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Incidence of *Phytophthora infestans* (Mont.) de Bary on potato and tomato in Maine, 2006–2010

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Abstract: Late blight, caused by *Phytophthora infestans* (Mont.) de Bary, is a devastating disease which is found worldwide. In Maine, United States (US), we recorded late blight on potato and tomato during the 2006–2009 cropping seasons. From 2006 to 2008, over 90% of the diseased samples were collected in potato fields from northern and central Aroostook County in Northern Maine, US. Then, in 2009, an unprecedented influx of inoculum on infected tomato transplants shipped to retail garden centers throughout the Northeast US significantly changed the late blight infection patterns. In 2009, 43% of diseased samples obtained were from tomato, and 57% from potato, and disease was found to occur all over the state. Moran's index and spatial autocorrelation analysis of disease occurrence, geographical locations, host factors, and infection levels from previous years, were not statistically significant ($p > 0.05$). Therefore, random distributions of late blight incidences were recorded across locations and years. Nearest neighbor analysis revealed that mean spatial distances for late blight occurrence ranged from 1.51 to 71.4 km from 2006 to 2008, and 7.4 to 126.5 km in 2009. The frequency and locations of late blight outbreaks in 2009 were substantially greater than in 2006, 2007, and 2008, as affected by the influx of inoculum and movement of infected tomato seedlings as well as conducive environmental conditions. All were contributing factors for late blight occurrence in Maine. In 2010, few disease samples were collected, indicating that the influx of inoculum in 2009 did not persist to cause widespread disease in 2010. The reduction of inocula sources, fungicide protection of susceptible hosts, and the removal and destruction of infected tomato seedlings and potato cull piles or volunteer plants, can greatly reduce late blight occurrences and improve potato production. These actions should be considered as an integral part of late blight management programmes in regions where late blight commonly occurs.

Key words: disease distribution, *P. infestans*, potato, spatial dependence, tomato

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Introduction

Potato late blight is caused by *Phytophthora infestans* (Mont.) de Bary and regarded as one of the most destructive plant diseases in the world (Birch and Whisson 2001; Judelson and Blanco 2005; Fry 2008). The annual costs for controlling late blight and the resulting losses to potato are approximated to be billions of dollars (Haverkort *et al.* 2008). The occurrence of *P. infestans* is of great significance due to foliar blight and tuber blight losses to potato production (Fry and Goodwin 1997). Significant economic losses or revenue losses of up to 210.7 million dollars are experienced by United States (US) potato growers, of which 77.1 million dollars are losses attributed to fungicide application costs alone (Guenthner *et al.* 1999, 2001). Similar economic losses amounting to millions of dollars may also impact tomato production as a result of late blight in the United States (Guenthmer *et al.* 2001).

Quantifying late blight among and within fields may enhance our ability to manage the disease. This task is complicated by the rapid progress of the disease on potato and tomato. In many cases, alternate hosts such as tomato (*Lycopersicum esculentum* L.), solanaceous weeds like hairy nightshade (*Solanum sarrachoides* Sendt.), volunteer potato plants, or potato refuse/cull piles have all been documented as significant inocula sources contributing to disease spread in potato (Erwin and Ribeiro 1996). Other mechanisms of inoculum dispersal and disease spread in potato have also been documented. Movement of *P. infestans* inocula within soil has been shown to be a contributing factor for the tuber blight phase of the disease (Fairclough *et al.* 1993; Mayton *et al.* 2007; Olanya *et al.* 2009b). Zoospores or sporangia have been implicated in short distance inoculum spread (Fairclough *et al.* 1993).

Aerial dispersal of *P. infestans* sporangia from lesions on leaves and stems of diseased potato or tomato plants is

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a major mechanism for dissemination of late blight from field to field over large areas (Zwankhuizen *et al.* 1998). Similarly, the dissemination of late blight through human activity accounted for the significant spread of the pathogen and diseased plants across regional and continental scales (Fry *et al.* 1993; Campbell 1999; Ristaino and Gumpertz 2000; Ristaino 2002).

Due to the devastating potential of *P. infestans* and the need to quickly control the potato disease (Duncan 1999), patterns of late blight spread within and among fields are not thoroughly documented. Several mechanisms of pathogen and disease spread may occur simultaneously resulting from combinations of aerial dispersal of sporangia and secondary spread, or movement of infected plant materials and alternate hosts.

Over the last several years, *P. infestans* has been reported in commercial potato fieldsin Maine. It has also been reported on tomato and potato in organic and home gardens throughout the state of Maine. In addition, the unique circumstances present in 2009, in which late blight pathogen inoculum was distributed throughout the state on infected tomato seedlings sold at garden centers, may have greatly affected the occurrence and distribution of late blight. Unlike other parts of the United States, late blight occurrence in Maine has been reported consistently every other year providing a residual inoculum reservoir. The broader context of this research is that the potato growing region of Maine is adjacent to the growing areas of the province of New Brunswick (Canada), and in close proximity to potato areas of the provinces of Quebec, Nova Scotia, and Prince Edward Island (Canada). The state of Maine produces seed potatoes that are shipped throughout the United States and to other countries. Changes in pathogen populations, cultural or management practices in Maine can therefore impact potato production elsewhere.

The objectives of this study were to: (1) assess the occurrence and geographical distribution of late blight on potato and tomato in 2006 through 2010, and (2) characterise the seasonal spread of potato late blight in relation to cultivar variation and cropping years. Details of the 2009 late blight pandemic in the eastern United States, as well as a discussion of its causes and results have been previously published (Fry *et al*. 2013).

Materials and Methods

Distribution of *P. infestans* in Maine

Samples of diseased potato or tomato plants provided by the University of Maine Cooperative Extension Service and collaborating growers, were used to document the occurrence and geographical location of late blight. Positive identification of *P. infestans* was based on symptoms typical of late blight and confirmed by pathogen isolation on Rye A media (Caten and Jinks 1968). Identification was followed by microscopic observations of colony morphology, sporangia and sporangiophores.

The locations and counties where diseased plants were obtained, the host or crop cultivar, and the date samples were collected were also recorded from the 2006–2010 growing seasons. Based on the collection record of diseased plants, the approximate geo-referenced field coordinates were determined.

Late blight infection events

Late blight infection events (the number of times late blight occurrence was reported on potato and tomato hosts) at various geographical locations, were recorded over time during each cropping year to document the number of field sites at a location (town) during the cropping cycle. Due to the risk of this disease devastating potato, no attempt was made to quantify late blight severity in the fields where disease was detected.

Frequency of *P. infestans* on tomato and potato

The frequency of *P. infestans* on symptomatic tomato and potato was recorded after positive isolation of the pathogen on Rye A selective medium (Caten and Jinks 1968). The *Phytophthora infestans* genotypes were characterized based on allozyme analysis (Goodwin *et al.* 1995) as well as for phenotypic characteristics such as mating types.

Sensitivity of isolates of *P. infestans* to various fungicides

The *in vitro* sensitivity of *P. infestans* isolates to protectant and systemic fungicides were assessed in laboratory experiments. The inhibition of *P. infestans* on Rye B medium amended with mancozeb, cymoxanil, and chlorothalonil, were investigated. Following the amendment of Rye B medium with mancozeb at 10 ppm, the pathogen isolates (00-87, 02-1, 06-29, 04-39, 99-1, and 99-33) were grown on the amended medium and the plates were incubated for 3 weeks (21 days). Similarly, the growth of isolate 06-29 (US-8 genotype) on Rye B medium amended with mancozeb, cymoxanil, and chlorothalonil at 10 and 100 ppm in comparison to the untreated control (0 ppm) were also investigated. Inhibition of *P. infestans* was computed based on the control treatment, and the results were compared graphically.

The fungicide effect on germination of zoospores and sporangia was also conducted on representative isolates of *P. infestans*. Germination of zoospores and sporangia of pathogen isolates 08-14 (100/111/122), 06-76 (111/122), 06-184 (100/122), and 07-8 (100/111/122) in the presence of mancozeb was investigated. The germination of both zoospores and sporangia was conducted on water agar amended with mancozeb at concentrations of 0 (control), 0.1, 1, and 2 ppm. For the germination of zoospores, sporangia harvested from Rye B medium and filtered to remove mycelia, were incubated in cold water (4°C) to stimulate zoospore release from sporangia after the cold shock treatment. Zoospore germination (%) was subsequently quantified on water agar amended with fungicides at 0 (control), 0.1, 1, and 2 ppm. The germination of sporangia (%) was also quantified at the same fungicide concentrations as zoospores.

Statistical analysis

Distribution of late blight occurrence was mapped separately for each year based on infection sites or locations. Spatial distances associated with late blight occurrences were computed based on nearest neighbor indices, which measures the distance between event locations using Geographical Information Systems (GIS) analysis (Nelson *et al.* 1994). The Z-statistics were used to determine significance of spatial distributions (Diggle 1983). The frequency of occurrence of *P. infestans* on diverse hosts (potato or tomato) and their relative occurrence on specific potato cultivars were computed for each year. Spatial autocorrelation analysis between occurrences of late blight to distances, host, and year was used to compute Moran's Index (I) by proc variogram. Data on fungicide effects, the inhibition of pathogen growth on Rye B medium and on the germination of sporangia and zoospores, were analysed by analysis of variance (ANOVA). Means and associated standard errors were computed and plotted graphically. All analyses were conducted using the Statistical Analysis System ver.9 (SAS Institute Inc., Cary, NC).

Results

Distribution of *P. infestans* in Maine

Late blight occurrence varied among cropping years and geographic locations in the northeast state of Maine. Based on positive disease diagnosis and pathogen isolations, the number of locations where disease occurred were [9, 4, 12 during 2006, 2007 and 2008 (Table 1) and 32 in 2009 (Table 2)]. The event locations (sites of late blight occurrence), showed that late blight occurred mainly in northern Maine (Aroostook County) in 2006, 2007, and 2008. The locations of disease occurrence exhibited departure from randomness ($p = 0.0037$) in the northern region during the 2006, 2007, and 2008 cropping years, but late blight was detected throughout Maine in 2009 (Fig. 1). Only three late blight samples, from two locations, both in coastal Maine (Waldeboro and Nobleboro) were collected in 2010, from both potato and tomato. Due to the low occurrence observed, no further analyses were conducted for that year.

The spatial distances of disease-spread computed among locations within each geographical region, were limited to approximately 95 km in 2006. In 2007 and 2008, the spatial distance of disease occurrence was less than 64 km in northern and central Aroostook county, except for a few occurrences in Penobscot county in 2006, as well as in Hancock and Oxford counties in 2008 (Tables 1 and 2). During 2009, the spatial distances of disease occurrences varied, but generally ranged from 7 to 91 km. Two occurrences of late blight disease revealed an average spatial geographical distance of 126 and 182 km from the mean center of occurrences (Table 1). The Moran's Index was non-significant ($p > 0.05$) and, therefore, no time series analysis were conducted.

Late blight infection events

The first outbreak of *P. infestans* occurred on July 13, July 2, July 16, June 25, and July 19, in 2006, 2007, 2008, 2009, and 2010, respectively. Infection events varied during the cropping season and among years. No infection events were recorded before the end of June except in 2009, where four infection events were recorded in June. The greatest number of subsequent new late blight infection events occurred during the weeks of July 15 to July 31 among the years (Fig. 2).

Frequency of *P. infestans* on tomato and potato

Except for the 2007 cropping year, *P. infestans* was detected on potato and tomato in all the years (Fig. 3). Disease recorded on tomato represented 4.5, 5.8, and 43% of late blight occurrences in 2006, 2008, and 2009, respectively. The frequency of *P. infestans* on potato was 96, 100, 94, and 57% in 2006, 2007, 2008, and 2009, respectively (Fig. 3). *Phytophthora infestans* was detected on various potato cultivars. In 2006, late blight was found on cultivars FL1879, FL1533 (early maturity), Shepody, Norwiss, Reba (mid-maturity), and Russet Burbank (late maturity). In 2007, late blight was reported on FL1879 and Russet Burbank. Similar results were observed in 2008 and 2009. In 2008, late blight was recorded on cultivars Superior, Ontario, and Norwiss (mid-maturity) as well as on Russet Burbank and Golden Russet (late maturity), and on tomatoes. In 2009, disease occurrences were recorded on diverse potato cultivars such as FL1533 and Dark Red Norland (early maturity), and on Russet Burbank, Kennebec (late maturity) as well as on unknown potato cultivars, and on tomato. Additionally, *P. infestans* was detected on tomato seedlings (transplants) from various locations as well in several stores and garden centers that sold tomato seedlings in 2009. Late blight was also observed on some potato cultivars that were not readily identified.

Based on allozyme analysis at the glucose-6-phosphate isomerase (*Gpi*) locus (Goodwin *et al*. 1995), the predominant genotype designations of *P. infestans* were 100/111/122 (US-8 genotype) during the 2006, 2007, 2008 cropping years. The percentages of isolates with 100/111/122 genotype designations were 93, 95, and 97% in 2006, 2007, and 2008, respectively. The 100/122 genotype designation was also observed, and constituted 6, 0, and 3% of the isolates for the same years, respectively. Other pathogen genotype designations, such as 100/111 and 111/122 were also observed in 2006 and 2008, at very low frequencies. In 2009, the percentage of isolates that were of 100/111/122 and 100/122 genotype designations were 57 and 43%, respectively. Isolates of the two genotype designations were recorded on diseased potato and tomato plants, and from various geographical locations in Maine. Overall, most of the isolates collected from the potato in northern Maine (mainly commercial fields) were the 100/111/122 (US-8) genotype, whereas most of the isolates on the tomato from central, coastal, and southern Maine were the 100/122 genotype. However, some isolates of 100/122 were found on the potato in

Table 1. Nearest neighbor analysis of average spatial distance of late blight (*Phytophthora infestans*) occurrences in Maine in 2006, 2007, and 2008

Location[a]	County	Sample date	Region	Mean spatial distance [km][b]
Fort Kent	Aroostook	7/13/2006	Northern	71.42
Mars Hill	Aroostook	7/18/2006	Northern	27.91
Bridgewater	Aroostook	7/18/2006	Northern	37.45
Mars Hill	Aroostook	7/27/2006	Northern	10.52
Washburn	Aroostook	7/27/2006	Northern	10.52
Easton	Aroostook	8/2/2006	Northern	14.71
Presque Isle	Aroostook	8/3/2006	Northern	6.36
Caribou	Aroostook	8/3/2006	Northern	14.24
Limestone	Aroostook	8/8/2006	Northern	25.62
Washburn	Aroostook	8/8/2006	Northern	10.52
Mapleton	Aroostook	8/9/2006	Northern	10.67
Caribou	Aroostook	8/10/2006	Northern	14.24
Westfield	Aroostook	8/10/2006	Northern	20.54
Presque Isle	Aroostook	8/14/2006	Northern	6.36
East Corinth	Penobscot	7/18/2006	Southern Interior	20.69
Leeds	Androscoggin	7/30/2006	Southern Interior	95.86
Corrina	Penobscot	7/31/2006	Southern Interior	5.16
Corinth	Penobscot	8/1/2006	Southern Interior	19.32
Orono	Penobscot	8/18/2006	Southern Interior	43.64
Littleton	Aroostook	7/2/2007	Northern	1.51
Houlton	Aroostook	8/15/2007	Northern	12.12
Littleton	Aroostook	8/17/2007	Northern	1.51
Presque Isle	Aroostook	8/23/2007	Northern	51.06
Caribou	Aroostook	7/16/2008	Northern	24.52
Grand Isle	Aroostook	7/21/2008	Northern	26.05
Washburn	Aroostook	8/14/2008	Northern	32.08
Westfield	Aroostook	8/11/2008	Northern	57.49
Fort Kent	Aroostook	7/30/2008	Northern	43.08
Grand Isle	Aroostook	7/30/2008	Northern	26.04
Van Buren	Aroostook	7/30/2008	Northern	14.81
Hamlim	Aroostook	7/23/2008	Northern	22.42
Mars Hill	Aroostook	8/6/2008	Northern	64.40
Easton	Aroostook	8/5/2008	Northern	50.11
Presque Isle	Aroostook	8/5/2008	Northern	44.11
Caribou	Aroostook	8/21/2008	Northern	24.52

[a] locations where occurrences of late blight were reported in 2006, 2007, and 2008

[b] spatial distance refers to mean distance from each location to mean center of locations of late blight occurrence in each region (of the state of Maine) based on the nearest neighbor index analysis

Table 2. Nearest neighbor analysis of average spatial distance of late blight (*Phytophthora infestans*) occurrences in Maine during the 2009 cropping year

Location[a]	County	Sample date	Region	Mean spatial distance [km][b]
Houlton	Aroostook	6/25/2009	Northern	55.74
Presque Isle	Aroostook	7/7/2009	Northern	7.39
Fort Fairfield	Aroostook	7/20/2009	Northern	21.30
Easton	Aroostook	7/31/2009	Northern	7.15
Sherman	Aroostook	7/31/2009	Northern	88.94
Presque Isle	Aroostook	7/31/2009	Northern	7.39
Presque Isle	Aroostook	8/5/2009	Northern	7.39
Mapleton	Aroostook	8/5/2009	Northern	14.75
Bangor	Penobscot	6/25/2009	Southern Interior	16.93
Bangor	Penobscot	7/12/2009	Southern Interior	16.93
Orono	Penobscot	7/13/2009	Southern Interior	25.82
Brewer	Penobscot	7/20/2009	Southern Interior	18.29
Waterville	Kennebec	7/14/2009	Southern Interior	59.35
Sangerville	Piscataquis	7/22/2009	Southern Interior	47.44
Dover Foxcroft	Piscataquis	7/22/2009	Southern Interior	43.85
Canton	Oxford	7/22/2009	Southern Interior	11.36
Newport	Penobscot	7/22/2009	Southern Interior	22.55
Fryeburg	Oxford	7/26/2009	Southern Interior	182.30
Exeter	Penobscot	7/26/2009	Southern Interior	19.92
Albion	Kennebec	7/31/2009	Southern Interior	48.58
Orono	Penobscot	7/31/2009	Southern Interior	25.83
Palmyra	Somerset	8/5/2009	Southern Interior	29.30
Sebec	Piscataquis	8/5/2009	Southern Interior	50.44
Lewiston	Andoscoggin	8/5/2009	Southern Interior	126.54
Ellsworth	Hancock	7/7/2009	Coastal	115.99
Dresden	Lincoln	7/12/2009	Coastal	4.23
Dresden	Lincoln	7/14/2009	Coastal	4.23
Bowdoinham	Sagadahoc	7/14/2009	Coastal	16.07
Warren	Knox	7/20/2009	Coastal	38.47
Warren	Knox	7/22/2009	Coastal	38.47
Lisbon Falls	Androscoggin	7/26/2009	Coastal	28.84
Dayton	York	7/31/2009	Coastal	91.48

[a] locations where occurrences of late blight were reported in 2009

[b] spatial distance refers to mean distances from each location to mean center of locations of late blight occurrence in each region (of the state of Maine) based on the nearest neighbor index analysis

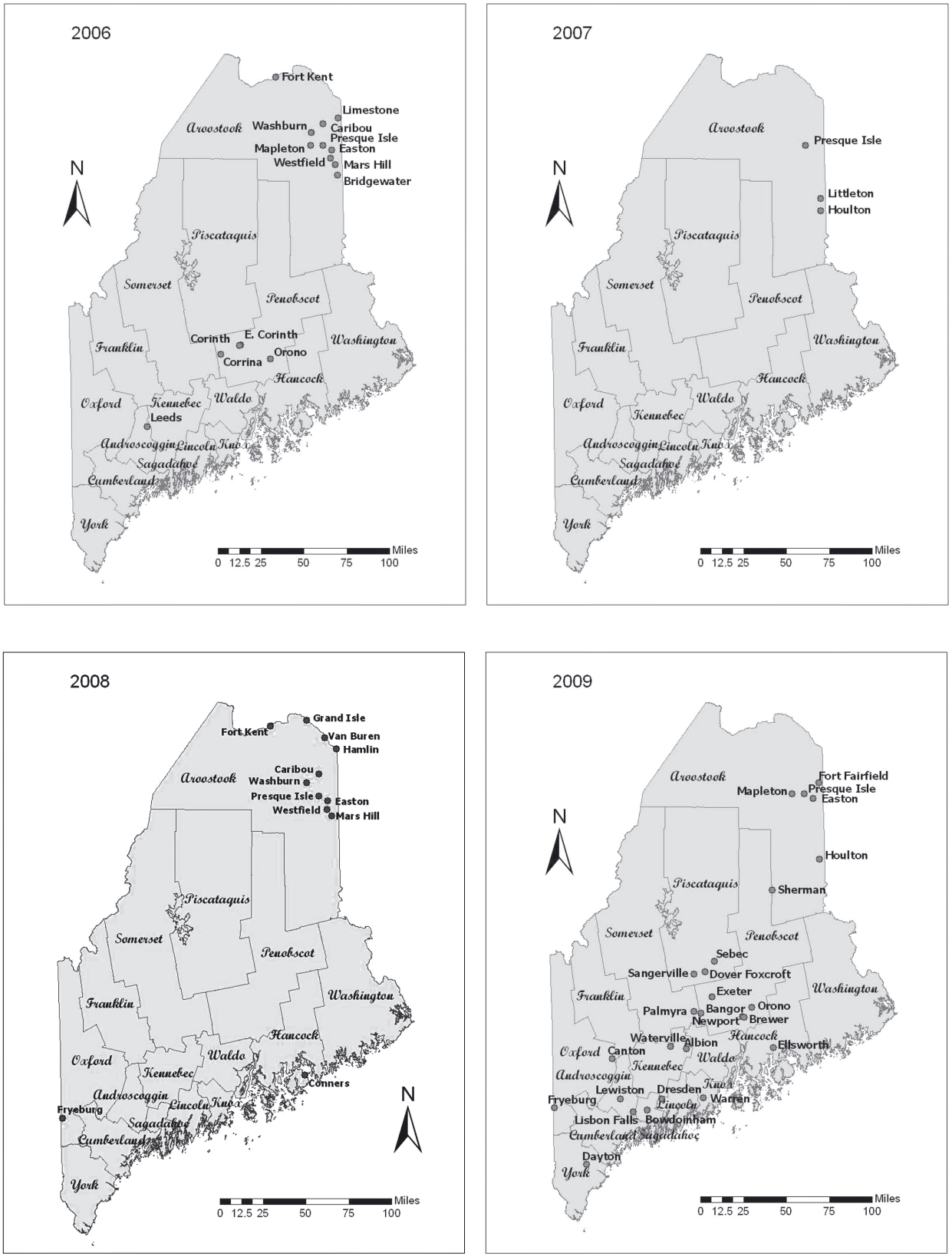

Fig. 1. Distribution of late blight on tomato and potato plants (late blight events) in fields at various geographic locations in Maine. The county names in the State of Maine (US) are in italics

northern Maine, and some isolates of 100/111/122 were also found on the tomato in coastal Maine. The pathogen mating type was A2 for all isolates collected in 2006–2009. Isolates of the 100/122 genotype collected in 2009 were later analysed by others using the RG57 DNA fingerprint probe (Goodwin *et al*. 1992) and determined to be of the new genotype designated US-22 (Hu *et al*. 2012; Fry *et al*. 2013).

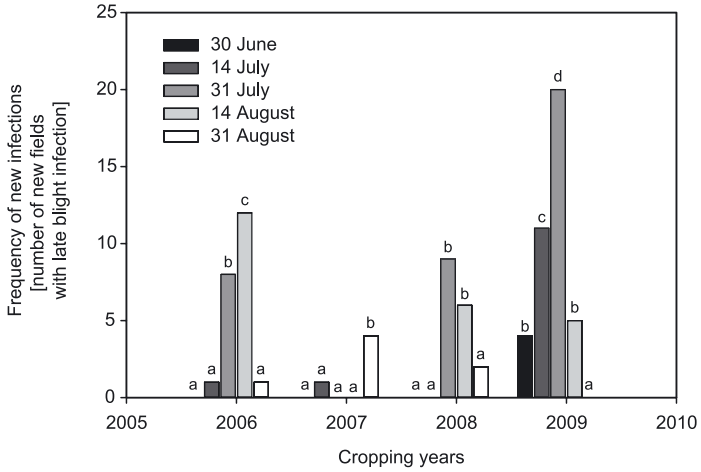

Fig. 2. *Phytophthora infestans* infection of potato and tomato hosts from 2006 to 2009 cropping years in Maine. Data refers to numbers of new infections recorded on both hosts within each assessment period. The bars with the same letters within each year indicate that the frequency of new infections at different assessment periods are not significantly different ($p > 0.05$)

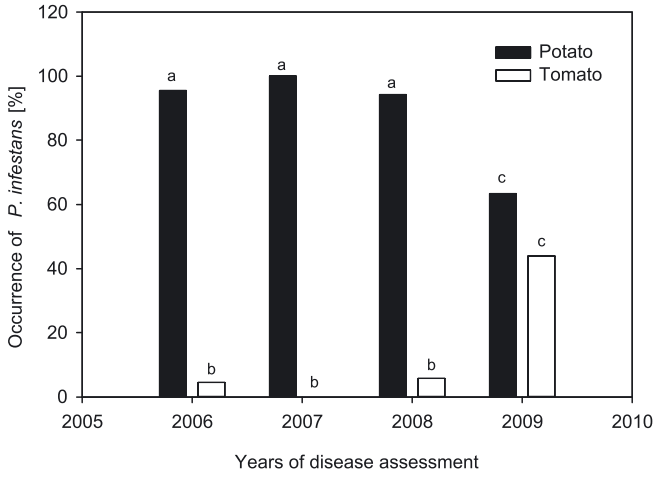

Fig. 3. Occurrences of *Phytophthora infestans* on potato and tomato from 2006 to 2009 cropping years. Data refer to the percentage of hosts infected with late blight during each year. Bars with different letters indicate significant differences ($p < 0.05$) in late blight occurrence between the two hosts within each year

Sensitivity of isolates of *P. infestans* to various fungicides

The inhibition of *in vitro* growth of *P. infestans* generally varied among isolates and ranged from 10–50% at day 4 to 62–80% at 21 days after incubation (Fig. 4). At twenty-one days of incubation, the greatest inhibition was recorded on isolate 99-1 (86/100, US-1) and the lowest inhibition was detected on 99-33 (100/111/122, US-8). When comparisons were made among mancozeb, cymoxanil, and chlorothalonil, at 100 ppm, the lowest inhibition of growth was detected on cymoxanil (Fig. 4).

The average germination of zoospores varied among pathogen isolates and ranged from 40–80% on the unamended medium (Fig. 5). On medium amended with mancozeb, at 1 and 2 ppm, complete inhibition of zoospore germination was detected and there was no significant difference in the inhibitory effect of fungicide concentrations on zoospore germination. Germination of sporangia in medium amended with fungicide such as mancozeb differed slightly among the tested pathogen isolates. Low germination of sporangia was detected across *P. infestans* isolates and ranged from 20–30% at 18°C (Fig. 5).

Discussion

The occurrences of late blight and *P. infestans* were recorded from the 2006 to 2010 cropping seasons in Maine. Late blight has been previously reported in potato-growing areas in Maine throughout the years (Groves 2002; Johnson 2005; Olanya *et al.* 2005) and in other potato growing regions with favorable environmental conditions for disease development (Fry and Goodwin 1997). Differences in the number of disease occurrences in various geographical locations from 2006 to 2009 suggest that variation of *P. infestans* inocula among locations and years are important contributing factors. Variations in pathogen spread and the effectiveness of disease-control measures may also have contributed to the differences observed in late blight occurrences among locations and years. The lowest number of late blight infections was observed in 2007,

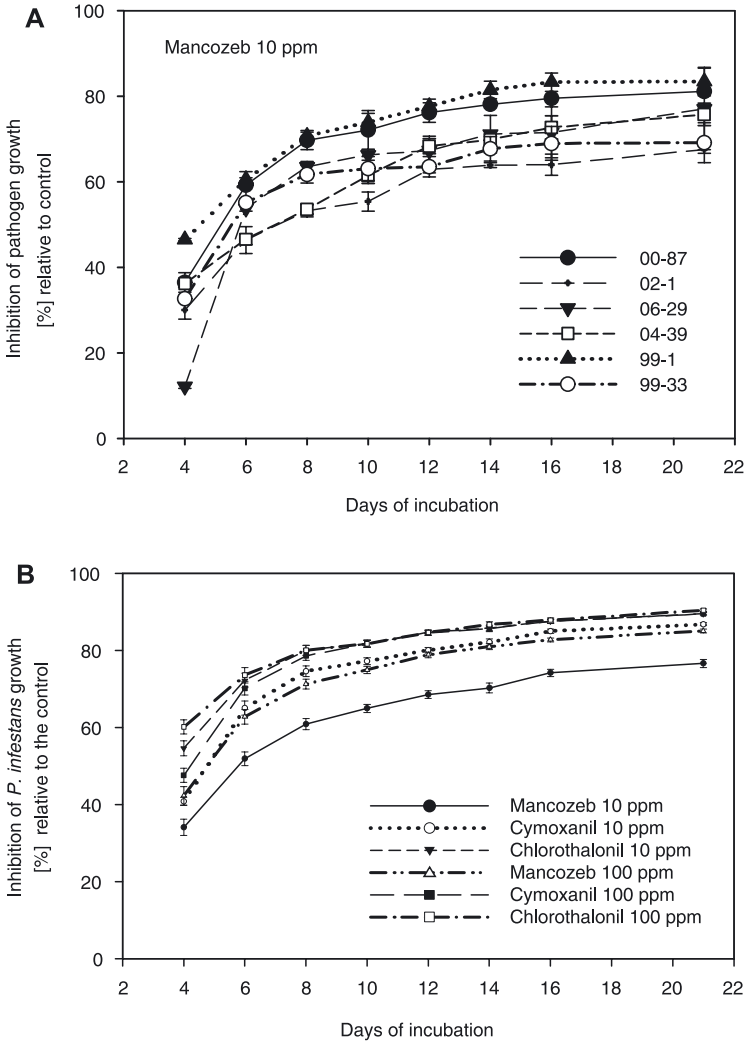

Fig. 4. Growth of *Phytophthora infestans* isolates on Rye B media (18°C) amended with mancozeb at 10 ppm. Isolates 00-87, 02-1, and 99-1 (US-1) represent genotype designations 100/100, 100/122, and 86/100, respectively. Isolates 06-29 and 04-39 are 100/111/122 (US-8) based on allozyme analysis. The reference isolates are 99-1 (86/100, US-1) and 99-33 (100/111/122, US-8). The inhibition of growth of 06-29 on Rye B medium amended with mancozeb, cymoxanil, and chlorothalonil fungicides at 10 and 100 ppm were also determined

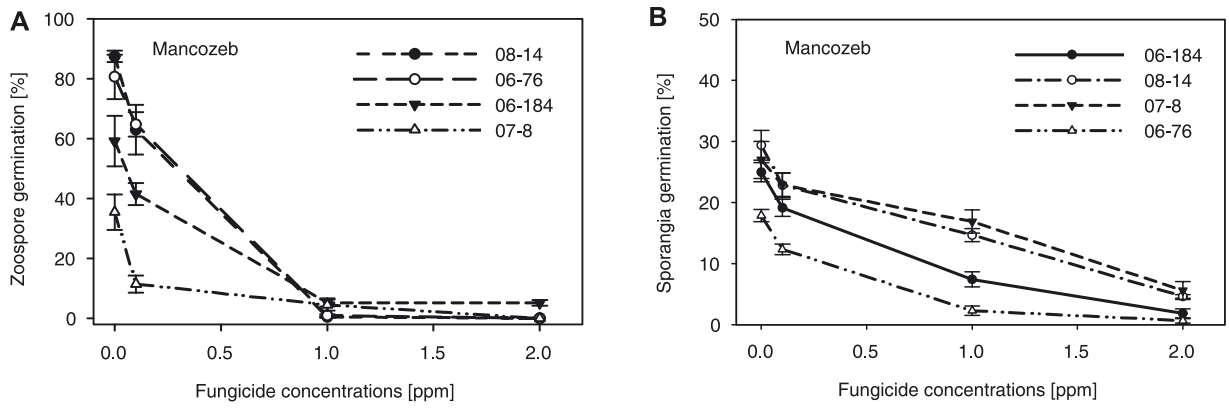

Fig. 5. Effects of fungicide (mancozeb) application on the germination of sporangia and zoospores of *Phytophthora infestans* isolates. The germinations of zoospores and sporangia were assessed on water agar amended with mancozeb at 0 (the control), 0.1, 1, and 2 ppm. The isolates and their genotype designations were: 06-76 (111/122), 06-184 (100/122), 07-8, and 08-14 (100/111/122)

suggesting that the presence of low levels of inocula, less suitable environmental conditions, effective disease control, or a combination of these factors were active. The greatest number of late blight occurrences observed in 2009, implies that there were greater inocula of *P. infestans* in the various growing regions, the presence of suitable environmental conditions for late blight development, and the possibility that the pathogen spread to various geographical regions, or a combination of these factors.

Variation in the spatial distances of late blight occurrences at diverse locations and years, suggests that *P. infestans* can spread across different spatial scales (Fry *et al*. 1993; Ristaino and Gumpertz 2000). In 2006, 2007, and 2008, the spatial distances among locations where late blight occurred were limited to 80 km. In 2009, spatial distances based on nearest neighbor distance analysis were about 40 km, although disease occurrences were widespread throughout the state of Maine and the entire northeastern United States. This implies that sources of inocula or mechanisms of disease-spread were more diverse during the 2009 cropping season compared to the other cropping years. In 2007 and 2008, late blight occurrences or spread may have been constrained by limited inocula sources, effective disease control, or by a less than optimum environment. In 2009, the unprecedented influx of inoculum and spread of disease on late blight-infected tomato seedling transplants sold at retail stores and garden centers throughout the northeast, drastically affected late blight patterns. This widespread inoculum source combined with very favorable environmental conditions for late blight development resulted in a very early disease development, widespread occurrence, and repeated outbreaks on both tomato and potato throughout the state.

Late blight was detected in June 2009. But by the time the extent of infection and the widespread distribution of the diseased transplants was determined, the disease had already been spread throughout the northeastern US (Fry *et al*. 2013). Effects of the 2009 late blight pandemic were most strongly felt by home gardeners and organic growers. Many people with backyard tomato plants, and home and organic potato fields found their plants destroyed as they were not prepared and able to deal with the consequences of this devastating disease (Fry *et al*. 2013). Thus, most of the isolates of *P. infestans* recovered from tomatoes and potatoes from central and southern parts of the state, were the same genotype and likely derived directly from the infected tomato transplants. However, this influx of inoculum, in general, had less impact on the late blight observed in commercial potato fields in northern Maine, which tended to be the same type (100/111/122, US-8) that has been predominantly observed in recent years in the state. Damage was also much less severe in commercial potato fields. This was due to the routine spraying of fungicides to control late blight and the ability to quickly respond to initial reports of disease outbreaks. Thus, the higher frequencies and occurrences of late blight in northern Maine in 2009, were probably more related to the conducive environmental conditions than the inoculum influx from tomato seedlings. However, the tomato strain of the pathogen was also observed on potato in northern Maine, and this strain also affected late blight in the north. Likewise, the potato isolate (US-8) was also observed in other parts of the state as well, although to a much lesser extent than the tomato isolate. These findings indicate that both pathogen genotypes were distributed statewide and affected the late blight epidemic. Experimental evidence for aerial dispersal of *P. infestans* on potato has shown that dispersal from field to field occurs within relatively limited distances (Ristaino and Gumpertz 2000). However, long distance spread has previously been attributed to human activity involving movement or transportation of diseased plant materials such as infected seed and transplants (Campbell 1999; Ristaino and Gumpertz 2000). In this case, the presence and distribution of diseased tomato seedlings across the state of Maine was a notable consequence of human activity and widespread infection from diverse inocula sources.

Infection of tomato by *P. infestans* has been previously documented in Maine and many other potato growing regions of the US and Canada (Vartanian and Endo 1985; Platt 1999; Olanya *et al.* 2009a). Overwintering and survival of *P. infestans* on tomato has also been documented in a California field (Vartanian and Endo 1985). Infection rates and sporangia production on diseased tomato plants by *P. infestans* were shown to be similar to that on potato under controlled environmental conditions (Olanya *et al.* 2009a). This suggests that diverse genotypes of *P. infestans* can infect potato or tomato. Tomato cultivation and production has been increasing in the state of Maine recently, due to home gardens and greenhouse/glasshouse operations. Because late blight can be disseminated via diseased tomato plants (seedling transplants), fruits, and seeds, precautions should be exercised in the production of tomato, transportation of seedlings as well as the management of late blight at local, state, and regional levels. Following the late blight epidemic in 2009, late blight in Maine in 2010 was limited, with only a few reported outbreaks which were very localised in nature. This indicates that the large influx of inoculum on tomato seedlings, and widespread occurrence of late blight in 2009, did not have large-scale lasting effects on late blight inoculum or the frequency and distribution of late blight in the following year. This may be due primarily to unfavorable environmental conditions in 2010, but also indicates that, for the most part, inoculum from 2009 did not survive through the winter to cause widespread disease in 2010.

With regard to fungicide sensitivity, differences in the reduction of the *in vitro* growth of pathogen isolates on medium amended with cymoxanil, chlorothalonil, and mancozeb fungicides, suggest that isolates differ in their sensitivities to fungicides. The result may be different levels of efficacy. Similarly, germination of zoospores and sporangia also varied among fungicides. Although protectant (chlorothalonil and mancozeb) and systemic (cymoxanil) fungicides may differ in their mode of activity, their efficacy on pathogen isolates based on *in vitro* experiments were similar. The low germination of zoospores and sporangia exposed to mancozeb, suggests that low concentrations of fungicidal compounds are needed for effective inhibition of the germination of zoospores and

sporangia. Various authors have previously documented the inhibition of the germination of sporangia and zoospores in response to fungicide compounds. Although US-22 (100/122 designation) was the dominant lineage in 2009 in the United States, in Maine this lineage and US-8 (100/111/122) were both recorded. The sensitivity of US-22 lineage to mefenoxam suggests that inoculum derived from this genotype can be easily destroyed by the fungicide. However, the predominate fungicides applied in potato fields in Maine consist of mancozeb and chlorothalonil (protectant), alternated with cymoxanil and occasionally mefenoxam (systemic) as well as some others. Therefore, it is possible that residual inoculum for lineage US-22 (100/122) could have persisted in subsequent years.

We conclude that occurrence and distribution of *P. infestans* on potato and tomato were impacted by various sources of inocula, dispersal mechanisms and patterns, and their interaction with crop and disease management practices or environmental factors. It appears that a combination of pathogen dissemination mechanisms and the dispersal of sporangia may have occurred simultaneously to account for disease spread. Due to favorable temperature, relative humidity and rainfall conditions during the potato cropping seasons in Maine, late blight spread readily occurred. Elimination or minimising sources of inocula, followed by prompt control measures would reduce the likelihood of late blight infections. Similarly, limiting the movement of potential Solanaceous hosts as well as tomato seedlings infected by late blight within a growing season, and following prudent cultural and chemical control strategies should also reduce the likelihood of infection or disease potential on potato.

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