Characterization of the *Phytophthora infestans* Population in the Columbia Basin of Oregon and Washington from 1992 to 1995

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ABSTRACT

Miller, J. S., Hamm, P. B., and Johnson, D. A. 1997. Characterization of the *Phytophthora infestans* population in the Columbia Basin of Oregon and Washington from 1992 to 1995. *Phytopathology* 87:656-660.

Isolates of *Phytophthora infestans* collected from 1992 to 1995 from potato fields in the Columbia Basin of Oregon and Washington were analyzed for compatibility type, metalaxyl sensitivity, and *glucose-6-phosphate isomerase* (*Gpi*) genotype. In 1992, 30 of 31 isolates were of the US-1 multilocus genotype. A single metalaxyl-resistant isolate of the US-6 (A1 *Gpi* 86/100) genotype was found near the end of the growing season. In 1993, only 2 of the 59 isolates collected were A1 isolates with *Gpi* 86/100. Ten isolates were of the A2 compatibility type, seven with

Gpi 100/111, two with *Gpi* 100/100, and one was undetermined. The remaining isolates were metalaxyl-resistant A1 compatibility types with either *Gpi* 100/100 or 100/111. The first A2 isolates in the Columbia Basin were found in 1993. In 1994, 10 of 18 isolates were of the US-1 genotype. The remaining isolates were US-6 and US-8 genotypes. In 1995, 97% of 268 isolates tested were of the US-8 genotype. Five isolates were A2 compatibility type with *Gpi* 100/122. One A2, metalaxyl-resistant isolate was *Gpi* 100/100/111, and two A1 isolates were *Gpi* 100/111/122. The population of *P. infestans* quickly changed between 1992 and 1995, from a population comprised almost exclusively of the US-1 genotype to a population represented by new or recombinant genotypes.

Late blight, caused by *Phytophthora infestans* (Mont.) de Bary, is one of the most destructive diseases of cultivated potato (*Solanum tuberosum* ssp. *tuberosum* L.). Evidence that the central highlands of Mexico is the center of origin for the pathogen has been provided by the widespread diversity in virulence characteristics and neutral allozyme markers in the Mexican population (9,14,24). Outside Mexico, a single clonal lineage existed worldwide until the 1980s (9,12,29). This lineage was characterized by the A1 compatibility type and sensitivity to the phenylamide fungicide metalaxyl. Isolates resistant to metalaxyl (3,4) and belonging to the A2 compatibility type (18,21) were detected in Europe during the 1980s. Since that time, numerous studies have reported the spread of the A2 compatibility type and metalaxyl resistance in potato-growing regions around the world. These newer strains have increased in frequency and replaced the old clonal strain in the Netherlands (8,29), Germany (29), Poland (29,30), and various regions of the United States (12,15). The spread of the A2 compatibility type to areas where the A1 compatibility type has existed creates the possibility of sexual recombination. Oospores produced by sexual recombination may provide another source of initial inoculum for initiating epidemics and lead to offspring with characteristics different from the parental types (9).

Allozyme analysis has become an effective tool for identifying variation in *P. infestans* populations (8,11,12,15,27-31). A study of isolates from the United States and Canada in 1992 identified 11 multilocus genotypes (15). The four most common multilocus genotypes, US-1, US-6, US-7, and US-8, were detected from more than three locations and were widespread in the United States. Before 1992, only two multilocus genotypes had been detected previously in the United States and Canada (US-1 and US-6).

Multilocus genotypes were based on compatibility type, allozyme genotype, and DNA fingerprints. These multilocus genotypes could be differentiated from each other on the basis of *glucose-6-phosphate isomerase* (*Gpi*) allozyme analysis alone. Allozyme analysis of *peptidase* (*Pep*) alleles gave only two allozyme genotypes, 92/100 and 100/100. The numbers represent the distance migrated by alleles relative to the most common allele during electrophoresis (1). In the case of *Pep* 92/100, the 92 allele migrates 92% as far as the 100 allele. Currently, 14 multilocus genotypes have been identified in the United States (11,15; S. B. Goodwin and W. E. Fry, *personal communication*). Four of the most recently identified multilocus genotypes (US-11 through US-14) can be identified by compatibility type and *Gpi* allozyme genotype.

Allozyme analysis was performed in the studies mentioned above by starch gel electrophoresis, which is a labor-intensive and time-consuming procedure (13). A more economical technique that uses cellulose acetate electrophoresis was developed in 1995 for use as a quick diagnostic tool to aid in control of late blight by providing information related to metalaxyl sensitivity and compatibility type identity (13). The relationship between allozyme genotype, metalaxyl sensitivity, and compatibility type was thought to be effective as long as populations of *P. infestans* remained clonal.

In the Columbia Basin of north-central Oregon and south-central Washington, late blight first was documented in 1947 (2). This region, comprising ~65,000 ha of cultivated potatoes in 1995, is surrounded by mountains on all sides, separating it from the potato-producing regions of western Washington and other areas of Oregon and from Montana, British Columbia, and southern Idaho. Late blight has caused severe damage in recent years, affecting ~25,000 ha in 1992, 31,000 ha in 1993, 770 ha in 1994, and 65,000 ha in 1995 (19,20). From 1947 to 1990, the population of *P. infestans* in this area was composed of the old clonal US-1 lineage (9,11). The situation changed when isolates resistant to metalaxyl appeared in 1991 (5) and when the A2 compatibility type was detected in 1993 (17). Almost all *P. infestans* isolates collected in 1992 were sensitive to metalaxyl (17). The frequency

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Publication no. P-1997-0418-04R

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of metalaxyl resistance and the A2 compatibility type were unknown, however.

This study was undertaken to determine the composition of the *P. infestans* population from 1992 to 1995 by characterizing isolates obtained from potato fields in the Columbia Basin for metalaxyl sensitivity, compatibility type, and *Gpi* genotype. A knowledge of the population structure of *P. infestans* in the Columbia Basin may supply information on metalaxyl resistance, sources of initial inoculum for epidemics, and potential for oospore production, which in turn will aid in developing better late blight control strategies.

MATERIALS AND METHODS

Isolate collection and maintenance. Isolates of *P. infestans* were obtained from naturally infected potato foliage throughout the Columbia Basin of Oregon and Washington from 1992 to 1995 (Table 1). This region extends for ~180 km, from Umatilla and Morrow counties in north-central Oregon to Grant and Adams counties in south-central Washington. Potato fields were monitored by field workers several times each week during the growing season. When late blight was first observed in any field, P. B. Hamm or D. A. Johnson usually was notified. Most infected fields were sampled during the low-incidence years of 1992 and 1994 from the time late blight was first reported in the region until mid-August to give a nearly complete sample of fields through mid-growing season. During the severe epidemic years of 1993 and 1995, when all fields in some counties were infected, representative isolations were done randomly in generalized locations throughout the Columbia Basin. In 1992 and 1993, only one isolate was obtained from each field, because populations within fields were believed to be clonal (10). In 1994 and 1995, multiple samples were taken from each field. Fewer samples were collected in the northern portion of the Basin, in Grant and Adams counties, because late blight was either not present or occurred at a lower incidence and later in the season than in the southern portion of the Basin. In all, 388 isolates were collected from 178 potato fields from 1992 to 1995.

Cultures were obtained by transferring sporangia from the margin of a single sporulating lesion to rye extract agar (26) amended with 100 µg each of penicillin, pimarin, and polymyxin per ml or by removing small areas of infected leaf tissue near the lesion margin and plating the tissue on rye extract agar containing 10 µl of pimarin, 250 units of ampicillin, and 10 µg of rifampicin per ml. Isolates were derived by transferring a hyphal tip or single sporangium to unamended rye extract agar. Isolates were maintained on rye extract agar at 18°C in darkness and were transferred every 4 to 5 weeks.

Isolate characterization. Each isolate was tested for compatibility type and metalaxyl sensitivity. Allozyme genotype was determined for all isolates collected in 1992, 1994, and 1995 and 36 of 59 isolates collected in 1993. Compatibility types were determined by pairing each isolate against two known A1 and two known A2 isolates on rye extract agar. Agar plates were kept in darkness at 18°C for 14 days and then examined for oospores with a dissecting microscope. Isolates Pi 94 (US-6) and Fi 2 (US-1) were used as A1 testers, and isolates 584 (US-8) and 302 (US-7) were used as A2 testers. The compatibility type and multilocus genotype of the tester isolates was confirmed by B. A. Fry (Cornell University, Ithaca, NY).

Metalaxyl sensitivity of isolates was evaluated by placing isolates singly on plates containing rye extract agar amended with 0.1, 1.0, 10.0, and 100.0 µg of technical-grade metalaxyl per ml. Classification of strains as sensitive or resistant was determined following a modification of the scheme used by Deahl et al. (6), using 10.0 µg of metalaxyl per ml as the critical value. Sensitive and resistant isolates displayed <10% and >10% growth, respectively, compared to the control. Isolates Fi 2 (US-1) and 382 (US-1)

and 584 (US-8) and 537 (US-11) were used as standards for sensitive and resistant responses, respectively. These standards were chosen because of their vigorous growth on rye extract agar in the absence of metalaxyl.

Isolates were analyzed for *Gpi* allozyme genotype (13). Assay samples were obtained by scraping mycelium from 2- to 3-week-old cultures with a spatula and placing the mycelium in a 1.5-ml centrifuge tube containing 1 ml of deionized distilled water. Tubes were stored on ice between steps for the remainder of the procedure. After centrifugation at 13,000 × *g* for 5 min, approximately half the supernatant in each tube was poured off and discarded. The pellet in each tube was ground with a plastic pestle attached to a high-speed electric hand-held drill until the sample mixture was homogenized. The tubes were centrifuged again at 13,000 × *g* for 5 min. Aliquots of supernatant were electrophoresed and stained with agar overlays for *Gpi* (13). The following standards (identified by B. A. Fry) were used: Fi 2 (US-1), Pi 94 (US-6), 301 and 302 (US-7), 584 (US-8), and 537 (US-11). Only two or three standards were run per plate, depending on the phenotype of the isolates being compared, i.e., 584, 301, and 302 with A2 isolates; 537 and Pi 94 with A1 metalaxyl-resistant isolates; and Fi 2 with A1 metalaxyl-sensitive isolates. When a combination of phenotypes was analyzed together, a combination of appropriate standards was used.

RESULTS

All isolates collected in 1992 were of the A1 compatibility type. Of 31 isolates, 30 were metalaxyl sensitive with *Gpi* 86/100 and belonged to the US-1 multilocus genotype. One isolate collected near the end of the growing season, on 18 August, was metalaxyl resistant with *Gpi* 100/100 and was a US-6 genotype (Table 2).

Preliminary analysis of samples by DNA fingerprinting indicated that isolates with the same *Gpi* allozyme genotypes collected in 1993 had significant genetic variation. This diversity within isolate groupings based on identical *Gpi* allozyme genotypes was not found in other years (R. W. Sandrock and W. E. Fry, *personal communication*). Because of this, no US designations were made for 1993 isolates.

In 1993, 59 isolates were collected from the Columbia Basin. Of these, 46 were A1, and 13 were A2 compatibility types (Table 2). All A2 isolates were resistant to metalaxyl. Of the A1 Columbia Basin isolates, 6 were sensitive, and the remaining 40 were resistant to metalaxyl. Allozyme analysis was done on 2 of the 6 A1 metalaxyl-sensitive isolates, 26 of the 40 A1 metalaxyl-resistant isolates, and 9 of the 10 A2 metalaxyl-resistant isolates. The isolates not analyzed for allozyme genotype did not survive in culture until the time when allozyme analysis was performed. The two A1 metalaxyl-sensitive isolates had *Gpi* 86/100 (Table 2). Of the A1

TABLE 1. Origin and number of *Phytophthora infestans* isolates collected from potato fields in the Columbia Basin of Oregon and Washington from 1992 to 1995

County of Origin	Number of isolates			
	1992	1993	1994	1995
Oregon				
Umatilla	2	30	8	113
Morrow	24	17	0	58
Washington				
Benton	4	6	0	43
Franklin	1	2	7	15
Walla Walla	0	4	1	11
Grant ^a	0	0	2	0
Adams ^a	0	0	0	5
Total	31	59	18	245
Number of fields	21	55	8	71

^a Late blight incidence was very low from 1992 to 1994.

metalaxyl-resistant isolates, 16 had *Gpi* 100/111, 4 had *Gpi* 100/100/111, and 6 had *Gpi* 100/100. The *Gpi* 100/111 allozyme genotype was found in 7 of the 10 A2 isolates, and the *Gpi* 100/100 allozyme genotype was found in 2 of the 10 A2 isolates.

In 1994, 10 of 18 isolates were metalaxyl sensitive and were of the US-1 genotype (A1 *Gpi* 86/100; Table 2). Of the remaining eight metalaxyl-resistant isolates, five were A2, and three were A1. All of the A2 compatibility type isolates were of the US-8 genotype (*Gpi* 100/111/122). All of the A1 metalaxyl-resistant isolates were of the US-6 genotype (*Gpi* 100/100).

In 1995, all isolates were metalaxyl resistant, and 97% of these isolates belonged to the US-8 genotype (Table 2). Five isolates with the A2 compatibility type had *Gpi* 100/122, and one A2 isolate had *Gpi* 100/100/111. Of 268 isolates, 2 were characterized as A1 with *Gpi* 100/111/122.

DISCUSSION

The population structure of *P. infestans* in the Columbia Basin of Oregon and Washington changed between 1992 and 1995. In 1991, metalaxyl resistance was detected in a limited area late in the season in the northern Columbia Basin of Washington (5). In our study, one metalaxyl-resistant isolate was found in 1992. This isolate was collected during late August, near the end of the growing season. The frequency of metalaxyl-sensitive isolates decreased from 97% in 1992 to 10% in 1993 and increased to 37% in 1994. Metalaxyl-sensitive isolates were not detected in 1995.

The reduced frequency of metalaxyl-sensitive isolates during this period probably was not due to selection by metalaxyl. Metalaxyl was not recommended to growers for late blight control after 1992. Metalaxyl was still applied, however, one to two times on potatoes planned for long-term storage, to control storage rots, making selection due to metalaxyl similar from 1993 to 1995. These applications for control of storage rots were made at the late flowering stage of the crop and again 2 weeks later. Fields already were infested by this time in 1995, particularly fields planted with susceptible cultivars where metalaxyl was usually not applied because tubers were not going to be placed in storage. In addition, very large numbers of volunteer potato plants that were not treated with metalaxyl were present during this period in the region due to mild weather conditions. A sample of 30 fields each year gave a mean of 9,023, 2,670, and 850 volunteers per ha for 1992, 1993, and 1994, respectively. These volunteers would have provided additional opportunities for spore production and dispersal of metalaxyl-sensitive strains. If metalaxyl-sensitive strains were present in 1995, they probably would have been found, es-

pecially considering the number of *P. infestans* samples collected and the widespread area from which they were collected.

Metalaxyl-resistant isolates were most likely introduced via infected potato seed brought into the Columbia Basin at the beginning of each growing season. Metalaxyl-resistant isolates have been reported from at least one seed-production area where seed is purchased for use in the Columbia Basin (6). In addition, a previous study dealing with metalaxyl resistance suggested that metalaxyl-resistant isolates migrated into the United States, rather than arising by mutation and then selection (16).

In our study, 10.0 µg of metalaxyl per ml was used as the critical value for determining metalaxyl resistance (6). Metalaxyl sensitivity, according to our criteria, corresponded to the level of sensitivity previously identified with compatibility type and *Gpi* genotype. Metalaxyl resistance is one of the defining characteristics of the US-8 genotype (15,16). All isolates with the A2 compatibility type and *Gpi* 100/111/122 in this study were resistant to metalaxyl. Deahl et al.'s (6) original classification defined isolates with 10 to 60% growth on metalaxyl-amended media (10.0 µg/ml) was metalaxyl intermediate. This classification seems too broad for practical use. In our study, the mean growth for isolates collected in 1995 when tested on 10.0 µg of metalaxyl per ml was 57% (variance = 0.028014) compared to the unamended control. Separating isolates at 60% growth compared to the control is arbitrary and does not help in classifying isolates. Only two isolates classified as metalaxyl resistant had growth less than 20% compared to the control. All US-1 isolates found in 1992 and 1994 had growth less than 10% of the control when tested on metalaxyl-amended agar, making the division between metalaxyl resistance and sensitivity at 10% logical. These findings are similar to those of Goodwin et al. (16) who analyzed metalaxyl resistance in US-8 isolates. The criteria used by Goodwin et al. (16) identified isolates as sensitive if growth on agar with 5 µg of metalaxyl per ml was less than 40% of growth on the control. They tested 49 US-8 isolates at a concentration of 5 µg of metalaxyl per ml and found the mean growth to be 62.3% of the growth of the control. The similarity between Goodwin et al.'s (16) test and our results indicates that metalaxyl resistance among isolates of the US-8 genotype is similar over wide geographic areas.

The A2 compatibility type was found for the first time in the Columbia Basin in 1993 (17). These new A2 isolates were *Gpi* 100/111 and *Gpi* 100/100. Additionally, A1 isolates with *Gpi* 100/100/111 and *Gpi* 100/111 also were detected for the first time in 1993. The isolates with these traits had not been found previously in the Columbia Basin and represent a genetically diverse group of isolates, as determined by DNA fingerprinting (R. W.

TABLE 2. Characteristics of *Phytophthora infestans* and number and percentage (in parentheses) of isolates of each genotype collected in the Columbia Basin of Oregon and Washington during each year from 1992 to 1995

Compatibility type	Metalaxyl sensitivity ^a	<i>Gpi</i> ^b genotype	1992	1993 ^c	1994	1995
A1	Sensitive	86/100	30 (97) ^d	2 (3)	10 (56) ^d	0
A1	Resistant	100/100	1 (3) ^e	5 (8)	3 (17) ^e	0
A2	Resistant	100/111	0	7 (12)	0	0
A2	Resistant	100/111/122	0	0	5 (28) ^f	260 (97) ^f
A1	Resistant	100/100/111	0	4 (7)	0	0
A1	Resistant	100/111	0	16 (27)	0	0
A2	Resistant	100/100	0	2 (3)	0	0
A2	Resistant	100/122	0	0	0	5 (2) ^g
A2	Resistant	100/100/111	0	0	0	1 (0.3) ^g
A1	Resistant	100/111/122	0	0	0	2 (0.7) ^g

^a Isolates were tested on rye extract agar amended with 10 µg of technical-grade metalaxyl per ml. Sensitive = <10% growth compared to the control; resistant = >10% growth compared to the control.

^b *Glucose-6-phosphate isomerase*.

^c Due to diversity in DNA fingerprinting within groups of isolates with the same *Gpi* genotype, genotype designations were not assigned for these isolates. Not all isolates survived in culture until the time when *Gpi* genotype was analyzed.

^d Isolates from the US-1 genotype.

^e Isolates from the US-6 genotype.

^f Isolates from the US-8 genotype.

^g Isolates not currently assigned to a genotype.

Sandrock and W. E. Fry, *personal communication*). The A2 *Gpi* 100/100 combination has been found in British Columbia (15). These novel A1 and A2 strains probably were transported into the Columbia Basin in infected seed, because the A2 compatibility type and the *Gpi* 111 allele had not been found in previous years. The novel strains either did not survive in the field during the winter of 1993 to 1994, were not reintroduced with infected seed, or did not become established in 1994, because none was detected after 1993. If any novel strains did escape detection in 1994, they probably did not survive until the 1995 growing season. The large sample size in 1995 greatly increased the chance of detecting any of these novel strains. In contrast, some isolates may have survived the winter, because A1 *Gpi* 86/100 and A1 *Gpi* 100/100 strains were found during all years between 1992 and 1994. These isolates could have overwintered in tubers, either in storage or in the field, or could have been established from infected seed.

During the spring of 1994, isolates were found with the A2 compatibility type and *Gpi* 100/111/122 for the first time. This strain also was most likely carried on infected seed from another location, because *Gpi* 122 had not been found previously in the Columbia Basin. Isolates of the A2 compatibility type and *Gpi* 100/111/122 dominated in 1995. These isolates had low DNA fingerprinting variation (15), indicating they were probably clonal, which is representative of the US-8 genotype. Isolates not of this genotype in 1995 were either A2 with *Gpi* 100/100/111 or *Gpi* 100/122 or A1 with *Gpi* 100/111/122. None of these combinations of traits had been found previously in the Columbia Basin. The A2 *Gpi* 100/122 could have arisen by the loss of the *Gpi* 111 allele from the US-8 genotype. The A2 *Gpi* 100/100/111 and the A1 *Gpi* 100/111/122 have not been given US designations at this point in time.

Sexual recombination could account for some of the new combinations of compatibility type and *Gpi* allozyme genotype found in 1993 and 1995. In a study analyzing isolates from Poland, a unique combination of *Gpi* alleles (90/90) was interpreted to be one line of evidence for sexual recombination (30). In the Columbia Basin, a cross between A1 *Gpi* 100/100 strains (present in 1992 and 1993) and A2 *Gpi* 100/111 strains (typical of the US-7 genotype, found previously in the United States) could have produced potential offspring with A2 *Gpi* 100/100 and A1 *Gpi* 100/111 strains. The potential offspring were found between 16 August and 9 September, near the end of the growing season. Both possible parents (A1 *Gpi* 100/100 and A2 *Gpi* 100/111) were found during mid-July in two counties in Oregon. Oospores of *P. infestans* can germinate and infect plant tissue as soon as 7 days after they are formed (7). Diseased tissue samples were not examined for oospores, however, so no physical evidence exists that supports sexual reproduction.

The sudden occurrence in 1994 and dominance in 1995 of US-8 genotype isolates may indicate this genotype is more fit than other strains. Isolates of the US-8 genotype infect young shoots from infected seed faster than other genotypes (22). This ability could provide the US-8 genotype with a competitive advantage in infecting new plant material during the spring. Additionally, the US-8 genotype is more aggressive in terms of tissue colonization, latent period, and spore production than other genotypes present in the Columbia Basin (23).

Analyzing the *Gpi* allozymes provided a fast method for characterizing isolates (13) but could not be used reliably in 1993 to designate genotypes due to the variation in DNA fingerprinting data, which was due to either sexual recombination or introduction of a diverse population on seed. Genotypes of isolates collected in all other years correlated well with the expected compatibility type and metalaxyl sensitivity for each isolate. In 1995, combinations of compatibility type and *Gpi* alleles not reported previously were found. Identification of genotype by *Gpi* allozyme assay alone, thus, was not sufficient to categorize isolates in the Columbia Basin. Additionally, compatibility and metalaxyl-

sensitivity tests should be continued. With both compatibility types present in the region, the potential exists for sexual recombination to occur. If recombination has occurred, the population of *P. infestans* is no longer clonal, and the use of *Gpi* genotypes for isolate identification may become obsolete. In addition, sexual recombination will lead to oospore formation in the field, potentially increasing the longevity of the pathogen in the field (7). As of yet, oospores have not been found in the Columbia Basin. If oospores do form, they may become a significant factor in the survival of *P. infestans* during winters and in the production of inoculum during the spring (7,25). Management of late blight then would be expected to be more difficult due to overwintering oospores and an aggressive population of *P. infestans*.

ACKNOWLEDGMENTS

Plant Pathology New Series 0249, Project 0678, Department of Plant Pathology, College of Agriculture and Home Economics Research Center, Pullman, WA. We thank B. A. Fry, S. B. Goodwin, and W. E. Fry for help with allozyme analysis; G. Pelter, T. Cummings, K. Pritchard, L. Douhan, J. Jaeger, and B. Geary for technical assistance; and L. M. Carris, D. A. Inglis, W. E. Fry, and S. B. Goodwin for comments on the manuscript.

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