

Breeding for Disease Resistance in Potato

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I. INTRODUCTION

The potato (*Solanum tuberosum* L.) is one of the world's five most important food crops in both total production and area cultivated (FAO 1999). Yield losses due to disease are particularly high in potato, due in part to asexual propagation. An estimated 22% of potato yield is lost per year due to diseases and pests (Ross 1986). Based on a total 1998 world production of nearly 300 million tonnes (FAO 1999), this amounts to over 65 million t of potatoes destroyed annually by pathogens and pests. In addition, costs of pesticide application to prevent further loss are significant. For example, a single application of fungicide cost potato growers in the United States \$5–11 million in 1994 (Stevenson 1994). The cost for control of early blight alone in the United States and Canada in 1994 was estimated to be \$21–44 million. Furthermore, although pesticides are used heavily for potato production, government agencies continue to evaluate pesticide labels with the goal of reducing their use. Therefore, the development of disease resistant potatoes is becoming increasingly important in potato breeding programs.

Reviews of potato breeding strategies have discussed breeding for disease resistance (Ross 1986; Tarn et al. 1992; Bradshaw and MacKay 1994). However, tremendous progress has been made in recent years. For example, genetic mapping of a number of resistance genes has created interesting insights into the evolution of resistance, screening for pathogens has become much more refined with the use of the polymerase chain reaction and advanced immunological techniques, and the introduction of the A2 mating type of *Phytophthora infestans* into Europe and the United States has prompted a renewed interest in breeding for late blight resistance. Consequently, this review considers publications from the 1990s. A recent review of ploidy manipulations in potato breeding (Ortiz 1998) lists germplasm resources for disease resistance and genetic analyses of disease resistance traits, but does not discuss them in detail. This review emphasizes diseases of major concern in North America, most of which are serious problems worldwide. Other

significant diseases, such as bacterial wilt, wart, and dry rot are reviewed elsewhere (Bradshaw and MacKay 1994; Ross 1986; Sequeira and Kelman 1976).

Because the cultivated potato is tetraploid, genetic progress is often slow. In addition, a lack of understanding of the physiology and genetic control of disease resistance has hindered progress in this area. However, tremendous genetic diversity exists in the wild relatives of potato, and resistance genes for every major disease have been found in this gene pool. An understanding of ploidy manipulations and the development of breeding strategies to utilize the genetic diversity in *Solanum* species are creating a significant impact on the development of disease resistant clones. In addition, the potato is particularly amenable to in vitro manipulations, so tissue culture screening methods and transgenic forms of disease resistance are also playing a role in breeding for disease resistance.

II. LATE BLIGHT

The fungus *Phytophthora infestans* (Mont.) de Bary causes late blight, which is considered to be the most destructive disease of potato (Wastie 1991). Late blight causes extensive foliar destruction during the growing season and tuber decay in storage. The pathogen is controlled mainly by the heavy use of fungicides (Freytag et al. 1994).

The introduction of the A2 mating type of *P. infestans* into Europe in the mid-1980s (reviewed by Fry and Goodwin 1997) and later into the U.S. and Canada (Deahl et al. 1991) has sparked renewed interest in breeding for resistance to late blight. In recent years, new genotypes of *P. infestans* have been detected and *P. infestans* populations are more genetically variable. In contrast, before 1990, the US-1 *P. infestans* genotype, which is sensitive to the fungicide metalaxyl and is an A1 mating type, was believed to predominate in North America (Inglis et al. 1996).

R-genes are responsible for major gene (race-specific) resistance to late blight. They exhibit Mendelian inheritance as single dominant factors and cause a hypersensitive response in potato (El-Kharbotly et al. 1994). However, the introduction of new *R*-genes into host plants has resulted in the development of corresponding virulent races of *P. infestans* that can overcome resistance. The number and frequency of complex *P. infestans* isolates has increased dramatically in recent decades, presumably due to the products of *R*-gene based breeding programs (Wastie 1991). Consequently, most breeders have abandoned strategies based on *R*-genes, and breeding efforts today emphasize "general" (race-

nonspecific) resistance, which appears to be polygenically inherited (Leonards-Schippers et al. 1994).

General resistance is a quantitative trait believed to be polygenically inherited. However, phenotypes of resistance due to *R*-genes and those caused by general resistance overlap (Meksem et al. 1995). General resistance to *F. infestans* is difficult to test and often cannot be separated from the effects of *R*-genes (Leonards-Schippers et al. 1992). Consequently, considerable effort in recent years has been directed toward the development of laboratory, greenhouse, and field assays that will recognize components of general resistance.

Late blight resistance does not appear to have been a major prerequisite for the success of current potato cultivars. However, due to the increasing frequency of *P. infestans* strains with fungicide resistance, the difficulty of controlling the fungus with conventional fungicides, and concerns about environmental effects of fungicides, many breeders now emphasize the development of late blight resistant cultivars (Wastie 1991).

A. Germplasm Resources

1. Cultivars and Advanced Selections. Wastie (1991) suggests that resistance genes within *S. tuberosum* Group Andigena and Group Tuberosum are sufficient for the development of late blight resistant cultivars. The limiting factor for the development of resistant cultivars is the availability of efficient, cost-effective resistance screening methods.

Several examples of resistance in cultivated germplasm have been reported in recent years. For example, Platt and Reddin (1994a,b) identified foliar and tuber resistance in a number of cultivars and breeding materials. Colon et al. (1995b) field-tested 22 *R* gene-free cultivars, introduced between 1900 and 1954, and found that some exhibited higher levels of partial resistance than those of currently grown field resistant cultivars. The partial resistance in these old cultivars is apparently durable, since the level of resistance today is similar to that at the time they were introduced. However, these clones are late maturing. In another study, utilizing 147 cultivars and breeding lines, 'Pike' and 'Snowden' were found to be more resistant than other North American cultivars, while 'Zarevo' was the most resistant of the European cultivars (Douches et al. 1997). Seven of the advanced breeding lines were equivalent to 'Zarevo' in infection levels. Swiezyński et al. (1993a) reported that *R*-alleles other than *R1*, *R2*, *R3*, *R4*, and *R10* (which are believed to be the only ones present in cultivars) exist in 'Bzura' because the cultivar was resistant to an inoculum with virulence 1.2.3.4.7.10.11.

The Canadian cultivar AC Brador (Tarn et al. 1995b) as well as the Cameroon cultivars CIPIRA, TUBIRA, and BAMIRA (Martin et al. 1995) exhibit extremely high field resistance to late blight. Finally, two potato breeding selections, B0718-3 and B0767-2, exhibit low levels of defoliation under late blight pressure (Goth and Haynes 1997). The resistance in these clones was presumably derived from PI 383470B, an introduction of *S. tuberosum* from India clone 1033. Partial resistance to foliar infection is present in the breeding clones AWN86514-2, B0692-4, B0718-3, and B0767-2 (Haynes et al. 1998).

In regions of the world where potato production from true seed is significant, populations with uniform levels of late blight resistance would be especially valuable. However, the level of resistance to late blight in 34 true potato seed lines tested by Ramaraj and Nanjan (1994) was low or moderately low. None of the lines was resistant or even moderately resistant.

Reports of tuber resistance are much less common than those of foliar resistance. Nevertheless, tuber resistance to late blight does exist in some commercial cultivars and advanced potato selections, such as AWN86514-2 (Platt and Reddin 1994a,b; Dorrance and Inglis 1998; Corsini et al. 1999).

It is possible that cultivars resistant to late blight before the introduction of the A2 mating type are no longer resistant to new *P. infestans* genotypes. However, Inglis et al. (1996) found that cultivars ranked nearly the same for resistance to new isolates of *P. infestans* as they did for the US-1 isolate (A1 mating type) before 1990. Kadish et al. (1990) observed that, while the fitness of metalaxyl-resistant isolates of *P. infestans* is higher than that of metalaxyl-susceptible isolates on susceptible cultivars, foliage resistance of some cultivars was still effective.

2. Wild and Cultivated Relatives. Three types of resistance have been identified in clones with wild *Solanum* species in their pedigrees (Swiezyński et al. 1991). The first is resistance due to major, dominant genes. This is the most common, is expressed in both foliage and tubers, and is sometimes, but not always, due to *R*-genes. The second is resistance in foliage and tubers associated with late maturity. And the third is resistance in whole tubers, but not tuber slices.

Colon et al. (1995a) reported higher levels of resistance among wild *Solanum* species than within *S. tuberosum*, especially for infection efficiency and lesion growth rate, and also for sporulation capacity. *Solanum verrucosum* plant introductions appear to contain a mixture of resistance mechanisms, which may include resistance due to *R*-genes (Tooley 1990). Rivera-Peña (1990) found that both complex race-specific

resistance and a high level of general resistance exist in *S. demissum*, *S. verrucosum*, *S. iopetalum*, and *S. brachycarpum*. Foliar resistance has also been identified in the cultivated diploid *S. tuberosum* Group Phureja (Hawkes 1990; Canizares and Forbes 1995) and *S. bulbocastanum* (Helgeson et al. 1998). According to Hawkes (1990), late blight resistance also exists in *S. berthaultii*, *S. bulbocastanum*, *S. circaefolium*, *S. demissum*, *S. microdontum*, *S. pinnatisectum*, *S. polyadenium*, *S. stoloniferum*, *S. tarijense*, *S. tuberosum* Group Andigena, *S. vernei*, and *S. verrucosum*. The Inter-Regional Potato Introduction Station (NRSP-6) reports resistance in 19 species, including Group Andigena, *S. bulbocastanum*, *S. demissum*, *S. tuberosum* Group Phureja, *S. pinnatisectum*, and *S. stoloniferum* (Bamberg et al. 1994).

B. Genetic Control of Resistance

1. Qualitative. The mapping of *R*-genes to potato chromosomes has led to interesting speculation about evolutionary and functional relationships among resistance genes. For example, the *R1* locus has been mapped to chromosome V, in the same genomic region as a locus conferring extreme resistance to potato virus X (*Rx2*) (Leonards-Schippers et al. 1992). In tomato, a gene controlling resistance to *Pseudomonas syringae* pv. tomato has also been mapped to chromosome V. Tomato and potato chromosomes are highly homeologous (Bonierbale et al. 1988; Gebhardt et al. 1991), so all three genes occupy similar regions. Resistance genes acting against different plant pathogens may be located in a similar genomic position because they are functionally related as allelic variants of the same locus or evolutionarily related as products of tandem duplication.

The *R*-gene allele *R3* has been mapped to a distal position on chromosome XI (El-Kharbotly et al. 1994). Alleles *R6* and *R7* have been mapped to the same region (El-Kharbotly et al. 1996b). The three DNA segments may be alleles of the same locus, or alleles at closely linked loci. The *R3*, *R6*, *R7* cluster occurs in the same chromosomal position as that of the *Fusarium oxysporum* resistance locus *I2*, which maps to the same segment of chromosome XI in tomato (Segal et al. 1992). Therefore, El-Kharbotly et al. (1996b) suggest that the genes encoding *I2* in tomato and *R3*, *R6*, and *R7* in potato are members of a homologous gene family.

In another example of apparent evolutionary similarities among resistance genes, Leister et al. (1996) used polymerase chain reaction (PCR) primers derived from an *Arabidopsis thaliana* bacterial resistance gene

to amplify potato DNA. One amplification product was linked without recombination to allele *R7*.

Although *R*-genes exhibit simple Mendelian inheritance, some researchers have reported deviant segregation ratios in inheritance studies. El-Kharbotly et al. (1996c) have demonstrated that the presence of additional genetic factors may cause distorted segregation of *R*-genes. The modifying gene in the case of *R1* appears to be a dominant suppressor. Ordonez et al. (1997) observed hypersensitivity, like that produced by dominant *R*-genes, in progenies of parents that did not express *R*-genes. Therefore, they analyzed segregation for resistance in progenies of parents with and without *R*-genes, and found that their data could not be explained solely by Mendelian inheritance of dominant *R*-genes. A more likely explanation is the presence of a dominant suppressor for *R*-genes, as described by El-Kharbotly et al. (1996c). Specific suppressors are elicited by specific isolates of the fungus, and the potato genes for production of these suppressors segregate independently of *R*-genes. Therefore, Ordonez et al. (1997) recommend testing for *R*-genes in progenies of putatively *R*-gene-free parents when breeding for general resistance. Colon et al. (1995c) also discovered a susceptible parent that produces resistant offspring, probably due to an epistatic interaction.

Isolation of *R*-genes is difficult because their products are unknown. El-Kharbotly et al. (1996a) suggest that insertional mutagenesis (transposon tagging) will be useful for the cloning of *R*-genes. They have created transgenic clones carrying the *Dissociation* transposable element at known positions on the potato restriction fragment length polymorphism (RFLP) map. These clones will be used for targeted transposon tagging experiments to locate *R*-genes and other potato resistance genes.

2. Quantitative. The genetic relationship between *R*-genes and genes responsible for horizontal resistance is unclear (Leonards-Schippers et al. 1992). However, a quantitative trait locus (QTL) contributing to late blight resistance is located in the same segment of potato chromosome V as allele *R1* (Leonards-Schippers et al. 1994). It appears, then, that *R*-genes may not be independent of genes for general resistance. However, Ordonez et al. (1998) suggest that at least some *R*-genes segregate independently of those for general resistance.

Using amplified fragment length polymorphism (AFLP) markers in a tetraploid population, Meyer et al. (1998) identified a multi-allelic locus at the proximal end of chromosome VIII that contributes significantly to late blight resistance. Although no late blight resistance genes or QTLs

have been mapped previously to this position, Leister et al. (1996) mapped a resistance gene analogue to this region in a diploid population.

Based on a comparison between genetic and phenotypic correlation coefficients between infection efficiency and area under the disease progress curve (AUDPC), infection efficiency and lesion size, and lesion size and spore density, Birhman and Singh (1995) concluded that environmental and/or non-additive genetic component(s) are important factors for late blight resistance. On the other hand, the correlations for lesion size, spore density, and AUDPC with spore production, and for lesion size with AUDPC, have negligible environmental and non-additive components. Broad-sense heritability was high for both lesion size (0.84) and spore density (0.75), so selection based on those traits should result in genetic gain. Ordonez et al. (1998) observed a significant genotype by isolate interaction ($P=0.0001$) when measuring lesion expansion rate, indicating isolate specificity in the expression of resistance.

Components of tuber blight resistance have been elucidated (see Section IIC1 and IID1). However, efforts to determine the inheritance of resistance in tubers have not been emphasized (Wastie 1991). Therefore, genetic studies of this important resistance trait are warranted.

C. Physiology of Resistance

There are numerous papers dealing with systemic induced resistance responses of potato plants when challenged with *P. infestans*. This review will consider reports of responses of genetically resistant clones to the pathogen.

1. Whole Plant. Potato fields may be affected by both late blight and early blight (see Section III). However, the biochemical pathways leading to late blight resistance appear to be unrelated to those for early blight resistance (Fry and Shtienberg 1990). Furthermore, compared to early blight, the effects of plant senescence on expression of late blight resistance are small. Therefore, genetic resistance has a more significant effect on expression of disease symptoms than does plant age.

There is a general belief that late-maturing cultivars are more resistant to late blight because their foliage is physiologically younger when the fungus appears (Dorrance and Inglis 1997). In addition, the early development of a dense canopy in early-maturing cultivars can create a microenvironment that encourages the reproduction and spread of *P. infestans* (Inglis et al. 1996). An alternative explanation for the relationship between maturity and resistance is proposed by Van Oijen

(1992b), who suggests that early maturity reduces yield loss due to *P. infestans*. Early cultivars escape part of the effect of the fungus by completing a larger proportion of their tuber bulking before foliage death ensues. Consequently, both the intensity and timing of epidemics affect relative yield loss in late versus early cultivars, and should be taken into account in breeding programs.

It is important to minimize oospore production because oospores are the products of sexual reproduction and, therefore, may contain genetic recombinants. Hanson and Shattock (1998) found that more oospores develop on clones with medium levels of race-nonspecific resistance than on susceptible cultivars. They suggest that the tissue of susceptible cultivars dies before oospores can form. In addition, cultivars with high levels of resistance keep oospore formation to a minimum. It is likely that both the levels and modes of nonspecific resistance mechanisms influence the ability of oospores to form in host tissue.

Although little is known about the physiology of tuber resistance, *P. infestans* enters unwounded tubers through eyes and lenticels. Presumably, resistance to penetration and spread is exercised at these sites (Wastie 1991). Seed potatoes infected with *P. infestans* exhibit reduced plant emergence. Plant emergence of resistant cultivars infected with the US-1 genotype is generally greater than that from susceptible cultivars (Platt et al. 1999). However, seed rot is much higher when infected with genotype US-8, so differences among cultivars are reduced. Clulow et al. (1995) suggest that tuber surface colonizing bacteria may be responsible for some differences in tuber resistance among clones. Some of these bacteria had a fungistatic effect on the growth of *P. infestans* in vitro.

2. Cellular. *R*-genes cause a hypersensitive necrotic response in leaf, stem, and tuber cells invaded by the fungus. At least in tuber cells, this response is associated with the formation of phytoalexins (Wastie 1991). In a video microscopy study, Freytag et al. (1994) observed the interaction between a potato clone carrying allele *R1* and *P. infestans* race 1 (compatible interaction) and race 4 (incompatible interaction). Differences between the compatible and incompatible interactions were minor at early stages of attack, when the fungus interacted with epidermal cells. Differences were more pronounced when the fungus penetrated mesophyll cells as more cells responded to fungal attack in the incompatible than in the compatible reaction. However, there were no differences between the compatible and incompatible reactions in the production of the defense-related gene products phenylalanine ammonia-lyase and pathogenesis-related protein 1. In contrast, Constabel and Brisson (1992) found that the defense-related STH-2 protein accumulated faster in

incompatible than compatible reactions, at least at low spore concentrations.

Koch et al. (1994) detected a change in photosynthetic electron transport in compatible, but not incompatible, reactions when detached leaves were challenged with a *P. infestans* spore suspension. The authors suggest that the fungus may be able to activate chloroplast metabolism specifically in compatible reactions, facilitating fungal growth.

Superoxide anion radicals are produced by plant tissues after fungal infection and may function as a resistance component. Ivanova et al. (1991) reported that the pattern of production of superoxide anion radicals in cultivars with high levels of general resistance was similar to that in incompatible reactions.

3. Induced Resistance. Induced late blight resistance was studied with three cultivars by pre-infecting lower leaves with *P. infestans* or with the non-pathogen *P. cryptogea* (Stromberg 1995). Induced resistance was observed in all cultivars using either inducer. Induced resistance by *P. infestans* on the most resistant cultivar, 'Matilda', suppressed penetration into the leaf, slowed growth inside the leaf, and reduced sporulation from lesions. The inducer inoculation probably caused the plant to recognize the fungus and activate its resistance genes. Then, when the second inoculation occurred, the plant had already mobilized its defense mechanisms. 'Matilda' was the best-protected cultivar, possibly because it was the most resistant and was, therefore, able to activate a larger number of resistance genes than the other cultivars in the study.

D. Breeding Strategies

1. Screening and Breeding Methods. Dorrance and Inglis (1997) have summarized published foliar late blight resistance tests. They stress the importance of using a set of known cultivars as standards to measure late blight epidemic potential in each test. Detached leaflet and leaf disk assays are useful for overall measurements of partial resistance. However, their usefulness is limited in screening large segregating populations in breeding programs because they require a large number of replications. The detached leaflet assay has been effectively used to separate germplasm into resistant and susceptible categories.

General resistance results from a complex of many host plant traits that work in combination to reduce the rate of disease spread. Components of resistance are affected by host plant and environmental factors, including leaf surface characteristics, plant growth habit, plant maturity,

canopy density, and canopy structure. These factors affect the level of moisture retention on plant surfaces and the ability of *P. infestans* propagules to reach target tissue (Wastie 1991). For example, incubation of detached leaflets at 10°C, rather than 20°C, tended to produce higher resistance scores (Harrison et al. 1994). Photoperiod and light intensity during growth of intact plants also affected detached leaflet responses to late blight infection. In addition, variation in visual estimates of blight severity can affect resistance scores. Forbes and Korva (1994) determined that precision and accuracy of late blight severity scores were greater when percentage severity was estimated directly than when a Horsfall-Barratt scale was used.

Researchers have attempted to separate resistance into components affecting sporulation and reproduction of the fungus. This component analysis strategy has sometimes been useful in determining mechanisms of resistance and, therefore, allowing the effective design of screening tests. In other cases, though, correlations between these screens and field evaluations have been weak (Wastie 1991). Partial resistance mechanisms include reductions in lesion size, infection efficiency, and sporulation capacity (Dorrance and Inglis 1997). While disease progress is most sensitive to lesion growth rate, genetic variation for infection efficiency is large enough for it to be used equally effectively in a resistance-breeding program (Van Oijen 1992a). Lesion growth rate may be an effective criterion for early generation selection, because it is strongly correlated with AUDPC (Colon et al. 1995b). However, Wastie (1991) suggests that post-infection characteristics, such as rate of fungus growth, rate of necrosis, and sporulation intensity, are too complicated to measure and, therefore, not suitable for screening large populations.

The most valuable resistance components to select in breeding programs appear to be lesion growth rate (Van Oijen 1990, 1992a; Colon et al. 1995a,b), infection efficiency (Van Oijen 1990, 1992a; Canizares and Forbes 1995; Colon et al. 1995a; Singh and Bhattacharyya 1995), lesion size (Birhman and Singh 1995), spore density (Birhman and Singh 1995), latent period (Singh and Bhattacharyya 1995), colonization rate (Singh and Bhattacharyya 1995), and sporulation (Singh and Bhattacharyya 1995). However, infection efficiency and lesion growth rate do not correlate with field performance in many wild genotypes (Colon et al. 1995a).

According to Van Oijen (1990, 1992a), disease progress is improved slightly by efforts to improve two components simultaneously, but the gain is not high enough to warrant breeding for both components. However, Birhman and Singh (1995) found that components of resistance are significantly interrelated and their effects are cumulative throughout an

epidemic. Therefore, they suggest that estimates of relative contributions of individual components to overall resistance probably cannot be made based on studies of a single component.

General combining ability (GCA) for foliar resistance, an estimate of additive gene action, correlates strongly with parental scores (Stewart et al. 1992; Bradshaw et al. 1995). Therefore, both parental phenotype and GCA can be used to select resistant parents for use in a breeding program. At least one parent should be a good general combiner in order to expect a high percentage of resistant seedlings among the offspring (Birhman et al. 1992). Recurrent selection could be effectively used to accumulate genes for foliar resistance. Although Stewart et al. (1992) could find no consistent differences among parents in specific combining ability (SCA), a measure of dominant and epistatic gene action, others have detected variation (Birhman et al. 1992; Bradshaw et al. 1995). Colon et al. (1995c) reported that GCA effects were predominant in *S. tuberosum*-wild species hybrids. Stewart et al. (1994a) detected a high mid-parent-offspring correlation ($r=0.93$, $P=0.02$) for foliar resistance and a moderate, but statistically nonsignificant, one ($r=0.76$, $P>0.10$) for tuber resistance.

Wastie et al. (1993) emphasize the importance of considering both the inheritance of resistance and visual preference when choosing parents for late blight resistance breeding. This reduces the amount of resistance lost during early stages of selection and facilitates the development of cultivars with both disease resistance and horticultural quality.

An understanding of the effect of partial resistance on chemical control strategies will help to reduce chemical inputs while protecting the crop. Shtienberg et al. (1994) found that defoliation in moderately resistant cultivars sprayed on a 14-day spray schedule was not significantly different from that in a susceptible cultivar on a 7-day schedule. Therefore, even partial host plant resistance should allow large reductions in the amount of fungicide applied. This could be accomplished without the risk of increased foliage or tuber infections, and it would decrease pressure on the fungus to develop resistance to fungicides.

Breeders tend to place less emphasis on tuber resistance than foliar resistance. However, rankings of foliage disease severity may differ from rankings of tuber disease severity (Inglis et al. 1996). Moderate correlations between GCAs for foliar resistance and tuber resistance have been reported (Bradshaw et al. 1995; Stewart et al. 1992; Wastie et al. 1993). These correlations are generally not high enough to justify screening progenies for only one type of resistance. Because the control of foliage blight does not guarantee a reduction in tuber blight, it is important to breed for this trait (Wastie 1991).

Components of field resistance to tuber blight include the depth at which tubers are formed, the ability of spores to wash into the soil from the canopy, the ability of periderm to form quickly, and tuber resistance to wounding (Wastie 1991). Evaluation for tuber blight resistance requires a test at harvest to determine the effects of tuber maturity and periderm formation on the expression of resistance, followed by a post-storage test to examine the effects of aging (Dorrance and Inglis 1998). Because the environmental component of variation for tuber resistance is greater than that for foliar resistance, Stewart et al. (1994a) suggest selecting for foliar resistance first and then for tuber resistance.

Platt and Tai (1998) reported that the incidence of late blight tuber rot is not a dependable resistance assay because disease and symptom expression may be sporadic. Instead, post-harvest inoculated tuber rot studies are preferable. Because the authors detected a moderate correlation ($r=0.646$) between foliar disease index and tuber rot, they suggest using tuber rot tests to pre-screen selections for resistance prior to field tests.

Dorrance and Inglis (1998) compared the effectiveness of a tuber slice method with that of a whole tuber inoculation method for tuber blight scoring. General tuber resistance was expressed in whole tubers but not slices of wild species tubers. A high degree of variation within and among individual experiments was observed. Therefore, because of the large number of replicates required for the tuber-slice assay, it is an undesirable method for screening large populations. The whole-tuber assay requires a greater number of tubers than the tuber-slice method, but is a more reliable test.

The expression of tuber resistance traits may cause adaptation by the fungus. Isolates of *P. infestans* from the cultivar Pimpernel caused more infection on tubers of 'Pimpernel' and 'Beate' than did other isolates (Bjor and Mulelid 1991). However, adaptation of the fungus to tuber resistance traits does not necessarily mean adaptation to foliar resistance traits.

Although most breeding programs emphasize general resistance, some *R*-gene breeding efforts continue. An argument has been made for using *R*-gene-containing clones in breeding programs because of a perception that they contribute additive quantitative ("residual") effects with a pyramid effect (Pederson and Leath 1988). However, Ordonez et al. (1998) found no relationship between ineffective *R*-genes and lesion expansion rates in most families studied. Meksem et al. (1995) have identified two RFLP markers flanking *R1*. These markers are useful for marker-assisted selection of the *R1* allele. In addition, they are located in a region of the potato genome to which several other resistance traits

have been mapped, including extreme resistance to PVX, quantitative resistance to *P. infestans*, and resistance to the root cyst nematode *Globodera pallida*. Therefore, they have the potential to be used for selecting other resistance traits.

The most effective and reliable methods for screening germplasm are natural infections or inoculated test plots in the field. Observations on blight intensity are made every few days during an epidemic to determine an overall rating (Wastie 1991), often based on AUDPC. In fact, other tests are usually compared to field results to determine their reliability. However, field trials are influenced by the environment, fungal pathotype, data collection methods, and data analysis procedures (Wastie 1991). For example, although no genotype $\times$ environment interaction was found for AUDPC in the presence of US-8 (Haynes et al. 1998), significant interactions have recently been reported (K. Haynes, pers. comm.). In addition, field-testing requires a site where late blight epidemics occur every year, as well as a *P. infestans* population representative of that where the potato cultivars will eventually be produced (Dorrance and Inglis 1997). A further complication is that the amount of blight on a plant is influenced by that of its neighbors. Because severely infected plants increase blight severity on adjacent plants, resistant cultivars are scored as less resistant in mixtures with other cultivars than in pure stands. Conversely, susceptible cultivars often appear more resistant in mixed stands (Connolly et al. 1995).

Field evaluations of tuber resistance are important because the arrangement of tubers in hills may be a component of resistance (Dorrance and Inglis 1998). Year-to-year fluctuations in timing of infection and distribution of inoculum are high and may lead to escapes. In addition, genotypic and environmental factors (tuber depth, distribution, and orientation in the hill; soil type; severity of the foliar epidemic; timing and amount of rainfall to wash sporangia into the soil, and soil temperature) affect tuber blight severity (Dorrance and Inglis 1998). Therefore, Dorrance and Inglis (1998) were unable to correlate field scores for tuber blight resistance across years.

Although resistance testing in the laboratory is less desirable than field-testing, a careful choice of screening techniques may allow collection of data that reflect field performance. As discussed above, lesion growth rate often correlates with field results. The aggressiveness index tested by Dorrance and Inglis (1997) was more significantly correlated with field results than was the area under the leaf expansion curve. Therefore, the strength of correlation between laboratory and field data may increase as more variables are considered.

Inoculation of whole plants in a greenhouse is a useful technique for inheritance studies, identification of resistance components, and choos-

ing parents for breeding. Genotypes can be placed into distinct categories and classified as highly susceptible, moderately susceptible, moderately resistant, or resistant (Dorrance and Inglis 1997). Ranking of genotypes may be similar under field and greenhouse conditions even though differences in absolute scores exist. For example, Colon et al. (1995a) observed that lesion growth rates and sporulation capacity in the field were lower than in the greenhouse. However, standard genotypes ranked similarly in the field and greenhouse, so both screening methods would lead a researcher to draw the same conclusions. On the other hand, Singh et al. (1997) concluded that a technique to screen seedlings in a greenhouse at the 4–5 week stage is unreliable. The correlation between seedling resistance scored in the greenhouse and adult resistance scored in the field was low (0.19).

Laboratory tests are advantageous because they allow for testing of several genotypes in a short period of time and can be performed regardless of the growing season. A detached leaf/leaflet assay can be useful and produce consistent results (Dorrance and Inglis 1997; Goth and Keane 1997). However, some genotypes do not respond as reliably as others do to a detached leaflet test. This may be because detached leaflets have no transpirational flow and the inoculum droplet remains on the leaflet for 2–3 days. Use of an entire leaf with its petiole may give more reliable results (Miller 1996).

An enzyme-linked immunosorbent assay (ELISA) has been developed to distinguish between hypersensitivity and susceptibility when potato leaflets are challenged with *P. infestans* (Harrison et al. 1991). The technique allows major resistance gene effects to be quantified. However, it is not able to separate the effects of *R*-genes from those of minor resistance genes.

Polymerase chain reaction-based tests for *P. infestans* have been developed for use with tuber tissue (Trout et al. 1997; Tooley et al. 1998). The fungus can be detected as early as 72 h after inoculation, before symptoms develop. The technique allows detection of the fungus at much higher frequencies than with plating on selective medium or moist incubation of tissue. Both the PCR and ELISA techniques provide rapid and accurate identification of a late blight infection, and are comparable in cost and ease of use. However, the PCR method provides a greater level of species specificity.

2. Natural Resistance. Although *R*-genes have been incorporated into many cultivars in recent decades, most potato breeders now prefer to utilize partial or rate-reducing resistance (Dorrance and Inglis 1997). However, this type of resistance is polygenically inherited, so it is much more difficult to incorporate, especially in a polyploid system (Wastie 1991).

Colon et al. (1995a) evaluated four components of resistance in *S. arnezii* × *hondelmannii*, *S. berthaultii*, *S. circaefolium*, *S. leptophytes*, *S. microdontum*, *S. sparsipilum*, *S. sucrense*, *S. vernei*, four hybrid progenies of *S. microdontum* × *S. tuberosum* and four *S. tuberosum* cultivars. In the wild species, components associated with general resistance can be improved through breeding because genetic variation exists for generation time, infection efficiency, lesion growth rate, and sporulation capacity. Genetic variation for sporulation capacity is also relatively large in *S. tuberosum*. In addition, variation among species was found for the relative importance of resistance components. For example, infection efficiency was the main resistance component in *S. berthaultii*, while sporulation capacity seemed to be the most important in *S. tuberosum*. Colon et al. (1995c) reported the expression of several major resistance genes in *S. tuberosum*–wild species hybrids, while Louwes et al. (1992) observed a high level of resistance in *S. tuberosum*–*S. circaefolium* subsp. *circaeifolium* hybrids.

Solanum nigrum (black nightshade) is highly resistant to *P. infestans*. Embryo rescue was used to create a sexual hybrid between *S. nigrum* and susceptible *S. tuberosum* ('Désirée') (Colon et al. 1993). Resistance was transferred as a dominant character to the offspring. In a detached leaf test, resistance was as high as that of the wild parent.

Somatic hybridization has been used to access wild species genes and to combine genes for late blight resistance. Tetraploid somatic hybrids between *S. tuberosum* and *S. circaefolium* subsp. *circaeifolium* were highly resistant to *P. infestans* (Mattheij et al. 1992). The somatic hybrids were sexually compatible with tetraploid clones, but the extent of chromosomal recombination between genomes is unknown. Resistance due to a hypersensitive response has been identified in somatic hybrids between *S. tuberosum* and *S. bulbocastanum*, and their backcross derivatives (Douches et al. 1997). In addition, somatic fusion between *S. tuberosum* haploids and *S. brevidens* produced hybrids that varied for late blight resistance, with some hybrids expressing higher levels of resistance than either fusion parent (Rokka et al. 1994). Late blight resistance in *S. brevidens* + *S. tuberosum* (Helgeson et al. 1993) and *S. bulbocastanum* + *S. tuberosum* (Helgeson et al. 1998) somatic hybrids was transferred sexually to offspring following crosses to cultivars.

Rasmussen et al. (1998) fused protoplasts of five haploid *S. tuberosum* clones with different levels of polygenically inherited resistance, but did not observe simple addition of resistance traits from the parents. In fact, there was considerable loss of quantitative resistance to both foliage and tuber blight in most hybrids compared to the parents. The authors sug-

gest that, in somatic hybrids, genes from one parent may inhibit the expression of resistance genes from the other parent.

3. Transgenic Resistance. Transgenic plants expressing the gene for glucose oxidase (which generates hydrogen peroxide) have been developed by Wu et al. (1997). The development of late blight lesions was significantly delayed in leaves of these plants. Transgenic plants that constitutively overexpressed osmotin (a pathogenesis-related protein) exhibited delayed development of disease symptoms in a growth chamber after inoculation with spore suspensions of *P. infestans* (Liu et al. 1994). Similarly, constitutive expression of osmotin-like proteins increased the tolerance of transgenic plants to *P. infestans*, especially at early phases of fungal infection (Zhu et al. 1996). Strittmatter et al. (1995) developed a system to generate necrotic lesions at fungal infection sites using two genes. The first gene, for the expression of the bacterial ribonuclease barnase, is driven by a promoter that mediates rapid, localized transcription in response to pathogen attack (Martini et al. 1993). The second gene expresses an inhibitor of barnase to reduce the negative effects of background barnase synthesis in the absence of fungal infection. Sporulation of *P. infestans* was reduced dramatically on transgenic plants expressing both genes.

Niederhauser et al. (1996) advocate a multilineal breeding strategy using *R*-genes. Because at least 16 distinct *R*-genes exist in *S. demissum* germplasm, the authors propose incorporation of *R*-genes, singly or in combination, into successful cultivars through gene isolation and transformation techniques. A cultivar would then be grown as a mixture of transgenic clones containing different *R*-genes.

4. In Vitro Breeding. Attempts to identify somaclonal variation for late blight resistance have met with only limited success. Cassells et al. (1991) screened somaclones of 'Bintje' for field resistance to *P. infestans* by comparing AUDPC to 'Bintje' and other reference cultivars. Although clones with improved field resistance were identified, most matured later than 'Bintje'. In addition, most of the resistant variants were unstable. This study underscores the importance of evaluating somaclones for stability, durability and economic traits along with late blight resistance.

In another in vitro study, cells of cultivars were selected using a culture filtrate of *P. infestans* (Cerato et al. 1993). Whole plants and detached leaves of regenerated plants were then screened for resistance. Some improvement in partial resistance was observed when cells were exposed to high concentrations of culture filtrate and screened using a

detached leaf test, but not when whole plants were screened. Not all plants derived from the same callus expressed the same level of resistance. Therefore, the culture filtrate may not have been able to take advantage of spontaneous somaclonal variation. The authors suggest that these results do not clearly demonstrate the usefulness of *P. infestans* culture filtrate for in vitro selection.

III. EARLY BLIGHT

Early blight, caused by the foliar fungus *Alternaria solani* Sorauer, is a significant disease of potato crops around the world. In North America, intensive use of fungicides is necessary to protect susceptible cultivars (Stevenson 1994). Although it can be argued that these fungicide costs are relatively small, there is increasing pressure on growers to reduce pesticide inputs. Consequently, cultivars with a high level of field resistance to early blight may become essential for potato production. The technology and germplasm are available for incorporating field resistance to early blight into cultivars (Stevenson 1994).

A. Germplasm Resources

1. Cultivars and Advanced Selections. Sources of host plant resistance to *A. solani* in *S. tuberosum* are relatively rare (Shtienberg et al. 1995). Moderate resistance has been identified in some cultivars and breeding materials (Platt and Reddin 1994a). Commercial cultivars exhibiting resistance to early blight are usually low yielding and late maturing (Rotem 1994). The cultivar AC Brador is highly resistant to early blight, but is late maturing (Tarn et al. 1995b). 'Russet Nugget' and 'Genesee' are reported to be moderately resistant (Holm et al. 1992) and resistant (Plaisted et al. 1993), respectively, to early blight. According to Stevenson (1994), 'Castile' also displays field resistance to early blight.

Boiteux et al. (1995) evaluated 934 potato cultivars and clones from breeding programs throughout the world in an attempt to find resistance not associated with late maturity. Only 27 clones had lower lesioned leaf areas than the resistant cultivar Aracy. Based on area under the disease progress curve (AUDPC), 22 genotypes were at least as resistant as 'Aracy'. There was a strong correlation between early blight resistance and late maturity. However, some genotypes with field resistance were not late-maturing. The correlation between early blight resistance and late maturity observed in previous studies may be a reflection of the narrow genetic base in potato cultivars.

2. Wild and Cultivated Relatives. Attempts to use *Solanum* species for the development of early blight resistant varieties have been minimal (Pavek and Corsini 1994). Ortiz et al. (1993) suggest that the cultivated diploids Group Phureja and Group Stenotomum are good sources of early blight resistance alleles. Evaluations by NRSP-6 (Bamberg et al. 1994) indicate that resistance exists in 42 *Solanum* species. Resistance was identified in several accessions of *S. acaule*, *S. chacoense*, Group Phureja, and *S. pinnatisectum*.

B. Genetic Control of Resistance

Both additive and non-additive gene action are important for early blight resistance (Brandolini 1992; Gopal 1998). Brandolini (1992) found non-additive gene action to be important for determining resistance in advanced breeding clones. However, Herriott et al. (1990) and Ortiz et al. (1993) reported that additive gene action was important in the diploid germplasm they studied. Although the tuber rot phase of early blight is rarely discussed, Pavek and Corsini (1994) have observed variability among clones for this trait. They suggest that one or more genes, with dosage effects, are responsible for susceptibility.

C. Physiology of Resistance

1. Whole Plant. Clones with early and medium maturity are generally more susceptible to early blight than are late-maturing genotypes (Johanson and Thurston 1990; Christ 1991; Brandolini 1992; Stevenson 1994). Pelletier and Fry (1990) suggest that receptivity of potato leaves (number of lesions per germinated conidium) to *A. solani* in various canopy strata is at least partially responsible for the maturity effect on resistance. According to Johanson and Thurston (1990), apparent immunity in physiologically young leaf tissue is due to inhibition of pathogen development on the host, rather than failure of the fungus to penetrate leaves.

Initial lesions develop at the same time on cultivars, whether they are resistant or susceptible to early blight (Shtienberg and Fry 1990; Johanson and Thurston 1990). In addition, secondary spread of the fungus begins simultaneously in different cultivars (Johanson and Thurston 1990). Therefore, resistance factors must come into play after this time.

2. Cellular. Schlagnhauser et al. (1997) reported that infection with *A. solani* results in the expression of two genes involved in ethylene production by the host plant. The genes are expressed in sequence, so they probably have different signal transduction and gene regulatory

mechanisms. Because ethylene is produced in plants in response to some biotic stresses, these gene products probably contribute to an early blight resistance mechanism.

3. Induced Resistance. Some resistance to *A. solani* can be induced by infection with PVX, at least in laboratory experiments (Kalra et al. 1992; Kalra et al. 1994). The level of PVX-induced resistance to *A. solani* depends on the time interval between viral and fungal inoculations, the viral strain, fungal inoculum concentration, host plant age, and host genotype. Susceptible cultivars exhibited the strongest interaction with the virus. The mechanism of this type of cross protection is not understood, but may be related to an increase in the production of defense compounds by the host plant in response to the virus. The authors suggest that an early blight resistance strategy might be to identify mild or attenuated viral strains that could provide induced early blight resistance without injuring the host. This might provide a second line of defense in resistant cultivars.

D. Breeding Strategies

1. Screening and Breeding Methods. As with most resistance screening, field evaluations are considered to provide the most reliable results. However, there are a number of complications when scoring for early blight resistance in the field. Early blight scores are difficult to interpret unless they are considered in conjunction with yield and maturity data (Stevenson 1994), so resistance trials often involve duplicate sprayed and unsprayed plots. Early blight generally progresses slowly over time, so multiple observations of disease symptoms are necessary to produce an accurate analysis of resistance. In addition, rate of disease progress varies among cultivars and does not necessarily correspond with maturity group. For example, disease progress on 'Castile' (mid-season maturity) is at least as slow as that on 'Russet Burbank' (late maturity). Therefore, comparisons of disease progress on each clone under evaluation are necessary to select genotypes with the slowest rate of disease progress within a maturity group (Stevenson 1994).

Christ (1991) found that ranking of cultivars for early blight resistance depends on the assessment method. The most informative and feasible approach appears to be estimation of percentage of leaf area (from the middle third of the plant canopy) covered with early blight lesions. This technique probably measures infection efficiency, which may be a component of resistance to *A. solani*. The amount of chlorosis on leaves with early blight lesions differed significantly among cultivars and cor-

related strongly with necrotic lesion severity ($r=0.96-0.99$). However, chlorosis was not always associated with early blight.

Greenhouse assessment of early blight resistance can be implemented by breeders (Stewart and Bradshaw 1993; H. E. Stewart et al. 1994b). In a greenhouse test, resistance was identified more effectively in adult plants from tubers than in true seedlings. When seedlings were evaluated, scores for older plants corresponded more closely to field scores than did scores for young seedlings. Selection of highly resistant seedlings was effective, but greenhouse and field scores were more similar to each other when families, rather than individual genotypes, were compared. Breeders could first select ideal parents for crossing and then select resistant seedlings in the progeny. Selected seedlings could then be multiplied and evaluated in the field.

A leaf disk assay has been developed that effectively reproduces the resistance reaction of cultivars with varying levels of susceptibility to early blight (Bussey and Stevenson 1991). High correlations between leaf disk early blight ratings and AUDPC from field trials were obtained when inoculated disks of juvenile leaf tissue were floated on phytohormones (naphthaleneacetic acid or 2,4-dichlorophenoxyacetic acid), which induced senescence. This method is much less time-consuming than field trials, may be less influenced by cultivar maturity than field trials, and is inexpensive. As with any screening test, it should include standards of known high and low susceptibility. This procedure may be especially valuable for breeding programs, because preliminary studies indicate that it can be used to screen for early blight resistance in *Solanum* species grown from true seed. Early generation screening may also be possible with an in vitro plantlet test developed by Lima et al. (1996). Resistant and susceptible genotypes could be identified, but results with a moderately resistant clone were inconsistent.

Gopal (1998) reported that the correlation between progeny means and SCAs was significant, while that between mid-parent resistance values and SCAs was not. Therefore, progeny tests are necessary when breeding for early blight resistance. Brandolini (1992) and Ortiz et al. (1993) also recommend progeny testing, combined with recurrent selection, to improve resistance to *A. solani* in breeding populations.

2. Transgenic Resistance. The gene encoding a strongly antifungal endochitinase from the mycoparasitic fungus *Trichoderma harzianum* was introduced into potato by transformation (Lorito et al. 1998). The fungal gene was expressed at high levels in plant tissues, but did not visibly affect plant growth and development. Selected transgenic lines were highly tolerant or completely resistant to *A. solani*, *Rhizoctonia solani*,

and other pathogens. Resistance was genetically stable and sexually transmitted.

3. In Vitro Breeding. Lynch et al. (1991) used the phytotoxic components of *A. solani* culture filtrate in an attempt to differentiate between resistant and susceptible genotypes in vitro. The culture filtrate test correlated with detached leaflet tests, but not with greenhouse and field tests. The poor correlation with whole plant tests may exist because the toxin effects are only part of a complex disease reaction. However, the significant correlation between detached leaflet tests and the culture filtrate test indicates that toxic metabolites may be useful in screening for at least a component of *A. solani* resistance.

IV. VERTICILLIUM WILT

Potato early dying disease, or *Verticillium* wilt, is caused primarily by the soil-borne fungi *Verticillium dahliae* Kleb. and *V. albo-atrum* Reinke and Berthold. It may cause severe losses of both yield and quality. Seed potato growers suffer further losses as infected plants produce infected and/or infested tubers. Colonization of plant tissues also causes an increase in soil-borne levels of inoculum and, therefore, increased disease potential in potato fields (Mol et al. 1996). *Verticillium* wilt results from planting infested seed or planting clean seed tubers into contaminated soil (Robb et al. 1994).

An important factor that undoubtedly has hindered efforts to control *Verticillium* has been the inability to adequately identify and quantify this pathogen (Robb et al. 1994). It is time-consuming and expensive to evaluate germplasm for *Verticillium* resistance in the field. In addition, disease symptoms may be confused with natural senescence and are influenced by environment. Therefore, a considerable amount of effort has been directed toward the development of efficient resistance screening assays.

The most widely grown commercial potato cultivars are not resistant to *Verticillium*. Vascular colonization is extensive in tolerant cultivars, but wilt symptoms are suppressed and yield reductions are limited to certain environmental conditions (Plasencia and Banttari 1997). However, tolerance is undesirable because it allows *Verticillium* propagules to accumulate in the soil, reducing the performance of future potato crops. Inoculum densities are lower in fields where resistant cultivars have been grown than in those where susceptible cultivars were planted (Davis et al. 1994).

Disease responses and yield losses due to *V. albo-atrum* tend to be more severe than those due to *V. dahliae* (Platt and MacLean 1994; Suganda 1995). In a yield trial, the greatest yield loss (63%) was detected in 'Kennebec' inoculated with *V. albo-atrum*. In contrast, the most severe yield loss due to *V. dahliae* was 45% in 'Russet Norkotah' (Suganda 1995).

In some regions, the root lesion nematode *Pratylenchus penetrans* (Cobb) Filipjev & Schuur. Stekh. interacts with *V. dahliae* to exacerbate disease symptoms (MacGuidwin and Rouse 1990). The involvement of *P. penetrans* in potato early dying disease is important because it results in the expression of disease symptoms even when soil populations of *V. dahliae* are so low that they would not otherwise cause disease (Bowers et al. 1996).

A. Germplasm Resources

1. Cultivars and Advanced Selections. There are no totally resistant cultivars, but some ('Katahdin', 'Targee', and 'Désirée') are tolerant (Sihachakr et al. 1996). Several recently released cultivars and breeding clones have been reported to express some level of resistance to *Verticillium*. 'Gemchip' is moderately late in maturity and resistant to *Verticillium* wilt (Pavek et al. 1991b). 'Frontier Russet', medium-early, is more resistant to *Verticillium* wilt than other early russets (Pavek et al. 1991a). 'Ranger Russet' is more resistant to *Verticillium* wilt than 'Russet Burbank' (Pavek et al. 1992). 'Russet Nugget' (Holm et al. 1992), 'Genesee' (Plaisted et al. 1993), and 'AC Brador' (Tarn et al. 1995b) are also reported to be resistant. 'AC Belmont' is highly resistant to *V. albo-atrum* (Tarn et al. 1995a). Corsini et al. (1990) have developed two russet *S. tuberosum* breeding clones with high levels of *Verticillium* wilt resistance and high yields under medium to long growing seasons. Another breeding clone, B0169-56, is reported to be highly resistant to both *V. albo-atrum* and *V. dahliae* (Goth et al. 1994). High levels of *Verticillium* wilt resistance have also been identified in germplasm release AWN86514-2 (Corsini et al. 1990).

2. Wild and Cultivated Relatives. According to Treadwell (1991), several sources of reasonably well-adapted *Verticillium* resistant germplasm are available, so the use of exotic germplasm is unnecessary for the production of new resistant cultivars. Nevertheless, wild *Solanum* species have been used as sources of *Verticillium* wilt resistance. Concibido et al. (1994) identified resistance to both *V. dahliae* and *V. albo-atrum* in *S. chacoense* and Group Phureja. Resistance to *V. albo-atrum*, but not

V. dahliae, was found in *S. raphanifolium*. Lynch et al. (1997) have reported resistance to *V. albo-atrum* in *S. berthaultii*, *S. chacoense*, and *S. tarijense*. Ochoa (1990) has also reported resistance in *S. tarijense*. Resistance to *Verticillium* wilt has been identified by NRSP-6 in 80 species (Bamberg et al. 1994). Resistant accessions are numerous in Group Andigena, *S. chacoense*, *S. gourlayi*, *S. microdontum*, and Group Phureja.

B. Genetic Control of Resistance

1. Qualitative. Lynch et al. (1997) were the first to describe single gene resistance controlling *Verticillium* wilt in potato. Based on inheritance studies, they determined that a single dominant gene controls resistance in a clone of *S. chacoense*. Hoyos et al. (1993) also suggested that simple inheritance of *Verticillium* wilt resistance exists, based on the observation that the low vascular colonization trait is transmitted to progeny with a fairly high frequency.

Ye (1994) used bulked segregant analysis to map genes in two populations for resistance to *V. dahliae*. The resistance of the two populations was derived from *S. chacoense* and Group Phureja. Two RFLP markers, both located on chromosome VIII, showed polymorphisms between the resistant and susceptible bulks of the *S. chacoense*-derived population but were not linked to resistance in the Group Phureja-derived population. Another marker on chromosome IV was associated with susceptibility in the Group Phureja-derived population. This probe was not polymorphic in the *S. chacoense*-derived population. Therefore, resistance from these two sources appears to be located on different chromosomes and controlled by dominant genes affected by modifying genes.

2. Quantitative. Most genetic studies indicate that resistance to *Verticillium* wilt is polygenic and complex. The most recent report of polygenic resistance, by Concibido et al. (1994), involved the evaluation of offspring from crosses between six diploid wild *Solanum* clones. Distributions were skewed toward resistance, indicating that dominance may be involved in the resistance reaction. An alternative explanation is that the various wild species used in these crosses have evolved different simply inherited resistance mechanisms. Crosses between heterozygotes would then produce complex segregation ratios. Tsrör and Nachmias (1995) also concluded that resistance in the cultivars Désirée, Kondor, and Alpha is not due to major genes. Pavek and Corsini (1994) support an additive gene model for *Verticillium* wilt resistance.

Lynch et al. (1997) reported that visual symptom expression was poorly correlated with the magnitude of stem invasion by *V. albo-atrum*. They emphasized the importance of distinguishing between tolerance and resistance. Tolerance was more common than resistance in the accessions they tested. They suggested that genetic mechanisms for the two disease reaction types are distinct in *S. chacoense*, with complex inheritance for tolerance and simple or complex inheritance for resistance.

C. Physiology of Resistance

1. Whole Plant. Although the mechanism by which *Verticillium* invades potato tissue has been the subject of several studies, information on host plant resistance mechanisms is sparse. A few comparisons between resistant and susceptible cultivars have provided some insight regarding incompatible host-pathogen interactions.

Grafting studies indicate that at least some component of resistance is present in potato roots. Hoyos (1990) observed that when susceptible 'Kennebec' scions were grafted onto resistant 'Reddale' rootstocks, colonization by *V. dahliae* was low in the (resistant) rootstock, but high in the (susceptible) scions. Suganda (1995) also performed grafting studies and observed similar results using *V. albo-atrum*. The number of propagules in both 'Reddale' and 'Kennebec' scions were consistently high, while that of both rootstocks was similar to the respective ungrafted control. Tsrör and Nachmias (1995) reported that when a susceptible scion was grafted onto a resistant rootstock, the number of *V. dahliae* propagules above the graft was approximately 25% that of the reciprocal graft.

Potato stems also influence the resistance reaction. Tsrör and Nachmias (1995) found that susceptible stems dipped in a *V. dahliae* spore solution developed ten times more propagules than those of the resistant genotype. Suganda (1995) reported that cut stems of 'Reddale' placed in a culture filtrate of *V. albo-atrum* exhibited the same degree of wilting as stems placed in a spore suspension. Therefore, mechanisms other than or in addition to vascular colonization may be involved in wilt symptom development. Hoyos (1990) suggested that vascular colonization occurs first and is followed by wilt symptoms.

While screening for *Verticillium* wilt resistance generally involves stems or whole vines, work by Vaughn and Lulai (1991, 1992) indicates that *V. dahliae* also elicits a wound response in tuber tissue. Consequently, even though *Verticillium* infection appears to occur largely through roots, a tuber response does occur as a result of fungal invasion.

2. Cellular. Both biochemical and physical resistance factors seem to be operating in resistant 'Reddale'. Vaughn and Lulai (1991) found that the rapid sealing-off of wounded tissue sites through hypersensitive browning, suberization, and wound periderm formation are essential *V. dahliae* resistance mechanisms. All three processes occurred more rapidly in 'Reddale' than in susceptible 'Kennebec' when they were exposed to the fungus. Maximal hypersensitive cell browning occurred much more quickly than maximal levels of the two other responses. Therefore, hypersensitive cell browning probably gives rapid, short-term protection against fungal penetration until wound periderm and suberin are produced. While these latter events take longer to develop, they provide greater protection.

Vaughn and Lulai (1992) also performed biochemical evaluations of tuber wound-healing responses to elicitors in 'Reddale' and 'Kennebec'. The elicitors consisted of either an autoclaved mycelial extract of *V. dahliae* or arachidonic acid (AA), a fungal component. Healing 'Reddale' tissues that were treated with the elicitors exhibited much more rapid and intense hypersensitive cell browning than did those of 'Kennebec'. Activities of enzymes believed to be involved in resistance reactions (lipoxygenase, LOX; polyphenoloxidase, PPO; and peroxidase, PER) were significantly lower in treated 'Reddale' tissues than in the control. This could have been due to inhibition of the wound response or the prevention of enzyme biosynthesis due to hypersensitive cell death. In contrast, in AA-treated 'Kennebec' tissue, LOX activity increased, while PPO and PER activities decreased compared to the control. The roles of the three enzymes in the resistance reaction are, therefore, unclear.

3. Root Lesion Nematode. While disease symptoms due to the synergistic interaction between *V. dahliae* and the root lesion nematode, *P. penetrans*, are well documented (MacGuidwin and Rouse 1990), the physiological nature of the interaction is not yet understood. Bowers et al. (1996) observed the infection and colonization of 'Superior' potato roots by *V. dahliae* when the roots were jointly exposed to *P. penetrans* and *V. dahliae*. Initial infection and colonization of potato roots by *V. dahliae* occurred in undifferentiated cells at the root tip and in the zone of elongation, and did not appear to be affected by nematode infection. Nematode feeding sites were not spatially related to points of entry of *V. dahliae* into roots. Therefore, feeding injury by nematodes does not appear to provide access points through which *V. dahliae* penetrates root tissue. However, root tips did become infected to a greater extent in the presence of nematodes. Perhaps nematode infection increases the production of root exudates or causes increased root branching. Both sce-

narios would increase the number of root contacts with *V. dahliae* microsclerotia. Powelson and Rowe (1993) also indicate that the fungal-nematode interaction is probably biological, rather than physical. They suggest that nematode feeding may alter a potato plant's physiology in such a way that favors fungal infection and/or colonization.

D. Breeding Strategies

1. Screening and Breeding Methods. It is common for potato breeders to conduct field evaluations of clones for *Verticillium* wilt symptoms. Platt and MacLean (1994) found that yield losses were positively correlated with wilt symptoms caused by either *V. dahliae* or *V. albo-atrum*. Wheeler et al. (1994) reported that, while the incidence of early dying symptom expression did not always reflect yield loss, there was general agreement between the two parameters. However, early dying symptoms are easily confused with normal plant senescence and confounded by other foliar diseases. In addition, breeding programs evaluate clones with a wide maturity range. If a breeder is unfamiliar with the vine-maturing pattern of a clone, it is difficult to distinguish between early dying and natural senescence. In addition, late-maturing susceptible clones may exhibit immature plant resistance. Comparisons between inoculated and uninoculated yield trials may identify valuable genotypes, but they are time-consuming, expensive, and cannot distinguish between tolerant and resistant clones.

Many potato production areas are affected by either *V. albo-atrum* or *V. dahliae*, but some regions contain both fungi. In these areas, it is important to characterize a clone's reaction to both species. Using the culture plate technique following inoculation with both species, Suganda (1995) rarely found high levels of both pathogens in the same tissue. Therefore, resistance should be evaluated in two comparable field sites, each with a high inoculum level of only one species. In his studies, some clones reacted differently to *V. albo-atrum* and *V. dahliae*. While 'Russet Norkotah' was the most susceptible to *V. dahliae*, 'Kennebec' was the most susceptible to *V. albo-atrum*.

One of the most widely used *Verticillium* screening techniques is the colony plate assay developed by Hoyos et al. (1991). They evaluated sap from basal stem sections of plants grown in *V. dahliae*-infested soil for approximately 100 days. Correlations between number of colony forming units (CFU) per ml plant sap and percentage of foliar wilt were high ($r=0.86-0.92$), even though there were differences in absolute numbers of CFU and percentage of foliar wilt across years. However, correlations were low ($r=-0.15-0.24$) in a diverse breeding population (Jansky and

Rouse 2000). Suganda (1995) reported that the correlation between CFU number and percent yield loss was intermediate ($r=0.66$) for both *V. dahliae* and *V. albo-atrum*. A significantly greater number of CFU/ml was recovered in samples taken from the main stem at the ground line than from the center or apex of the plant (Hoyos et al. 1991). A strong correlation ($r=0.93-0.95$) exists between CFU/ml and number of infected stems (Jansky and Rouse 2000).

The colony plate assay is similar to that developed by Davis et al. (1983), except that it uses fresh sap rather than dried stems. While the sap method can be performed more rapidly in the laboratory, it must be done within days of sample collection. Dried stems, on the other hand, can be processed later in the season, after other field operations have been completed. In contrast to PCR-based assays or ELISA procedures, the culture plate method measures the number of *Verticillium* propagules capable of growing. Dead propagules or those with reduced viability will not be detected (Suganda 1995). In a further refinement of the technique, Suganda (1995) developed a medium that can differentiate between *V. dahliae* and *V. albo-atrum* in culture, allowing scoring for both species.

Environmental effects may exert a strong influence on resistance assays. There is often a poor correlation between field and greenhouse tests. Hoyos et al. (1991) reported a low correlation between CFU/ml recovered from field-grown plants and that from greenhouse-grown plants, with more CFU/ml recovered from the field. A better approximation to field results was obtained when large (30 cm) diameter pots were used. There was no correlation between resistance scores in the field and those in greenhouse trials in the study by Ye (1994).

Hoyos et al. (1991) reported stronger environmental effects on vascular colonization scores for 'Russet Burbank' and 'Kennebec' than for 'Reddale'. Under normal temperature conditions, a substantial increase in vascular colonization occurred between 100 and 130 days after planting in 'Kennebec'. The increase in vascular colonization in 'Russet Burbank' during that time period was even more pronounced. Vascular colonization by *V. albo-atrum* in both 'Kennebec' and 'Reddale' was greater when plants were grown at an air temperature of 16°C than 20°C. Wilt symptoms in 'Kennebec', however, were higher when grown at 20°C (Suganda 1995).

Treadwell (1991) used a vascular wilt colonization rating scale to score plates developed by the Hoyos et al. (1991) procedure. It allows many thousands of isolation plates to be scored in a short period of time. Because considerable non-genetic variation in CFU/ml exists, many samples are needed to accurately characterize a genotype. Genotypes are

easily misclassified if a small number of stems is evaluated (Treadwell 1991; Jansky and Rouse 2000). The colonization rating scale is valuable because the accuracy of a genotype score is more dependent on evaluating many samples than on exactly quantifying the number of CFU from each sample. Since stem colonization screening requires a large number of replications to effectively score a clone, it was suggested that the technique be reserved for selected (advanced) genotypes in a breeding program. One breeding strategy would be to evaluate small families for *Verticillium* resistance. Then, crosses that produced a high percentage of resistant clones could be repeated and larger numbers of genotypes grown for selection based on yield and tuber characteristics. Advanced selections from these crosses could then be tested in *Verticillium* trials, with the expectation that many would be resistant. An alternative would be to cross resistant parents with those that have other valuable traits. Then, mild selection could be carried out for tuber traits, followed by selection for *Verticillium* resistance.

Another method of evaluating the degree of vascular colonization is through the use of an indirect competitive ELISA (Sundaram et al. 1991; Plasencia et al. 1996; Plasencia and Bantari 1997). The assay detects four or more nanograms of total fungal protein, and exhibits a linear relationship between total protein and absorbance. The assay is able to distinguish between colonized and non-colonized plants and can differentiate susceptible 'Kennebec' from resistant 'Reddale'. In contrast to the culture plate technique, an instrument is used to quantify results so subjectivity is substantially reduced. In addition, samples can be processed quickly, in 2–4 days instead of two weeks. This is important if retesting is necessary or desired during the growing season. The cost of the assay is half of that for the culture plate technique. Although this assay is not more sensitive or reliable than the culture plate method, it allows for the screening of a larger number of samples at no extra cost. Ye (1994) identified a statistically significant, but low correlation [$r=0.4$ (field) and 0.5 (greenhouse)] between data from the culture plate technique and the ELISA procedure. Suganda (1995) pointed out that the ELISA method assay uses a monoclonal antibody that reacts to both *V. dahliae* and *V. albo-atrum*, so it cannot be used to determine the relative proportion of each species in a stem. In addition, it is not sensitive enough to detect less than 10^4 conidia per milliliter of plant sap. This inoculum level is fairly high and cannot adequately distinguish among genotypes unless they differ widely in their reaction to *Verticillium*.

The PCR-based assay developed by Hu et al. (1993) can be used to detect *Verticillium* infection and is capable of distinguishing between *V. albo-atrum* and *V. dahliae*. This assay uses species-specific primers

generated from differences in base sequence in the internal transcribed spacer regions of ribosomal genes. However, the PCR reaction may be inhibited by natural inhibitors in plant tissues (Suganda 1995).

There are several advantages to developing cultivars with different sources of *Verticillium* resistance on each side of the pedigree. Having resistance in both parents increases the proportion of resistant offspring (Treadwell 1991). This strategy might also reduce inbreeding depression and create highly heterozygous offspring, which is important for yield and vigor. In addition, if the two sources contributed different resistance genes, then a pyramid effect would be created. This might result in a clone with more durable resistance than one containing genes from a single resistance source.

Effective early generation selection for *Verticillium* resistance would enable breeders to efficiently incorporate the trait into potential cultivars. Hoyos et al. (1993) assayed vascular colonization by *V. dahliae* in seedling transplants and identified clones for further tests. However, only 8.6% of the putatively resistant clones selected as seedlings were as resistant as 'Reddale'. A more rigorous first-year test is probably needed to eliminate susceptible seedlings. This would allow the susceptible seedlings to be detected visually. Seedlings with higher levels of resistance could be differentiated using a vascular colonization assay. Corsini and Pavek (1996) found that early screening strictly for resistance tended to eliminate clones with other desirable traits. The authors suggest that large population sizes are needed in order to combine *Verticillium* resistance with other traits. They also emphasize the need to screen for resistance under tuberizing conditions to avoid selecting a large population of late-maturing clones. Late-season vigor appears to be one of the key factors in a potato plant's ability to survive disease pressure long enough to produce a good yield of high-quality tubers. However, it is critical to combine this vigor with normal tuber bulking (Corsini and Pavek 1996).

2. Natural Resistance. *Verticillium* resistance has been reported in interspecific hybrids containing *S. tuberosum* Group Tuberosum and the wild diploid species *S. berthaultii*, *S. bukasovii*, *S. chacoense*, *S. gourlayi*, *S. sparsipilum*, and *S. tarijense* (Jansky and Rouse 2000). Five clones were identified with early maturity, low symptom expression, and low levels of stem colonization.

Jadari et al. (1992) fused haploid *S. tuberosum* with *S. torvum*, a non-tuber-bearing, sexually incompatible species. Although the four somatic hybrids were much less vigorous than their parents, all were resistant

to fungal filtrate. However, their field resistance and sexual compatibility with potato have not been evaluated.

3. Transgenic Resistance. Chang et al. (1993) transferred the coding sequence from a pea disease resistance gene (DRR 49) into 'Shepody'. When grown on *Verticillium*-infested soil, two of the transgenic lines expressing mRNA from that gene had higher tuber yields and larger tuber size than did the nontransformed control.

4. In Vitro Breeding. Sebastiani et al. (1994) screened for *Verticillium* resistance in regenerated plants following callus culture of 'Désirée' stem explants. One clone with apparent resistance was identified when nodal explants were grown on medium containing *V. dahliae* filtrate. This clone did not show symptoms when inoculated and grown in a growth chamber. Nachmias et al. (1990) evaluated the potential for *Verticillium* resistance screening using *V. dahliae* toxin. However, calli from susceptible 'Blanka' and tolerant 'Alpha' and 'Spunta' were not differentially affected by the addition of partially purified *V. dahliae* toxin to the agar. Similarly, there was no dramatic effect of the toxin on protoplasts. The partially purified toxin did reduce the viability of suspension cells from 'Blanka' and 'Russet Burbank' (susceptible), but not those of 'Alpha'. In addition, partially purified toxin killed root tips in root cultures of 'Blanka', while roots of tolerant 'Désirée' and 'Alpha' were not affected. Therefore, the toxin may be at least partially responsible for the stunting of plants in the field. The toxin also differentially affected the growth of tissue culture plantlets. Fresh weight, height, and root development of toxin-treated 'Russet Burbank', but not 'Alpha', plantlets were reduced significantly compared to untreated controls. In contrast, Mohan et al. (1990) found no correlation between the reaction of excised leaves to the acetone precipitate of the toxin and the level of wilt resistance in the field or greenhouse. The authors suggest that the toxin evaluated in this study might not be a pathogenicity factor. Alternatively, wilt resistance in this germplasm may be due to the restriction of colonization of the vascular system by the fungus. The latter hypothesis is supported by a highly significant correlation between the level of wilt susceptibility and the degree of colonization of stem apices (Mohan et al. 1990).

5. Root Lesion Nematode. *Pratylenchus penetrans* (Cobb) Filipjev & Schuurmans Stekhoven can be an important component of the potato early dying disease complex. Synergism between *V. dahliae* and *P.*

penetrans may occur, causing foliar symptoms and reductions in yield greater than would be expected from that caused by the organisms acting separately. The combination of *V. dahliae* and *P. penetrans* reduced yield of 'Russet Burbank' by up to 36% and also reduced specific gravity (MacGuidwin and Rouse 1990). Similar initial population levels of the nematodes and fungus interact differently from year to year, influencing the relationship between joint infection and yield loss.

Microplot studies conducted over three years examined the influence of *V. dahliae* and *P. penetrans* on the *Verticillium* susceptible cultivars 'Kennebec' and 'Superior', and the resistant cultivars 'Reddale' and 'Russette' (Wheeler et al. 1994; Wheeler and Riedel 1994). While *V. dahliae* alone decreased yield of the susceptible cultivars during two of the three years, an interaction between *V. dahliae* and *P. penetrans* resulted in even greater yield losses during one year for 'Kennebec', 'Reddale', and 'Russette'. This interaction resulted in an earlier high incidence of *Verticillium* wilt symptoms. The major effect of the two pathogens on yield and early dying was due to *V. dahliae* alone, followed by the interaction between *V. dahliae* and *P. penetrans*, and then *P. penetrans* alone. It appears, therefore, that screening of potato germplasm for resistance to *Verticillium* in the presence of *P. penetrans* is not necessary, because resistance is still detectable in the presence *V. dahliae* alone. However, results of field evaluations should indicate whether *P. penetrans* was present. If it was, then cultivars resistant to *V. dahliae* alone may be misclassified as partially susceptible.

The effect of *V. dahliae* isolates on disease severity may be influenced by the presence of *P. penetrans*. Botseas and Rowe (1994) observed that isolates of *V. dahliae* from vegetative compatibility group (VCG) 4A were more virulent than those from VCG 4B when roots were infected simultaneously with *P. penetrans*. When plants were grown in soil infested only with *Verticillium*, disease severity caused by the two isolates did not differ.

V. COMMON SCAB

The soil-borne actinomycete *Streptomyces scabies* (Thaxter) causes corky lesions on tuber surfaces. Common scab is a major cause of tuber quality degradation in potato, making it an important trait for resistance breeding. Efforts to date, however, have proceeded with almost no understanding of the physiology and inheritance of resistance (Wastie 1994). For 70 years, attempts have been made to elucidate the mechanism by which the pathogen induces the development of lesions in

potato tubers. Recent progress may result in the development of improved methods for the selection of resistant genotypes.

A. Germplasm Resources

1. Cultivars and Advanced Selections. No commercially significant cultivars are immune to infection by *S. scabies* (Haynes et al. 1997a). Recent reports of clones with resistance to scab include 'Russet Nugget' (Holm et al. 1992), 'Genesee' (Plaisted et al. 1993), 'AC Chaleur' (De Jong et al. 1995b), 'Krantz' (Goth et al. 1995), 'AC Novachip' (De Jong et al. 1995a), 'Ontario' (Haynes et al. 1997a), 'Andover' (Plaisted et al. 1998a) and 'Pike' (Plaisted et al. 1998b). Singh and Singh (1992) screened a collection of 129 clones for resistance to *S. aureofaciens* and identified nine that were highly resistant and 17 moderately resistant.

2. Wild and Cultivated Relatives. Resistance to common scab exists in *S. chacoense*, *S. commersonii*, and *S. yungasense* (Hawkes 1990), as well as some cultivated relatives, such as Group Phureja (DeMaïne et al. 1993).

B. Genetic Control of Resistance

1. Qualitative. Based on progeny of $4x \times 2x$ crosses, Murphy et al. (1995) suggest that resistance results from homozygosity of a dominant allele (Sc_1) at one locus or a homozygous recessive condition ($sc_2 sc_2$) at a second locus, as proposed by Alam (1972). Resistance was transmitted effectively from diploid parents producing pollen by first division or second division restitution. Tetraploid parents were also effective in contributing resistance to progeny of $4x \times 4x$ crosses. The correlation between mid-parents and families was moderately high ($r=0.765$), indicating that selection of parents can be based on their scab resistance.

2. Quantitative. Haynes et al. (1997a) estimated broad sense heritabilities for amount of surface area covered (AI) and type of lesion (LI) caused by *S. scabies* in a tetraploid *S. tuberosum* population. High estimates (0.89 for AI and 0.93 for LI) indicate that breeding efforts will improve scab resistance. Because these broad-sense heritability estimates are high, scab resistance may be simply inherited. For both AI and LI, the genotype $\times$ environment interaction was significant and the genotype $\times$ environment variance was only one-half to one-third that of the variance due to environment. However, disease pressure was high in this study, so under less severe conditions, the genotype $\times$ environment variance

might be greater as the more susceptible genotypes separate into different categories.

C. Physiology of Resistance

1. Whole Plant. Mechanisms of resistance to common scab have not been elucidated. However, at least one component of resistance appears to be associated with the ability of the periderm to prevent the spread of the fungus into the cortex (Wastie 1994).

Keinath and Loria (1991) studied the interaction between *S. scabies* and potato in a greenhouse assay. Three cultivars with different levels of scab resistance were inoculated with four concentrations of each of two strains of *S. scabies*. In most cases, scab severity increased linearly with an increase in $\log_{10}$ of the inoculum, but both cultivar resistance and pathogen virulence also affected it. At high inoculum densities, the moderate resistance in clone NY 64 effectively reduced scab severity. At low densities, however, scab severity was similar to that of susceptible 'Chippewa'. 'Superior' was consistently the most highly resistant clone. Disease severity in all three clones increased with increasing levels of inoculum.

Cultivars with varying levels of scab resistance did not consistently affect the rhizosphere (roots with adhering soil) population of *S. scabies* (Keinath and Loria 1991). The pathogen was also found in the rhizoplane (washed roots), but at much lower levels than in the rhizosphere.

2. Cellular. Characterization of the pathogen's effect on host plants may lead to an understanding of resistance mechanisms. Lawrence et al. (1990) have isolated two phytotoxins, thaxtomin A and thaxtomin B, from scab-inoculated tubers. These appear to be the compounds responsible for the development of lesions on scab-infected tubers. Natsume et al. (1996) also isolated the phytotoxins concanamycins A and B from *Streptomyces*. Bukhalid and Loria (1997) have cloned a putative pathogenicity factor from *S. scabies*. The 9.4-kb DNA fragment allows the nonpathogen *S. lividans* to necrotize and colonize potato tuber slices, producing scab-like symptoms on minitubers.

D. Breeding Strategies

1. Screening and Breeding Methods. Several rating systems have been developed to evaluate scab resistance because the type of lesion and the amount of surface area covered by lesions vary from tuber to tuber within a clone (Haynes et al. 1997a). Goth et al. (1993) used cluster

analysis to compare scab resistance of uncharacterized germplasm with that of cultivars used as disease standards. Under conditions of very poor or very severe scab development, data on proportion of scabby tubers did not discriminate among clones. Cluster analysis on the combination of surface area index (based on surface area infected) and lesion index (based on lesion severity) effectively classified relative resistance of clones in relation to known cultivar standards. This method was determined to be superior to use of an overall scab index based on lesion type and surface area infected.

Scab resistance has been effectively transmitted from diploid parents to tetraploid offspring via $4x \times 2x$ matings (Murphy et al. 1995; Tai et al. 1996). Tai et al. (1996) suggest that selection for scab resistance could be based on either the best individual seedlings or the best families. However, within-family selection of resistant genotypes was found to be an undesirable method for this trait because estimates of response to selection were lowest for this selection method.

VI. SOFT ROT AND BLACKLEG

Gram-negative *Erwinia* bacteria cause soft rot and blackleg diseases in potato. In storage, soft rot can destroy an entire crop. In the field, it can cause significant yield depression. Hide et al. (1996) reported that blackleg reduced yield by 28%, mainly by decreasing tuber number. The three species that affect potato are *E. carotovora* subsp. *carotovora* (Jones) Dye (Ecc), *E. carotovora* subsp. *atroseptica* (van Hall) Dye (Eca), and *E. chrysanthemi* (Burkholder, McFadden, and Dimock) (Ech). These bacteria produce pectic enzymes that macerate plant tissues (Carputo et al. 1996). *Erwinia* species are facultative anaerobes, and potato tissues are most susceptible to *Erwinia* damage under anaerobic conditions (Bulow et al. 1999). Because there is no practical means of chemical control of this pathogen, the development of resistant cultivars is especially important (Bains et al. 1999). No commercially significant cultivar is highly resistant to bacterial soft rot (Dorel et al. 1996), and breeding for resistance to blackleg has also been unsuccessful (Allefs et al. 1996).

A. Germplasm Resources

1. Cultivars and Advanced Selections. Cultivars from Europe and the United States do not contain high levels of resistance (Rabot et al. 1994; Carputo et al. 1996; Bains et al. 1999; Reeves et al. 1999). However, some resistance has been reported. In a study of 14 cultivars, Tzeng et al.

(1990) found 'Butte', 'Russet Burbank', and 'Norgold Russet' to be the most resistant. 'Kennebec' and 'Sebago' are also reported to express resistance to bacterial soft rot development (Sturz and Matheson 1996). Reeves et al. (1999) detected resistance in breeding clones AF 522-5 and AF 1424-7. Wright et al. (1991) reported that many New Zealand breeding lines were more resistant to soft rot caused by *Eca* than were commercial cultivars.

2. Wild and Cultivated Relatives. Several authors (Hawkes 1990; Bamberg et al. 1994; Rousselle-Bourgeois and Priou 1995; Bains et al. 1999) have reported *Erwinia* resistance in *S. chacoense*. Resistance also exists in accessions of *S. bulbocastanum*, *S. demissum*, *S. hjertingii*, *S. leptophytes*, *S. microdontum*, *S. megistacrolobum*, *S. pinnatisectum*, Group Andigena and Group Phureja (Hawkes 1990; Bamberg et al. 1994). Bamberg et al. (1994) report resistance to blackleg in 72 species. Carputo et al. (1996) screened 12 accessions of *Solanum* species and found the most resistant to be in *S. canasense*, *S. multidissectum*, and *S. tarijense*. Levels of resistance varied among accessions within a species. Rousselle-Bourgeois and Priou (1995) reported resistance in Group Andigena, Group Phureja, and *S. vernei*. Resistance was identified in *S. boliviense* and *S. sanctae-rosae* by Bains et al. (1999) and in hybrids between *S. tuberosum* and *S. brevidens* (Zimnoch-Guzowska et al. 1999).

B. Genetic Control of Resistance

1. Qualitative. Because resistance was transferred easily from somatic hybrids to offspring, Bains et al. (1999) suggested that soft rot resistance is simply inherited. The relationship between genes for resistance to tuber soft rot and those for resistance to blackleg has yet to be determined. However, because correlations between blackleg and tuber soft rot are not significant (Allefs et al. 1995a; Carputo et al. 1997), different genes may control resistance in tubers and stems (Tzeng et al. 1990). Although Zimnoch-Guzowska et al. (1999) detected a significant association between tuber and stem resistance, they suggest that the genetic control of resistance in tubers is partially independent of that for stems. On the other hand, resistance could be controlled by the same gene under different regulatory control in stems than in tubers. The genes conferring resistance to *Eca* appear to be the same as those for resistance to *Ecc*, or at least are tightly linked to them. Carputo et al. (1996, 1997) found significant correlations between resistance reactions to the two *Erwinia* subspecies for both blackleg and tuber soft rot.

C. Physiology of Resistance

1. Whole Plant. Although a relationship between tuber specific gravity and incidence of blackleg and tuber soft rot has been suggested, recent studies have found no strong correlation (Wolters and Collins 1995; Carputo et al. 1997; Haynes et al. 1997b). In addition, Tzeng et al. (1990) and Wolters and Collins (1995) could not correlate tuber calcium levels with cultivar rankings for incidence of bacterial soft rot. However, in a multiple linear regression model, tubers were more likely to be resistant if they contained high levels of both calcium and dry matter (Tzeng et al. 1990).

In some situations, resistance may result in part from the ability of endophytic bacteria to influence *Erwinia* growth and disease development (Sturz and Matheson 1996). They observed a negative correlation between populations of *Erwinia*-inhibiting endophytic bacteria and bacterial soft rot development. It is possible, then, that *Erwinia* resistance may sometimes involve the host genotype's predisposition to become colonized by certain beneficial endophytes.

2. Cellular. Anaerobic conditions exacerbate the *Erwinia* disease reaction due to increased synthesis of pectate lyase enzymes that macerate cell walls (Hugouvieux-Cotte-Pattat et al. 1992). In anaerobic environments, the bacterium also alters plant cell membrane integrity. Stewart et al. (1994) observed extensive lignification, starch degradation, and cell wall degradation by *Ecc* under both aerobic and anaerobic conditions. However, extensive membrane degradation occurred only under anaerobic environments.

Pectate lyases appear to play a key role in *Erwinia* pathogenicity and are, therefore, important to consider in studying resistance mechanisms. Although *Ech* appears to grow equally well in tissues of resistant and susceptible clones, the level of pectate lyase activity is significantly lower in resistant tubers due to limited transcription of pectate lyase genes by the bacterium or inhibition of the enzyme (Dorel et al. 1994). In addition, a highly methylated form of pectin, which is difficult to degrade by *Erwinia* pectinases, is found in tubers (Dorel et al. 1996) and stems (Marty et al. 1997) of resistant clones. The pectin content is also higher in cell walls of resistant clones (Marty et al. 1997). A high level of pectin esterification in stem and tuber cell walls was identified in a highly resistant clone (McMillan et al. 1993). The esterified form of pectin is probably more resistant to pectate lyase.

Additional studies of molecular components involved in the resistance reaction have also been initiated. Deighton et al. (1992) have

demonstrated that oxygen is required for a potato tuber to express resistance to Ecc because it is involved in free radical formation, an apparent host response to *Erwinia* challenge. Lopez-Lopez et al. (1995) determined that acetylsalicylic acid (ASA) at low concentrations does not inhibit Ecc growth, but at higher concentrations, total inhibition occurs. It appears that ASA alters host plant metabolism, rather than affecting bacterial viability. Comparisons between compatible and incompatible potato/*Erwinia* interactions led Hindenbrand and Ninne-mann (1994) to conclude that soft rot resistance in tubers may be due, at least in part, to the speed at which phytoalexins accumulate and the concentrations that develop in infected tissue. Following infection with Eca, Abenthum et al. (1995) detected substantial levels of phytoalexins in stems, roots, and stolons. However, a relationship between resistance and phytoalexin levels in whole organs was not detected. Studies of concentrations at sites of infection are underway.

The behavior of *Erwinia* in potato stems has also been studied using serological and plating methods. Using immunofluorescence colony staining (IFC), Van Vuurde et al. (1994) observed colonization of vascular, cortical, and epidermal cells as well as intercellular spaces, following infection with Ecc and Eca. Vascular browning also correlated with infection by Ecc and Eca. However, *Erwinia* bacteria were also found at large distances from regions with significant browning.

3. Induced Resistance. Under anaerobic conditions, wounding of tubers prior to inoculation with Ecc induces resistance to soft rot (Vayda et al. 1992). While the mechanism of this type of resistance is unknown, it is probably related to the wound-induced production of mRNAs, proteins, and compounds associated with host defenses. Cultivars that quickly develop wound-induced resistance are more resistant to soft rot than are those that require a longer period to accumulate defense compounds.

D. Breeding Strategies

1. Screening and Breeding Methods. Several whole tuber tests for soft rot resistance have been developed and tested recently. In a comparison between a point titration test (inoculating tubers with pipette tips) and a mist chamber-bruise test (inoculating at a bruise site), both tests produced similar results and were determined to be effective for resistance screening (Lojkowska and Kelman 1994). The mist-chamber bruise test is easy to use, but requires more tubers than the point titration test. Tzeng et al. (1990) evaluated resistance by immersing tubers in inoculum and determining percent surface area decayed after incubation in a mist

chamber. While the most resistant and susceptible cultivars ranked similarly across years and soil types, relative rankings of intermediate clones shifted. Reeves et al. (1999) suggest that percent weight loss of inoculated tubers is a better measure of resistance than incidence of infection because interaction effects are smaller. Koppel (1993) suggests that no single test is appropriate for measuring soft rot resistance, because each mimics different storage and handling conditions. Therefore, it is probably best to evaluate clones using several methods.

The reliability of the tuber slice test has received mixed reviews. Because of the variability of results, Lojkowska and Kelman (1994) question the acceptability of this test. However, Haynes et al. (1997b) found the method to be acceptable, with the best variables being diameter of macerated tissue and $(\text{initial tuber weight} - \text{weight of tissue after removal of macerated tissue} / \text{initial tuber weight})^{1/2}$. They suggest that the problems associated with variability are due to an inadequate understanding of the sample size necessary to detect differences between treatment means. Allefs et al. (1995b) effectively used the tuber slice assay to evaluate cultivars for resistance to *Erwinia*. Correlation coefficients between the tuber slice and whole tuber assays were 0.71 for Eca and 0.64 for Ecc. Although the site of inoculation (medullary tissue vs. cortex) did not affect cultivar rankings, incubation environment (air vs. anaerobic conditions) did. The authors stress the importance of standardizing oxygen concentration during incubation.

Although previous reports have indicated a relationship between soft rot resistance and both high specific gravity and calcium concentration (Tzeng et al. 1990; Conway et al. 1994), Wolters and Collins (1995) found these correlations to be weak. Therefore, they suggest that selection of clones based on specific gravity or calcium content will not substantially improve soft rot resistance.

It is important for breeders to determine whether it is necessary to score clones for resistance to soft rot and blackleg. However, this question has not been answered unequivocally in the literature. Rabot et al. (1994) suggest that susceptibility to tuber rot generally corresponds with susceptibility to blackleg. Zimnoch-Guzowska et al. (1999) also reported that a positive relationship ($r=0.725$) exists between tuber and stem resistance to *Erwinia*. On the other hand, Arce et al. (1999) and Wu et al. (1995) found no correlation in transgenic plants.

In a large, multi-institutional study, four inoculation methods for measuring blackleg resistance were compared (Gans et al. 1991). They included inoculation with a needle, inoculation with a high-pressure needle-less jet injector, vacuum infiltration, and an inoculum dip of damaged tubers. Both the needle and jet-injector methods were able to

discriminate between cultivars for susceptibility to blackleg symptoms. Infiltration was a less desirable technique because results varied, probably due to differences in lenticel receptivity during inoculation. Jet inoculation may be too severe and needle inoculation not severe enough to measure differences in resistance. A high incidence of blackleg was also observed in water-inoculated controls, presumably because the inoculation procedure caused expression of latent infection. Half-tuber tests were found to be more precise than whole tuber tests in differentiating intermediate levels of resistance (Rabot et al. 1994).

Stem immersion and stem inoculation techniques produce similar results when used to screen for blackleg resistance (Carputo et al. 1996). The stem immersion technique is superior for analyzing large numbers of genotypes, because it is rapid, highly reproducible, and requires little space. However, Rabot et al. (1994) argue that stem tests are not as appropriate as tests using inoculated tubers because they do not allow cultivar resistance to be fully expressed.

Allefs et al. (1996) studied clonal differences in "stem base resistance" to blackleg. The development of blackleg results from rotting mother tuber tissue reaching the points of stem attachment to tubers. Therefore, a partial resistance mechanism is one in which the stem base acts as a barrier against the bacteria in the rotted mother tuber. In this study, only a small amount of the variance for blackleg resistance was due to stem tissue resistance. However, this may simply reflect a small amount of variation among clones for this trait. The most important resistance component appears to be a reduction in mother tuber decay. However, screening for this trait is expensive, time-consuming, and requires a large number of tubers. The authors conclude that stem base resistance to Ech is the most reliable component to select for when breeding for blackleg resistance.

An effective petiole test has been developed for large-scale blackleg resistance screening (Bisht et al. 1993). Inoculation and incubation of leaf petioles with Eca and Ecc produced rankings of five cultivars that agreed with their reported field reactions. The authors suggest that this method could be used for large-scale blackleg screening in wild *Solanum* species and large breeding populations.

The effect of subspecies on *Erwinia* resistance screening results varies among studies. Wolters and Collins (1994) reported that selection of resistant clones was more effective when inoculated with either Ecc or Ech than with Eca. Disease development after inoculation with strains of Eca was not severe enough to distinguish among moderately resistant and resistant clones. The strains of Eca used in their study may have been less virulent than those of Ecc and Ech. However, clones resistant

to one species were also resistant to the other two. Similarly, Carputo et al. (1996) found that genotypes more resistant to Eca were also more resistant to Ecc. Allefs et al. (1995a) reported that an interaction exists between soft rot resistance and *Erwinia* subspecies. On the other hand, Haynes et al. (1997b) did not detect a significant interaction between cultivar and *Erwinia* subspecies. In addition, Zimnoch-Guzowska et al. (1999) found that tuber resistance to Eca was highly correlated with tuber resistance to Ech ($r=0.815$).

Erwinia resistance scores are consistent across strains of subspecies (Wolters and Collins 1994; Allefs et al. 1995a). Clones resistant to one strain of an *Erwinia* species were found to be resistant to a second strain of that species. According to Wolters and Collins (1994), selection for resistance with a specific strain of any of the three subspecies will produce clones that maintain their resistance in areas where different strains and/or species are present.

Kohm and Eggers-Schumacher (1995) used reverse passive haemagglutination to detect four strains of Ecc with a detection limit comparable to ELISA. This technique is reliable, easy to perform, and produces results faster than ELISA does.

The polymerase chain reaction can be used to detect *Erwinia* species (Darrasse et al. 1994; Smid et al. 1995; Frechon et al. 1998). Primers have been developed that are specific for Eca under a wide range of conditions and produce results similar to those from viable counts on a selective medium, the most sensitive alternative method tested (Frechon et al. 1998). A set of primers capable of distinguishing between Eca and other *Erwinia* species has also been created by Smid et al. (1995).

2. Natural Resistance. *Erwinia* resistance from wild species has been transferred into cultivated germplasm. Diploid interspecific hybrids from crosses between Group Tuberosum haploids and *S. canasense*, *S. multidissectum*, and *S. tarijense*, along with tetraploid and hexaploid *S. tuberosum* + *S. commersonii* somatic hybrids, were screened for resistance to blackleg and tuber soft rot caused by Eca and Ecc. In the five families tested, resistance to blackleg was identified in four families and resistance to tuber soft rot was found in three families. One tetraploid somatic hybrid was resistant to both blackleg and tuber soft rot (Carputo et al. 1997). Resistance has also been identified in tubers of *S. tuberosum* + *S. brevidens* somatic hybrids and their sexual progeny (Helgeson et al. 1993; Zimnoch-Guzowska and Lojkowska 1993, 1999; Lojkowska and Kelman 1994; Bains et al. 1999). Resistance in the second backcross of *S. tuberosum* + *S. brevidens* hybrids to *S. tuberosum* was reduced compared to the first backcross generation (Allefs et al. 1995b). *Erwinia*

resistance was not observed in somatic hybrids developed by Rokka et al. (1994) using different *S. brevidens* genotypes.

Some *S. tuberosum* haploids express higher levels of blackleg resistance than the tetraploid clones from which they are derived (Carputo et al. 1996). This is in accordance with Peloquin et al. (1991), who suggest that genetic variability hidden in polyploids can be revealed by haploids.

3. Transgenic Resistance. Martin (1994) described a transgenic system that appears to have some effectiveness against *Erwinia* bacteria. A chimeric construct containing a lysozyme gene and a sequence for a signal peptide was used to transform potato. Lysozyme catalyzes the breakdown of bacterial cell walls, while the signal peptide facilitates the transport of lysozyme into the apoplast where the bacteria reside (During et al. 1993). In laboratory and greenhouse tuber disc assays, transformed potatoes exhibited significantly less tissue maceration than control potatoes following inoculation. Another transgenic approach is the introduction of the gene for glucose oxidase, which generates hydrogen peroxide, a product of plant defense responses (Wu et al. 1995). Transgenic plants produced elevated hydrogen peroxide levels in leaves and tubers, but grew normally. Tubers were highly resistant to soft rot caused by Ecc under both aerobic and anaerobic conditions. The development of lesions caused by *P. infestans* was also significantly delayed in leaves of these plants. Arce et al. (1999) transformed potato with genes encoding either attacin protein or a cecropin analog. Attacins are proteins that alter the permeability of the outer membrane of Gram-negative bacteria. Cecropins are peptides that alter the inner membrane of bacteria, killing both Gram-positive and Gram-negative species. Several of the transgenic clones expressed an increased level of resistance to *Erwinia* infection. Expression of the cecropin analog appears to be more effective than attacin in conferring resistance. In contrast, plants transformed with a gene construct coding for the cecropin B peptide did not produce detectable levels of the peptide, nor were they resistant to Eca or Ecc (Allefs et al. 1995c).

Bulow et al. (1999) have identified a promoter that is active in potato leaves, stems, roots, and tubers under anaerobic conditions. This is especially valuable for the development of transgenic *Erwinia* resistant clones, because promoters that are commonly used (such as the cauliflower mosaic virus 35S promoter) are switched off under anaerobic conditions.

4. In Vitro Breeding. Taylor et al. (1993) screened 25 'Crystal' somaclones for soft rot resistance and found one clone to be significantly more resistant than 'Crystal'. Because a resistant clone was found in a

small population of somaclones, the authors suggest that this technique might be valuable for generating *Erwinia* resistant germplasm.

VII. RING ROT

Bacterial ring rot is caused by the Gram-positive bacterium *Clavibacter michiganensis* subsp. *sepedonicus* (Spieck. & Krotth.) [syn. *Corynebacterium sepedonicum* (Spieck & Krotth.)]. It causes severe damage to foliage and tubers in many cultivars (De Boer and McCann 1990). In an effort to control the bacterium, a zero tolerance policy for import and export of seed potatoes has been established in Canada, the United States, and European countries (Mogen et al. 1990; Xiang and DeBoer 1995; Kawchuk et al. 1998). However, the bacterium has yet to be eradicated. The production of tolerant (symptomless) cultivars is discouraged in the North American seed industry, because they may serve as a source of bacterial inoculum (De Boer and McCann 1990).

A. Germplasm Resources

1. Cultivars and Advanced Selections. Cultivars vary in the degree to which they express symptoms of bacterial ring rot (De Boer and McCann 1990). Cultivars with low levels of symptom expression include 'BelRus', 'Rose Gold', and 'Désirée', but all of these cultivars harbor significant bacterial cell populations following inoculation with *C. m. sepedonicus*.

2. Wild and Cultivated Relatives. In order to avoid the development of symptomless carriers, researchers have attempted to find immunity to the bacterium. Although it does not exist in cultivars, immunity has been identified in wild *Solanum* species. Kurowski and Manzer (1992) evaluated 156 accessions from the United States potato collection for immunity to bacterial ring rot. Although each accession had been rated previously as highly resistant, symptoms were observed in all plants of 57 accessions. Cells of *C. m. sepedonicus* could not be detected in 321 of 1,000 plants tested in the remaining accessions. Some of those plants may be immune. Kriel et al. (1995a) screened 26 accessions of 14 different diploid and tetraploid *Solanum* species and, using indirect immunofluorescence antibody staining, found immunity in nine accessions of tetraploid *S. acaule*. These accessions maintained their immunity during testing over an eight-month period. Immunity was verified in one *S. acaule* clone and also in one *S. tuberosum* Group Phureja clone by Ishimaru et al. (1994).

B. Genetic Control of Resistance

In *S. acaule*, immunity appears to be conferred by dominant alleles at two loci (Kriel et al. 1995b). Phenotypic expression of bacterial ring rot in nonimmune progeny ranged from susceptible to resistant, probably due to minor or modifying genes.

Mapping of the genes responsible for ring rot immunity appears feasible, but has not yet been accomplished. Ishimaru et al. (1994) suggest that markers from RFLPs and randomly amplified polymorphic DNA could be employed to detect polymorphisms around the gene(s) for bacterial ring rot immunity.

C. Physiology of Resistance

1. Whole Plant. Manzer and Kurowski (1992) suggest that large differences in disease reaction are not due to mode of entry of the bacterium. Their conclusion is based on the observation that, even when roots were inoculated considerably later than seed pieces, symptom expression occurred at approximately the same time for each. However, bacterial cell populations were higher and symptom development more pronounced in root-inoculated plants.

2. Cellular. Plant defense responses to *C. m. sepedonicus* toxin have been characterized by Romanenko et al. (1997a, 1997b, 1998). Bacterial toxin added to suspension cultures of 'Lugovskoi' (resistant) and 'Luk'yanovskii' (susceptible) enhanced both peroxidase and phenylalanine ammonia-lyase (PAL) activities in cells of the resistant cultivar, and decreased those of both enzymes in the susceptible cultivar (Romanenko et al. 1997a). In addition, receptors of the ring rot toxin have been detected in cell walls of a susceptible, but not a resistant, cultivar (Romanenko et al. 1997b, 1998).

D. Breeding Strategies

1. Screening and Breeding Methods. There is disagreement regarding the use of foliar versus tuber symptoms when scoring plants for resistance to ring rot. Nelson et al. (1992) suggest that it is important to consider both foliar and tuber symptoms. In their study, correlations between foliar severity and tuber symptom data were significant, but not high enough to adequately predict tuber symptoms based on foliar symptom expression. Kawchuk et al. (1998) did not detect a significant correlation between tuber and foliar symptoms during four years of their 11-year study. On the other hand, Westra and Slack (1994) suggest that, while

tuber symptom expression is not appropriate for resistance scoring because its expression is highly variable among years, foliar symptom expression is constant among cultivars and can be used in all potato growing regions.

Symptom expression, whether in foliage or tubers, may not be appropriate for resistance screening. De Boer and McCann (1990) compared bacterial population densities in stems and tubers of cultivars differing in expression of ring rot symptoms. Although the clones differed significantly in symptom expression, the incidence of latent infection among clones was similar. Therefore, clones that appeared resistant were actually tolerant, because they supported multiplication of the bacterium while remaining symptomless. Similarly, Manzer and Kurowski (1992) compared cell populations of *C. m. sepedonicus* in symptomless cultivars with those that produced symptoms. Some cultivars failed to show symptoms 11 weeks after inoculation, but contained high cell populations only 7 weeks after inoculation. The relationship between cell populations and levels of symptom expression was not always clear.

Immunofluorescence has been widely used to detect ring rot bacteria in potato stems (Gudmestad et al. 1991; De Boer et al. 1992; De Boer et al. 1994; Drennan et al. 1994). However, primers have been created to allow the use of PCR to detect *C. m. sepedonicus* (Firrao 1990; Mirza et al. 1993; Schneider et al. 1993; Drennan et al. 1994; Hogue and Roy 1994; Rademaker and Janse 1994; Karjalainen et al. 1996). In most cases, both techniques produce similar results. However, cross-reactions sometimes give false positive results with immunofluorescence, but clear negative results with the PCR reaction. Ultrasensitive detection of *C. m. sepedonicus* is possible with a nested PCR assay (Lee et al. 1997). This method detects the bacterium at levels that are not easily detected by direct PCR. Kohm and Eggers-Schumacher (1995) used reverse passive haemagglutination to detect *C. m. sepedonicus* with a detection limit comparable to ELISA. This technique is reported to be reliable, easy to perform, and produces results faster than ELISA does.

2. Natural Resistance. Because immunity to bacterial ring rot appears to be simply inherited, it may be relatively easy to transfer resistance to valuable clones in breeding programs. *Solanum acaule* is self-pollinating, so the development of clones homozygous for dominant immunity alleles should be straightforward. These clones would be valuable in breeding programs, because 100% of the offspring from crosses to *S. tuberosum* would be expected to be immune to bacterial ring rot (Kriel et al. 1995b).

VIII. POTATO LEAFROLL VIRUS

Potato leafroll luteovirus (PLRV) is the most important potato viral pathogen worldwide (Huisman et al. 1992; Wilson and Jones 1993a; Valkonen et al. 1998). Yield losses can be significant, and the net necrosis that appears in tubers of infected plants renders them unacceptable for processing. The economic impact of the virus is due to both its effect on yield and the difficulty in preventing its spread to other plants (Barker et al. 1992). The virus is confined to phloem tissues and is transmitted persistently via aphid vectors (Kawchuk et al. 1997). It is also transmitted through infected seed pieces. The virus is controlled using insecticides to kill transmitting insects, and vine killers to prevent infection of tubers.

PLRV is a spherical virus that contains a positive sense single-stranded RNA (Kawchuk et al. 1990). The PLRV coat protein gene encodes a 23 kD protein composed of 208 amino acids (Keese et al. 1990) and contains an internal open reading frame encoding a 17 kDa single-stranded nucleic acid-binding protein (Tacke et al. 1991).

Breeding for resistance to PLRV has been limited by complex genetic control of resistance and difficulty in screening for resistance using field trials (Barker and Woodford 1992). Three forms of resistance are recognized: resistance to infection (I^R), resistance to virus accumulation (A^R), and decreased viral movement from infected foliage to tubers (Wilson and Jones 1992). There is no evidence that resistance to infection is associated with resistance to accumulation (Solomon-Blackburn and Barker 1993).

A. Germplasm Resources

1. Cultivars. While partial resistance to PLRV exists in some cultivars, none are immune to infection (Brown et al. 1997). Resistance to PLRV has been reported in 'Delcora', 'Omega', and 'Whitu' (Wilson and Jones 1993b), as well as 'Bismark' ($I^R A^S$), 'Omega' ($I^R A^R$), 'Spunta' ($I^R A^R$), and 'Delaware' ($I^S A^R$) (Wilson and Jones 1992). Resistance to phloem transport also exists in 'Bismark' (Wilson and Jones 1992). Relatively high resistance to PLRV has been reported in 'AC Brador' (Tarn et al. 1995b).

2. Wild and Cultivated Relatives. Series *Etuberosa* exhibits extremely high resistance to PLRV. Valkonen et al. (1992) found that all 26 accessions of *S. brevidens*, one accession of *S. etuberosum* and one accession of *S. fernandezianum* tested were extremely resistant to PLRV. Resistance to PLRV also exists in series *Tuberosa*. Resistance has been iden-

tified in *S. chacoense* (Brown and Thomas 1994) and Group Phureja (De, Maine et al. 1993). Bamberg et al. (1994) report resistance in 62 species. They indicate that a large number of accessions of *S. acaule*, Group Andigena, *S. demissum*, *S. gourlayi*, and *S. stoloniferum* are resistant.

B. Genetic Control of Resistance

1. Qualitative. Resistance to PLRV is generally considered to be simply inherited. A single dominant gene (R_{LRhc}) controls extreme resistance to PLRV in *S. chacoense* (Brown and Thomas 1994). In contrast, two unlinked dominant complementary genes (Rl_1 , Rl_2) are required for resistance in two *S. tuberosum* clones (Barker et al. 1994b). Minor genes appear to modify expression of resistance in these clones. Swiezynski et al. (1993b) also suggest that resistance genes probably act in a dominant, complementary fashion. Similarly, Brown et al. (1997) observed a high percentage of resistant plants in progenies of highly resistant parents, indicating major gene control of resistance.

2. Quantitative. Quantitative inheritance of resistance appears to be operating in progenies of moderately resistant and susceptible clones (Brown et al. 1997). Wilson and Jones (1993b) also reported that quantitative resistance exists for resistance to PLRV. Narrow sense heritability in advanced breeding material was found to be 0.72, indicating a high level of additive genetic variance for PLRV resistance (Brown et al. 1997).

C. Physiology of Resistance

1. Whole Plant. There appears to be a maturity effect on PLRV resistance. Three cultivars differing in susceptibility to PLRV ('Russet Burbank'—highly susceptible, 'Kennebec'—moderately resistant, and 'Cascade'—resistant) were increasingly resistant to PLRV in the field as plants aged (DiFonzo et al. 1994).

The presence of PVX sometimes significantly increases the accumulation of PLRV in susceptible clones, and this effect is most pronounced in mature leaves of older plants (Wilson and Jones 1993a). However, the presence of PVX does not increase the number of plants that become infected with PLRV.

2. Cellular. Transgenic and host plant resistances that restrict virus multiplication seem to operate by similar mechanisms (Derrick and Barker 1992). In PLRV-susceptible plants, many cells in phloem bundles are

virus-infected. In contrast, PLRV-resistant plants, whether conventionally or transgenically derived, contain fewer virus-infected cells and they are primarily restricted to internal phloem bundles. Therefore, one of the underlying mechanisms in PLRV resistance in primarily infected (Derrick and Barker 1997) and secondarily infected (Derrick and Barker 1992) tissue is a reduction in the number of cells that become infected in external phloem bundles. Intercellular spread of PLRV is under simple genetic control, so a limited number of proteins must be involved in the process (Barker and Solomon 1990). Derrick and Barker (1997) have proposed the following model for the movement of PLRV. In resistant and susceptible plants, the virus moves long distances passively in assimilate flow in sieve elements. A PLRV-encoded protein that can gate plasmodesmata mediates short distance movement between sieve tube elements and companion cells in susceptible clones. Short distance movement in resistant plants is reduced due to ineffective production or function of this protein.

Transgenic plants that produce PLRV coat protein are able to restrict PLRV multiplication even when that protein represents a very small proportion of total leaf protein (Kawchuk et al. 1990; van der Wilk et al. 1991). The translational efficiency of coat protein RNA may be very low or the protein may be unstable, at least outside phloem tissue. If protein levels are low even in phloem, then protection may result from interference of viral replication by the transcript.

D. Breeding Strategies

1. Screening and Breeding Methods. Because PLRV-infected plants do not express strong necrotic symptoms, classification into resistant and susceptible categories is more difficult than with other viruses (Barker et al. 1994b). In addition, symptom severity does not necessarily correlate with PLRV concentrations (Valkonen and Makarainen 1993).

It is common to determine PLRV resistance rankings using field trials in which rows of infected tubers are planted to provide sources of infection, and then the extent of infection in exposed plants is assessed in harvested tubers. These field exposure trials with infector plants detect I^R (resistance to infection) but not A^R (resistance to accumulation) (Wilson and Jones 1993a). Gallo et al. (1994) found that field tests using infector plants provided consistent PLRV resistance ratings despite year-to-year variation in PLRV pressure. Current season infection, based on ELISA of foliage, was detected in less than 2% of the plants and was, therefore, inadequate as a measure of secondary PLRV incidence. Using various clones as PLRV infector plants and neighboring 'Maris Piper' receptor

plants, Barker and Woodford (1992) determined that virus spread is limited severely from clones in which virus accumulation is restricted, whether or not they are resistant to infection.

Assessment of field resistance to PLRV is complicated by factors such as inconsistent infection pressure across environments and variation among clones in symptom expression. In contrast, the assessment of resistance to PLRV multiplication in secondary infection following graft inoculation is relatively straightforward. In a number of clones, the expression of this type of resistance is fairly consistent in different environments (Solomon-Blackburn and Barker 1993).

Resistance to PLRV increases as the season progresses and attempts are being made to quantify these changes. Eventually, action thresholds for green peach aphid (*Myzus persicae* Sulz.) may be modified to reflect the dynamics of PLRV resistance (DiFonzo et al. 1994). Green peach aphid action thresholds for control of PLRV should increase as plants mature.

Field trials are generally more effective than greenhouse evaluations for identifying undesirable plants and observing PLRV disease symptoms (Kawchuk et al. 1997). However, greenhouse assays have been used to classify potato cultivars into PLRV resistance categories corresponding to field resistance ratings. Some clones behave differently in the greenhouse than in the field. For example, 'Cascade' was not as resistant in a greenhouse test as it was in field trials (DiFonzo et al. 1994). The field resistance of 'Cascade' to PLRV is probably too complex to assess in the greenhouse. Greenhouse progeny tests were able to effectively identify families with PLRV resistance, but were not able to identify the most resistant clones within families (Solomon-Blackburn et al. 1994).

2. Natural Resistance. Clones that restrict virus accumulation (A^R) are not necessarily those most resistant to infection in the field (I^R), and there is no association between the two types of resistance. However, both types of resistance are found in some clones. Solomon-Blackburn and Barker (1993) identified a clone that remained virus-free in all field exposure trials and was also the most resistant to PLRV multiplication. The combination of these two types of resistance in cultivars should be especially valuable for eliminating the spread of PLRV.

A combination of rescue pollinations and embryo rescue was used to produce diploid sexual hybrids between diploid series *Tuberosa* clones and diploid non-tuber-bearing series *Etuberosa* clones (Watanabe et al. 1995). Following graft inoculation, some putative hybrids contained undetectable levels of PLRV based on ELISA tests. Resistance was presumably derived from *Etuberosa*.

Host plant resistance can be effective against diverse isolates of PLRV. Clones developed in Scotland with host-mediated resistance to virus multiplication were found to be resistant to a Peruvian isolate of PLRV (Barker 1995). Although the molecular basis of this resistance has not been characterized, it is apparently effective against a broad spectrum of strains.

Resistance to PLRV from *S. brevidens* has been incorporated successfully into *S. tuberosum* germplasm via somatic hybridization (Pehu et al. 1990). Resistance in tetraploid hybrids ranged between that of parental scores and appeared positively related to the extent of expression of *S. brevidens* morphological traits. Hexaploid hybrids tended to be either resistant or susceptible, with resistance found in those clones containing two doses of *S. brevidens* genes.

Triploid anther culture haploids derived from somatic hybrids between two PLRV resistant clones (*S. tuberosum* and PLRV resistant *S. brevidens*) retained the level of resistance that was present in the parents and somatic hybrids (Rokka et al. 1995). Protoplasts of these haploids have been fused with *S. tuberosum* to combine PLRV resistance with tuberization.

3. Transgenic Resistance. Major cultivars with transgenic resistance to PLRV have been created. Some are commercially available and are marketed by Monsanto as NewLeaf Plus® clones (Duncan et al. 1999). Transgenic plants expressing the PLRV coat protein gene exhibit a titer reduction rather than resistance to infection (Barker et al. 1992; Barker et al. 1994a; Kawchuk et al. 1991; van der Wilk et al. 1991; Brown et al. 1995; Presting et al. 1995). Transformation of 'Désirée' with the PLRV coat protein gene conferred resistance independent of the inoculum level (Kawchuk et al. 1990). Furthermore, both positive- and negative-sense transcripts were effective sources of resistance (Kawchuk et al. 1991; van der Wilk et al. 1991). Resistance in transgenic plants probably occurs at an early stage of infection, when low levels of both positive and negative RNA strands of the virus exist. Antisense RNA-mediated resistance is desirable because plants do not commit any energy to translation of the coat protein gene and transencapsidation is not a concern.

Field resistance and reduced tuber symptoms exist in plants expressing the PLRV coat protein cDNA. Thomas et al. (1997) transformed 'Russet Burbank' with two copies of the PLRV coat protein gene, each driven by a different promoter. For most lines, spread of PLRV from an infected plant to adjacent transgenic plants was severely restricted in the field.

Presting et al. (1995) transformed 'Ranger Russet' with three different constructs. One contained the native PLRV coat protein gene. The second contained a modified coat protein gene designed to optimize protein expression. The third contained the vector plasmid without the coat protein gene. All three populations exhibited resistance to PLRV accumulation. Plants with the modified coat protein gene were no more resistant than were those with the native gene. Because resistance was observed following transformation with the vector control, a component of PLRV resistance must be associated with vector DNA or the tissue culture process.

The DNA sequence of the PLRV coat protein gene is somewhat conserved, so coat protein-mediated resistance may be effective against a broad spectrum of strains (Barker 1995). A Scottish coat protein transgene conferred resistance to a Peruvian isolate of PLRV.

Yield reductions in transgenic plants indicate that constitutive expression of PLRV cDNA may be metabolically expensive (Kawchuk et al. 1997). In addition, constitutive expression increases the risk for transencapsidation or viral recombination. Therefore, because PLRV replicates exclusively in phloem, tissue-specific promoters have been used for expression of PLRV coat protein genes (Graham et al. 1997). Virus resistant plants were obtained when the *RolC* promoter (which drives high-level gene expression in phloem tissue, bundle sheath cells, and vascular parenchyma) was used to control expression of the PLRV coat protein gene. In contrast, use of the *Sh* promoter (which expresses genes exclusively in phloem tissue) did not produce PLRV-resistant transgenic plants.

Following regeneration, putative virus-resistant transgenic plants are usually transferred from tissue culture to a greenhouse or growth chamber to be screened for resistance to infection and disease development. To reduce initial screening costs and time, tissue culture methods have been developed for mechanical and graft inoculation (Russo and Slack 1998). They were validated by comparisons with greenhouse screens using putative PVY⁰ replicase-mediated resistant regenerants of 'Atlantic'. Although disease symptoms were not obvious on plants infected in vitro, ELISA detected the virus. Compared to greenhouse testing, in vitro screening required less time and space.

Combining host-mediated resistance with coat protein-mediated resistance substantially reduces PLRV multiplication compared to either type of resistance alone (Barker et al. 1994c). Plants expressing both types of resistance are nearly immune to PLRV, so the spread of virus in the field should be reduced or even eliminated.

4. In Vitro Breeding. Somaclonal variants of 'Russet Burbank' have been identified with improved field resistance to PLRV and reduced tuber disease symptoms (Kawchuk et al. 1997). In field trials, reductions in PLRV infection were similar to those of plants expressing the coat protein gene. Furthermore, in contrast to transgenic lines, the somaclonal variant did not exhibit the yield reduction that is presumably associated with expression of PLRV cDNA.

5. Resistance Breaking. Knowledge of how other pathogens interfere with the expression of genes conferring PLRV resistance is important for the selection of types of resistance to be incorporated and predicting their durability. Salazar et al. (1995) have reported that the presence of potato spindle tuber viroid (PSTVd) can lead to a decrease in the level of resistance to PLRV.

IX. POTATO VIRUS X

Potato virus X potexvirus (PVX) is the most widespread of the potato viruses. It may be latent, with subtle effects on plant foliage and vigor (Xu et al. 1995). The virus is transmitted mechanically and both the virion and viral RNA are infectious.

PVX is a single-stranded plus sense virus, so its RNA functions in translation as a typical eukaryotic mRNA. The PVX genome contains five open reading frames (ORFs). ORF 1 may encode the viral replicase protein; ORFs 2, 3, and 4 are referred to as the triple block genes because their reading frames overlap; ORF 5 encodes the capsid protein (Braun and Hemenway 1992). Dominant major genes for resistance to PVX include those for extreme resistance (*Rx*) and hypersensitivity (*Nb*, *Nx*).

A. Germplasm Resources

Wild *Solanum* species have historically served as a source of PVX resistance genes. Resistance continues to be identified and characterized in this germplasm (Bamberg et al. 1994). For example, Saladrigas et al. (1990) reported that many *S. acaule* genotypes and a few *S. tuberosum* clones contain the gene *Xⁱ*, which confers extreme resistance to most PVX strains. Extreme resistance to infection was identified in an advanced Group Phureja-Group Stenotomum diploid population (Vallejo et al. 1994). Extreme resistance was also found in *S. × chaucha*, *S. × curtilobum*, *S. × juzepczukii*, and *S. vernei* (Querci et al. 1995).

According to Hawkes (1990) PVX resistance is present in *S. acaule*, *S. chacoense*, *S. × curtilobum*, *S. suurense*, *S. tarijense*, *S. sparsipilum*, Group Andigena and Group Phureja. In addition, Valkonen et al. (1992) reported that genes conferring resistance to PVX exist in *S. brevidens* and *S. etuberosum* and are probably different from those currently used by breeders. Pehu et al. (1990) and Gibson et al. (1990) have also reported resistance in *S. brevidens*.

B. Genetic Control of Resistance

Most reports of major gene resistance to PVX are those involving dominant monogenic control. For example, Ortiz et al. (1994) reported that a single dominant gene is responsible for immunity in haploids with resistance derived from *S. stoloniferum*. However, Vallejo et al. (1995) propose that the inheritance of resistance to infection by PVX in Group Phureja-Group Stenotomum diploids is controlled by at least two genes in a dominant type of epistatic interaction. Resistant plants are produced when dominant alleles of genes *X₁* and *X₂* are present. If the dominant allele of one gene is absent, then plants are resistant only when the dominant allele at the other gene is homozygous (Vallejo et al. 1995).

Mapping studies of PVX resistance genes are underway. Resistance genes *Rx1* and *Rx2* have been mapped to the distal end of chromosome XII and the middle of chromosome V, respectively (Ritter et al. 1991). However, Bendahmane et al. (1997) mapped *Rx* to a different location on the distal end of chromosome XII in cultivar 'Cara'. 'Cara' may contain an *Rx* gene other than *Rx1*, or it may possess a chromosomal inversion. The *Nb* gene has also been mapped to the distal end of chromosome V (De Jong et al. 1997). Because *Nb* is very close to *Rx2*, the authors could not exclude the possibility that *Nb* and *Rx2* are alleles of the same gene. The region containing *Nb* and *Rx2* also includes other resistance genes (*R1*, against *P. infestans* and *Gpa*, against the nematode *Globodera pallida*). As discussed in late blight Section IIB1, this clustering of resistance genes may be the result of duplication and divergence events (De Jong et al. 1997). Recently, *Gpa2* was found to be linked tightly to *Rx*, lying approximately 0.02 centiMorgans away (Van der Voort et al. 1999). The authors suggest that the two genes may share common components in their signal-transduction pathways. The gene *Nx_{phu}*, conferring hypersensitive resistance in Group Phureja, has been mapped to the distal end of the long arm of chromosome IX (Tommiska et al. 1998). This region also contains the gene *Sw-5* for resistance to tomato spotted wilt virus in *Lycopersicon peruvianum* (Stevens et al. 1995).

C. Physiology of Resistance

Because resistance breaking events result from changes in the viral coat protein, they are key to understanding interactions between resistance gene products and viral coat proteins. Kohm et al. (1993) reported that *Rx*-mediated resistance depends on an active feature of the PVX coat protein that is absent in the resistance-breaking strain of PVX_{HB}. Therefore, the *Rx* interaction is a protein-mediated, rather than an RNA-mediated effect (Goulden et al. 1997). Similarly, Bendahmane et al. (1995) conclude that the coat protein appears to be the elicitor of *Rx1*-mediated resistance. The recognition phase of *Rx* resistance is highly specific; it is active only in the presence of specific forms of the PVX coat protein. The response phase, in which viral RNA accumulates, does not appear to be virus specific (Bendahmane et al. 1995). The mechanism of recognition of the PVX coat protein seems to be the same for seven *Rx* genes (*Rx*, *Rx_{acl}*, *Rx_{adg}*, *Rx_{cha}*, *Rx_{cur}*, *Rx_{juz}*, and *Rx_{vrn}*) tested by Querci et al. (1995). The viral coat protein has been found to be the major determinant of the host response when *Nx* and *Rx1* confer resistance (Kavanagh et al. 1992; Goulden et al. 1993; Santa Cruz and Baulcombe 1995). Goulden et al. (1993) showed that a threonine residue at position 121 in the coat protein is critical for *Rx*-mediated resistance.

Resistance may also be due to effects on viral transcription, translation, or replication. Based on protoplast studies, Saladrigas et al. (1990) suggest that the gene product of *X_i* (probably = *Rx_{acl}*) restricts transcription or translation of the viral genome. Kohm et al. (1993) observed that *Rx* resistance in protoplasts results in an almost complete suppression of viral RNA accumulation. Genes *Rx_{adg}* and *Rx_{acl}* both inhibit PVX RNA accumulation in infected cells, so *Rx*-mediated resistance is probably due to inhibition of viral replication (Kavanagh et al. 1992). Similarly, Obuchi et al. (1992) reported that resistance in 'Saco' (progeny of an *Rx_{adg}* clone) protoplasts affects virus replication after uncoating. Goulden et al. (1993), however, suggest that the effect of *Rx*-mediated resistance in protoplasts is just one of multiple components. They described a mutant form of PVX that can overcome *Rx*-mediated resistance in protoplasts, but not in intact plants. The mechanism of resistance to PVX in *S. brevidens*, which has not been characterized previously, appears to be restricted cell-to-cell spread of the virus rather than restricted replication (Valkonen et al. 1991).

D. Breeding Strategies

1. Natural Resistance. Valkonen et al. (1995b) used rescue pollination to create hybrids between a *S. tuberosum*-*S. multidissectum* hybrid and

S. brevidens. Although neither parent was resistant to PVX, two hybrids exhibited a hypersensitive resistance response. Either the pollinator parent (IvP35—hypersensitive to PVX) contributed genes to the hybrids, or transgressive or complementary gene action was expressed in the hybrids.

Resistance to PVX from *S. brevidens* has been incorporated successfully into *S. tuberosum* germplasm via somatic hybridization (Pehu et al. 1990). Resistance in tetraploid hybrids ranged between that of the parents and appeared positively related to the extent of expression of *S. brevidens* morphological traits. Hexaploid hybrids tended to be either resistant or susceptible, with resistance found in those clones containing two doses of *S. brevidens* genes, as was also observed with PLRV.

Based on studies of *Gomphrena globosa*, Goulden and Baulcombe (1993) suggest that homologs of *Rx* may exist even in non-hosts for PVX. Therefore, it may be necessary to reevaluate the concept of host range limitation to include the possibility that a plant may be a non-host for a pathogen because it recognizes the products of the pathogen as if they are avirulence determinants. Consequently, "non-hosts" may be valuable sources of resistance genes.

2. Transgenic Resistance. Efforts to develop transgenic plants with PVX resistance have been successful. Transgenic plants express levels of PVX resistance in the field similar to the levels found in plants with major genes for resistance. It appears that transgenic PVX resistant cultivars can be developed while maintaining the qualities of the original clones (Lawson et al. 1990; Jongedijk et al. 1992; Van den Elzen et al. 1993; Xu et al. 1995; Spillane et al. 1998).

Transgenic lines expressing the PVX coat protein gene exhibit delayed symptom development and/or reduced virus accumulation upon primary infection (Kaniowski et al. 1990; Lawson et al. 1990; Spillane et al. 1998) as well as lower frequencies of secondary infection (Jongedijk et al. 1992). In clones that contain genes for both the coat protein and movement protein, resistance appears to be due to the coat protein (Xu et al. 1995). In contrast, expression of a modified PVX movement protein gene was reported to confer resistance to PVX (Seppanen et al. 1997). In this case, the production of dysfunctional movement proteins in transgenic plants probably caused resistance by competing with the wild-type movement protein.

Expression of a gene encoding a component of the mammalian interferon-induced antiviral system (2'-5' oligoadenylate synthetase) conferred resistance to PVX under field conditions (Truve et al. 1993).

Virus concentrations were lower than those of nontransgenic controls and transgenic plants expressing the PVX coat protein gene.

During the production of transgenic clones, virus genes are usually linked to the cauliflower mosaic virus 35S promoter, which provides a high level of constitutive expression. However, disease defense mechanisms, including ethylene biosynthesis, are often induced by virus infection. Feher et al. (1992) used an ethylene-inducible promoter to produce virus-resistant plants expressing a PVX coat protein gene. Ethephon treatment resulted in a substantial increase in coat protein mRNA.

3. Resistance Breaking. The occurrence of resistance-breaking PVX strains is rare, making single gene resistance relatively durable in potato. According to Goulden et al. (1997), two simultaneous mutations are required to convert an *Rx*-sensitive strain into an *Rx*-breaking strain. While this provides a partial explanation for the durability of *Rx* genes, the authors believe that other unidentified factors are also significant. Resistance breaking events have been reported, however, as discussed in Section IXC1. Recently, a resistance-breaking isolate of PVX (PVX^{MS}) was detected in Argentina. This isolate is able to replicate in both resistant (*Rx*) and susceptible genotypes (Tozzini et al. 1994).

X. POTATO VIRUS Y

Potato virus Y potyvirus (PVY) is the second most important viral pathogen of potato, behind potato leafroll luteovirus (Valkonen et al. 1998). Plants are often more severely damaged by PVY than by PLRV (Ross 1986). Yield losses due to PVY may approach 80% (Valkonen et al. 1998). PVY-infected volunteer potato plants or weeds usually exist near potato crops, and aphids may carry the virus to the field (Hämäläinen et al. 1997). Therefore, even if a field is planted with virus-free seed, current-season (primary) infection of PVY can reduce yields (Novy and Helgeson 1994). Furthermore, weak strains can be latent or cause mild symptoms that are difficult to detect (Ross 1986).

The virus is mechanically and aphid-transmitted in a non-persistent manner (Huisman et al. 1992). The RNA of PVY contains one large open reading frame for a putative protein that is cleaved during translation into smaller functional proteins (Huisman et al. 1992).

There are three major groups of strains. Most of the strains are PVY^O (ordinary strains), while other strains are members of groups PVY^N (tobacco vein necrosis strains) or PVY^C (stipple streak strains). Breeders are generally not concerned with PVY^C strains because they are not

insect-transmitted and most cultivars contain a major gene (*Nc*) for resistance to these strains.

Hypersensitive resistance controlled by *Ny* genes is often PVY strain group-specific, but extreme resistance controlled by *Ry* genes is effective against all strains of PVY (Jones 1990). Following infection with PVY, plants expressing *Ny* become necrotic in systemically infected parts. Plants expressing *Ry* generally remain symptomless following infection (Hämäläinen et al. 1997).

A. Germplasm Resources

1. Cultivars and Advanced Selections. Several cultivars contain major genes for PVY resistance. For example, Valkonen and Makarainen (1993) evaluated PVY resistance in cultivars commonly grown in Finland. Hypersensitive resistance was found to exist in 10 of the 13 cultivars tested following infection with PVY^O. However, none of the cultivars exhibited extreme resistance. Cultivars 'Pirola', 'Barbara', and 'Fabal' are immune to replication by PVY (Barker 1996). A high level of resistance to PVY has been reported in 'AC Chaleur' (De Jong et al. 1995b) and the germplasm release AWN86514-2 (Corsini et al. 1999). Moderate resistance exists in 'AC Novachip' (De Jong et al. 1995a), 'AC Belmont' (Tarn et al. 1995a), and 'AC Brador' (Tarn et al. 1995b).

2. Wild and Cultivated Relatives. Extreme resistance to PVY has been identified in several wild *Solanum* species. Genes controlling extreme resistance have been identified in *S. stoloniferum*, *S. hougassii*, and Group Andigena (Swiezynski 1994). Extreme resistance has also been found in 26 accessions of *S. brevidens*, one accession of *S. etuberosum*, and one accession of *S. fernandezianum* (Valkonen et al. 1992), and in an advanced Group Phureja-Group Stenotomum diploid population (Vallejo et al. 1994). Gibson et al. (1990) has also reported resistance in *S. brevidens*. Resistance to PVY exists in Group Phureja (De, Maine et al. 1993) and Valkonen et al. (1995b) suggest that it may be transferred when it is used as a pollinator for embryo rescue studies. PVY resistance has been reported by Bamberg et al. (1994) in 27 species. Numerous resistant accessions exist in *S. chacoense* and *S. stoloniferum*. Resistance to PVY^N has been reported in *S. acaule*, *S. megistacrolobum*, *S. stoloniferum* and Group Andigena (Singh et al. 1994).

Valkonen (1997) also reported several novel forms of resistance to PVY in eight wild potato species. These included hypersensitive resistance to PVY^O in *S. megistacrolobum* and *S. polyadenium* and to PVY^N in *S. stoloniferum*, systemic acquired resistance in *S. chacoense* following

infection with PVY^N, and low temperature expression of hypersensitive resistance to PVY^N in *S. sparsipilum* and *S. suurense* and to PVY^O in 'Pito'.

B. Genetic Control of Resistance

Monogenic dominant *Ry* genes generally confer extreme resistance to PVY. However, genes for extreme resistance in series *Etuberosa* are probably different from those currently used by plant breeders (Valkonen et al. 1992).

Extreme resistance to PVY apparently is not always due to a single dominant gene. Flis (1995) studied segregation for extreme resistance to PVY (derived from *S. stoloniferum*) in progenies derived from crossing extremely resistant clones to clones differing in resistance. A single dominant gene model could explain the extreme resistance observed in some, but not all, families. Extreme resistance may sometimes, therefore, be determined by the cumulative action of several dominant genes, which may in turn be influenced by other genes. Clones with non-parental types of resistance to PVY were also observed in this study. Vallejo et al. (1995) proposed another multiple gene model for PVY resistance. They suggested that resistance to infection by PVY in Group Phureja-Group Stenotomum diploid potatoes is controlled by at least two complementary genes. Clones are susceptible if they are recessive for either gene pair. In another study, based on progeny of somatic hybrids containing *S. etuberosum*, Novy and Helgeson (1994) concluded that the PVY resistances in series *Etuberosa* species are not simply dominant in their expression. A dominant temperature-dependent modifier (*Tdm*) gene has been found to alter the expression of the PVY-induced hypersensitive reaction at higher temperatures, resulting in leaf drop symptoms (Valkonen et al. 1998).

Recent molecular mapping studies have determined the location of two *Ry* genes. Both *Ry_{adg}* (derived from Group Andigena) and *Ry_{sto}* (derived from *S. stoloniferum*) have been mapped to similar locations on the proximal end of chromosome XI (Hämäläinen et al. 1997 and Brigneti et al. 1997, respectively).

The expression of *Ry_{adg}* is epistatic to the expression of *Ny_{adg}* (Valkonen et al. 1994a). Although the location of *Ny_{adg}* is not yet known, it is not linked to *Ry_{adg}* (Hämäläinen et al. 1997). In addition, Hämäläinen et al. (1998) reported that three DNA fragments homologous to a tobacco virus resistance gene co-segregated with *Ry_{adg}*, again indicating evolutionary similarities among diverse resistance genes.

Genes for hypersensitivity to PVY^O are independent of gene *Nc*, which confers hypersensitive resistance to PVY^C (Jones 1990). In addition, the gene for immunity derived from Group Andigena (*Ry_{adg}*) is independent of that for *Ny* (Ortiz et al. 1994).

C. Physiology of Resistance

1. Whole Plant. Plants inoculated late in the growing season are less likely to produce infected tubers than are early-inoculated plants. This phenomenon is coined mature plant resistance, and is probably due to changes in plant metabolism as well as a shortened window of opportunity for the virus to spread to tubers. Gibson (1991) reported that mature plant resistance against PVY^O and PVY^N exists in the cultivars 'King Edward', 'Record', 'Maris Piper', and 'Désirée'.

2. Cellular. Protoplasts have been utilized to determine mechanisms of virus resistance. Hinrichs et al. (1995) determined that *Ry_{sto}*-resistance to PVY^N does not function at the protoplast level because the *Ry* gene does not inhibit PVY multiplication in protoplasts. It appears necessary for *Ry* in the infected cell to interact with surrounding tissue. PVY^O replicates in initially infected cells of *Ry_{sto}* plants and then moves into adjacent cells before the resistance reaction is able to stop viral movement and replication (Hinrichs et al. 1998). Therefore, *Ry_{sto}* does not impede infection of epidermal cells or short-distance movement of PVY from those cells. However, it does limit the movement of PVY in mesophyll tissue, probably through a hypersensitive reaction (Hinrichs et al. 1998).

Transgenic plants can also serve as useful tools for virus resistance studies. RNA-mediated resistance occurs when transgenes expressing untranslatable sense RNAs confer virus resistance. Smith et al. (1995) developed plants with transgenes that expressed different versions of the PVY coat protein open reading frame. Transgenic plants producing either mRNA and coat protein, or only an untranslatable mRNA, were highly resistant. An inverse correlation between transgene transcript accumulation and virus resistance was often observed, whether translatable or untranslatable versions of the PVY coat protein open reading frame were expressed. Most highly resistant lines had multiple transgenes that accumulated RNA and/or protein to low levels. Resistance appears to be due a host cell-mediated response.

Transgenics have also been used to study *S. brevidens* plants that accumulate very low levels of PVY, except in the presence of tobacco

mosaic virus (TMV). Because a virus-coded movement protein mediates cell-to-cell movement of TMV, transgenic *S. brevidens* lines were created to complement a strain of TMV with defective movement functions. PVY accumulation was enhanced in three of the four lines, indicating effective expression of the movement protein. However, enhancement of PVY accumulation seemed to be associated with the effects of tissue culture rather than expression of the movement protein gene (Valkonen et al. 1995a). Valkonen et al. (1991) suggest that the resistance of *S. brevidens* to PVY is due to slow cell-to-cell movement rather than slow virus replication. However, PVY was detected in uninoculated leaves of inoculated plants of *S. brevidens*, so long distance movement may not be affected (Valkonen et al. 1991).

3. Induced Resistance. Infection with PVY^O results in cross-protection against PVY^N (Singh and Singh 1995). Protection was complete in *S. brachycarpum* and partial in *S. tuberosum* cultivars, so the effect depends in part on host genotype. The cross protection in *S. brachycarpum* against PVY^N is heterologous because PVA-infected plants are partially protected. However, this cross-protection is not general, because protection does not occur in plants pre-infected by the nonpotyviruses PVM, PVS, and PVX.

D. Breeding Strategies

1. Screening and Breeding Methods. Several authors have stressed the importance of using test crosses to determine the most suitable parents for virus resistance breeding (Flis 1995; Wastie et al. 1992; Solomon-Blackburn and MacKay 1993). Producing and using parents that are multiplex for resistance genes can increase the efficiency of breeding for dominant major gene resistance to PVY, especially in tetraploids (Wastie et al. 1992). Because hypersensitive resistance is simply inherited as a dominant trait, segregation studies can be used to determine the genetic status of clones. For example, the segregation ratio from the cross 'Desirée' (hypersensitive) × 'Maris Piper' (non-hypersensitive) was close to 1:1, indicating that 'Desirée' is probably simplex for a single dominant hypersensitive resistance gene (Ny_{tbr}) (Jones 1990). 'Pito' was also determined to be simplex for Ny (Valkonen et al. 1998). When multiplex clones are crossed with susceptible parents chosen for other desirable traits, nearly all of their progeny will be resistant. Therefore, virus resistance screening is unnecessary. In addition, the probability of finding a genotype with a complex combination of desirable characteristics, including PVY resistance, is improved (Solomon-Blackburn and MacKay

1993). The value of developing multiplex parents is illustrated by Mendoza et al. (1996). They identified two triplex clones from crosses between a pair of duplex clones. Approximately 96% of the progeny from crosses between these triplex clones and susceptible clones are expected to be immune to PVY.

Flis (1995) recommends the use of PVY^N for evaluating test families. Since this strain quickly reaches a high concentration, the possibility of susceptible escapes is reduced, along with the time needed for assessment. Susceptible cultivars vary in their suitability for use as test cross parents, and their value may also vary for use with different resistant parents. Therefore, Solomon-Blackburn and MacKay (1993) recommend using at least two different susceptible cultivars for test crosses with each resistant parent.

The identification of the chromosomal location of *Ry* genes (Brigneti et al. 1997; Hämäläinen et al. 1997) has provided the first step toward marker-assisted selection for PVY resistance in potato breeding programs. Restriction fragments of chloroplast DNA might also provide markers for the identification of PVY resistant clones (Palilova and Pyko 1997).

2. Natural Resistance. Valkonen et al. (1995b) used rescue pollination to create hybrids between a *S. tuberosum*-*S. multidissectum* hybrid and *S. brevidens* with extreme resistance to PVY. Two of the four hybrids were as resistant as their *S. brevidens* parent. The remaining hybrids were moderately resistant, indicating that epistasis, complementary gene action, or transgressive expression of resistance was operating.

Somatic fusion has been used in an attempt to combine resistance genes from diverse backgrounds. A high level of resistance has been obtained in *S. tuberosum* + *S. brevidens* somatic hybrids (Pehu et al. 1990; Rokka et al. 1994; Valkonen et al. 1994b). However, somatic hybrids produced from two PVY resistant clones, *S. brevidens* (2x) and 'Pentland Dell', did not express the extreme resistance of the *S. brevidens* parent (Valkonen and Rokka 1998). The authors suggest that factors responsible for mediating virus transport in 'Pentland Dell' may compete with those of *S. brevidens* in the hybrids, resulting in enhanced virus transport and increased virus levels in leaves. In addition, *S. brevidens* genes interfered with the expression of Ny contributed by 'Pentland Dell', because limited necrosis developed in hybrids infected with PVY. This study demonstrates that the expression of virus resistance in somatic hybrids combining two different resistance mechanisms may be different from that of the parental genotypes.

Novy and Helgeson (1994) created somatic hybrids between a PVY resistant *S. etuberosum* clone and a susceptible diploid clone. Although

most hybrids were significantly more resistant than their susceptible parents, none were as resistant as the *S. etuberosum* parent. Following crosses of the hybrids to cultivars, progeny were neither as resistant as the somatic hybrid parent, nor as susceptible as the cultivar parent.

3. Transgenic Resistance. The development of transgenic plants with PVY resistance has been approached through the expression of viral coat protein genes. Major cultivars with transgenic resistance are now commercially available and are marketed by Monsanto as NewLeaf Y® clones (Duncan et al. 1999). The PVY coat protein appears to suppress initial infection by the virus, rather than reducing its spread and multiplication (Lawson et al. 1990). Transgenic plants expressing the PVY coat protein gene are phenotypically normal (Lawson et al. 1990). Okamoto et al. (1996) created transgenic plants expressing the coat protein gene of PVY^N. Four clones were completely resistant to PVY and two of those clones contained only one copy of the transgene. In this example, resistance is probably operating through an RNA-degrading mechanism. Malnoe et al. (1994) also created transgenic plants carrying the PVY^N coat protein gene. A clone with two copies of this coat protein gene expressed complete resistance to a PVY^N strain and good resistance to a PVY^O strain. Coat protein-mediated resistance in this clone also inhibited the spread of the virus through the tubers.

In a variation of coat protein-mediated resistance, the lettuce mosaic virus coat protein gene was inserted into 'Spunta' and 'Claustar' to test for the possibility of heterologous protection against PVY (Hassairi et al. 1998). Immunity to PVY^O was observed in four clones. In addition, two clones exhibited partial resistance as a delay in symptom expression. There was no correlation between immunity and the level of transgene RNA/protein. Although the expression of coat protein genes can be achieved by adding an AUG translation start codon to the coat protein sequence, Wefels et al. (1993) were able to induce expression of the capsid protein using a protease to release it from the precursor protein. Transgenic plants resistant to PVY^N, due to expression of the coat protein gene for that strain, contain undetectable levels of the coat protein (Farinelli et al. 1992). However, the PVY^N coat protein accumulated in those plants when they were infected with PVY^O. Heterologous encapsidation of PVY^O RNA by the PVY^N coat protein was detected. The authors suggest engineering resistance using truncated coat protein transgenes to avoid transencapsidation events.

As an alternative to coat protein-mediated resistance, Pehu et al. (1995) transformed 'Pito' with the non-structural PVY gene *P1*. A high

level of resistance to systemic infection by PVY^O was observed in these transgenic plants.

XI. MULTIPLE VIRUS RESISTANCE

A potato crop is often infected by more than one virus, and disease severity usually increases with multiple virus infections. For example, rugose mosaic symptoms result when plants are infected with both PVX and PVY. These plants exhibit a synergistic increase in disease symptoms. Therefore, breeders are interested in developing clones with resistance to multiple viruses. It has been difficult, however, to combine resistance to PLRV, PVX, and PVY (Ross 1986).

A. Breeding Strategies

1. Natural Resistance. Barker (1997) suggests that *Ry_{sto}* (from *S. stoloniferum*) is responsible for broad-spectrum resistance to several potyviruses (PVY, PVA, and PVV) or it is actually a family of closely linked genes for resistance to each virus. Similarly, a single gene in 'Pirola' and 'Barbara' appears to confer resistance to both PVY and PVA (Barker 1996).

Expression of PVX resistance does not appear to affect the expression of PVY resistance. Somatic hybrids between clones containing dominant genes for extreme resistance to PVX and PVY generally express resistance to both viruses (Thach et al. 1993). However, expression of PLRV resistance may require the presence of extreme resistance to PVX and PVY. Brandolini et al. (1992) found that susceptibility to PLRV infection increased when plants were also infected with PVX and PVY.

Series *Etuberosa* appears to be especially valuable as a source of multiple virus resistance (Valkonen et al. 1992). Clones of *S. brevidens* are often resistant to PLRV, PVY, and PVX, indicating a possible common resistance mechanism (Gibson et al. 1990; Pehu et al. 1990). Furthermore, somatic hybrids between *S. brevidens* and *S. tuberosum* tend to be either resistant or susceptible to all three viruses, indicating a possible genetic linkage or a common genetic basis for resistance (Pehu et al. 1990). Although Valkonen et al. (1994b) agree that genes for PVX resistance are linked to those for PVY resistance in *S. brevidens*, they suggest that resistance to PLRV is under different genetic control.

Corsini et al. (1994) have been successful in combining PLRV, PVY, and PVX resistance, using germplasm from Group Andigena and from complex hybrids containing wild *Solanum* germplasm. In addition, they found that the cultivars 'Abnaki', 'Bzura', 'Granola', and 'Pilica' have resistance to PLRV along with some degree of resistance to PVY and/or PVX.

2. Transgenic Resistance. Transgenic 'Russet Burbank' plants containing genes for both PVY and PVX coat proteins expressed both genes (Lawson et al. 1990). However, PVY coat protein levels were lower than those for PVX. Plants became infected with PVX only when they were co-infected with PVY, so it appears that the PVX coat protein may be interacting with PVY. Kaniewski et al. (1990) have also developed a transgenic clone with a high level of resistance to PVX and PVY due to the expression of both coat protein genes. A single gene construct that confers resistance to PLRV, PVY, and PVX has also been developed (Tacke et al. 1996). This construct contains a mutant allele of the PLRV gene *ORF4* that encodes a 17-kD protein (pr17). This protein is localized to the plasmodesmata of sieve element-companion cell complexes and is believed to interfere with systemic spread of viruses by inhibiting movement between cells.

XII. SUMMARY

The potato is an intensively managed crop in most parts of the world. Although resistance breeding has been practiced for well over a century, heavy inputs of pesticides are still required to realize the yield potential of current cultivars. A number of constraints to resistance breeding have prevented the development of successful cultivars with high levels of multiple disease resistance traits. These include the polyploid nature of the crop, the need to incorporate other quality traits, a lack of understanding of inheritance of resistance, inadequate resistance screening methods for large populations, and insufficient knowledge of the effects of interactions among resistance mechanisms. In addition, established cultivars are difficult to displace in a conservative industry.

In recent years, efforts to improve the efficiency of disease resistance breeding in potato have paid large dividends. Several resistance genes have been mapped and can be used for marker-assisted selection. Quantitative trait loci have also been identified, allowing selection for potentially more durable forms of resistance. Wild relatives of potato have been screened for resistance traits, so breeders now know where to concentrate their efforts. Improved methods of resistance screening have been developed, using in vitro and greenhouse techniques to evaluate large segregating populations. The use of PCR and ELISA provide objective data to supplement visual scores of disease reactions. Finally, the development of transgenic plants has provided access to novel forms of resistance that can complement resistance genes present in the genus *Solanum*.

Numerous breeding strategies have been utilized for the development of disease resistant clones. Resistance genes from many wild and cultivated relatives have been readily incorporated into advanced breeding material using ploidy manipulations. In addition, somatic fusion and embryo rescue techniques have been successful in accessing resistance genes in species that are not sexually compatible with *S. tuberosum*. However, somaclonal variants have not been significant sources of disease resistant germplasm. Recent reports of successful mutation breeding for resistance are also lacking. Finally, transgenic clones are becoming significant, especially in the virus resistance arena. However, a lack of public acceptance of transgenic plants appears to be a major constraint to their use in some countries.

Although recent progress in potato disease resistance breeding has been impressive, the real payoff is yet to come. In the future, we should see the release of cultivars with significant levels of resistance to multiple diseases. If these traits can be combined with the quality characteristics that are required for success, then major reductions in pesticide use for potato production should be feasible.

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