A Test of Taxonomic Predictivity: Resistance to White Mold in Wild Relatives of Cultivated Potato

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ABSTRACT

A major justification for taxonomic research is its assumed ability to predict the presence of traits in a group for which the trait has been observed in a representative subset of the group. Taxonomy is regularly used by breeders interested in choosing potential sources of disease-resistant germplasm for cultivar improvement. We designed this study as an empirical test of prediction by associating resistance to white mold [caused by the fungal pathogen Sclerotinia sclerotiorum (Lib.) de Bary] to diverse potato (Solanum spp.) taxonomies and biogeography, using 144 accessions of 34 wild relatives of potato in Solanum sections Petota and Etuberosum. Tremendous variation for resistance to white mold occurs both within and among species. No consistent association was observed between white mold resistance and taxonomic series (based on a phenetic concept), clades (based on a cladistic concept), ploidy, breeding system, geographic distance, or climate parameters. Species and individual accessions with high proportions of white-mold-resistant plants have been identified in this study, but both often exhibit extensive variation and designation of either as resistant or susceptible must take this variation into account. Therefore, taxonomic relationships and ecogeographic data cannot be reliably used to predict where additional sources of white mold resistance genes will be found.

A primary function of classification is to construct classes about which we can make inductive generalizations (Gilmour, 1951).

One of the greatest assets of a sound classification is its predictive value (Mayr, 1969).

Taxonomy has long strived to construct predictive classifications (Vavilov, 1922; Gilmour, 1951; Michener, 1963; Rollins, 1965; Warburton, 1967; Mayr, 1969; Sokal, 1985; Stuessy, 1990; Daly et al., 2001). Warburton (1967) stated this goal clearly as, “[Prediction] means that one can describe a trait as characteristic of all members of a taxon before it has been verified for all. It also means that if organisms have been classified together as a taxon because they have all been found to share certain traits, they will later be found to share other traits as well.” For example, plant breeders use taxonomy to make their initial choice (or avoidance) of related (or unrelated) germplasm based on such statements as Species X is resistant to a particular disease, by choosing other accessions of this or related species. Clearly, not all accessions of a species share traits, but lacking prior evaluation data, taxonomy provides a useful guide to make inferences on unevaluated accessions, based on knowledge of a limited subset of the group.

The idea that traits should be associated with related organisms is perhaps universally accepted. As stated by Warburton (1967), “[This idea] is so deeply ingrained in the common sense of biologists that it sounds strange when formally stated.” This very utilitarian predictive component of taxonomy has long been used to justify taxonomy grant proposals, and cladograms serve as useful hypotheses to associate to a variety of traits as diverse as morphology, anatomy, ecology, development, disease epidemiology, and behavior (Harvey et al., 1966; Baum et al., 2005). While the association of traits to taxonomy is only as reliable as the taxonomy, revised molecular-based phylogenetic results are uncovering previously unexpected associations and have shown tremendous economically important discoveries of use to society. For example, Daly et al. (2001) summarize the enhanced predictive component of new phylogenies to synthesis of a nonprotein amino acid, glucosinolates production, N fixation, and taxol biosynthesis. Adams et al. (2001) show associations to new phylogenies and the loss of a telomere repeat in the plant order Asparagales. Crandall (1999) shows the use of an AIDS phylogeny to detect viral recombination and to identify modes of transmission.

The present study is the first test of the association of potato taxonomy to disease resistance (here, to the fungal disease white mold) and, to our knowledge, the first experiment designed to test prediction without prior knowledge of traits. For example, while the studies mentioned above clearly demonstrate the enhanced value of molecular phylogenies and prediction, they do not mention the many instances surely overlooked where prediction does not occur. All of the above studies (except the AIDS example) show associations at the ordinal level, and the present study investigates associations at a much lower taxonomic level (sections Petota and Etuberosum of Solanum). Perhaps the predictive power of taxonomy varies with traits or taxonomic levels.

While white mold is not a widespread problem in potato, it can be a serious disease in certain environments. It is becoming more important in production systems with high fertility regimes and sprinkler irrigation, where dense foliage can remain wet long enough for the fungus to become established (Powelson, 2001). White mold is a serious and widespread disease in soybean [Glycine max (L.) Merr.] (Kim et al., 2000), so it is especially prevalent in potato in regions where soybean is grown as a rotation crop. While white mold is controlled to some extent with frequent fungicide applica-

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Abbreviations: PDA, potato dextrose agar; PET, potential evapotranspiration; QTL, quantitative trait loci.

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tions, host plant resistance would provide an effective, environmentally safe, and inexpensive method of control of the disease. Our study is the first to screen wild potato species for resistance to white mold. Resistance to every major potato disease has been found in wild \textit{Solanum} species (Jansky, 2000), and these species often contribute resistance to multiple diseases (Jansky and Rouse, 2000). Most of the species are directly crossable to the cultivated potato, so resistance genes can be accessed easily.

We chose a disease test because the wild potato species provide a wide range of resistances to diseases, they are readily transferred to cultivated potato, and disease resistances in wild species provide one of the major justifications for collecting and storing germplasm in genebanks. Species-specific statements of the breed-disease resistances in wild species provide one of the most dramatic examples of the potential for resistance in wild species. Most of the species are directly crossable to every major potato disease has been found in wild \textit{Solanum} species (Jansky, 2000), and these species often contribute resistance to multiple diseases (Jansky and Rouse, 2000). The purpose of our study is to produce a comprehensive and statistically significant set of data for disease resistance to \textit{S. sclerotiorum} in wild species of sections \textit{Petota} and \textit{Etuberosum}, to search for associations of these resistances to taxonomic and biogeographic fac-
tors, and to make these data available as a future resource to test other possible associations (for example, alternative taxonomies).

\section*{MATERIALS AND METHODS}

\subsection*{Plant Material}

The \textasciitilde5000 cultivated and wild potato accessions in the U.S. genebank in Sturgeon Bay, WI, were originally collected in 12 countries in the Americas, covering most of the distribution area of wild potatoes that occur from Colorado (USA) to Chile and Uruguay. Species richness is high in central Mexico at 20° N lat, and in the southern hemisphere, particularly in the Andean highlands between 8 and 20° S lat (Hijmans and Spooner, 2001). Our study represents 14 of the 19 tuber-bearing series of Hawkes (1990) and all four clades of sect. \textit{Petota} based on chloroplast restriction site phylogenies as discovered by the combined studies of Spooner et al. (1993), Rodríguez and Spooner (1997), Spooner and Castillo (1997), and Castillo and Spooner (1997) (Fig. 1). We obtained true potato seed of each accession from the NRSP-6 Potato Genebank, and studied between one and seven accessions of each of 31 ingroup (sect. \textit{Petota}) and three outgroup (sect. \textit{Etuberosum}) species (Fig. 1: accessions examined and data are available in the Supplemental Table 1). Species names followed recent taxonomic changes as outlined in Spooner and Hjimans (2001) and Spooner et al. (2004).

\subsection*{Disease Screening}

Seeds were soaked in 1500 ppm gibberellic acid for 24 h to break dormancy. They were then sown in flats in a peat-based potting mix (Pro-Mix, Premier Horticulture, Dorval, QC, Canada) and grown in a greenhouse with a 18-h photoperiod. After 3 wk, each seedling was transplanted to an individual cell of a 50-cell flat. Half of the seedlings of an accession were placed in one flat and half in another flat, to construct two replications. Seedlings were inoculated 2 to 3 wk after transplanting. Seed germination and seedling survival varied, and the number of seedlings evaluated per accession ranged from four to 224 (mean 37).

The inoculation procedure was adapted from a soybean protocol that correlates with field evaluations (Kim et al., 2000). White mold inoculum was prepared by placing a sclerotium of \textit{S. sclerotiorum} [nap bean (\textit{Phaseolus vulgaris} var. vulgaris) isolate] onto a plate of potato dextrose agar (PDA) (30 g L$^{-1}$) and allowing it to germinate in the dark at room temperature. A plug was removed from the edge of the colony and placed on a fresh PDA plate. The second PDA plate contained a ring of sterile filter paper disks (8-mm diam.)
along the outer edge. The expanding colony was observed twice daily. As soon as the colony grew over the ring of disks, the disks were used to inoculate seedlings.

Seedlings were first misted thoroughly with distilled water. Then, each plant was inoculated by placing a paper disk on an older leaflet, mycelial side down. When all the seedlings in a flat were inoculated, the plants were misted again, taking care to avoid disturbing the paper disks. A plastic dome was thoroughly misted inside and placed over the flat. Each flat was placed in a growth room with diffuse light and a temperature of 21°C. Flats were not disturbed until the fourth day after inoculation. Four days after inoculation, the dome was removed from each flat and each plant was scored for its response to the fungus. A plant was given a score of one if it was alive and zero if the fungus had caused the stem to collapse and the plant to die. This process was repeated every day for 14 d. A survival score was determined for each plant on the basis of the number of days it remained alive.

**Statistical Analysis**

Because resistance evaluations were terminated after 14 d of observation, the survival data were considered to be censored (data collection stopped at a specific time). Censored data are most appropriately analyzed using nonparametric methods based on rank scores. The Mann–Whitney test (Mann and Whitney, 1947) was used when a comparison between two groups was made. The Kruskal–Wallis test (Kruskal and Wallis, 1953) was used when comparisons among more than two groups were made. Post hoc pairwise comparisons following a significant Kruskal–Wallis test were performed using the Mann–Whitney test with an appropriate Bonferroni correction. To determine the relative contributions of species, accession, and individual plants to the variation seen in the days to infection, a linear model was fit with random effects of species and accession. The contribution due to individual plants was reflected in the residual variability. Because the data consisted of discrete counts (and were thus of questionable normality), as well as censored at 14 d, the results of the model were used as a guide in quantifying the variability present.

To test the question of whether geographic provenance of samples is a predictor of disease resistance, we analyzed biogeographic variables. The analyzed response variables were the accumulated percentages of dead plants on each day (Days 1 to 14), resulting in a time course. For each point in time, we analyzed both spatial and environmental variables.

A caveat with spatial data concerns was the (expected) similarity between neighboring data points for a given variable called autocorrelation (the closer the more similar). Patterns of autocorrelation in data can create false positive results in spatial analysis, but not necessarily. In any case, the analysis of spatial autocorrelation can be a useful tool to investigate mechanisms at different spatial scales (Diniz-Filho, 2003). Diniz-Filho et al. (2003) suggested the use of a general approach to test for spatial autocorrelation in data by conforming to the same resolution. The accumulated percentages of dead plants on each day for those accessions divided by the number of classes (Bonferroni criterion, Diniz-Filho et al., 2003).

The spatial filter method consists basically of extracting positive eigenvectors from a connectivity matrix and using them directly as spatial predictors or filters in a regression framework. Eigenvectors with high positive eigenvalues tend to capture large-scale spatial effects; those with smaller values have more local patterns. The method is related to a spectral decomposition (Fourier analysis) and the classical statistical factor analysis. Likewise, the spatial factors or eigenvectors are used to capture and express latent variables. The method allows adding eigenvectors until the residuals minimize autocorrelation as tested by the Moran coefficient.

Finally, we used a linear partial regression analysis model (filtered by spatial factors). Thus, the linear partial regression model could take into account the effect of predictor variables unbiased by spatial effects as well as determining the effect of space alone.

Several environmental factors that have been shown to be associated with large-scale richness gradients were used to assess the effect of environmental provenance on disease response in the partial regression analysis: (i) potential evapotranspiration (PET); (ii) actual evapotranspiration; (iii) mean daily temperature in the coldest month; (iv) annual mean temperature; and (v) annual rainfall. The dataset is available for the whole western hemisphere (Rangel et al., 2005) and was constructed for a 1° grid resolution from sources at www.sage.wisc.edu/atlas (verified 27 July 2006; T.F.L.V.B. Rangel, 2005, personal communication). This dataset was combined with our data by conforming to the same resolution. The accumulated percentages of dead plants on each day for those accessions falling into the same grid-cell were averaged. A final database of 100 grid-cells was established and used for further analysis.

The library spdep spatial dependence of the package R (http://www.r-project.org; verified 27 July 2006) was used for exploratory spatial analysis; for eigenvector filtering and partial regression the package SAM (Rangel et al., 2005).

**RESULTS**

**Comparisons among Species**

Significant ($P = 0.05$) differences in mean rank scores were detected among the 34 species in the trial. Within the context of our study, the most resistant species was *S. commersonii*, while the most susceptible was *S. infundibuliforme* (Fig. 2).

When pairwise comparisons were made between species, significant differences ($P = 0.05$, Bonferroni correction for multiple comparisons) were detected for 247 of the 561 comparisons (44%). The most resistant species, *S. commersonii*, was significantly more resistant than all other species. The most susceptible species, *S. infundibuliforme*, was more susceptible than 29 of the 33 species.

**Comparisons among Accessions within Species**

Mean rank score comparisons were made among accessions within each of the 25 species in which three or
more accessions were evaluated for white mold resistance. Significant mean rank score differences among accessions were detected for 22 (88%) of the species. The large amount of interaccession variation for white mold resistance can be visualized using box plots. Each plant score is based on the number of days that the plant survived during the 14-d observation period. A box plot of six accessions of *S. commersonii*, the most resistant species, illustrates inter- and intraaccession variation (Fig. 3). While the median score of five of the accessions is 14 d, one accession (PI 498418) had a much higher proportion of susceptible plants, with a median score of 1 d. In addition, highly susceptible plants were observed in all accessions. *S. palustre*, the second most resistant species, showed a similar pattern, with five accessions having median scores of 14 and one accession with a median score of one. An even greater amount of inter-accession variability is illustrated among six accessions of *S. bulbocastanum* (Fig. 3). All plants in one accession (PI 558379) survived for the entire 14-d observation period. In contrast, the median score for two accessions (PI 310960 and PI 347757) was 1 d. In addition, the range in variation among accessions is apparent. In one accession (PI 545751) 50% of the plants survived for a narrow range of 8–10 d, while in another (PI 347757) the range was 0 to 14 d.
of our study, the most resistant series was $\text{S. commersonii}$, and most susceptible ($\text{S. infundibuliforme}$) species, respectively. When series containing multiple species were evaluated, however, species were not consistently grouped based on resistance. For example, series Tuberosa contains $\text{S. verrucosum}$, $\text{S. kurzianum}$, $\text{S. brevicaule}$, and $\text{S. microdontum}$, which are among the most susceptible species. However, it also contains $\text{S. sparsipilum}$, which is among the most resistant species (Fig. 2). Similarly, rank scores of species in the series Conicibaccata range from low ($\text{S. longiconicum}$) to high ($\text{S. violaceimarmoratum}$).

There were 10 pairs of rank score comparisons among the five chloroplast clades, and four (40%) were significantly different at $P = 0.05$ using the Bonferroni correction. Chloroplast clade 5 was significantly more resistant than each of the other four clades. The remaining six paired comparisons revealed no differences. Clade 5 is composed of three outgroup species, two of which are among the most resistant species ($\text{S. etuberosum}$ and $\text{S. palustre}$). However, the other member of the clade ($\text{S. fernandezianum}$) is not among the most resistant species.

Ploidy groupings indicated that tetraploid species were more susceptible than both diploid and hexaploid species. Mean rank scores for the diploid group were not different from those of the hexaploid group. Five of the six tetraploid species were grouped among the most susceptible species. However, $\text{S. acaule}$ is among the resistant species. One hexaploid species ($\text{S. schenckii}$) was among the most resistant species, while the remaining two ($\text{S. ehrenbergii}$ and $\text{S. demissum}$) were not.

### Biogeography

Figure 4 shows average days to survival and the distribution of the accessions examined. Examination of this map reveals no striking geographic pattern for resistance. This is confirmed by statistical analysis. Following the basic protocol outlined in (Diniz-Filho and Bini, 2005) and confirmed by exploratory data analysis, a first set of 20 spatial filters was established for use in partial regression analysis. This was then reduced to only significant filters for each day of the disease response. The correlograms show that most of the spatial structures were captured by the eigenvectors except for distances less than 500 km (see Fig. 5 for a representative example). Interestingly, the Moran $I$ coefficient of the response variable oscillates around zero between relatively high values and at significant levels. This may indicate a checkerboard-like pattern.

Further analysis of a combined data set of spatial and environmental variables in a linear partial regression revealed that the spatial component alone (as captured by the eigenvectors) has only an average level of 2% decreasing from 5.6% on Day 2 to 0.7% on Day 12 (see Fig. 6). This trend coincides with the expectation and the low levels suggest a very minor role of the spatial geometry.

The regression model as a whole explains, on average, <40% of the variation. From the remaining predictors, no consistent pattern could be established for the envi-
Environmental variables of interest except for PET, although the trend across time of explained variability seems to suggest so. Even in the case of PET, the regression coefficient is low (<0.01) during the time course. In summary, the spatial and environmental origins of accessions show no clear relationship with the accumulated percentages of dead plants on each day.

DISCUSSION

Breeders are constantly searching for useful sources of genetic variability, and wild species have provided much of this resistance (Lenne and Wood, 1991). If a breeder can use taxonomic or geographic information to identify the most likely sources of valuable genes, then the efficiency of the search can be improved dramatically. However, it does not appear that, for white mold resistance, these tools can be used to significantly refine the screening process.

Significant variation in mean rank scores for white mold resistance was identified among taxonomic series, chloroplast clades, and ploidy levels. However, none of these grouping criteria consistently identified the most resistant species. Therefore, a breeder would not be able to focus on a series, clade, or ploidy level with the expectation that all species in the group will be resistant.

Significant variation in mean rank scores was also identified among species. The most resistant species were *S. commersonii* and *S. palustre*. Consequently, a breeder will most likely find white mold resistance genes in plants of these species. However, while the species may be characterized as resistant, it is important to emphasize that considerable variation exists among accessions and among plants within each accession. Knowledge of intra-accession variation has led to a process of fine screening, whereby individuals within resistant accessions are screened and the most resistant individuals are maintained clonally for use by breeders (Bamberg et al., 1986; Douches et al., 2001; Zlesak and Thill, 2004). This concept can be extended to most of the species included in this study, as 84% were variable among accessions. So, while...
resistant plants are common in *S. commersonii* and *S. palustre*, some plants (and in some accessions, many plants) will be susceptible to white mold. Conversely, the most susceptible species contained few, if any, resistant plants. Considering the most susceptible species *S. infundibuliforme* and the second most susceptible species *S. hjertingii*, two out of five accessions and two out of six accessions, respectively, did not contain any plants that survived the 14-d observation period. Searching for resistant plants in these species would not be efficient. However, accessions of some intermediately resistant species contain a high proportion of resistant plants. For example, *S. bulbocastanum*, *S. chomatophilum*, *S. polyadenium*, and *S. stenophyllidium* all had one accession with a median score of 14 d, so a breeder would expect to find strong resistance in half of the plants screened.

Little is known about the original distribution of *S. sclerotiorum*, as the published distributional ranges are based on only very scattered records. The Commonwealth Mycological Institute (1986) has published maps of fungal distributions, and Farr et al. (2002) represents the latest compilation of host–fungal pathogen records. All are based on only scattered reports worldwide, and the fungus used in this study infects a wide range of plants in different families.

The genetic basis of white mold resistance has not yet been determined in potato. In soybean, resistance is considered to be a quantitative trait. Some components to resistance are due to avoidance mechanisms that result from plant morphology, while others are physiological (Ender and Kelley, 2005). Quantitative trait loci (QTL) for both types of resistance have been identified. Similarly, in rapeseed, three QTL have been found to be associated with leaf resistance in seedlings, and three with stem resistance in mature plants (Zhao and Meng, 2003). Physiological resistance in sunflower may be more simply inherited. Rönike et al. (2004) identified two AFLP fragments linked to white mold resistance. In our study, we looked only at a single component of resistance (physiological resistance) in leaf tissue of seedlings. The plant–pathogen interaction resulted in two distinct outcomes. In resistant plants, a lesion began to form, but within a few days it ceased to expand and the plant recovered from the inoculation. In susceptible plants, the lesion continued to expand, eventually reaching the stem, where it girdled and killed the plant.

If resistance to this fungus represents convergent evolution, then we would expect resistant phenotypes to be restricted to regions of selection pressure, independent of species. In the case of a widespread potato species, one would expect resistant phenotypes to appear (or increase in frequency) as the selection pressure increases; this contrasts with the taxonomic prediction expectation. The strength of selection pressure due to this disease would be important in both taxonomic and geographic prediction models. *Sclerotinia sclerotiorum* may exert weak selection pressure on host plants, especially compared with environmental stresses that provide more consistent selection pressure, such as frost or drought tolerance.

Complex inheritance can also reduce predictivity with taxonomy or geography, as the phenotype (disease resistance) may be caused by diverse and nonhomologous genes. The complex inheritance of white mold resistance in other crops suggests that several genes may be involved in potato as well. Despite the lack of predictivity, the existing taxonomic information, combined with our identification of accessions with high levels of resistance, can help identify putatively diverse sources of resistance genes.

Geography and ecology have also been associated with other traits in potato. For example, associations have been found between the altitude of origin and the frost.
killing temperature of *S. acaule* (Li et al., 1980); between altitude and resistance to potato leafhopper [*Empoasca fabae* (Harris)] (Flanders et al., 1992); and between altitude and glycoalkaloid content (Ronning et al., 2000). Van Soest et al. (1983) found that there was a concentration of wild potato species with cyst nematode (*Globodera pallida*) resistance near Potosí, Bolivia. Van Soest et al. (1984) concluded that wild potatoes with resistance to late blight occur near the tropics of Capri- corn and Cancer. Flanders et al. (1992) found that species from hot and arid areas were associated with resistance to Colorado potato beetle [*Leptinotarsa decemlineata* (Say)], potato flea beetle (*Epitrix subcrinita* LeConte), and potato leafhopper. Species from cool or moist areas tended to be resistant to potato aphid [* Macrosiphum euphorbiae* (Thomas)]. Flanders et al. (1997) found differences between geographic areas for insect resistances in potatoes. Hijmans et al. (2003) investigated the extent to which taxonomic, ecological, and geographic factors can be used to predict frost tolerance in wild potato species. They used screening data for 1646 samples from 87 species that had been collected in 12 countries in the Americas. They found a strong association of frost tolerance with species and to a lesser extent with taxonomic series. They also found significant geographic clustering of areas with wild potatoes with similar levels of frost tolerance. Moreover, there was a greater chance of finding wild potatoes with high levels of frost tolerance in areas with a yearly mean minimum temperature below 3°C than in warmer areas. However, temperature was only a weak predictor of frost tolerance.

The complexity of disease resistance introduces challenges to the search for predictive associations to disease. Genomic studies have revealed a suite of common patterns in the evolution of disease resistance genes. (i) Many disease resistance genes exist in tandemly duplicated gene clusters that give rise to resistance gene families. (ii) Disease resistance gene variation can arise from both intra- and intergenic recombination and gene conversion. Such recombination provides a mechanism for generating new race specificities either by reshuffling existing genes or by the creation of novel resistances to biotypes to which neither parental allele was resistant. (iii) Resistance gene clusters appear to evolve very rapidly. (iv) Several resistance genes show remarkable similarity to previously identified recognition specificities from a diverse group of organisms, with a common motif being leucine-rich repeats. These are particularly subject to adaptive selection. (v) Transposable elements have been associated with some resistance gene clusters. They may become activated by pathogen-induced stress to provide a selective advantage through further allelic diversity and genetic plasticity in gene complexes. (Grube et al., 2000; Richter and Ronald, 2000; Hulbert et al., 2001). It is possible, therefore, that disease gene evolution may occur faster than plant speciation, disrupting a concordance between resistance and a phylogenetically-based taxonomy. Related to this is the as-yet-unproven concept that such rapid genetic reshuffling of disease resistance genes may provide a common evolutionary origin to resistances to different pathogens. The tight clustering of a dual resistance gene in tomato (*Lycopersicon esculentum* Mill.) (resistance to a nematode and a potato aphid) suggests that this may be the case (Rossi et al., 1998). Also related to this is the knowledge that at least in *Arabidopsis*, disease resistance genes form a huge (an estimated 1%) part of the genome (Meyers et al., 1999) and likely form a complex set of duplicated and rearranged regions.

Despite these complications of complex genetic control, almost all traits are complex, and there are no empirical studies or theory to suggest which traits are likely to be subject to taxonomic prediction. The very traits cited as examples of the predictive power of taxonomy by Daly et al. (2001) are all under polygenic control. For example, glucosinolate production has been shown to be under the control of three to seven genes, depending on the cross (Magrath et al., 1993; Mithen et al., 1995; Li et al., 2001; Soedhi et al., 2002). A dozen genes are involved in the control of disease resistance genes (reviewed in Jansky, 2000). Major dominant genes also confer resistance to *Potato virus X*, *Potato virus Y*, and *Potato leaf roll virus* (reviewed in Jansky, 2000). In addition, one or two major genes appear to provide resistance to common scab (*Streptomyces scabies*) (Alam, 1972), ring rot (*Clavibacter michiganensis* subsp. *sepedonicus*) (Kriel et al., 1995) and Verticillium wilt (*Verticillium dahliae* and *V. albo-atrum*) (Lynch et al., 1997; Jansky et al., 2004). There are no published reports on the inheritance of resistance to white mold in potato, although inheritance studies are underway.

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