

History and consequences of migrations, changes in epidemiology and population structure of potato late blight, *Phytophthora infestans*, in Finland from 1845 to 2011

Doctoral Dissertation

Asko O. Hannukkala



**History and consequences
of migrations, changes in
epidemiology and population
structure of potato late blight,
Phytophthora infestans, in Finland
from 1845 to 2011**

Doctoral thesis in plant pathology

Asko O. Hannukkala

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History and consequences of migrations, changes in epidemiology and population structure of potato late blight, *Phytophthora infestans*, in Finland from 1845 to 2011

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Potato late blight, caused by *Phytophthora infestans* (Mont.) de Bary, is one of the most destructive diseases of potato globally, including Finland. The history of late blight occurrence in Finland from 1845 to the 1980s is described based on newspaper articles (from 1845 to 1900) and scientific reports (from 1910 to 1982). The occurrence and severity of potato late blight from 1982 to 2011 is based on monitoring untreated plots of cv. “Bintje” in annual variety trials and untreated plots in fungicide efficacy trials at 6 to 9 Research Stations of MTT Agrifood Research Finland, and similar experiments at the Potato Research Institute. The effect of climatic factors on first late blight outbreaks was modelled for the period 1993–2002. From 1999 to 2002 aspects of suspected soil-borne epidemics were studied in detail. In total 4927 *P. infestans* isolates were collected from 1990 to 2010. Mating type (2703 isolates), R-gene virulence race (1100 isolates) and response to fungicides metalaxyl (3912 isolates) and propamocarb-HCl (2541 isolates) were determined. Haplotype based on mitochondrial DNA was determined for 154 isolates collected from 1992 to 2000. Development in fungicide use from 1953 to 2010 is described based on statistics for fungicide sales and the area devoted to potato growing in Finland.

Leaf and tuber symptoms on potato most probably caused by the late blight pathogen were first described in eastern Finland in 1845. In 1847 and 1848 the disease was

widespread, occurring in various parts of the country. From 1849 to the 1980s one to five severe late blight epidemics were reported per decade. During this period late blight usually appeared in the fields during the latter part of August or early September. At the end of the 1990s there was a rapid shift towards early outbreaks of late blight and since then the first late blight outbreaks have been reported at the end of June or during the first week of July. The shift towards early epidemics has led to significant increase in fungicide applications in the 1990s and 2000s. The mating type A2 was first found in 1992 in Finland and its proportion in the population, until the end of the 1990s, was close to 20%. From the end of the 1990s the A1:A2 mating type ratio in the Finnish *P. infestans* population has been close to 50:50. The change in mating type ratio coincides with the shift towards early outbreaks of late blight, suggesting an increasing role of oospores as a source of primary inoculum in Finland. During the monitoring period from 1992 onwards, only representatives of the new *P. infestans* population have been detected among the isolates studied. The Finnish *P. infestans* population is very diverse based on the spectrum of R-gene virulence races and genetic marker studies. No clonal lineages can be detected in the population. Metalaxyl-resistant isolates dominated the population in the early 1990s, but after adapting anti-resistance strategies and introduction of numerous new fungicides, metalaxyl resistance has almost

disappeared. There has not been any sign of resistance to propamocarb-HCl in the Finnish *P. infestans* population.

Currently potato late blight is under good control in conventional potato production as long as effective fungicides are available. There is increasing public demand for decreasing use of pesticides in agriculture and therefore in future more effort should be put on developing potato cultivars with durable resistance against late blight. Also cultural practices, including crop rotations, should be developed to re-

duce sources of primary inoculum in future potato production. Regular monitoring should be implemented at least every few years due to the potential for rapid changes in potato late blight epidemiology and population properties.

Key words:

Potato, Solanum tuberosum, potato late blight, Phytophthora infestans, epidemiology, population diversity, mating type, virulence race, mitochondrial haplotype, pesticides, fungicide resistance

Perunaruton (*Phytophthora infestans*) leviämisen, epidemiologisten ja patogeenipopulaatiossa tapahtuneiden muutosten historia ja seuraukset Suomessa vuodesta 1845 nykypäivään

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P*hytophthora infestans*- (Mont.) de Bary munasiemen aiheuttama perunarutto on maailmanlaajuisesti, kuten myös Suomessa, yksi perunan tuhoisimmista taudeista. Taudin levinneisyyttä ja ankaruutta Suomessa kartoitettiin vuosina 1845–1900 julkaistuista suomen- ja ruotsinkielisistä sanomalehtiartikkeleista, joita Suomen Kansalliskirjaston digitoimasta artikkelikokoelmasta löytyi yli 500. Vastaavia tietoja vuosilta 1910–1982 koottiin erilaisista tutkimusraporteista. Vuosina 1983–2011 perunaruton alkamista ja epidemioiden etenemistä tutkittiin 6–9 MTT:n tutkimusaseman ja Perunantutkimuslaitoksen perunan lajikekokeista ja perunaruton torjuntakokeista tehtyjen ruttohavaintojen perusteella. Miltei kaikki ruttohavainnot tehtiin taudinarasta Bintje-lajikkeesta, jota ei ollut käsitelty rutton-torjunta-aineilla. Vuosina 1991–2011 perunaruton esiintymistä tarkkailtiin lisäksi erilaisilla perunapalstoilla ja ammattiviljelmillä. Säätekijöiden vaikutusta perunaruton alkamisajankohtaan mallinnettiin vuosina 1993–2002. Vuosina 1999–2002 selvitettiin tarkasti munaitiöiden aiheuttaman maatartunnan merkitystä tautiepidemian alkulähteenä. Vuosina 1990–2010 perunaruttoon sairastuneista kasveista kerättiin 4927 *P. infestans*-kanta tarkempiin tutkimuksiin. Näistä 2703 kannasta määritettiin pariumistyyppi, 1100 kannasta virulenssivotu. Kestävyys metalaksyyliä vastaan tutkittiin 3912 ruttokannasta ja propamokarbi-hydrokloridia vastaan 2541 kannasta. Lisäksi vuosina 1992–2000 ke-

rätyistä perunaruttokannoista tutkittiin 154 kannan mitokondrion DNA:n haplotyyppi, joka kertoo ruttokantojen perinnöllisestä taustasta. Perunaruton torjuntaan rekisteröityjen kasvinsuojeluaineiden käyttömäärien ja vaikutustavaltaan erilaisten tehoaineiden suosion muutoksia vuosina 1953–2010 selvitettiin torjunta-aineiden myyntitilastojen ja perunan viljelypinta-alojen muutosten perusteella.

Sanomalehdissä julkaistujen tarkkojen perunaruton oirekuvausten perusteella näyttää ilmeiseltä, että tautia esiintyi Suomessa Viipurin ympäristössä muutamilla viljelyksillä jo vuonna 1845–vain vuotta myöhemmin kuin se oli löydetty ensimmäisen kerran Euroopassa Belgiasta. Vuosina 1847 ja 1848 perunaruton kerrottiin aiheuttaneen suurta tuhoa perunapelloilla ja etenkin mukularuttona varastoissa Turun ja Viipurin seuduilta pohjoiseen Tornionjokilaaksoa myöten. 1800-luvun puolivälistä 1980-luvulle maanlaajuisia perunaruttoepidemioita on ollut enintään muutamana vuotena vuosikymmentä kohden. Tänä ajanjaksona tauti ilmaantui perunapelloille yleensä elokuun puolivälin jälkeen ja satotappiot johtuivat pääsääntöisesti mukuloiden pilaantumisesta kellareissa. 1900-luvulle asti Suomen maaseudulla asuva valtaväestö ei ollut erityisen riippuvainen perunasta, sitä kasvatettiin ensisijaisesti karjan rehuksi. Täten perunarutto ei Suomessa koskaan aiheuttanut maanlaajuisia nälänhätää. Ruttovuosista joutuivat eniten kärsimään köyhät tilattomat ja

orastavien teollisuuspaikkakuntien työväki, joiden pääasiallinen energianlähde oli palstaviljelmillä kasvatettu peruna.

1900-luvulla on julkaistu muutama tutkimusraportti perunaruton esiintymisestä eri vuosikymmenillä ja niiden perusteella ruton kehitys on ollut varsin samanlaista aina 1990-luvulle asti—ensimmäiset ruttovahainnot yleensä elokuun jälkipuoliskolla ja enintään muutama paha ruttovuosi vuosikymmenessä. 1950-luvulta alkaen kemiallisia rutontorjunta-aineita on aktiivisesti tarjottu viljelijöille, mutta kemiallinen torjunta alkoi yleistyä vasta 1970-luvulla. Ruton hallitsemiseksi tuolloin tarvittiin enintään 2–3 käsittelyä elo-syyskuussa.

1990-luvun lopulla perunaruton epidemiologia muuttui radikaalisti ja tauti ilmaantui pelloille jo kesä-heinäkuun taitteessa. 2000-luvulla perunarutto on ilmaantunut kasvustoihin useimmiten heinäkuun ensimmäisellä viikolla. Ruttoepidemioiden aikaistuminen ja viljelyn ammatti- maistuminen on nelinkertaistanut rutontorjuntaruiskutusten määrän 1980-lukuun verrattuna. Perunaruton A2 pariutumismuoto löydettiin Suomesta 1992 ja sen osuus oli 1990-luvulla noin 20% tutkituista perunaruttokannoista. Aivan 1990-luvun lopussa A1- ja A2-muotojen osuus alkoi lähestyä 50:50 suhdetta, mikä on luonteenomaista suvullisesti lisääntyville populaatioille. Tämä muutos tapahtui kutakuinkin samaan aikaan ruttoepidemioiden alun aikaistumisen kanssa. On mahdollista, että maassa säilyvien munaitiöiden merkitys taudin alkulähteenä lisääntyi tuolloin merkittävästi. Vuodesta 1992 lähtien ruton alkamisajankohta ja A1/A2 pariutumismuotojen määrällinen suhde näyttäivät vakiintuneen. Kaikki tässä tutkimuksessa määritetyt *P. infestans*-kannat ovat kuuluneet uuteen Meksikosta Eurooppaan 1980-luvulla levinneeseen populaatioon. R-geeni virulenssirotujen ja

erilaisten geneettisten markkereiden perusteella Suomessa ja muissa Pohjoismaissa esiintyvä perunarutto on monimuotoisempi kuin rutto muualla Euroopassa. Muualla ruttopopulaatiot koostuvat muutamasta vallitsevasta kloonista, kun Pohjoismaissa miltei kaikki kannat ovat perimältään erilaisia yksilöitä. 1990-luvun alussa pääosa tutkituista *P. infestans*-kannoista oli valikoitunut kestäviksi metalak-syyli-torjunta-ainetta vastaan. Resistenssiä ehkäisevien toimenpiteiden ja uusien vaikutustavaltaan erilaisten torjuntavalmisteiden ansiosta metalaksyyliä kestävät kannat ovat miltei hävinneet. Perunaruton kestävydestä muita Suomessa käytettyjä rutontorjunta-aineita vastaan ei ole näkynyt merkkejä.

Tällä hetkellä perunarutto on hallinnassa tavanomaisessa viljelyssä, mutta se edellyttää runsasta kasvinsuojeluaineiden käyttöä. Uusi EU:n direktiivi (128/2009 EC) ja laki kasvinsuojeluaineista (1563/2011) velvoittavat siirtymään integroituu kasvin-suojeluun ja vähentämään kasvinsuojelu-aineiden käyttöä. Lähitulevaisuudessa on kehitettävä ja otettava viljelyyn nykyistä rutonkestävämpiä perunalajikkeita. Myös perunan viljelykäytäntöjä, kuten kasvin-vuorotusta, on kehitettävä niin että perunaruton alkutartuntalähteet saadaan nykyistä paremmin eliminoitua. Myös perunaruton esiintymisen ja taudinaiheuttajapopulaation muutosten tarkkailuun tulisi taata voimavaroja. Muutoin perunaruton mahdollisiin tuleviin muutoksiin ei pystytä ajoissa varautumaan.

Avainsanat:

peruna, Solanum tuberosum, perunarutto, Phytophthora infestans, epidemiologia, population monimuotoisuus, pariutumistyyppi, virulenssirotu, haplotyyppi, kasvinsuojeluaineet, torjunta-aineresistenssi.

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The publications are referred to in the text by their Roman numerals. The published articles are reprinted with the kind permission from the publishers, John Wiley and Sons, Inc. and The Scientific Agricultural Society of Finland.

ABBREVIATIONS

AFLP	amplified fragment length polymorphism
a.i.	active ingredient
AIR	apparent infection rate
ATP	adenosine triphosphate
CAA	carboxylic acid amide
C_i	virulences per isolate
C_p	virulences per pathotype
cv.	cultivar
DAP	days after planting
DNA	deoxyribonucleic acid
DPC	disease progress curve
DSS	decision support system
ELISA	enzyme-linked immunosorbent assay
EVIRA	Finnish Food Safety Authority
GmbH	Gesellschaft mit beschränkter Haftung
Gpi	glucose-6-phosphate isomerase
HCl	hydrochloride
H_s	Shannon diversity index
H_{SR}	normalized Shannon diversity index
IPM	integrated pest management
MtDNA	mitochondrial deoxyribonucleic acid
MTT	Agrifood Research Finland
NAP	national action plan
Pep	peptidase
PCR	polymerase chain reaction
rAUDPC	relative area under disease progress curve
RFLP	restriction fragment length polymorphism
RNA	ribonucleic acid
SASA	Scottish Agricultural Science Agency
SSR	simple sequence repeat
TUKES	Finnish Safety and Chemicals Agency
UV	ultraviolet
QiI	quinone inside inhibitor
QoI	quinone outside inhibitor
qPCR	quantitative polymerase chain reaction

Contents

1	Introduction	11
1.1	Potato late blight and <i>Phytophthora infestans</i> (Mont.) de Bary	11
1.1.1	Potato late blight disease.....	11
1.1.2	The causal agent of potato late blight.....	11
1.2	Phenotypic and genetic variation in <i>P. infestans</i> populations	14
1.3	Markers used for characterising the diversity of <i>P. infestans</i> populations.....	15
1.3.1	Mating type.....	15
1.3.2	Virulence race (pathotype).....	15
1.3.3	Genetic markers	16
1.4	Ecology and epidemiology of <i>P. infestans</i> in potato	17
1.4.1	Primary sources of inoculum	17
1.4.2	Primary infection and colonisation	19
1.4.3	Secondary spread infection and colonisation of aerial potato tissues	19
1.4.4	Survival in tubers.....	20
1.4.5	Survival as oospores	20
1.5	Source of origin of <i>P. infestans</i> and its migrations to Europe	21
1.5.1	First migration of potato late blight to Europe.....	21
1.5.2	Second migration of potato late blight to Europe and elsewhere	23
1.6	Disease management by cultivar resistance.....	23
1.6.1	Progress in breeding late blight resistant cultivars.....	23
1.6.2	Potato breeding, cultivars and late blight resistance in Finland.....	25
1.7	Disease management by fungicides	26
1.7.1	General development of fungicides against late blight.....	26
1.7.2	Biochemical modes of action of the most widely used late blight fungicides.....	29
1.8	Integrated approach to late blight management	31
1.9	Objectives of the thesis	32
2	Materials and methods	34
2.1	Historical and recent development of blight epidemics in Finland	34
2.1.1	Review of historical literature and surveys on late blight incidence from 1845 to 1980	34
2.1.2	Systematic monitoring of late blight outbreaks from 1983 to 2011	34
2.1.3	Modelling weather as a possible cause of early late blight outbreaks	36
2.1.4	Modelling the progress of late blight epidemics	36
2.1.5	Monitoring suspected soil derived epidemics	36
2.2	Collection and characterisation of <i>P. infestans</i> isolates	37
2.2.1	Collection and selection of isolates	37
2.2.2	Mating type determination.....	38
2.2.3	Determination of virulence races	38
2.2.4	Assessment of response to systemic fungicides	39
2.2.5	Mitochondrial DNA haplotype determination	39
2.3	Sales and estimated usage of different late blight fungicides	39
2.3.1	Sales of active ingredients	39
2.3.2	Estimated potato area treated annually	40
2.3.3	Estimated number of annual applications per potato hectare	41

3	Results and discussion	42
3.1	Historical and recent development of late blight epidemics in Finland.....	42
3.1.1	Potato late blight incidence and severity from 1845 to 1900	42
3.1.2	Knowledge on the cause of potato late blight in Finland prior to 1900	45
3.1.3	Potato late blight from 1901 to the 1980s	46
3.1.4	Monitoring the late blight outbreaks from 1983 to 2011.....	46
3.1.5	Modelling weather as a possible cause for the shift to early outbreaks.....	48
3.1.6	Modelling progress of late blight epidemics	49
3.2	Sexual reproduction and diversity in Finnish <i>P. infestans</i> population.....	52
3.2.1	Mating type ratio and oospore production.....	52
3.2.2	Diversity and complexity of R-gene virulence races	55
3.2.3	Genetic diversity	58
3.3	Consequences of sexual reproduction and earlier starting epidemics to fungicide use.....	59
3.3.1	General development of fungicide sales and use.....	59
3.3.2	Multi-site protectant fungicides.....	60
3.3.3	RNA-synthesis inhibitors and metalaxyl resistance	60
3.3.4	Fungicides disturbing cell membrane assembly.....	63
3.3.5	Fungicides inhibiting mitochondrial respiration	64
3.3.6	Protecting expanding leaves and apical growth after fungicide application	64
4	Conclusions and future prospects	66
5	Acknowledgements	67
6	References	69

1 Introduction

1.1 Potato late blight and *Phytophthora infestans* (Mont.) de Bary

1.1.1 Potato late blight disease

Potato late blight, caused by *Phytophthora infestans* (Montagne) de Bary, is one of the most destructive plant diseases to affect mankind since potato became an essential part of the human diet worldwide. The pathogen has vast potential for reproduction and genetic flexibility and has provided unpleasant surprises, including the Irish and Continental European potato famines, since it first migrated to Europe via the USA from Central or South America in the 1840s (Bourke 1964, Turner 2005, Zadoks 2008). Even today the human costs resulting from the disease can be huge, and during epidemic years late blight can still drive modern farmers out of business, especially in developing countries (Fry and Goodwin 1997). The direct annual monetary costs of control and lost production are estimated at €1,000,000,000 worldwide (Haverkort et al. 2008). Globally these costs are likely to increase as potato production areas are rapidly increasing in developing countries like China, India and the highlands of several African countries (Reviewed e.g. by Fry 2008, Vleeshouwers et al. 2011).

In contrast to several adverse effects on society, the first migration of the pathogen in the 1840s also was a driving force in the development of mycology, plant pathology and resistance breeding as discrete sciences, as well as in development of chemical and cultural practices for plant disease control. The second migration in the late 1970s or early 1980s resulted in rapid development of molecular tools to investigate this “scientifically fascinating and ever challenging pathogen”, as stated by Birch

and Whisson (2001). It remains to be seen whether the recent appearance and rapid invasion of aggressive genotypes “13_A2” and “6_A1” in Great Britain indicates the next globally significant change in the *P. infestans* population and its epidemiology (Nyongesa et al. 2010, Cooke et al. 2011).

Potato late blight symptoms are evident in nearly all parts of the potato plant: leaflets, stems, stolons and tubers (de Bary 1853, de Bary 1876, Smart and Fry 2001). The infection typically appears first as small irregular necrotic spots (Figure 1a), which rapidly turn to lesions with a dark brown necrotic centre surrounded by collapsed pale green or chlorotic tissue in leaflets (Figure 1b, 1c). In dry conditions the first symptoms can be necrotic lesions on stems (Figure 1d) (Birch and Whisson 2001, Fry 2008). Gradually, almost all of the shoot tissue becomes degraded (Figure 2a) and only hollow dead stems remain in the field (Figure 2b) (Smart and Fry 2001). Also tubers may become infected and rot (Figure 2c) before and during storage (Nyankanga et al. 2007). The destruction is completed by secondary fungi and bacteria that easily invade the damaged tuber tissue (Smart and Fry 2001).

1.1.2 The causal agent of potato late blight

In Europe symptoms of potato late blight were probably first noticed in Belgium in 1844 and in 1845 severe late blight outbreaks were reported in Ireland and several regions of Continental Europe, as described in numerous review articles. It took, however, over 40 years before the science fully agreed about the causal agent of potato late blight disease (Bourke 1964, Dowley et al. 1995, Turner 2005, Fry 2008, Zadoks 2008).



Figure 1. Typical symptoms caused by *P. infestans* on potato during the early phase of an epidemic. 1 a. Irregular necrotic spots, 1b and 1c. Advanced chlorotic and necrotic lesions, 1d. Necrotic stem lesions caused by *P. infestans* on potato. (Photos Asko Hannukkala).



Figure 2. Severe damage caused by *P. infestans* at the advanced stage of an epidemic. 2a. Seriously damaged shoots. 2b. Fully destroyed plants. 2c. Brown dry rot on tubers (Photos Asko Hannukkala).

Already in autumn 1845 David Moore in Ireland clearly pointed out that mould constantly accompanied the appearance of decay in dying potato plants. Originally he considered the mould to be an accompaniment to some mysterious disease that had weakened the plants. Correspondence in 1845–1846 with M. J. Berkeley gradually convinced Moore about the “fungal theory” and by 1847 Berkeley and Moore suggested the mould to be the cause of the decay of potato (Berkeley 1846, Nelson 1995). J.F.C Montagne in 1845 named the mould *Botrytis infestans* (Turner 2005, Zadoks 2008). During the following decades

there was serious disagreement among scientists, some of whom claimed the mould to be the causal agent of potato “blight” or “murrain” and others who believed that the mould appeared after the potato crop was injured for various reasons, including cosmic radiation and the sins of mankind (Peterson 1995, Turner 2005).

De Bary (1853), in his first book on plant diseases, strongly supported the “fungal theory”. In classic papers from 1861 and 1863, de Bary described microscopically penetration, subsequent invasion of intercellular tissues and further sporangiophore

and sporangia production in healthy potato leaves artificially inoculated with the spores of the pathogen, then called *Peronospora infestans* (Turner 2005). Finally de Bary (1876) published his extensive laboratory, greenhouse and field studies on the biology and morphology of the potato late blight pathogen and on the basis of particular morphology of the pathogen's sporangiophores created a new genus within the Family *Peronosporaceae*: *Phytophthora infestans*. In spite of de Bary's convincing evidence, scientific debate on the cause of potato blight continued until the end of the 1800s before most scientists accepted *P. infestans* to be the causal agent of potato late blight disease (Peterson 1995, Turner 2005).

P. infestans has been taxonomically classified as a representative of the Phylum *Oomycota* and Class *Oomycetes*. Historically, taxonomists have grouped *Oomycota* in the Kingdom Fungi, mainly due to their filamentous growth (Brasier and Hansen 1992). However, many features distinguish *Oomycetes* from true fungi: cell walls are composed of cellulose compounds and glucans instead of chitin, the vegetative state is diploid, zoospores have two morphologically dissimilar flagella, one with mastigonemes, and finally antheridia and oogonia are produced as gametangia (Money 1998). Molecular genetics studies have revealed that *Oomycetes* form a monophyletic group in the Kingdom, termed *Stramenopila*, clustering together with others in the *Chromalveolata* super-group. True fungi belong to a different super-group, the *Ophisthokonta* (Gunderson et al. 1987, Adl et al. 2005). *Oomycetes* are closely associated with diatoms and brown algae (Gunderson et al. 1987, Birch and Whisson 2001, Fry 2008).

Current phylogenetic classification of *P. infestans* according to Birch and Whisson (2001):

- Kingdom: *Stramenopila* (*Chromista*)
- Phylum: *Oomycota*
- Class: *Oomycetes*
- Order: *Peronosporales*
- Family: *Peronosporaceae*
- Genus: *Phytophthora* of which *P. infestans* is the type species

The mycelium of *P. infestans* is multinucleate and aseptate. Asexual sporangia are formed on sporangiophores that grow from infected tissue or growth media (Figure 3a, 3b). In moist conditions motile wall-less, biflagellate zoospores are released from the sporangia. After a short (often less than 60 min) motile phase, zoospores are encysted on the leaf surface. Encysted zoospores, sometimes called cystospores, germinate directly via a germ tube and penetrate leaf or stem tissue (Marks 1965, Erwin and Ribeiro 1996).

P. infestans is a heterothallic, near obligatory, pathogen with two mating types (sexes) named A1 and A2 (Gallegly and Galindo 1958, Fry 2008), though A2 mating type and sexual reproduction was not recorded in nature outside Mexico until the early 1980s (Fry et al. 1993, Goodwin et al. 1994, Ristaino et al. 2001). In the laboratory, the pathogen can be grown on artificial media, but under field conditions it cannot grow outside living plants (Caten and Jinks 1968, Smart and Fry 2001). The pathogen is bisexual and in the presence of both mating types A1 and A2 strains can produce male antheridia and female oogonia (Figure 3c) (Gallegly and Galindo 1958, Fry 2008). Also self-fertile strains capable of sexual reproduction without the corresponding mating type exist. 'Apparent' self-fertility can result from mixtures of A1s and A2s when tested isolates do not originate from a single sporangium (Fyfe and Shaw 1992).

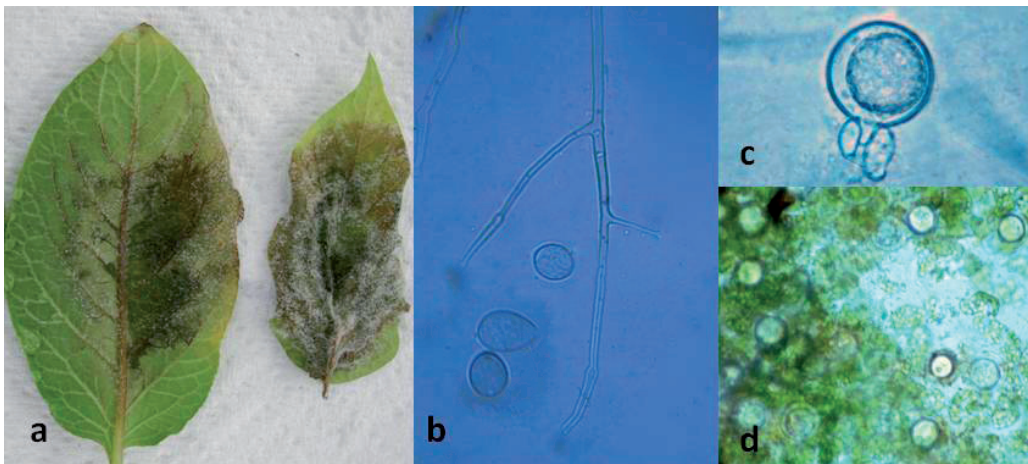


Figure 3. Macroscopic and microscopic structures of *P. infestans*. 3a. Mycelia and sporangiophores sporulating on potato leaflets. 3b. Sporangiophore and sporangia. 3c. Oogonium and antheridium. 3d. Oospores in infested potato leaf tissue. (Photos Asko Hannukkala).

The formation of oogonia and antheridia is stimulated by a volatile hormone described as a diterpene. Thus mycelia of opposite mating types need not be in direct contact with each other to initiate sexual reproduction. Additionally, several external factors, including growth media, mechanical wounding and the presence of other microbes, can stimulate gametangia production (Ko 1988, Judelson 1997, Fry 2008). The antheridia of *P. infestans* are amphigynous (the oogonial stalk is surrounded by the antheridium). Fertilisation results in formation of a thick-walled oospore that serves both as a survival structure and as a source of genetic variation via sexual recombination (Drenth et al. 1995, Turkensteen et al. 2000, Smart and Fry 2001).

1.2 Phenotypic and genetic variation in *P. infestans* populations

P. infestans has the potential to produce large amounts of variation in progeny via both asexual and sexual reproduction mechanisms (Brasier 1992, Brasier and Hansen 1992), which have been relatively poorly understood until recently (Judelson 1997, Birch and Whisson 2001). The entire genome of *P. infestans* has recently been sequenced (Haas

et al. 2009), providing valuable tools for further genetic research of the pathogen and its interactions with the potato and the environment (Fry 2008).

From the plant pathology, and especially the epidemiology, point of view, genetic and phenotypic diversity and changes in diversity in population level is more interesting than variation among individual strains as such (Goodwin et al. 1994, Fry 2008, Cooke et al. 2011, Gisi et al. 2011), although deeper understanding of the basic biochemical and genetic processes behind genetic variation can improve development of resistant potato varieties and novel modes of action and target sites for late blight fungicides (Judelson 1997, Birch and Whisson 2001, Gisi et al. 2011).

Interest in population studies increased during the 1980s and 1990s when *P. infestans* populations outside Mexico started to show various signs of changes (Fry et al. 1993, Goodwin et al. 1994, Flier et al. 2003a) and new phenotypic and genetic markers were developed to study and compare different *P. infestans* populations (Tooley et al. 1985, Goodwin et al. 1992). The concept of clonal lineage established for other plant pathogens at the beginning of the 1990s (as reviewed

by Smart and Fry 2001) became an important means to understand the population biology and epidemiology of *P. infestans*. The identification of a clonal lineage requires one or more genetic markers. One clonal lineage includes all strains of the pathogen possessing a single genotype based on these markers (Anderson and Kohn 1995). All members of a clonal lineage are descendants of a single individual, but variation within a lineage can be caused by mutations or mitotic recombination (Goodwin 1997). Characterisation of clonal lineages in *P. infestans* is based on mating type, allozyme genotypes and DNA-fingerprints. Originally DNA-fingerprints were based on restriction fragment length polymorphisms (RFLPs) (Goodwin et al. 1992, Goodwin 1997), but currently microsatellite markers, also termed simple sequence repeats (SSR), are increasingly used to determine a clonal lineage (Lees et al. 2006, Cooke et al. 2011).

Though *P. infestans* has vast potential to produce genetic and phenotypic variation, until recently in most parts of the world prevailing populations have had rather narrow and uniform genetic backgrounds (Fry et al. 1992). *P. infestans* populations in most potato-producing regions are subjected to genetic bottlenecks: only few random genotypes are able to survive to reproduce during the next season. This is the case in regions where harsh environmental conditions, sanitation practices, lack of wild host species and lack of oospores reduce the survival of the pathogen (Goodwin et al. 1994). Also in sexually reproducing populations, where survival is not normally a severe bottleneck, the majority of the oospore-derived progenies are less fit and only very few fit genotypes gradually become dominant in the population (Mayton et al. 2000, Knapova et al. 2002, Lee et al. 2002)

It is characteristic of *P. infestans* that prior to the 1980s the population outside Mexico was dominated by one clonal lineage, US-1 (Goodwin et al. 1994). Thereafter populations have become more diverse, undergo-

ing rapid seasonal changes. Moreover, in certain regions populations have become almost clonal, or consist of very few dominant clonal lineages (Cooke et al. 2011). Particularly in northern Europe, current *P. infestans* populations consist of numerous unique or almost unique genotypes among which no obvious clonal lineages exist (Brurberg et al. 2011).

1.3 Markers used for characterising the diversity of *P. infestans* populations

1.3.1 Mating type

Mating type (A1 and A2), apart from rare self-fertile strains of *P. infestans* (Fyfe and Shaw 1992), has proven to be a robust and reliable marker used in most population studies. The assay is based on pairing unknown isolates with known A1 and A2 tester strains, following screening of oospore production. An axenic culture required for each isolate restricts the number of samples that it is practically possible to include in the screening process (Cooke and Lees 2004). Therefore, standard molecular assays have been developed and used in recent surveys. In the case of conflicting results, the bioassay is still regarded as more reliable than the molecular one (Gisi et al. 2011).

1.3.2 Virulence race (pathotype)

Since the 1950s vertical resistance genes found in *Solanum demissum* Lindl. and deployed in *Solanum tuberosum* L. have been utilised to determine corresponding genes for virulence/avirulence in *P. infestans* (Black et al. 1953, Toxopeus 1956, Malcomson and Black 1966, Peters et al. 1998). Based on the ability of different *P. infestans* strains to overcome a range of the specific R-genes in potato, they can be classified into numerous physiological races compatible with the same set of R-genes in potato (Malcomson and Black 1966, Peters et al. 1998). The terminology for the 'races' has been inconsis-

ent in the published literature, including e.g. physiological race, compatibility race, pathotype and virulence race. According to a suggestion of Andrivon et al. (2011), the term virulence, as determined by Van der Plank (1968), is used in this thesis. By the end of the 1960s eleven R-genes from *S. demissum*, R₁ to R₁₁, and numerous corresponding combinations of virulence factors in *P. infestans*, had been detected (Malcomson 1969).

Virulence race is usually determined by bioassay, where leaflets of a series of a genetically defined differential set of potato clones, each carrying one of the 11 specific R-genes, is inoculated with sporangia or zoospores of the *P. infestans* isolate under study. After an incubation period, compatible or incompatible reactions are scored and the isolate's race is determined by the numbers of R-genes overcome in the differential set. For example *P. infestans* virulence race 1.3.4.7.10.11 is capable of overcoming R-genes 1, 3, 4, 7, 10 and 11 in the potato differential set (Stewart 1990, Peters et al. 1998, Cooke and Lees 2004).

The numbers and frequencies of different virulence races in certain populations can further be used for calculating several different statistical population diversity and race complexity estimates (Andrivon 1994). These estimates have been utilised in numerous studies comparing temporal and spatial variation in *P. infestans* populations between single fields, between geographical regions within countries, between countries and between larger geographical units (e.g. Malcomson 1969, Shattock et al. 1977, Drenth et al. 1994, Peters et al. 1998, Cooke and Lees 2004, Cooke et al. 2011). It is also important to keep in mind that additional R-genes have been found after establishment of Black's differential set in the 1960s (Trognitz 1998). Recent genome-wide registers of *P. infestans* effectors enable approaches that accelerate cloning and specificity-pro-

fling of R-genes (Haas et al. 2009, Vleeshouwers et al. 2011).

1.3.3 Genetic markers

Isozymes (isoenzymes, allozymes) were widely used to study genetic variation in *P. infestans* populations (Tooley et al. 1985) before DNA-based molecular markers were developed. Isozymes are variants of the same enzyme. Variation in isozyme banding patterns at the glucose-6-phosphate isomerase (Gpi) and peptidase (Pep) loci was applied in *P. infestans* population studies (Cooke and Lees 2004). The method is based on measuring and comparing relative distances of movement of different Gpi and Pep isozymes in electrophoresis in comparison to allele 100 (Tooley et al. 1985, Fry and Goodwin 1997). The old asexual *P. infestans* population includes Gpi types 86/100 or 100/100 and Pep types 92/100, 100/100 and 92/92 (Spielman et al. 1991, Goodwin et al. 1994).

Polymorphisms at various regions of the mitochondrial genome are one widely used tool to study evolutionary biology of different organisms. The mitochondrial genome has a slow mutation rate, is inherited usually from the female parent and has a uniform genetic background (Avila-Adame et al. 2006). Properties of the mitochondrial genome have been applied to study the origin, migration and population diversity of *P. infestans* (Carter et al. 1990, Griffith and Shaw 1998, Gavino and Fry 2002). Four mitochondrial haplotypes, named Ia, Ib, IIa and IIb, have been distinguished for *P. infestans* by RFLP, employing different restriction enzyme combinations (Carter et al. 1990). The old asexual *P. infestans* has an Ib mitochondrial haplotype, while the new sexually reproducing population consists of haplotypes Ia, IIa and IIb (Smart and Fry 2001).

The RFLP probe RG57, which has been extensively used in *P. infestans* population studies, yields a genetic fingerprint

of 25–29 bands (Forbes et al. 1998). It has proven to be a useful tool in population studies (Cooke and Lees 2004) and in tracking inoculum sources of *P. infestans* (Zwankhuizen et al. 2000). Amplified Fragment Length Polymorphism (AFLP) markers yield many loci per primer combination and have played a central role in genetic mapping of *P. infestans* (Cooke and Lees 2004). They have also been used in population studies to study intra-population diversity (Knapova and Gisi 2002, Flier et al. 2003a). The method is said to be sensitive to changes in DNA quality and comparison of results between laboratories is very difficult (Cooke and Lees 2004).

Simple sequence repeat (SSR) markers or microsatellites are short fragments of DNA in which motifs of 1–6 bases occur repeatedly throughout the genome. Recently they have been used in different genetic studies of *P. infestans* (Lees et al. 2006, Brurberg et al. 2011). Microsatellites can be used to detect individual *P. infestans* isolates in a population and the method is said to be repeatable between laboratories (Cooke and Lees 2004).

1.4 Ecology and epidemiology of *P. infestans* in potato

1.4.1 Primary sources of inoculum

Until the 1980s in Europe the only primary sources of inoculum for asexually reproducing clonal populations of *P. infestans* were infected tubers (Figure 4) that managed to overwinter in storage, soil or dumps (Hirst and Stedman 1960, Mäkelä 1966, Zwankhuizen et al. 2000). The invasion of a sexually reproducing population during the 1980s and 1990s into Europe represented a new primary inoculum source; *P. infestans* oospores able to survive in the soil without a living host (Andrivoon 1995, Drenth et al. 1995). The relative importance and impact of epidemic progress

of different potential sources of primary inoculum differs considerably between European countries and globally (Fry 2008). In the Netherlands and the UK (Cooke et al. 2011) waste potato in dumps was recognised to be the most dangerous source of inoculum for potato production and very strict rules were imposed in 1999—all dumps had to be covered before growing season with black plastic in the Netherlands (Zwankhuizen et al. 2000). After successful elimination of dumps, latently infected seed tubers became the major source of primary inoculum (Cooke et al. 2011).

Also in the UK and Ireland (as reviewed in Cooke et al. 2011), and Germany (Möller et al. 2009), infected seed potato is regarded as one of the most important sources of primary inoculum. It is suspected that latently infected seed tubers in particular might be the main source of inoculum because up to 20% of tubers in certain commercial certified seed lots have been latently infected by *P. infestans* (Appel et al. 2001). In fact, symptomatic tubers are not a very efficient source for initiating late blight epidemics. In studies of Hirst and Steadman (1960), only 21 tubers out of 3260 (0.6%) in five successive years were able to produce symptomatic stems. Similar results have been obtained in Finland (Mäkelä 1966). Also in our own small exercise in 1999, only 27 of 400 symptomatic tubers planted in the field germinated. Altogether 135 individual shoots were produced and all of them remained symptomless to mid-July when typical air-borne late blight lesions appeared on upper leaves of the plants (Asko Hannukkala and Ari Lehtinen, unpublished).

Though both mating types A1 and A2 and new sexually reproducing *P. infestans* populations have been recognised throughout Europe, oospores are regarded as being of minor importance as a source of primary inoculum in most countries (Möller et al. 2009, Cooke et al 2011). Only in

the Nordic Countries (Andersson et al. 1998, paper II, Widmark et al. 2007) and the Netherlands (Turkensteen et al. 2000, Zwankhuizen et al. 2000, Cooke et al. 2011) are oospores (Figure 4) regarded as an important source inoculum in practical potato production. From the Nordic Countries there is no estimate of the frequency of oospore-derived epidemics, but according to a Dutch survey 18% of the cases of early infections in monitored potato fields in 2003–2005 were driven by oospores (Evenhuis et al. 2007).

Alternative hosts also may serve as a primary source of inoculum. *P. infestans* has been traditionally regarded as a pathogen with a limited host range (Fry 2008). In

North America and Europe the most important cultivated hosts are potato and tomato (*Solanum lycopersicum* L.) (Berg 1926, Legard et al. 1995). Also certain Solanaceous weeds in North America and Europe and numerous wild *Solanum* species in Central and South America can be infected (Flier et al. 2003a, Fry 2008). Until recently the role of alternative hosts in the epidemiology of late blight in Europe has been regarded as minor (Flier et al. 2003b). The importance of certain Solanaceous weeds like *Solanum nigrum* L. and *S. physalifolium* Rusby could increase in northern Europe in the future as a result of climate change. These weeds are currently very rare in Finland, but occur frequently in southern Scandinavia, where *S. physali-*

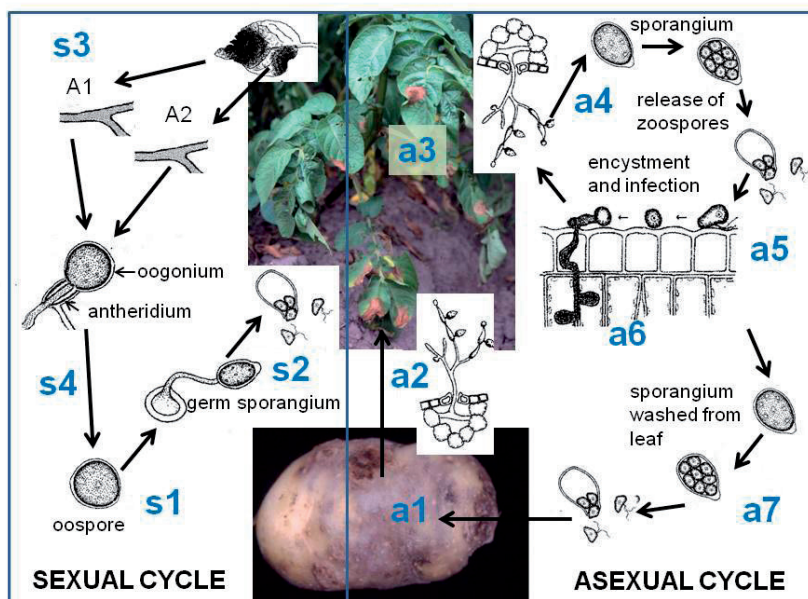


Figure 4. Life cycle of *P. infestans*. Asexual cycle: a1 *P. infestans* overwinters in infected tubers. a2. Primary infection takes place via mycelia invading developing shoot or via sporangia and zoospores formed on tuber surface. a3. Primary lesions are formed in infected stems or leaves. a4. Sporangia produced in lesions transmit the disease in potato canopy. a5. Sporangia entering potato tissue release zoospores, zoospores are encysted and infect the tissue. a6. Several generations of sporangia are formed in crop during season. a7. Part of the sporangia are washed into soil and zoospores formed in soil infect tubers. Sexual life cycle: s1 Oospores overwinter in soil. s2. In moist soil oospores form one sporangium and zoospores infecting potato. Thereafter life cycle continues asexually from phase a3. s3. When mycelia of both mating types A1 and A2 get in close contact oogonium and antheridium are formed. s4. As result of fertilization new oospores are formed and released in soil in plant debris. Portions are adapted from Agrios (2005) and constructed from drawings in Judelson (1997).

folium has been shown to be a very favourable host for oospore production of *P. infestans* (Andersson 2007).

1.4.2 Primary infection and colonisation

Infection of shoots via infested tubers can be caused by mycelium growing from the tuber into the developing stem tissue or via sporangia and zoospores formed on the tuber surface (Figure 4) under moist conditions (Hirst and Stedman 1960). It has been shown with ELISA (Schlenzig et al. 1999) and PCR techniques (Appel et al. 2001) that *P. infestans* can be detected from sprouts and the lower parts of the stem, indicating systemic growth of the pathogen from tubers to stems. In symptomatic potato tubers sporulation has been detected within 19 hours after they have been taken from cold storage to a warmer environment and high moisture. Zoospores formed in seed tubers in soil can infect neighbouring plants, stems and the lowest leaves of the shoot produced by infested seed tubers (Hirst and Stedman 1960, Johnson 2010).

Oospores in soil germinate and form one sporangium, which then produces motile zoospores. Zoospores can swim in wet soil towards stems and leaves that touch the ground and thereby cause infection (Figure 4). Oospores and zoospores can also be transmitted to lower parts of the crop in soil splashed by raindrops. Germination of oospores is favoured by wet soil and temperatures of 10–20 °C (Pittis and Shattock 1994, Turkensteen et al. 2000, Stömberg et al. 2001).

1.4.3 Secondary spread infection and colonisation of aerial potato tissues

The driving force of destructive late blight epidemics is the ability of the pathogen to produce rapidly enormous quantities of asexual spores, sporangia (Crosier 1934).

It is estimated that more than 300,000 sporangia per day can be produced in a single lesion on a potato leaflet (Legard et al. 1995, Fry and Goodwin 1997). Recent Nordic studies show that the most aggressive Nordic strains under favourable conditions can produce more than 1000 sporangia/mm² infested leaf (Lehtinen et al. 2009). The sporangia produced in primary lesions are spread by rain splashes within the plant and into the neighbouring plants (Figure 4). Winds and aerosols of water vapour created during rain showers carry infectious sporangia within fields and also promote long-distance dispersal over wider geographic regions. During heavy rains sporangia in air are washed into the ground outside potato fields and are mostly eliminated. Sporangia are also very vulnerable to UV radiation and lose their infectivity within minutes in direct sunshine (Harrison 1992, Fry 2008).

Sporangia landing on susceptible host tissue in relatively dry and warm conditions can germinate directly, forming a germ tube that penetrates actively through the host cell wall or via open stomata. In moist conditions sporangia attached to the host surface produce numerous motile zoospores. Zoospore formation is further enhanced by chilly temperatures of around 4 to 6 °C. Within 30 to 60 minutes zoospores are encysted and in favourable conditions cysts germinate as germ tubes, which actively penetrate through the host epidermis (Figure 4). The mycelia of *P. infestans* within host tissue grow in the spaces between cells and produce haustoria, taking up nutrients from host cells. Gradually host tissue collapses as seen in visible symptoms. The pathogen starts to produce the next generation of sporangia at the tips of sporangiophores growing out of stomata on the lower lamina of the potato leaf. Secondary sporangia are furthermore spread by wind, rain splashes and aerosols induced by rains and the disease in the crop increases at an exponential rate with increasing generations of sporangia if

the disease development is not restricted by fungicides, unfavourable environmental conditions or lack of susceptible host tissue at the end of the epidemic (As reviewed by e.g. Harrison 1992, Fry 2008). In current Nordic *P. infestans* populations a new generation of sporangia can be produced in favourable conditions every 3 to 6 days depending on the aggressiveness of the isolate (Lehtinen et al. 2009).

1.4.4 Survival in tubers

For the asexually reproducing *P. infestans* population the ability to infect tubers was its only mechanism to ensure its survival to the next season. Tuber infection is induced by sporangia and zoospores produced in the canopy and washed off into the soil by rain showers or dew. No systemic growth of the pathogen via stolons to tubers is known to occur. Oospores in soil can, however, infect tubers. Tubers are infected mainly by motile sporangia in wet soil via open lenticels, eyes, growth cracks or wounds (Croxall and Smith 1976, Darsow 2005, Olanya et al. 2009). Additionally, tuber infection can take place during harvest if during lifting tubers are mixed with infested foliage. Tubers can further be infected between harvest and storage if they are exposed to air-borne sporangia of *P. infestans* from surrounding potato fields. Chemical or mechanical haulm killing before harvest reduces the risk of tuber blight (Miller et al. 2002, Nærstad et al. 2007).

Tuber infection is affected by a range of biotic and abiotic factors, as reviewed by Nyankanga et al. (2007) and Olanya et al. (2009). The level of late blight infestation in a canopy is not very well correlated with the amount of tuber blight. The risk for tuber blight is highest if disease progress in the canopy is slow, resulting in a long period of sporulating lesions present in the crop, and if simultaneously frequent rain showers wash off sporangia into the soil and high soil moisture, crucial for zoospore formation, is maintained

(Croxall and Smith 1976). Dispersal of *P. infestans* from diseased tubers to healthy ones before harvest in soil is common in moist soil (Olanya et al. 2009). Under normal low temperature storage conditions tuber blight does not markedly spread from one tuber to another, but if storage temperature rises above 8 to 10 °C sporangia produced on diseased tuber surfaces can spread rapidly throughout the stored tubers (Dowley and O'Sullivan 1991, Johnson 2010).

After infection of a tuber the survival of an asexual *P. infestans* population is fully dependent on the viability of the tuber in storage, soil or waste pile. The survival ability over winter in a cool climate has probably been one of the most serious genetic bottlenecks reducing the diversity of *P. infestans* populations before the era of oospores. In the northern European climate tubers stored outside have traditionally frozen during winter, but warmer winters have changed this situation (as reviewed in Cooke et al. 2011). Also, improved storage technologies, including rapid cooling, improves the ability of infested tubers to remain viable or even symptomless during storage (Olanya et al. 2009). In studies of survival of *P. infestans* in artificially infested tubers in Finnish conditions carried out in the early 1960s (Mäkelä 1966), practically none was infectious after storing or burial in soil under natural conditions. Hirst and Stedman (1960) and Croxall and Smith (1976) reported comparable results for a slightly warmer European climate.

1.4.5 Survival as oospores

If both mating types A1 and A2 are present in a single leaflet of potato, or stem tissue, oospores can form in abundance in a potato canopy in the Nordic climate (Strömberg et al. 2001). Oospores formed in potato tissue are incorporated into soil attached to potato residues and survive in the absence of a living host in soil after

degradation of host tissue (Andrison 1995, Drenth et al. 1995). The reported longevity of oospores differs between individual studies, but in most cases it is shown to be long enough for survival of the pathogen over winter (Pittis and Shattock 1994, Andrison 1995, Drenth et al. 1995, Medina and Platt 1999, Turkensteen et al. 2000, paper II, Cooke et al. 2011). In studies of Turkensteen et al. (2000) oospore contaminated soils remained infectious for 3–4 years depending on the soil type in the Dutch environment. Nordic *P. infestans* oospores produced by crossing Danish, Finnish, Norwegian and Swedish isolates, and buried in soils in the respective countries, also remained viable for 3–4 years under natural conditions, though the proportion of viable oospores was very low after 3 years of burial (Cooke et al. 2011).

The formation and germination of oospores is favoured by relatively cool temperatures. Also the ability of *P. infestans* to produce oospores is different for different potato cultivars, but this ability is not clearly connected to field resistance of the cultivar (Strömberg et al. 2001). The importance of the oospores for the survival of the pathogen in the Nordic climate is enhanced by frozen soil, which conserves the oospores in conditions that would kill tubers. It also seems obvious that in spring there is a germination peak of oospores that coincides with the planting and emergence of the potato crop (Widmark et al. 2011).

1.5 Source of origin of *P. infestans* and its migrations to Europe

1.5.1 First migration of potato late blight to Europe

A disease resembling potato late blight is known from ancient times in South and Central America. Initially, from the end of the 1800s it was generally assumed that the pathogen originated in the Andean re-

gion of South America. At the end of the 1920s questions were raised about this theory and the centre of origin of *P. infestans* was suggested to be in central Mexico (Reviewed by Fry 2008). Subsequently, and up to the present time, the centre of origin of late blight has been debated vigorously (Reddick 1939, Zentmyer 1988, Goodwin et al. 1994, Abad and Abad 1997, Grunvald and Flier 2005, Gomez-Alpizar et al. 2007). There are reviews of historical articles and studies on old herbarium material examined using modern DNA-based genetic markers that support both theories and the controversy seems to continue (Ristaino et al. 2001, Fry 2008).

In Europe, potato late blight was first noticed in Belgium in 1844 (Zadoks 2008). The year 1845 was the first severe blight year, although from the meteorological point of view it was not an extreme one. The disease rapidly radiated out through Central Europe and countries from Germany to Ireland were affected by October 1845. The consequences of the potato murrain for Ireland have frequently been reviewed and the history of the Irish famine has been told by several authors (Bourke 1964, Gray 1995). The first comprehensive retrospective study of the impacts of potato blight for several countries in Continental Europe in 1845–1848 was published recently by Zadoks (2008). Within one year the disease was established in Europe and became a constant threat to potato ever since, whenever weather is favourable for a disease epidemic (Bourke 1964). The disease had already caused economic hardship for potato growers throughout north-eastern USA during the early 1840s but nothing comparable with the ‘Great Hunger’ in Europe had been faced previously (Stevens 1933, Turner 2005, Zadoks 2008).

The first years of invasion of potato late blight in Europe in 1845–1849 led to one of the most severe human, social, economic and political catastrophes for Eu-

rope, which was, in its own way, comparable with the upheavals resulting from World Wars I and II. Potato in the 1800s had become the principal source of nutrition of the poor people due to its ability to produce twice as many calories per hectare as cereals (Stevens 1933, Zadoks 2008). In several consecutive years the European potato yield was almost completely destroyed. Ireland became a frightening example of the vulnerability of a society where nutrition of the majority of the population was based on a single crop and only one cultivar (“Lumper”) at that. Lumper was very high yielding but very susceptible to late blight. Also serious political conflicts between and within Ireland, Great Britain and Continental European countries arose because of slow acting governments that were unable to provide an alternative food source for their starving populations (Gray 1995, Zadoks 2008).

Due to the potato famine, Ireland lost 2–3 million of its citizens through emigration and famine. Numerous poor emigrants died due to the primitive circumstances on ships carrying them away from Ireland. The large number of immigrants from Ireland and Continental Europe also had severe social and demographic impacts on American and Australian societies (Bourke 1964, Dowley et al. 1995, Fry and Goodwin 1997, Smart and Fry 2001). The Irish Famine has been highlighted repeatedly, but according to Zadoks (2008) the parallel Continental Famine caused a comparable number of deaths and contributed to the revolutions in 1848 following rural pauperisation and urban discontent. In the words of Zadoks (2008), “The Continental Famine was not only caused by poor harvest of potato due to late blight, but also by losses of grain due to frost, drought, rust, voles, inopportune rains, floods and hailstorms. The Famine was enhanced by hoarding, speculation, and poor governance and the hunger was followed by infectious diseases”.

After the severe consequences of potato late blight in the late 1840s, the disease somehow stabilised and lost its power (Turner 2005, Fry 2008, Zadoks 2008). One reason must have been the common practice in Europe to propagate seed potato on farms from true seeds, aimed at providing virus-free potatoes (Colon et al. 1995). A large part of the potato germplasm that had evolved by the 1840s from repeated use of true seed was eliminated in the blight attacks of 1845–1846. Part of these potato populations survived and their descendants were subjected to continuous natural selection in the presence of the disease. This ‘natural’ selection continued through the second half of the 1800s, building field resistance in cultivated potato sufficient to prevent further pan-European catastrophes (Glendinning 1983).

Locally severe epidemics were frequently reported throughout Europe during the second half of the 1800s during rainy seasons. It was typical of the blight damage in the blight years of the 1800s that they had the most detrimental effect on the rural poor and landless labourer populations, who were dependent on the yield from their small, often rented, potato plots (Turner 2005, Zadoks 2008). Throughout the 1900s until the 1970s potato late blight severely reduced potato yields regionally in Europe when moist periods were long enough and when there was enough inoculum carried over from one season to the next (Cox and Large 1960, Croxall and Smith 1976, Kolbe 1982, Zwankhuizen and Zadoks 2002, Dowley et al. 2008).

The population of *P. infestans* introduced to the United States and Europe in the 1840s, according to DNA studies of herbarium specimens, was genetically rather diverse (Ristaino et al. 2001). Some occasional and unknown genetic bottlenecks directed *P. infestans* populations to be dominated by a few clonal lineages in many parts of the world (Fry 2008). *P. infestans* populations outside Mexico un-

til the 1980s had been particularly dominated by a single clonal lineage referred to US-1 (Goodwin et al. 1994, Fry and Goodwin 1997).

1.5.2 Second migration of potato late blight to Europe and elsewhere

The first indication of possible changes in the potato late blight population was the rapid selection of phenylamide resistant *P. infestans* strains at the beginning of the 1980s, soon after commercialisation of the group of phenylamide fungicides (Gisi and Cohen 1996). Phenylamide fungicides such as mefenoxam (metalaxyl-M), metalaxyl and benalaxyl inhibit ribosomal RNA synthesis, specifically RNA polymerisation. In the Netherlands (Davidsse et al. 1983) and Ireland (Dowley and O'Sullivan 1981) metalaxyl resistant *P. infestans* strains caused a severe blight epidemic and considerable crop losses in 1980, after only two seasons of intensive use of metalaxyl products.

The next indication of some serious change in potato late blight was the discovery of A2 mating types in potato fields in 1981 in Switzerland. A2 mating type had never before been reported in potato stands outside Mexico (Hohl and Iselin 1984). Subsequently, there were more reports on the occurrence of A2 mating type strains in Europe (Shaw et al. 1985, Fry et al. 1991). By the beginning of the 1990s occurrence of the A2 mating type had been reported throughout Europe, as well as in Asia, Africa and South America (Fry et al. 1993).

Genetic studies of Spielman et al. (1991), using two polymorphic allozyme loci (Gpi-1 and Pep-1), showed that new genotypes of *P. infestans* that were not found in the old clonal A1 population, were present in several European regions where A2 mating type had been introduced. New genotypes of both A1 and A2 mating types were found in Europe, indicating displacement of the old population in certain re-

gions. According to the review of Fry et al. (1993), the new population in Europe had fully replaced the old population in only a few years, while in the United States and Canada the replacement took place 10–20 years later (Fry 2008).

The origin of the new European population was concluded to be Mexico, from where large quantities of potato had been imported in the late 1970s (Fry et al. 1993). The new North American *P. infestans* population also originates from Mexico, but the migration is most probably linked to infected tomato fruits and therefore the North American population structure is very distinct from that in Europe, Asia and South America (Fry 2008). During the 1990s the existence and establishment of the new population was confirmed by numerous DNA-based studies using e.g. RFLP (RG57) and AFLP markers and mitochondrial haplotypes, as reviewed by Fry and Goodwin (1997).

1.6 Disease management by cultivar resistance

1.6.1 Progress in breeding late blight resistant cultivars

The first attempts in the mid-19th century to control potato blight were efforts to find or breed disease resistant or tolerant potato cultivars. At that time selection of cultivars from seedlings produced from seeds from natural crossings was common practice and some level of resistance was obtained automatically because seedlings were exposed to continuous blight attacks. Thomas Andrew Knight, already in 1807, proposed cross-pollination as a method for combining characters, but cross-pollination only became a common method at the end of the 1800s and the beginning of the 1900s (Glendinning 1983).

In the early 1900s discovery of R-genes, providing apparent immunity against blight in *Solanum demissum*, created op-

timism that late blight problems were finally and forever over (Fry 2008). R-genes code for proteins that recognise specific pathogen effectors and by so doing initiate a series of events leading to complete disease resistance and a hypersensitivity reaction in potato (Glendinning 1983, Colon et al. 1995, Vleeshouwers et al. 2011). During the 1920s and 1930s numerous breeding programmes were carried out to transfer R-genes from *S. demissum* to existing *S. tuberosum* cultivars and from the 1930s onwards R-gene-containing commercial cultivars were introduced (Toxopeus 1956, Colon et al. 1995). Unfortunately, it was rapidly discovered that the new resistant cultivars were very soon attacked by newly arising races of *P. infestans* (Toxopeus 1956).

In the 1950s four R-genes from *S. demissum* were identified and named R₁ to R₄, and were deployed in cultivars (Black et al. 1953, Toxopeus 1956). In the 1950s and 1960s seven more R-genes, R₅ to R₁₁, were discovered but simultaneously it was found that many strains of *P. infestans* were compatible to the new R-genes even if they had never been used in grown cultivars (Malcomson 1969). This finding in the UK, and later all over Europe, decreased considerably the enthusiasm for using R-genes as a source of resistance in breeding programmes, but some breeders still keep faith in finding new durable R-genes (Fry 2008).

There has been much debate among potato breeders since the 1950s as to whether to use R-genes or field resistance, which does not provide complete resistance, but which represents a more durable basis for good tolerance to late blight (Toxopeus 1956, Mastenbroek 1966, Umaerus et al. 1983, Colon et al. 1995, Fry 2008). Rapid progress in development of new modes of actions of fungicides and application techniques, especially in the 1980s and 1990s, further decreased interest in breeding for resistance against late blight and potato

production in the Western World became fully dependent on chemical blight control (Zwankhuizen and Zadoks 2002, Turner 2005, Gisi et al. 2011).

Global resistance to *P. infestans* has been one of the most important targets in numerous potato breeding programmes (Mastenbroek 1966, Umaerus et al. 1983, Park et al. 2009, Jacobs et al. 2010). Since the 1980s the increased concern on the adverse environmental effects and risks for consumer health of applying agricultural pesticides has increased pressure to develop more resistant cultivars (Allefs et al. 2005, Haverkort et al. 2008). The pressure of recent EU pesticide legislation (Directive 2009/128 EC) to reduce the use of pesticides and make National Action Plans further increases the need for more resistant cultivars (Barzman and Dachbrodt-Saaydeh 2011). Until recently the strong linkage between foliar field resistance to late blight and late foliage maturity under long-day conditions has been a problem in developing the desired cultivars. In fact, due to the long growth period, varieties with moderate resistance need more protective fungicide applications than early-maturing less resistant cultivars (Allefs et al. 2005).

Sequencing genomes of both potato and *P. infestans* (Haas et al. 2009) has provided tools to study structure and mechanisms of functions of the resistance genes in potato (Jacobs et al. 2010). Many new R-genes additional to R₁ to R₁₁ have been recognised in wild *Solanum* species. It has also become possible to engineer R-genes and introduce new types of R-gene-based resistance in cultivated potato, which is also believed to be more sustainable than that achieved using the old R-genes (Vleeshouwers et al. 2011). It has been concluded by Allef et al. (2005) that due to fast progress in understanding the function of resistance/avirulence genes in *P. infestans* and potato, and development of novel molecular tools for potato breeders, breeding for

late blight resistance in potato has only just begun. The economic and societal benefits achieved by growing potato varieties with durable resistance were recently highlighted by Haverkort et al. (2008).

1.6.2 Potato breeding, cultivars and late blight resistance in Finland

Interest in potato cultivars started to increase in the 1890s when it was generally realised that potato germplasm in Finland was old and degenerate. Many regional Farmer Associations started to collect funds to import new cultivars from Europe, like cv. “Magnum Bonum”, which was believed to be relatively blight resistant (e.g. Newspaper *Uusi Suometar* March 2nd 1892). The systematic comparisons of different European potato cultivars were started at the Finnish Agricultural Experimental Institute in 1911 (Reviewed by Yllö 1963).

Potato breeding in Finland was started in 1920 at Hankkija Potato Breeding Institute and in 1926 at the State Institute for Agricultural Research, later the Agricultural Research Centre. Plant breeding in Finland, including for potato, was reorganised in 1994 when all breeding activities were fused into the independent Boreal Plant Breeding Company. The main goals of potato breeding in Finland have been and remain: early varieties for summer use, early main crop varieties and early high yielding starch varieties with high starch content (Varis 2001, <http://www.boreal.fi>). Unfortunately, earliness is very strongly associated with high susceptibility to blight (Mastenbroek 1966, Seppänen 1971). Good adaptation to the cool Nordic climate and long-day conditions and reasonable resistance to the main pests, have been taken into consideration in Finnish potato breeding programmes (Varis 2001). The resistance to potato late blight has been taken into account by monitoring blight incidence and severity in varie-

ty field trials (Yllö 1963, Seppänen 1971). Currently the first progenies of crossings are also subjected to intense potato late blight pressure and the most susceptible ones are automatically discarded (Leena Pietilä, personal communication).

The first Finnish potato cultivar was Tammiston Aikainen (Tammisto Early), released in 1930. It was, according to its name, a very early variety. It was relatively widely grown until the 1980s. Two Finnish table potato cultivars that had success were Jaakko (1951) and Pito (1964), but they are no longer grown (Varis 2001). Probably the most successful Finnish cultivar has been Hankkijan Timo (1975), a very early variety, which still constitutes approximately 5% of the potato growing area (Table 1). It is the dominant cultivar in early potato production, especially under fleece (Varis 2001). For an early variety Hankkijan Timo has good late blight resistance, especially against tuber blight (Table 1). Another Finnish variety of some importance is Hankkijan Tanu, which is an early starch variety (Varis 2001). Currently it is an important cultivar for the starch industry to get the starch factories running as early as possible in the season (Ossi Paakki, personal communication).

Finnish potato production throughout history has been dependent on foreign cultivars though the seed potatoes are largely multiplied in Finland. Until the 1970s cultivars originating from Great Britain and Germany dominated production, and were accompanied by a few Dutch cultivars like Bintje. Since the 1970s Dutch varieties such as Record replaced the British cultivars and some Swedish cultivars, like Sabina and Matilda, gained in popularity (Varis 2001). Currently Dutch varieties dominate the main crop potato and processing industry production, with the exception of the German cultivar Nicola. Most of these cultivars have medium level resistance to leaf blight, but they are rather resistant to tuber blight (Table 1).

Table 1. The most commonly grown potato cultivars in Finland from 2005 to 2009 as a percentage of total potato growing area, their country of origin and estimated resistance to leaf and tuber blight. Growing areas: Information Centre of Ministry of Agriculture and Forestry, TIKE; Origin and estimates for resistance properties: The European Cultivated Potato Database; <http://www.europotato.org>

Cultivar	Origin	Leaf blight resistance	Tuber blight resistance	2005 %	2006 %	2007 %	2008 %	2009 %
Posmo	DK	Low	Medium	10.2	11.3	12.0	12.3	11.8
Nicola	DE	Medium	High	8.8	9.1	9.7	9.4	9.4
Van Gogh	NL	Medium	High	14.4	14.5	13.3	10.9	8.1
Saturna	NL	Medium	High	12.5	11.9	10.3	9.1	7.7
Asterix	NL	Medium	High	6.6	6.1	5.7	5.9	6.3
Hankkijan Timo	FI	Medium	High	4.8	4.9	5.1	5.3	5.4
Kardal	NL	High	High	3.5	4.4	3.7	3.8	4.9
Melody	NL	Medium	High	0.0	0.1	0.4	1.7	4.1
Rikea	DE	Low	Medium	2.2	2.2	2.4	3.1	4.0
Victoria	NL	Medium	High	2.9	2.9	3.2	3.0	3.4
Fambo	NL	Low	High	3.5	2.9	2.6	2.6	3.2
Inova	NL	Low	High	0.0	0.8	1.7	2.5	3.1
Hankkijan Tanu	FI	Low	Low	4.4	4.3	3.9	4.0	3.1
Others				26.2	24.6	26.0	26.4	25.5
Total				100	100	100	100	100

1.7 Disease management by fungicides

1.7.1 General development of fungicides against late blight

Soon after the first migration of potato late blight in Europe, the effect of different inorganic compounds was tested to reduce the disease. The first attempts were not very successful because the epidemiology of the disease was not well understood. For example, a mixture of copper sulphate, lime and table salt was suggested as a soil treatment to prevent tuber blight (Turner 2005). Globally, chemical late blight control became routine after the Second World War (McCallan 1967). Currently in Europe it is difficult to manage potato production without frequent applications of fungicides against late blight. Fungicides used for late blight control are not toxic to potato. Modern compounds are engineered to interfere with vital metabolic pathways in *Oomycetes* and many of them do not prohibit growth of true fungi (Gisi and Sierotzki 2008).

The first universal fungicide, ‘Bordeaux Mixture’, was developed by Dr. Millardet (French) and was officially introduced in 1886. Bordeaux Mixture is a mixture of copper sulphate and slaked lime (McCallan 1967). This compound dominated the world fungicide market until the 1940s when it was replaced by less toxic fixed-copper fungicides and the first organo-metallic compounds (Turner 2005). Bordeaux Mixture and copper fungicides are not absorbed or transferred in plant tissue. They should be applied prophylactically and only complete coverage of all plant tissues provides efficient control (Finckh et al. 2008). Copper and other inorganic products in Europe during recent decades have mainly been used in organic potato production. Gradually through the 2000s they have been banned over environmental concerns in most European countries, including Finland (Speiser et al. 2006).

The development of organic chemistry during and after the Second World War, part-

ly by accident provided new compounds for chemical blight control. Dithiocarbamates (Table 2) were patented in the USA already in 1934, but it was only in 1946 when their efficacy in potato late blight control was realised. Manganese ethylenbisdithiocarbamates, maneb (1954) and mancozeb (1961), during the 1960s became the most commonly used fungicides against late blight (McCallan 1967). They are still globally the most widely used late blight fungicides as straight products and in pre-packed mixtures with numerous other compounds (Gullino et al. 2010). After application dithiocarbamates remain on the surface of the plant and are not absorbed by plant tissues (Edgington et al. 1980). Dithiocarbamates can be redistributed from the upper leaf layers to lower leaf layers (Bruhn and Fry 1982). There is also evidence that under favourable moisture conditions mancozeb can be redistrib-

uted and protects new growth better than expected for a contact fungicide (Evenhuis et al. 2006). Heavy rain or irrigation however washes mancozeb from the plant into the soil and protection efficacy is rapidly lost under these conditions (Schepers 1996).

The next significant improvement in chemical late blight control was the development of fungicides capable of penetrating and moving within plant tissue (Cohen and Coffey 1986, Wong and Wilcox 2001). Systemic fungicides penetrate the plant via roots or aerial parts and are transported within vascular tissue, also protecting new growth after treatment (Edgington et al. 1980, Cohen and Coffey 1986). Locally systemic compounds, often termed translaminar fungicides, penetrate leaf tissue from a sprayed to an unsprayed lamina in concentrations sufficient to provide disease

Table 2. Biochemical mode of action, fungicide group (FRAC 2011), active ingredients and pre-packed mixtures of fungicides used for potato late blight control in Finland.

Biochemical mode of action	Fungicide group (FRAC classification, 2011)	Active ingredients	Commercial products straight or in mixture with another active ingredient
Multi-site contact activity	Inorganic compounds	Copperoxychloride	straight
	Dithiocarbamates	Maneb Mancozeb	straight straight, metalaxyl, metalaxyl-M, fenamidone, dimethomorph, zoxamide, cymoxanil
Inhibiting ribosomal RNA synthesis	Phenylamides	Metalaxyl Metalaxyl-M	mancozeb mancozeb, fluazinam
Disturbing cell membrane assembly	Carbamates	Propamocarb-hydrochloride	mancozeb, fenamidone
	Carbocyclic acid amides	Dimethomorph Mandipropamid	mancozeb straight
Inhibiting mitochondrial respiration	Dinitroanilines	Fluazinam	straight, metalaxyl-M
	Quinone outside inhibitors	Famoxadone Fenamidone	cymoxanil mancozeb, propamocarb-HCl
	Quinone inside inhibitors	Cyazofamid	straight
Inhibiting mitosis and cell division	Benzamides	Zoxamide	mancozeb
Unknown mode of action	Cyanoacetamideoximes	Cymoxanil	mancozeb, famoxadone

control on the unsprayed side. They usually also move towards new growth after treatment at leaf edges, but are not transported in vascular tissues towards the apex (Ypema and Gold 1999, Wong and Wilcox 2001).

The first fungicide for late blight control with translaminar properties was cymoxanil, which was launched on to the market in 1977 (Douchet et al. 1977, Cohen and Grinberger 1987, Johnson et al. 2000). The first truly systemic fungicide, metalaxyl (Table 2), was also introduced on to the market in 1977 (Urech et al. 1977). It belongs to a group of phenylamides that have protective, curative and eradicated antifungal activity, controlling different diseases caused by *Peronosporales*, including *P. infestans* (Cohen and Coffey 1986). The weakness of phenylamides is the risk of rapid development of fungicide resistance in *P. infestans* populations (Dowley and O'Sullivan 1981, Davidse et al. 1983, Gisi and Cohen 1996, Dowley et al. 2002).

Due to resistance problems faced a few years after intensive use of phenylamides for late blight control (Dowley and O'Sullivan 1981, Davidse et al. 1983), several new types of fungicides were developed and introduced in the 1980s and 1990s. The systemic fungicide, propamocarb-hydrochloride (HCl), was introduced into commercial use in 1978 and was primarily used as a soil treatment against *Oomyctes* in ornamentals and vegetables. In the 1980s it was additionally adapted for potato late blight control to replace phenylamides (Cooke et al. 1981, Burden et al. 1988, Löchel and Birchmore 1990). Propamocarb-HCl belongs to the carbamate group of chemicals (Gisi and Sierotzki 2008).

The first carboxylic acid amide (CAA) fungicide, dimethomorph (Table 2) was introduced in 1988 (Albert et al. 1988) and commercialised as a pre-packed mixture with mancozeb during the beginning of

the 1990s (Cohen et al. 1995). Dimethomorph is a translaminar fungicide fully absorbed within a few hours after application into leaf tissue and the adjacent leaf lamina (Cohen et al. 1995, Cohen and Gisi 2007). Another CAA fungicide, mandipropamid (Table 2), was introduced and commercialised in 2005 (Huggenberger and Kuhn 2006, Lamberth et al. 2006). Mandipropamid has both contact and translaminar properties. Within two hours after application the fungicide is totally absorbed into the wax layer of the leaf surface, providing good rain-fastness and persistent efficacy. Thereafter mandipropamid is gradually transported into leaf tissues, contributing slower curative and translaminar activities than dimethomorph as well as protection of new growth at the leaf edges (Huggenberger and Kuhn 2006, Cohen and Gisi 2007). The fungicide is also redeposited in the apex, providing reasonable protection for new apical growth, but the mechanism of transport is not yet fully understood (Huggenberger and Knauf-Beiter 2009).

Also new types of contact fungicides were developed in the 1980s and 1990s. Fluzinam, a dinitroaniline compound (Table 2), was first registered for potato late blight control in Europe in the Netherlands in 1992 (Anema et al. 1992). Fluzinam is tightly absorbed into wax layers of the leaf surface within two hours after application, providing good rain-fastness in comparison to dithiocarbamates (Schepers 1996). The compound is persistent in epidermal wax layers where it is deposited for at least thirteen days (Mathiasen et al. 1997). However, the fungicide is not redistributed to the new growth of leaf edges or apex and during the period of rapid potato growth at high infection pressure a safe application interval is between 5 and 7 days, depending on the application rate (Cooke and Little 2006). Another new contact compound, zoxamide, was introduced for potato late blight control in 1998 (Egan et al. 1998) and commercialised in Europe as a pre-packed mixture with man-

cozeb in the early 2000s (Cohen and Gisi 2007). Zoxamide belongs to the benzamide chemical group, but in plants it behaves similarly to fluazinam: after application zoxamide is very strongly bound into the wax layer of the plant epidermis (Bacci et al. 2007). This provides good rain-fastness, but the fungicide has no translaminar or systemic properties and it is not re-distributed after application to any new growth of plant leaves or to the apex (Egan et al. 1998).

The majority of novel fungicides developed after 1999 are inhibitors of the mitochondrial respiration chain (Table 2). They belong to several different chemical groups, but many of them are strobilurins (Bartlett et al. 2002, Gisi et al. 2011). The majority of these compounds have been developed for control of true fungi (Bartlett et al. 2002), but a few compounds also are effective against *Oomycetes* (Gisi and Sierotzki 2008). Famoxadone was announced in 1996 (Joshi and Sternberg 1996) and commercialised in 1997 (Bartlett et al. 2002). Fenamidone was first introduced in 1998 (Mercer et al. 1998) and commercialised as different pre-packed mixtures with other compounds in 2001 (Bartlett et al. 2002). Cyazofamid was introduced in 1998 (Mitani et al. 1998) and registered in Europe for potato late blight control in 2001 (Mitani et al. 2001).

Famoxadone is a protectant contact fungicide with no translaminar movement or curative action against *P. infestans* (Joshi and Sternberg 1996). The residual effect in trials has lasted up to 13 days after fungicide application (Andrieu et al. 2001). Fenamidone penetrates crop tissues rapidly and is transported translaminarily, providing also curative action against *P. infestans* (Musson et al. 2004). Cyazofamid is a protective contact fungicide that is absorbed by the epidermal wax layers within one hour after application, being completely rain fast (Ebersold 2002). Residual efficacy lasts for 7–14 days after applica-

tion, depending on the infection pressure (Ohshima et al. 2004, Mitani et al. 2005).

1.7.2 Biochemical modes of action of the most widely used late blight fungicides

Fungicides with a multisite biochemical mode of action do not have any specific target site in the metabolic pathways, but rather disturb several fundamental processes in the life-cycle of the pathogen (Gisi and Sierotzki 2008). Inorganic fungicides are protectant multi-site inhibitors (Table 2) of a wide range of pathogens. The efficacy of copper fungicides is based on passive accumulation of copper in pathogen cells, causing non-specific denaturation of proteins and enzymes (Somers 1963). Dithiocarbamates (Table 2) like mancozeb interfere with at least six different fatal biochemical pathways within the fungal cell cytoplasm and mitochondria. After almost 50 years of intensive use no sign of resistance development against mancozeb or other dithiocarbamates in any pathogen has been detected (Gisi and Sierotzki 2008, Gullino et al. 2010).

Phenylamide fungicides (Table 2) such as mefenoxam (metalaxyl-M), metalaxyl, oxadixyl and benalaxyl are systemic single site fungicides inhibiting ribosomal RNA synthesis, specifically RNA polymerisation (Davidse 1988). Due to the extremely narrow target site in the metabolic processes of pathogens, phenylamides are vulnerable to development of complete resistance in pathogen populations. Moreover, cross-resistance against all compounds in this fungicide class normally develops (Gisi and Cohen 1996). Phenylamide resistance in field isolates of *P. infestans* and failure of these products in late blight control was detected in the Netherlands (Davidse et al. 1983) and in Ireland (Dowley and O'Sullivan 1981) after only two seasons of intensive use. Thereafter resistance has been reported from all over the world (Deahl and Demuth 1992, Gisi and Co-

hen 1996). Since anti-resistance strategies initiated in the 1980s and 1990s (Staub 1991) are followed, phenylamides can be safely used as a part of spray programmes against late blight (Gisi and Cohen 1996, Dowley et al. 2002, Gisi et al. 2011).

Fungicides belonging to the carbamate group and CAA fungicides (Table 2) severely disturb assembly of cell membranes (Gisi and Sierotzki 2008). Propamocarb-HCl is reported to affect permeability of cell membranes of *oomycetes* and cause leakage of cell components (Papavizas et al. 1978). The precise biochemical mode of action of carbamate fungicides has not been published to date. There are certain synergistic effects between propamocarb and other fungicidal compounds (Couch and Smith 1991, Johnson et al. 2000, Mayton et al. 2001, Tafforeau et al. 2009). Propamocarb-HCl fungicides have been relatively widely used worldwide for potato late blight control for almost thirty years without resistance having been detected in field isolates of *P. infestans*, although resistance in certain *Pythium* species has been reported (Gisi and Sierotzki, 2008).

In the presence of CAA fungicides the morphology and ultrastructure of cell walls of *Phytophthora*-species show many remarkable changes (Kuhn et al. 1991, Johnson et al. 2000). However, the primary biochemical inhibitory mode of action and target site of CAA fungicides in *oomycete* cells is still unclear (Cohen et al. 1995, Cohen and Gisi 2007, Gisi and Sierotzki 2008). According to Griffiths et al. (2003) biosynthesis of cell membrane components like phospholipids and lecithin is prohibited. Gisi and Sierotzki (2008) suggested that most likely the inhibitory processes are membrane-bound and take place at the interface between the plasmalemma and cell wall. In spite of intensive monitoring of resistance in *P. infestans* populations to dimethomorph since its commercialisation, no resistant field isolates of the pathogen have been found (Gisi and Sierotzki

2008, Cooke et al. 2011). Relatively short practical experience in monitoring of development of resistance against mandipropamid does not indicate any resistance problems (Cohen et al. 2007, Cooke et al. 2011, Gisi et al. 2011).

The biochemical mode of action of fluazinam is based in an uncoupling effect of oxidative phosphorylation (Table 2) that interrupts the fungal cell energy production process (Anema et al. 1992, Tucker et al. 1994). Based on extensive worldwide efficacy tests and practical experiences throughout the 1990s, fluazinam proved to be an effective alternative to other fungicides available at that time for leaf blight control. It also controlled tuber blight more efficiently than any other fungicide at that time (Weiterstadt 1997, Cooke et al. 1998). Response of *P. infestans* populations to fluazinam has been monitored frequently worldwide since the early 1990s. No sign of resistance or increased tolerance to fluazinam has been reported in field populations of *P. infestans* (Cooke et al. 1998, Cooke and Little 2006, Gisi and Sierotzki 2008, Gisi et al. 2011).

Most fungicides developed after 1999 inhibit electron transport at cytochrome b by binding to the Qo or Qi site, the ubiquinol oxidising pocket. This is located on the outer side of mitochondrial membranes (Gisi and Sierotzki 2008). Famoxadone and fenamidone (Table 2) are chemically distinct from strobilurins, but they have similar biochemical modes of action and target site and they belong to the same cross-resistance group as strobilurins (Barlett et al. 2002). Cyazofamid blocks the electron transferin mitochondrial cytochrome bc₁ complex. The binding site of cyazofamid is the Qi centre of the enzyme. Therefore, cross-resistance with strobilurin type products acting at the Qo-site is improbable (Ohshima et al. 2004). Fenamidone or cyazofamid resistant field strains of *P. infestans* have not yet been detected (Gisi et al. 2011).

The biochemical mode of action of zoxamide (Table 2) is unique among *Oomycete* fungicides. Zoxamide inhibits nuclear division of *Oomycete* cells by disruption of cellular microtubules as the result of highly specific covalent binding to the β -subunit of tubulin (Egan et al. 1998, Young and Slawewski 2001). A similar mode of action is known for relatively old benzimidazole fungicides e.g. benomyl and thiophanatemethyl, active against many representatives of true fungi (Davidse 1986). Zoxamid has no cross-resistance with other chemical groups of fungicides (Egan et al. 1998). The theoretical risk for resistance development in *P. infestans* against zoxamide is considered relatively low (Young et al. 2001, Cooke et al. 2002, Bacci et al. 2007). The biochemical mode of action of cymoxanil is unknown. Resistance against cymoxanil has not been reported in field populations of *P. infestans* (Gisi and Sierotzki 2008).

1.8 Integrated approach to late blight management

Potato late blight control in conventional potato production in Europe is currently highly dependent on frequent fungicide applications (Cooke et al. 2011). Recent EU Pesticide legislation (Directive 2009/128 EC) and corresponding Finnish legislation (Laki kasvinsuojeluaineista 1563/2011) emphasise sustainable use of pesticides and integrated pest management (IPM) practices. National action plans (NAP) for sustainable use of pesticides must be made in each EU country by the end of 2012. Considerable reductions in pesticide use are suggested in published NAPs (Barzman and Dachbrodt-Saayeh 2011). According to the directive (2009/128 EC) all professional growers must implement principles of IPM in their production by January 1st 2014.

In potato late blight management IPM and reduction of application of late blight fungicides can be achieved by reducing sourc-

es of primary inoculum, using cultivar resistance, improving crop rotations, using late blight forecasts and decision support systems (DSS) and improving crop management technologies (Hewson and Sagenmüller 2000, Hansen et al. 2002, Fry 2008, Cooke et al. 2011).

In most parts of Europe infected seed potato is one of the most important sources of primary inoculum (Zwankhuizen et al. 2000, Cooke et al. 2011). The incidence of symptomatic tubers in certified seed potato is strictly limited, but the occurrence of latent infections is normally not inspected. In Germany up to 20% of tubers in certain commercial seed lots have been latently infected (Appel et al. 2001). In current high quality seed potato storage at temperatures below 4 °C tuber blight symptoms rarely develop, though the pathogen survives in latently infected tubers to the next season. PCR-based tests of seed potato to detect latent infections should be applied to improve seed quality (Johnson and Cummings 2009).

In regions where waste piles are regarded as the main source of primary inoculum, covering dumps with black plastic has proven to be a very effective way to reduce blight (Zwankhuizen et al. 2000, Cooke et al. 2011). Volunteer potato growing as weeds in other crops may represent a source of primary inoculum though their main effect seems to be accelerating the epidemic rather than being a primary inoculum source. Survival of volunteer plants can be reduced by herbicides and crop rotations (Boonekamp 2005, Cooke et al. 2011).

In regions like Finland, where oospores play an important role as a primary source of inoculum (Evenhuis et al. 2007, paper I), it is important to prevent production of oospores by keeping the crop as healthy as possible until harvest. Also a long enough crop rotation reduces the amount of oospores in the soil (Andrivon 1995, Turken-

steen et al. 2000). It was shown in paper I and by Bødker et al. (2006) that in fields with crop rotation late blight starts considerably later than in potato monoculture or where very short rotations are used, which can reduce the need for control by one to two fungicide applications.

There is much potential for reduction of fungicide applications if cultivars with good field resistance were to be grown more extensively than now. In Western Europe the choice of cultivar has so far been based on high yielding capacity and other cultural and technical properties rather than late blight tolerance or resistance. Moreover, varieties with reasonable field resistance can be grown safely with low fungicide input at low or moderate infection pressure, but there is risk of failure in blight control at high disease pressure (as reviewed in Cooke et al. 2011).

DSSs can organise and integrate all available information on the epidemiology of *P. infestans*, historical weather data and forecasts, potato growth, properties of fungicides, cultivar resistance and modelled disease pressure to make decisions concerning successful late blight management (Fry et al. 1983, Hansen et al. 2002, Cooke et al. 2011). Potato experts and advisors have utilised numerous DSSs developed in their extension work, but among ordinary potato growers the systems have not been very popular (Hansen et al. 2002). It is possible that the new EU directive (128/2009 EC) encourages improvement and application of these DSSs also in practice.

1.9 Objectives of the thesis

The aim of this thesis is to summarise historical and recent migrations of *P. infestans* into Europe and especially into Finland. The changes due to the migrations in disease epidemiology, phenotypic traits of the pathogen population and their consequences for disease management practices

are emphasised. Impacts on genetic traits of the pathogen population are only briefly reviewed.

The main questions and hypothesis to be tested were the following:

1. How potato late blight incidence, severity and onset of epidemics in Finland have fluctuated after first disease outbreaks in 1845?

Hypothesis 1: There are random annual fluctuations in late blight incidence and severity but no systematic change before 1990s.

Hypothesis 2: Late blight epidemics since the end of the 1990s have started clearly earlier than before that.

Hypothesis 3: There have been less years since the end of the 1990s when late blight is fully absent than before that.

2. Has warming temperature and increased precipitation since the mid 1990s favoured early late blight outbreaks?

Hypothesis 1: Early starting late blight epidemics are due to warm weather and high precipitation at the period preceding disease outbreak.

3. If the late blight epidemics have started earlier since the mid 1990s, have the other epidemiological parameters changed?

Hypothesis 1: Relative area under disease progress curve has increased.

Hypothesis 2: The angle of apparent infection rate has become steeper.

Hypothesis 3: The time of disease progress from 5% to 95% leaf blight severity has become shorter.

4. Do oospores, sexual reproduction, increased phenotypic and genetic diversity play role in the epidemiology of late blight in Finland?

Hypothesis 1: Both mating types A1 and A2 are present in Finland.

Hypothesis 2: Oospores are formed in potato crop.

Hypothesis 3: Oospores are responsible for early starting late blight outbreaks.

Hypothesis 4: Short crop rotations increase risk for oospore derived late blight outbreaks.

Hypothesis 5: Diversity and complexity of virulence races has increased in *P. infestans* population.

Hypothesis 6: Genetic diversity as indicated by different DNA-markers has increased in *P. infestans* population. This hypothesis is only shortly reviewed on base of co-author Nordic papers not included as original papers in this thesis.

5. Has the fungicide use against late blight increased due to obvious shift towards early starting late blight epidemics and has this increased the risk of fungicide resistance?

Hypothesis 1: Sales of fungicides and number of fungicide applications against late blight have increased since the 1990s

Hypothesis 2: Resistance against fungicides metalaxyl and propamocarb hydrochloride has increased as a result of their intensive use

The objective of paper I was to quantify incidence and earliness of late-blight ep-

idemics over the years and determine the factors associated with the increase in the disease from the 1930s to 2002. In paper II the aim of the study was to verify that oospore-derived epidemics actually occur in Finland. Here these data are used to discuss whether the observed early *P. infestans* attacks are influenced by the almost simultaneous finding of oospores as a potential source of primary inoculum. In papers III, IV and V the aim was to compare temporal and spatial changes in phenotypic traits of Finnish and Nordic *P. infestans* populations from 1992 to 2003, with the emphasis on mating type, virulence races and fungicide resistance.

These aspects have been complemented by previously unpublished information on epidemic development in Finland during the second half of 1800s and from 2003 to 2011. Also the development of fungicide use against potato late blight is updated. In this thesis only Finnish data have been used and completed with corresponding data collected from 2006 to 2010. The aim is to show how at national level the obviously increased sexual reproduction has affected late blight epidemiology and *P. infestans* population structure. Also the connections between fungicide use and development in fungicide resistance are highlighted as well as the impact of epidemiological changes on overall late blight fungicide use.

2 Materials and methods

2.1 Historical and recent development of blight epidemics in Finland

2.1.1 Review of historical literature and surveys on late blight incidence from 1845 to 1980

The historical review of blight incidence and severity in Finland from 1845 to 1980 is based on various published sources in newspapers and scientific papers. A systematic survey on late blight epidemic development at six fixed observation sites from 1931 to 1962 was published by Professor Seppänen (1971). The original data from this survey provided by Professor Seppänen were somewhat reorganised for this thesis. Late blight incidence in that period was used as reference data for late blight development from 1983 to 2011.

The late blight incidence and estimated severity prior to 1900 was studied from the Finnish National Library's digital collection of historical newspapers <http://digi.kansalliskirjasto.fi/sanomalehti>. This collection contains most newspapers published in Finland from 1771 to 1910. At that time the articles in newspapers were often written by university professors, teachers and other highly educated experts. Therefore, descriptions of disease development are probably relatively accurate though the reports were based on disease symptoms and the causal agent had not been determined in Finland before 1873.

Simple searches using keywords “perunarutto”, “potaattirutto”, “perunatauti”, “potaattitauti”, “potatissjuka”, “bladmögel” and “brunröta”—terms for potato late blight in Finnish and Swedish—identified over 1000 articles from 1845 to 1900. The searches in the newspaper collection were fuzzy searches, which mean that the server program also searched for words

that resembled the search terms. Only articles containing information on late blight incidence in Finland or describing general biology of the pathogen were included. These articles were sorted by year and the blight incidence was roughly estimated by number of articles published each year. In years when more than 10 articles were published, each article was evaluated to get an impression of late blight incidence and severity in different parts of Finland. Based on these articles epidemic severity for each year was subjectively estimated at local, regional or nationwide levels.

Estimation of potato late blight incidence and severity from 1900 to 1930 and 1964 to 1982 is based on a few occasional published surveys from University of Helsinki, Agricultural Experimental (later Research) Centre and potato breeding institutes. In period 1931–1958 a systematic survey of late blight incidence in variety trials carried out by the Agricultural Research Centre in Tikkurila, southern Finland is available (Yllö 1963). A similar survey at six experimental sites from 1931 to 1962 in different parts of Finland is also used as reference data (Seppänen 1971).

2.1.2 Systematic monitoring of late blight outbreaks from 1983 to 2011

The data on the incidence, severity and late blight progress from 1983 to 2011 are based on observations made on cv. Bintje in unprotected variety trials, untreated experimental plots in fungicide efficacy trials and from 1992 onwards in additional untreated monitoring plots at several monitoring sites in different parts of Finland.

From 1983 to 1991 information on late blight outbreaks is based on potato variety and fungicide efficacy trials carried out

at nine experimental stations of MTT Agrifood Research Finland (formerly MTTK Agricultural Research Centre) and the Potato Research Institute (Table 3). In these trials occurrence of blight was regularly monitored 1–2 times a week from the end of June until the end of the growing season. For this study the date of the first blight outbreak was considered to be when blight lesions were found in unprotected experimental plots of cv. Bintje. The data were obtained from original observation sheets provided by the experimental sites. More detailed description of the methodology and monitoring sites is given in paper I.

From 1992 to 2002 additional and more accurate observation sites were represented by different blight monitoring and warning projects carried out at that time. At each experimental site mentioned above there were specific observation plots (cv. Bintje) that were monitored 2–3 times a week for the appearance of first blight lesions. The leaflets containing lesions were sent to MTT, Plant Protection, where the presence of *P. infestans* was verified by microscopic observation. In addition, participants in the blight warning project mon-

itored selected organic potato fields and home gardens within approximately 50 km radius of fixed monitoring sites. A more detailed description of the monitoring system is given in paper I.

From 2003 to 2011 there were six fixed monitoring sites, Jokioinen, Lammi, Mikkelä, Ylistaro, Ruukki and Rovaniemi (see map attached to Table 3), where the appearance of first blight lesions at the critical period was monitored almost daily. In addition, advisors inspecting potato fields around Finland reported their observations on first late blight outbreaks to MTT, Jokioinen. This has been part of the European blight monitoring network (Euro-Blight, www.EuroBlight.net) and the dates of first observations have also been reported online on the map available on the Euroblight home pages.

Additional information, like types of first symptoms, further blight development on the site, cultural practices and cropping history was collected for each monitoring site from 1992 to 2011. Some of this information has been used as explanatory variables to establish reasons for different types of late blight outbreaks.

Table 3. Fixed monitoring sites for potato late blight epidemics in Finland in 1931–2011 and their location on the map of Finland.

Name of the monitoring site	Coordinates		Elevation above sea level m	Fixed monitoring sites for potato late blight outbreaks 1931 - 2011			
	Latitude N	Longitude E		1931–1962	1983–1991	1992–2002	2003–2011
Kokemäki	61°16′	22°15′	38		x	x	
Jokioinen	60°49′	23°30′	104	x	x	x	x
Pälkäne	61°20′	24°13′	106	x			
Lammi	61°05′	25°01′	100		x	x	x
Mikkeli	61°41′	27°12′	101		x	x	x
Ylistaro	62°56′	22°29′	26	x	x	x	x
Maaninka	63°09′	27°19′	88	x	x	x	
Ruukki	64°41′	25°06′	48	x	x	x	x
Rovaniemi	66°35′	26°01′	106	x	x	x	x



2.1.3 Modelling weather as a possible cause of early late blight outbreaks

Simple plotting of days of first blight outbreaks over years revealed that blight epidemics in the 1990s clearly started earlier than during the 1980s (Figure 6). Therefore, extensive modelling work was started to find out if this change could be explained by changes in meteorological factors like temperature and precipitation. The daily weather data for the fixed monitoring sites 1983–2002 were obtained from the Finnish Meteorological Institute, which maintains weather stations at these sites. The hypothesis to be modelled was: did the epidemics during the period 1993–2002 start earlier than during 1983–1992, and if so can this change be explained by an increase in precipitation or temperature? The modelling work was fully carried out by MSc. Timo Kaukoranta. Details of the several different regression approaches, including logistic regression, are described in detail in paper I.

2.1.4 Modelling the progress of late blight epidemics

The obvious shift towards earlier epidemics in the monitoring data raised the question as to whether some overall changes in the shape of late blight disease progress curves (DPC) over the period had taken place. The disease progress assessments over seasons 1991–2002 in cv. Bintje were collected from variety trials and unprotected plots in fungicide efficacy trials at MTT, Jokioinen and the Potato Research Institute, Lammi. These trials were carried out using randomised complete block designs with 4 replicates, which allows much more accurate comparison of epidemics than field observations. For DPC comparisons relative area under disease progress curve (rAUDPC) and apparent infection rate (AIR) were calculated for each trial and replicate. Only trials where at least five consecutive disease ratings were made

were included in calculations. Additional epidemiological measurements calculated from the data were number of days from planting to appearance of the first blight lesions, number of days from first lesions to 5% of blighted leaf area and number of days from 5% to 95% defoliation. There were altogether 48 experiments that were included in this comparison, allowing comparison of 192 different DPCs. Details of calculations and statistical analyses are given in paper I.

2.1.5 Monitoring suspected soil derived epidemics

During the first years of the 2000s when increasing indirect indications of soil-borne late blight epidemics had accumulated from Finland, and especially from Sweden (Andersson et al. 1998), special emphasis was placed on examining the possible inoculum sources for the very first late blight attacks of the year. An extensive number of both plant and soil samples were collected and analysed from first late blight foci, especially from the early crop produced under fleece cover. The details of the procedures applied are described in paper II.

In addition, a 9.5 x 36 m² field plot was established in 1999 to get material for studying different aspects of soil-derived epidemics. The field at Jokioinen where the observation plot was established had been used for potato trials since 1983 with only a few occasional seasons without potato. The field had to be abandoned for normal potato late blight experiments due to too heavy and spatially extremely uneven disease outbreaks. This plot provided a considerable part of the soil and plant samples studied in 2000–2002 for paper II, and it was maintained for further studies until 2011.

The infectivity of soil samples obtained from first late blight foci in growers' fields and experimental plots was studied by a

modification of a soil baiting bioassay described by Drenth et al. (1995). Details of modifications are given in paper II.

The occurrence of latent *P. infestans* infections and progress of the infection within one single plant to separate between tuber-derived infection and other inoculum sources was studied using real-time PCR techniques to detect and quantify *P. infestans* DNA in plants. The qPCR procedure was optimised and modified by Dr. Terhi Rantanen on the basis of methods developed by Judelson and Tooley (2000) and Appel et al. (2001). The detailed procedure is described in paper II.

2.2 Collection and characterisation of *P. infestans* isolates

2.2.1 Collection and selection of isolates

P. infestans isolates for this study were collected from potato fields between 1990 and 2010. Due to the long time frame the aims for monitoring and sampling, strategies were changed during the studies. From 1990 to 1996 the main aim was to monitor phenylamide resistance using metalaxyl as indicator fungicide. Samples were predominantly taken at an advanced stage of epidemic (20 to 50% of foliage diseased) and additionally from yield tubers. From 1997 to 2001 a major effort was put into collecting the very first late blight lesions to monitor possible effects of suspected oospore-derived infections on *P. infestans* populations, but a considerable number of isolates were additionally sampled during later stages of epidemics. From 2003 to 2010 the objective of sampling was to get an overall impression of *P. infestans* population on year and country level basis and few isolates per single field from as many fields as possible were sampled before exponential expansion of leaf blight in the crop (disease level 1 to 20% of leaf area affected). The details of sam-

pling procedures and aims for each period are described in papers III, IV and V.

To reduce the obvious sampling error effect on apparent properties of the population only isolates obtained from a single lesion on one leaflet were included in the data used here. From isolates collected between 1990 and 2006, only those taken from crops with less than 30% of blighted leaf area were included. In the period from 1997 to 2001 isolates collected from the very first lesions were excluded. For these reasons the numbers of isolates analysed for different properties shown in Table 4 differ slightly from the corresponding figures shown in papers III and IV. A total number of 4927 isolates collected from 470 fields between 1990 and 2010 were included in this study (Table 4).

The leaflets containing one single late blight lesion were collected from fields individually and placed in small plastic bags or on moist filter paper in Petri dishes and sent to MTT Plant Protection for analyses. Details of the procedure developed during the monitoring period are described in detail in papers III, IV and V. The determinations of R-gene virulence race, response to fungicides and part of mating type determinations in 1997–2001 tested on potato leaf disks were carried out as soon as possible after the arrival of the isolates. Part of the isolates were transferred to rye agar and tested after 2–3 months culturing on agar.

From 1990 to 2001 only a subset of the isolates were transferred on to a modified rye medium and tested later for further studies. From 2003 to 2010 all isolates were further cultured on rye medium. The procedure for culturing field isolates on agar media is described in detail in papers III and IV. The isolates obtained before the 2000s were stored on rye agar in test tubes at 4°C and re-cultured annually. Isolates surviving the storage and new iso-

Table 4. Total number of monitored fields and *P. infestans* isolates collected, and number of isolates included in mating type, MtDNA, R-gene virulence race and fungicide response determinations from 1990 to 2010.

Year	Number of fields monitored	Number of isolates collected	Mating type determination	MtDNA determination	R-gene virulence race determination	Response to metalaxyl	Response to propamocarb-hydrochloride
1990	33	92	0	0	0	92	0
1991	39	418	0	0	0	418	0
1992	18	481	30	9	20	481	19
1993	17	408	9	0	20	408	14
1994	13	206	119	20	156	206	206
1995	5	74	1	0	71	74	74
1996	12	142	16	9	14	142	142
1997	22	602	447	28	66	589	589
1998	45	539	510	31	101	517	517
1999	23	179	169	12	35	134	129
2000	51	765	599	45	0	164	164
2001	12	258	162	0	139	0	0
2003	91	325	218	0	42	257	257
2006	12	105	98	0	104	104	104
2007	26	116	110	0	115	110	110
2008	33	126	126	0	126	125	125
2009	10	54	53	0	54	54	54
2010	8	37	36	0	37	37	37
Total	470	4927	2703	154	1100	3912	2541

lates since 2000 were preserved in a cryo-freezer at approximately -140 °C.

2.2.2 Mating type determination

From 1992 to 2010 the mating types of a total of 2703 *P. infestans* isolates were determined (Table 4). The tester isolates 90209 (A1) and 88055 (A2) were obtained in autumn 1994 from Cyanamid Forschung GmbH, Germany and they were originally isolated from potato in The Netherlands in 1990 and 1988 respectively. The mating type of each isolate was determined by pairing it with tester isolates on rye medium as described in papers III, IV and V. In addition, some of the isolates collected between 1997 and 2002 were analysed by pairing on floating leaf disks to avoid rather laborious isolation for pure cultures on rye agar. The details of the floating leaf disk method are described in paper IV. The presence of oospores was ex-

amined under a compound microscope. The mating type of the isolate under study was determined to be opposite to the mating type of the tester isolate when oospores were formed.

2.2.3 Determination of virulence races

The R-gene virulence race of 1100 isolates (Table 4) was determined by inoculating leaf disks obtained from Black's differential set of R-gene-containing potato clones, incubating leaf disks in moist conditions at 16–18 °C for 7 days and scoring the rate of growth and sporulation as described in papers III, IV and V. Interaction was determined to be compatible if sporangio-phores were visible on at least one of the six leaf disks. A test was repeated if fewer than four leaf disks sporulated on clone R₀ or cv. Bintje. The mean number of virulences per isolate (C_v), virulences per pathotype

(C_p) and normalised Shannon diversity index H_{SR} were calculated using formulas described by Andrivon (1994).

Isolates collected from 1990 to 2001 were tested on a differential set obtained as tubers from The Scottish Agricultural Science Agency (SASA), excluding R-gene 9. Test material was propagated from seed tubers. For the period 2003–2010 meristem cultures of Black's differential set were acquired from SASA. Test plants were propagated in green house from these microplants. The set was tested several times to ensure that plants from tubers and meristem cultures provided consistent results (Andrivon et al. 2011).

To compare the changes in diversity of populations between years the Shannon diversity index was calculated as follows:

$H_s = -\sum_j (p_j * \ln p_j)$, $j = 1 \dots N_p$, where p_j is the frequency of the j th race in the population and N_p is the total number of races in the population.

The bias created for HS by differences in sample sizes between populations was corrected by using relative Shannon diversity index as follows:

$H_{SR} = H_s / \ln N_p$, where N_i is the number of individuals in the population (Andrivon 1994).

The complexity of races in different populations was estimated as C_i =average number of virulence genes per single isolate and C_p =average number of virulence genes per one individual race as reviewed by Andrivon (1994).

$C_i = \sum_j (p_j * v_j)$, $j = 1 \dots N_p$

$C_p = (\sum_j v_j) / N_p$, $j = 1 \dots N_p$, where v_j is the number of virulence genes of j th race and p_j and N_p as described above for the Shannon diversity index.

2.2.4 Assessment of response to systemic fungicides

The response of 3912 isolates to metalaxyl and 2542 isolates to propamocarb-HCl (Table 4) was tested using the floating leaf disk method (Sozzi et al. 1992). Applied fungicide dilutions were for metalaxyl (Ridomil 25 WP, CGA 48988, and since 1999 Novartis experimental compound (metalaxyl-M), CGA 329351) 1, 10 and 100 mg L⁻¹, and for propamocarb-HCl (Previcur N, Batch number 03445549, Schering AG, Germany) 10, 100 and 1000 mg L⁻¹. Dilution series were prepared in distilled water. Sporangia were multiplied on leaves of cv. Bintje and collected in distilled water with a paintbrush. The spore concentration was adjusted to 1x10⁵ sporangia mL⁻¹. Leaf disks were inoculated with a droplet of 20 µL of sporangial suspension in the centre of the leaf disk. Details of the testing procedure are described in papers III, IV, V.

2.2.5 Mitochondrial DNA haplotype determination

Very small numbers of the isolates (154) collected from 1992 to 2000 (Table 4) were studied for their mitochondrial DNA haplotype to get an approximate idea about the possible presence of the old US-1 haplotype 1b in the Finnish *P. infestans* population. MtDNA types were determined by the PCR-RFLP method developed by Griffith and Shaw (1998). Details of the procedure are given in paper IV.

2.3 Sales and estimated usage of different late blight fungicides

2.3.1 Sales of active ingredients

Statistics for annual pesticide sales have been maintained by various authorities in Finland since 1953. Currently TUKES, the Finnish Safety and Chemicals Agency, is responsible for the work. The data for the pesticide sales from 1953 to 1970s were

obtained from databases of MTT Agri-food Research Finland and the Potato Research Institute collected from original annual statistics published by the authorities. From the 1970s to the beginning of the 2000s the statistics for pesticide sales were published in the Finnish journal *Kemia-Kemi* ("Chemistry") and thereafter until 2008 on the internet pages of the Finnish Food Safety Authority, EVIRA. The data for sales of potato late blight fungicides were collected from these sources and statistics for 2009 and 2010 have been provided by and used with the permission of authorities at TUKES.

From 1953 to 1985 only copperoxychloride and dithiocarbamates were used for late blight control and it has been estimated here that 70% of the total sales were used for potato. From 1985 to 2010 the pesticide sales were reported on a commercial product basis and potato late blight products, except straight dithiocarbamates and copperoxychloride, were not used on other crops. For 1985 to 2010 it has been assumed that 70% of dithiocarbamates and copper fungicides were used for potato late blight control. The annual sales have been reported in kg or tonnes of pre-packed products and as kg of active ingredients in the products.

To get an overview of the changes in use of different types of fungicides, the annual sales of active ingredients were classified on the basis of their biochemical mode of action in the pathogen cell. Multi-site protectant fungicides include dithiocarbamates and copper products. Metalaxyl and metalaxyl-M (mefenoxam) are classified as RNA-synthesis inhibitors. Propamocarb-HCl, dimethomorph and mandipropamid were classified as cell membrane assembly disturbing compounds. Fluazinam, cyazofamid, famoxadone and fenamidone were classified as mitochondrial respiration inhibitors, though they belong to separate chemical groups. The rest of the active ingredients registered in Finland, including

cymoxanil and zoxamide, were classified as 'other modes of action' (Table 2).

2.3.2 Estimated potato area treated annually

The fungicide sales in kilograms of active ingredients do not give a representative picture of the magnitude of their use because pre-packed products contain different concentrations of active ingredients. Moreover, application rates of different products range between 0.2 and 4.0 L/ha. Therefore amounts of annually sold pre-packed products were used to estimate how widely the products were used. The amounts of sold products were divided by application rate to estimate how many hectares could have been treated with each product. On-label application rate for each product was used in calculations. For some products on-label application rate is given as a range e.g. 2–4 kg/ha. The average of minimum and maximum application rate (3 kg/ha in this case) was used in calculations. In Finland farmers generally acquire some of the pesticides for the next season already during last months of the previous year. Therefore, two year moving averages of treated hectares were used as estimates for the annually treated area.

Rain-fastness and mobility of fungicidal compounds after application are crucial for the efficacy. It was of interest to examine whether these properties affected fungicide sales. Therefore fungicides were classified as products not absorbed in leaf tissue (straight dithiocarbamates), products absorbed but not translocated after application, products containing components translocated within the leaf and products containing components translocated within leaf and further transported into apical growth. The classification is based on fungicide ratings maintained by the EuroBlight consortium at <http://www.euroblight.net/FungicideComparison.asp?language=UK> and also published in the review of Cooke et al. (2011).

2.3.3 Estimated number of annual applications per potato hectare

To estimate how many applications per season could be theoretically be done with the total amount of late blight fungicides sold, over the total potato crop area, the estimated number of hectares treated was divided by the annual total potato growing area. The official statistics for annual growing areas of crops are maintained by the

Finnish Ministry of Agriculture and Forestry. These statistics are published annually by the Information Centre of the Ministry of Agriculture and Forestry in the 'Yearbook of Farm Statistics'. Yearbooks from 2003 to 2010 are available at <http://www.maataloustilastot.fi/>. The potato areas from 1953 to 2002 were collected from printed versions of these yearbooks, but they are not listed in the references.

3 Results and discussion

3.1 Historical and recent development of late blight epidemics in Finland

3.1.1 Potato late blight incidence and severity from 1845 to 1900

In the period 1845 to 1900 over 1000 newspaper articles, where potato late blight was mentioned, were found from the historical newspaper library maintained by the Finnish National Library. Of these articles 281 reported occurrence of late blight in different parts of Finland and are used in Figure 5. The remaining articles were general descriptions of late blight occurrence in Europe or America, or descriptions of general biology and manage-

ment of the disease. The newspaper articles mentioned here have not been included in the references. The citations in Finnish and Swedish have been translated into English by the author.

Potato late blight obviously reached Finland relatively soon after its migration to Ireland, Great Britain and Continental Europe. Probably the first report on the occurrence of potato late blight in Finland was published on November 15th 1845 in the newspaper Kanawa (No. 45). It was written: "Se kulkewa potaattitauti näkyy uhkaawaan Suomessaki ruweta liikkumaan. Wiipurin luona se jo on monella tilalla pilannut pian koko wuoden tuloksen, ehkei siitä täällä ole oltu paljo milläänkän, siksi kuin täällä potaatin

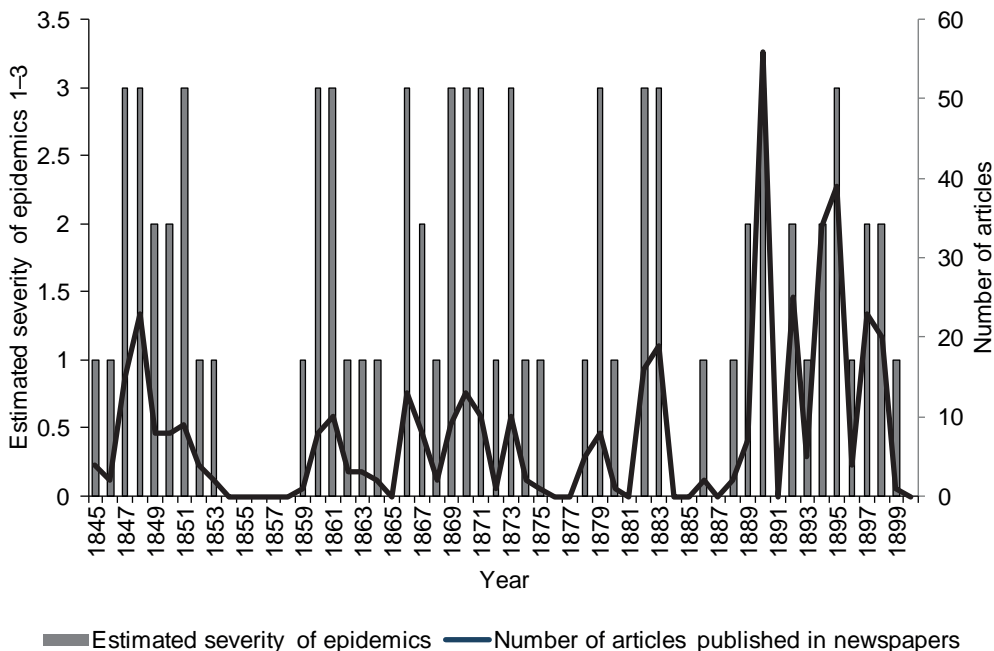


Figure 5. Number of articles published in Finnish newspapers on potato late blight incidence in 1845–1900 and the estimated severity of the epidemic: 0=no articles published, 1=local, 2=regional, 3=nationwide.

wiljelys warsin wähsen wasta on harjotettu". (The potato disease epidemic is threatening to spread in Finland. In Wiipuri region the disease already has spoiled the entire yield on many farms, but the public seems not to be worried because potato is not yet very widely grown here). Thereafter in the article the occurrence and symptoms of the disease are described in detail and this description fits well with potato late blight. Before that Finland's Allmänna Tidning (No. 242, October 18th 1845) had published an article "Några ord om potatis-sjukdomen" (Some words on potato disease), where many properties of the disease described in European media were described in detail. This article probably awakened awareness of the potato disease and helped the audience to recognise late blight symptoms in the field and in storage.

The first comprehensive report on the occurrence of potato late blight in Finland was published in the newspaper Finland's Allmänna Tidning (No. 222) on September 9th in 1847, in Swedish, under the title "Potatis-sjukan i Finland" (Potato disease in Finland). According to the article, in Finland tuber rot at that time was the main concern because due to early frost leaf blight epidemics seldom advanced to a serious stage. It is written in the article: "Potatis-sjukan synes således icke hos oss hafva haft alldeles samma utvecklingsgång, som i andra länder; men resultatet är dock detsamma, nemligen en felslagen skörd och mörka utsigter för de arbetande classerne, som till så betydlig del lifnära sig af potäter". (The potato disease here seems not to have the same development cycle as in other countries; but the result is nevertheless the same, spoiled yield and dark prospects for the working class, whose nutrition is highly dependent on potato). Basically the same article was published in Finnish in the newspaper Suometar (No. 43) October 27th 1847.

According to several newspaper reports, as reviewed by Mäkelä (1966), potato late blight was rather widely present in southern Finland by 1847. In 1848 the disease had spread further to central parts as well as the coastal areas of Ostrobothnia, extending as far as Tornio (66°3' N, 24°22' E). Locally the disease caused marked crop losses in 1847 and 1848. In certain regions one third of the tubers were destroyed and some growers lost their entire crop.

Grotenfelt (1910) over the 61-year period from 1847 to 1908 reported that there were 18 years when late blight caused severe damage throughout the country. At that period blight often caused severe damage in 2 to 3 consecutive years at two to nine years intervals. A similar result can be seen in Figure 5 for the period 1845 to 1900, based on articles written in Finnish newspapers in Finnish or Swedish. After the severe late blight years of 1847 and 1848 (Mäkelä 1966) and 1851 (Suometar No. 41, October 14th 1851), there were only seven articles written on the occurrence of late blight until 1860 (Figure 5).

The years 1860 and 1861 were very severe late blight years as reported in the newspaper Suometar (No. 43, November 2nd 1860) and summarised in the newspaper Suometar (No. 47, November 22nd 1861): "Potaatti-rutto kuuluu uhkaawan maatamme suuremmalla turmiolla kuin koskaan ennen" (Potato late blight threat to our country seems to be more severe than ever before). Thereafter in the article advice to inspect potato storage regularly is given to keep an eye open for tuber rot. There was a serious concern that due to decomposition there would be no seed potato left and therefore it was suggested in the article to select small but healthy tubers and keep them in a dry place to provide seed potato for the next spring.

Famines caused by frost and other crop damage were relatively common in Finland during the first half of the 1800s. The

last large-scale famine in Finland occurred in 1867–1868. These were rather severe blight years, but the main reason for the famine was that grain yields were severely damaged by early frosts (Varis 2001). The severity and consequences of the shortage of potato and grain is described in the article “Apua nälänhätään” (Famine relief) in the newspaper *Tapio* (No. 6, February 8th 1868). Until the 1880s potato in the countryside was mainly grown for cattle feed, but it was very important food for the poorest working class (Finlands *Allmänna Tidning* No. 222, September 9th 1847) and its importance increased towards the end of the 1800s (*Päivälehti* No. 228, October 2nd 1890).

From 1873 to 1890 there was period with only a few severe late blight years (Figure 5). In 1890 potato late blight appeared early in the crop and probably caused more damage than ever before or after. According to newspaper articles the epidemics in 1890 appear to be even worse than reported on the next extremely severe blight year 1953 (Pohjakallio 1954). The appearance of late blight in the potato crop in south-west Finland was reported in the newspaper *Satakunta* (No. 91) August 7th 1890. By the end of August over 40 articles were published in different newspapers reporting late blight incidence all over Finland. Many more reports on decaying potato in storage were given in September and October. The overview of the situation was given for example in the newspaper *Maamme* (No. 124, October 16th 1890): “Yli koko Suomen maan on nimittäin perunarutto suurimmaksi osaksi turmellut perunain tulon. Tosin sanotaan muutamissa osissa maatamme perunain tänä vuonnaakin säilyneen perunarutolta, waan sellaisia paikkakuntia ei saata olla monta” (Over the whole Finland potato late blight has destroyed most of potato yields. However, it is said that some regions have been spared from blight though these sites cannot be many). Similar reports were given in paper *Oulun Ilmoituslehti* (No. 91, October

31st) in an article “Mitä nyt on tehtävä kun perunat pahenewat? (What to do now when potatoes are decaying?).

In the last decade of 1800s late blight was locally present every year, but only year 1895 seems to have been a severe blight year. Late blight was commonly present already on the first half of August. The season was favourable for all crops and good potato yields were expected. Tuber blight however caused harm, as stated in the newspaper *Aamulehti* (No. 217, September 19th 1895): ”Perunoita on jo aljettu ottaa ja tulis niitäkin hywin, mutta perunaruton pahentamia on paljon” (Potato harvesting has been started and yields would be otherwise good, but plenty of tuber blight is present).

Potato late blight from 1845 to 1900, based on newspaper articles, usually started at the end of August or beginning of September and the most serious consequence was decay of tubers during storage. One of the earliest outbreaks in the 1800s is reported in the newspaper *Keski-Suomi* (No. 31) on August 5th 1871 at Jyväskylä, in Central Finland: “Yleisen märkyyden tähden sawimaille on potaatti-ruttokin ilmaantunut” (Due to overall wetness potato late blight has appeared on clay soils). Also in 1890 blight started at several sites during the first half of August, as mentioned in the previous section. In 1895 late blight was reported in Central Finland on August 7th (Suomalainen No. 89). In 1870 late blight was reported in eastern parts of Finland on August 13th (*Tapio* No. 32): “Potaatti-tautia mainitaan jo ilmestyneen näillä tienoilla muutamissa wesi-itkuisissa maalaaduissa” (Potato disease is already mentioned to appear in these region on certain soil types suffering from wetness). Also in 1864 newspaper *Tapio* (No. 33) on August 13th states from eastern Finland: “Potaatin warsissa on jo walitettawasti nähty ruosteen merkkiä” (Unfortunately signs of potato late blight have already been seen on potato stems).

3.1.2 Knowledge on the cause of potato late blight in Finland prior to 1900

In the 1800s there were no actual research activities on potato late blight in Finland. The pathogen occurring in diseased potatoes in Finland was scientifically soundly identified as *P. infestans* in 1873 by Karsten (1873). In the 1800s scientific studies published elsewhere in Europe were referred to and the “fungal theory” seems to have been readily accepted in Finnish newspapers.

The newspaper Finlands Allmänna Tidning (No. 242, October 18th 1845) refers to an article published in a Swedish newspaper, Svenska Post- och Inrikes Tidningarne (No. 234, October 9th 1845), on the cause of potato late blight. It was written: “Den verkliga orsaken till detta onda, säger Hr Morren (Professor i jordbruksvetenskapen vid universitet i Lüttich), är en svamp eller mögel, som de lärde räkna till arten af Botrydes (svamp-klassen) men som landtmännen icke anse för annat än ett slags Brand, och som de tillskriva än för mycken väta, än för mycken torka, än en elak vind, än insekter” (The real reason for this evil, says Mr Morren (Professor of agricultural sciences at the University of Lüttich), is a fungus or mould, which is classified as a species of Botrydes (fungal class), but which farmers only see as a kind of blight, and which is described to be spread by too much moisture, too much drought, evil winds and insects). A similar article was published in Finnish in the newspaper Maamiehen Ystävä (No. 6, February 7th 1846).

Further progress in research on the causal agent of potato late blight in Europe was reported in the newspaper Suometar (No. 42) October 26th 1860. It was written: “Syy potaatti-ruttoon on luultu löydettyksi olewan yksi ruokalais-mato (parasit-mask), jota latinaksi toiset kutsuvat Botrytis infestans, toiset Perenospora in-

festans. Mainitun ruton syytä on Ranskassa oppineet tutkineet sitte w. 1846, jolloin määrättiin erinäinen tutkijakunta tätä warthen” (The cause of potato blight is believed to be a parasite called *Botrytis infestans* or *Perenospora infestans* [the name is written as it appears in the article]. In France scholars have studied the cause of blight since 1847, when a specific research group was appointed for the purpose). Basically the same news was disseminated later in the 1860s in several other newspapers.

Later the newspaper Päivätär (No. 2) January 14th 1865, in an article “Potaatin viljely” (Potato production), reported on the new findings of de Bary: “Kolme luonnontutkijata de Bary, Speerschneider ja Hoffman todeksi näyttiwät, että potaattitauti on mahia (*Peronospera infestans*), jonka siemenidut tunkeutuwat potaattitaimenen terweisiin osiin, ja näiden häwiöllä kehtyen matkaansaattawat taudillisen tilan” (Three scientists, de Bary, Speerschneider ja Hoffman proved that the potato disease is a parasite (*Peronospera infestans*) [the name is written as it appears in the article], whose germ tubes enter into healthy parts of potato plants and induce the disease). Thereafter in the article the life cycle and epidemiology are described in detail. It is further highlighted: “De Bary on selwästi toteen näyttänyt, että mahi on taudin syy eikä päinwastoin. Joku aika luultiin mahin niin kuin muutamien muidenkin sienikaswaksien ilmaantuen wasta sitte, kuin tauti jo on päässyt wälälle.” (De Bary has clearly shown that the parasite is the cause of the disease and not vice versa. Some time ago it was thought that the parasite was some other fungus, appearing only after the plant had already become diseased).

The progress of de Bary’s studies was further reported in the newspaper Päivälehti (No. 210, September 11th 1891) “Saksalainien tiedemies Anton de Bary wuonna 1861 tekemiensä wiljelyskokeiden kautta todis-

ti että perunaruttoon on syynä pieni lois-sieni. Tälle sienelle, joka jo oli kerennyt saada montakin nimeä, antoi Anton de Bary vuonna 1876 nimeksi *Futophthora infestans*, joka juuri merkitsee tuon sienen perunaa tuhoavaa vaikutusta” (German scientist Anton de Bary, based on experiments carried out in 1861, proved that the cause of potato late blight is a small parasitic fungus. For this fungus, that already had been given several names, Anton de Bary gave name *Futophthora infestans* [the name is written as it appears in the article], which means destructive effect of the fungus on potato). In this article the morphological properties of the pathogen and how to use a microscope to identify it are also described in detail.

3.1.3 Potato late blight from 1901 to the 1980s

In the early 1900s the blight occurrence in Finland must have been rather similar to that in the 1800s. According to Grotenfelt (1910), from 1901 to 1908 blight was present every year, but there were only two years when late blight had severe consequences. Liro (1917), in the first textbook on plant pathology in Finnish, describes *P. infestans* and blight disease as one of the most dangerous potato diseases, but he does not give any detailed description of the prevalence of the disease. Jamalainen (1933) and Rainio (1937) described one severe blight epidemic per decade in the 1920s and 1930s, but only a single year when no blight was present. Yllö (1963) described 8 severe blight years, observed in variety trials in Tikkurila in southern Finland from 1931 to 1958. In his studies an average of 85% of the leaf area was affected in most susceptible varieties in severe blight years. This is quite different from the current situation, as shown in Figure 8.

According to Seppänen (1971) in the period 1931–1962 leaf blight was present on average in four out of five years and one year in three was a severe blight year

in southern Finland. He defined a severe blight year as being if blight started by August 20th and the haulm was destroyed within 2–3 weeks. Beyond 63° N the severity of leaf blight was considerably reduced, primarily due to more frequent occurrence of frost and weather conditions less favourable to blight.

From the 1960s to the beginning of the 1980s there is very fragmented information on blight onset and incidence in Finland, but according to Seppänen (personal communication) the situation was very similar to that reported by Seppänen in 1971. Because late blight incidence had been stable for such a long time, and new effective fungicides were registered for late blight control, it was justified that Professor Seppänen (1987) shared the common view among other potato experts: ‘Potato late blight was the most important disease until the 1970s in Finland, but now the problem is overcome for ever and we do not have to worry about late blight any more’.

3.1.4 Monitoring the late blight outbreaks from 1983 to 2011

During the period 1931–1962, which is used as reference, no obvious changes in dates of first late blight outbreaks are evident as reconstructed from data published by Seppänen (1971). Late blight usually appeared in the crop during the latter half of August or the beginning of September (Figure 6). During that period there were only three years, 1934, 1935 and 1938, when late blight was present in the fields at the end of July. These were exceptionally warm seasons (Seppänen 1971).

From 1983 to 1987 there was no change in the dates of first late blight outbreaks in comparison with the period 1931–1962. Between 1988 and 1996 late blight appeared in the fields at the end of July and beginning of August (Figure 6). In 1997–1998 there was a sudden change to

ward earlier epidemics and in the early 2000s the first late blight symptoms were found normally during the last week of June. Since 2005 late blight has usually appeared in potato fields during the first week of July. Early appearance of blight in the crop does not necessarily indicate early epidemic progress. For example, in 1999 late blight was detected at the end of June and in 2010 at the beginning of July (Figure 6), but later in the season the weather became unfavourable for late blight devel-

opment and at the end of season there was only a very slight blight attack on the potato crop (data not shown).

For the period 1983–2002 various regression equations can be calculated to predict the dates of first late blight outbreaks. The scatter plot in Figure 6 can also be interpreted as there being a regular trend towards earlier epidemics from 1983 to 1996. In 1997–1998 there was a very sudden shift towards early starting epidemics

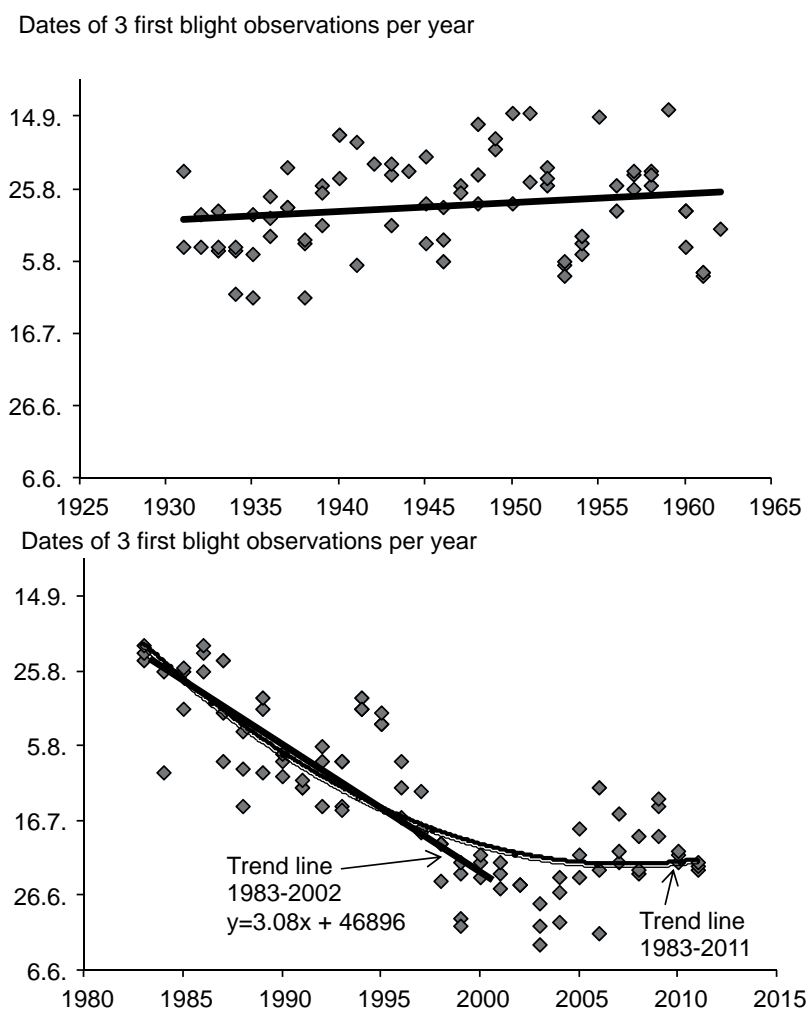


Figure 6. Dates of three earliest late blight observations in Finland annually from 1931 to 1962 (data provided by Seppänen 1971) and from 1983 to 2011. Regression for period 1983–2002 is presented as in paper I.

and from thereon the onset of epidemics stabilised around first week of July.

The observed systematic shift towards earlier epidemics in Finland seems to be unique in Europe. Exceptionally early late blight outbreaks were frequently observed in southern Sweden in the late 1980s and early 1990s, but they were restricted to specific early potato production systems (Andersson 2007). Schepers (2004) collected statistics on the dates of first late blight outbreaks in most European countries in the years 1999–2003. There was considerable temporal variation in dates of blight onset and also individual very early outbreaks occurred, but no trend towards earlier starting epidemics in any country was noted. Zwankhuizen and Zadoks (2002) studied progress of annual late blight epidemics from 1950–1996 in the Netherlands. They found high seasonal variation in disease intensity, but no data are presented on the dates of onset of the late blight epidemics. Croxall and Smith (1976) studied disease progress curves of potato late blight from 1923 to 1974 in the East Midlands, UK. The shapes of disease progress curves varied greatly from year to year, but no regular change in dates of disease outbreak were recorded during that period. Also Kolbe (1982) studied progress of potato late blight in Bavaria, Germany between 1943 and 1982, and no systematic changes in onset of blight epidemics were established.

It can be argued that the less frequent monitoring practice in the period 1983–1991 than from 1992 onwards explains the observed results. The data for the period 1983–1991 were obtained from variety and fungicide efficacy trials, where detecting the very first late blight symptoms was not the main objective. Moreover, the trials were monitored only once or twice a week. From 1992 onwards the monitoring was specifically aimed at determining the first date of late blight outbreak and observation plots were monitored almost daily.

Theoretically this could contribute approximately a maximum of 5 days to delay in detecting the first symptoms in 1983–1991 in comparison with the period from 1992 onwards, while the observed change was in the range of 30–40 days. In addition, there are records of relatively early outbreaks in 1983–1991 and late outbreaks since 1992.

3.1.5 Modelling weather as a possible cause for the shift to early outbreaks

From the monitoring data (Figure 6) it is apparent that late blight at the end of the period 1983–2002 appeared in the crop earlier than at the beginning of the period. Temperature and humidity are known to be driving forces for *P. infestans* infection and epidemic development (Crosier 1934). Therefore, it was reasonable to study whether a change in some climatic factor affecting infection and symptom development could explain the shift towards earlier late blight outbreaks.

The details of the relatively complex modelling process are given in paper I, and only the main issues behind the final model and conclusions are described here. The prediction was based on daily mean temperature and precipitation because daily duration of humid periods, the basis of all modern blight models (Fry et al. 1983), was not available for the monitoring sites at that time. The parameter to be modelled was number of days after potato planting to observation of the first late blight symptoms (DAP).

The daily weather data for 1983–1992 were used to estimate regression parameters for predicting date of late blight onset DAP and the developed regression model was applied to examine how well these parameters could predict blight onset during 1993–2002. Tested regression parameters consisted of different temperature–precipitation regimes during the 40-day period after potato planting. For these data none

of the linear regression approaches were satisfactory in predicting the date of late blight outbreak.

Finally, logistic regression was used to secure at least a qualitative prediction. This means that it was only possible to predict whether the late blight epidemic was starting early or late. The epidemic was defined as “early” if late blight lesions were found 77 DAP or earlier. The epidemic was defined as “late” if late blight was found later than 77 days after potato planting. The model that best assigned the cases to the correct “earliness” category and was biologically meaningful was Logit (P) = intercept + B' [T₃ T₃P₃ T₃²P₃ T₄P₄ T₄²P₄], where T₃ and T₄ are the mean temperatures and P₃ and P₄ number of rainy days during 21 to 30 and 31 to 40 DAP respectively (paper I).

In the period 1992–2002, both the observed and predicted early potato late blight outbreaks were more common than

during 1983–1991. Since 1997 late blight appeared early in the crop much more often than predicted (Figure 7), suggesting that additional factors besides temperature and precipitation affected the infection and late blight onset. Many observations and studies presented later in this thesis provide at least circumstantial evidence that sexual reproduction and oospores as a new source of primary inoculum can explain the observed shift towards earlier late blight outbreaks.

3.1.6 Modelling progress of late blight epidemics

The disease progress curves (DPC) for Lammi and Jokioinen between 1991 and 2002 were of relatively uniform shape (Figure 8). The exponential phase of DPC at Jokioinen was clearly earlier in the epidemics assessed in 1996 or subsequently than before 1996. At Lammi there was more deviation, but also there the exponential phase was earlier in epidemics an-

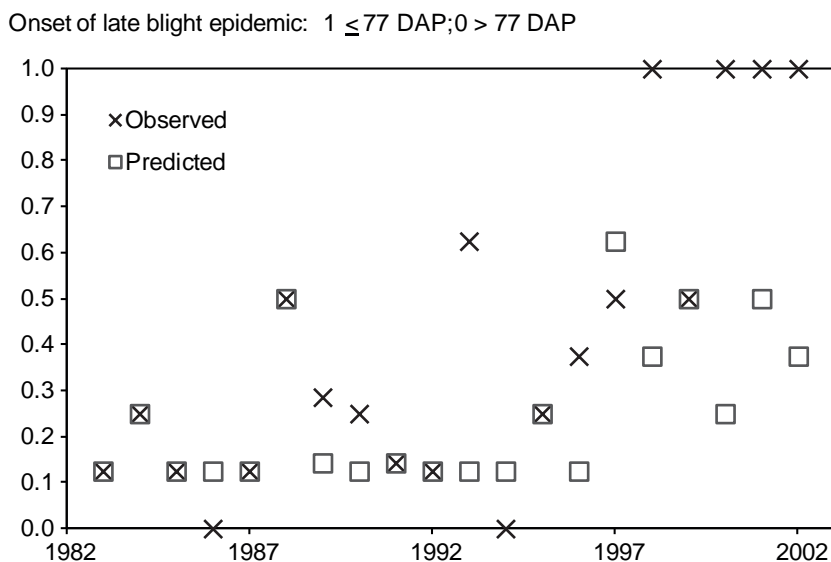


Figure 7. Proportion of potato late blight outbreaks detected early (< 77 days after planting, DAP) in all monitored fields as observed and predicted by a logistic regression model based on parameters calculated from daily temperatures and numbers of rainy days during the period 40 DAP.

alysed in 1998–2002 than in other years (Figure 8). The graphics of DPCs indicate a general tendency towards earlier starting epidemics from 1991 to 2002, but prevailing seasonal weather conditions in particular probably determine the onset of the exponential phase of epidemic development.

The monitoring period was divided into two parts, 1991–1995 and 1996–2002, for statistical comparison of some components of the epidemics. Pair-wise comparisons where other years were compared to 2002 also were carried out. The major change observed in epidemic profiles

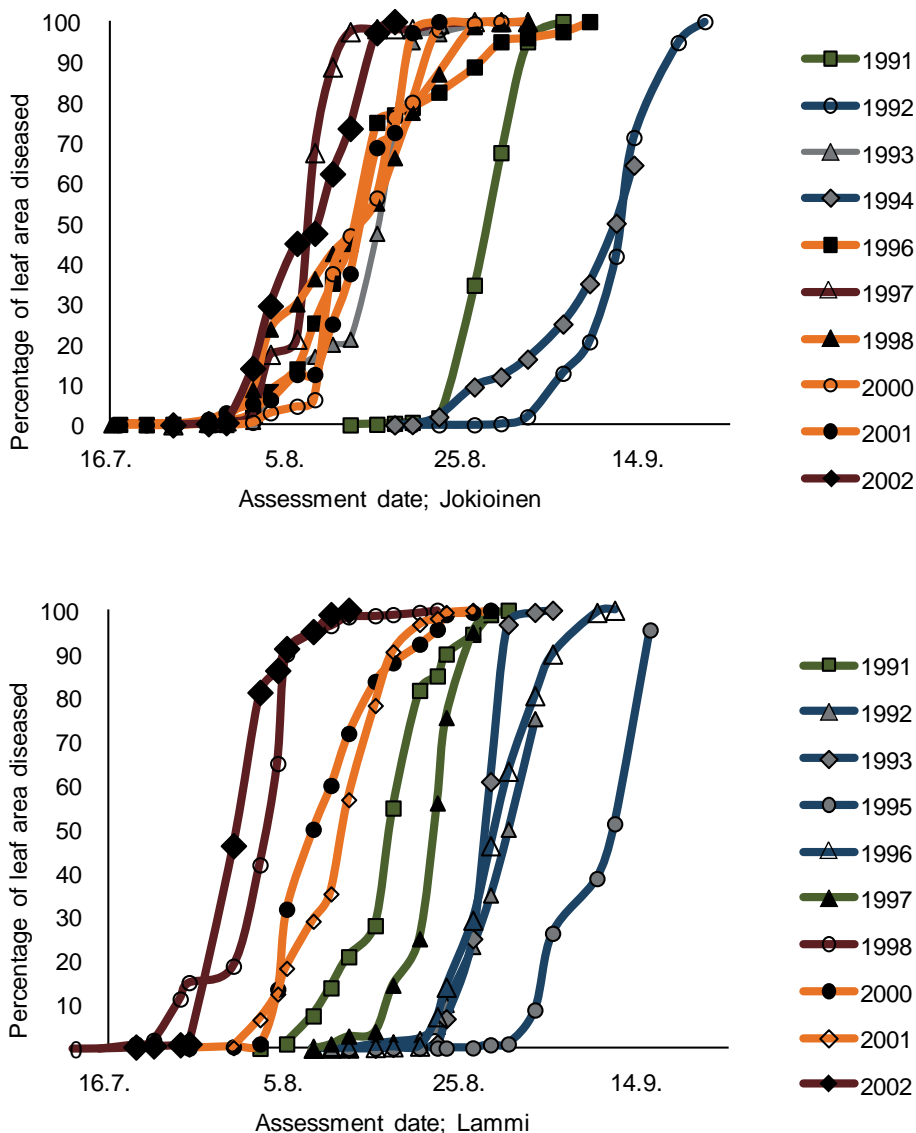


Figure 8. Average annual disease progress curves based on percentage of diseased leaf area at Jokioinen and Lammi in 1991–2002. In 1994 and 1999 at Lammi, and in 1995 and 1999 at Jokioinen, DPC is not shown due to very low disease severity.

was that in 1996–2002 the first blight lesions were found on average 15 days earlier than in the 1991–1995 period (Table 5). In all years from 1991 to 1995, except 1993, blight was found statistically significantly later than in 2002 (p values <0.0001). In this respect, the years 1996–2001 did not however differ from 2002 (p values 0.036–0.808).

The progress of the disease from first lesions to 5% diseased leaf area in both periods on average took 8–9 days (Table 5). This indicates that no change in multiplication capacity of the pathogen had taken place at the linear phase of epidemic progress. After the disease severity reaches the 5% level exponential expansion of the disease usually starts. Due to the overall earlier disease outbreak in 1996–2002, in comparison with 1991–1995, also the onset of the exponential phase of the epidemics began 13–14 days earlier.

Also the duration of the exponential phase (5–95% diseased leaf area) of the epidemics was similar throughout the period 1991–2002, namely 13–14 days (Table 5). This indicates that the aggressiveness of *P. infestans* during this monitoring period had not changed. The unaltered aggressiveness of the pathogen is further supported by the fact that relative area under the disease progress curve and apparent infection

rates were similar from one year to another during the monitoring period. The average final disease rating from 1991 to 1995 was 70% and 99% from 1996 to 2002. This indicates that during 1991–1995, when epidemics started late, there was not enough time before harvest for the disease to reach the 100% level.

Aggressiveness components, infection efficacy, duration of latent period, lesion growth rate and sporulation capacity, of the Nordic *P. infestans* population were studied comparing 25 single lesion isolates from each of four countries; Denmark, Finland, Norway and Sweden (Lehtinen et al. 2009). Estimates obtained for some of the components, especially duration of latent period, differed markedly from those used in standard late blight forecast and simulation models (Crosier 1934, Fry et al. 1983, Hermansen and Amundsen 2003). There were no statistically significant differences in the aggressiveness components of *P. infestans* isolates among the Nordic countries, but within each country variation among isolates was considerable. Comparable studies had not been carried out in Nordic countries before and therefore nothing can be concluded on the possible change in aggressiveness components over time (Lehtinen et al. 2009, Cooke et al. 2011).

Table 5. Statistical comparison of some components of epidemic profiles in 1991-95 and 1996-2002 at Lammi and Jokioinen.

Epidemiological measurement	Monitoring period		Estimate for difference b-a	p-value for b-a	Number of trials
	1991–1995 (a)	1996–2002 (b)			
Days from planting to first symptoms	81	66	-15	<0.0001	48
Days from first symptoms to 5 % blighted leaf area	9	8	-1	0.3269	45
Days from 5 % to 95 % blighted leaf area	13	14	1	0.6388	37
Relative area under disease progress curve (rAUDPC)	0.50	0.53	0.03	0.2928	35
Apparent infection rate (AIR)	0.46	0.45	-0.01	0.8774	45
Final disease rating (%)	70	99	29	0.0007	48

In the Netherlands increased aggressiveness of *P. infestans* has been reported, resulting in shorter life cycles, shorter infection period, greater temperature range and increased occurrence of stem blight (Flier and Turkensteen 1999). As a consequence, shorter spray intervals have been adopted than for the 1990s (Cooke et al. 2011). Also increased aggressiveness of tuber blight has been shown (Flier et al. 1998). In the UK and Ireland increased use of late blight fungicides and the recent appearance and rapid spread of completely new genotypes indicate changes in aggressiveness (Cooke et al. 2011).

3.2 Sexual reproduction and diversity in Finnish *P. infestans* population

3.2.1 Mating type ratio and oospore production

Mating type studies were initiated in 1997, when also the mating types of isolates collected in 1992–1996 were determined. Both mating types A1 and A2 were de-

tected among *P. infestans* isolates collected in 1992 (Figure 9). Unfortunately, accurate time of migration of the population containing mating type A2 into Finland cannot be estimated because isolates collected before 1992 are not available.

From 1992 to 1998 the proportion of A2 mating type in the *P. infestans* population remained steady at between 20–30%. Annual variations can be explained by sampling error and small sample size in some years. A considerable number of isolates produced oospores with both A1 and A2 tester strains, indicated as the A12 mating type (Figure 9). These are probably mixtures of isolates due to sampling procedure. Single lesion leaflets were originally collected from fields, but this does not rule out the occurrence of latent infections in collected leaflets. The leaflets were collected in plastic bags and during mailing the lesions had often expanded, covering the whole leaflet. On arrival of the leaflet in Jokioinen it was sometimes impossible to locate accurately the initial lesion.

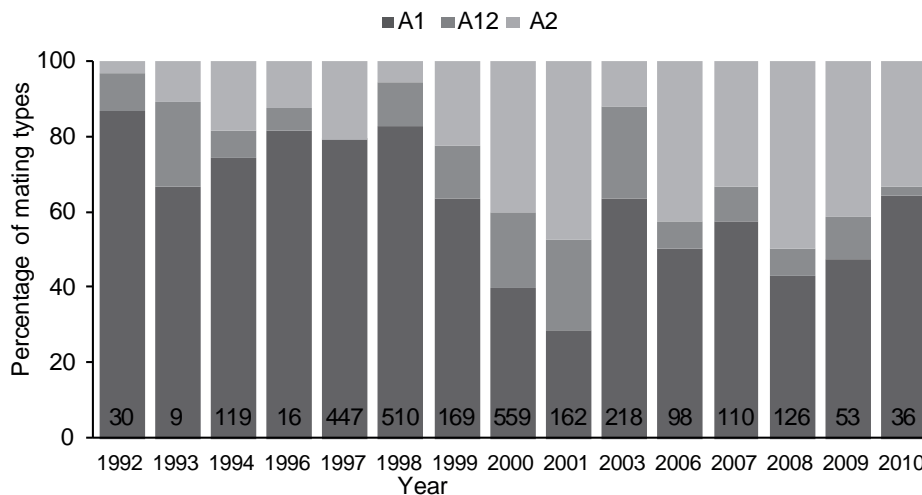


Figure 9. Occurrence of mating types A1 and A2 of *P. infestans* in 1992–2010 in Finland. Numbers within the bars indicate the number of isolates for which mating type was determined each year. A12 mating types produced oospores in pairings with both tester isolates. They are most probably mixtures of isolates rather than true self fertile isolates.

In 1999 the proportion of A2 (especially if A12 mating types are interpreted as containing an A2 component) increased to almost 40% in the population, and thereafter from 2000 to 2010 the proportion of both mating types fluctuated roughly between 40% and 60%. The general shift towards close to a 1:1 A1:A2 mating type ratio seems to take place some years later than the shift towards earlier outbreaks of potato late blight since 1997 (Figure 9). However, since 1997 both mating types have been present in all fields where the earliest late blight outbreaks have been reported and more than two isolates have been determined for mating type.

The frequency of both mating types at the population level (Figure 9) and frequency of fields where both mating types are present (Figure 10) suggests that sexual reproduction in potato fields is possible and

even probable. In paper II at least indirect evidence for sexual reproduction in potato fields was shown: oospores could be found (microscopically) in plants collected from potato fields and potato leaflets showing symptoms (Figure 11) produced numerous oospores after a few days of incubation in moist conditions (Figure 3d).

Accurate direct proof for sexual reproduction of *P. infestans* under field conditions is very hard to get. It is generally accepted that the new population that migrated pan-globally from Mexico in the 1980s and 1990s (Shaw et al. 1985, Spielman et al. 1991, Fry et al. 1993, Andrivon et al. 1994, Goodwin et al. 1994, Fry and Goodwin 1997, Knapova and Gisi 2002, Cooke et al. 2011) is sexually reproducing and capable of producing oospores in the potato crop.

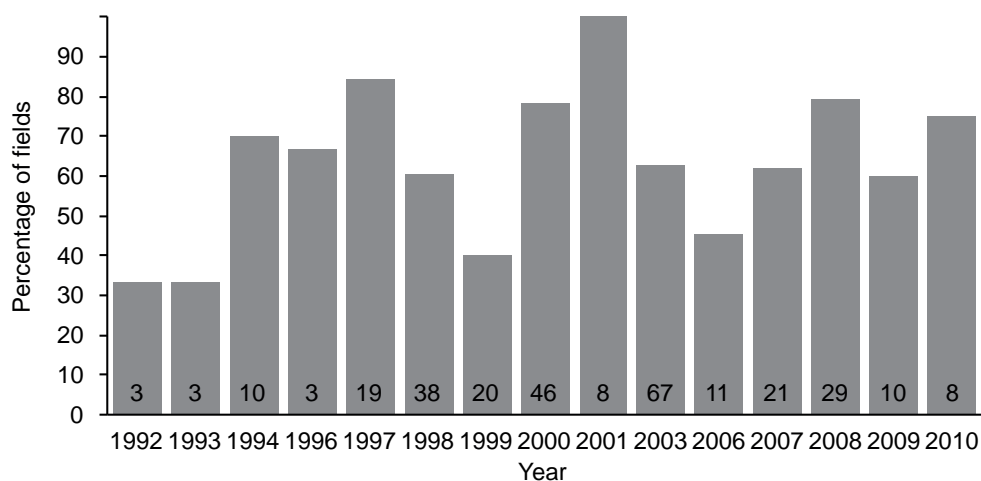


Figure 10. Proportion of fields where both mating types A1 and A2 of *P. infestans* were detected in 1992–2010 in Finland. Numbers within the bars indicate the number of fields where two or more isolates for mating type were determined. In most fields only 3–6 isolates were analysed. Detection of both mating types among very few isolates indicates high frequency of both mating types in these fields.

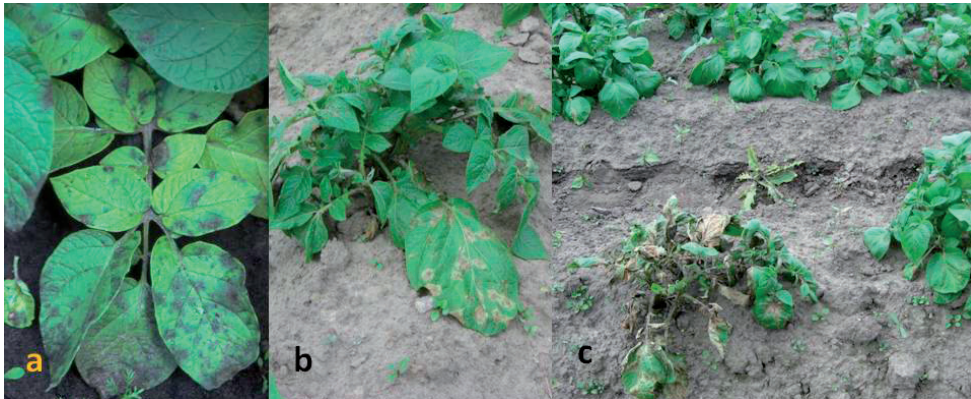


Figure 11. Potato late blight symptoms associated with suspected soilborne infection. 10a. Mosaic-like atypical late blight symptoms, often found on the lowest potato leaf touching the ground after heavy rains in fields after several years of consecutive potato growing. These leaflets usually contain numerous oospores after a few days of incubation in wet conditions. 10b. More typical late blight lesions on lowest leaflets in fields with similar cropping history as for Figures 10a. and 10c. Severe late blight damage to potato seedling two weeks after emergence in potato monoculture. Seed tubers were symptomless and no *P. infestans* DNA was detected in them using real-time PCR-assay. Photos Asko Hannukkala.

The role of oospores as a primary source of infection was studied and discussed rather intensively among *P. infestans* researchers in the 1990s and 2000s. It has been shown that oospores are capable of surviving in the soil for some years and infecting following potato crops (Pittis and Shattock 1994, Andrivon 1995, Drenth et al. 1995, Andersson et al. 1998). In our own studies (paper II) *P. infestans* was able to be frequently isolated from potato leaflets of plants incubated in soils suspected of containing soil-borne inoculum. To remove all other types of propagule other than oospores, soil samples were repeatedly subjected to cycles of freezing, wetting and drying.

Typical circular late blight lesions (Figure 11b) often appear on the lowest leaves touching the ground a few days after heavy rains in connection with suspected soil-borne epidemics. Often mosaic-like symptoms (Figure 11a) appear in the lowest leaves. Also young plants can be almost completely destroyed by *P. infestans* soon after emergence (Figure 11c). These types of symptoms were first reported from Sweden in 1996 (Andersson et al. 1998)

and later in several fleece-protected early potato crops in southwest Sweden (Widmark et al. 2007). In Finland new types of symptoms were first detected in the early 2000s (paper II) and thereafter they have become relatively common in cases of early outbreaks of late blight (Hannukkala, unpublished). Similar early atypical symptoms were found in Denmark (Bent Nielsen, Jens G. Hansen, personal communication) and Estonia (Eve Runno-Paurson, Mati Koppel, personal communication), but not in Norway (Arne Hermansen, personal communication), the United Kingdom (Louise Cooke, David Cooke, David Shaw, personal communication), Ireland (Lesley Dowley, personal communication), The Netherlands (Huub Schepers, Geert Kessel, personal communication), France (Didier Andrivon, Serge Duvachell, personal communication), Germany (Michael Zellner, personal communication) and Poland (Jozefa Kapsa, Renata Lebecka, personal communication).

The symptoms shown in Figure 11a, b and c could theoretically be initiated by sporangia and zoospores produced on infected seed tubers (Schlenzig et al. 1999). In our

studies, seed tubers were symptomless and no *P. infestans* DNA was detected in seed tubers or in stem bases below ground or on upper parts of stems and leaves, while DNA concentrations were originally very high in leaves touching the ground (Rantanen et al. 2002, paper II). In studies of Appel et al. (2001), DNA was detected in all lower parts of the stem when blight-infected seed was used.

Our findings (paper II) suggest that in Finnish potato production oospores and soil-derived epidemics play a significant role and greatly affect the observed early outbreaks of late blight epidemics. This is enhanced in typically narrow crop rotations in Finnish potato production. In paper I it is shown that late blight appears in the potato crop on average 9 days earlier if the preceding crop is potato rather than a break crop. Similar results were obtained in other Nordic countries and it is concluded in the review by Cooke et al. (2011) that in the Nordic climate oospores latently infected seed potatoes are the main source of primary infection. Elsewhere in

Europe the role of oospores is not very significant and seed, volunteer plants and waste piles are the major source of primary inoculum.

3.2.2 Diversity and complexity of R-gene virulence races

All eleven virulence factors against Black's differential set of eleven R-genes have been detected in Finnish *P. infestans* populations. Almost all current *P. infestans* isolates in Finland are compatible with R-genes 1, 3, 4, 7, 10 and 11. Frequencies of virulence factors 7 and 10 in the population in particular have considerably increased since the first half of the 1990s (Figure 12). Virulence factors 2, 5, 6, and 8 in recent studies have been detected in 10–20% of isolates and the incidence of virulence 2, 5 and 6 has significantly increased since the 1990s. Virulence 9 has been found only a few times since 2006, but the potato clone containing R-gene 9 was included in the test set only since 2003.

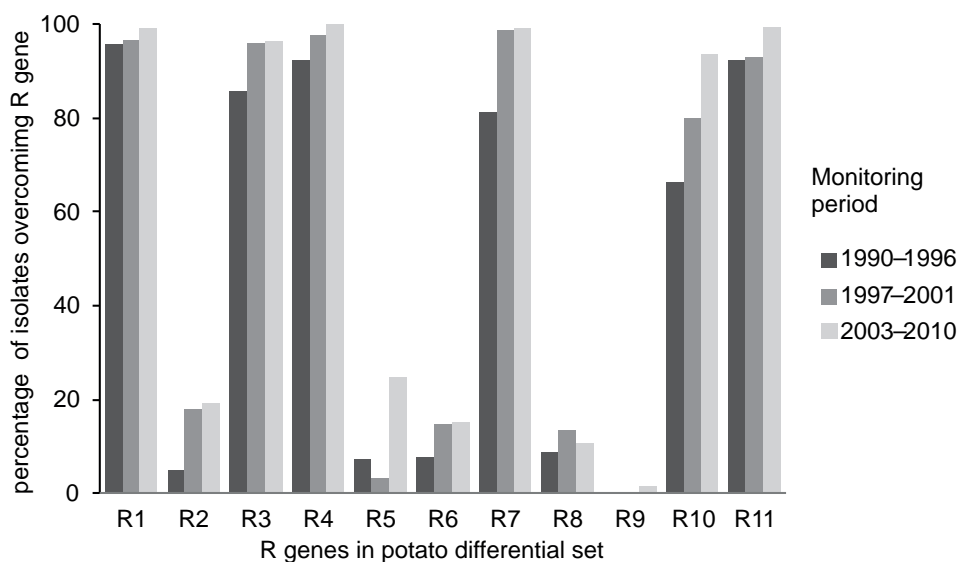


Figure 12. Percentage of *P. infestans* isolates compatible with R-genes 1–11 in Black's differential set of potato clones in 1990–2010. R-gene 9 was included in the set only during the period 2003–2010.

There is no obvious biological explanation for the increase in virulence factors in the *P. infestans* population. Potato varieties cultivated on a large-scale in Finland do not contain R-genes and few varieties ever grown during the monitoring period contain R-genes 1, 3 or 10. The origin of the potato differential set used for the studies differed between 1990–2001 and 2003–2010, which could affect the results (Stewart 1990, Andrivon 1994, Andrivon et al. 2011). However, the difference in overall virulence factor composition was much greater between 1990–1996 and 1997–2001, when the same differential set was used, whereas between 1997–2001 and 2003–2010 a new differential set and a new propagation approach was employed (Figure 12).

The frequencies of different virulence factors are very similar in the Nordic countries, as reported in papers III and V and confirmed in a more recent survey carried out in 2008 (Asko Hannukkala, Björn Andersson, Arne Hermansen and Bent Niels-

en, unpublished). The frequencies of virulences 2, 5, 6, 8 and 9 in Finnish and Nordic *P. infestans* populations are much lower than reported elsewhere (Andrivon et al. 1994, Peters et al. 1998, Elansky et al. 2001, Knapova and Gisi 2002, Śliwka et al. 2006, Runno-Paurson et al. 2009), but also the testing protocols have differed between publications. According to a recently published ring test with uniform testing protocol among 12 European laboratories, including MTT, Finland (Andrivon et al. 2011), all laboratories were in good agreement in determining virulences 1, 3, 4, 7 and 11, but less consistent regarding results in determination of other virulence factors for some *P. infestans* isolates.

The spectrum of virulence races in the Finnish *P. infestans* population seems to have evolved in two directions. The overall population has become less diverse and the individual virulence races more complex from the early 1990s to the 2000s (Figure 13). The decrease in diversity can be explained by the fact that in 1990–1996 over

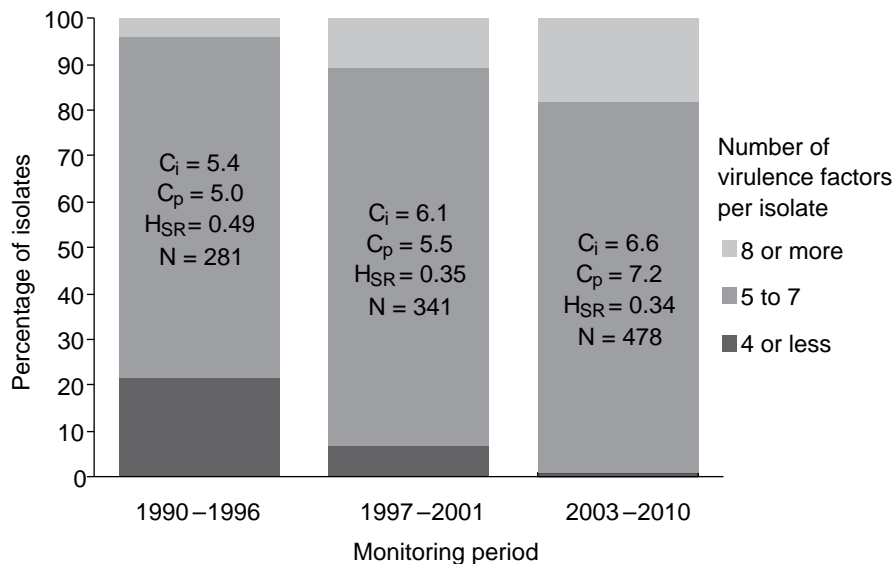


Figure 13. Percentage of *P. infestans* isolates containing 4 or less, 5 to seven and eight or more virulence factors in 1990–2010 in Finland. C_i is the average number of virulences per isolate, C_p the average number of virulences per individual race (pathotype), H_{SR} Shannon's normalised diversity index and N the total number of isolates analysed during each time period. R-gene 9 was included in the differential set only during the period 2003–2010.

13% of the population consisted of unique races lacking one or two of the virulences 3, 4, 7 or 10 and 50% of the individuals in the population (Normalised Shannon diversity index $H_{SR}=0.49$) were of a different virulence race. By the 2000s the proportion of unique races had decreased to 4–5% and almost all isolates had acquired virulences 3, 4, 7, and 10, in addition to 1 and 11 (Figure 12). Only one third of individuals represented different virulence races ($H_{SR}=0.35-0.34$). Purely mathematically the situation greatly reduces the theoretical number of virulence combinations in comparison with a situation where the proportion of each individual virulence factor would be more even. The connection was clearly shown by Andrivon (1994) in comparisons of C_p , C_p and H_{SR} values calculated for several *P. infestans* populations collected and reported by various authors in different parts of the world between the 1960s and early 1990s.

Simultaneously the complexity of races increased, both measured as average num-

ber of virulence factors in each isolate (C_i) and in each separate virulence race (C_p). It is also clear (Figure 13) that isolates containing 4 or fewer virulence factors disappeared and were displaced by isolates containing 8 or more virulence genes from the 1990s to the 2000s. During the period 2003–2010 a few races containing all virulence factors, one to eleven, were also detected (data not shown).

One single virulence race (1.3.4.7.10.11) has been fully dominant throughout the monitoring period, comprising almost half the *P. infestans* population (Figure 14). The most frequently detected races increased in their prevalence from the 1990s to the 2000s and seem to have accumulated additional virulence factors. The incidence of a race lacking virulence 10 decreased during the period, indicating that some of the most complex 'new' races have been very successful in the *P. infestans* population.

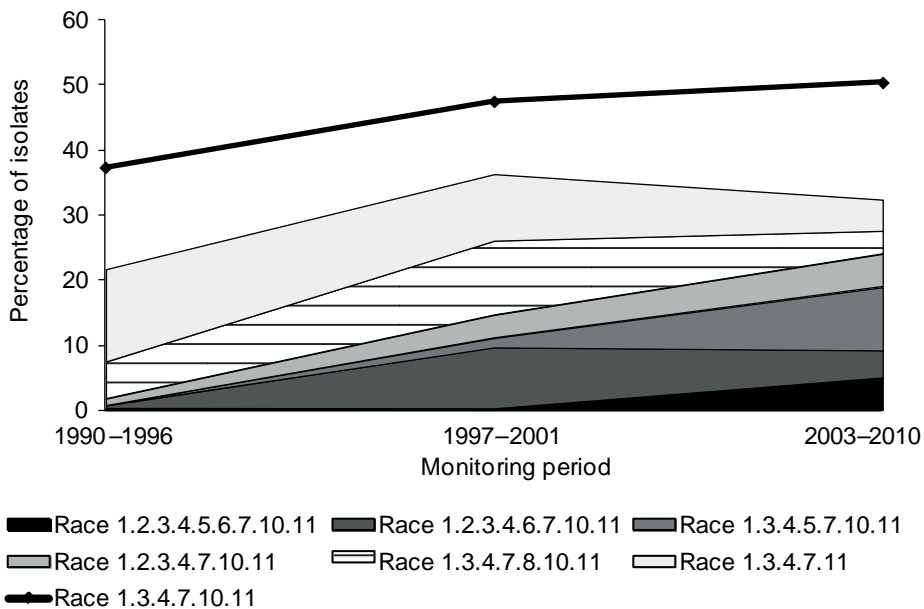


Figure 14. Changes in the prevalence of the seven most common virulence races in Finnish *P. infestans* populations over the time periods 1990–1996 to 2003–2010.

The shift towards more and more complex races is shown to be typical for a sexually reproducing population. According to Umaerus et al. (1983) in the 1960s races containing 1 to 3 virulence factors were dominant and during the 1970s races possessing up to 5 virulence factors were sometimes found in isolates collected from R-gene collections of cultivars. In most populations from the 1970s to the 1990s, restudied by Andrivon (1994), C_i and C_p values were between 1 and 4, with the exception of Mexican and East German populations. In the Mexican sexually reproducing population these values were between 5 and 6 and in the East German between 4 and 7, probably due to collection of isolates from potato breeding material with a high incidence of R-genes. Also in Canada and the USA old asexual US-1 genotypes contained 1 to 4 virulences, while up to 9 virulence genes were found in sexually reproducing populations of *P. infestans* (Peters et al. 1998).

According to some theories, highly complex pathogen races carrying many 'unnecessary' genes for virulence in a host population without corresponding R-genes should be less fit than races with one or few virulence genes (Van der Plank 1968). This has been shown to be the case in other host-pathogen systems, as reviewed e.g. by Cooke and Lees (2004) and Fry (2008). According to this thesis, and based on papers III, IV and V, the fitness cost associated with race complexity of *P. infestans* is not obvious. In Dutch *P. infestans* populations from three separate regions no statistically significant correlation was found between number of virulence factors per isolate and aggressiveness components (Flier and Turkensteen 1999). Furthermore, in most population studies on sexually reproducing *P. infestans* populations, the most complex races have been among the most aggressive ones (Goodwin et al. 1995, Leberton et al. 1998, Fry 2008)

3.2.3 Genetic diversity

All the Finnish *P. infestans* isolates characterised since 1992 have represented the new population that migrated to Europe in the 1980s. The A2 mating type has been present and only mitochondrial haplotypes Ia and IIa have been found (paper III, Andersson et al. 2007). Moreover, the phenotypic and genetic diversity among Finnish *P. infestans* isolates has been high, as in other Nordic countries (paper IV). Thus no evident clonal lineages have been detected, in contrast to the USA (Goodwin et al. 1994, Fry 2008), the United Kingdom and many continental European regions (Cooke et al. 2011).

Papers on studies of genetic diversity of Finnish and Nordic *P. infestans* populations are not included in the thesis because most of the analyses were carried out in Sweden, Norway and the Netherlands (Brurberg et al. 1999, Andersson et al. 2007, Brurberg et al. 2011). A short overview is given here to complement other aspects of the Finnish population described above. Studies based on molecular markers also indicate that diversity in populations, measured using various methods, increased from the 1990s to the 2000s and enhanced the significance of sexual reproduction in Finnish and Nordic potato late blight populations.

In 2002-2006 RFLP marker RG57 revealed 25 different multilocus genotypes among 39 isolates characterised, 18 of which were found only once (Brurberg et al. 1999). AFLP fingerprinting of 116 *P. infestans* isolates collected in 1992-2000 detected over 80 distinct AFLP fragments, although many of these were monomorphic for all isolates. Among isolates collected in different years and sites fewer than 20% displayed an identical AFLP-banding pattern (Hannukkala, Lehtinen and Joutsjoki, unpublished). On the other hand, all isolates collected from a single field were in three cases almost identical in their AFLP-

banding pattern, while in two cases almost all isolates produced a unique AFLP-banding pattern (Andersson et al. 2007). This indicates that in certain conditions one especially fit isolate survived the genetic bottleneck or was capable of taking over temporarily in a population, as shown in studies of Goodwin et al. (1994).

The studies of genetic variation of *P. infestans* in the Nordic countries using standardised, validated simple-sequence repeat (SSR) protocols (Lees et al. 2006) revealed extreme genetic diversity among isolates collected from the four Nordic countries in 2003 (Brurberg et al. 2011). The number of different alleles detected at nine SSR loci (Pi02, Pi04, Pi16, Pi26, Pi33, 4B, 4G, D13 and G11) in total was 49 among 200 isolates analysed. Fifty Finnish isolates analysed represented 49 distinct multilocus genotypes and one genotype occurred twice. All these findings support the hypothesis that sexual reproduction is very common in Finnish and Nordic *P. infestans* populations, and current populations are genetically more diverse than elsewhere in the Europe, USA and Canada.

3.3 Consequences of sexual reproduction and earlier starting epidemics to fungicide use

3.3.1 General development of fungicide sales and use

Potato late blight fungicide sales and their use in late blight management dramatically increased from 1953 to 2010 due to intensification of potato production and changes in *P. infestans* epidemiology. There are reliable statistics of fungicide sales in Finland from 1953 onwards. The need for chemical late blight control was slowly recognised during the 1960s and the percentage of treated area gradually increased from 2% in 1960 to 14% in 1969. Fungicide treatments to protect the crop were frequently recommended in the 1970s (Seppänen 1979). Fungicide sales rapidly increased at the beginning of the 1970s to early 1980s to level of 50 tonnes of active ingredient per year, mostly multi-site protectant dithiocarbamates (Figure 16). Theoretically the amount of fungicides sold was sufficient for one spray application to 80% of the total potato area, which then was 40,000 ha (Figure 15).

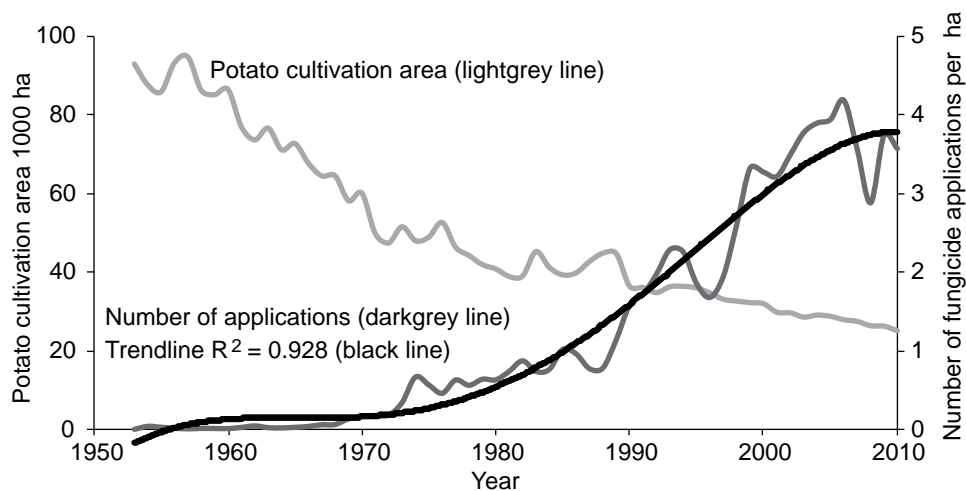


Figure 15. Development of potato cultivation area and estimated annual number of late blight fungicide applications per cultivated hectare from 1953 to 2010, and the calculated trend for increase in number of applications.

During the 1980s an increasing number of potato growers started to develop their production systems to fully specialise in growing potato as their main cash crop. According to Seppänen (1987) those farmers specialised in potato production applied fungicides 2–3 times per season to their fields. The migration of a sexually reproducing *P. infestans* population followed by early starting epidemics during the 1990s has strongly affected both fungicide use and sales. The increase in fungicide use coincides well with earlier starting blight epidemics observed during the same period (Figure 6). The annual number of fungicide applications per hectare has increased from one in the early 1990s to four in the 2000s. In recent years the average number of applications seems to have stabilised to 3–4 annual applications per hectare (Figure 15).

During the 2000s the area treated with fungicides stabilised at approximately 100,000 hectares, though considerable annual fluctuations in estimated fungicide-related potato area are obvious, especially at the end of the 1990s and in the early 2000s (Figure 19). This is partly explained by different late blight pressure in different seasons. The estimate for treated area based on fungicide sales for an individual season is not very accurate, even where a two-year moving average has been used, because after an economically profitable growth season farmers usually buy fungicides to be used in coming years for tax benefits. Furthermore, for seasons with low blight pressure, part of the fungicides reserved for applications are held back to be used in following years. In spite of its limitations, this simple estimate represents a reasonable overview of the trend in fungicide use in Finnish potato production.

3.3.2 Multi-site protectant fungicides

In Finland inorganic multi-site fungicide, Bordeaux Mixture, was known already at the beginning of the 1900s. Researchers

recommended it for late blight control in 1930s (Jamalainen 1933). Due to substantial fluctuations in potato late blight incidence between years, inadequate application technology and difficulties in correct timing of Bordeaux Mixture application, chemical blight control was not regarded as economically profitable in the 1930s (Roivainen 1939). In the early 1950s another inorganic multi-site protectant fungicide, copperoxychloride was registered for late blight control and until the 1960s in it was the only late blight fungicide available. The use and sales of the fungicide were low (Figure 15, Figure 16).

In the beginning of 1960s organic multi-site protectant fungicides, dithiocarbamates, were registered. From the 1960s to the 1980s the sales of multi-site protectant fungicides increased steadily (Figure 16). Dithiocarbamates constituted 95% of the sales of multi-site protectant fungicides. At the end of the 1980s and beginning of the 1990s sales of multi-site protectants increased dramatically (Figure 16). This is mainly due to increased use of RNA-synthesis inhibiting fungicide metalaxyl. In the new commercial product 94% of the active ingredient content was dithiocarbamate, mancozeb, and only 6% metalaxyl.

In 1990s sales of dithiocarbamates again decreased because new fungicides with other modes of action and better rain-fastness were introduced to market. In recent years the decrease in sales of dithiocarbamates has been rapid (Figure 16). The development is in concordance with EU policy according to which the use of dithiocarbamates should be restricted due to risks for human health (Cooke et al. 2011).

3.3.3 RNA-synthesis inhibitors and metalaxyl resistance

The registration of the fully systemic RNA-synthesis inhibiting phenylamide fungicide, metalaxyl, in 1985 was regarded as a revolutionary improvement in late

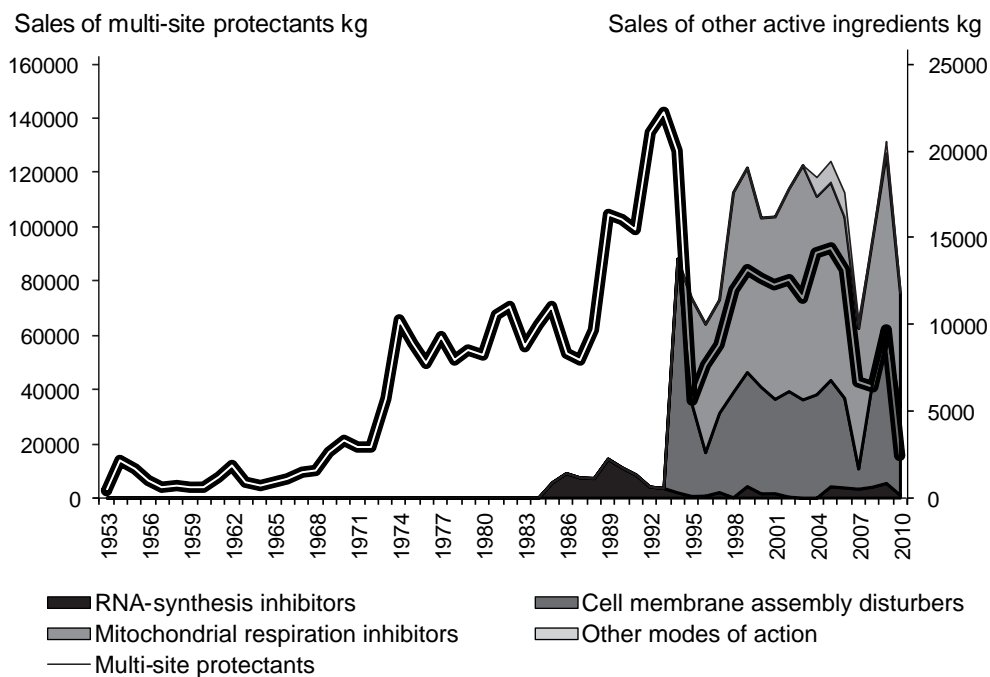


Figure 16. Sales of active ingredients of potato late blight fungicides kg/year, classified by their biochemical effect on pathogen metabolism. Sales of multi-site protectants (copper and dithiocarbamates) are shown on a different scale to the others.

blight control. Sales of the pre-packed mixture of metalaxyl and mancozeb rapidly increased and at the end of the 1980s. In figure 16 the amount of sold RNA-inhibiting active ingredient looks rather low. Due to low dose rate the amount was sufficient for more than 50% of the potato area to be treated with the metalaxyl-mancozeb mixture (Figure 17).

The rapid development of resistance in *P. infestans* to phenylamides and loss of efficacy of the fungicide in Ireland and the Netherlands (Dowley and O'Sullivan 1981, Davidse et al. 1983) was known at the time of registration of metalaxyl in Finland. It was, however, believed that no resistance development could take place in Finland while metalaxyl was sold as a pre-packed mixture with a high concentration of mancozeb. One to two applications with metalaxyl-mancozeb mixture were

recommended as the basic blight management strategy. At high disease pressure additional applications of straight dithiocarbamates were recommended (Seppänen 1987). During the initial years after registration applications with a metalaxyl product were recommended to be started after visible blight lesions were present in the crop, and only after 1987 were recommendations changed to emphasize the importance of preventive use of metalaxyl product (Hannukkala 1994).

The use of a mancozeb–metalaxyl mixture was not enough to prevent rapid built up of metalaxyl resistance in Finnish *P. infestans* strains, while all other rules for anti-resistance strategies (Staub 1991) were profoundly violated. The monitoring of metalaxyl resistance was started at MTT Agrifood Research Finland in 1990 through the initiative of the pesticide in-

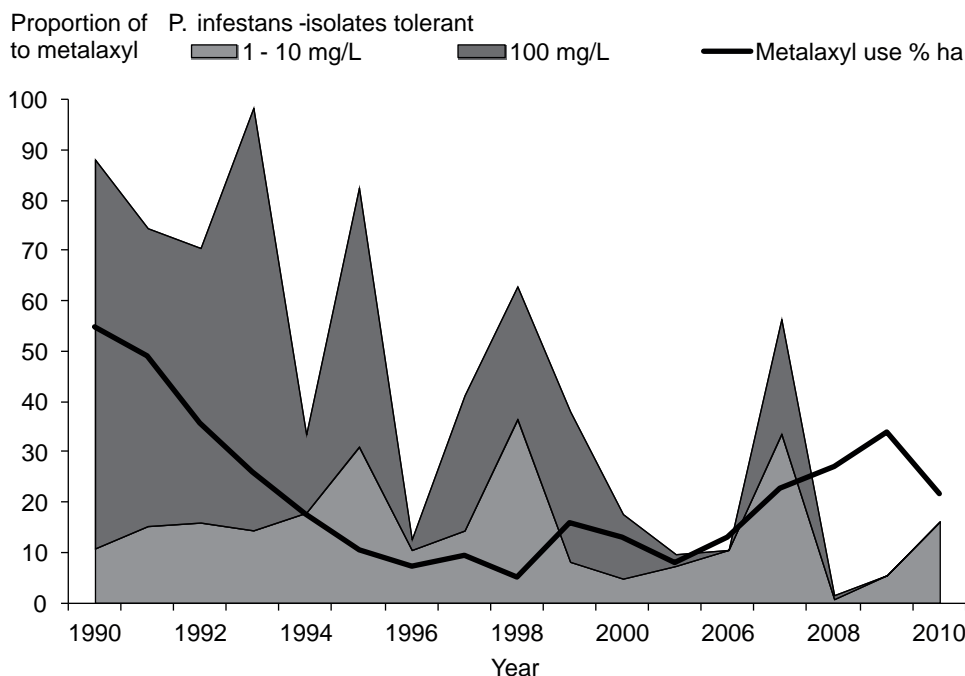


Figure 17. Proportion of *P. infestans* strains growing and sporulating normally at 1–10 mg/L (intermediately resistant) and 100 mg/L (resistant) concentration of metalaxyl in 1990–2010 and percentage of cultivated potato area treated with metalaxyl containing products.

dustry to prove that no resistance was present, but it transpired that among 91 isolates tested in 1990 only 11 (12%) were sensitive to metalaxyl (Figure 17). The resistance level in the *P. infestans* population remained high for the following few years.

Loss of efficacy of metalaxyl under practical field conditions was never scientifically soundly proven in Finland, in contrast to the case for Ireland (Dowley and O’Sullivan 1981) and the Netherlands (Davidse et al. 1983). However, Finnish farmers, based on their own judgement of failures in late blight control, dramatically reduced metalaxyl use (Figure 17) and replaced it with straight dithiocarbamates while other alternatives were not available (Figure 16). Simultaneously strict restrictions in use of metalaxyl products were given in collaboration with authorities and the pesticide industry following anti-resist-

ance actions adapted elsewhere in Europe (Staub 1991, Gisi and Cohen 1996).

Both the occurrence of phenylamide resistant strains (Dowley and O’Sullivan 1981) and the presence of the A2 mating type (Hohl and Iselin 1984) of *P. infestans* in Europe were detected in 1981. Frequency of phenylamide resistance and A2 mating type in *P. infestans* populations rapidly increased globally throughout the 1980s and 1990s. Also in Finland the presence of phenylamide resistance as indicated by resistance to metalaxyl and A2 mating type were detected almost simultaneously in the first years of the 1990s (paper III). These two events most probably are coincidental and independent of each other. The very first phenylamide-resistant strains found in the Republic of Ireland were later shown to belong to the ‘Old’ US-1 population (Cooke et. al. 2011).

During the first half of the 1990s overall fungicide sales (Figure 17) and use (Figure 15) rapidly increased. As a result of changes in chemical late blight control practices the proportion on metalaxyl-insensitive *P. infestans* strains started to decrease during the second half of the 1990s (Figure 17) (paper III). The proportion of insensitive strains has remained low during the 2000s (paper IV), with occasional seasonal fluctuations that are considered as normal behaviour of *P. infestans* populations (Cooke et al. 2011). Metalaxyl resistant strains of *P. infestans* in the 2000s in the Nordic Countries have been rare in spite of moderate use of metalaxyl fungicides (paper V). The Nordic resistance situation differs from that in many other countries where, in spite of careful anti-resistance actions, the proportion of metalaxyl resistant strains has remained typically at the level of 10–40% of the population (Cooke et al. 2011, Gisi et al. 2011).

3.3.4 Fungicides disturbing cell membrane assembly

To combat metalaxyl resistant strains of *P. infestans* fungicides with alternative biochemical modes of action were introduced in the 1990s. A fungicide disturbing assembly of cell membranes in *P. infestans*, propamocarb-HCl in mixture with mancozeb, was registered in 1994. This can be seen as a peak in sales of fungicides affecting cell membranes in 1994–1995 (Figure 16, Figure 18). Following years fungicides with similar mode of were registered. In 1997 CAA-fungicide, dimetomorph, in mixture with mancozeb was registered. More recently in 2008 another CAA-fungicide, mandipropamid was introduced to Finnish markets. The overall sales of cell membrane assembly disturbing fungicides have been rather stable in 2000s (Figure 16, Figure 18).

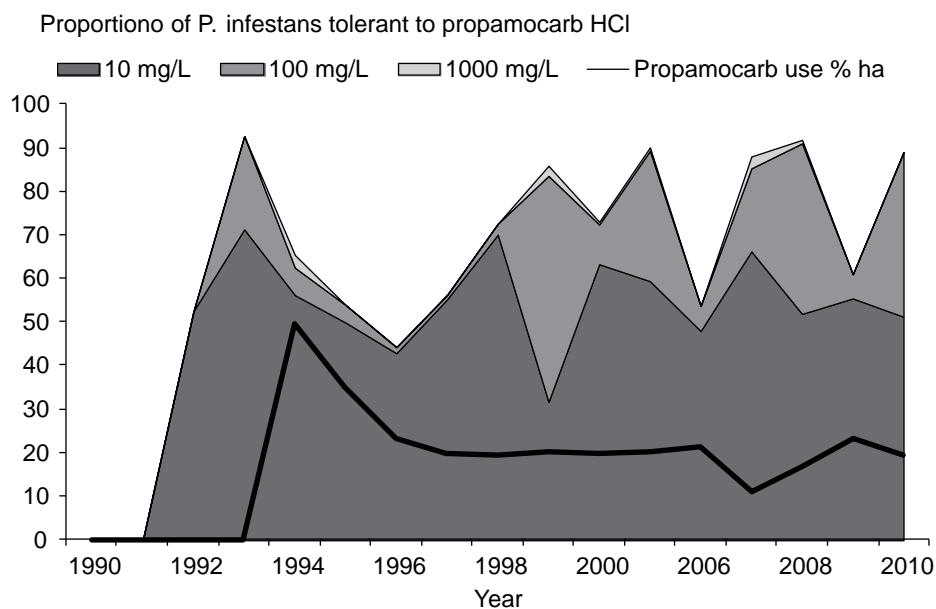


Figure 18. Proportion of *P. infestans* strains growing and sporulating normally at 10 mg/L, 100 mg/L and 1000 mg/L concentration propamocarb-HCl of the fungicide in 1992–2010 and the percentage of cultivated potato area treated with propamocarb-HCl containing products.

Although the risk of resistance development against propamocarb-HCl was estimated low (Löchel and Birchmore 1990) *P. infestans* isolates collected for metalaxyl resistance studies were also tested for their response to propamocarb-HCl since 1994. Additionally, 33 isolates collected in 1992 and 1993 were tested to determine the sensitivity baseline of the population before propamocarb-HCl was used on a field-scale.

There were isolates growing and sporulating in the presence of up to 100 mg/L propamocarb-HCl already before the fungicide was commercialised in Finland. There have been considerable annual fluctuations in the occurrence of isolates with some tolerance to the fungicide, but no trend towards increased tolerance is evident in Figure 18. This is in good agreement with the original evaluation of propamocarb-HCl as a fungicide with a low risk of resistance development (Löchel and Birchmore 1990, Bardsley et al. 1998). After almost 20 years of experience in use of propamocarb for late blight control in Europe and America no signs of resistance in *P. infestans* or failures in blight control have been reported (Gisi and Sierotzki 2008, Cooke et al. 2011).

Among the cell-membrane-active compounds propamocarb-HCl has a different biochemical mode of action and target site from the CAA-fungicides dimethomorph and mandipropamid (Cohen et al. 1995, Griffiths et al. 2003, Lamberth et al. 2006, Gisi et al. 2007). Theoretically, dimethomorph and mandipropamid belong to the same cross-resistance group (Cohen and Gisi 2007), but no signs of increased insensitivity against these compounds in field isolates of *P. infestans* have been recorded (Cooke et al. 2011, Gisi et al. 2011).

3.3.5 Fungicides inhibiting mitochondrial respiration

Several fungicides inhibiting mitochondrial respiration in *P. infestans* cells have been registered since 1995. Their share in

fungicide sales rapidly increased after their introduction and has remained high with yearly fluctuations (Figure 16.) Also most compounds that inhibit mitochondrial respiration differ clearly in their biochemical mode of action and target site. Fluazinam currently comprises 70% of the sales of respiration inhibitors in Finland. The mechanism of inhibition of ATP production at the presence of fluazinam differs from disruption of electron transport in cytochrome b caused by QoI- and QiI-fungicides (Tucker et al. 1994, Mitani et al. 1998, Barlett et al. 2002). No resistance in field isolates of *P. infestans* to fluazinam has been detected in several European monitoring projects (Cooke et al. 1998, Cooke and Little 2006, Gisi and Sierotzki 2008, Cooke et al. 2011).

Famoxadone and fenamidone, among the QoI-compounds, belong to the same cross-resistance group as strobilurins (Barlett et al. 2002), but are in a separate resistance group to QiI-compound cyazofamid (Ohshima et al. 2004). Rapid resistance build-up against strobilurins and related compounds has been common for several pathogens of field crops (Fernández-Ortuño et al. 2008). Therefore, it is necessary to be careful and strictly follow anti-resistance instructions. It has been shown that there is no cross-resistance between RNA-synthesis inhibitors, cell-membrane-active and respiration-inhibiting fungicides (Gisi and Sierotzki 2008).

3.3.6 Protecting expanding leaves and apical growth after fungicide application

The change in typical late blight epidemic onset from August to the beginning of July also highlighted a new problem: how to protect new growth efficiently after fungicide application during the period of intensive vegetative growth of potato? At the stage of rapid vegetative growth, the potato aerial biomass can increase by 10–20% within one week (Gayler et al.

2002), which is the typical application interval for many late blight fungicides.

Until 1995 there were only available products not penetrating in leaf tissues (dithiocarbamates), and fully systemic RNA-synthesis inhibiting (dithiocarbamate + metalaxyl) products (Figure 19).

Applications were mainly done after potato flowering when vegetative growth has ceased. The problem with dithiocarbamates was that they were easily washed into soil during heavy rains. Metalaxyl-product protecting apical growth lost its efficacy due to fungicide resistance.

The use of the products absorbed into wax layer of leaves rapidly increased after their registration in 1995 (Figure 19). At the end of the 1990s when spray applications had to be started at the period of fast vegetative growth of potato application interval of these products had to be reduced from 7–10 days to 5–7 days. Products are tightly bound into wax and not redeposited to the expanding leaf after application. This resulted in increase in the number of an-

nual sprays per hectare (Figure 15). The next improvement in control efficacy was introduction of products capable to move within leaf after application. The proportion of fungicides used with different mobility properties in potato was relatively stable from 1996 to 2006 (Figure 19).

New fungicides providing good protection of apical growth, cyazofamid (2006), mandipropamid and a mixture of propamocarb-HCl and fenamidone (2008), have replaced less mobile fungicides in spray programmes (Figure 19). Simultaneously the number of applications per unit area appears to have decreased during the most recent seasons (Figure 15). Recent reduction in late blight fungicide use might be the first step to fulfil the requirements of EU (Directive 128/2009 EC) and corresponding Finnish pesticide legislation (Laki kasvinsuojeluaineista 1563/2011). There is still a long way to reach the aims of published National action plans (NAP) for sustainable use of pesticides (Barzman and Dachbrodt-Saayeh 2011), where 25–50% reductions in pesticide use within few years has been suggested.

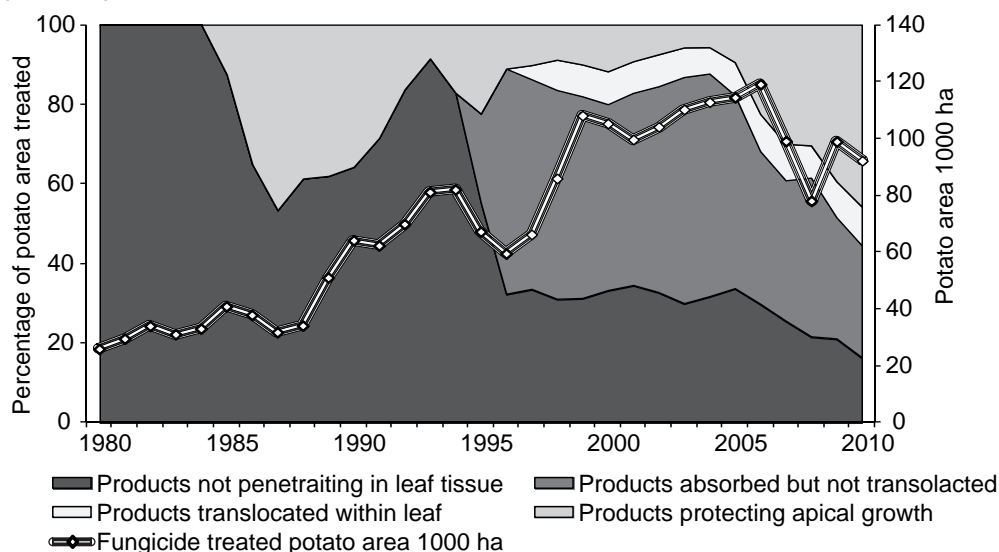


Figure 19. Use of products not penetrating into leaf tissue, products absorbed in leaf but not traslocated, products translocated within leaf tissue and products additionally protecting apical growth, as a percentage of the entire fungicide-treated potato area. The lines indicate fluctuations in annual potato area treated with fungicides in Finland.

4 Conclusions and future prospects

The incidence and severity of potato late blight, since its migration in 1847, was relatively stable in Finland until the 1990s. The epidemics normally started towards the end of August and there were few severe blight years in a decade. The rapid change in epidemiology and population structure took place during the 1990s when a new sexually reproducing *P. infestans* population invaded and displaced the asexual A1 clonal lineage. Throughout the 1990s the onset of late blight outbreaks shifted from the end of August to the end of June or beginning of July. Also the mating type ratio simultaneously stabilised close to 50/50 for mating types A1 and A2 in the *P. infestans* population. This indicates a high probability for oospore formation in diseased crops and consequent accumulation of infective oospores in the soil. Soil-borne primary inoculum currently plays a much bigger role in potato late blight epidemiology in Finland and the Nordic Countries than elsewhere in Europe. The diversity of R-gene virulence races decreased while the complexity of races increased considerably. Also the genetic diversity of the Finnish *P. infestans* population is very high as measured with RFLP, AFLP and SSR markers. There is no direct evidence of overall increased aggressiveness in the Finnish *P. infestans* pop-

ulation, although the range in aggressiveness components between individuals is very wide. Earlier starting late blight epidemics have resulted in increased use of fungicides to protect the crop. Three to four more fungicide applications are currently needed for reliable late blight control than in the 1980s. The increased use of phenylamide fungicides at the end of the 1980s rapidly created the problem of an almost fully resistant *P. infestans* population. Improved anti-resistance strategies and introduction of several fungicides with different modes of actions were sufficient to eradicate phenylamide-resistant strains from the *P. infestans* population. Currently potato late blight is under full control in conventional potato production, as long as effective fungicides are available. There is increasing public demand for decreased use of pesticides in agriculture, and therefore in the future more effort should be put into developing potato cultivars with durable resistance against late blight. Also cultural practices, including crop rotations, should be developed to reduce sources of primary inoculum in future potato production. Due to the potential for rapid changes in potato late blight epidemiology and population properties, regular monitoring should be implemented at least in a few years' intervals.

5 Acknowledgements

The studies included in this dissertation have been carried out at MTT Agrifood Research Finland, and Potato Research Institute (Petla). The data gathered before 1990s have been produced by MTT's and Petla's budgeted funding. From the 1990s onwards data for this thesis has been obtained from many domestic and international potato late blight related projects funded mainly by the Ministry of Agriculture and Forestry, MTT and Petla. Great deal of the late blight monitoring data has been collected from variety and fungicide efficacy trials principally paid by seed, breeding and chemical companies. I am honestly thankful for all the material resources, mental support and patience provided by the numerous collaborators and colleagues that have made it possible to compose and finalise this long term study on potato late blight in Finland.

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At the finalizing phase of this work my supervisors and colleagues acting Professor Minna Pirhonen and Professor Jari Valkonen have provided me a great opportunity to work as a university lecturer in University of Helsinki for the major-

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