to be the most susceptible lines, and 552 the most resistant/tolerant one. PI180693 and 902131 had a resistance/tolerance behaviour similar to 552 at the first date but were much more attacked at the second date. MN313, which displayed an intermediate resistance behaviour at the first scoring date, was much more attacked at the second date.

DS in growth chamber and RDS in the field chamber were correlated (Figure 1), although not significantly ($P = 0.137$ and 0.06 in 1999 and 2000, respectively). Conversely, DS and aerial indices were strongly and significantly correlated, for AI1 ($P = 0.028$ and 0.008 in 1999 and 2000, respectively) and AI2 ($P = 0.02$ and 0.038 in 1999 and 2000, respectively).

Discussion

The new set of pea differentials proposed in this study, is composed of five partially resistant lines (Capella, MN313, 902131, 552, PI180693) and one susceptible variety, Baccara. Its originality is based on the germplasm used. From the previous sets (presented in Table 8), only MN313 (Malvick and Percich, 1998a) and PI180693 (Beute and Lockwood, 1967) were

Table 6. Disease severity values incited by Ae5 on the set of pea genotypes, over nine experiments

Experiments	Pea genotypes					
	BACCARA[1]	CAPELLA	MN313	902131	PI180693	552
CV2	4.20 ***bc***	3.47 ***a***	3.40 ***a***	3.15 ***a***	2.75 ***ab***	2.95 ***ab***
CV3	4.15 ***bc***	3.30 ***a***	3.20 ***a***	3.00 ***a***	2.90 ***ab***	2.75 ***ab***
CV4	4.80 ***a***	3.22 ***ab***	3.25 ***a***	3.25 ***a***	3.10 ***a***	3.05 ***a***
V1	3.40 ***d***	3.37 ***a***	3.10 ***a***	3.10 ***a***	3.00 ***ab***	2.95 ***ab***
V2	4.30 ***b***	3.10 ***ab***	3.25 ***a***	3.07 ***a***	2.50 ***b***	2.95 ***ab***
V3	3.40 ***d***	2.80 ***b***	2.93 ***a***	2.87 ***a***	2.95 ***ab***	2.62 ***ab***
V4	4.20 ***bc***	3.05 ***ab***	3.00 ***a***	3.07 ***a***	2.95 ***ab***	2.35 ***b***
V5	3.65 ***cd***	3.10 ***ab***	3.20 ***a***	3.20 ***a***	3.00 ***ab***	2.90 ***ab***
V6	3.40 ***d***	3.15 ***ab***	3.15 ***a***	2.97 ***a***	2.50 ***b***	2.60 ***ab***
Mean[2]	**3.94 *a***	**3.17 *b***	**3.16 *b***	**3.07 *b***	**2.83 *c***	**2.79 *c***

[1]Mean values followed by the same letter in a column are not significantly different (Newman–Keuls test, $p = 0.01$).
[2]Mean values followed by the same letter in a row are not significantly different (Newman–Keuls test, $p = 0.01$).

Table 7. Comparison of the behaviour of the pea-set in growth chamber (inoculated with Ae5) and in field trial in 1999 and 2000

Genotypes	Growth chamber (Ae5)	Field results 1999 (Courtacon)			Field results 2000 (Courtacon)		
	DS[1]	RDS[2]	AI1[3]	AI2[4]	RDS[2]	AI1[3]	AI2[4]
BACCARA	3.94 ***a***	3.88 ***a***	4.50 ***a***	5.50 ***a***	3.63 ***a***	6.33 ***a***	7.33 ***a***
CAPELLA	3.17 ***b***	4.08 ***a***	3.50 ***ab***	4.50 ***a***	3.65 ***a***	5.33 ***ab***	6.00 ***a***
MN313	3.16 ***b***	3.46 ***ab***	3.00 ***ab***	4.00 ***a***	3.14 ***ab***	4.66 ***bc***	6.67 ***a***
902131	3.07 ***b***	2.53 ***ab***	2.00 ***bc***	2.67 ***a***	3.25 ***ab***	4.00 ***c***	4.67 ***b***
PI180693	2.82 ***c***	2.78 ***b***	1.00 ***c***	3.00 ***a***	2.84 ***b***	3.00 ***c***	4.33 ***b***
522	2.79 ***c***	1.96 ***ab***	2.50 ***bc***	2.50 ***a***	2.56 ***b***	3.00 ***c***	3.00 ***c***

[1]DS = Mean disease severity, assessed according to a 0–5 scale – See the text for details.
[2]RDS = Mean root disease severity, assessed in the field on 11 May 1999 and 2000, according to a 0–5 scale – See the text for details.
[3]AI1 = Mean aerial index assessed on 27 May 1999 and 6 June 2000, according to a 1–9 scale.
[4]AI2 = AI assessed on 9 June 1999 and 15 June 2000, according to a 1–9 scale.

found to be differentials. During preliminary experiments under controlled conditions, lines from these sets were found susceptible to French isolates: PI175232 from the set of Beute and Lockwood (1967); 86-2236, Mn108, WI8902 and WI8904 from the set of Duerst (1996); 90-2079 and WI8904 from the set of Malvick and Percich (1998a).

The second originality of this new set relies on the set up process. In previous sets, differential pea lines were only chosen according to their different resistance levels (Lockwood and Ballard, 1960; Beute and Lockwood, 1967; Duerst, 1996; Malvick and Percich, 1998a). Our strategy evaluated general resistance levels of the pea genotypes, but also assessed their host–pathogen interaction patterns. Primarily, most of the resistant sources characterised elsewhere were screened, to retain germplasm of good resistance level and of different genetic background. The results showed that the available resistance sources displayed an incomplete level of resistance to the French *A. euteiches* isolates, but were more resistant than the tested European varieties. Furthermore, the presence of genotype × isolate interactions was investigated within a restricted group of cultivars and isolates, with the aim of including each of the interaction patterns observed in the final differential set.

The resistance criterium was the second important thing to choose in the development of the set, since this criterium has to give a faithful interpretation of the host–pathogen interaction. Resistance of pea to *A. euteiches* was previously assessed by mortality occurence (Beute and Lockwood, 1967; Sundheim, 1972), a criterium which confuses resistance and tolerance and is highly sensitive to environmental variation. Percentage of plant fresh weight has been also used as resistance criterium. Kraft et al. (1994) considered resistant any line having a fresh biomass of at least 70% of its uninoculated control. This criterium appeared poorly discriminating in our screening of pea germplasm. Moreover, this measure, expressing an overall response of the plant to the infection, may involve both resistance and tolerance components (*sensu* Rapilly (1991)).

Global resistance observed in these genotypes was actually a mix of true resistance (defined by Parlevliet and Zadoks (1977) as 'resistance acting to hinder or prevent the establishment on or in and the colonization of the host by the pathogen') and escape resistance ('preventing or hindering contact of the host with the pathogen'), which can involve factors independent of the defence mechanisms of the plant (larger root systems (Kraft and Boge, 1996) for example). As a consequence, many of the lines released by breeders are actually tolerant, and do not have a good level of intrinsic resistance. That is why, in the step relating to host–pathogen interactions, we chose to limit our resistance evaluation to DS, a criterium directly linked to resistance to the pathogen (true resistance *sensu* Parlevliet and Zadoks (1977)) that Malvick and Percich (1998, 1999) already used.

For the design of an international differential set, care must be taken on the choice of the differential genotypes, of the differential isolates, and on the choice of methodology for assessing the genotype–isolate interaction. This study took into account as far as possible the results of the previous studies for the choice of the differential genotypes, and chose isolates from Europe and outside Europe. Most of the previous

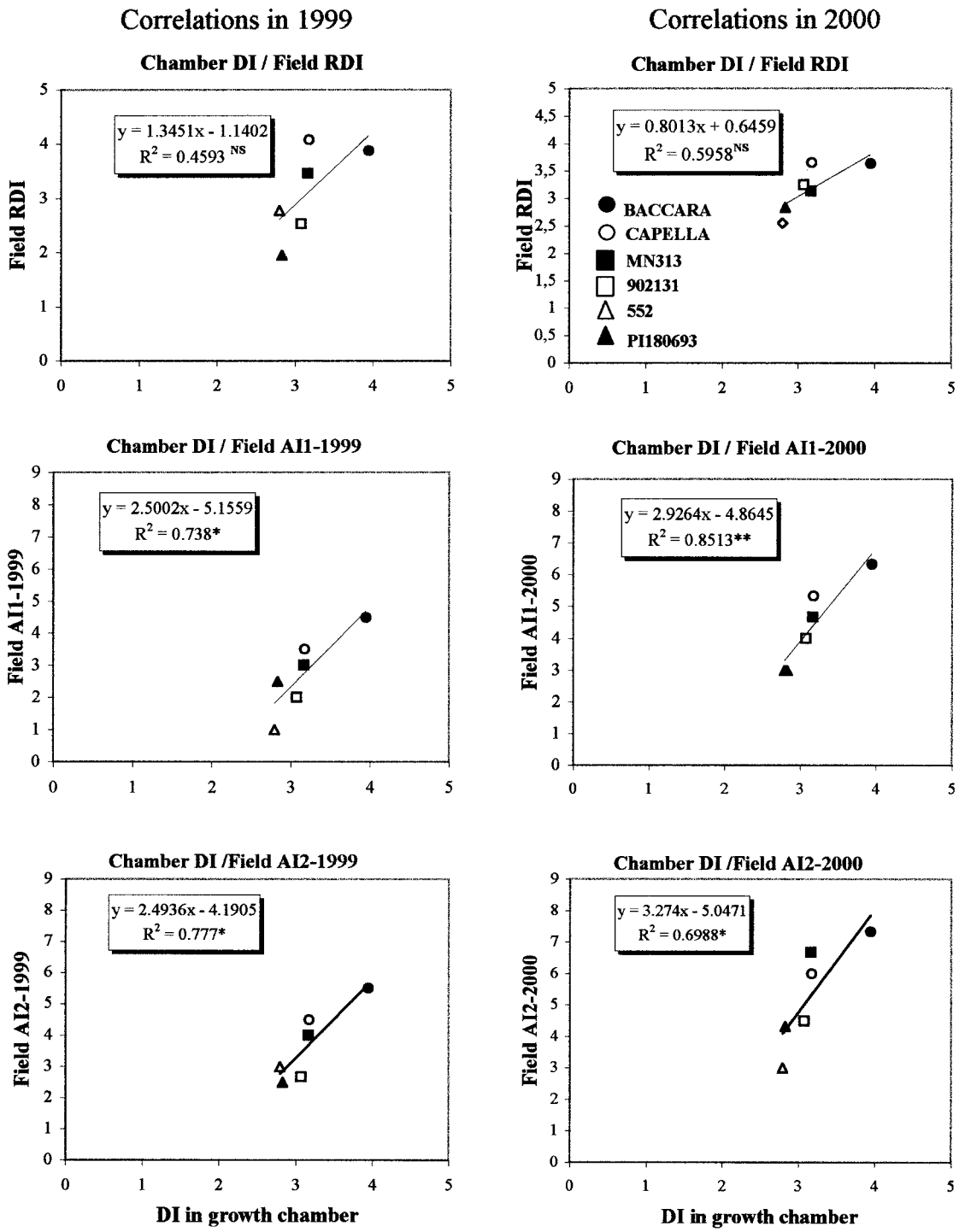

Figure 1. Correlations between the results of the set of pea genotypes in controlled conditions (disease severity (DS) incited by Ae5) and in field trials (Courtacon) in 1999 and 2000, in term of root disease index (RDI) and aerial indices assessed at two dates (AI1, AI2). DI: Mean disease index evaluated in growth chamber, 7 days after inoculation with Ae5 (0–5 scale). RDI: Root disease index assessed in the field (0–5 scale). AI1: Aerial index assessed in the field, at the 27 May 1999 and the 6th of June 2000 (0–9 scale). AI2: Aerial index assessed in the field, at the 9 June 1999 and the 15th of June 2000 (0–9 scale).

Table 8. Differential pea sets developed in previous studies on resistance to *Aphanomyces euteiches*

References	Pea genotypes (from susceptible to resistant)
Beute and Lockwood (1967), Sundheim (1972), Manning and Menzies (1984)	Miragreen, Early Perfection, PI175232, PI169604, PI180693, PI166159
Duerst (1996)	EP77, CSC8221, PI175232, WI8902, WI8904, MN108, 86-2231
Malvick and Percich (1998)	Little Marvel, WI8904, 90-2079, MN313 and MN314

differential isolates (Sundheim, 1972; Malvick and Percich, 1999) were unfortunately unavailable at this time (Wicker and Rouxel, 2001).

In this study, the definition of resistance patterns was allowed by both quantitative and qualitative analysis of the disease data: observation of statistically significant quantitative interactions, and characterisation of susceptible and resistant interactions by the use of a pathogenicity threshold, fixed at DS = 1. Fixed thresholds of DS = 2.5 and even 3 (on a 0–5 disease scale) were already used on this pathosystem to distinguish virulence groups among pea-infecting isolates (Malvick and Percich, 1998a,b). Our threshold was considered as the most biologically relevant one since up from this note, the pathogen could be reisolated from the roots (i.e. was saprophytically active) and did form oospores in the roots (i.e. could fulfill its sexual reproduction). A similar methodology was used on the interaction *Brassica napus*/*Plasmodiophora brassicae*: the threshold 25, chosen on 0–100 scale, corresponds to the formation of small galls on the lateral roots, that cannot lead to the formation of clubs on the taproot (Some et al., 1996; Maria Manzanares-Dauleux, personal communication, 2001). Our qualitative approach allowed to distinguish resistance factors expressing at the pre-infection and infection stages of the interaction, whereas the quantitative approach took into account the resistance phenomenon occurring after the penetration (resistance to the pathogen colonisation).

Disease severity was assessed at a given date, and with a given inoculum dose and form. The *A. euteiches*/pea interaction involves quantitative components (Shehata et al., 1983), which may express at different stages of the infection process. Thus, it could be more informative to follow the progression of the DS over time by several disease assessment, leading to criteria such as the area under disease progression curve. Another way of accessing the different resistance mechanisms expressing over time would be to distinguish the main stages of the infectious process (pre-infection, infection, colonisation of the roots tissues) and thus assess whether any resistance factor expresses during one of these stages. This approach will need further methodological work, but may be very fruitful in future research.

The set we propose comprises six genotypes: Baccara, Capella, MN313, 903131, 552 and PI180693, corresponding to five 'resistance patterns'. Whether each of these patterns corresponds to a distinct resistance factor, or a combination of factors, is not known. Moreover each factors give no indication on the number of genes involved in resistance (Person, 1959). Flor (1956) showed that the precise knowledge of the genetic basis of host–parasite systems requires knowledge of the genetics of both the host and the parasite. Resistance to *A. euteiches* is quantitative and polygenically inherited (Shehata et al., 1983), but the number of genes involved is unknown. Recent studies indicated that resistance of line 902079 could be explained by a major Quantitative Trait Locus (QTL) (Pilet-Nayel et al., 2001). One genetic factor linked to the field partial resistance of MN313, was expressed differentially among different nurseries, and was hypothesized to be isolate-specific (Weeden et al., 2000).

In this study, the seedling reaction of the differential genotypes in controlled conditions was correlated with their behaviour in the field. The weak correlation of the DS in growth chamber with the RDI scored in the field could be related to the effect of other root pathogens, as *Fusarium solani*, *F. oxysporum* and *Phoma medicaginis* var. *pinodella*. However, the good correlation of the chamber results with the aerial symptoms in the field indicates that resistance assessed in growth chamber is a good indicator of the resistance/tolerance expressed in natural conditions. Whether this correlation is due to a delay in the infection of the resistant lines or to a slowed pathogen progression within the plant tissues, as Kraft tended to indicate in the case of PI180693 (Kraft and Boge, 1996), remains to be determined.

In future research, the specificity of the resistance of pea to *A. euteiches* will have to be further characterised. Parlevliet and Zadoks (1977) showed that quantitatively expressed resistance may be composed of genes of minor effects acting in a gene-for-gene manner with minor avirulence genes of the pathogen. The genetic basis of pea–*A. euteiches* interactions should be investigated, by the characterization of the different components of pea resistance, the monitoring of their

kinetics of expression, and of genes or QTLs involved in each of these. This new set will be a very useful tool for that purpose, and any new developments would help to improve the set. It will be necessary to work with single-spore isolates of *A. euteiches* to assess whether the resistance patterns correspond to gene-for-gene interactions.

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