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Influence of competition and host plant resistance on selection in *Phytophthora infestans* populations in Michigan, USA and in Northern Ireland

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Competition between genotypes of *Phytophthora infestans* was studied by inoculating potato cultivars with differing susceptibility to late blight in field experiments over three years in Northern Ireland, UK, and Michigan, USA. Multiple isolates of six genotype groups of *P. infestans* were chosen from the local populations in both N. Ireland and Michigan for inoculation of separate field trials planted in 2003, 2004 and 2005. Four cultivars were used in each trial; two (susceptible cv. Atlantic and the partially resistant cv. Stirling) were common to both locations, whereas the two additional cultivars (with partial resistance to late blight) were cvs Santé and Milagro in N. Ireland and cvs Pike and Jacqueline Lee in Michigan. Single-lesion isolates of *P. infestans* were obtained from leaves at 1% level of infection, characterized using pre-assigned markers and re-assigned to their respective genotype groups. Extreme selection occurred within the population of genotypes of *P. infestans* in N. Ireland in each year, with different genotype groups dominating the infection of different cultivars. Selection was observed on all cultivars tested, but was greatest on the more resistant cultivars. Over the 3 years, all of the 114 isolates obtained from cv. Milagro belonged to a single group, whereas among the 118 isolates from cv. Atlantic all six groups were represented. By contrast, in Michigan, the US-8 genotype dominated infection in all cultivars in each year; only 12 of 374 isolates characterized belonged to other genotypes (11 US-14 and a single US-10 isolate).

Keywords: aggressiveness, cultivar resistance, potato, potato late blight, virulence

Introduction

Late blight, caused by the oomycete *Phytophthora infestans*, remains the most destructive disease to affect the potato worldwide, and causes significant damage to the industry in terms of crop loss and chemical control. In both Northern Ireland, UK and the state of Michigan, USA, conditions are typically ideal for its spread, with periods of high humidity, frequent rainfall and moderate temperatures (Goodale *et al*., 1998; Baker *et al*., 2005). Nonetheless, most commercially grown cultivars in both regions are highly susceptible to foliar late blight (Inglis *et al*., 1996; R. Martin, personal communication). The increased number and severity of epidemics over recent decades, which have been associated with new migrations of the pathogen (Goodwin *et al*., 1998), demonstrate the increased need for commercially acceptable cultivars that possess greater levels of late-blight resistance.

Since the late 1970s, a number of migration events have caused major population changes globally, during which the previously pan-global US-1 clonal lineage has been displaced and genotypically distinct populations have emerged in Europe and the United States (Spielman *et al*., 1991; Goodwin *et al*., 1998). It has been suggested that these new genotypes are competitively fitter owing to increased aggressiveness, both on the foliage and in the tuber (Spielman *et al*., 1991), and that the increased variation that has been observed in the new populations could lead to greater selection for strains that are able to overcome cultivar resistance to potato late blight (Flier *et al*., 2003).

Selection within populations can occur rapidly, particularly if there is a considerable difference in fitness (defined as the ability of a phenotype to contribute to the next generation; Antonovics & Alexander, 1989) between or within genotypes. Fitness is therefore an important

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predictive measurement of the potential for selection within populations. However, owing to the difficulty of determining long-term fitness, single-generation measurements that are associated with aggressiveness, such as infection frequency, lesion size and sporulation, are commonly used to predict fitness in the field (Antonovics & Alexander, 1989; Day & Shattock, 1997), although these parameters do not assess or take into account any possible direct competitive interactions.

Competition has been defined by Newton *et al.* (1997) as the effect of one propagative unit of one pathogen strain on the reproductive output of another strain, relative to the effect of a unit of that other strain on its own reproductive output. The potential impact of competition will therefore depend on the fitness of individuals both in the presence and absence of potential competitors.

Much of the supporting evidence for selection within late-blight populations has come from detached leaflet experiments, whole-plant inoculations and tuber studies, because of a lack of appropriate markers for tracking populations, with few competitive tests carried out in the field. In Israel, Kadish & Cohen (1988) showed that metalaxyl-resistant isolates competed successfully against sensitive isolates on whole plants even if their initial frequency was as low as 1% in mixed inoculum, and Cohen & Samoucha (1990) studied competition between *P. infestans* isolates using mixed inoculum in polytunnels and reported that, even in the absence of the fungicide, oxadixyl-resistant isolates were competitively superior to sensitive ones. Miller & Johnson (2000) used allozyme genotyping to assess the comparative fitness of the US-1 genotype and the US-8 genotype on cv. Russet Burbank in transplanted greenhouse-grown plants; most of the isolates that were recovered were of the US-8 lineage, which indicated that this genotype had a fitness advantage. This study, however, did not investigate the effect of cultivar resistance on competition and selection.

Quantitative or 'horizontal' resistance, also known as race non-specific or field resistance, confers a general reduction in disease and is exhibited towards all races that are capable of causing more than a hypersensitive reaction on the host (Niederhauser, 1961). Field resistance to late blight has been the subject of numerous investigations, with most suggesting it is highly durable (e.g. Forbes *et al.*, 2005). However, Flier *et al.* (2003), using a combination of detached leaflets, whole tubers and field experiments, detected differential isolate–cultivar interactions for both foliar and tuber blight resistance and concluded that some adaptation to partial resistance exists in populations of *P. infestans* from the Netherlands. Several studies have investigated the isolate–cultivar interaction by sampling and comparing the aggressiveness of isolates from the naturally infected foliage of cultivars that have differing levels of partial resistance. Lozoya-Saldaña *et al.* (2006) characterized isolates from five potato cultivars grown in the Toluca Valley, Mexico; limited host–pathogen specificity was observed for only two cultivars and this was not related to host resistance. Similarly, Montarry *et al.* (2006) sampled cultivars that exhibited differing levels of late blight resistance from field trials in France over a period of 2 years. This study supported the hypothesis of gradual adaptation to partial resistance, but genotypic diversity was low and no correlation was detected between pathogenicity traits and the selection of genotypes. Andrivon *et al.* (2007) compared *P. infestans* isolates that were obtained from potato crops in France and Morocco on detached leaves of the potato cvs Bintje (commonly grown in France) and Désirée (more commonly grown in Morocco than France), and detected a higher level of aggressiveness in French isolates to Bintje and in Moroccan isolates to Désirée; it was proposed that differences were due to adaptation to the locally grown cultivar.

The aim of the present study was to determine the influence of the potato cultivar on selection in *P. infestans* populations in a competitive field environment, using cultivars that have differing levels of resistance to potato late blight. Trials were conducted at Michigan State University, USA, and Belfast, N. Ireland, using local *P. infestans* genotypes from those areas. Both populations comprise genotypes that are easily identifiable by commonly used phenotypic and genotypic tools. The population of *P. infestans* in Michigan is composed of a number of distinctive genotypes, but is dominated by the highly aggressive US-8 lineage (Goodwin *et al.*, 1998; W.W. Kirk, unpublished observations). By contrast, the population in N. Ireland consists of a limited number of clones that are related to, but differentiated from, the populations that are found in mainland Britain and elsewhere in Europe (Cooke *et al.*, 2006) and are distinct from the current USA populations. The distinctive and variable nature of the two populations makes them valuable case studies for the present investigation.

Materials and methods

Isolate selection, characterization and maintenance

Isolates were obtained from collections maintained at the Agri-Food & Biosciences Institute in Belfast, N. Ireland, and Michigan State University, USA. All were originally collected from the naturally infected foliage of commercially grown potatoes between the years 1996 and 2002. All isolates used in the study were derived from single zoospores of these original cultures using the method of Caten & Jinks (1968), as modified by Cooke *et al.* (2006). Cultures were maintained at 18°C in the dark, on either rye A agar (Caten & Jinks, 1968) amended with the antibiotics rifampicin (25 mg L^{-1}) and natamycin (25 mg L^{-1}), or pea agar (Hollomon, 1965). Isolates were passaged through detached leaflets of a susceptible potato (cv. Désirée in N. Ireland and FL1879 in Michigan) 1 month prior to the inoculation of each field trial to maintain aggressiveness.

Six groups, which contained multiple isolates of *P. infestans*, were chosen from each local population in N. Ireland and Michigan to represent the phenotypic and genotypic variation that exists within the two regions

Table 1 Characteristics of parental isolates of *Phytophthora infestans* used to inoculate potato field trials in Northern Ireland, UK in 2003, 2004 and 2005

Isolate	Group number	Metalaxyl sensitivity[a]	*Pep* genotype	mtDNA haplotype	R-gene phenotype	RG57 fingerprint	Genotype[b]
43/02	1	R	*100/100*	IIa	1,3,4,5,7,8,10,11	100 010 001 100 110 100 011 001 1	NI-1
30/01	1	R	*100/100*	IIa	1,3,4,5,7,8,10,11	100 010 001 100 110 100 011 001 1	NI-1
35/02	1	R	*100/100*	IIa	1,2,3,4,7,8,10,11	100 010 001 100 110 100 011 001 1	NI-1
41/02	1	R	*100/100*	IIa	1,3,4,5,7,8,10,11	100 010 001 100 110 100 011 001 1	NI-1
62/00	2	S	*100/100*	IIa	1,3,4,5,7,8,10,11	100 010 001 100 110 100 011 001 1	NI-1
19/00	2	S	*100/100*	IIa	4,5,11	100 010 001 100 110 100 011 001 1	NI-1
11/01	2	S	*100/100*	IIa	1,2,3,4,5,7,8,10,11	100 010 001 100 110 100 011 001 1	NI-1
46/02	2	S	*100/100*	IIa	1,3,4,5,7,8,9,11	100 010 001 100 110 100 011 001 1	NI-1
2/00	3	R	*100/100*	Ia	1,3,4,7,10,11	110 010 000 100 110 010 011 101 1	NI-2
26/00	3	R	*100/100*	Ia	1,4,7,8,10,11	110 010 000 100 110 010 011 101 1	NI-2
21/00	3	R	*100/100*	Ia	1,2,3,4,7,8,10,11	110 010 000 100 110 010 011 101 1	NI-2
Orla 4E	3	R	*100/100*	Ia	1,2,3,4,5,7,8,10,11	110 010 000 100 110 010 011 101 1	NI-2
50/98	4	S	*100/100*	Ia	1,3,4,5,6,7,8,10,11	110 010 000 100 110 000 011 101 1	NI-2b
7/02	4	S	*100/100*	Ia	1,3,4,7,8,11	111 010 100 100 110 100 011 101 1	NI-3
64/02	4	S	*100/100*	Ia	3,4,7	111 010 100 100 110 100 011 101 1	NI-3
47/02	4	S	*100/100*	Ia	1,3,4,5,7,8,11	111 010 100 100 110 100 011 101 1	NI-3
6/01	5	R	*96/100*	Ia	1,4	111 010 100 000 110 010 011 101 1	NI-4
1/99	5	R	*96/100*	Ia	1,4,5,7,10,11	111 010 100 000 110 010 011 101 1	NI-4
35/99	5	R	*96/100*	Ia	R0 only	111 010 100 000 110 010 011 101 1	NI-4
23/01	5	R	*96/100*	Ia	non-pathogenic	111 010 100 000 110 010 011 101 1	NI-4
Milagro BL2/02	6	R	*83/100*	Ia	1,2,3,4,5,6,7,8,9,10,11	101 011 110 100 110 100 111 001 1	NI-5
Remarka BL2/02	6	R	*83/100*	Ia	1,2,3,4,5,6,7,8,9,10,11	101 011 110 100 110 100 111 001 1	NI-5
Santé BL2/02	6	R	*83/100*	Ia	1,2,3,4,5,6,7,8,9,10,11	101 010 110 100 110 100 111 101 1	NI-5a

[a]S, sensitive; R, resistant.
[b]Genotype as designated by Cooke *et al.* (2006).

Table 2 Characteristics of parental isolates of *Phytophthora infestans* used to inoculate potato field trials in Michigan, USA in 2003, 2004 and 2005

Isolate	Group number	Mating type	*Gpi* genotype	mtDNA haplotype	R-gene phenotype	RG57 fingerprint	Genotype[a]
Pi 95-3[b]	1	A1	*86/100*	Ib	5	101 010 101 100 110 100 011 001 1	US-1
Pi 96-2[b]	2	A1	*100/100*	Ib	1,3,5	101 010 101 100 110 100 011 001 1	US-1·7
Pi 95-2	2	A1	*100/100*	Ib	1,9	101 010 101 100 110 100 011 001 1	US-1·7
Pi 02-007[b]	3	A2	*100/111/122*	Ia	1,2,3,4,5,6,7,8,9,10,11	100 010 000 100 110 100 011 011 1	US-8
Pi 95-7	3	A2	*100/111/122*	Ia	1,3,4,5,6,7,10,11	100 010 000 100 110 100 011 011 1	US-8
Pi 02-006	3	A2	*100/111/122*	Ia	3,4,5,6,7,10,11	100 010 000 100 110 100 011 011 1	US-8
Pi 00-003	3	A2	*100/111/122*	Ia	3,5	100 010 000 100 110 100 011 011 1	US-8
SR83-84[b]	4	A2	*111/111/122*	Ia	1,2,4,5,10	100 010 000 100 110 100 011 011 1	US-10
Pi 96-1[b]	5	A1	*100/100/111*	Ia	5	101 011 100 000 110 101 011 001 1	US-11·2
Pi 00-001[b]	6	A2	*100/122*	Ia	1,6	100 010 000 100 110 100 011 011 1	US-14
Pi 94-2	6	A2	*100/122*	Ia	1,2,3,4,6,7,10,11	100 010 000 100 110 100 011 001 1	US-14·1
Pi 98-1	6	A2	*100/122*	Ia	1,5	100 010 000 100 110 100 011 011 1	US-14
Pi 99-2	6	A2	*100/122*	Ia	1,5	100 010 000 100 110 100 011 011 1	US-14

[a]Genotype as designated by Goodwin *et al.* (1995).
[b]Six isolates used to inoculate the first Michigan field trial of 2003.

(Tables 1 and 2). All had been previously characterized and named according to these differences (Goodwin *et al.*, 1998; Cooke *et al.*, 2006). Four isolates were included for each group if possible. For the purposes of this investigation, these isolates are referred to as parental isolates, as opposed to isolates that were collected from inoculated field trials, which are referred to as progeny isolates.

Parental isolates from N. Ireland were distinguished by differences in peptidase (*Pep*) allozyme genotype (Goodwin *et al.*, 1995), mitochondrial DNA (mtDNA) haplotype (Griffith & Shaw, 1998) and sensitivity to the fungicide metalaxyl (leaf disc method described by Cooke *et al.* 2006). Parental isolates from Michigan were distinguished by mating type (see Cooke *et al.*, 2006),

glucose-6-phosphate isomerase (*Gpi*) allozyme genotyping (Goodwin *et al*., 1995) and mtDNA haplotype (as above). Parental isolates and a subset of progeny isolates from both N. Ireland and Michigan were also characterized by RG57 fingerprint, using the method of Goodwin *et al*. (1992) modified by Cooke *et al*. (2006).

Parental isolates from N. Ireland represent the major genotypes that have been detected in this country over the past 10 years [designated NI-1 to NI-5 by Cooke *et al*. (2006)]. Isolates within genotypic groups were identical to each other with respect to these markers and all were of the A1 mating type, as no A2 mating type isolates were found in N. Ireland between 1996 and 2004. Although there was a strong association between the individual isolates that were chosen for each group and their phenotypic and genotypic markers, there were some minor variations in their RG57 fingerprints (Table 1). Isolates from Michigan (Table 2) represent the local population (genotype groups designated US-1, US-1·7, US-8, US-10, US-11 and US-14 by Goodwin *et al*., 1998). Three groups of isolates were A1 mating type and three were A2 mating type. RG57 fingerprints confirmed these genotypic identifications except for minor variations in two isolates (Pi 96-1 and Pi 94-2).

All parental isolates from the N. Ireland and Michigan field trials were tested for virulence against 11 single R-gene differentials (R1–R11), using detached leaflet assays; R-gene differentials were obtained from the Scottish Agricultural Science Agency (SASA) and the United States Department of Agriculture (USDA) Potato Germplasm Unit. A susceptible cultivar, lacking R1–R11 genes, was included in each virulence test as a control. Leaflets from healthy, glasshouse-grown plants were detached, placed in transparent plastic boxes that had a layer of moistened paper towel in the base to maintain high humidity, and inoculated abaxially with a single 20 μL drop of a zoospore/sporangial suspension that contained approximately 2×10^4 sporangia mL^{-1}. Boxes were incubated for 7 days at 15–18°C in a naturally lit room and, subsequently, each leaflet was assessed for the presence or absence of sporulating lesions. The presence of a non-sporulating hypersensitive response was scored as being negative for virulence against the gene. Isolates were assayed twice on separate occasions, with three replicate leaflets tested per assay for each differential and the control. A positive or negative result was recorded if two out of the three leaflets produced identical scores in both assays.

All isolates from N. Ireland, except 23/01, were pathogenic and able to infect leaflets from the R0 and R1 host differentials. Virulence complexity was high, regardless of originating group, with each group containing an isolate virulent on at least six R-gene differentials. Only Group 6 isolates contained virulence factors for all 11 R-genes. Generally, Group 5 isolates possessed the fewest virulence factors, whereas all other groups contained isolates that were pathogenic towards at least eight R-gene differentials.

The virulence complexity of isolates from Michigan was more variable than for isolates from N. Ireland with little consistency observed amongst isolates from the same group. The US-8 group was generally the most complex, with three out of four isolates containing seven or more virulence factors; one isolate was virulent against all 11 R-genes.

Inoculum production

Sporangial/zoospore suspensions were derived from each isolate. In N. Ireland, suspensions were made using 5-day-old sporulating lesions from detached leaflets of a susceptible cultivar. Spores were brushed from the surface of the leaf into sterile distilled water using a wet sable brush. In Michigan, 7 to 10-day-old cultures growing on rye A agar (Caten & Jinks, 1968) plates were used. Spores were brushed into sterile distilled water from the surface of the agar plate using a rubber policeman. Suspensions were standardized to approximately 2×10^4 sporangia mL^{-1}, cooled at 5°C for 2 h and examined under the microscope to confirm the release of zoospores. Isolates were then combined into their respective groupings (Tables 1 and 2) in equal volume (described above).

Field trial planting and inoculation

Field trials were planted at sites in Belfast, N. Ireland and Laingsburg, Michigan, during May or early June in 2003, 2004 and 2005. Six field trials were planted in total. Four cultivars that had differing resistance ratings to foliar late blight were included in each trial, except for the Michigan field trial of 2005, in which eight cultivars were used. Resistance ratings were based on National Institute of Agricultural Botany (NIAB), Cambridge, UK ratings or breeders' estimates for foliar blight resistance, using a 1–9 scale on which 9 is maximum resistance (Anonymous, 1999). The commercial cultivars Atlantic (NIAB rating 3) and Stirling (NIAB rating 8) were included at both sites; cvs Santé (NIAB rating 7) and Milagro (breeder's estimate 8) were planted only in the N. Ireland field trials, and cvs Pike (breeder's estimate 3) and Jacqueline Lee (breeder's estimate 8; Douches *et al*., 2001) were planted only in the Michigan trials. In 2005, in Michigan, Dakota Crisp (breeder's estimate 3), MSI049-A, MSI152-A and MSJ461-1 (breeder's estimate of 6, 7 and 8, respectively) were also included.

Each cultivar was replicated four times using a randomized complete block design, in which each block contained four rows of 10 tubers each and each row was planted 0·3 m apart. Each plot was 3 m long and a 1·5 m path was incorporated between each block. Six rows of susceptible infector plants (cv. Désirée in N. Ireland and FL1879 in Michigan) were planted flanking the trial plots, two on each side and two in the centre. Irrigation was applied to supplement natural rainfall through the use of overhead sprinklers. Each trial plot was inoculated with all six groups except for the N. Ireland trial of 2005 in which Groups 2 and 5 were omitted owing to the loss of pathogenicity. Each infector row of trial plots was inoculated

with equal amounts of standardized inoculum from each group and all six groups were inoculated separately onto each row. For the N. Ireland trials, every fourth plant was inoculated with an individual group that was chosen at random. Multiple leaflets from two leaves per plant were inoculated. For the Michigan trials, the two infector rows that were adjacent to each trial plot were divided into six sections and sprayed with equal amounts of an individual group that was chosen at random. An epidemic was established and allowed to spread to surrounding test cultivars.

Epidemic assessment and characterization

Plots were monitored for first disease symptoms in the susceptible spreader rows and each inoculated plant/section was inspected for successful infection as indicated by the presence of sporulating lesions. Disease assessments were conducted every 4 days following detection of the first disease symptoms in the susceptible cultivar plots using the ADAS blight assessment key (Anonymous, 1976) for foliar infection amended by the addition of the 0·01 and 10% disease severity categories. Single-lesion leaflets were removed from each plot once approximately 1% infection was reached by arbitrarily selecting 10 leaflets from different plants within each plot. Thus, sampling was carried out based on the infection of individual plots rather than on the infection of particular cultivars. This stage of the epidemic was chosen for sampling because secondary spread of the pathogen had occurred within the cultivar plots and, although sufficient lesions were present for sampling, extensive foliage death had not occurred. If cultivars were highly resistant, and therefore unlikely to reach the 1% symptom level, lesions were removed once 100% defoliation was reached in the susceptible cultivars. The spreader host varieties in both N. Ireland and Michigan were also sampled in 2005. Ten single-lesion leaflets were removed arbitrarily from each of the six spreader rows when each reached approximately 1% infection.

Samples were incubated for 24–48 h under high humidity to encourage sporulation before isolation into pure culture using amended rye A media (described above). This was achieved either by the transfer to media of sporangia from sporulating leaflets using agar cubes (*c.* 3 mm) or directly using small sections of infected leaf material. Isolates were subsequently maintained on pea agar before being characterized using previously described tests and reassigned to their respective groups. All progeny isolates from N. Ireland were distinguished by differences in *Pep* allozyme genotyping, mtDNA haplotype and sensitivity to the fungicide metalaxyl. Progeny isolates from Michigan were primarily characterized by *Gpi* allozyme genotype, with all isolates in 2003 and a subset in 2004 and 2005 tested for mating type. A subset of progeny isolates from both locations in 2003 (selected to include representatives of each group from each cultivar) and a subset of Group 4 isolates from the 2004 Northern Ireland trial were tested for RG57 fingerprint.

Data analysis

Relative area under the disease progress curves (RAUDPCs) were calculated for individual cultivars in each trial using evaluations for mean percentage foliar late blight as key temporal reference points (Madden & Hughes, 1995). The following formula was used for all calculations

$$\text{RAUDPC} = \frac{\sum_{i=0}^{\text{final}} (T_i - T_{i-1})(P_{i-1}) + \frac{(T_i - T_{i-1})(P_i - P_{i-1})}{2}}{(T_{\text{final}} - T_0)(100)}$$

in which T_0 was day of inoculation, T_i was the ith day after inoculation when an estimation of percentage foliar late blight was made, T_{final} was the number of days after inoculation at which time the most susceptible cultivar had reached 100% defoliation and P_i was estimated percentage foliar late blight at T_i (Stein & Kirk, 2002). Evaluations were continued until 100% foliar infection was reached in the most susceptible cultivar. Significant differences between cultivars in separate years were calculated by the use of two-way analysis of variance with the Tukey multi-comparison pairwise test (Tukey, 1953). Differences across years were also analyzed by ANOVA with mean RAUDPCs for each year treated as a separate block. The statistical software SigmaStat, version 2·03 (Systat Software Inc.) was used for all ANOVA tests.

Pearson chi-squared analyses for goodness-of-fit (Chernoff & Lehmann, 1954) were used to test for the significance of associations between the observed number of isolates of each genotype group from individual cultivars, as derived from the total number of isolates recovered, and the expected number based on the proportion of each group in the inoculum. These tests were based on the assumption that each group became established in the spreader rows to the same extent. If the expected number of isolates per group was fewer than five, chi-squared analysis was not reported as the sample was considered to be too small to be representative. The expected proportions were 0·17 if six groups were inoculated and 0·25 if four groups were inoculated. Thresholds of significance for all tests were $P < 0{\cdot}05$.

Results

Field trial epidemic characterization

Assessment of the spreader rows in each trial for the presence of sporulating lesions showed that all were successfully infected and that each group established an infection. Late-blight epidemics spread quickly through all six inoculated field trials (Fig. 1) and significant differences were observed between the RAUDPCs of cultivars from each trial (Table 3). Across years there was a significant difference in the average RAUDPC values of cultivars planted in both N. Ireland and Michigan field trials (Table 3). In N. Ireland, Atlantic was the most

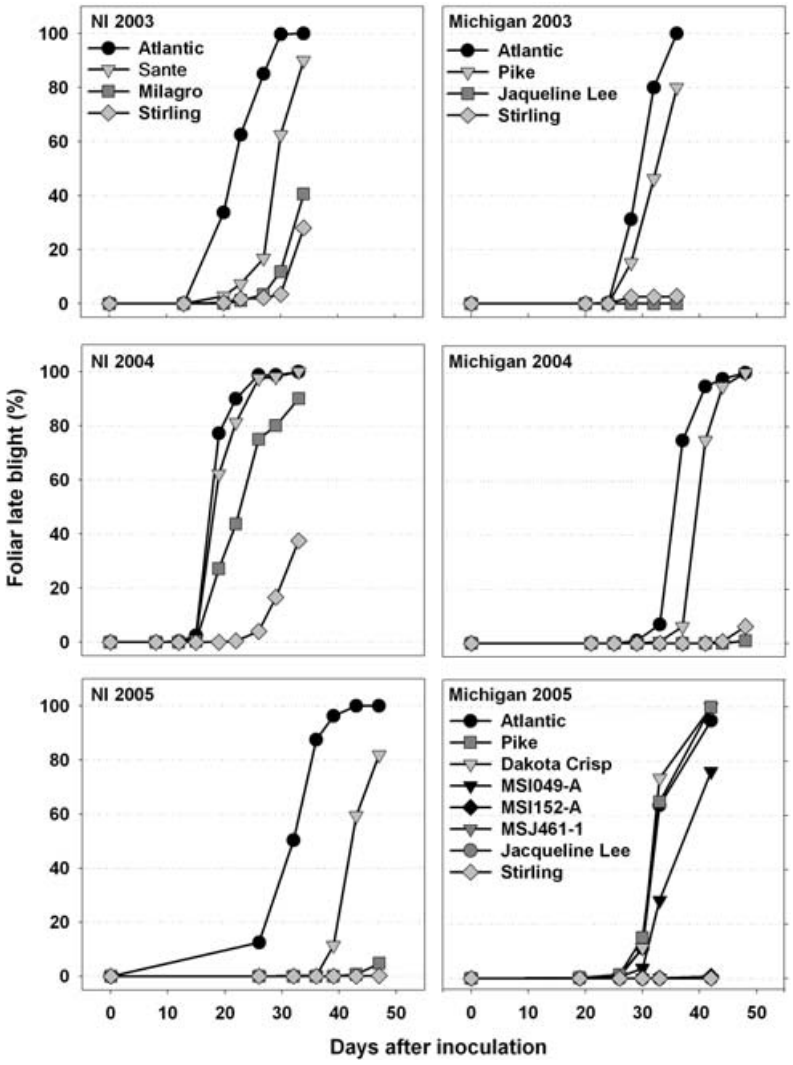

Figure 1 Mean percentage foliar late blight of potato cultivars in Northern Ireland, UK (NI) and Michigan, USA infected with a mixed inoculum of *Phytophthora infestans* in 2003, 2004 and 2005 (characteristics of inoculum are listed in Tables 1 and 2 for N. Ireland and Michigan, respectively).

susceptible cultivar, whereas cvs Santé and Milagro were intermediately susceptible. Stirling was more resistant than Atlantic and Santé. However, there was no significant difference across years between Milagro and Stirling, which were the two most resistant cultivars in all three trials. In field trials conducted in Michigan, cvs Atlantic and Pike were the most susceptible. Atlantic was significantly more susceptible than Pike in two of the three field trials, but across years there was no significant difference between them. Stirling and Jacqueline Lee exhibited consistently high levels of resistance and there was no significant difference between the two in any trial. Of the extra cultivars planted in the Michigan field trial of 2005, Dakota Crisp was the most susceptible (not significantly different from Atlantic and Pike), advanced breeding line MSI049-A exhibited intermediate susceptibility, whereas MSI152-A and MSJ461-1 were highly resistant to foliar late blight (not significantly different from cvs Stirling and Jacqueline Lee).

Competitive selection

The number of lesions removed from each cultivar during the six field trials (data not shown) and the number of isolates successfully sub-cultured onto media for subsequent characterization were variable (Table 4). In total, 496 isolates from N. Ireland and 374 isolates from Michigan were characterized from the main plots. Furthermore, 50 isolates from N. Ireland and 40 isolates from Michigan were recovered and characterized from the susceptible spreader rows. In general, fewer isolates were recovered from the more resistant cultivars. This was due either to a lack of lesions in the field or the difficulty in sub-culturing from more resistant leaflets, which generally produce fewer sporangia. Chi-squared analyses compared the observed number of isolates from each group with the expected number, which was derived from the total number of isolates recovered from a particular cultivar.

Table 3 Relative area under the disease progress curve (RAUDPC) values for potato cultivars exposed to mixed inoculum of *Phytophthora infestans* in field trials in Northern Ireland, UK and Michigan, USA in 2003, 2004 and 2005

Location	Cultivar/ Breeding Line	RAUDPC[a] 2003	2004	2005	Mean
Northern Ireland	Atlantic	36·3 a	45·0 a	30·1 a	37·2 a
	Santé	14·6 b	42·1 a	3·8 b	20·2 b
	Milagro	4·1 c	29·5 b	0·0 b	11·2 bc
	Stirling	2·5 c	4·5 c	0·0 b	2·3 c
	S.E.[b] (9 d.f.)	1·85***	1·50***	1·76***	6·64**
Michigan	Atlantic	40·4 a	25·3 a	20·4 a	28·7 a
	Pike	25·4 b	17·1 b	21·5 a	21·2 a
	Stirling	1·6 c	0·3 c	0·0 c	0·6 b
	Jacqueline Lee	0·0 c	0·0 c	0·0 c	0·0 b
	Dakota Crisp	n/a	n/a	22·3 a	n/a
	MSI049-A	n/a	n/a	12·7 b	n/a
	MSI152-A	n/a	n/a	0·1 c	n/a
	MSJ461-1	n/a	n/a	0·0 c	n/a
	S.E. (9 d.f.[c])	1·62***	0·12***	1·19***	4·10***

[a]Relative area under the disease progress curve value accompanied by a letter that indicates significant difference grouping as measured by the Tukey multiple comparison method; cultivars sharing a common letter were not significantly different from others assessed at the same site in the same year. All comparisons were tested at $P < 0{\cdot}05$.
[b]Standard error of the mean.
[c]Except for 2005 Michigan trial; d.f. = 21.
n/a not applicable.
$^{**}P < 0{\cdot}01$; $^{***}P < 0{\cdot}001$.

Table 4 Number of isolates of *Phytophthora infestans* derived from each potato cultivar in field trials in 2003, 2004 and 2005 in Northern Ireland, UK and Michigan, USA with corresponding foliar blight resistance ratings

Location	Cultivar	2003	2004	2005	Resistance ratings[a]
Northern Ireland	Atlantic	40	40	38	3[b]
	Santé	37	40	39	7[b]
	Milagro	34	40	40	8[c]
	Stirling	29	35	33	8[b]
Michigan	Atlantic	40	40	29	3[b]
	Pike	40	39	19	3[c]
	Stirling	40	22	4	8[b]
	Jacqueline Lee	6	10	2	8[c]
	Additional cvs[d]	n/a	n/a	43[e]	see footnote[d]

[a]For foliage blight (1–9 scale; 9 = maximum resistance).
[b]National Institute of Agricultural Botany (NIAB) rating.
[c]Breeder's estimate.
[d]Four additional cultivars included were Dakota Crisp, MSI049-A, MSI152-A and MSJ461-1 with foliar blight resistance ratings of 3, 6, 7 and 8, respectively (breeders' estimates).
[e]30, 12, 1 and 0 isolates from Dakota Crisp, MSI049-A, MSI152-A and MSJ461-1, respectively.

A clear pattern of selection was observed for all three N. Ireland field trials (Fig. 2), with fewer genotype groups being recovered from the more resistant cultivars. There was a significant difference between the expected number of isolates per group and the number of isolates that were found to have infected the cvs Atlantic, Santé and Milagro in all three trials. In 2003, all six groups infected cv. Atlantic ($\chi^2 = 11{\cdot}89$, d.f. = 5, $P < 0{\cdot}05$), whereas in 2004 only Groups 2, 4 and 6 were detected on this cultivar ($\chi^2 = 70{\cdot}36$, d.f. = 5, $P < 0{\cdot}001$). In 2005, in which only the most aggressive four groups (2, 3, 4 and 6) were inoculated, all four were detected ($\chi^2 = 63{\cdot}47$, d.f. = 3, $P < 0{\cdot}001$). Different groups dominated the infection of cv. Atlantic for all three seasons, with little consistency observed for this highly susceptible cultivar. In 2003 and 2004, Group 6 dominated the infection of cv. Santé (2003: $\chi^2 = 98{\cdot}73$, d.f. = 5, $P < 0{\cdot}001$; 2004: $\chi^2 = 166{\cdot}71$, d.f. = 5, $P < 0{\cdot}001$), and few other groups were detected. In 2005 ($\chi^2 = 22{\cdot}23$, d.f. = 3, $P < 0{\cdot}001$), all four groups were detected, but Groups 2 and 6 were detected in greatest number. Extreme selection was observed in the infection of cv. Milagro, with only Group 6 isolates being detected in all three years (2003: $\chi^2 = 170$, d.f. = 5, $P < 0{\cdot}001$; 2004: $\chi^2 = 199{\cdot}9$, d.f. = 5, $P < 0{\cdot}001$; 2005: $\chi^2 = 186{\cdot}67$, d.f. = 3, $P < 0{\cdot}001$). Likewise, few groups were found to infect the comparatively resistant cv. Stirling in 2004 and 2005 (2004: $\chi^2 = 142{\cdot}09$, d.f. = 5, $P < 0{\cdot}001$; 2005: $\chi^2 = 64{\cdot}09$, d.f. = 3, $P < 0{\cdot}001$); the expected number of isolates for each group was less than five in 2003 and therefore considered to be too small for analysis. The same trend was observed in all three years, whereby Group 4 isolates were found in greatest number. In 2003 and 2005, Group 3 was also able to infect Stirling to varying degrees; Group 3 differs from Group 4 only in its resistance to the fungicide metalaxyl. Small numbers of Group 6 were also detected in 2004 and 2005. In 2005, one isolate that was identical to Group 4 but possessed the *Pep 96/100* allozyme genotype was recovered. The origin of this isolate is unclear and therefore it was not included in the analysis of Stirling in 2005. Significant differences were detected from the susceptible spreader cv. Désirée in 2005 ($\chi^2 = 36{\cdot}88$, d.f. = 3, $P < 0{\cdot}001$). Groups 2 and 4 were dominant, but smaller numbers of all the other inoculated groups were detected.

In all three Michigan field trials 97% of the isolates recovered were US-8 (Fig. 2). Only two other genotype groups were recovered (US-10 and US-14) and these were found only in small number. In 2003, one US-14 isolate was recovered from Pike. In 2004, one isolate of the US-10 lineage and one isolate of the US-14 lineage were recovered from cv. Atlantic, and in 2005 eight US-14 isolates were recovered, of which four were from cv. Pike, two were from MSI049-A and two were from the susceptible spreader rows. Both US-10 and US-14 are mating type A2 and mitochondrial haplotype Ia. Significant differences were observed in 2003 and 2004 in the observed number of the six inoculated groups that were found on the susceptible cvs Atlantic (2003: $\chi^2 = 199{\cdot}9$, d.f. = 5, $P < 0{\cdot}001$; 2004: $\chi^2 = 176{\cdot}81$, d.f. = 5, $P < 0{\cdot}001$) and Pike (2003: $\chi^2 = 188{\cdot}2$, d.f. = 5, $P < 0{\cdot}001$; 2004; $\chi^2 = 195$, d.f. = 5, $P < 0{\cdot}001$). The same trends were observed

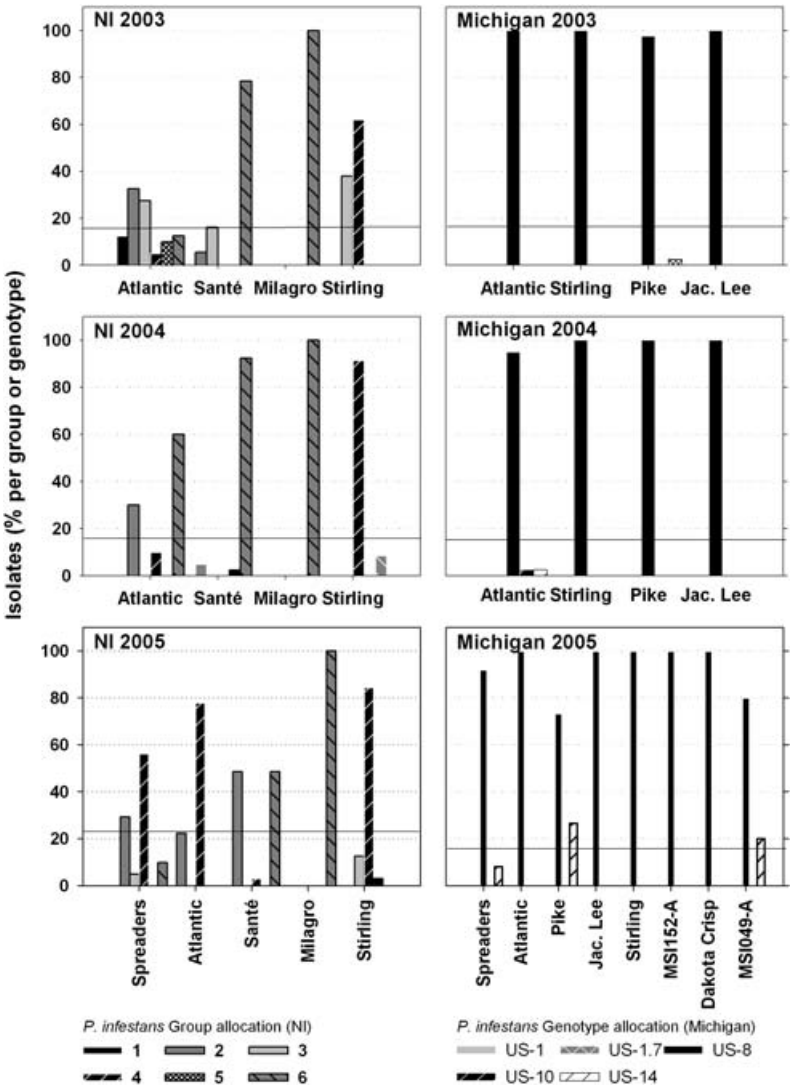

Figure 2 Percentage of isolates of individual groups and genotypes of *Phytophthora infestans* identified from inoculated potato cultivars in field trials in Northern Ireland, UK (NI) and Michigan, USA, respectively, in 2003, 2004 and 2005. The expected percentage frequency per group or genotype is indicated by the horizontal line.

in 2005, although the sample size was too small for statistical analysis. Significant differences were observed in 2003 on the resistant cv. Stirling (χ^2 = 199·9, d.f. = 5, P < 0·001). In 2004 and 2005, sample size was again too small for analysis although the same trends were observed. Jacqueline Lee showed a high level of resistance in the field and consequently few lesions were available for characterization (Table 4). However, the only group found to infect this cultivar was US-8. Of the four extra cultivars planted in 2005, significant differences were observed only in the susceptible cv. Dakota Crisp (χ^2 = 150, d.f. = 5, P < 0·001). All isolates recovered were US-8. The potato advanced breeding lines, MSI049-A, MSI152-A and MSJ461-1 were highly resistant to foliar late blight and few lesions were detected. Significant differences were detected from the susceptible spreader FL1879 in 2005 (χ^2 = 166·7, d.f. = 5, P < 0·001). Again US-8 was dominant with a small number of US-14 isolates being detected. All Michigan progeny isolates tested for mating type proved to be A2. Additionally, with one exception (Table 5), all progeny isolates from both N. Ireland and Michigan that were characterized by RG57 analysis had fingerprints that were identical to those commonly found in their designated group.

Discussion

To investigate selection and competition between *P. infestans* genotype groups in the field as influenced by cultivar, it is necessary to establish an inoculum source. In the present study, sporangial/zoospore suspensions were used to inoculate spreader rows so that the amount of inoculum of each group could be equalized for each trial. Moderately susceptible cultivars that would not be killed too quickly and would provide a continuing source of inoculum for the test plots, but yet could be infected by all the isolate groups, were chosen for these rows. However, the drawback to this method is that although each

Table 5 RG57 fingerprints for selected progeny isolates of *Phytophthora infestans* from Northern Ireland, UK and Michigan, USA field trials in 2003 and Northern Ireland field trial in 2004

Location	Year	Group number[a]	Number of isolates tested	RG57 fingerprint	Genotype[b]
Northern Ireland	2003	1	1	100 010 001 100 110 100 011 001 1	NI-1
		2	2	100 010 001 100 110 100 011 001 1	NI-1
		3	3	110 010 000 100 110 010 011 101 1	NI-2[c]
		4	7	111 010 100 100 110 100 011 101 1	NI-3
		5	1	111 010 100 000 110 010 011 101 1	NI-4
		6	11	101 011 110 100 110 100 111 001 1	NI-5
Northern Ireland	2004	4	12	111 010 100 100 110 100 011 101 1	NI-3
Michigan	2003	3	20	100 010 000 100 110 100 011 011 1	US-8
		6	1	100 010 000 100 110 100 011 011 1	US-14

[a]Based on *Pep* allozyme genotype, mtDNA haplotype and sensitivity to metalaxyl in N. Ireland (Table 1) and *Gpi* allozyme genotype in Michigan (see Table 2).

[b]Genotype as designated by Cooke *et al.* (2006) for N. Ireland isolates and Goodwin *et al.* (1998) for Michigan isolates.

[c]One of the three isolates from Group 3 had a novel fingerprint that differed from Group 4 isolates by the presence of band 21, and was designated NI-3a.

genotype group could be shown to have successfully infected the spreader cultivars, quantitative levels of establishment could not be determined and therefore the amount of inoculum from each group to reach the test cultivars was unknown. Ideally, the amount of inoculum of each group to which the test cultivars were exposed would have been equalized, but in practice this would be virtually impossible. To address the issue of possible differences in establishment of genotype groups, in 2005 the proportions of each group in the spreader rows were determined by sampling and characterizing isolates at 1% infection, to indicate relative establishment. All four groups used in that year were detected from the N. Ireland spreader cultivar, albeit in unequal proportions, but only the US-8 and US-14 lineages were detected in Michigan. The most probable explanation is that in both countries certain genotype groups were fitter than others even on the susceptible spreader rows and became established to different extents. Nonetheless, the possibility that some aspect of inoculum production or of the inoculation process may have favoured one pathogen group over another cannot be completely ruled out.

Extreme selection was observed within genotype groups of the N. Ireland *P. infestans* population on all the test cultivars and this was not consistent with differences in the initial inoculum availability (as indicated by sampling from the spreader rows). Consistent trends were observed across all three N. Ireland trials, whereby different groups dominated the infection of different cultivars. Selection was most marked, and most consistent between years, on the more resistant cvs Milagro and Stirling. Significant cultivar effects were also observed on the susceptible cv. Atlantic and intermediately resistant cv. Santé, but with some variation between years in the groups that were selected. It is thus likely that the *P. infestans* population in N. Ireland is strongly influenced by the cultivar grown.

The genetic mechanisms that underlie the observed N. Ireland cultivar–pathogen genotype interactions are unclear. The susceptible cv. Atlantic is known to contain only the R1 resistance gene (Rubio-Covarrubias *et al.*, 2005) and the resistant cv. Stirling contains one R-gene that is probably R7 (Stewart *et al.*, 2003), but it is not known if the other cultivars used in these trials carry any R-genes. However, in laboratory testing, using detached leaflets of the test cultivars, at least one isolate from each group inoculated was virulent and capable of infecting each cultivar to some extent (G. K. Young, unpublished observations). In addition, in the present study, one group containing isolates that were virulent to the 11 known *Solanum demissum* R-genes was introduced into each N. Ireland trial, and this group, although dominant on cvs Santé and Milagro, was not selected to a large extent on the other cultivars. Virulence complexity was also high for the other five N. Ireland groups inoculated. However, it cannot be ruled out that the specific selection observed was attributable to interactions with other unidentified R-genes.

Specific selection was not observed within the Michigan competitive field trials, in which the US-8 genotype dominated the infection of all cultivars tested, including the spreader row breeding line FL1879, and few isolates of other genotype groups were recovered. The US-8 genotype was first detected in New York State, USA in 1992 and spread rapidly during the years 1994 to 1996 to become the most commonly isolated lineage in most regions of the USA (Goodwin *et al.*, 1998). Detached leaflet studies indicated that the US-8 genotype was significantly more aggressive than the 'old' US-1 genotype (Miller *et al.*, 1998). Although the fitness of the US-8 genotype has been compared with the older US-1 lineage (Miller *et al.*, 1998), the present study is the first competitive field comparison of the US-8 genotype with other 'new' genotypes of *P. infestans*. These results demonstrate

that the US-8 genotype has a higher foliar competitiveness than other genotypes found in Michigan over the past 10 years, which has probably led to its dominance within the population. It is therefore proposed that cultivar-specific selection of genotypes is unlikely to be important in Michigan at this time. However, because the US-8 genotype was not successful in infecting a number of the resistant cultivars that were used in these trials, there may be potential for such selection in the future.

Although both mating types were used in the Michigan trials, there was no evidence of any sexual recombination or generation of variation by asexual mechanisms, and all progeny isolates shared common genotypes by both allozyme and RG57 fingerprint with those of the parental isolates. Both mating types have existed in the United States since the introduction of the A2 mating type in 1992 (Goodwin *et al*., 1998), but previous studies have indicated that, in general, opportunities for sexual reproduction are limited, with most populations being composed of a single mating type (Goodwin *et al*., 1998). Even in areas in which both mating types coexist, sexual reproduction seems to be infrequent, although certain lineages may have been produced by rare sexual recombination events (Goodwin *et al*., 1998). In this study, the US-8 lineage, which has the A2 mating type, dominated the foliar epidemics to such an extent that the possibility of sexual recombination was effectively excluded; such dominance may explain, at least in part, why sexual reproduction of *P. infestans* in the United States is so infrequent.

It is not clear if the cultivar-specific selection of *P. infestans* genotypes that was found in the N. Ireland trials would lead to the erosion of cultivar resistance. Van der Plank (1971) reviewed the stability of field resistance and found little evidence for a differential interaction between specific cultivars and races of *P. infestans*. However, this work pre-dated the emergence of the new *P. infestans* populations, which possess greater genetic variability. Theoretical population models have been inconclusive as to predictions regarding the potential breakdown of partial resistance. Gandon & Michalakis (2002) predicted that the use of hosts that have partial resistance to a particular pathogen would increase the selection of aggressive isolates and lead to the breakdown of such resistance, whereas Gould *et al*. (1991) argued that aggressiveness would evolve only slowly in such situations. Although Harrison (1992) proposed that for *P. infestans* the degradation of partial resistance was likely, owing to its high multiplication rate and the polycyclic nature of epidemics, in practice there is little evidence to support this hypothesis. Most studies have reported that partial resistance has been highly durable and robust over time and in differing locations (Forbes *et al*., 2005) and cultivars have maintained relative resistance rankings even when changes have occurred in the pathogen population (Grünwald *et al*., 2001).

A number of studies have reported significant pathogen isolate × cultivar interactions and some have associated these with a potential erosion of partial resistance. Using detached leaflets in laboratory assays, Day & Shattock (1997) found a higher level of aggressiveness of isolates of the A2 mating type on detached leaflets of cv. Cara compared with isolates of the A1 mating type. Similarly, Carlisle *et al*. (2002) found that one isolate of the US-8 lineage (also of the A2 mating type) was specifically aggressive to cv. Cara compared with isolates from N. Ireland, although it was not more aggressive than other isolates to cvs Binje and Stirling. Flier *et al*. (2003) detected significant differences in the susceptibility of cultivars that were grown in field-trial conditions and inoculated with isolates of differing aggressiveness, which was interpreted as evidence for the erosion of partial resistance. Montarry *et al*. (2006), who studied French populations of *P. infestans*, showed that the local populations were structured by host cultivars for both qualitative (virulence) and quantitative (aggressiveness) components of pathogenicity. In a recent study that compared isolates sampled from France and Morocco on detached leaves of the potato cvs Bintje (commonly grown in France) and Désirée (more commonly grown in Morocco than France), it was proposed that differences in aggressiveness were due to adaptation to the locally grown cultivar (Andrivon *et al*., 2007). Jinks & Grindle (1963) and Hussain (2003) reported specific adaptation to particular cultivars during 'passaging' experiments in the laboratory, which suggests that some isolates of *P. infestans* might evolve rapidly in response to growth on different cultivars, thereby leading to adaptation within populations. In general, cultivars from the present study exhibited similar resistance levels to foliar late blight to those predicted by NIAB or breeders' ratings. The exception was cv. Santé, in which the observed resistance levels were consistently lower than that expected from the comparatively high NIAB resistance rating of 7 for foliar late blight. This difference in resistance was also recorded in a study of foliar resistance in Latvia, from which it was suggested that cv. Santé had been previously misclassified (Hansen *et al*., 2005). Thus, despite the extreme *P. infestans* genotype × potato cultivar effects that were observed during all three N. Ireland field trials, the resistance levels of the potato cultivars chosen were not observed to have been eroded significantly over time.

The mechanisms by which pathogen genotypes compete are not well understood. It is possible that different genotypes induce the production of defence proteins at different times in particular cultivars, but yet still successfully infect that cultivar (e.g. Wang *et al*., 2008). However, in a competitive situation, genotypes that induce defence proteins earlier than others and are better able to overcome this earlier induction would have the competitive advantage, which, coupled with the subsequent inhibition of other genotypes, would explain the competitive selection that was shown in this study. This could occur even though isolates are not differentially aggressive on specific cultivars in the absence of the competitive interaction. An investigation of the individual and competitive aggressiveness of the genotype groups that were used in this study on detached leaflets will be reported in a subsequent paper.

Results from the present study indicate marked cultivar-specific differences in fitness between the genotype groups from N. Ireland, but not Michigan. Most notably, the relatively new resistant cv. Milagro (which has not been commercialized to date) was infected only by NI-5 (Group 6) isolates, a genotype that was capable of infecting the susceptible cvs Atlantic and Désirée, but was detected in extremely low proportions on these cultivars compared with other genotypes. The NI-5 genotype is infrequent in N. Ireland (Cooke *et al*., 2006) and has been isolated only from natural infections of partially resistant potato cultivars and has not been found in commercial crops (L. R. Cooke, unpublished observations). Its apparent competitive disadvantage in infecting susceptible cultivars, which has also been noted on tubers (J. M. Thompson, personal communication), might help to explain its rarity in N. Ireland, where most of the potatoes grown are extremely susceptible to late blight. If cv. Milagro or related cultivars were grown on a larger scale in N. Ireland, the impact on the local *P. infestans* population could be marked. However, the extreme selection for US-8 that was observed in Michigan suggests that unless another fit genotype emerges in the pathogen population, the US-8 genotype will probably continue to dominate there regardless of the cultivars that are grown.

Although it is clear that the choice of cultivar can substantially impact the surrounding late-blight population, its impact on the control and management of epidemics remains unknown. Emergence and selection of particular genotypes (e.g. US-8) can lead to serious consequences for the potato industry (Goodwin *et al*., 1998). If a genotype were selected through an enhanced ability to infect a particular cultivar that was not previously grown in the area, other linked characteristics could impact greatly on the severity of the epidemic; this has previously been described as the 'hitch-hiking' effect (Kojima & Schaffer, 1967). For example, a genotype might be more aggressive on tubers than existing genotypes, or a different mating type could be selected that allows sexual reproduction to take place, which ultimately could lead to further selection of new aggressive genotypes: this may have already occurred, as in Great Britain in 2005. After the present study had been completed, a new, highly aggressive genotype of the A2 mating type was identified (Shaw *et al*., 2007) and was found in 65% of outbreaks sampled in 2006 (Cooke *et al*., 2007). This genotype was identified for the first time in N. Ireland in 2007 (Cooke *et al*., 2008); it is not yet clear how this may affect the *P. infestans* population in N. Ireland. It is important that new cultivars are tested in a competitive environment, not only to measure resistance levels but also evaluate the potential impact on the surrounding late blight populations.

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